THE NUTRITIONAL STATUS OF PREGNANT WOMEN IN RELATION TO ALCOHOL CONSUMPTION DURING PREGNANCY, AND PREGNANCY OUTCOME

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Confidentiality: Grade A

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DECLARATION OF AUTHENTICITY

I, Ingrid Klinger, hereby declare that the work contained in this thesis is my own original work and that I have not previously, in its entirety or in part, submitted it at any university for a degree.

Signature: Date:

SUMMARY

Introduction: Heavy alcohol consumption during pregnancy is teratogenic.[49-51] A woman's nutritional requirements increase during pregnancy.[4] The dangers of heavy drinking in the presence of malnutrition may put the fetus at a further disadvantage to normal development and life.

Objectives: To determine the nutritional status of pregnant women in relation to alcohol consumption during pregnancy, and pregnancy outcome. To relate the combined effect of maternal alcohol consumption and nutritional status to pregnancy outcome.

Study design: Prospective, longitudinal and cohort.

Study population: Pregnant women attending Hanover Park MOU for pre-natal care. They were classified as subjects (heavy drinkers) or controls (light drinkers or abstainers), and 15 pairs were matched according to race, parity and gestational age at the onset of their participation in the study.

Methodology: A skilled FARR worker determined the alcohol consumption of the individuals through a validated questionnaire, whilst the investigator gathered the study data blinded to the participants' alcohol consumption status. The investigator conducted three interviews with the pregnant individuals. Anthropometrical, clinical and biochemical investigations were done and questionnaires completed to determine dietary intake, socio-demographics, health and eating habits.

An experienced FARR paediatrician examined the newborns, assessing their anthropometric status, health and the presence of any alcohol-related signs.

Results: The nutritional status of the matched subjects and controls did not differ significantly in terms of dietary intake, anthropometric or clinical assessment. There were significant differences between the 2 groups' serum vitamin A values ($p<0.0097$). Significant associations were found between the mother and newborn data; specifically energy intake and gestational age at birth ($p<0.0083$), MUAC and birth weight ($p<0.04$), and weight gain and

weight for age ($p<0.0056$). The participants' energy intake also had a significant correlation with their weight gain during pregnancy ($r=0.0389$, $p<0.01$). The prevalence of FAS in the total population was 6.67%; a finding that confirms previously reported data in nearby Wellington, Western Cape.[49]

Conclusion: Some mothers' good nutritional status did not protect their offspring against alcohol's teratogenic effects. Nutritional status did have a few statistical significant effects on pregnancy. However, the investigator is of the opinion that the few significant findings were not enough to accept or reject the hypothesis; therefore, making the results inconclusive.

OPSOMMING

Inleiding: Swaar alkoholgebruik tydens swangerskap is teratogenies.[49-51] Vroue se voedingsbehoeftes verhoog met swangerskap.[4] Die gevare van swaar alkoholgebruik in die teenwoordigheid van wanvoeding mag die fetus verhoed om normal te ontwikkel en groei.

Doelwitte: Om die voedingstatus van swanger vroue te bepaal in verhouding met alkoholgebruik tydens swangerskap, en die swangerskapsuitkoms. Asook om die gekombineerde effek van die moeder se voedingstatus en alkoholgebruik op haar swangerskapsuitkoms te bepaal.

Studie-ontwerp: Prospektief, longitudinaal en kohort.

Studiepopulasie: Swanger vroue wat Hanover Park MOU besoek vir voorgeboorte sorg. Hulle is of as toetslinge (swaar drinkers) of as kontroles (ligte drinkers of geheelonthouers) geklassifiseer, en 15 pare is gepaar na aanleiding van ras, pariteit en gestasionele ouderdom by die aanvang van deelname aan die studie.

Metodologie: ‘n Ervare SAVN lid het die individue se alkoholgebruik bepaal deur middel van ‘n gevalideerde vraelys. Die navorser was geblind vir die individue se alkoholgebruik. Die navorser het drie onderhoude gevoer met elke individu. Antropometriese, kliniese en biochemiese ondersoeke is gedoen. Vraelyste is voltooi om dieetinname, gesondheid, sosio-demografiese en eetgewoonte- inligting te versamel.

‘n Ervare SAVN pediater het alle pasgeborenes ondersoek om hulle antropometriese status, gesondheid en die teenwoordigheid van enige alkohol-verwante tekens te bepaal.

Resultate: Die voedingstatus van die gepaarde toets-en kontrolegroepe het nie beduidend verskil in terme van dieetinname, antropometriese of kliniese evaluering nie. Daar was ‘n beduidende verskil tussen die 2 groepe se serum vitamien A vlakke ($p<0.0097$). Beduidende assosiasies is gevind tussen die moeder en pasgebore se data; naamlike tussen energie-inname en gestasionele ouderdom by geboorte ($p<0.0083$), bo-armomtrek en geboortegewig ($p<0.0056$), en gewigstoename tydens swangerskap en die baba se gewig vir ouderdom

($p<0.0056$). Die deelnemers se energie-inname het ook ‘n beduidende positiewe korrelasie met hul gewigstoename tydens swangerskap gehad ($r=0.0389$, $p<0.01$). Die prevalensie van FAS in die totale populasie was 6.67%; wat ook gevind is onlangs in die nabygeleë Wellington, Weskaap.[49]

Gevolgtrekking: Sommige moeders se goeie voedingstatus het nie hul kinders teen alkohol se teratogeniese effekte beskerm nie. Voedingstatus se effek op swangerskapsuitkoms was statisties beduidend in ‘n paar gevalle. Tog is die navorser van mening dat daar nie genoeg beduidende bewyse is om die nulhipotese te aanvaar of verwerp nie; dus is die gevolgtrekking onopgelos.

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LIST OF ABBREVIATIONS

ADA	American Dietetic Association
ADH	Alcohol dehydrogenase
ADH2	Alcohol dehydrogenase-2
ADH2*2	Alcohol dehydrogenase 2*2
ARBD	Alcohol related birth defects
ARND	Alcohol related neurological defects
BAC's	Blood alcohol concentrations
BMI	Body mass index
CNS	Central nervous system
CV	Coefficiant of variation
DHA	Docosahexanoic acid
EDD	Expected delivery date
EFA's	Essential fatty acids
EPA	Eicosapentanoic acid
FAE	Fetal Alcohol Effects
FARR	Foundation for Alcohol Related Research
FAS	Fetal Alcohol Syndrome
FBC	Full blood count
FFQ	Food frequency questionnaire
FGR	Fetal growth restriction/retardation
GFR	Glomerular filtration rate
GIT	Gastro-intestinal tract
Hb	Haemoglobin
HC	Head circumference
HC/A	Head circumference for age
HCT	Haematocrit
ID	Iron deficiency
IDA	Iron deficiency anaemia
IOM	Institute of Medicine
IUGR	Intra-uterine growth restriction/retardation
LBW	Low birth weight

MEOS	Microsomal ethanol-oxidising system
MOU	Midwifes and obstetrics unit
MT	Metallothionein
MUAC	Mid-upper arm circumference
n	Number
NTD	Neural tube defects
PAWC	Provincial Administration of the Western Cape
PIH	Pregnancy induced hypertension
PPW	Pre-pregnancy weight
q1-q3	Interquartile range
RDA	Recommended dietary allowance
RTHC	Road to health chart
SAVN	*Stigting vir Alkohol-verwante Navorsing*
SGA	Small for gestational age
SOD	Superoxide dismutase
Std Dev	Standard deviation
TB	Tuberculosis
UCT	University of Cape Town
W/A	Weight for age
WHO	World Health Organization

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1. INTRODUCTION

1.1 The importance of a good maternal nutritional status regarding pregnancy outcome

Throughout history it had been found that the incidence of miscarriages, still births, neonatal deaths, malformations and low birth weight (LBW) all increased in infants conceived during famine.[1] Maternal nutritional status is therefore an important predictor of infant birth weight. LBW is a major factor in infant deaths and long-term health problems such as developmental disabilities and learning disorders as determined in the U.S.A.[2,3]

Fortunately, both pre-pregnancy nutritional status and weight gain during pregnancy (which both affect birth weight) are factors that can be modified to improve pregnancy outcome.[4,5] Of the weight gained during pregnancy less than half comprises of the fetus, amniotic fluid and the placenta. It has been found that maternal size predicts placenta size. In turn, placental size determines the amount of nutrition given to the fetus. Exchange of gasses and removal of waste products also take place through the placenta.[4] The rest of the weight gained consists of maternal reproductive tissues, fluid, blood and stores. The increase of blood volume serves to meet the needs of oxygen and nutrient transport to the fetus and maternal tissues.[6] Uterine muscle growth takes place to facilitate delivery, and breast tissue growth for optimal lactation.[7] The stores accumulated consist mostly of fat, which acts as an energy reserve during pregnancy and thereafter.[4] After pregnancy, the fat stores are used for lactation and energy for the mother since the newborn's demands on her make it difficult to take in adequate nutrition.[8]

Therefore, a good pre-pregnancy nutritional status and adequate weight gain during pregnancy is essential for the proper development and growth of the fetus, and to accommodate the mother's needs.[3]

1.1.1 The effect of maternal malnutrition on pregnancy outcome

The Second World War provided some of the first scientific evidence that nutrition influences reproduction outcome. Retrospective studies done in Holland, Leningrad and Japan showed that famine was associated with a dramatic drop in fertility. In Holland specifically, 50% of women of childbearing age developed amenorrhoea, and the national birth rate dropped by 53%.[9] A recent retrospective report found that people who were exposed in utero (in the 1st trimester) to the Dutch famine, had stunted brain development that could be a risk factor for developing schizophrenia.[10]

Maternal malnutrition leads to decreased expansion of blood volume, which causes poor placental perfusion. This impairs the development of the placenta, which is the pipeline for nutrients from the mother to the fetus. These changes may be more important in the etiology of fetal growth restriction (FGR) than the decreased nutrient availability due to the mother's lacking diet.[8]

Epidemiological studies conducted mostly in the United Kingdom (U.K.) indicate that maternal malnutrition may predispose the offspring to chronic lifestyle diseases, such as hypertension, Diabetes mellitus (DM), ischaemic heart disease and hypercholesterolemia.[3,11] DJP Barker and co-workers, using past databases such as national birth registers, neonatal and infant mortality rates, have documented associations between these figures and more present day rates of mortality from ischaemic heart disease, stroke and chronic bronchitis. These studies suggested possible relations between early influences during pregnancy and later diseases, which could be explained by "programming": an early stimulus or insult, operating during a sensitive or critical period, results in long-term changes in the structure or function of specific organs. The "insult" in question was poor maternal health and physique, which is in turn related to malnutrition and/or disease.[11]

Barker and his team found that the death rate from ischaemic heart disease fell with increasing birth weight, suggesting that maternal factors (which determine birth weight) are related to the later risk of ischaemic heart disease. There are various risk factors for ischaemic heart disease, such as hypertension, high plasma levels of clotting factors such as fibrinogen and factor VII, android fat distribution and DM, which were all investigated individually.[11]

Individuals with lower birth weight were more likely as adults to have increased blood pressure or hypertension. The possible mechanism behind this association is thought to be a rise in the pressure in the fetal circulation in order to maintain placental perfusion (an inverse relationship was found between lower birth weight and higher placental weight), which is maintained after birth.[11]

Lower birth weight was also associated with later glucose intolerance or DM. The Barker group proposes that individuals with impaired fetal growth had impairment of growth of the pancreatic islet cells, and therefore a reduced insulin production capacity.[11]

Reduced fetal and infancy growth (measured as lower birth weight and weight at one year) was strongly associated with high plasma levels of fibrinogen and factor VII in adult life. This may be a persisting response to impaired liver development during a critical early period.[11]

Another adverse response to growth failure in fetal life and infancy is the tendency to store fat abdominally in adult life. Android fat distribution increases risk of cardiovascular disease and DM. It is independently associated with increased blood pressure, serum cholesterol and triglycerides, as well as plasma glucose and fibrinogen.[11]

Preventing intra-uterine growth restriction could therefore prevent many adult diseases, which was originally thought to be only responsive to lifestyle changes.[11]

A good deal of attention has been paid to the influence of maternal size (pre-pregnancy weight and height) on pregnancy outcome; both under- and overweight affect maternal and newborn outcomes (*Table 1.1*).[9]

Table 1.1: Influence of maternal size on pregnancy outcome

	Underweight	**Overweight**
Newborns	Prematurity LBW/Small for gestational age (SGA) Increased perinatal mortality	Macrosomia (>4500g) Multiple births Increased perinatal mortality
Mothers	Hypertension Anaemia	Hypertension Gestational DM Labour complications Post-partum haemorrhage

Source: Brown JE. Preconceptional Nutrition and Reproductive Outcomes. In: Maternal Nutrition and Pregnancy Outcome, CL Keen, A Bendich, CC Willhite (Eds.). Annals of the New York Academy of Sciences, New York, 1993:286, 287.

Women of short stature have smaller pelvises than taller women, which have been associated with premature labour and FGR.[12]

The pattern of weight gain also influences the pregnancy outcome. According to the American Dietetic Association (ADA), only 30 to 40% of pregnant women gain weight according to the specified guidelines.[3] Poor weight gain dating from early in pregnancy, results in a "symmetrically" growth-retarded infant, who is uniformly small. Poor weight gain in the third trimester causes asymmetrical growth restriction, affecting weight and body length more than head size. Poor weight gain at the very end of pregnancy results in a thin baby who is underweight, but whose length and head circumference are normal.[11] Premature labour is also associated with lower weight gain late in pregnancy.[13]

1.1.1.1 Macronutrients

Appropriate nutrition during pregnancy will result in appropriate weight gain and improved pregnancy outcome.[4] Energy requirements increase during pregnancy from the second trimester onwards. An intake of 2200kcal/day is recommended in the first trimester (same as non-pregnant recommendation), and an additional 300kcal/day for the second and third trimesters.[3,4] Protein requirements increase from 50g/day when non-pregnant to 60g/day when pregnant.[4] When an energy-restricted diet is followed in pregnancy, there is increased competition between the mother and the fetus for nutrients. The body seems to protect the mother's stores, and FGR occurs. Protein and energy restrictions usually occur together; therefore it is difficult to separate their consequences. Intervention studies have shown that there was an increase of birth weight when the diet was supplemented with energy. No further increase in birth weight occurred when protein was added to the energy supplement.[4]

As with macronutrients, the Recommended Dietary Allowance (RDA) (1989) for micronutrients increases during pregnancy. Micronutrient deficiency and toxicity may lead to an adverse outcome for the mother and the fetus.[4]

A recent review article cited that although multiple micronutrient intervention studies have been done worldwide, it is still not possible to say conclusively that it helps improve pregnancy outcome.[14] The investigator acknowledges this, but has chosen to highlight the literature that discusses micronutrient deficiencies and toxicities and the successes of supplementation.

1.1.1.2 Vitamins

- *Folic Acid/Folate*

During pregnancy there are increased demands for folate because of increased maternal erythropoïesis and fetal-placental growth. The RDA during pregnancy is 400µg/day compared to 180µg/day before pregnancy (women aged 25-50 years).[4]

Women are at risk for folate deficiency include those with moderate to heavy alcohol use, smoking, certain drugs (anticonvulsants, methotrexate, valproic acid), pregnancy, lactation

and oral contraceptive use.[4,13] Suboptimal folate status during pregnancy could potentially impair cell growth and replication of the fetus and placenta, which in turn could increase the risk for LBW and premature delivery. Scholl and Hediger (1997) observed that a suboptimal folate intake (<60% of the RDA) carried a threefold increased risk for premature delivery and LBW.[15] A recent study in Nepal showed an increase in birth weight when a folic acid-iron supplement was taken antenatally.[16]

There is conflicting evidence, regarding the relationship of folate deficiency and miscarriage, LBW, prematurity and neural tubular defects (NTD) such as Spina Bifida and anencephaly.[4] The greatest controversy exists around the relationship between folate deficiency and NTD's. In animal studies, it was found that the anti-metabolites of folate are very teratogenic to the central nervous system (CNS); and that folate supplementation protected the CNS against the development of NTD's.[17]

In human studies it was found that there is a tenfold increased risk of NTD's occurring among first-degree relatives, indicating a possible genetic trait in its occurrence (the highest prevalence of NTD's are among the Irish, and the lowest among Blacks and Indian population groups).[17,18] Occurrence could also be sensitive to environmental factors, since the incidence is different in various socio-economic groups and has seasonal variations.[18] The use of aminopterin (folate antagonist anticonvulsant) has been associated with NTD and an altered folate metabolism. The result of Schorah and his team's intervention study was that subjects with a NTD history had an altered folate metabolism and that minimal folate supplementation could prevent NTD's.[17] The exact amount to be supplemented and which cases to supplement remains controversial. Amounts have varied from 360μg to 500mg, all with different results.[19] Bendich pointed out that since women at risk for NTD pregnancies have an altered folate metabolism, the form in which folate is supplemented, will play a role in prevention. The polyglutamate form of folate occurs in food, and needs to be enzymatically converted to the monoglutamate form to be taken up by cells. If the supplementation is in the monoglutamate form, the increased bioavailability could successfully raise folate status.[19]

Mills and Raymond (1993) raised various questions regarding folate supplementation of pregnant women:

- Should only at risk women or the general obstetric population be supplemented?

- If folate was to be vehicled as a fortificant in foods, what would the effects be? There is little experience of treating people with high doses (4mg/day or more) of folate over prolonged periods. If lower doses were used, would it still be effective?
- If supplementation was only to be given to women at risk for NTD pregnancies, what kind of effective screening can be used to target these women? When a woman has a history of NTD pregnancies, she could be targeted for supplementation, but how about those who have no knowledge or previous obstetric history of NTD risk?[20]

Various factors, therefore, need to be assessed before it can be decided who has to be supplemented, how much of the supplement and in what form the supplement of folate must be to prevent NTD. According to a recent ADA report, all women of childbearing years and pregnant women should supplement their normal, varied diets with 400µg a day of folate from fortified foods or supplements to prevent NTD's.[3]

- *Vitamin B6*

Increased vitamin B6 requirements during pregnancy are necessary for non-essential amino acid synthesis and other important roles played in carbohydrate and fat metabolism. The RDA during pregnancy is 2,2mg/day compared to 1,6mg/day before pregnancy (women aged 25-50 years).[4] Pregnant women at risk for vitamin B6 deficiency are drug and alcohol abusers, adolescents and those with multifetal pregnancies.[13] It has been suggested that a substantial number of pregnant women with normal diets develop biochemical abnormalities suggestive of a vitamin B6 deficiency. Unsatisfactory Apgar scores have also been associated with lower levels of vitamin B6.[4]

B6 supplementation has shown some success in the management of nausea and vomiting during pregnancy although it is not known whether the placebo effect or the actual effect on neurotransmitter production produces the relief.[4]

- *Vitamin C*

An increase in the vitamin C requirements is necessary to aid with increased metabolism, iron absorption and collagen synthesis during pregnancy. The RDA during pregnancy is 70mg/day compared to 60mg/day before pregnancy (women aged 25-50 years).[4] The vitamin C requirements increase with 10mg/day during pregnancy and it is easily met within most

diets. In the case of alcohol use, smoking and a multifetal pregnancy, the requirements increase.[13] With a multifetal pregnancy, an additional 50mg is needed and with smoking an additional 35mg of vitamin C is needed according to the Institute of Medicine (IOM).[21,22]

Although vitamin C deficiency has not been associated with adverse pregnancy outcomes in large population studies, a few have associated it with preeclampsia.[23] Serum ascorbate concentrations had been weakly associated with gestational duration.[14]

- *Vitamin A*

The RDA for women does not change in pregnancy because of accumulated maternal stores of vitamin A, which easily meets the fetus' and mother's needs.[13] The RDA for pregnant and non-pregnant women (women aged 25-50 years) is 800μg Retinol units/day.[4]

In 1932, F Hale discovered the first birth defect caused by a nutrient deficiency, when he fed pregnant sows a vitamin A free diet that produced offspring with no eyeballs.[18] Vitamin A deficiency in animals has been proven to be teratogenic, but no confirmatory evidence is available about the teratogenicity in humans.[4]

However, hypervitaminosis A has been associated with adverse pregnancy outcomes, such as hydrocephalus, microcephaly, cardiovascular abnormalities and altered growth.[13,24] Studies in the U.S.A. have determined that pregnant women's average vitamin A intake is higher than the RDA.[24] Impaired growth has occurred with supplementation of 25 000 IU vitamin A (or more) a day, or with isotretinoin (vitamin A analog for the treatment of Acne Vulgaris) use.[4,25]

A small study done in the U.S.A. on a group of women experiencing amenorrhea, found that they consumed an average of half a kilogram of carrots a day and very little white meat (chicken and fish). They had very high serum carotene levels and decreased prolactin, luteinising hormone, and follicle stimulating hormone. When a diet modification was introduced, most of the women's menses returned and a few conceived as well.[12]

1.1.1.3 Minerals

- *Iron*

Iron requirements increase substantially during pregnancy from 15mg/day (women aged 25-50 years) to 30mg/day. The mother's bone marrow needs an additional 500mg elemental iron for the increased erythrocyte production during pregnancy. The placenta and fetus needs 250-300mg elemental iron during pregnancy.[4]

Iron deficiency is common among women of reproductive age. With the increased needs of pregnancy, iron deficiency and subsequent anaemia are therefore very real problems.[26] Worldwide statistics confirm this: 51% of pregnant women globally suffer from anaemia, and 56% of pregnant women in developing countries. More than half of anaemias of pregnancy are attributed to iron deficiency.[27]

Factors contributing to iron deficiency, especially in developing countries, are:
- poor dietary iron intake (little red meat),
- poor dietary iron bio-availability (non-haeme iron in cereal-based diets and black tea taken with meals),
- increased iron requirements for reproductive demands, and
- iron losses due to parasitic infections.[27]

Moderate anaemia has been associated with shortened gestational periods and slower fetal development. Severe anaemia have been associated with prematurity and increased neonatal and maternal mortality.[28] The hypothesis behind this is that poor iron consumption leads to inadequate production of haemoglobin, which is the oxygen carrier on erythrocytes. This causes poor oxygen delivery to the placenta and fetus, and the mother's body tries to compensate by increasing the cardiac output, which adds extra work-load to the mother's heart, which stresses her other organs.[12]

The World Health Organisation (WHO) advocates iron supplementation in developing countries during pregnancy, especially the second half of it.[28] A recent study in the U.S.A.

showed that even the diet of middle- to upper-income pregnant women did not provide enough iron. The authors of this also recommend using an iron supplement.[29]

Counter arguments include cost, unpleasant side-effects and accidental poisoning of children.[28] The argument holding the most ground though is the lack of evidence that iron supplementation is beneficial in decreasing the prevalence of iron deficiency anaemia or iron deficiency during pregnancy.[26] In a study undertaken in the Cape Peninsula, South Africa where selective iron supplementation was given to pregnant women, it seemed to prevent haemoglobin from dropping below 10g/dl, but it did not decrease the prevalence of anaemia and iron deficiency.[28]

Possible explanations for the ineffectiveness of iron supplementation are poor compliance, and when combined with calcium and magnesium supplementation, iron absorption is decreased. Large doses of elemental iron are also associated with diminished absorption and more frequent side-effects (heartburn, nausea, diarrhoea, constipation and dark stools).[26]

The following can be done to improve absorption and compliance with iron supplementation during pregnancy:

- explain the benefits of taking supplements,
- start early in the second trimester,
- do not combine intake with calcium, tannins (in black ceylon tea) and phytates (in the outer husks of cereal grains),
- take the supplement with meals,
- give in the form of a one a day supplement, and
- inform the mother of the side-effects.[26,28]

To address this problem more effectively, prevention through nutrition education and advanced identification of women at risk for anaemia and iron deficiency are essential. Those at increased risk are:

- pregnant women aged 18 – 22 years, since the growth period of adolescence just ended (iron stores may be low or depleted),
- primigravidas since they usually fall in the 18 – 22 years age group,

- more than 3 previous pregnancies increases the risk, as a result of the maternal depletion syndrome: because of the increased iron demands of pregnancy, consecutive pregnancies without sufficient iron intake can have an accumulative depletion effect,[28] and
- pregnant women who had consecutive pregnancies less than 2 years apart. The strain that pregnancy places on her iron stores, requires at least 2 years for her to replace the lost iron. Taking iron supplements will speed this up.[12]

Nutrition education to prevent inadequate iron stores in women of childbearing age must focus on:

- increasing iron intake: food sources rich in iron include organ meats, meats, poultry, seafood, legumes, whole wheat, green leafy vegetables and nuts; and
- increasing iron absorption: the absorption of plant sources of iron can be increased by concurrently consuming vitamin C-rich foods and/or meat, poultry, fish; and by avoiding tea and coffee with meals.[27,28]

- Zinc

Zinc is associated with enzymes in carbohydrate, protein and fat metabolism, and DNA, RNA and protein synthesis.[13] Zinc finger proteins are the largest class of DNA-binding proteins. Zinc finger controlled gene expression is a major part of development, growth and other processes.[30] The RDA during pregnancy is 15mg/day, compared to 12mg/day before pregnancy (women aged 25-50 years).[4]

In animal studies, zinc deficient rats have given birth to offspring with cleft lip, cleft palate, brain, eye, heart, lung, and urogenital abnormalities. Even short periods of deficiency during pregnancy have shown to influence the embryo. The rapid effects of a zinc-deficient diet during pregnancy have been, in part, attributed to the lack of pools of tissue zinc that can be mobilized in response to a deficient diet. The mechanisms by which zinc deficiency can cause the above-mentioned defects are not well understood. A possible mechanism suggested, is that a zinc deficient diet is associated with an energy deficit. However, most investigators agree that the gross structural defects associated with zinc deficiency cannot be explained by decreased energy intake, since food deprivation is not so typically teratogenic. Additional possible mechanisms are: decreased protein and nucleic acid synthesis, increased

oxidative damage, cellular membrane defects, altered gene expressions, and decreased binding of hormone and transcription factors dependant on zinc finger proteins.[31]

Studies done regarding zinc deficiency in humans, have shown that clinical signs of zinc deficiency are the result of decreased protein synthesis; such as slowed growth, slow wound healing, skin lesions, and an impaired immune function.[26]

In the U.S.A., the zinc intake of a high number of women is below the RDA. This may suggest a significant subpopulation of pregnant women with a marginal zinc status, although it must be noted that an intake less than the RDA is not necessarily a deficient diet.[31]

According to Keen and fellow-investigators, the primary cause of zinc deficiency is a zinc-deficient diet, and the secondary causes are tissue injury and maternal stress. Regarding the secondary causes, the following hypothesis states the proposed mechanism: certain drugs and environmental challenges cause an acute phase reaction, which includes metallothionein (MT) synthesis. MT binds zinc and copper in the liver, which causes a decrease in serum zinc.[31] Alcohol especially, is one of the drugs that causes an increase in MT synthesis. It is a diuretic as well, which increases dietary zinc excretion.[26,31]

There are 3 reasons why zinc deficiency may be teratogenic in humans:

- Females with the genetic disorder, *Acrodermatitis enteropathica* (disorder characterised by signs of zinc deficiency), have a high prevalence of offspring with congenital malformations related to zinc deficiency unless given zinc supplements.
- Women with decreased serum zinc levels in early pregnancy have a higher frequency of pregnancy complications than women with adequate or higher serum zinc levels.[31] Some complications experienced during pregnancy, labour or abnormalities in the offspring associated with a zinc deficiency are: LBW, prematurity, pregnancy-induced hypertension, prolonged labour, intrapartum haemorrhage, and congenital malformations.[26]
- Some prospective studies have shown a decrease in pregnancy complications when given zinc supplements.[31] Specifically, a 30mg/day supplement of elemental zinc given to a group of pregnant teenagers led to a lower incidence of prematurity and the need for respiratory support for the infant.[26] A recent randomised, placebo controlled study in Bangladesh used a 30mg a day elemental zinc supplement from 12 to 16 weeks of

pregnancy until delivery. The aim was to assess the effect of daily zinc supplements on the mothers' offspring's growth and morbidity. The interpretation of the study's findings is that there was no increase in birth weight, but there was a reduction of health risks among the LBW infants.[32]

- Calcium

Calcium regulating hormone levels alter during pregnancy to increase calcium absorption in the intestines.[13] Approximately 30g of calcium have to accumulate during pregnancy; 25g in the fetal skeleton and 5g in the mother's skeleton for lactation demands. The RDA during pregnancy is 1200mg/day, compared to 800mg/day before pregnancy (women aged 25-50 years).[4]

The increased requirements for calcium during pregnancy can be met by most diets, but high-risk groups such as adolescents and women suffering from lactose intolerance may need supplementation to meet the RDA.[3,13]

It has been suggested that supplementation of more than 2000mg calcium/day may be protective in preventing pregnancy induced hypertension (PIH), since ecclampsia (a severe outcome of PIH) is similar to tetany as a result of hypocalcaemia.[13] The proposed underlying mechanism of calcium supplementation preventing PIH is that it reduces vascular sensitivity to angiotensin II (raises blood pressure by causing peripheral vasoconstriction).[12] Leg cramps occurring during pregnancy, thought to be related to decreased serum calcium, and a calcium/phosphorus imbalance, may also improve with calcium supplementation.[4,13]

Osteomalacia of the mother and newborn, caused by calcium deficiency, may occur in severe cases of a calcium-deficient diet.[4]

- Iodine

Iodine is an essential part of thyroid hormones that are produced by the thyroid gland. Thyroid hormones play an important part in cell proliferation and the nerve system (synapse and denditric formation).[14,26] The RDA during pregnancy is 175µg/day, compared to 150µg/day before pregnancy (women aged 25-50 years).[4]

During pregnancy, the levels of oestrogen increase, which causes increased serum thyroxine levels and speeds up the glomerular filtration rate (GFR). This leads to greater urinary iodine losses. It is unnecessary to take supplements though, since using iodised salt meets the requirements of pregnancy.[26] (Salt is iodised by law in RSA.[33])

In other countries where salt is not iodised and drinking water does not contain sufficient iodine, iodine deficiency has occurred. Severe iodine deficiency in pregnancy can cause an extremely debilitating syndrome in the offspring known as cretinism.[4,26] It is characterised by mental retardation, deaf mutism, spastic diplegia or quadriplegia, a characteristic shuffling gait, shortened stature and hypothyroidism. The degree of cretinism is determined by the degree of iodine deficiency of the pregnant mother.[4] Iodine supplementation decreased infant mortality rates in the country formerly known as Zaire and Algeria.[14]

- *Magnesium*

The increase in RDA for pregnancy is to compensate for the needs of fetal and maternal tissue growth. The RDA during pregnancy is 320mg/day, compared to 280mg/day before pregnancy (women aged 25-50 years).[4]

In 1990, the National Academy of Sciences of the U.S.A. advised that magnesium supplementation during pregnancy may reduce the incidence of pre-eclampsia and intra-uterine growth retardation. At that time, the available data was inadequate to recommend changes to the RDA.[2] The Magpie trial collaborative group recently published results showing that magnesium sulphate supplementation caused a 58% reduced risk of eclampsia among women who had pre-eclampsia in pregnancy.[34]

Kurzel (1993) raised the question whether magnesium deficiency can predispose to premature labour. Certain prospective studies have shown that magnesium supplementation reduces the incidence of preterm delivery and premature rupture of the membranes. Magnesium plays an important part in muscle contractions; calcium being the stimulator and magnesium is the inhibitor of muscle contractions. Hypomagnesaemia, therefore, may initiate uterine contractility or irritability thereby initiating labour. Kurzel *et al.* determined through their own studies that from the beginning of pregnancy until approximately 35 weeks of gestation serum magnesium steadily rises, and then drops again until labour. They found that women

who experienced premature labour had a significantly lower serum magnesium concentration, determined between 21-33 weeks gestation, than those with term labour. Since women with a history of premature labour are statistically more likely to experience a recurrence, Kurzel suggests magnesium supplementation may prevent prematurity.[35] Kurzel's findings are supported in a randomised, controlled trial done by Crowther (2003) and colleagues. They found that giving a magnesium sulphate supplement to pregnant women who were at risk of premature delivery improved important paediatric outcomes (mortality, cerebral palsy and combined), although not significantly so. These findings are not consistently supported by other studies.[36]

- Essential Fatty Acids (EFA's)

EFA's were discovered in 1960 when babies who were fed a skim milk formula developed signs of deficiency. The EFA's are: linoleic acid (omega 6 family), a precursor for prostaglandins, thromboxanes and arachidonic acid, and linolenic acid (omega 3 family), a precursor for docosahexanoic acid (DHA) and eicosapentanoic acid (EPA) respectively. EFA's are important membrane components and are essential for several physiological processes.[37]

Regular intake of fatty fish (a good source of omega 3 fatty acids) during pregnancy was associated with a fall in the rate of premature delivery, according to a Danish study. Since prematurity is the most common cause of LBW, infant morbidity and mortality, the possibility that a single dietary measure can help prevent this, is good news. The high content of omega 3 fatty acids in fish led investigators to believe that these EFA's acts on gestation length and parturition.[38] This is supported by studies conducted in Kansas City and Glasgow where the investigators supplemented the mothers' diets with DHA-rich eggs and fish oil supplements respectively during the second half of pregnancy, with significant increases of gestation duration.[39,40] The proposed mechanism is that an increased prostacydin or a decreased prostaglandin production is associated with an increased omega 3 fatty acid intake.[15]

Since the fetus has a decreased ability to convert linolenic acid to DHA, which is essential for normal eye and brain development, it is dependant on the mother for an adequate supply of this essential fatty acid.[37]

1.1.2 Improving maternal nutritional status and pregnancy outcome

The most important step in improving maternal nutritional status and thereby pregnancy outcome is to identify the mothers who are at risk of being malnourished, and who will be unable to consume adequate nutrition during pregnancy. There has been sufficient evidence to conclude that maternal nutrition influences reproductive performance, especially of women who are at high risk to have LBW babies. Birth weight, as a reflection of reproductive performance, is thought to determine the child's potential for future health and survival. Children who were born with a LBW are more likely to be more frequently hospitalised for illnesses, to have more visual and hearing disabilities, behavioural disorders and to experience more learning problems at school. LBW is also an etiological factor in cerebral palsy, epilepsy and mental retardation.[12]

In the U.S.A., the Institute of Medicine (IOM) has established nutrition risk criteria to determine who should receive food assistance from the Special Supplement Program for Women, Infants and Children. Because these markers were derived from epidemiological data in the U.S.A., representing the majority of at risk women, the investigator is certain that it could be used to identify at risk women in population groups outside the U.S.A. as well. A summary of these at risk criteria or markers for pregnant women are discussed below.[12,41]

- Age

The age of a mother determines her reproductive efficiency. The very young (≤ 15years old) do not have the physiological maturity to handle the stresses of pregnancy. The bodies of older women (≥ 49 years old) are also not viable for pregnancy when experiencing the effects of ageing and menopause. Mothers between the ages of 25 and 34 years have the best pregnancy outcomes.[12]

- Parity

Most problems are experienced with a first pregnancy, such as PIH and delivery complications. The risk of LBW increases substantially after 5 or more pregnancies. The risk of LBW is further increased if subsequent pregnancies are closely spaced.[12] Closely spaced

pregnancies are defined as those with an interconceptal interval of 6 months or 9 months if the mother is concurrently lactating.[41]

- Past obstetric performance

Problems experienced in a previous pregnancy increases the chances of them occurring again. The chance of a LBW infant is greater when past pregnancy outcomes were poor. This implies that a poor pregnancy outcome is not merely coincidental, but that there are some underlying causes that place women at a continuous risk each time they are pregnant.[12]

- Race

In the U.S.A., maternal and perinatal mortality and morbidity rates are 2 to 3 times higher among non-whites than whites. There are also a higher percentage of LBW infants among non-whites than whites. The reasons for this are complex and not well understood. In the above-mentioned investigation, the average non-white mother was younger, had a poor education level (a marker of socio-economic status) and had less perinatal care than the average white mother. Even when maternal smoking, stature, pregnancy weight gain, all combinations of age and parity, and history of previous LBW offspring were controlled for, black mothers still had and increased risk of having LBW offspring.[12]

- Social class

Social class is usually determined by a person's income, occupation and education according to western culture. In South Africa as well as the U.S.A., social class can be confounded by race, specifically that people of the non-white ethnic groups generally fall in a lower social class than whites. It is the social and physical environment which factors such as income and education represent that affects pregnancy outcome. Housing, sanitation, diet and health care are known to vary with different socio-economic status.[12]

- Prenatal care

U.S.A. data strongly supports the suspicion that inadequate prenatal care significantly increases a woman's risk for a poor pregnancy and pregnancy outcome.[12] According to the nutrition risk criteria, inadequate prenatal care is defined as the 1st visit to a antenatal health facility after the 1st trimester or "long" intervals between visits.[41]

To summarise, if women have one or more of the following risk factors,

- of non-white race
- impoverished
- lack of education
- under 17 or over 35 years of age
- 1st pregnancy or high parity
- pregnancies less than one year apart
- prior obstetric complications
- previous fetal-infant death or disability,

they are more likely to have LBW infants and they can be targeted for special intervention.[12]

The effects of smoking and alcohol use during pregnancy are also very significant in causing LBW, and are discussed later separately.

1.2 Physiological changes during pregnancy that may influence nutritional status

1.2.1 Changes that affect absorption and utilisation of nutrients during pregnancy

Many changes occur in the gastro-intestinal tract (GIT) and kidneys that affect nutrition during pregnancy. These changes are caused by the release of hormones and the increase in intra-abdominal pressure. Ultimately, these changes occur to increase the absorption and utilisation of essential nutrients.[8]

Symptoms such as an increase in appetite, nausea and vomiting occur in most cases. Changes in the GIT include decreased intestinal secretions, increased nutrient absorption and decreased motility of the GIT. The latter is caused by an increase in progesterone levels and causes esophageal regurgitation, slowed emptying time of the stomach and reverse peristalsis.[3,12] This in turn, leads to heartburn and constipation. Constipation is also worsened by the swelling uterus compressing the intestines and pressing up against the stomach.[12] The benefit of slowed emptying time of the stomach is that the rate of glucose absorption is reduced, and

this in turn prevents peaks in insulin levels. The decreased intestinal motility is beneficial since there is more time for the absorption of micronutrients. The disadvantage of this is that more water is absorbed in the colon, which in turn leads to constipation.[8]

During pregnancy, there is increased workload placed on the kidneys. The increased metabolic demands of the mother's body and the fetal waste products have to be processed by the kidneys. Hormones, an increased blood volume, the mother's posture and nutrient intake cause the change in renal function. The glomerular filtration rate increases by thirty to fifty percent during pregnancy.[12] This results in greater glucose, amino acids, nicotinic acid, vitamin C and folate losses because the tubules cannot reabsorb these nutrients efficiently.[3,8,12] The increased glucose excretion also increases the mother's chances of developing urinary tract infections.[12]

The high levels of progesterone during pregnancy cause a decreased emptying time of the gallbladder and slightly higher serum cholesterol. This could lead to gallstone formation.[12]

1.2.2 Changes that affect nutrient intake during pregnancy

Hormones are usually named as the reason for eating habits changing during pregnancy. However, other factors also play a role, such as ethnic preferences, individual preferences (vegetarianism, allergies, dislikes), religious considerations and economic constraints.[7] The fear of gaining weight or advice from the pregnant woman's physician, her friends and family mitigate food intake as well. Regardless of all the factors that influence nutrient intake during pregnancy, the primary determinant of intake will be the pregnant woman herself.

During pregnancy, the ability to taste is altered, and a lower sensitivity for sweet and salty tastes develops.[8,12] This can manifest itself as cravings. Some foods that are commonly craved are chocolate, citrus fruits, pickles, chips and ice-cream.[3] Unpleasant gastro-intestinal symptoms, such as nausea and heartburn can cause certain food aversions to develop. Some foods that are commonly disliked during pregnancy are meat, coffee and spicy or fatty foods.[3,8]

Pica is a compulsion to consume non-food substances or unsuitable substances having no or little nutritive value.[3,7] Some of the more common substances consumed are clay, soil, ice, freezer frost, laundry starch, soap, ashes, chalk, paint, burnt matches, corn starch and baking soda.[3] Although pica can occur within both sexes and in different races and cultures, it does manifest in pregnancy more frequently. The origin or compulsion of pica is unknown. Some believe that hormones or iron deficiency predispose to pica. Pica can be dangerous during pregnancy. Malnutrition can occur if the unsuitable substance is eaten instead of food. In cases where clothes starch is eaten, obesity can occur. Lastly, poisoning can occur when paint and charcoal, for example, are eaten.[7]

1.3 Alcohol and its effects on maternal nutritional status, pregnancy and pregnancy outcome

1.3.1 The effect of alcohol on maternal nutritional status

Alcohol can affect nutrition in several ways. Alcohol, which contains 7kcal/g, can displace energy from more nutritious foods leading to deficiencies of essential nutrients.[21,42] The effect of alcohol on nutrition is also dependant on dose and the length of time it has been used.[42] Heavy drinking for a long period of time therefore affects nutritional status more adversely than light drinking for a short period. The ways in which heavy drinking can cause malnutrition are as follows:

- Inadequate food intake

There is no single established dietary pattern for people who regularly drink alcohol. Moderate and social drinkers usually have a good daily food intake and the alcohol consumed adds extra energy to an adequate diet. This can lead to weight gain.[42] In contrast, heavy drinkers often eat poorly, since food intake may be less important to them than their alcohol intake.[21,42,43] Some heavy drinkers take as much as 50% of their daily energy as alcohol;[43] therefore intakes of protein and micronutrients may be severely inadequate.[42] This can lead to changes in body composition, and reduced lean body mass and fat stores have been reported.[44]

- Inadequate digestion and absorption

Alcohol inhibits the breakdown of nutrients by decreasing secretion of digestive enzymes from the pancreas. Inadequate secretion of pancreatic enzymes leads to steatorrhoea, which causes loss of fat-soluble vitamins and calcium.[43] Alcohol also impairs nutrient absorption by damaging the stomach and intestinal mucosa, causing thiamin and cobalamine to be malabsorbed. Severe mucosal damage can result in gastro-intestinal bleeding, which can cause iron losses.[43,45] Malabsorption is common in heavy drinkers, with both alcohol use and malnutrition affecting absorption.[44] For example, folate deficiency alters the lining of the small intestine, which in turn impairs absorption of water, glucose, sodium and folate.[43] Alcohol also affects absorption by delaying or increasing gastric emptying. The effect is dose-dependant, ≤6% ethanol concentration either accelerates or have no effect on gastric emptying, while ≥10% concentration delays gastric emptying. Alcohol can increase intestinal motility, which causes diarrhoea.[44]

- Impaired nutrient utilisation

Ethanol interferes with the metabolism and utilisation of various nutrients. There is interference with the body's blood glucose control. When a fasting or malnourished person drinks alcohol, hypoglycaemia can occur. When there is no food to supply energy, stored sugar is depleted, and the products of alcohol metabolism inhibit gluconeogenesis. Long-term drinking triggers the microsomal ethanol-oxidising system (MEOS). MEOS is an inefficient alcohol metabolism system, which uses more energy than it produces, therefore causing loss of energy that the body needs. Ethanol impairs protein nutrition by inhibiting protein breakdown to amino acids, impairing the processing of amino acids by the small intestine and liver, impairing synthesis of protein from amino acids and impairing protein secretion by the liver.[43] There is interference with the conversion of thiamin to its active form as well as decreased utilisation of the active form. Ethanol interferes with the hepatic formation and release of S-methyltetrahydrofolic acid, conversion of pyridoxine to its active form, and the activation of vitamin A.[44]

- Increased requirements

Long-term heavy drinkers have wastage of energy as a result of the increased use of MEOS. Long-term alcohol use is also associated with an increased metabolic rate and oxygen use by

the MEOS. Energy requirements are increased to prevent a mildly malnourished heavy drinker becoming severely malnourished. Protein requirements are also increased because of malabsorption and impaired utilisation. Folate, vitamins B6 and B12 are involved in tissue growth and repair, and requirements are increased in the heavy drinking individual due to the damage caused by alcohol to the gastro-intestinal tract and related organs. Folate requirements are increased to correct the reduced haemopoiesis seen in heavy drinkers. A high ethanol and carbohydrate load increases thiamin requirements.[44]

- Decreased stores

With fatty infiltration of the liver (the first stage of liver cirrhosis and alcoholic liver disease) lower concentrations of vitamins thiamin, folate, nicotinic acid, B2, B6, B12, pantothenic acid and vitamin A have been found in the liver.[44,45,46] The decreased stores may simply be due to reduced storage space due to fat, fibrosis and cellular necrosis.[43]

- Increased excretion and other losses

Intestinal bleeding leads to iron loss.[43,45] The release of vitamins, including folate, from hepatic stores may be increased through urinary excretion.[44] In heavy drinkers, increased urinary losses of zinc, calcium, magnesium and phosphate occur, together with increased faecal losses of nitrogen and magnesium that have been reported.[43,44,47]

1.3.2 The effect of alcohol on pregnancy

1.3.2.1 The potential mechanisms of alcohol's teratogenic effect on pregnancy

Alcohol has many different types of effects on the body, and it is unlikely that they are all mediated by the same mechanisms.[42] With the helpful results from animal studies, there are several mechanisms explaining alcohol's teratogenicity on the mother and fetus during pregnancy.[48]

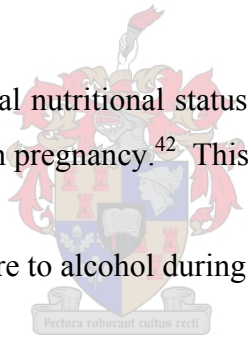
Alcohol and its primary metabolite, acetaldehyde, are known tissue toxins that may directly interfere with fetal cellular growth and metabolism.[42,48]

Alcohol-induced hypoxia could be responsible for some of the adverse effects of maternal alcohol use. One well-established consequence of fetal hypoxia is growth restriction, since inadequate oxygen can cause cell death and/or slowed cell growth. Large amounts of oxygen are needed to metabolise alcohol via the MEOS.[42]

Maternal alcohol use can interfere with placental growth and function. A study on monkeys showed that alcohol given intravenously led to the collapse of the umbilical vasculature. This alcohol-induced impairment of umbilical circulation impairs oxygen transport that leads to fetal hypoxia and acidosis.[12] Impaired nutrient transport to the fetus may also occur. The placenta itself may also oxidise ethanol, producing toxic amounts of acetaldehyde.[42]

Fetal hypoxia may also result from premature placental separation, amniotic fluid infections, anaemia and antepartum bleeding, all of which are more common in women who drink heavily.[42]

Finally, alcohol can affect maternal nutritional status adversely, and could be responsible for the teratogenic effect of alcohol on pregnancy.[42] This will be discussed later on.



1.3.2.2 Critical periods of exposure to alcohol during pregnancy

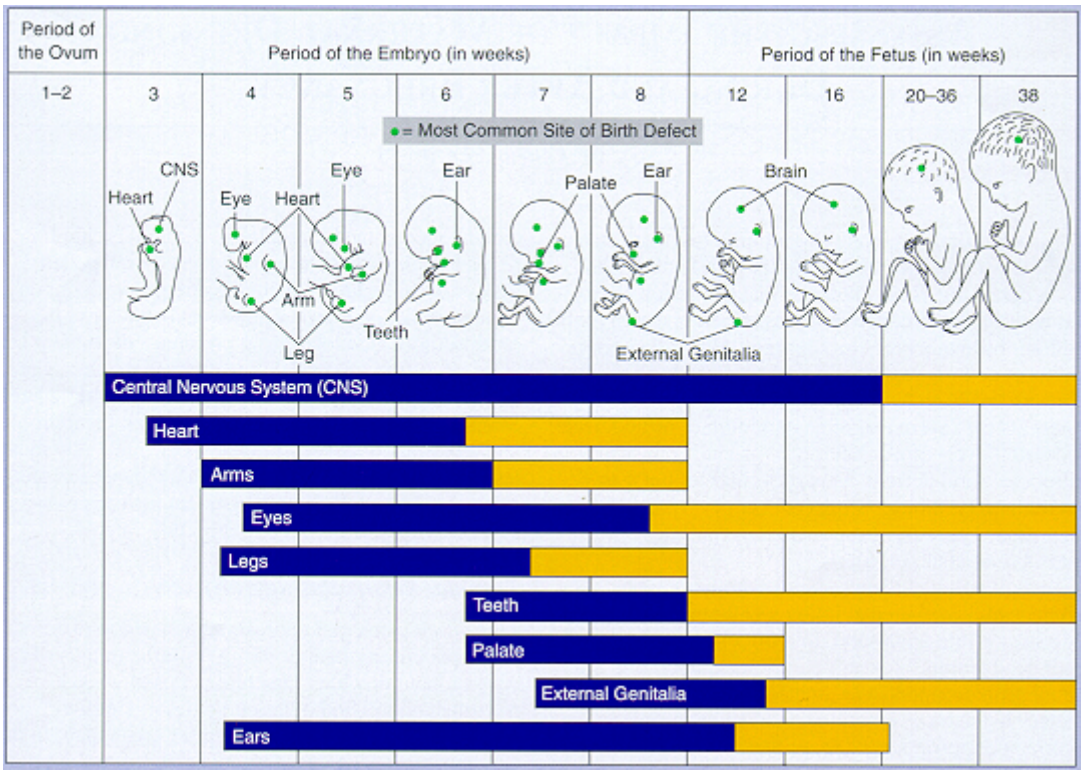
Alcohol consumption during any time of pregnancy is potentially harmful to the fetus.[49] The specific body systems affected by alcohol exposure depends on the time of the exposure coinciding with the critical development of the specific body system (*Figure 1.1*).[50]

The first trimester (0-12 weeks) seems to be the most critical period for structural abnormalities to occur. Alcohol may affect the way in which cells grow and arrange themselves as they multiply, thereby causing abnormalities in the embryo.[49] Abnormalities of the face, heart, brain (neural tube defects in severe cases), limbs and urogenital system can occur.[48,50]

Alcohol exposure in the second trimester (12 - 28 weeks) may result in miscarriage.[49]

During the third trimester (28 weeks - end) the fetus grows rapidly, and alcohol exposure can result in FGR.[49]

The brain continues to grow throughout pregnancy and during the post-partum period, and remains sensitive to alcohol's teratogenic effect. The entire pregnancy is therefore a critical period during which alcohol exposure can affect brain development.[49]



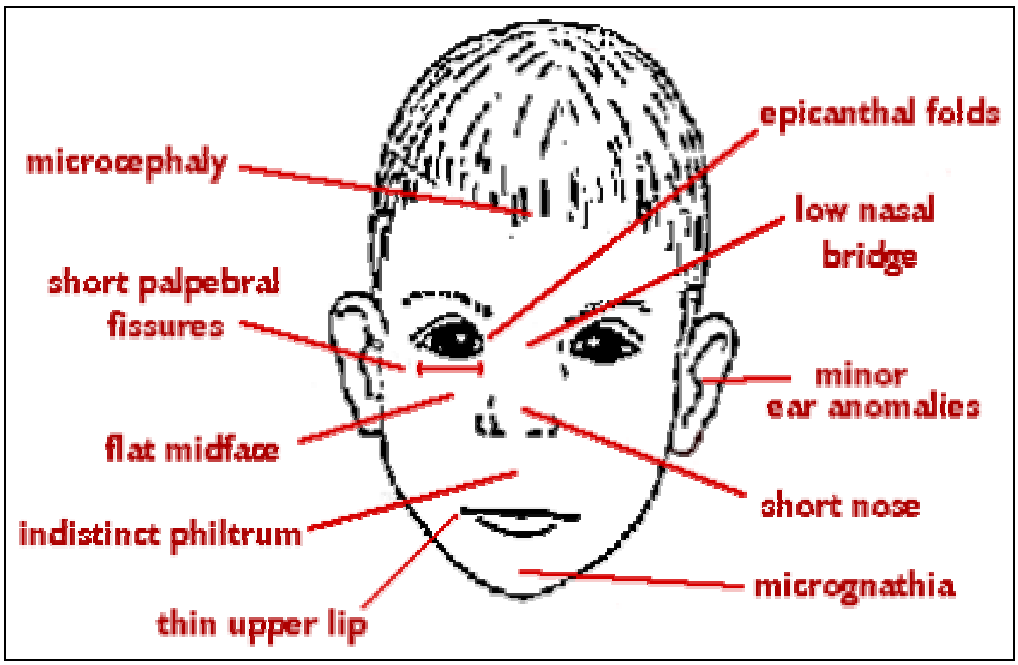
Source: Jacobson SW. Assessing the impact of maternal drinking during and after pregnancy. Alcohol health and research world 1997; 21(3):199-200

Figure 1.1: The periods of development of specific body systems of the fetus during pregnancy

1.3.2.3 Fetal Alcohol Syndrome (FAS)

FAS is a birth defect caused by heavy alcohol consumption during pregnancy. It is the most common cause of mental retardation, and the only preventable one.[51] FAS is diagnosed when the full spectrum of features are present, namely:

- Low Birth Weight and growth retardation
- Facial features (*Figure 1.2*) such as - microcephaly
  - short palpebral fissures
  - epicanthic folds
  - low nasal bridge
  - long, flat philtrum
  - thin upper lip
- Mental retardation and behavioural abnormalities
- Other organ system involvement including cleft lip and palate, spina bifida, renal anomalies and cardiovascular defects.[42,49,51,52]



Source: Randels JP, Streissguth AP. Fetal Alcohol Syndrome and Nutrition Issues. Nutrition Focus for children with special health care needs May/June 1992; 7: 3

Figure 1.2: The characteristic facial features of FAS

In addition to these features, a diagnosis is dependant on a positive history of heavy drinking during pregnancy.[49]

The original term, Fetal Alcohol Effects (FAE), also related to maternal alcohol consumption, was divided into 2 new categories of birth defects by the Institute of Medicine in 1996:[51] The 2 new categories are Alcohol Related Birth Defects (ARBD), which is characterised by physical abnormalities, and Alcohol Related Neurological Defects (ARND), which is characterised by physical and central nervous system abnormalities.[49] These 2 birth defects usually occur with smaller amounts of alcohol consumption during pregnancy.[51] The latest phrase coined is "Fetal Alcohol Spectrum Disorder". It is preferred by the U.S.A.'s National Institute for Alcohol Abuse and Alcoholism and is meant to include all forms of defects and disorders that resulted from all levels of prenatal alcohol exposure.[52]

A study by Viljoen and Croxford (1999) assessed alcohol consumption of 600 pregnant women attending antenatal clinics in the Western Cape. It was found that almost 1 in 4 women were drinking heavily and thereby placed their unborn children at high risk for FAS.[51] The incidence of FAS, ARBD and ARND in South Africa have only been determined in certain areas. The Foundation for Alcohol Related Research (FARR) has determined an incidence of 6.6% in the Western Cape and 4.7% in Gauteng of FAS among school-entry children. The former is the highest reported in the world. Further research is currently underway in the Northern Cape, where even higher prevalences are being observed.[49,53]

Certain factors must play a role in the etiology of FAS since not all women who drink heavily during pregnancy have FAS-affected offspring.[42] As with other teratogens, outcome of pregnancy can be modulated by various biological and environmental factors including exposure to alcohol above certain threshold levels, the pattern and timing of exposure to alcohol, genetic vulnerability of the fetus and mother and the nutritional status of the mother.[54]

Women and men metabolise and absorb alcohol very differently. Women will have higher blood alcohol concentrations (BAC's) after consuming the same amount of alcohol as men. This is because women generally have less body water (mostly muscle) than men. Women also may have less alcohol dehydrogenase (ADH) activity in the stomach, causing

more of the ingested alcohol to reach the blood circulation.[55] Older women and women with higher parity have been found to be more likely to have FAS-affected offspring. With increasing age, a woman's body fat increases and her body water drops through loss of body muscle, which could lead to higher peak BAC's. Older women with a history of heavy drinking, can also metabolise alcohol quicker and have increased levels of acetaldehyde, which is highly teratogenic.[56] Undernourished women may have less muscle than appropriately nourished women, causing their bodies to have higher peak blood alcohol levels.[42]

Since there is a difference in incidence of FAS among different ethnic groups, it seems that a genetic susceptibility may play a role.[56] Recent research done among the mixed ancestry population in the Western Cape, investigated certain known polymorphisms of the alcohol dehydrogenase-2 (ADH2) gene making isozymes with different alcohol oxidizing capacities, as being possible candidates for influencing the risk for FAS. Results showed that the alcohol dehydrogenase-2∗2 (ADH2*2) allele was found significantly more in the control group than in the mothers of FAS-affected children and the children themselves. It would seem therefore, that ADH2*2 allele may be a marker for some kind of protection against FAS.[57]

Worldwide, it was found that FAS had the highest incidence in low socio-economic status settings.[56] Poverty, which is associated with poor education, undernutrition, smoking, drug abuse, lower religiosity and poor prenatal care, can exacerbate the effect of alcohol on the fetus.[49,56]

1.3.3 The possible synergistic effect of maternal alcoholism and maternal malnutrition on pregnancy

Alcohol itself is directly toxic to the developing fetus and pregnant mother, and an accompanying nutritional deficiency can compound the risk for adverse pregnancy outcomes.[43] A number of reasons have been proposed to explain this apparent synergy. The increased use of MEOS in heavy drinkers causes increased free radical production. Free radicals are known to cause cellular damage in heavy drinkers. Animal studies have shown high levels of superoxide dismutase (SOD), an enzyme produced to protect cells from free

radical damage, in mother's and fetal livers, suggesting that the tissues of heavy drinkers and of their unborn children may be experiencing alcohol-induced free radical damage. Since the fetus' anti-oxidant defense system is not fully developed, it is very susceptible to free radical damage.[48] Anti-oxidant nutrients in the mother's diet are therefore very important to protect the fetus against the documented background that the nutritional status of the heavy drinking mother may be inadequate, and deficiencies of anti-oxidants have been described.[56] Specifically, zinc deficiency has been investigated (caused by increased urinary losses in heavy drinkers) because of the similarities between zinc deficiency and alcohol's teratogenicity, and women of FAS-affected offspring had significantly lower zinc levels than women of non-FAS-affected babies.[42] Zinc, as part of SOD, can displace metal ions from membranes, and is associated with metallothionein. Animal experiments support that free radical damage, caused by alcohol in a zinc deficient animal, is greater than free radical damage by either zinc deficiency or heavy drinking.[48] This suggests that zinc deficiency can potentiate alcohol's teratogenicity.[41]

Zachman and Grummer's (1998) hypothesis states that alcohol and its effect on vitamin A status is key to the pathogenesis of FAS.[58] Heavy drinking inhibits the conversion of vitamin A to retinoic acid, which is essential for brain, head and limb formation.[59] The hypothesis is supported by the phenotypic similarities between FAS and vitamin A toxicity and deficiency.[58]

Alcohol interferes with the activities of certain folate-containing enzymes. Some of the brain deficits of FAS may be due to the enzymes' decreased activity that is responsible for nucleic acid and protein synthesis.[59]

Therefore, although evidence exists that alcohol abuse and micronutrients deficiencies interact to ameliorate adverse pregnancy outcomes, it is far from conclusive and not well understood. More animal research is definitely warranted to better understand the mechanisms.[21]

1.4 Cigarette smoking

A recent study done among the mixed ancestry race group in the Western Cape, described characteristics of mothers of FAS-affected offspring compared to a control group. Since 83.9% of subjects admitted to smoking during pregnancy, compared to 45.2% of the controls, the investigator found it necessary to include information on the effect of smoking on pregnancy and pregnancy outcome.[60]

Cigarette smoke contains carbon monoxide, nicotine, cyanides and other compounds that cause insufficient oxygen to reach the fetus and placenta.[12,21] The major outcome of this is FGR. Smoking pregnant women have a double risk of having a LBW baby.[12] Smoking also seems to play a role in causing spontaneous abortions, especially in the 1st trimester, premature placenta abruption, premature delivery and sudden infant death syndrome.[21]

Smokers tend to have poorer eating habits than non-smokers. For example, in a U.S.A. study, almost twice the number of smokers versus non-smokers didn't eat breakfast.[59] Cigarette smoke also suppresses appetite and plays a role in inadequate food intake.[21] Smokers need more vitamin C, vitamin B6, folate and beta-carotene to maintain adequate serum levels of the nutrients. Although inadequate intake of the above nutrients may occur, smoking could decrease the absorption and increase the utilisation of the nutrients, resulting in lower serum levels.[21,59] There is very little evidence that cigarette smoking affects mineral status like it does that of the above vitamins.[21]

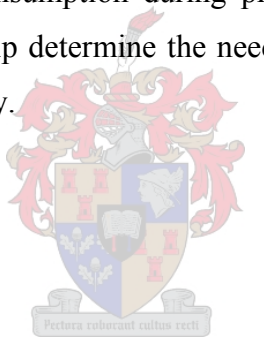
Although the mother's smoking during pregnancy has been related to FGR, spontaneous abortion and prematurity, there is no evidence that it causes congenital malformations. In contrast, the father's smoking has been associated with birth defects and childhood cancers in the offspring.[61]

Smoking, together with alcohol consumption, seemed to have an additive effect on reducing fetal growth as observed in a Danish study.[12]

1.5 Problem Identification and Motivation

There is little doubt from the available evidence that heavy alcohol consumption during pregnancy has teratogenic effects on the fetus.[49] The effect of alcohol on fetal development is dose-dependant, and varies from miscarriage to behavioural and cognitive abnormalities later in life.[54] The nutritional requirements of a pregnant woman increase to compensate for the growth of the fetus and the needs of her body.[4] It is therefore reasonable to assume that the dangers of heavy drinking in the presence of malnutrition can therefore place the fetus at a further disadvantage to normal development and life.

In view of the latter, the purpose of the study was to assess the nutritional status of pregnant women in relation to alcohol consumption during pregnancy and pregnancy outcome. The findings of the study will contribute to the better understanding of the interaction of nutritional status and alcohol consumption during pregnancy, and its effect on pregnancy outcome. The study will also help determine the need for nutritional support for those who consume alcohol during pregnancy.



2. METHODOLOGY

2.1 Aim of the study

The aim of the study was to determine nutritional status in pregnancy and alcohol consumption during pregnancy in relation to the pregnancy outcome.

2.2 Objectives

1. To determine the nutritional status of pregnant women attending the Hanover Park Midwife and Obstetrics Unit (MOU) and to relate it to the pregnancy outcome.

2. To relate alcohol consumption during pregnancy to the pregnancy outcomes.

3. To relate the combined effect of maternal alcohol consumption and nutritional status to pregnancy outcome.

2.3 Hypotheses

1. Null hypothesis

Pregnancy outcome is not affected by maternal nutritional status and alcohol consumption.

2. Alternative hypothesis

Pregnancy outcome is affected by maternal nutritional status and alcohol consumption.

2.4 Approval for the study

The study was approved by the Ethics Committee of the Faculty of Health Sciences, Stellenbosch University (*Appendix 1).* Approval for use of Hanover Park MOU as the study area was obtained by the Foundation for Alcohol Related Research (FARR) and from the

community health services organization of the Provincial Administration of the Western Cape (PAWC).

2.5 Study design

This was a prospective, longitudinal, cohort study.

2.6 Study area

The FARR study group had predetermined Hanover Park MOU as the study area in view of its long-standing relationship with this MOU, and the established trust that patients and employees have with FARR health workers.

2.7 Study population

The patients of Hanover Park MOU reside mainly in the Hanover Park, Phillipi and Mannenberg areas of Cape Town. The inhabitants are mostly of the Mixed Ancestry (Coloured) race group. A high proportion of the inhabitants live in poor socio-economic conditions and have limited formal education. The population group is therefore more prone to alcohol abuse.[56]

2.7.1 Selection of the study population

The study population was recruited from Hanover Park MOU by one FARR health worker during 3 morning visits a week (average) during the period of October 2000 to February 2002. Recruitment days were not always the same days of the week. Because of the well-established relationship between FARR and the staff of this MOU, the on-duty nurses who were responsible for prenatal care informed the FARR investigator of mothers who registered for prenatal care at the MOU for the 1st time, was less than 22 weeks pregnant, and did not have any serious medical conditions. While these mothers waited for their prenatal care, the FARR investigator approached each of them separately to ask if they would be interested in partaking in FARR's research studies. After the FARR investigator determined if the woman met all the inclusion criteria and was willing to take part, a consent form was signed.

The study population selected was, therefore, one of convenience. This study's population was the same as the one used for FARR's other pre-existing studies (the Maternal-Infant Study and the Ultrasound Study) but within this present study's specified time frame.

FARR's Ultrasound Study used ultrasound to determine whether the fetus had been affected by alcohol use in utero. Three to four ultrasound investigations were done with pregnant mothers, of which the first had to be done before 22 weeks gestation. After birth, the infants were investigated to determine whether the ultrasound investigation results could be verified by the clinical findings at one, six and 12 months of age. FARR's Maternal-Infant Study consisted of three interviews with the pregnant mother to determine her health, obstetric history, socio-demographics, alcohol use and smoking during pregnancy. After birth, a paediatrician and a developmental pschycologist examined the mother's infant at one month, six months and 12 months of age. The health, physical and neurological development of the infants were scrutinised in order to determine whether they had been affected by alcohol use during pregnancy.

Pregnant women were included in the study, if they met the following criteria:

- 18-40 years old;
- heavy drinkers, i.e. 2 or more drinks* /day, >5 drinks/week taken on a single occasion (binge), or >45 drinks per month [62] comprised the subjects of the study;
- light drinkers (up to 2 drinks/day and no binge drinking) or abstainers[62] served as controls;
- provided informed consent to having anthropometric measurements and a blood sample taken, as well as providing the necessary information for the completion of the questionnaires; and
- provided informed consent to having anthropometric measurements and clinical assessment conducted on their infants at the age of one month.

The exclusion criteria for the study were:

- the presence of renal and/or liver disease, epilepsy, hypertension or diabetes mellitus; as well as cardiac disorders or any other chronic disease receiving treatment, or other drug use with known nutrient interactions (*Table 2.1*).[63,64]

* One drink contains 15mls of absolute alcohol.[62]

The lower age limit of 18 years was chosen because of the limitations of using the Body Mass Index and MUAC for the assessment of nutritional status in adolescents. The upper age limit of 40 years was chosen since it is documented in the literature that the chances of poor reproductive outcome increase thereafter.[12]

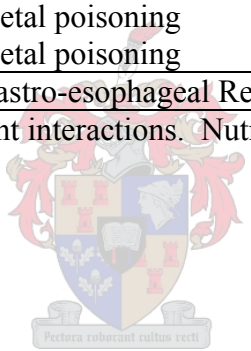
The diseases listed in the exclusion criteria were cited as they are known to alter the ingestion, absorption, metabolism and excretion of nutrients, which in turn may affect nutritional status. Moreover, pregnant women with uncontrolled diabetes mellitus are known to be predisposed to having children with fetal macrosomy, which would make anthropometric assessment of the newborn inaccurate.[6,63]

Alcohol consumption status (heavy drinkers or light drinkers/abstainers) was determined by means of a questionnaire (*Appendix* 2) by the same trained health professional of FARR who recruited the pregnant women. The women were classified only according to the frequency of alcohol consumption they drank during pregnancy before the interview. Because of this approach by FARR, quantitation of alcohol intake per se was not feasible.

Table 2.1: Drug-Nutrient Interactions as a criteria for exclusion from the study

Medication	*Disease used for*	*Possible Vitamin/Mineral Deficiency*
Mineral Oil Cholestyramine Neomycin	Constipation Hypercholesterolaemia Hypercholesterolaemia	Vitamin A
Cimetidine Colchicine Para-aminosalicylic Acid	Ulcers Gout T.B.	Vitamin B_{12}
Sulfasalazine Trimethoprim Pyrimethamine Anti-convulsant Drugs: • Phenytoin • Phenobarbitol • Primidone	Ulcerative Colitis Antibacterial Antiprotozoal Epilepsy/Convulsions	Folate
Chronic antacid use EDTA D-penicillamine	Gastro-esophageal Reflux Metal poisoning Metal poisoning	Zinc
Chronic antacid use	Gastro-esophageal Reflux	Iron

Source: Thomas JA. Drug-nutrient interactions. Nutrition reviews 1995; 53(10): 271-279



2.8 Methods Schedule

The pregnant women who met the entry criteria were included in this study. Having given consent, the women were collected by a FARR driver at their homes or in the immediate vicinity thereof and taken to the FARR offices, on the University of Cape Town (UCT) medical school premises, for data collection. Various information was collected at the different visits to the FARR offices as tabulated below:

Table 2.2: Methods Schedule

Persons Involved	*Time and Place*	*Information Collected*
Mother and FARR investigator	Hanover Park MOU for "booking"; mothers < 22 weeks pregnant.	Recruitment by FARR investigator. Consent letter for recruitment signed, and Full Blood Count done, use Hb and HCT values. Alcohol consumption questionnaire completed.
Mother and study investigator	FARR, UCT Medical School, ± 22 weeks pregnant.	Consent form signed. Blood sample drawn (15ml), full anthropometric assessment, clinical assessment, food frequency questionnaire, and 24 hour recall completed.
Mother and study investigator	FARR, UCT Medical School, ± 30 weeks pregnant.	Clinical assessment, questionnaire, 24 hour recall and weight appraisal completed.
Mother and study investigator	FARR, UCT Medical School, ± 36 weeks pregnant..	Clinical assessment, food frequency questionnaire, 24 hour recall and weight appraisal completed.
Newborn and paediatrician	FARR, UCT Medical School during Maternal-Infant study; at ±1 month of age.	Information gathered from baby's Road to Health Chart and MOU's birth summary (birth weight and head circumference, mention of any delivery problems, or physical/mental defects); clinical assessment done by FARR paediatrician for alcohol-related signs.
Study investigator and FARR investigators	End of data collection.	Share information between studies (i.e. this study and FARR's studies); especially relating to alcohol consumption status of mother, and relevant questionnaire data.

The health worker from FARR (headquarters at UCT Medical School) had previously determined the alcohol consumption status (light, heavy drinker or abstainer) and other demographic parameters of individuals. The investigator of this study collected and analysed all data blinded to the alcohol consumption status of the women. All information was shared with FARR after the completion of data collection and analysis. The data in this thesis remains the property of FARR and Stellenbosch University.

2.9 Methods

All questionnaires and nutritional status assessment methods were tested and validated in a pilot study. The investigator is a qualified dietician who is trained in these methods.

2.9.1 Questionnaires

The questionnaires (*Appendix 3*) were used to determine the normal dietary intake, and factors that could influence it:

- socio-demographics questionnaire (age, occupation, finances);
- health history and lifestyle questionnaire (parity, illnesses, medications, supplements, exercise); and
- dietary assessment questionnaire (food security, eating behaviour, food frequency questionnaires, 24 hour recalls).[63]

2.9.1.1 Socio-demographics

Socio-demographics, although giving no direct information about nutritional status, can indicate risk for nutritional disorders, such as poor living conditions or insufficient funds for proper nutrition.[63]

2.9.1.2 Health

Health history reveals disorders or medication that can affect a subject's nutritional status. Psychological disorders (e.g. depression, eating disorder) and certain medications, can act as appetite suppressants. Certain diseases such as cystic fibrosis and DM can alter the metabolism and utilisation of nutrients.[63] Previous obstetric problems (e.g. LBW, premature delivery) are also of concern because nutritional disorders may have been contributing factors.[12]

Information concerning lifestyle has a direct impact on nutrient requirements and their intake. High physical activity for instance implies increased nutritional needs.[63] The use of vitamin

and/or mineral supplements may give a mother a false sense of nutritional security and she may neglect her food intake. On the other hand supplements could also help in supplying nutrients otherwise lacking. Cigarette smoking has an effect on the appetite of the mother, and has been shown to increase the risk for LBW infants.[59,63]

2.9.1.3 Dietary assessment

Food security reflects the adequacy of food supplies at home, and also whether there are adequate facilities available for hygienic food preparation and storage. Eating behaviour provides information regarding meal pattern and any limitations (self- or otherwise induced) of the individual's diet. Gastro-intestinal symptoms (e.g. nausea) experienced during pregnancy may affect food intake.[63]

Food intake was determined using the Food Frequency Questionnaire (FFQ) and 24 hour recall methods.[4,12] The FFQ enquires about the frequency of foods consumed (e.g. every day, twice a week, seldom) and gives a good indication of a individual's usual food intake over a long period of time.[65] Foods are grouped together under headings such as "breads and spreads", "vegetables" and "porridges". Since the list covers a wide variety of foods, it helps the individual to remember items not often eaten, or "hidden" food items such as condiments.[4,12]

The FFQ was undertaken at the 1st interview to determine usual food intake since the start of pregnancy until the time of the 1st interview. A FFQ was repeated at the last interview.

The 24 hour recall was undertaken at all 3 interviews. The 24 hour recall asks the individual to list all the food and drink consumed in the last 24 hours.[66] Ideally, two 24 hour recalls are recommended for weekdays and one for a weekend day, but because this study had to be incorporated time wise with the FARR studies, this was not always possible. The average of three 24 hour recalls gives a good representation of an individual's usual food intake over the study period. By using both methods of dietary intake assessment, a more accurate estimate of usual food intake was obtained for this study population.[4,12]

Only the investigator completed the questionnaires with the individuals. She is a qualified dietician and trained in administering FFQ's, 24 hour recalls and indicating portion sizes. Food models, serving spoons, plates, bowls, cups etc were used to help the individuals indicate how much of a certain food was consumed.

The questionnaire was validated as described in the pilot study and validation study sections.

2.9.2 Anthropometric Assessment

Measurements were taken according to the schedule in *Table 2.2*.

- Weight

For the purposes of this study, if the pre-pregnancy weight was not known and or could not be obtained from clinic records, then the weight recorded during the first trimester of pregnancy, either at Hanover Park MOU or during the first visit to FARR, was used.[67] Weight appraisal was done at each visit to FARR, i.e. three times in total. If the pre-pregnancy weight was known, pre-pregnancy Body Mass Index (BMI) was calculated, and used to determine if weight gain between visits was adequate in the different trimesters of pregnancy.

A portable Soehnle scale (Leifheit©) was used to determine the individuals' weights.
Individuals were weighed as follows:

- without shoes;
- under standard conditions (before meals, after emptying bladder);
- to the nearest 0.1kg;
- scale was standing on a flat, hard surface and was calibrated regularly – every time it was moved and after every 10-20 measurements using 5kg bags of sugar;
- person stood relaxed in the middle of the platform of the scale without support, person looked straight ahead; and
- repeated three times and the average of the three readings is taken as the weight.[67]

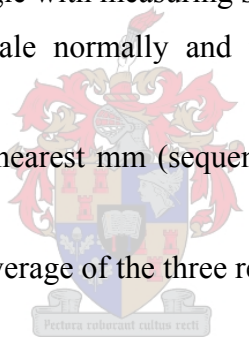
- Height

If pre-pregnancy height was not known, the woman's height was measured during the first visit to FARR. Height was used to calculate BMI.[63,67] Height does not change significantly within the first half of pregnancy, and can be used accurately as pre-pregnancy height. During the second half of pregnancy posture changes occur because of the extra weight being carried that will influence height.[63] Therefore, height was only measured during the 1st interview of the study.

A Leicester height measure (CMS weighing equipment©) standing perpendicular to the floor was used to measure height.

Individuals were measured as follows:

- no shoes, hair coverings, hair bands and/or combed up hair;
- person stood up straight with her feet together and arms relaxed alongside the body;
- the knees were together and legs straight;
- Frankfurt lines formed 90° angle with measuring stick;
- person was requested to inhale normally and measurement was taken on maximum inspiration;
- the reading was taken to the nearest mm (sequential measurements was not to differ by more than 5mm); and
- repeated three times and the average of the three readings is taken as the height.[67]



- Mid-upper Arm Circumference (MUAC)

This measurement reflects maternal fat and lean tissue stores. It correlates well with pre-pregnant weight, because it is less sensitive to short-term changes in nutritional status.[67,68]

A non-tear, stretch-resistant, plasticised measuring tape was used to determine MUAC. It is an easy, inexpensive way to measure nutritional status.[67]

Individuals were measured as follows:

- on the right arm of the patient;
- reading was taken on the horizontal line at the mid-point of the arm, with the arm hanging relaxed at the patient's side;
- the mid-point of the arm was determined by measuring the distance between the individual's acronium and olecranon, while the arm was held to the side of the body, the

elbow bent at a 90˚ angle with the palm facing upwards. The distance is halved and this distance from the acronium is taken as the mid-point of the arm;

- soft tissue was not compressed;
- measurement was read to the nearest mm; and
- repeated three times and the average of the three readings is taken as the MUAC.[67]

2.9.3 Clinical Assessment

The clinical examination (*Table 2.3*) focused on the detection of muscular wasting and signs of nutrient deficiencies, specifically vitamins A, B's, C, folate, iron and zinc.

Table 2.3: Clinical signs of specific nutrient deficiencies and toxicities

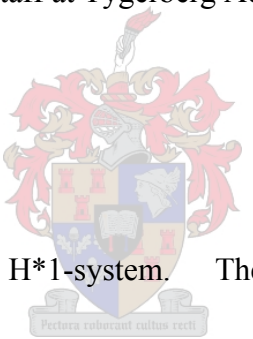
DEFICIENCIES/TOXICITY	CLINICAL SIGNS
Muscle wastage	Musculature of thumb, fore arm, upper arm, face (temporal and orbital muscles), chest, upper and lower legs
Carotenoids	Palms: (very rare) Hypercarotenodermia
Vitamin A	Fore arm and upper leg: Follicular Hyperkeratosis Eyes: Night blindness, Xerosis, Bitot's Spots, Xeropthalmia
Thiamin (Vitamin B1)	Eyes: Nystagmus Lower leg: calf muscle tenderness Feet: "Foot drop"
Pyridoxine (Vitamin B6)	Lower leg: calf muscle tenderness Eyes: redness and fissuring of eyelid corners
Vitamin C	Scurvy (very rare): Follicular Hyperkeratosis, swollen and inflamed gums, loss of hair, dry itchy skin Skin: bleeding, i.e. petechiae, purpura, ecchymoses
Folate	Mouth: Glossitis
Zinc	Head: hair loss Skin: lesion (impaired wound healing)
Iron	Iron deficiency Anaemia: Spoon-shaped nails Anaemia (e.g. Iron deficiency): pale conjunctiva

Source: Mahan LK, Escott-Stump S. Krause's Food, Nutrition and Diet Therapy, 9th Ed. WB Saunders, Pennsylvania, 1996: 963, 964

2.9.4 Biochemical Assessment

For the purposes of this study serum haemoglobin (Hb), haematocrit (HCT), ferritin, plasma vitamin A, folate and zinc were determined. A qualified nurse of FARR drew blood during the 1st visit at FARR. In total 3 tubes of blood (15ml) were needed, a purple EDTA K3 vacutainer, green lithium LH vacutainer, and a red Z serum clot activator vacutainer. Hb and HCT were tested for during the initial registration or "booking" visit at the MOU; a full blood count (FBC) was done as part of the protocol at Hanover Park MOU. Biochemical analysis of the FBC was undertaken by provincial laboratories that have recently been assimilated nationally as the National Health Laboratory Services. Plasma vitamin A, folate and serum ferritin were analysed for this study by the staff of the human nutrition laboratory of Tygerberg Academic hospital. Plasma zinc was analysed for this study by the department of Chemical Pathology's laboratory staff at Tygerberg Academic hospital.

2.9.4.1 Biochemical methodology



- Hb

Hb was determined using the H*1-system. The haeme fraction is read using the colorimeter.[26]

- HCT

HCT is not directly measured and analysed, but was calculated from measured Hb, RBC and Mean Corpuscular Volume(MCV). The HCT is calculated using the following formula.[26]

$$\text{Haematocrit (\%)} = \frac{\text{MCV x RBC (10)}}{10}$$

- Ferritin

Serum ferritin levels were determined using Radioimmunoassay kits.[69] The coefficient of variation (CV) was 3.4%.

- Vitamin A

Plasma levels were determined using High Performance liquid Chromatography according to the Vuilleumier method.[69] The CV was 4.6%.

- Folate

Plasma levels were determined using the solid-phase no-boil dual-count radioassay kit (which usually determines the vitamin B12 value simultaneously).[69] The CV was 3.8%.

- Zinc

Plasma levels were determined by the Perkin Elmer method on standard atomic absorption.[69] The CV was 2.37%.

2.9.5 Assessment of the newborn

Mothers visited FARR with their babies at 1, 6, and 12 months of age as part of the Maternal-Infant Study. For the purposes of this present study, measurements were taken at the 1-month visit. The following information was collected from each baby:

- Termination of pregnancy, prematurity and multiple births

1. Terminated in abortion or miscarriage at what gestational age?
2. Stillbirth?
3. Term (38-42 weeks) or premature birth (<38 weeks) and what was the gestational age?
4. Singleton or multiple birth?

- Anthropometric assessment of newborn

The birth weight of the baby was obtained from the mother's patient summary or the baby's Road to Health Chart (RTHC). The obstetric staff of Hanover Park MOU are trained in taking babies' weights and the scale at Hanover Park is regularly calibrated and in good working order.

The weight of each baby was determined as follows using a Seca baby scale (Precision Weighing Balances©):

- zero calibration was done before taking the measurement;
- baby was weighed without clothes or shoes;
- wait till the baby was lying still; and
- reading taken to the nearest g.[67]

The head circumference of each baby was determined as follows with a non-tear, stretch-resistant, plasticised measuring tape:

- all head coverings were removed;
- tape measure was correctly placed (just above the supra-orbital ridge and over the occiput giving the highest reading.[67]

Weight and head circumference of each baby was determined at the 1 month visit at the FARR offices by a paediatrician trained to recognise signs in the young relating to maternal alcohol abuse. He is experienced in taking anthropometric measurements on infants.

- Diagnosis of Fetal Alcohol Syndrome (FAS)

A paediatrician from FARR experienced in the diagnosis of FAS evaluated the babies (at one month of age) to determine whether or not they were affected with FAS, or had any alcohol related signs. However, it is clinically difficult to diagnose FAS in the infant. An accurate diagnosis is made more easily between 3-10 years of age.[56] Any other diagnosis made during the time of data collection is included in the results. Follow-up in years to come will give a more accurate reflection of the incidence of FAS or it's sub-classifications in this population. The status of the baby was classified as "yes" (child has FAS), "no" (the child does not have FAS) or "deferred" (the child has significant features of FAS but they are insufficient for a confident diagnosis of FAS) (*Appendix 4*).

- Other physical or mental defects

Any other defects, either noted by the obstetric staff of Hanover Park MOU or by the paediatrician from FARR were documented. Specific attention was paid to the identification of Spina Bifida.

- Delivery

Problems that have been previously associated in the literature with heavy drinking and/or nutrient-deficiencies were also noted, such as:

- pre-eclampsia (vitamin C);[23]
- intra-partum haemorrhage (zinc);[26] and
- low apgar scores (heavy drinking).[56]

2.10 Pilot study

The pilot study (*Appendix 5*) was performed at Bishop Lavis MOU. After approval was obtained from the senior medical superintendent (*Appendix 6*), community health services organization of PAWC, the investigator visited the MOU and interviewed 3 women on 3 consecutive Fridays. All methodology, except the biochemical and alcohol consumption assessment was conducted during the pilot study. The women were all between the ages of 18 and 40 years, of the Mixed Ancestry race group and pregnant.

Analysis of the pilot study data, indicated significant differences in the nutrient intake as obtained from the 24 hour recall and the FFQ questionnaires (*Appendix 7*). In theory, the FFQ values were to be compared with the average of three 24 hour recalls, but in practice the pilot study's constraints made that impossible, because only one 24 hour recall was completed. The study leaders recommended that the investigator had to conduct a separate trial to determine the validity and reliability of the dietary questionnaires.

The dietary questionnaires were retested on a conveniently selected sample of 9 women in the validation study (*Appendix 8*). The women were between the ages of 18 and 40 years, of the Mixed Ancestry race and non-pregnant. The questionnaires were administered on 3 occasions over a period of a week. Analysis of the data (*Appendix* 9) under these experimental conditions indicated that the nutrient intake values obtained from the 2 types of dietary questionnaires were indeed comparable and consistent and, therefore, valid in determining dietary intake. Due to time limitations the questionnaires could not be validated over a longer period of time that would have been more similar to the research study's time frame.

2.11 Research study

2.11.1 Times and dates

Times for data collection for the study were chosen to coincide with those chosen by FARR for their studies, to prevent unnecessary expenditure. Appointments were either in the mornings or early afternoon. Data collection started on the 26th October 2000 with the recruitment of the first individual and ended on the 11th July 2002 with the last individual and her baby's postnatal visit and interview. The study's duration was 1 year and 10 months.

2.11.2 Area

The study area was predetermined by FARR as Hanover Park MOU. Data collection took place at the FARR offices on UCT medical campus.

2.11.3 Sample

Initially a sample of 50 matched subjects and controls were to be included in the study. Due to time constraints and less recruitments at the time of the study, the sample size was later reduced to 15 matched subjects and controls, derived out of a total population of 47 individuals. The subjects and controls were matched according to gestational age, parity and ethnic group.

2.11.4 Procedure

Each individual was recruited by a FARR health worker at Hanover Park MOU, and an interview was conducted with her at the MOU or at another convenient location. This study's investigator conducted 3 interviews with the women at the FARR offices. Each individual and her baby visited the FARR offices for a postnatal visit and interview. The interviews are discussed separately (*Appendix 10*).

2.12 Analysis of data

2.12.1 Statistical analysis

Consulting statisticians advised the use of descriptive statistics.

Means, standard deviations, medians and interquartile range values were obtained of all dietary data. The non-parametric sign test was used to determine the difference (p-values) between the subject and control populations' dietary intake. This test is preferred with small sample sizes, and it was a pair-wise comparison between subjects and controls.

Correlations were determined with the Spearman correlation test, also used because of the sample's small size. The Spearman correlation test determines whether the null hypothesis ($p=0$) or the alternative hypothesis ($p\neq0$) is true. If $p<0.05$, then the null hypothesis is rejected and the correlation is meaningful.

The non-parametric Kruskal-Wallis test, Willcoxon rank test and Fischer's Exact test were used on the rest of the data to determine any significance between different groups of data. It also has significance if these tests give a $p<0.05$. This test is preferred with small sample sizes. The Willcoxon rank test is preferred when 2 groups of data are compared, and Kruskal-Wallis test is preferred if there are multiple comparisons made.

2.12.2 Analysis of dietary data

The dietary intake sheets were processed with the help of the MRC Food Composition Tables so that food portion sizes were expressed as grams of a certain foodstuff per day.[70] The dietary analysis programme, Food Finder 2©, was used to analyse the food intake of the 24 hour recalls and FFQ's into nutrient values. The following nutrient values were of specific interest to the investigator, because of its role in pregnancy and alcohol-related nutrition: energy, protein, carbohydrates, fat, vitamins A, D, E, K, C, thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, calcium, phosphorous, magnesium, iron, zinc, iodine and selenium.

The 24 hour recalls and FFQ's were analysed separately. The absolute values of nutrients were compared to the 1989 values of RDA's to be expressed as a percentage value of the RDA. Cut-off values of < 66.67% and > 133.33% of the RDA were used to indicate a possible inadequate (deficient or toxic respectively) intake of the specific nutrients.

2.12.3 Anthropometric Data Processing

Weight and height is used to calculate BMI.[68]

$$BMI = \frac{\text{weight (kg)}}{\text{height (m)}^2}$$

BMI is a simple, reliable and easy accessible measurement of nutritional status in adult persons.[67] The BMI was then classified (*Table 2.4*) according to the indices of the World Health Organisation (WHO).[71]

Table 2.4: BMI Classification

	Undernourished			***Normal***	***Overnourished***		
	Severe	*Mild*	*Slight*		*Grade 1*	*Grade 2*	*Grade 3*
Women	<16	16-16,9	17-18,49	18,5-24,9	25-29,9	30-39,9	>40

Source: WHO Expert Committee. Physical status: The use and interpretation of anthropometry. Report of a WHO expert committee, WHO, Geneva, 1995

According to the pre-pregnancy BMI of the women, there are specific weight gain goals to be attained during pregnancy (*Table 2.5*).

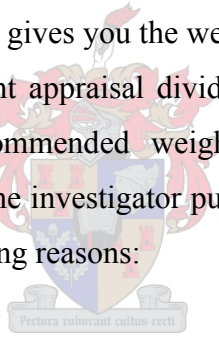
Table 2.5: Recommended Weight Gain during Pregnancy based on B.M.I.

Weight Category based on BMI	**Total Weight Gain (kg)**	**1st Trimester* Gain (kg)**	**2nd & 3rd Trimester* weekly Gain (kg)**
Underweight (BMI < 19,8)	12,5-18	2,3	0,49
Normal Weight (BMI = 19,8-26)	11,5-16	1,6	0,44
Overweight (BMI > 26-29)	7-11,5	0,9	0,3
Obese (BMI > 29)	6		

Source: Kaiser LL, Lindsay Allen. Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome. JADA Oct. 2002; 102(10): 1479-1490

***** 1st trimester = 0-12 weeks
2nd trimester = 12-28 weeks
3rd trimester = 28 weeks – end of pregnancy

Weight appraisals were done 3 times theoretically. The difference of the 2nd from the 1st, and the 3rd from the 2nd weight appraisal gives you the weight gained, or in some cases, lost over a certain period of time. That weight appraisal divided by the period of time (in weeks), is interpreted according to the recommended weight gain (*Table 2.5*) according to the individual's BMI. In more detail, the investigator put individuals in the different weight gain interpretation groups for the following reasons:



- "correct": when the weight gained was ± 0.05kg/week from the recommended weight gain,
- "positive": when weight gain did take place during pregnancy, but was below the recommended weight gain,
- "fast": when weight was more than 0.05kg/week from the recommended weight gain (The average increased weight gain for the "fast" group was 0.2kg/week.), and
- "negative": when there was no weight gain, or in some cases when there was weight loss during pregnancy.

If pre-pregnancy weight or a weight measurement in the first trimester was not available, BMI was not used to determine nutritional status. Instead, MUAC was used to determine nutritional status. There are percentile tables (*Table 2.6*) available for classifying MUAC values as an individual's nutritional status.[72]

Table 2.6: Nutritional Status classification according to MUAC

Nutritional Status for Females	Mid-upper Arm Circumference
Undernourished	* < 5th percentile
Risk for Undernutrition	* 5th – 10th percentile
Borderline Undernutrition	* 10th – 25th percentile
Normal	* 25th – 75th percentile
Borderline Overnutrition	* 75th – 90th percentile
Risk for Overnutrition	* 90th – 95th percentile
Overnutrition	* > 90th percentile

Source: Frisancho AR. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981; 34: 2540

2.12.4 Biochemical analysis

Blood Hb, HCT and serum ferritin values are used to determine iron deficiency or iron deficiency anaemia (IDA).[63]

Anaemia cut-off values are indicated in *Table 2.7*:

Table 2.7: Indices for Anaemia during Pregnancy

PREGNANCY STATUS	Haemoglobin(g/l)	Haematocrit(%)
1st trimester	110	33
2nd trimester	105	32
3rd trimester	110	33

Source: West SC. Nutritional assessment of the pregnant woman. Clin Obs & Gyn 1994; 37(3): 502-511

There are also adaptations that must be made if the women were smokers (*Table 2.8*) or live at high altitudes.[63] It was not necessary to adapt for altitude in this study, since Cape Town is situated at the coast.

Table 2.8: Hb and HCT values when smoking during pregnancy indicating anaemia

PREGNANCY STATUS	Cigarettes per day					
	0-9		10-20		21-40	
	Hb(g/l)	HCT(%)	Hb(g/l)	HCT(%)	Hb(g/l)	HCT(%)
1ST Trimester	110	33	113	34	115	34.5
2nd Trimester	105	32	108	33	110	33.5
3rd Trimester	110	33	113	34	115	34.5

Source: West SC. Nutritional assessment of the pregnant woman. Clin Obs & Gyn 1994; 37(3): 502-511

Anaemia accompanied by a low serum ferritin (<12.0µg/l) indicates IDA.[26] Serum ferritin of <20µg/l indicated iron deficiency, if the Hb value was >105g/l. It is not recommended in the literature to determine serum ferritin during the 3rd trimester, because the serum ferritin concentration decreases independently of iron status due to the expansion of plasma volume.[63]

Criteria was determined for plasma vitamin A, folate and zinc values during pregnancy at which possible deficiency consequences could arise (*Table 2.9*).[69]

Table 2.9: Criteria for Nutrient deficiencies in Pregnancy

Nutrient:	High Risk	Moderate Risk	Low Risk
Plasma Zinc(µmol/l) 1st & 2nd Trimesters	<7.9	7.9-10.5	>10.6
Plasma Folate(nmol/l)	<6.8	6.8-13.5	>13.6
Plasma Vitamin A (µmol/l)	<0.35	0.35-1.04	>1.05

Source: Ackurt F, Wetherilt H, Loker M, Hacibekroglu M. Biochemical assessment of nutritional status in pre- and post-natal Turkish women and outcome of pregnancy. Eur J Clin Nutr 1995; 49: 615

2.12.5 Newborn anthropometric assessment

Birth weight was classified according to the following parameters, that are independent of sex and gestational age at birth:[67]

- low birth weight (LBW)= < 2500g;
- very low birth weight (VLBW)= <1500g; and

- extremely low birth weight (ELBW)= <1000g.

Weight for age (W/A) and head circumference for age (HC/A) were determined at the age of ± 1 month using sex-specific percentile charts for birth to 36 months age (*Appendices 12, 13*). The indices used for classifying the babies' anthropometric values are:

- <5th percentile (underweight);
- 5th – 50th percentile (bottom range of normal);
- 50th – 95th percentile (upper range of normal); and
- >95th percentile (overweight).[67]

Through research, piloting the methodology and sharing information with FARR, the data that was acquired in this study is of very high quality.

3. RESULTS

3.1 Sample

Of the 47 individuals recruited in this study, 15 subjects could be matched with controls (*Table 3.1*). The individuals were matched according to their race, gestational age at the onset of their participation in the study, and their parity. It was not necessary to match the women according to ethnicity as all of the women were of the Mixed Ancestry population.

Table 3.1: Fifteen matched pairs of subjects and controls according to their gestational age and parity.

SUBJECT NUMBER	Parity	Gestational age	CONTROL NUMBER	Parity	Gestational age
3	0	16	5	0	15
4	0	21	42	0	21
8	0	18	30	0	18
15	0	18	37	0	18
16	0	19	11	0	19
21	1	8	2	1	12
22	1	12	9	1	14
23	1	21	14	1	18
24	2	19	18	2	20
25	1	20	40	1	18
31	0	24	19	0	21
34	1	19	29	1	18
39	0	19	44	0	17
46	0	18	32	0	19
47	3	20	13	3	20

Therefore there were 17 unmatched women who contribute to the total study population, and were not allocated as either subjects or controls (*Appendix 14*).

The findings of the study are presented for the paired subjects and controls (n=15 in each group) as well as the total study population (n=47). The most important results are displayed forthwith. *Appendix 15* contains a more comprehensive version of the results chapter.

3.2 Questionnaire

Forty-six individuals completed the questionnaire (*Appendix 3*). The statistical significance of differences and correlations is indicated when it exceeded the 5% level of significance.

3.2.1 Socio-demographics

One hundred percent of the total population (n=47) were of the Mixed Ancestry ethnic group. The control mothers were inclined to be slightly older than the subjects (*Table 3.2*).

Table 3.2: Descriptive statistics regarding age of the different population groups

Subjects (n=15)		Controls (n=15)		Total (n=46)	
Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)
24.93 (4.43)	26.00 (21.00 – 28.00)	25.07 (6.70)	24.00 (19.00 – 30.00)	25.23 (5.89)	24.00 (20.00 – 29.00)

The living space of the subjects was a mean of 5.8 people living together in a house, compared to the controls who were 5.4 people per household (*Table 3.3*).

Table 3.3: Descriptive statistics regarding people per house and per bedroom of the different population groups

	Subjects (n=15)		Controls (n=15)		Total (n=46)	
	Per house	Per bedroom	Per house	Per bedroom	Per house	Per bedroom
Mean	5.80	3.80	5.40	3.07	5.72	3.28
Std Dev	3.14	2.70	3.68	1.75	3.51	1.96
Median	5.00	3.00	5.00	3.00	5.00	3.00
q1-q3	3.00- 8.00	3.00- 4.00	3.00- 7.00	2.00-4.00	3.00- 7.00	2.00- 4.00

Of the total population (n=46), 21 mothers were working during pregnancy (45.7%). Thirty-three point three percent of the subject population (n=15) and 46.7% of the control population (n=15) were employed during pregnancy. Therefore, approximately half of the total

population had no income (*Table 3.*4) of their own and were dependant on their spouses or family to support them.

Table 3.4: Descriptive statistics regarding the income of individuals in Rands of the different population groups

	Subjects (n=15)	Controls (n=15)	Total (n=46)
Mean	474.00	1094.57	643.87
Std Dev	966.60	1379.92	1049.63
Median	0.00	488.00	0.00
q1 – q3	0.00 – 600.00	0.00 – 1496.00	0.00 – 900.00

3.2.2 Health history

The majority of the population was healthy before pregnancy. Four subjects had chronic diseases such as asthma, hypertension or Tuberculosis. One control had asthma and anaemia. The only severe acute illness in the population was a control who had had meningitis.

3.2.3 Obstetric history

Of the total population (n=46), 45.6% were expecting their first child at the time of the study. The remaining women had one to five offspring (*Table 3.5*).

Table 3.5: The incidence of different parities of the population groups

Parity:	0	1	2	3	5
Subjects (n=15)	9	4	1	1	0
Controls (n=15)	8	5	1	1	0
Total (n=46)	21	15	6	3	1

3.2.4 Lifestyle

Most mothers did not consume supplements during pregnancy (60.9% of the total population, 80% of the subjects and 46.7% of the controls), but among those who did mineral (specifically iron sulphate) and multivitamins was the commonly consumed supplement. (*Figure 3.1*)

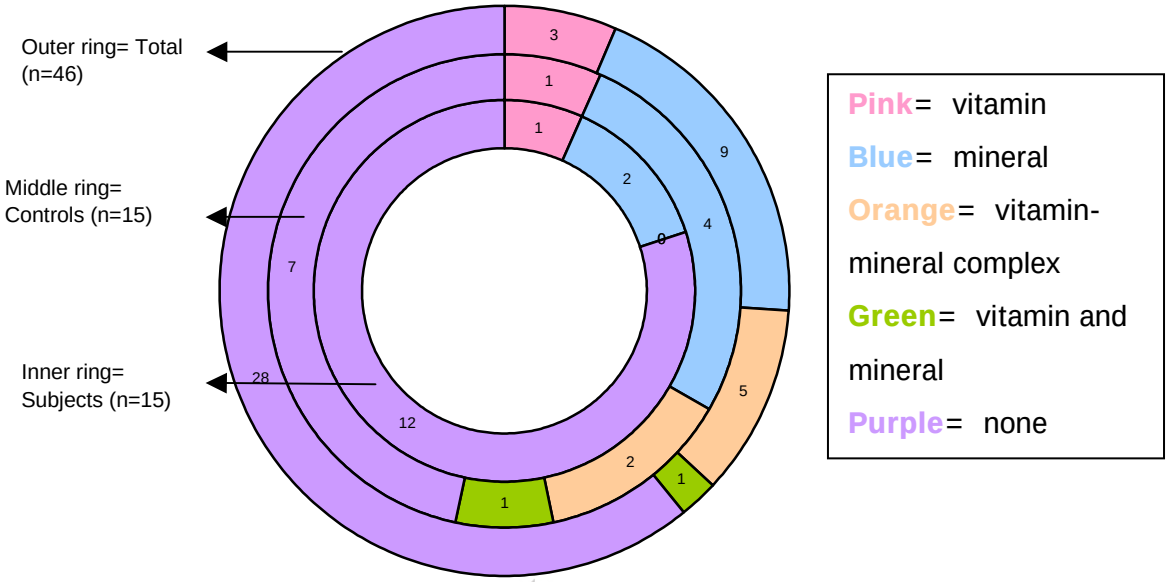


Figure 3.1: The comparison of micronutrient supplement practices during pregnancy among the different population groups

A recent study within the same area and type of study population as this one documented a very high incidence of cigarette smoking during pregnancy.[60] This, especially in the subject group, was also found by the investigator (*Table 3.*6).

Table 3.6: The incidence of smoking and the amount smoked per day by individuals in the population groups

Number of cigarettes smoked per day:	Subjects (n=15)	Controls (n=15)	Total (n=46)
1-4	6	4	15
5-10	7	1	17
Non-smoker	2	10	15

3.2.5 Food security

The availability of certain facilities as specified below (*Table 3.7*), influences the household's ability to safely store, prepare and consume food. The majority of the population groups had all the specified facilities.

Table 3.7: The availability of facilities among the different population groups

Facilities	Subjects (n=15)		Controls (n=15)		Total (n=46)	
	Yes	No	Yes	No	Yes	No
Running water	11	4	14	1	37	9
Electricity	13	2	15	0	41	5
Stove (electrical or gas)	14	1	15	0	45	1
Fridge	11	4	15	0	38	8
Toilet	11	4	10	5	33	13

Through a questionnaire (*Appendix 3, section E.2*), it was possible to determine the individual's food security status.[73] A significantly lower number of subjects was classified as being food secure when compared with controls (*Table 3.8*).

Table 3.8: The incidence of individuals who had different food security statuses in the population groups

Food security status:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Food secure	7*	14*	32
Food insecure	4	0	8
Food insecure with hunger evident	3	1	5
Food insecure with severe hunger evident	1	0	1

* Significant difference according to Fischer's Exact test of $p < 0.0233$.

3.2.6 Eating habits influenced by pregnancy

When asked whether the women thought they ate more, less or the same during their pregnancies than before the present pregnancy, approximately half (46.7%) of the total population felt that they ate more during pregnancy. Thirty five point five percent felt that they less during pregnancy, with only 18% of the individuals reporting that they ate the same amount of foods during pregnancy than before.

Only one woman claimed not to have experienced any gastro-intestinal symptoms during her pregnancy. The remainder of the total population experienced nausea (47.83%), constipation (47.83%) and heartburn (65.22%) as the most frequent gastro-intestinal symptoms. (Many women experienced more than one symptom.) There was a statistical significant difference of a higher incidence of gastro-intestinal symptoms in the subject population than the controls, according to the Fischer's Exact test ($p<0.0019$) and the Chi-square test ($p<0.04$).

Food aversions that developed during pregnancy was not as common as developing a food craving (56.5% compared with 87% of the total population). The top 3 foods most avoided were spicy foods, meats and fish. The top 3 foods most craved for were vegetables, sweets and chocolates and dairy products.

3.3 Dietary intake

The study aimed at having three 24 hour recalls and two FFQ's completed with each individual, which was realized except in a few cases (*Table 3.9*).

Table 3.9: Numbers of different dietary questionnaires completed in the different population groups

Dietary questionnaires	Subjects (n=15)	Controls (n=15)	Total (n=47)
24 hour recall no. 1	15	15	47
24 hour recall no. 2	15	15	46
24 hour recall no. 3	14	15	42
FFQ no. 1	15	15	46
FFQ no. 2	15	15	43

The analysis of the individual questionnaires is presented in *Appendix 15*. The ensuing table concentrate on the average intake of nutrients of the 24 hour recall questionnaires and the FFQ's. The nutrient values are expressed as absolute values (*Table 3.10*). The nutrient values were also expressed as percentages of the 1989 RDA, since DRI's were not available at the time the study findings were analysed (*Figures 3.2-3.5*). Overall, the nutrient intake was higher when obtained by the FFQ, compared with the 24 hour recall questionnaire. However, none of these differences were statistically significant. In terms of adequacy,

vitamin D, folate, calcium and iron had values below 66.67% of the RDA in all the population groupings. On the other hand vitamin B12 and C had values far exceeding 133.33% of the RDA. The only significant differences between nutrient values of the subjects and controls were in vitamin D according to the 1st FFQ and in selenium according to the 2nd FFQ questionnaire (*Appendix 15*), which was considered to be incidental rather than of any nutritional significance. The contribution of micronutrient supplements was not included in the nutrient intake analysis.

Table 3.10: The mean, SD, median and q1-q3 absolute values of the specified nutrients as the averages of the three 24 hour recalls and 2 FFQ's

Variable as absolute values	Subjects		Controls		Total	
	24 hour recall	Food frequency	24 hour recall	Food frequency	24 hour recall	Food frequency
	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)
Energy (kilo-joules)	13246.81 (11241.28) 9472.33 (7241.33-14354.67)	17724.73 (9893.34) 15344.00 (11908.50-21640.00)	9612.04 (3119.10) 9824.67 (7431.00-10621.00)	18615.54 (18066.81) 13613.50 (12377.50-16908.00)	10731.17 (6985.43) 9699.50 (7431.00-11455.67)	16900.28 (11867.95) 13873.00 (11908.50-17225.00)
Protein (g)	84.00 (27.17) 81.19 (64.68-96.58)	119.57 (61.90) 106.22 (83.99-131.99)	82.28 (25.95) 80.20 (66.97-94.81)	98.17 (17.12) 98.22 (83.72-105.26)	82.40 (25.55) 82.48 (66.97-95.83)	100.48 (42.39) 96.62 (80.43-117.03)
Carbo-hydrates (g)	244.05 (72.45) 222.43 (192.98-285.93)	435.81 (170.73) 399.35 (375.80-478.95)	311.71 (99.65) 314.47 (227.03-341.83)	419.15 (79.64) 381.45 (377.40-487.90)	271.93 (76.74) 273.67 (196.57-305.33)	426.93 (114.29) 404.35 (374.05-487.90)
Fat (g)	67.75 (23.75) 62.85 (49.95-80.85)	118.66 (41.43) 127.70 (97.10-133.10)	98.11 (39.69) 82.47 (74.90-127.43)	117.58 (28.63) 118.00 (99.70-146.60)	79.63 (30.59) 76.63 (56.13-92.40)	121.03 (32.22) 125.48 (97.10-146.60)
Alcohol intake was not quantitated by FARR or by the investigator. The investigator had to be blinded to alcohol intake of study participants.						
Vitamin A (µg RE)	786.37 (798.85) 406.95 (255.81-904.24)	2051.14 (1732.35) 1532.44 (1092.48-2157.96)	745.69 (541.05) 502.05 (404.00-840.32)	1587.59 (901.19) 1334.48 (752.80-2418.96)	680.02 (576.61) 487.28 (347.20-745.60)	1629.75 (1265.34) 1213.72 (768.40-2157.96)
Vitamin D (µg)	4.61 (3.72) 3.55 (2.03-4.84)	8.57 (4.07) 8.57 (4.51-106.2)	3.43 (1.62) 3.14 (2.44-4.67)	5.28 (2.07) 5.31 (3.18-7.42)	4.13 (2.72) 3.48 (2.44-4.87)	6.94 (3.63) 6.98 (4.06-9.41)

Variable as absolute values	Subjects		Controls		Total	
	24 hour recall	Food frequency	24 hour recall	Food frequency	24 hour recall	Food frequency
	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)
Vitamin E (mg α-TE)	8.49 (3.21) 8.87 (5.39-11.23)	19.90 (10.09) 16.98 (13.02-24.49)	10.85 (9.83) 8.27 (6.71-11.47)	15.49 (6.61) 13.98 (9.40-20.16)	10.26 (6.73) 9.16 (6.65-11.94)	16.19 (7.72) 15.47 (11.42-19.37)
Vitamin K (μg)	47.33 (31.43) 29.76 (22.51-75.28)	74.80 (38.34) 64.32 (58.00-77.85)	44.49 (25.26) 40.76 (22.86-66.81)	92.08 (73.34) 69.45 (58.39-85.12)	44.80 (27.59) 33.93 (22.58-69.62)	77.98 (50.38) 67.21 (55.82-85.12)
Vitamin C (mg)	128.71 (188.85) 52.78 (31.39-158.46)	254.79 (188.28) 164.47 (89.31-426.51)	144.70 (118.95) 104.25 (55.68-246.14)	275.76 (217.91) 190.97 (111.76-357.91)	116.74 (135.86) 73.83 (34.22-145.58)	220.76 (177.77) 160.41 (95.07-291.04)
Thiamin (mg)	1.10 (0.63) 0.89 (0.68-1.33)	1.86 (0.99) 1.47 (1.23-2.49)	1.15 (0.49) 1.05 (0.84-1.38)	1.71 (0.45) 1.60 (1.41-1.91)	1.04 (0.49) 0.95 (0.70-1.16)	1.63 (0.73) 1.47 (1.25-1.84)
Riboflavin (mg)	1.41 (1.03) 1.12 (0.68-1.67)	2.95 (1.53) 2.63 (2.03-3.39)	1.57 (0.92) 1.35 (0.99-1.75)	2.56 (0.81) 2.51 (1.82-3.22)	1.42 (0.91) 1.27 (0.81-1.68)	2.53 (1.19) 2.17 (1.82-2.99)
Niacin (mg NE)	22.19 (9.11) 19.10 (17.09-26.93)	28.07 (15.61) 23.75 (19.13-32.57)	21.92 (6.49) 22.84 (17.95-26.24)	25.29 (4.59) 24.48 (22.78-27.42)	21.04 (7.68) 19.58 (16.88-26.24)	24.32 (10.60) 22.86 (19.26-27.42)
Vitamin B6 (mg)	1.79 (1.02) 1.54 (1.23-1.89)	2.74 (1.51) 2.04 (1.79-3.40)	1.75 (0.78) 1.69 (1.18-2.33)	2.43 (0.48) 2.39 (2.06-2.79)	1.66 (0.82) 1.57 (1.14-1.97)	2.37 (1.06) 2.09 (1.75-2.79)

Variable as absolute values	Subjects		Controls		Total	
	24 hour recall	Food frequency	24 hour recall	Food frequency	24 hour recall	Food frequency
	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)
Folate (µg)	236.39 (109.32) 219.40 (180.63-263.84)	482.91 (278.88) 405.60 (343.18-477.24)	249.62 (122.12) 211.84 (175.89-299.87)	431.64 (203.23) 343.80 (318.60-453.00)	231.84 (108.88) 219.40 (175.89-276.19)	409.18 (214.21) 356.02 (307.52-453.00)
Vitamin B12 (µg)	4.33 (2.71) 3.55 (2.39-5.91)	8.50 (4.25) 7.53 (5.35-10.22)	3.84 (1.66) 3.51 (2.84-5.04)	6.65 (2.67) 5.74 (4.71-7.89)	4.13 (2.08) 3.74 (2.74-5.12)	7.36 (3.56) 6.95 (4.77-9.32)
Calcium (mg)	572.08 (394.82) 445.06 (273.40-754.80)	1252.82 (975.73) 1148.40 (744.00-1305.42)	744.11 (509.57) 584.00 (352.52-871.40)	1222.31 (363.05) 1076.70 (973.98-1382.40)	614.62 (414.62) 473.28 (352.52-754.80)	1096.27 (639.08) 1002.48 (753.00-1277.64)
Phospho-rous (mg)	1127.86 (467.09) 983.20 (770.00-1283.60)	1978.71 (1294.50) 1732.92 (1258.80-2143.98)	1190.35 (466.58) 1128.20 (865.08-1401.76)	1726.40 (400.34) 1633.20 (1447.26-1868.04)	1119.05 (437.29) 1037.22 (811.60-1283.60)	1682.97 (843.35) 1593.00 (1262.04-1868.04)
Magne-sium (mg)	273.06 (110.65) 244.44 (189.24-317.12)	502.51 (342.53) 404.70 (308.00-580.24)	277.19 (98.13) 271.47 (206.72-318.78)	438.97 (124.64) 425.76 (354.91-474.88)	265.43 (99.08) 253.24 (199.25-317.12)	422.68 (229.59) 397.65 (306.05-461.97)
Iron (mg)	11.26 (5.79) 9.99 (7.24-13.87)	19.29 (9.86) 15.21 (13.47-22.83)	11.21 (4.64) 10.38 (8.88-13.03)	17.69 (6.72) 14.68 (13.35-19.86)	10.64 (4.57) 10.06 (7.49-12.21)	16.46 (7.69) 14.34 (12.30-19.68)
Zinc (mg)	11.36 (4.88) 9.92 (7.95-13.41)	17.23 (9.93) 14.69 (11.99-19.07)	11.65 (3.60) 11.53 (9.23-13.44)	14.58 (3.41) 14.26 (11.74-17.18)	11.04 (3.84) 9.94 (8.53-12.38)	14.58 (6.67) 13.95 (11.37-17.08)

Variable as absolute values	Subjects		Controls		Total	
	24 hour recall	Food frequency	24 hour recall	Food frequency	24 hour recall	Food frequency
	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)	Mean (Std Dev) Median (Inter-quartile range)
Iodine (µg)	49.52 (20.11) 46.74 (38.42-59.79)	88.22 (51.39) 81.90 (64.30-95.73)	47.93 (22.79) 39.68 (35.35-67.62)	63.45 (21.35) 62.21 (46.31-74.29)	48.36 (25.91) 41.09 (34.24-59.79)	71.83 (37.22) 66.25 (46.31-87.48)
Selenium (µg)	60.28 (20.01) 55.61 (50.85-71.00)	96.00 (42.62) 84.66 (71.92-100.95)	55.86 (17.49) 54.01 (43.23-71.98)	68.55 (22.33) 64.21 (56.49-78.33)	56.81 (19.86) 55.61 (45.12-71.00)	77.06 (33.78) 74.52 (57.28-89.57)

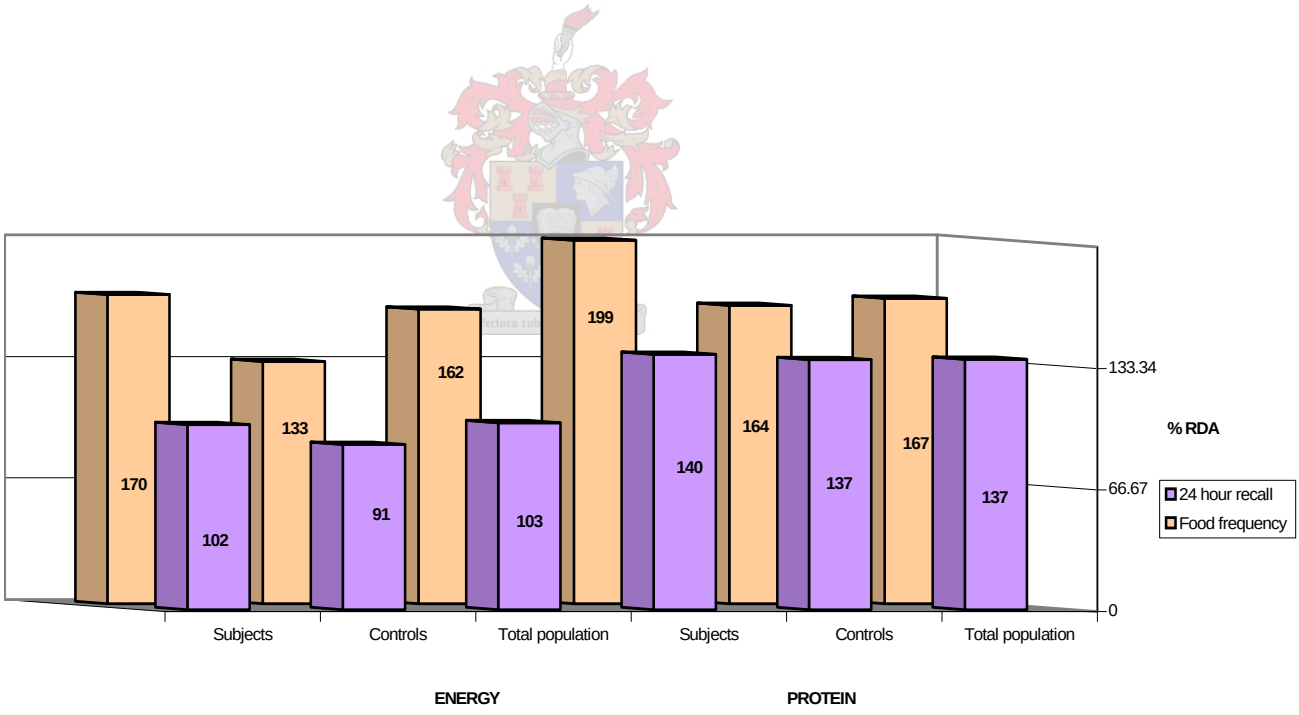


Figure 3.2: The mean % RDA values of the different population groups' macronutrients

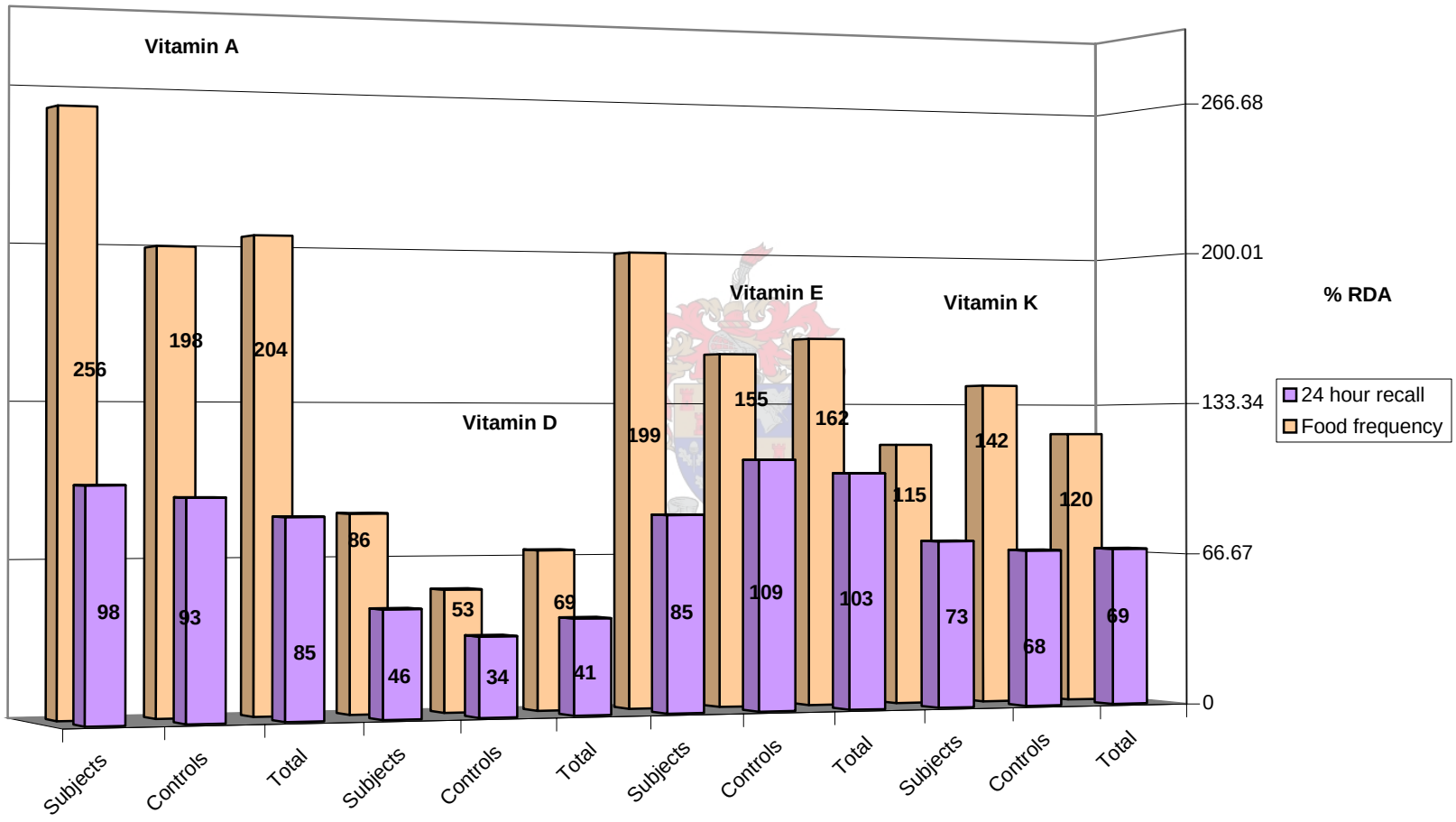


Figure 3.3: The mean % values of the different population groups' fat-soluble vitamins intake

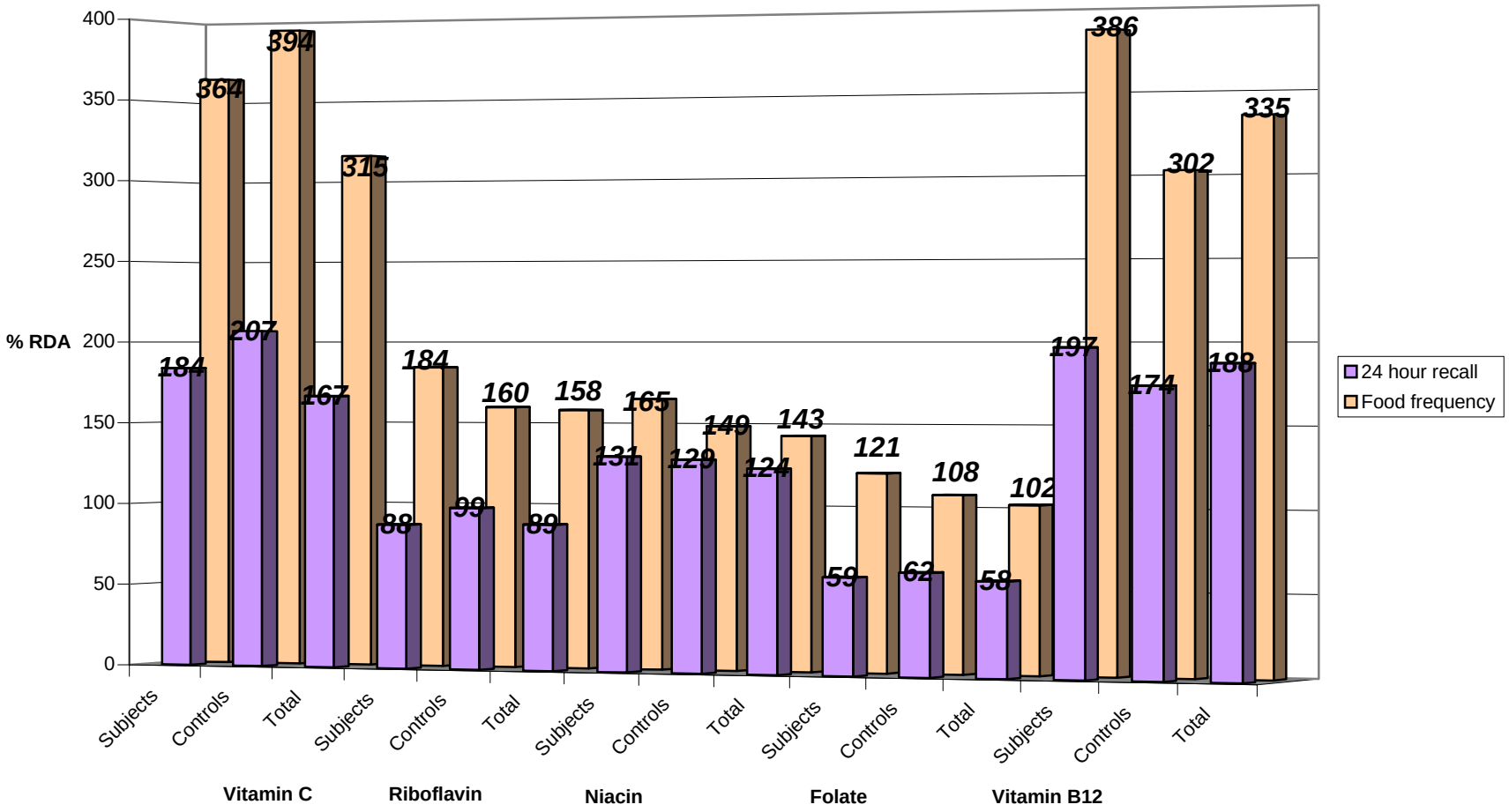


Figure 3.4: The mean % values of the water-soluble vitamins of which some intake values were inadequate

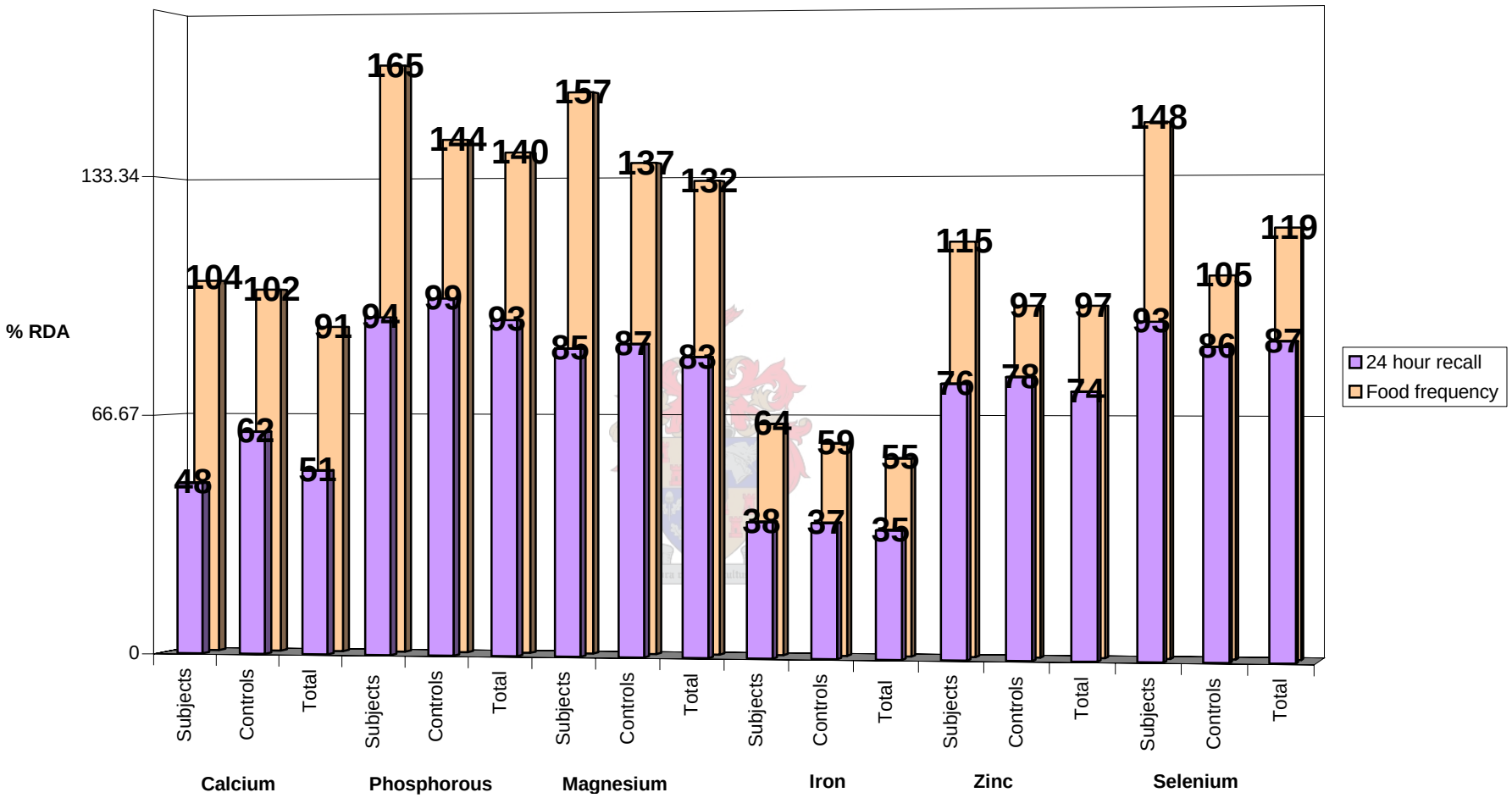


Figure 3.5: The mean % values of the different population groups' minerals and trace elements intake

Adequate daily intake is 66.67 – 133.33% RDA, inadequate intake is < 66.67% RDA and > 133.33% RDA. In *Appendix 15* is the distribution of the number of individuals in the subject, control and the total population in the different RDA groups of adequacy/inadequacy are presented. There was only a significant difference between the subject and control population's calcium intake according to the FFQ. *Figures 3.6 and 3.7* show the nutrients of which the population groups had a majority of inadequate intakes, such as low intake of iron, zinc, folate and vitamin D; high intake of protein, niacin, vitamin E, C and B12. This corresponds well with the values in *Table 3.*10. There is an apparent discrepancy regarding vitamin A, as a majority of the populations had a low intake according to the 24 hour recalls, and a majority that had a high intake according to the FFQ's.

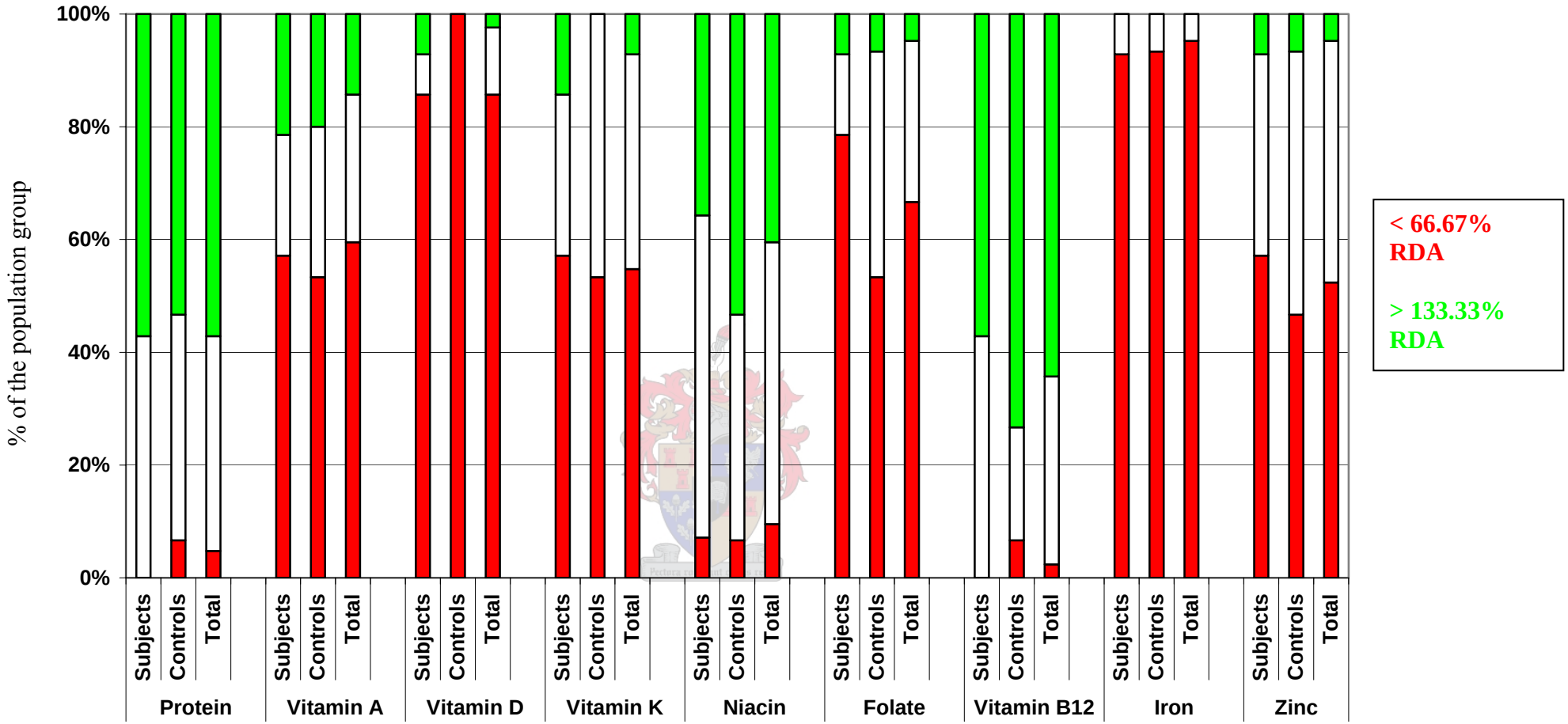


Figure 3.6: The nutrients for which there was a majority of inadequate intakes in the different study groups according to the 24 hour recall analysis

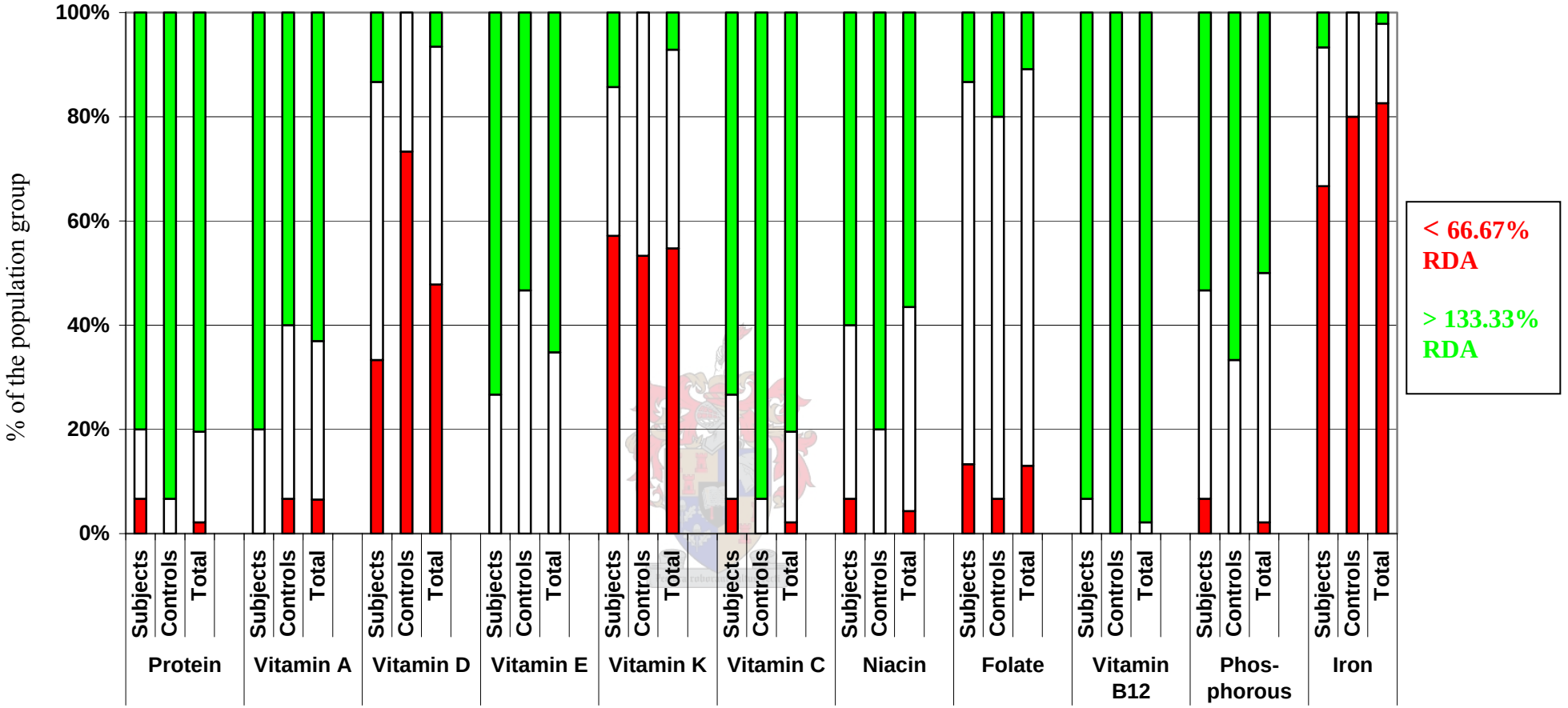


Figure 3.7: The nutrients for which there was a majority of inadequate intakes in the different study groups according to the FFQ analysis

The recommended energy distribution according to the prudent guidelines were: 55% energy from carbohydrates, 15% from protein and 30% from fat. The macronutrients' contribution to total energy as obtained from the 24 hour recalls and FFQ's (*Figures 3.8 & 3.9*) show that more fat than the recommended 30% was consumed by the study groups. There was no significance to the slight difference in energy distribution of the subjects and controls.

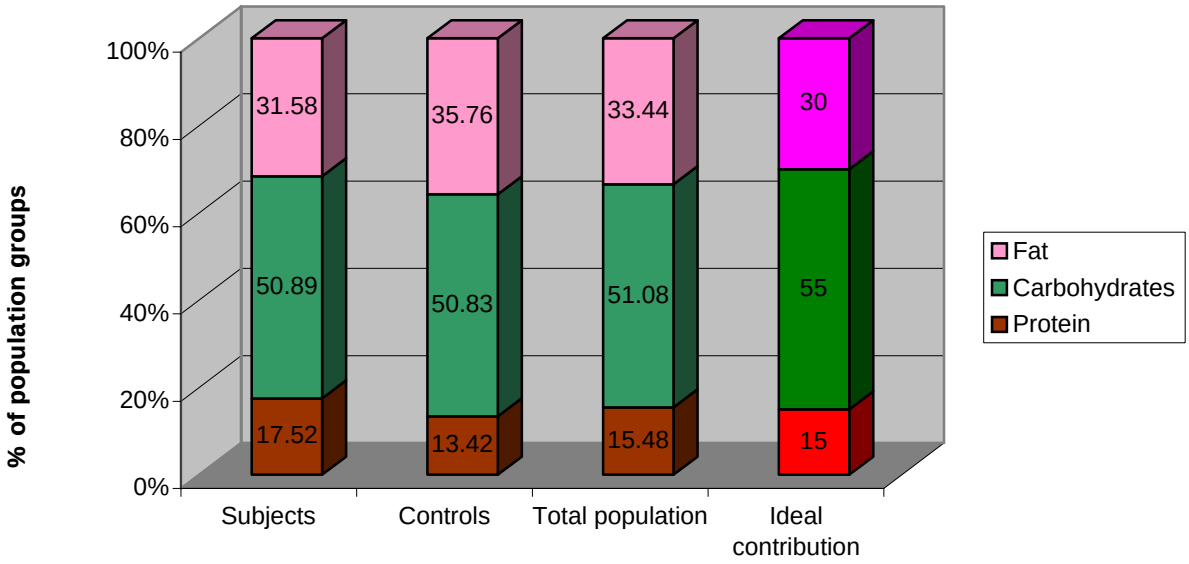


Figure 3.8: Energy contribution of macronutrients as % within different population groups according to the 24 hour recalls

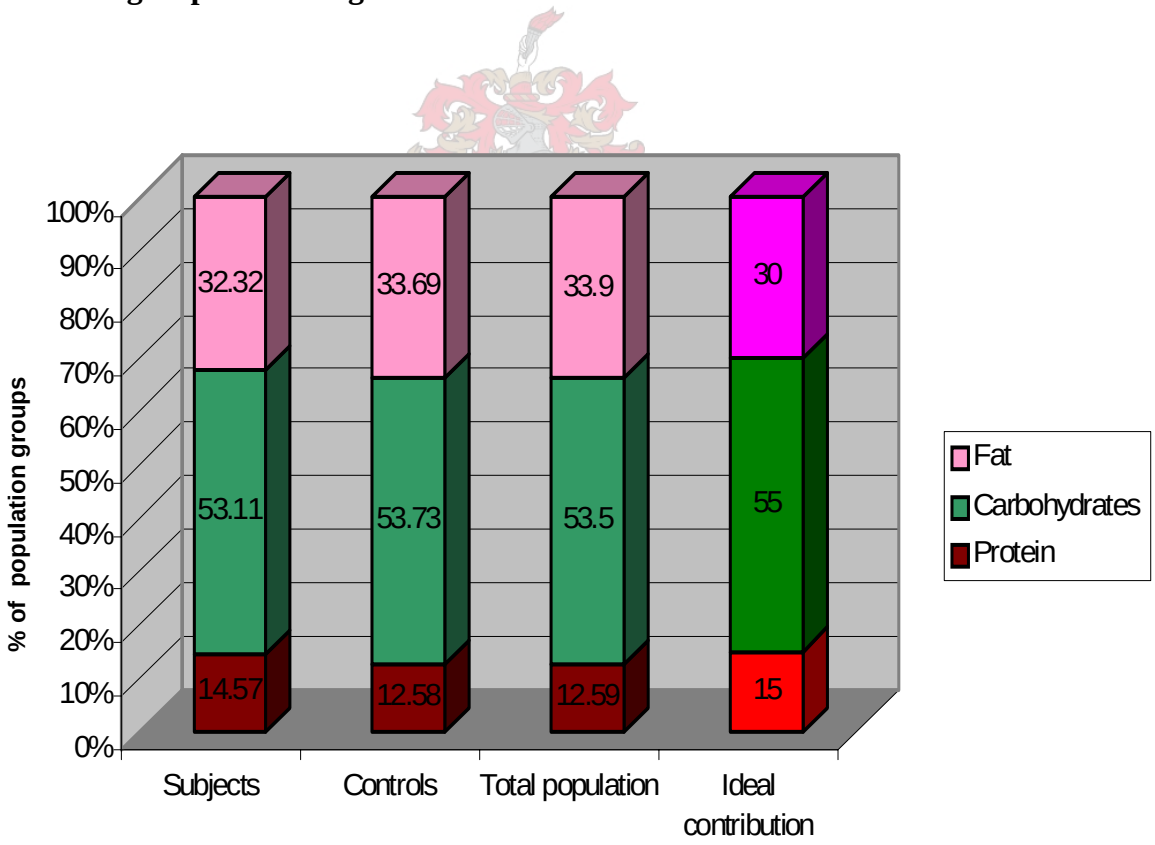


Figure 3.9: Energy contribution of macronutrients as % within different population groups according to the food frequency questionnaires

Another source of energy in the diet is alcohol. The contribution of alcohol to energy intake is not included in the above figures, since the investigator did not determine alcohol intake in her dietary questionnaire, as she had to be blinded to alcohol drinking status of the individuals.

3.4 Anthropometric results

Body Mass Index (BMI), can only be used as an index of nutritional status if pre-pregnancy weight (PPW) is known or if a weight appraisal is done within the 1st trimester of the pregnancy.[67] Almost half of the total population did not know their PPW or had a weight appraisal done in the 1st trimester (*Table 3.11*). This was problematic since pre-pregnancy nutritional status must be known to interpret if weight gain during pregnancy was appropriate or not.

Table 3.11: The number of individuals of whom the PPW or a 1st trimester weight appraisal value was known of in the different population groups

PPW or weight in the 1st trimester?	Subjects (n=15)	Controls (n=15)	Total (n=47)
"Yes"	6	8	25
"No"	9	7	22

However, the mid-upper arm circumference (MUAC) was also determined as a measure of nutritional status. MUAC does not have to be done before pregnancy or within the 1st trimester, as it is less sensitive to the relatively short-term weight changes that accompany pregnancy.[67]

3.4.1 Nutritional status distribution according to anthropometric measurements

The nutritional status distribution according to BMI (< 18.5 being undernourished, 18.5 – 25 normally nourished, and > 25 overnourished) did not differ significantly between the subject and control groups (*Table 3.12*); although there is a trend that only subjects had BMI's below 18.5. The other method of nutritional status assessment was measuring the MUAC.

Similarly, there was no statistical significance to the difference in distribution, but again only subjects had MUAC measurements of less than the 5th percentile (*Table 3.13*).

Table 3.12: Nutritional status distribution according to BMI of the different population groups

BMI classifications:	Subjects (n=6)	Controls (n=8)	Total (n=25)
< 18.5	2	0	3
18.5 – 25	3	6	16
> 25	1	2	6

Table 3.13: Nutritional status distribution according to MUAC of the different population groups

MUAC percentile groups	Subjects (n=15)	Controls (n=15)	Total (n=46)
< 5	3	0	4
5 – 10	0	0	3
10 – 25	4	3	8
25 – 75	6	9	21
75 – 90	2 *	3	7
90 – 95	1	0	3 *
> 95	0	0	0

* Two individuals with BMI's over 30, which could make their MUAC's inaccurate.

Three weight appraisals were done by the investigator on each individual. The difference between the 3 appraisals was interpreted according to which BMI grouping and trimester of pregnancy as either "correct"[a], "positive"[a], "fast"[a], or "negative"[a] (*Table 3.14*). Significantly more controls (2 compared to 0) had "fast" weight gain during pregnancy than subjects.

a:

- "correct": when the weight gained was ± 0.05kg/week from the recommended weight gain,
- "positive": when weight gain did take place during pregnancy, but was below the recommended weight gain,
- "fast": when weight was more than 0.05kg/week from the recommended weight gain (The average increased weight gain for the "fast" group was 0.2kg/week.), and
- "negative": when there was no weight gain, or in some cases when there was weight loss during pregnancy.

Table 3.14: The distribution of individuals in the different population groups according to weight gain interpretation groups

Weight gain interpretation:	Subjects (n=6)	Controls (n=8)	Total (n=25)
"Correct"	1	1	4
"Fast"	0*	2*	4
"Negative"	1	0	4
"Positive"	4	5	12
"Positive", but individual obese	0	0	1

* The statistical significance according to Chi-Square test is $p < 0.0411$.

3.5 Biochemical data

All study participants were asked to volunteer a blood sample (*Table 3.15*) in which serum folate, vitamin A, ferritin, plasma zinc and haemoglobin and haematocrit was determined.

Table 3.15: The number of individuals of the different population groups who volunteered a blood sample

Volunteer?	Subjects (n=15)	Controls (n=15)	Total (n=47)
"Yes"	11	10	30
"No"	4	5	17

3.5.1 Biochemical data distribution

The mean, standard deviation, median and interquartile range (q1-q3) of zinc, folate, vitamin A and ferritin was determined of the subject, control and total population groups presented in *Tables 3.16 – 3.19*. The values did not differ significantly between the groups, with the exception of serum vitamin A significantly lower in the control group.

Table 3.16: Zinc's values of the different population groups

Zinc (µmol/l):	Subjects (n=8)		Controls (n=10)		Total (n=25)	
	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)
	14.70 (1.88)	14.80 (13.20- 6.00)	15.14 (2.53)	14.80 (14.00- 18.00)	14.25 (2.24)	14.10 (12.30- 15.80)

Table 3.17: Folate's values of the different population groups

Folate (nmol/l):	Subjects (n=11)		Controls (n=10)		Total (n=30)	
	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)
	17.06 (18.55)	11.10 (7.48- 19.27)	12.19 (7.85)	9.97 (8.16- 18.10)	15.14 (12.98)	11.21 (8.16- 18.10)

Table 3.18: Vitamin A's values of the different population groups

Vitamin A (µmol/l):	Subjects (n=11)		Controls (n=10)		Total (n=30)	
	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)
	5.72* (13.27)*	1.58 (1.51- 2.54)	1.20* (0.31)*	1.04 (0.98- 1.30)	3.24 (8.24)	1.47 (1.06- 1.76)

* The statistical significance according to Kruskal-Wallis test is $p < 0.0097$.

Table 3.19: Ferritin's values of the different population groups

Ferritin (ng/ml):	Subjects (n=11)		Controls (n=9)		Total (n=30)	
	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)	Mean (Std Dev)	Median (Interquartile range)
	24.92 (16.73)	22.80 (14.40- 29.40)	23.83 (12.37)	25.70 (20.90- 29.90)	24.00 (15.34)	22.10 (14.40- 29.40)

Similarly, the classification of folate, vitamin A and zinc during pregnancy as "low risk", "moderate risk" and "high risk"[69] for deficiency revealed no significant differences (*Table 3.20 –3.22*).

Table 3.20: The distribution of individuals among the different population groups according to their risk value grouping of zinc

Zinc's classification:	Subjects (n=8)	Controls (n=7)	Total (n=25)
Low Risk	8	7	25
Moderate Risk	0	0	0
High Risk	0	0	0

Table 3.21: The distribution of individuals among the different population groups according to their risk value grouping of folate

Folate's classification:	Subjects (n=11)	Controls (n=9)	Total (n=30)
Low Risk	3	5	11
Moderate Risk	5	3	15
High Risk	2	1	4

Table 3.22: The distribution of individuals among the different population groups according to their risk value grouping of vitamin A

Vitamin A 's classification:	Subjects (n=11)	Controls (n=9)	Total (n=30)
Low Risk	11	4	25
Moderate Risk	0	5	6
High Risk	0	0	0

Ferritin in conjunction with haemoglobin is considered to be a readily available and good measure of iron status even in pregnancy. The cut-off points of ferritin < 20µg/l and haemoglobin > 105g/l, indicates iron deficiency.[63] There was no statistical significant difference between the subjects' and controls' incidence of anaemia and/or iron deficiency; despite the fact that more subjects had iron deficiency and anaemia when compared with controls (*Tables 3.23 & 3.24*). If anaemia coincides with a ferritin value < 12µg/l, then the individual has an iron deficiency anaemia.[26] Only one of the three subjects who had anaemia had the appropriate ferritin value to have iron deficiency anaemia.

Table 3.23: The distribution of iron deficiency among the different population groups

Iron deficiency?	Subjects (n=10)	Controls (n=9)	Total (n=30)
"Yes"	4	2	13
"No"	6	7	17

Table 3.24: The distribution of individuals with anaemia among the different population groups

Anaemia?	Subjects (n=15)	Controls (n=14)	Total (n=46)
"Yes"	3	0	3
"No"	12	14	43

3.6 Clinical signs

Individuals were examined for clinical signs of nutrient deficiencies and/or toxicities that were relevant to this study (*Table 3.25*). Because of the low incidences of clinical signs, significance could not be tested.

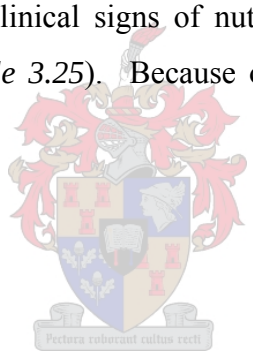


Table 3.25: The incidence of different clinical signs among the population groups

DEFICIENCIES/ TOXICITY	CLINICAL SIGNS		Total (n=47)	Subject (n=15)	Control (n=15)
MUSCLE WASTAGE (ENERGY AND PROTEIN DEFICIENCY)	Thumb muscle		0	0	0
	Fore arm		4	2	0
	Upper arm		3	2	0
	Face		0	0	0
	Chest		0	0	0
	Upper leg		2	2	0
	Lower leg		4	2	0
VITAMIN A TOXICITY	Palms: Hypercarotenodermia		0	0	0
VITAMIN A DEFICIENCY	Fore arm and upper leg: Follicular Hyperkeratosis		0	0	0
	Eyes	Night blindness	2	1	0
		Xerosis	0	0	0
		Bitot's Spots	0	0	0
		Xeropthalmia	0	0	0
THIAMIN (VITAMIN B1) DEFICIENCY	Eyes: Nystagmus		0	0	0
	Lower leg: calf muscle tenderness		0	0	0
	Feet: "Foot drop"		0	0	0
PYRIDOXINE (VITAMIN B6) DEFICIENCY	Lower leg: calf muscle tenderness		0	0	0
	Eyes: redness & fissuring of eyelid corners		0	0	0
VITAMIN C DEFICIENCY	Scurvy	Follicular hyper-keratosis	0	0	0
		Dry itchy skin	3	2	0
		Swollen and inflamed gums	0	0	0
		Loss of hair	1	1	0
	Skin: bleeding		0	0	0
FOLATE DEFICIENCY	Mouth: Glossitis		0	0	0
ZINC DEFICIENCY	Head: hair loss		1	1	0
	Skin: impaired wound healing		0	0	0
IRON DEFICIENCY	Iron deficiency anaemia: spoon-shaped nails		1	0	0
	Anaemia (e.g. iron deficiency): pale conjunctiva		12	4	3

Other clinical signs that were observed were eczema (n=3), opaque nails (n=4) and a possible case of unconfirmed pellagrous dermatitis.

3.7 Newborn data

Upon visiting the FARR offices with their newborns (at approximately one month of age), forty-five mothers had their babies examined by a paediatrician who had experience in identifying alcohol-related signs in children.

3.7.1 Newborn statistics

No significant differences were seen in the distribution of gender, birth weights or percentile distribution of weight and head circumference of the babies (*Tables 3.26 – 3.*28).

Table 3.26: The sex distribution of the newborns in the population groups

SEX	Subjects (n=15)	Controls (n=15)	Total (n=45)
Female	6	10	24
Male	9	5	21

Table 3.27: The distribution of birth weights between the different population groups

Weight classifications	Subjects (n=15)	Controls (n=15)	Total (n=45)
Normal birth weight (> 2500g)	10	12	34
Low birth weight (< 2500g)	5	3	11

Table 3.28: The distribution of newborns according to their weight and head circumference in the different population groups

Anthropometric measurement and classification		Subjects (n=14)	Controls (n=15)	Total (n=45)
Weight for age	< 5	1	0	3
	5-50	7	11	23
	50-95	6	4	18
	> 95	0	0	1
HC for age	< 5	0	0	2
	5-50	7	6	18
	50-95	7	9	24
	>95	0	0	1

The estimated date of delivery (EDD), determined by the ultrasound examination each mother underwent, revealed no differences in premature deliveries between the study groups (*Table 3.29*).

Table 3.29: The distribution of newborns being born term or prematurely in the different population groups

Gestational age	Subjects (n=15)	Controls (n=15)	Total (n=45)
Term	13	14	39
Premature	2	1	6

The paediatrician, classified all newborns into either confirmed Fetal Alcohol Syndrome (FAS), normal or "deferred" (*Figure3.10*). Defer, in this context, means to delay the diagnosis until a later date. (As mentioned in the methodology, it is very difficult to make an accurate diagnosis of FAS in infancy, and that a re-examination from three years of age would make for a better judgment.) There was no statistical significant difference in the incidence of FAS or "deferred" newborns between the subjects and controls.

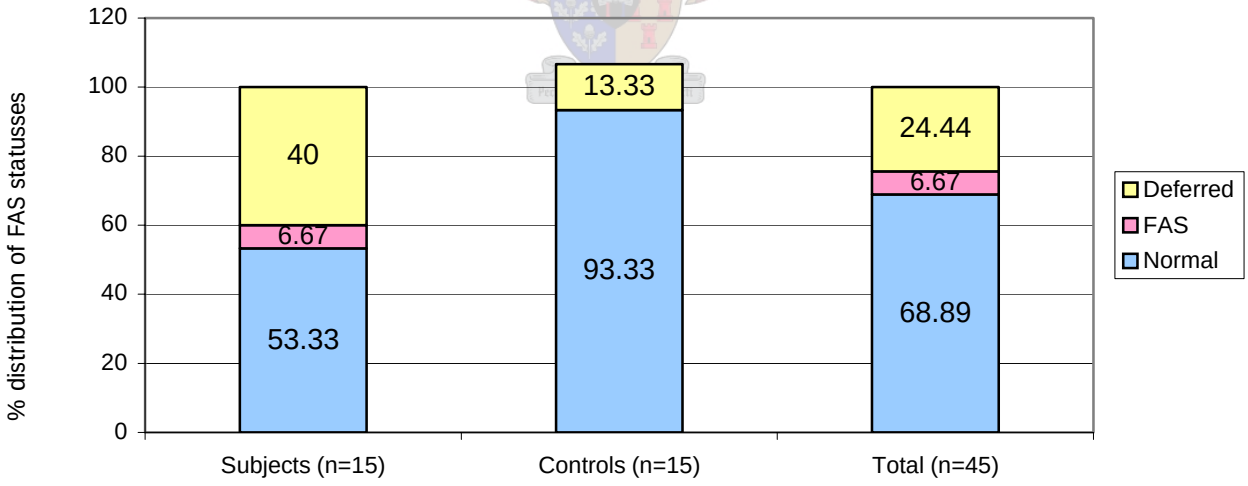


Figure 3.10: The percentage of the alcohol-affected newborns compared with normal newborns of the different population groups

In the birth summary, any complications that were experienced during the pregnancy or delivery had been noted, and no statistically significant difference was found (*Tables 3.30 & 3.31*).

Table 3.30: The presence of pregnancy or delivery complications among the individuals and their newborns of the different population groups

Pregnancy or delivery complications *	Subjects (n=15)	Controls (n=15)	Total (n=45)
Yes	6	4	12
No	9	11	33

*The complications experienced were: emergency caesarian sections (n=6), fetal distress (n=1), pregnancy induced hypertension (n=6), intra uterine growth restriction (IUGR) (n=1), cord around neck (n=1), gestational diabetes mellitus (n=1).

Table 3.31: The presence of physical defects among the newborns of the different population groups

Physical defects *	Subjects (n=15)	Controls (n=15)	Total (n=45)
Yes	4	5	13
No	11	10	32

* The physical defects found were: heart murmurs (n=3), hirsutism (n=3), strabismus (n=1), oral thrush (n=1), seborrhoeus dermatitis (n=1), bulbous impetigo (n=1), pulmonary artery stenosis (n=1), torticollis (n=1).

In one case, an ultrasound investigation showed a case of possible spina bifida. A blood investigation was done which confirmed this, and subsequently the mother decided to terminate the pregnancy.

3.8 Analysis of selected data of the pregnant women

3.8.1 Dietary data

The mothers were grouped together according to BMI's nutritional status indices (< 18.5 being undernourished, 18.5 – 25 normally nourished, and > 25 overnourished); and their dietary intake (according to the averages of the three 24 hour recalls and the two FFQ's) was compared (*Table 3.32*). There was no statistical significance when the mean values of different nutrients were compared among the different BMI classification groups. It is interesting to note that the under- and overnourished groups' energy and protein intake were higher than the normally nourished.

Table 3.32: Mean nutrient intake of the different BMI classification groups according to the average of three 24 hour recalls and two FFQ's

NUTRIENTS Mean values in % of the RDA	BMI < 18.5 (n=3)		BMI 18.5 – 25 (n=16)		BMI > 25 (n=6)	
	24 hr recalls	Food frequency	24 hr recalls	Food frequency	24 hr recalls	Food frequency
Energy	103.63	145.93	88.04	133.60	105.79	188.83
Protein	137.96	164.71	130.10	158.08	161.21	218.80
Vitamin A	72.12	266.93	82.46	192.26	106.27	207.11
Vitamin D	70.65	74.84	32.49	62.18	45.09	95.48
Vitamin E	120.74	159.10	99.59	154.04	96.83	207.89
Vitamin K	74.90	94.84	71.45	128.46	55.16	128.35
Vitamin C	209.06	286.72	167.85	329.35	126.13	330.82
Thiamin	79.89	105.68	64.21	103.13	78.32	141.07
Riboflavin	87.85	155.88	84.50	149.50	104.99	206.23
Niacin	132.56	136.58	118.32	136.61	134.46	185.41
Vitamin B6	82.56	104.49	67.45	101.54	95.81	142.28
Folate	69.23	108.51	54.62	99.53	59.36	117.04
Vitamin B12	217.60	362.02	166.24	313.25	233.54	416.04
Calcium	38.57	77.75	51.74	87.66	60.55	125.51
Phosphorous	88.89	131.18	89.44	132.66	109.97	188.37
Magnesium	85.66	125.89	78.70	125.98	94.91	171.97
Iron	39.63	53.54	33.52	53.03	38.43	68.07
Zinc	74.66	95.74	68.48	92.49	90.10	124.53
Iodine	30.57	40.23	24.40	36.57	35.97	60.78
Selenium	105.02	137.84	78.84	108.11	100.88	148.22

The different weight gain interpretation groups' average dietary intake of the three 24 hour recalls and the 2 FFQ's were compared. There was no statistical significance when the mean values of different nutrients were compared among the different weight gain groupings, but visually there was a trend when the average energy intake according to 24 hour recall, was compared with the actual weight gained (*Figure 3.11*). The data was cleaned by removing 3 values, thereby making the trend significant ($p<0.0100$). When the subjects and controls were

split (*Figure 3.12*), only the subjects' weight gain and energy intake correlated significantly. None of the macronutrients correlated significantly with weight gain.

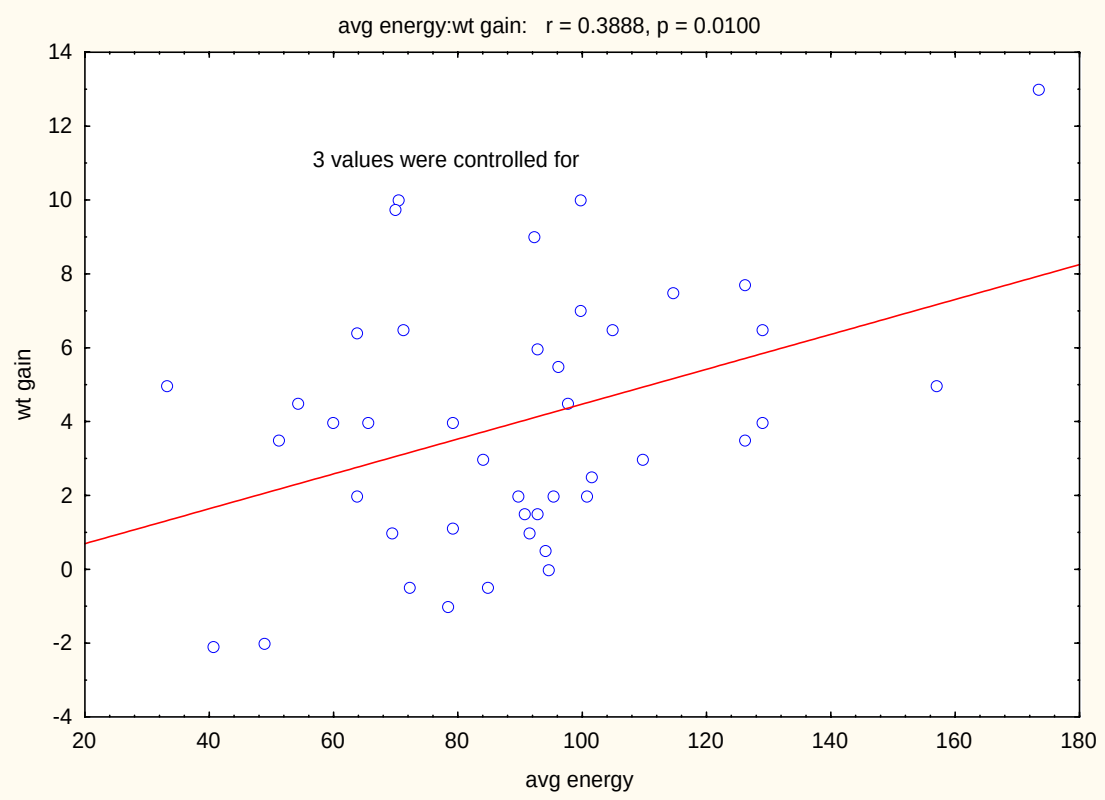


Figure 3.11: Average energy intake as % of the RDA according to 24 hour recall compared with actual weight gain during pregnancy in kilograms

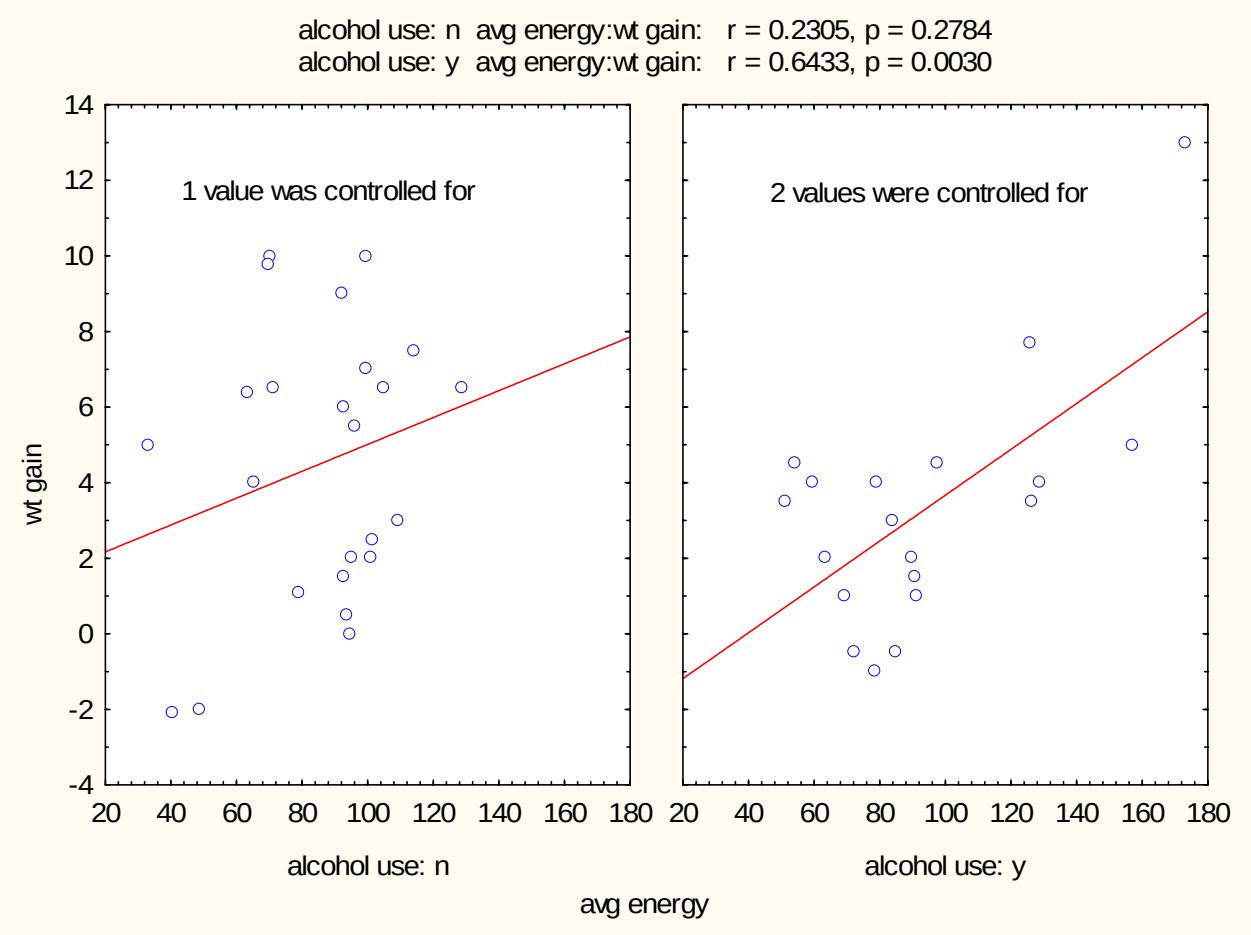


Figure 3.12: Average energy intake as % of the RDA according to 24 hour recall compared with actual weight gain during pregnancy in kilograms of the subjects and controls separately

3.8.2 Biochemical data

The summation of the subject and control population's zinc, folate and vitamin A's biochemical values and dietary intake were compared and grouped accordingly (*Figures 3.13 & 3.14*). Please note the varying n values identifying what number of women had the relevant biochemical tests done and completed the relevant dietary questionnaires. No statistically significant correlation was found between biochemical values and nutrient intake categories. Appropriate or inappropriate intake of the nutrients did not always coincide with the relevant biochemical classification.

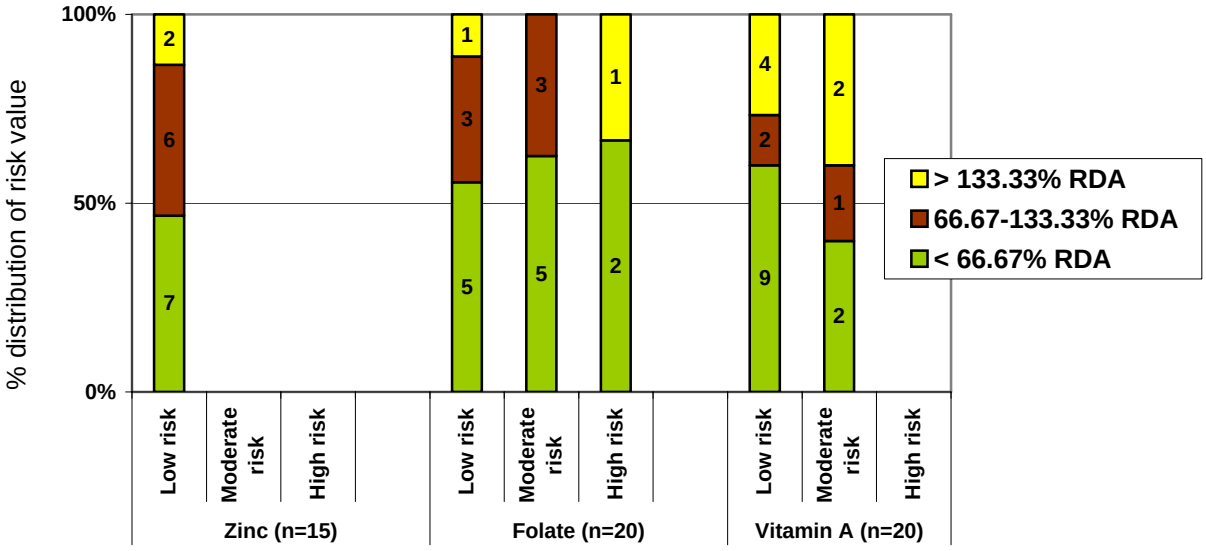


Figure 3.13: Women categorised according to their biochemical and 24 hour recall nutrient values

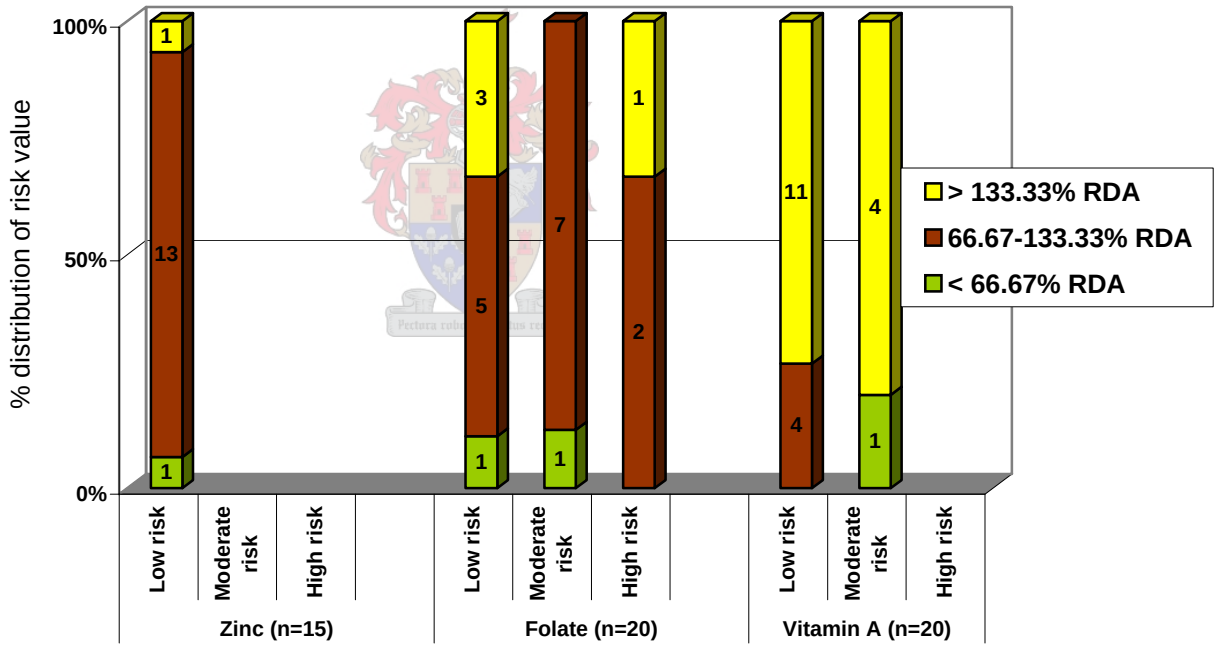


Figure 3.14: Women categorised according to their biochemical and FFQ nutrient values

The presence or absence of iron deficiency, anaemia and IDA was compared to iron intake in the low, adequate and high groupings of the RDA (*Tables 3.33-3.35*). There was no statistical significance in the distribution of individuals who had iron deficiency and iron deficiency anaemia despite the majority having reported an iron intake of less than 66.67% of the RDA.

Table 3.33: Comparison of the incidence of iron deficiency to the iron intake of individuals

Iron deficiency? (n=29)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Yes"	6	5	0	1	-	0
"No"	20	16	2	6	-	1

Table 3.34: Comparison of the incidence of anaemia to the iron intake of individuals

Anaemia? (n=20)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Yes"	2	2	1	1	-	0
"No"	16	14	1	2	-	1

Table 3.35: Comparison of the incidence of iron deficiency anaemia to the iron intake of individuals

Iron deficiency anaemia? (n=20)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Yes"	1	1	0	0	-	0
"No"	17	15	2	3	-	1

According to literature, there is a possible relationship between anaemia, iron deficiency anaemia and undernutrition.[4] The presence of anaemia and iron deficiency anaemia was compared to the distribution of nutritional status according to BMI and MUAC, and the rates of weight gain. No statistically significant relationships was found:

Table 3.36: The incidence of anaemia of individuals compared with their BMI classification

BMI classification	Anaemia?	
	"Yes"	"No"
< 18.5 (undernourished)	2	2
18.5 – 25 (normally nourished)	0	11
> 25 (overnourished)	1*	4

* Individual that has IDA.

3.9 Analysis of selected data of the newborns

3.9.1 Newborn data

Of the total population (n=45), a significant difference in birth weight status and weight for age and HC for age was found (*Tables 3.37 - 3.41*). This confirms that with a normal birth weight the chances increase of having an adequate and above-adequate weight and HC later in life. No statistical significant relationship was found between birth weight and FAS status, even though the literature suggests that FAS children usually experienced IUGR (*Table 3.38*).[42, 49, 51, 52] HC and FAS status however did have a significant relationship (*Table 3.40*). Although it was not a statistically significant relationship, more LBW newborns were born prematurely than normal birth weight newborns (*Figure 3.15*).

Table 3.37: Comparison of the birth weight and weight for age data taken at the age of one month

Birth weight classification	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
LBW	3	7	1*	0
Normal	1	15	17*	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0127$.

Table 3.38: Comparison of newborns' FAS status with birth weight

FAS status	Birth weight	
	Normal birth weight	Low birth weight
"deferred"	6	5
FAS	2	1
Normal	26	5

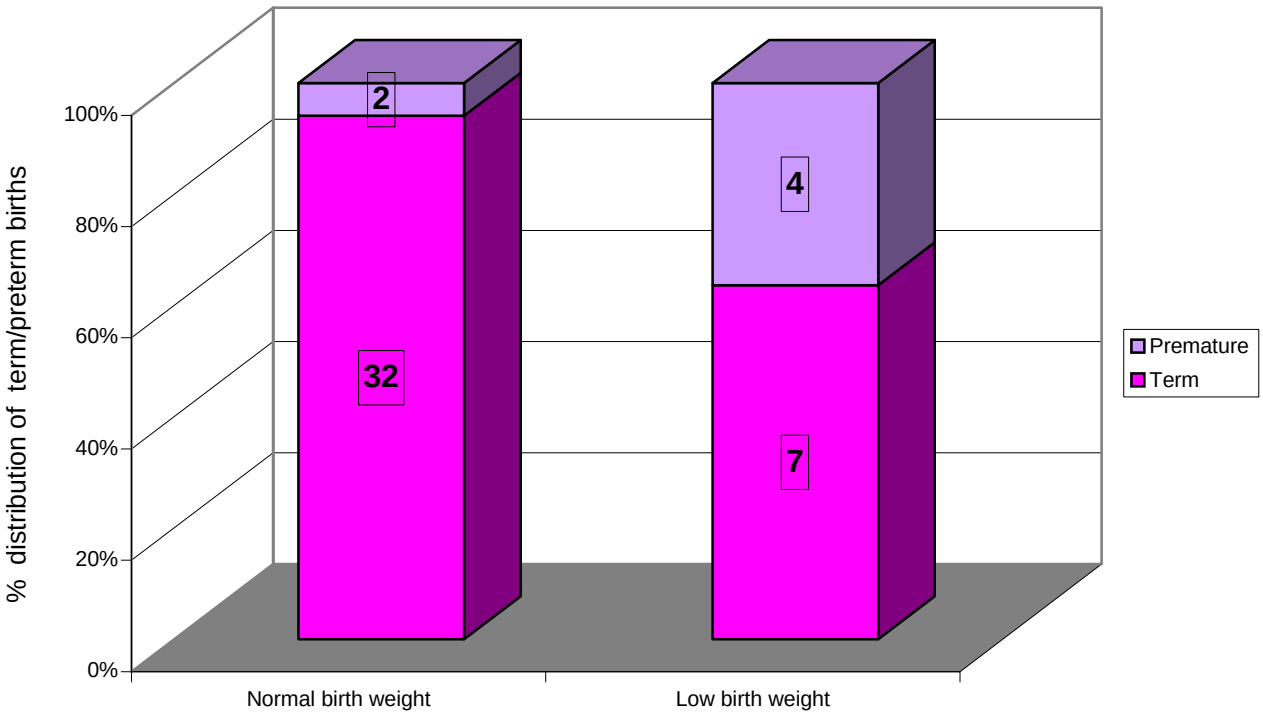


Figure 3.15: The comparison of the newborns' birth weight with their gestional age at birth

Table 3.39: Comparison of head circumference for age classification to the birth weight classification of the newborns

Birth weight classification	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
LBW	2	7	2*	0
Normal	0	11	22*	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0041$.

Table 3.40: Comparison of head circumference for age classification to the "FAS status" of the newborns

FAS status	Head circumference for age (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
"deferred"	0	7	4*	0
FAS	1	2	0*	0
Normal	1	9	20*	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0471$.

Table 3.41: Comparison of head circumference for age classification to the gestational age at birth of the newborns

Gestational age at birth (in weeks)	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
31	2	0	0	0
32	0	1	0	0
34	0	0	2*	0
36	0	1	0	0
Term	0	16	22*	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0030$.

3.9.2 Newborn data compared to the mother's questionnaire data

The interaction of smoking by the mother and the effect of it on the newborn was investigated. Smoking (and how much) versus non-smoking was compared with the incidence of LBW and the newborn's FAS status. No significant difference was found; however a higher proportion of LBW and FAS-affected offspring's mothers smoked.

Table 3.42: The incidence of low birth weight and normal birth weight of the newborn compared with the smoking status of the newborn's mother

Birth weight:	Smoking status:		
	Non-smoker	1-4 cigarettes/day	5-10 cigarettes/day
Low birth weight	1	6	4
Normal birth weight	13	8	13

Table 3.43: The smoking status of the mother compared with the FAS status of her newborn

FAS status:	Smoking status:		
	Non-smoker	1-4 cigarettes/day	5-10 cigarettes/day
Normal	13	7	11
"deferred"	1	6	4
FAS	0	1	3

A statistically significant correlation (Spearman) of $p < 0.0.03$ was found between the mother's income and the birth weight of her child (*Figure 3.16*).

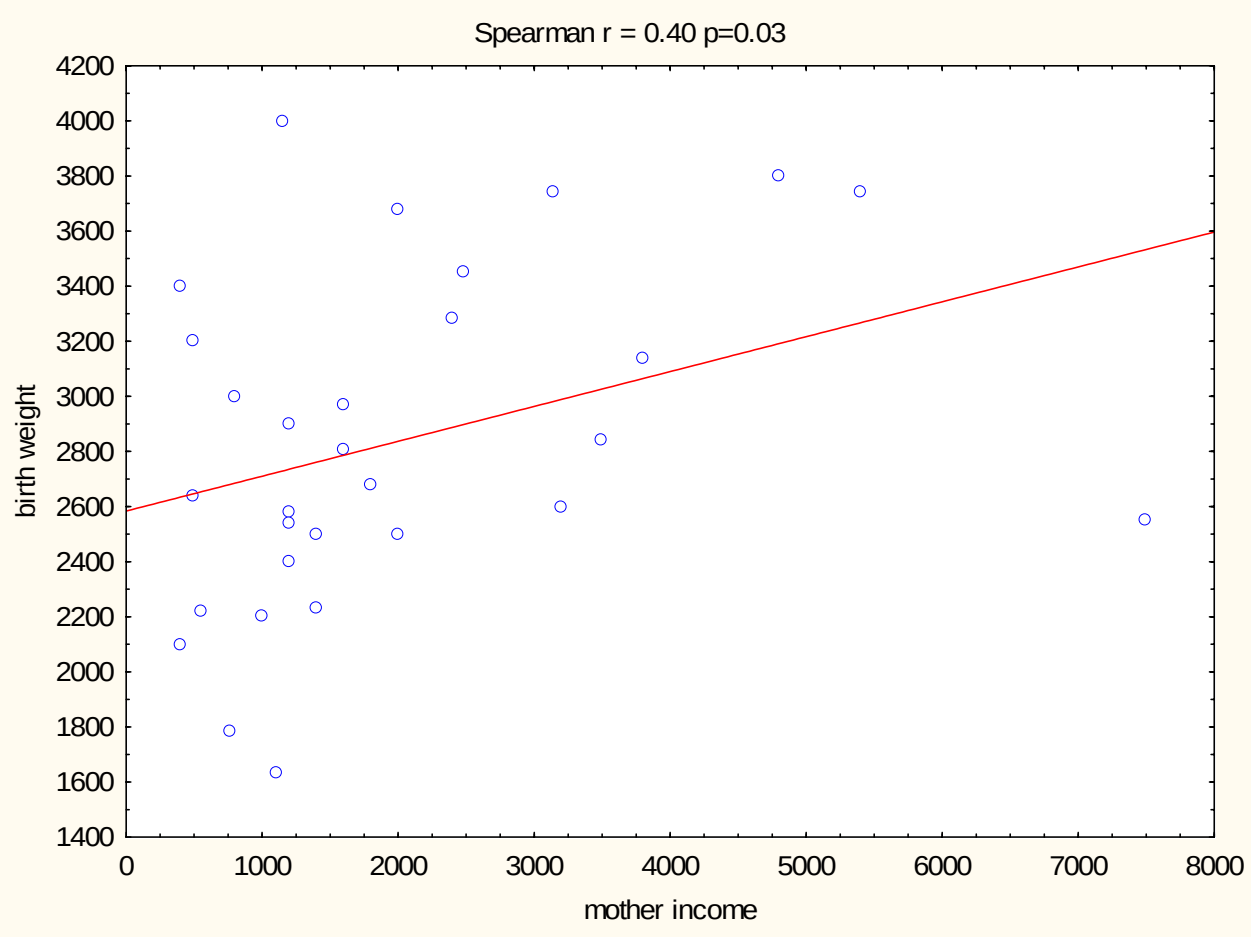


Figure 3.16: Correlation of the mother's own income to the birth weight of the newborn

This result was not confirmed when the investigator compared the food security status of the participant to the newborn's birth weight. However, there was a trend for significance when food security status of the participants was correlated to the newborns' birth weights (*Figure 3.17*).

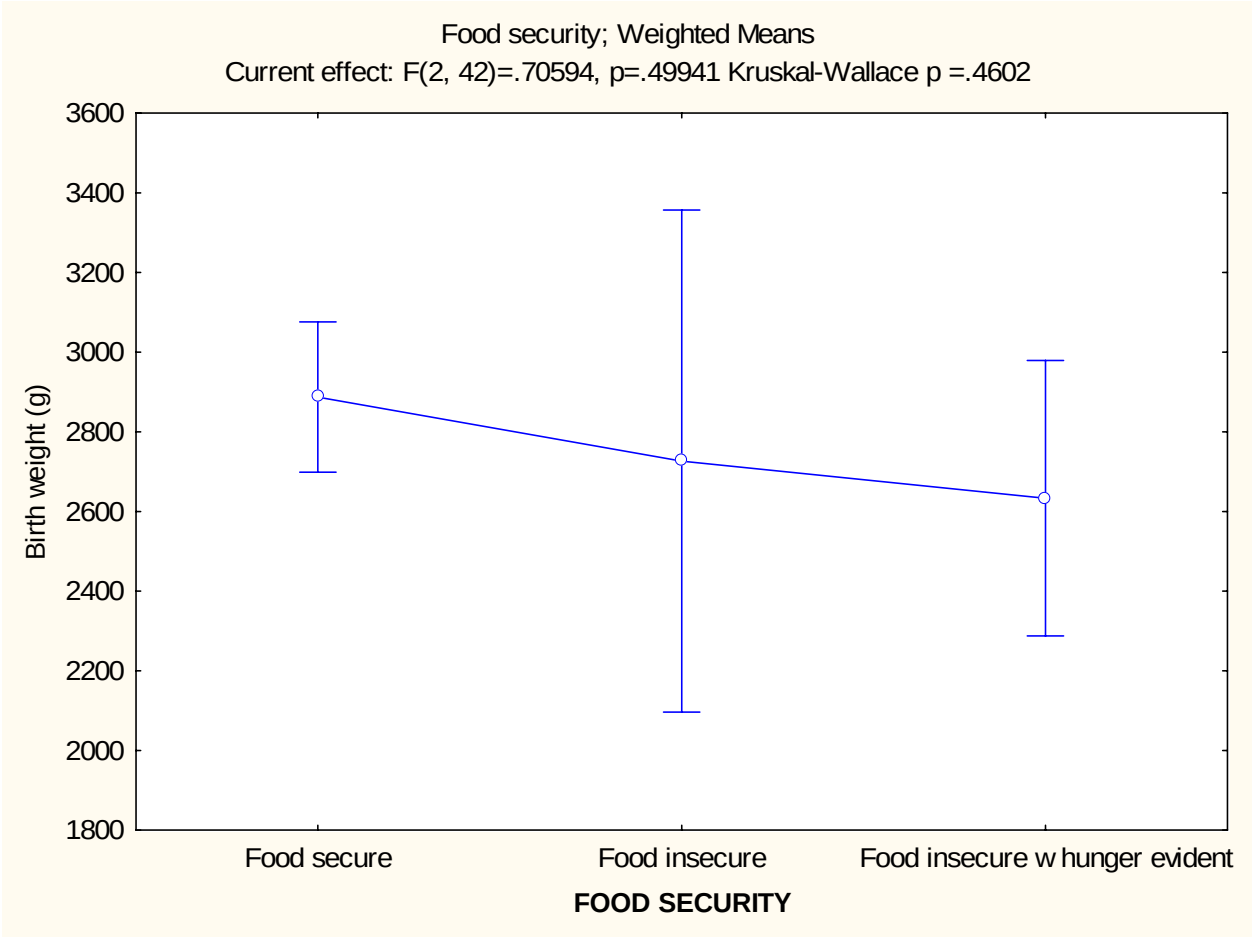


Figure 3.17: Food security of the participant during pregnancy compared with her newborn's birth weight

Literature cites older women and women with higher parities being more likely to have FAS-affected offspring[56]; a trend which has also been documented by the present study (*Figure 3.18*). The correlation is not significant ($p < 0.4602$), but would most probably have been if the population was larger.

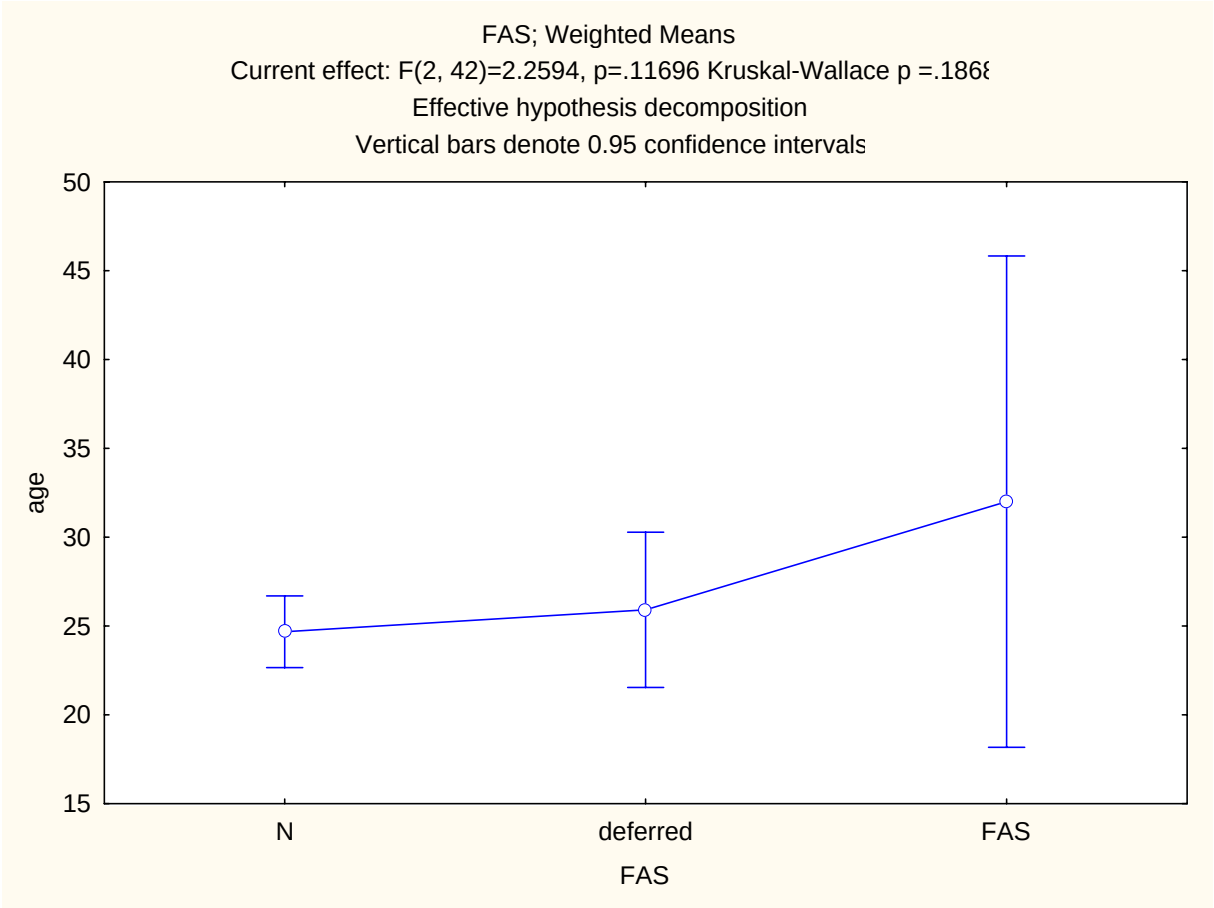


Figure 3.18: Correlation between the participant's age and the newborn's FAS status

3.9.3 Newborn data compared to the mother's dietary data

When comparing the newborns' weight for age, HC for age and birth weight with the mothers' dietary intake, no statistical significant difference was found using the Fischer's Exact test.

When comparing the newborns' gestational age at birth with the mothers' dietary intake, a statistical significant difference was found with energy intake ($p < 0.0083$) on the basis of the 24 hour recalls (*Table 3.44*).

Table 3.44: Comparison of the mother's energy intake to the newborn's gestational age at birth

Gestational age at birth in weeks	Energy (< 66.67% RDA)	Energy (66.67 – 133.33% RDA)	Energy (> 133.33% RDA)
31	1	1	0
32	0	1	0
34	0	0	2
36	0	1	0
Term (> 38 weeks)	7*	28*	4*

* Statistical significance according to Fischer's Exact test is $p<0.0083$.

When comparing the newborns' alcohol related signs status (FAS, "deferred", normal) to the mothers' dietary intake, no statistical significance was found. However, the FAS-affected offspring's mothers did have a general lower nutrient intake when compared with the normal and "deferred":

Table 3.45: Comparison of the nutrient intake of the mothers of newborns with different FAS statuses

NUTRIENTS' mean values in % of the RDA	Normal offspring (n=31)		"deferred" offspring (n=11)		FAS-affected offspring (n=3)	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Energy	95.80	142.32	94.83	168.59	63.90	130.31
Protein	132.99	162.39	131.17	197.46	97.97	167.47
Vitamin A	70.67	201.22	122.87	197.65	44.79	320.50
Vitamin D	34.32	63.04	58.21	82.06	54.09	113.65
Vitamin E	103.59	156.88	94.15	190.47	56.10	165.17
Vitamin K	67.01	121.90	71.59	116.79	31.47	151.11
Vitamin C	191.22	344.04	109.88	298.77	38.89	175.57
Thiamin	70.61	109.09	61.63	119.82	37.88	98.91
Riboflavin	90.47	156.79	71.89	176.56	47.42	159.77
Niacin	122.77	141.66	111.38	160.76	80.03	140.55
Vitamin B6	75.21	107.63	70.84	120.46	42.74	97.25
Folate	57.48	102.97	53.89	107.39	33.82	110.54
Vitamin B12	174.68	305.74	199.26	407.09	116.42	487.66
Calcium	51.19	90.11	43.34	105.22	31.65	84.29
Phosphorous	90.96	135.23	87.81	168.92	64.46	135.43
Magnesium	80.61	129.61	78.81	152.64	59.05	127.19
Iron	35.41	55.55	31.41	57.62	21.53	35.75
Zinc	71.31	94.59	67.64	115.02	53.16	92.11
Iodine	25.96	37.48	29.36	52.21	25.89	51.86
Selenium	81.70	115.03	94.49	140.44	72.64	115.36

The comparison (Kruskal-Wallis test) of the mean dietary intake of mothers whose newborns were normal weight compared to those whose newborns were LBW (*Table 3.46*) was not significant except in the case of vitamin D. LBW's mean values of most nutrients (15 out of 20) according to 24 hour recall questionnaires were higher than normal birth weight; but the reverse picture was the case on the basis of the FFQ questionnaires (4 out of 20).

Table 3.46: Comparison of the mean nutrient intake of mothers with LBW newborns to those of normal birth weights

NUTRIENTS' mean values expressed as % of the RDA	LOW BIRTH WEIGHT (<2500g) (n=11)		NORMAL BIRTH WEIGHT (>2500g) (n=34)	
	24 hour recall	**FFQ**	**24 hour recall**	**FFQ**
Energy	96.05	135.69	93.51	150.77
Protein	134.73	158.92	138.56	176.49
Vitamin A	115.08	233.81	77.02	202.94
Vitamin D	56.43*	73.27	37.10*	69.43
Vitamin E	133.39	160.29	94.05	166.74
Vitamin K	72.96	156.42	68.79	113.43
Vitamin C	196.97	325.71	159.12	329.39
Thiamin	66.49	99.12	70.44	116.65
Riboflavin	101.03	144.80	86.63	170.36
Niacin	122.46	126.26	124.72	153.78
Vitamin B6	74.50	97.16	77.02	115.92
Folate	64.78	102.39	55.95	105.96
Vitamin B12	209.15	332.08	179.91	346.40
Calcium	61.63	87.14	48.55	97.72
Phosphorous	99.56	133.36	91.72	148.63
Magnesium	89.29	126.79	81.11	139.71
Iron	35.89	54.06	35.44	57.35
Zinc	73.99	92.19	73.82	102.92
Iodine	34.28	44.95	25.85	41.36
Selenium	91.62	111.37	86.24	124.89

* There is a statistical significant difference according to Kruskal-Wallis test ($p<0.0288$).

The mother, who had a termination because her fetus was diagnosed with spina bifida, had a normal to high intake of folate according to the one 24 hour recall (81% RDA) and food frequency questionnaire (144% RDA) that was completed with her.

3.9.4 Newborn data compared to the mother's anthropometric data

The mothers' BMI, MUAC and weight gain was compared with the newborns' birth weight, gestational age at birth, weight for age and HC for age taken at one month, for significance testing. The following results had significant associations for the total population (n=45):

Table 3.47: The newborn's birth weight compared with the mother's MUAC classification.

MUAC classification (in percentiles)	Normal birth weight (> 2500g)	Low birth weight (< 2500g)
< 25	10	5
25 – 75	14	5
> 75	10*	1*

* The statistical significance according to the Fischer's Exact test is $p < 0.0449$.

Table 3.48: The newborn's weight for age classification compared with the mother's weight gain interpretation groups.

Weight gain	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
"Correct"	0*	0*	5*	0*
"Fast"	0	5	3	0
"Negative"	0	3	5	0
"Positive"	4	14	5	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0056$.

Using the Spearman's correlation test a positive and significant association ($p < 0.04$) was found between birth weight and mid upper arm circumference (*Figure 3.19*).

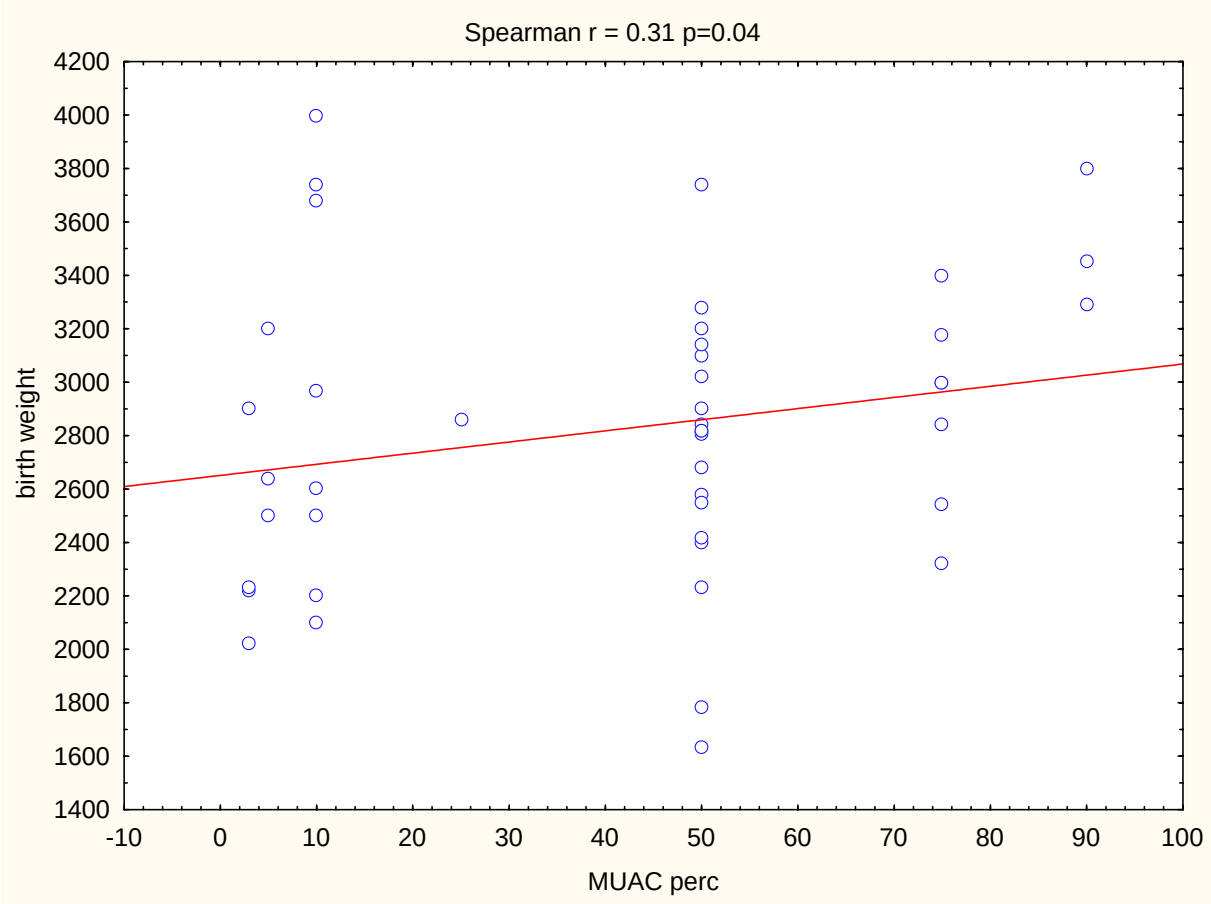


Figure 3.19: Correlation of newborn's birth weight (g) with the mother's mid-upper arm circumference (percentile)

The significant differences and relationships found between nutritional status, pregnancy outcomes and alcohol-related signs of the subjects and the controls will be weighed against the aims and objectives of the study in the next chapter .

4. DISCUSSION

This cohort, controlled study assessed the nutritional status of pregnant women in relation to their alcohol consumption and pregnancy outcome. The nutritional status of the matched subjects and controls did not differ significantly in terms of dietary intake, anthropometric or clinical assessment. The prevalence of FAS in the total population group was a staggering 6.67%; a finding that confirms previously reported data in nearby Wellington, Western Cape.[49] As described in other literature, smoking and older age was also associated with FAS in the study.[49,56,60]

A very important significant correlation showed that energy intake positively influences weight gain ($p < 0.0100$). Another significant difference ($p < 0.0083$) regarding energy intake is that 28 out of the 39 women who had a term birth as opposed to a preterm birth, had an adequate energy intake of 66.67% - 133.33% RDA. The Spearman's correlation and Fischer's Exact tests showed a significant positive association between MUAC, an index of the mother's nutritional status and the birth weight of her infant. Weight gain during pregnancy and the baby's weight for age also had a significant relationship.

To determine the incidence of FAS and newborns with alcohol abuse-related signs was one of the pregnancy outcome parameters in this study. It was quite alarming that only eight of the fifteen subjects' newborns were given a diagnosis of normality. Of the remainder, only one had an outright diagnosis of FAS. (There were three FAS diagnoses in the total population.) Six subjects' newborns were given a "deferred" status. This does not mean necessarily that they have FAS, but in the opinion of an experienced paediatrician there is enough cause for concern. Regarding the control population, it is noticeable that two controls' newborns had a "deferred" status. This could either mean that those control mothers were not completely honest when answering questions about how much they drank during pregnancy, or it could mean that as controls they were light drinkers of alcohol and that that was enough to damage their unborn children. Another possibility is that the facial characteristics, upon which a FAS diagnosis relies partially, were passed on by previous generations where FAS might have been present.

When FAS status was compared with HC for age, FAS newborns had a significantly higher risk of having a smaller HC ($p < 0.0471$). This finding is supportive that intra-uterine alcohol exposure adversely affects brain growth. One of the diagnostic criteria for FAS is in fact a HC for age below the 3rd percentile.[51] However, the criteria of LBW was not confirmed by the results of the present study, since no association was found between FAS status and the newborns' birth weight. Two of the three FAS newborns and 6 of the 11 "deferred" newborns had a normal birth weight.

The trend discussed in the literature that FAS offspring's mothers are generally older was also found in this study. Although there was no significance to the association ($p < 0.186$), the 3 mother's ages were 27, 31 and 38 years, in comparison with the mean age of the total population which was 25.23 years.

All except one of the FAS and "deferred" newborns had mothers who smoked. This could contribute to IUGR, which is usually associated with FAS.

Anthropometrically, the BMI's, MUAC's and weight gain of the mothers with FAS-affected newborns did not differ greatly from mothers with normal or "deferred" offspring. Their average energy intake was lower, albeit no significantly so, than that of the mothers with normal offspring (63.9% compared to 95.8% RDA according to the 24 hour recalls).

Two subjects had a BMI < 18.5 and 3 subjects had a MUAC < 5th percentile. Although this difference between the subject and control population was not significant, it does indicate that undernutrition was more of a problem in the subject population.

Nutritional status during pregnancy is believed to be of key importance in mitigating pregnancy outcome.[1-4] Long-term heavy alcohol consumption has proved to be very teratogenic to the human body and is documented to adversely affect nutritional status.[21,42-44] Thus the impact of heavy drinking on the pregnant woman's nutritional status is assumed to affect the pregnancy outcome significantly.[21] However, the present study's results only seem to verify alcohol's direct teratogenicity on the fetus, as the majority of the subject mothers had a good nutritional status (according to MUAC and nutrient intake) which was neither protective nor responsible for the adverse the pregnancy outcome.

The evidence from literature is far from conclusive about the synergistic effect of alcohol abuse and malnutrition in adverse pregnancy outcomes. Alcohol does affect nutritional status by influencing dietary intake (increasing or suppressing appetite and using the food budget to buy alcohol), have adverse affects on absorption, digestion and metabolism of nutrients, impairs storage of nutrients in the liver and increases the excretion of certain nutrients.[21,42-47] Alcohol metabolism creates free radicals, and so increase anti-oxidant requirements.[48] The potential resultant nutrient deficiencies and the alcohol abuse can interact to cause adverse pregnancy outcomes. Zinc deficiency have been discussed in the literature as possibly interacting with heavy drinking by not being able to counteract the free radicals produced by alcohol metabolism and that it could lead to birth defects.[42,48] Zinc intake was adequate in the subject population (due to their good intake of meat, fish, poultry, milk and milk products and cereal[3]) and all the plasma zinc levels were in the "low risk" range. Therefore, it seems unlikely that this heavy drinking subpopulation's adverse pregnancy outcomes were caused by zinc deficiency.

Vitamin A intake was adequate for all the population groups according to the 24 hour recall's average. This can be attributed to their general fondness of yellow vegetables (carrots are popular snack foods, and some form of pumpkin was consumed more than once weekly) and chicken liver. Chicken liver was generally eaten once a week or every second week. Mean plasma vitamin A was significantly higher ($p < 0.0097$) for the subjects than for the controls. Zachman and Grummer's hypothesis states that heavy drinking inhibits the conversion of vitamin A to retinoic acid, that is essential for brain, head and limb morphology.[58] Also, in animal studies, it was found that heavy alcohol consumption led to the mobilisation of the liver's vitamin A stores in the fetus that caused organ damage and subsequent defects.[21] Thus vitamin A status seems to be a potential culprit within this study as ameliorating alcohol abuse's damage in utero. However, although the subjects' mean vitamin A was significantly higher than that of the controls, all subjects' vitamin A values were in the "low risk" grouping.

Therefore, although in a few cases subjects had been undernourished (according to BMI, MUAC and nutrient intake) and possibly vitamin A status was a mitigating factor in the etiology of alcohol-related defects, the majority of subjects had a good nutritional status; showing the direct toxicity of heavy drinking caused adverse pregnancy outcomes.

The dietary intake of the different population groups was generally very good when compared to the 1989 RDA. It was planned that the average of the two FFQ's would represent the average of the total dietary intake during pregnancy. The 1st FFQ would be representative of the first half of pregnancy, prior to the individual entering the study, and the 2nd FFQ or the average of the three 24 hour recalls would represent the remainder of the pregnancy spent in the study environment. The 2nd FFQ and the average of the three 24 hour recall questionnaires could be compared for validity.[74] Literature states that FFQ mostly overestimates the usual dietary intake,[75] but the difference between the present study's 24 hour recall averages and the 2nd FFQ was quite large in some cases. The investigator had validated the dietary questionnaires beforehand and the resultant data corresponded well. The FFQ is a difficult diet recall questionnaire to complete, especially when the individuals have to think back over a long period of time. Since the cut-off was 22 weeks pregnant or less for this study, this meant that some women had to think back over a period of 5 and a half months. They may have overestimated their intake since the FFQ was quite thorough and asked about a variety of foods and drinks. It was decided not to combine the 24 hour recall and FFQ data, but to leave them separately, so that they can be assessed on their own as the average and above-average dietary intake of the population.

The population mostly ate plenty of fresh fruits and vegetables that helped boost the micronutrient values. Although most of them could not afford a wide variety of foods, the diet would be considered in general terms to have been a balanced one. Upon investigation of the different population groups' dietary questionnaires, it seemed that the subjects ate more "staple foods" such as bread, rice and porridges than the controls. This could explain their higher energy intake.

A nutrient whose 24 hour recall mean value was far below 66.67% of the RDA, which is an extremely important nutrient during pregnancy for both the mother and the developing fetus, is iron.[4,11] Good sources of iron are organ meats, meat, poultry, dried fruits and fortified bread and cereals. Low dietary iron intake is not uncommon, and an otherwise balanced and adequate diet can fall short of adequate dietary iron.[4] This population did consume meat and poultry daily or every second day, but perhaps not enough of the best sources, such as organ meats. The low dietary iron is cause for concern, since so many dietary and physiological factors affect iron absorption and utilisation negatively, and in the end the dietary iron may not be utilised optimally.[27]

As with iron, vitamin D, folate and calcium's intake was less than 66.67% of the RDA according to the 24 hour recalls. Vitamin D plays an important role in the absorption of calcium. The richest sources of vitamin D are fatty fish, such as herring and salmon, chicken liver, egg yolks and milk.[4] The population did not eat salmon and herring, but they did eat a lot of canned pilchards (once in a week or every second week), which is also an excellent source of vitamin D. Although most individuals consumed liver, eggs and milk weekly, the intake was not sufficient for an adequate vitamin D value. However, there is no cause for concern over a possible vitamin D shortage, since vitamin D is manufactured in the skin with the abundant sun exposure in South Africa.

Since folate is such an important nutrient during pregnancy, the low levels of folate according to the 24 hour recalls should be cause for concern. An adequate folate intake should be easily consumed in a varied diet. Good sources are liver, dark green vegetables such as spinach and broccoli, lean beef, potatoes and whole-wheat bread.[4] The population's intake of spinach, broccoli and whole-wheat bread was poor. Because of the micronutrient's importance, the ADA recommends that women should take folate supplements if their diets are lacking.[3] Nine women of the total population (n=46) took either a multivitamin or multi-vitamin and – mineral supplement which had folate in it. Whether the woman who had a termination due to spina bifida took a folate-containing supplement is unknown, since she had not completed the questionnaire, as she was withdrawn from the study before her 2nd interview.

The richest sources of calcium are milk and milk products, dark green leafy vegetables, sardines and pilchards.[4] Although the population had a good intake of pilchards, they had a poor intake of green leafy vegetables such as broccoli and spinach, and an inadequate intake of milk. The investigator noted during data collection that milk and milk products were only bought when the budget allowed it in the poorer households.

The nutrients that had an intake above the 133.33% of the RDA mark were vitamins C and B12. As mentioned earlier, the women in the study did eat enough fresh fruits. Oranges, naartjies, lemons, guavas and tomatoes were very popular and could be bought relatively cheap. This was fortunate as a great number of the population were smokers, and the body then requires extra vitamin C.[12]

The vitamin B12 values were much higher than the upper level adequate level of intake. Since vitamin B12 is abundant in most animal protein sources such as organ meats, meat, milk and eggs,[4] it was expected the population would have high values of vitamin B12, because of the surfeit of protein in their diets. However, vitamins C and B12 are water-soluble vitamins and excesses are excreted via urine.

Iodine also needs mention, as the investigator feels that the dietary analysis of the study does not truly reflect the iodine intake of the population. Salt is iodised in South Africa, and is by far the biggest contributor of iodine in the diet and makes it easy to consume enough of it with regular salt use.[33] Other sources are shellfish, sardines and certain smoked foods.[4] Assessment of salt intake is made difficult by issues such as determining salt content of food that was not prepared by the person herself, that participants in research have to quantify the numbers of "shakes" from the salt container over foods, sizes of "pinches" of salt and that salt shakers do not have uniform amount of holes. Since the chance of an iodine deficiency was highly unlikely within the setting and the unreliability in determining salt intake, the investigator did not do an accurate assessment of salt intake. Thus the values of iodine intake were considered inaccurate, since it only represented the iodine in the foods ingested.

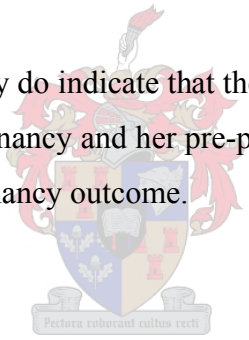
The macronutrient distribution of the subpopulations was close to the ideal of 15:55:30 of protein: carbohydrates: fat. The only mentionable value was the 35% energy contribution of fat in the control population's diet. This could lead to unnecessary weight gain and hyperlipidemia.[4] As mentioned in the results section, alcohol intake was not incorporated into the nutrient analysis. FARR determined whether the women were heavy, light drinking or abstaining during pregnancy at the recruitment interview. The investigator was not confident to express that classification as a daily alcohol intake as it surely varied from day to day, and after recruitment into this study, the women may have been more aware of their alcohol intake and altered its "usual intake". For the study's purposes, alcohol use was determined only to classify women as subjects (heavy drinking) or controls (light drinking or abstinence) and to be aware that extra energy would be ingested from alcohol. Even without the added energy of alcohol, the subjects still ingested more energy (101.58% versus 90.69% of the RDA in the 24 hour recalls) than the controls, which could lead to overnutrition.

The actual weight gained in pregnancy had a positive significant correlation with average energy intake ($r= 0.389$, $p< 0.0100$) for the total population. Also, mothers who had term

births had a significantly adequate energy intake during pregnancy ($p< 0.0083$). These 2 significant associations with energy intake show that dietary intake during pregnancy does influence the pregnancy outcome.

A very important statistical significant association in this study is that the mother's nutritional status according to her MUAC classification (< 25 undernourished, 25 –75 normally nourished and > 75 overnourished) ($p< 0.0449$) and absolute percentile value ($p< 0.04$) had a positive effect on the newborn's birth weight. This supports the literature's finding that a mother's pre-pregnancy nutritional status influences fetal growth.[9] The other case of statistical significance was when the mother's weight gain during pregnancy was compared with the newborn's weight for age. It showed that the women with "correct" weight gain had a baby with a weight for age between the 50th and 95 percentile ($p< 0.0056$). This supports the literature's finding that weight gain during pregnancy directly influences fetal growth and growth thereafter.[12]

Therefore certain data of this study do indicate that the mother's behaviour (dietary intake and alcohol consumption) during pregnancy and her pre-pregnancy nutritional status (MUAC) did influence the pregnancy and pregnancy outcome.



5. CONCLUSION

The nutritional status of the total study population was, based on means, very good. More heavy drinking subjects[a] were undernourished than light drinking or abstaining controls[a] according to BMI and MUAC, and the subjects had a better mean nutrient intake than controls. Although these differences were noticeable between the subjects and controls, they were not statistically significant.

Various questions are left unanswered at the conclusion of this research. As one of the study objectives was "to relate the combined effect of maternal alcohol consumption and nutritional status to pregnancy outcome", it was important to try and determine whether nutritional status could intervene with alcohol abuse's teratogenic effects. It is felt that this was not sufficiently answered by the study's results. The 3 alcohol-drinking mothers who had FAS-affected newborns, had adequate nutritional statuses according to anthropometry, clinical and biochemical assessment. Still, most of the subject participants also had adequate nutritional statuses according to the above parameters, but did not have FAS-affected babies. The women with FAS children did however have a sub-optimal nutrient intake when it was compared to that of mothers with normal and "deferred" offspring. but it did not differ significantly.

The picture painted socio-demographically, shows that the living conditions and health of the subjects were poorer than that of the controls, although not significantly different. The subjects lived in more crowded dwellings, had fewer facilities available, and more were unemployed and food insecure than the controls. The controls were less likely to smoke and more of them consumed supplements during pregnancy. Therefore, an unhealthier lifestyle and living environment seems to be associated with heavy alcohol consumption in this study.

[a] Heavy drinkers are defined as having 2 or more drinks/day, >5 drinks/week taken on a single occasion (binge), or >45 drinks per month are defined as subjects. Light drinkers (up to 2 drinks/day and no binge drinking) or abstainers served as controls.[62]

The null hypothesis adopted states "pregnancy outcome is not affected by maternal nutritional status and alcohol consumption". No significant associations were found between alcohol consumption and birth weight, gestational age at birth, weight for age or FAS and "deferred" incidences. No significant associations were found between maternal nutritional status and weight for age, HC for age, FAS and "deferred" incidences.

Findings in the present study which concur with the literature indicating that maternal alcohol consumption and nutritional status influence pregnancy outcome, are few. Alcohol use during pregnancy can cause "Fetal Alcohol Spectrum Disorder" as seen by the high prevalence of FAS and "deferred" infants. More subjects than controls were undernourished according to BMI and MUAC which may indicate that as a group the heavy alcohol consumers are more nutritionally at risk. Dietary intake during pregnancy was proven important as energy intake significantly influenced the weight gain and gestational age at birth. The mother's own (pre-pregnancy) nutritional status was shown to be just as important in influencing pregnancy outcome, as MUAC correlated positively and significantly with the newborn's birth weight. The latter indicates that a woman's nutritional status during and before pregnancy can influence her pregnancy and the health and well-being of her child.

However, the investigator is of the opinion that there were not enough significant findings to accept or reject the hypothesis, making the results inconclusive.

6. LIMITATIONS & RECOMMENDATIONS

The major draw back of this study was the small size of the study population. With a larger sample, more statistical significant results could have been obtained and the study group would have been more representative of the women attending Hanover Park MOU.

More manpower would have greatly reduced the time span of the study. The time spent (four years plus) on the study does not justify the size of the study population.

In a more ideal situation, the participants for such a study must be followed up more regularly and from an earlier time in the pregnancy. This would increase the accuracy of the retrospective dietary questionnaires, calculating BMI, better weight appraisals and earlier blood samples to prevent haemodilation to influence values.

If more women volunteered a blood sample, the biochemical investigations could have been more meaningful.

A longer follow-up period after birth, would give the study more meaning. It would be easier to correctly identify FAS, ARBD and ARND from three years old onwards. Longer follow-up would also give the investigator more opportunity to monitor the growth of the newborns.

When the effect of nutritional status in relation to alcohol consumption during pregnancy is investigated, it might be more useful if a cohort between 2 different socio-economic groups of women is used. One group, being as well nourished as the majority of women in Hanover Park MOU, and another group from a poverty-stricken area such as a rural town where undernutrition is more prevalent. This way it would perhaps be easier to determine whether nutritional status plays a role in the occurrence of alcohol-related birth defects.

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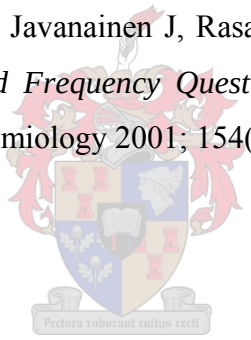

8. APPENDICES

Appendix 1

UNIVERSITEIT VAN STELLENBOSCH
UNIVERSITY OF STELLENBOSCH

7 Junie 2000

Me I Klinger
Dept Menslike Voeding

Geagte me Klinger

NAVORSINGSPROJEK: **"THE NUTRITIONAL STATUS OF PREGNANT WOMEN IN RELATION TO ALCOHOL CONSUMPTION DURING PREGNANCY AND PREGNANCY OUTCOME"**

PROJEKNOMMER : 2000/C027

My brief van 12 Mei 2000 verwys.

Subkomitee C van die Navorsingskomitee het die voorlopige goedkeuring van bogenoemde projek op 29 Mei 2000 bekragtig.

Met vriendelike groete

CJ VAN TONDER
n ADJUNKREGISTRATEUR (TYGERBERGKAMPUS)

CJVT/ev

C:\DOCUMENTS\NAVC\PROJEKTE\20002000-C027-002.DOC

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Appendix 2

MATERNAL ANTENATAL QUESTIONNAIRE

Interviewer: ..... Date of first interview:

Clinic: ..... ___/___/_____

Province: .....

PART 1: INFORMATION OBTAINED FROM INTERVIEWEE:

1. Name: .....
2. D.O.B.: ___/___/_____ 3. Folder number: .....
4. Marital status:

Married	common law	single	single+living together	Other .....

5. Address: .....
   .....
   .....
6. Telephone: (Home) ..... (Work) .....
7. Religious affiliations: .....
8. Ethnic group: .....
9. Maternal education: .....
10. **Father of baby**: Name: .....
    Highest educational level achieved: .....
11. **Family income**: i. Income: .....
    ii. Source of income: .....

12. OBSTETRIC HISTORY G: ..... P: .....

    a) **Previous Obstetric History**
       i) Hospital admissions: .....
       ii) Problems: .....
       .....

    b) **Current Obstetric history**
       i) Estimated gestation: .....
       ii) LMP: .....
       iii) Vaginal bleeding: .....

Date:
BP:
Weight:
Height:
Urine:
LMP
EDD
SF
GA
VDRL
Rh
g%

iv) Hypertension: .....

v) Morning sickness: Yes No

If Yes, Duration: ..... Frequency: .....

vi) Additional information: .....

.....

13. Previous medical history

a) Operations: .....

b) Serious illnesses: .....

14. Family history

a) Any genetic disorders in the paternal/maternal family? Yes No

b) Disorder: .....

c) Relationship of interviewee to affected person: .....

15. Medication usage

a) Are any regular medications used? Yes No

b) Drug(s): .....

c) Dosage: .....

16. Smoking

a) Does the patient smoke? Yes No

b) How many cigarettes per day ..... / day

Or

How many packets of tobacco in a week? No of packets: .....

Size of packet: .....

17. Alcohol History

a) How old were you when you first started drinking alcohol? ..... years

If interviewee has never drunk alcohol, ASK:

i) Is there any particular reason why you have never drunk any alcohol?

.....

.....

If interviewee has stopped drinking, ASK:

ii) When did you stop? .....

iii) Why did you decide to stop drinking? .....

.....

b) What type of alcohol do you usually drink? .....

.....

c) When do you usually drink? .....

.....

d) How much do you usually drink on each occasion? .....

.....

.....

e) Just before and around the time you became pregnant, what did you usually drink in a typical week? (*Start with Friday and ask for details for each day)*

(If interviewee does not drink every week, record drinking pattern for a week when she does drink and then note how many times in a month she does drink.)

	BEER		WINE		SPIRITS		OTHER (specify)	
	No. of Drinks	mls/ drink	No. of Drinks	mls/ drink	No. of Drinks	mls/ drink	No. of Drinks	mls/ drink
FRIDAY								
SATURDAY								
SUNDAY								
MONDAY								
TUESDAY								
WEDNESDAY								
THURSDAY								

Number of weeks in a month when Interviewee drinks: .....

f) Has your drinking pattern changed in any way since ..... (month of conception) YES NO *If NO, skip to Question 17(j)*

g) When did your drinking habits change? .....

h) How did your drinking habits change? .....

i) Why did you change you drinking pattern? .....

.....

j) Have there been times during your pregnancy (even before you knew you were pregnant) when you went to a party or were upset etc and when you may have drank more than is typical for you (as you have just described to me)?

YES NO

If YES, ASK:

i) When did this happen? .....

ii) Please describe to me what and how much you drank? .....

.....

iii) About how many times has this happened? .....

iv) COMMENTS: .....

.....

k) Do you usually eat when you are drinking? YES NO

If YES, Specify: a) .....

.....

l) Over what period of time do you usually drink? *(Calculate this by asking the question in stages, eg. "When do you start drinking on a Friday?", "When have you usually had enough to drink?", etc.)* ..... hours

COMMENTS: .....

.....

.....

18. **Drug History**

Have you ever used any drugs, such as marijuana (dagga), mandrax or anything else? YES NO

If YES, ASK:

a) What have you used? .....

b) When last have you used any _____? .....

c) How often do you usually use _____? .....

COMMENTS: .....

.....

19. **Awareness**

a) Is the patient aware of any factors which may be harmful to the growth or well-being of her baby? YES NO

b) Which factors are harmful? .....

c) Is the patient aware of any factors which may improve the baby's health prior to delivery? YES NO

d) Which factors are beneficial? .....

PART 2: INFORMATION OBTAINED FROM MEDICAL SOURCES

1. **EDD** by dates: ___/___/____ by palpation ___/___/____ by U/S___/___/____
2. Height: ..... 3. Preconception weight: .....
4. Booking visit:

   Gestational age: ..... Weight: .....

   BP: ..... Hb: .....
5. VDRL: ..... 6. Hypertension: .....
7. Vaginal bleeding: ..... 8. Proteinuria: .....
9. U/Sound findings: Date performed ___/___/_____ Gestational age .....

   Comments .....

   .....
10. Medication/supplements dispensed by clinic: .....
11. Additional relevant history.....

.....

12. Assessment .....

.....

TWEAK AND T-ACE

i) Het 'n vriend of familielid al ooit vir jou vertel van goed wat jy gesê of gedoen het terwyl jy gedrink het, wat jy nie kan onthou nie?
Ja Nee

ii) Het goeie vriende of familie die afgelope jaar al gekla of was hulle bekommerd oor jou drinkgewoontes? Ja Nee

iii) Het jy al ooit vroeg in die oggend nodig gehad om te drink om op te hou bewe of vir 'n " hangover"? Ja Nee

iv) Het jy al ooit gevoel jy moet minder drink? Ja Nee

v) Het mense jou al kwaad gemaak deur jou drinkgewoontes te kritiseer?
Ja Nee

vi) Hoeveel drankies het jy nodig om "lekker" te voel? _____
(OF VRA: " Voordat jy die effek van alkohol begin voel")

a) Wanneer jy se aantal drankies, hoeveel bedoel jy? _____

b) Hoeveel is een drankie vir jou? _____ml

vii) Hoeveel drankies kan jy drink? _____
Hoeveel kan jy drink voordat jy uit-"pass" of aan die slaap raak?
_____

Appendix 3

Questionnaire

Section A: Patient Details

A.1 Patient's Name: _____
A.2 Patient Code: _____
A.3 Date of Recruitment: _ _ /_ _ / 2000
A.4 Gestational Age of baby at time of recruitment: _ _ weeks

Section B: Socio-Demographics

B.1 Ethnic Group
Caucasian [ ] Mixed Ancestry [ ] Black [ ] Indian [ ]
B.2 Date of Birth: _ _ /_ _ /_ _ _ _
B.3 Age : _ _
B.4 Living arrangements:
B.4.1 Number of People in house
[1] [2] [3] [4] [5] [6] Other: _____
B.4.2 Adults [1] [2] [3] [4] [5] [6] Other: _____
Children [0] [1] [2] [3] [4] [5] [6] Other: _____
B.4.3 Number of Bedrooms in house
[1] [2] [3] [4] [5] [6] Other: _____
B.4.4 People per Bedroom
[1] [2] [3] [4] [5] [6] Other: _____
B.5 Does your house have Indoor Plumbing? [Yes] [No]
B.6 Occupation: _____
B.7 Income/month: _____
B.8 Financial Situation in home:
B.8.1 Does household share expenses? [Yes] [No]
(If yes, ask B.8.2, and skip B.8.3, B.8.4.)
B.8.2 What is the Total Income of the Household per month?
_____
B.8.3 Does your family pay it's own bills? [Yes] [No]
(If yes, ask B.8.4.)
B.8.4 What is the Total Income of the family per month?
_____

B.8.5 Compare income to Household Subsistence Level (*Appendix 11*):
Specify: _____ [<], [>], or [same] as HSL?

Section C: Health History

C.1 Do/did you have any chronic illnesses?
[T.B.] [D.M.] [Hypertension] [Cancer] Other: _____
C.2 Did you have any severe illnesses in the past? [Yes] [No]
If yes, specify: _____
C.3 Did you have any operations in the past? [Yes] [No]
If yes, specify: _____

C.4 How many children do you have?
[0] [1] [2] [3] [4] [5] [6] Other: _____

C.5 How old are they? (Starting with the Eldest)

	Age	**Delivery Problems**	**Health Problems**	**School Problems**
Child 1				
Child 2				
Child 3				
Child 4				
Other:				

C.6 Were there any problems with the delivery/ies of your child/ren? [Yes] [No]
If yes, indicate the nature of the delivery problem on table above.

C.7 Are they all healthy? [Yes] [No]
If no, indicate the health problems on table above.

C.8 Those children that attend school, are they experiencing any problems at school? [Yes] [No]
If yes, specify the nature of problem on table above.

Section D: Lifestyle

D.1 Do you do exercise? [Yes] [No]
If yes, specify Type _____
Times/week _____
Minutes/session _____

* Consult Appendix 8 for Intensity of exercise _____, and calculate into Food finder results.

D.2 Consult Appendix 9 for Intensity of occupation _____ and calculate into Food finder results.

D.3 Do you take any supplements? [Yes] [No]
If yes, specify type and dosage:

[ ] Vitamin and mineral supplement _____
[ ] Vitamin supplement _____
[ ] Mineral supplement _____
[ ] Other _____

D.4 Do you smoke? [Yes] [No]
If yes, specify _ _ /day

D.5 Drinking Status:
Heavy Drinker [Yes] [No]
Quantity/day: _____
Light Drinker [Yes] [No]

Quantity/day: _____

Section E: Food Security

E.1 Do you have the following in your house?

Running Water [Yes] [No]
Electricity [Yes] [No]
Stove [Yes] [No]
Fridge [Yes] [No]

E.2 The following questions are in the form of statements; indicate whether they are applicable in your situation or not. (Draw results from food security at the end.)

E.2.1 Sometimes people lose weight because they don't have enough to eat. In the last year did you lose weight because there wasn't enough food? [Yes] [No]

E.2.2 In the last year, did you ever eat less than you felt you should because there wasn't enough money to buy food? [Yes] [No]

E.2.3 In the last year, did you (or other adults in the house) ever cut the size of your meals or skip meals because there wasn't enough money to buy food? [Yes] [No]

E.2.4 How often did this happen?

[ ] Almost every month
[ ] Some months but not every month
[ ] Only 1 or 2 months

E.2.5 In the last year, were you ever hungry but didn't eat because you couldn't afford enough food? [Yes] [No]

E.2.6 In the last year, did you (or other adults in the house) ever not eat for a whole day because there wasn't enough money for food? [Yes] [No]

E.2.7 How often did this happen?

[ ] Almost every month
[ ] Some months but not every month
[ ] Only 1 or 2 months

E.2.8 In the last year, did you ever cut the size of your child/ren's meal because there wasn't enough money for food? [Yes] [No]

E.2.9 In the last year, did your children ever skip a meal because there wasn't enough money for food? [Yes] [No]

E.2.10 How often did this happen?

[ ] Almost every month
[ ] Some months but not every month
[ ] Only 1 or 2 months

E.2.11 In the last year, was your child/ren ever hungry but you couldn't afford more food? [Yes] [No]

E.2.12 In the last year, did your child/ren ever not eat for a whole day because there wasn't enough money for food? [Yes] [No]

*Indicate whether these questions/statements are "often true", "sometimes true", or "never true".

E.2.13 I worried whether my food would run out before I got money to buy more. Was that....

[ ] Often true
[ ] Sometimes true
[ ] Never true

E.2.14 The food that I bought just didn't last, and I didn't have money for more. Was that....
[ ] Often true
[ ] Sometimes true
[ ] Never true

E.2.15 I/we couldn't afford to eat balanced meals. Was that....
[ ] Often true
[ ] Sometimes true
[ ] Never true

E.2.16 The child/ren wasn't eating enough because I just couldn't afford enough food. Was that.....
[ ] Often true
[ ] Sometimes true
[ ] Never true

E.2.17 I relied on only a few kinds of low-cost food* to feed the child/ren because I was running out of money for food. Was that.....
[ ] Often true
[ ] Sometimes true
[ ] Never true

* rice, beans, pastas, bread, potatoes

E.2.18 I/we couldn't feed the children a balanced meal because I/we couldn't afford it. Was that...
[ ] Often true
[ ] Sometimes true
[ ] Never true

E.3 Is anyone in the house on the PEM Scheme? [Yes] [No]
If yes, specify whom and what: _____

Section F: Eating Behaviour

F.1 Since your pregnancy, have you changed any of your eating, drinking or smoking habits? [Yes] [No]
If yes, specify:
_____

F.2 Have you experienced any stomach problems since your pregnancy? [Yes] [No]
If yes, specify Type & Frequency:
_____

F.3 Do you skip meals? [Yes] [No]
If yes, specify: [ ] Breakfast
[ ] Lunch
[ ] Supper
Frequency/week: _____

F.4 Do you avoid certain foods? [Yes] [No]
If yes, specify: _____
Why? [ ] Dislike [ ] Allergic [ ] Religious reasons Other _____

F.5 Since your pregnancy, have you had “cravings” for food or non-food substances?
[Yes] [No]
If yes, specify Type & Frequency: _____

Food frequency questionnaire

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
PORRIDGE	Maize-meal Porridge Brand:_____ _____ Enriched? Y/N; Specify: _____	Stiff Pap (Enriched)	3400 4278	1C = 250g 1T = 75g						
		Soft Pap (Enriched)	3399 4277	1C = 250g 1T = 75g						
		Crumbly Pap (Enriched)	3401 4279	1C = 140g 1T = 30g						
	Mabella Porridge	Stiff/soft	3437	½C = 125g						
	Maltabella Porridge	Stiff/soft	3241	½C = 125g						
	Oats Porridge	Brand name: _____	3239	½C = 125g						
	Other Cooked Cereals	Specify:_____ _____								
	Milk on Porridge (Tick off appropriate answer)	None								
		Whole/Fresh 2% Skim Milk Blend	2718 2772 2775 2771	little = 30g med = 60g much = 125g						
		Condensed (Whole) Condensed (Skim)	2714 2744	1t = 10g						
		Evaporated (Whole) Evaporated (Low Fat)	2715 2827	1t = 3g						
		Non-Diary Creamer	2751	1t = 4g						
	Is Sugar added to porridge? (Tick off)	None								
		White Brown	3989 4005	1t = 6g						
		Syrup Honey	3998 3984	1t = 15g						
		Sweetner, Type: _____	P001 6							

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
	Is Fat added to porridge? (Tick off)	None								
		Animal Fat/Butter Hard Margarine Soft Margarine/PM Soft Margarine/Med	3479 3484 3496 3531	1t marg/oil = 5g						
		Sunflower Oil	3507							
		Peanut Butter	3485	1t = 12g						
BREAKFAST CEREALS	Breakfast Cereals	Specify:_____		*****						
	Milk on Cereals	Specify:_____		********						
	Is Sugar added to Cereal?	Specify:_____		*******						
	Is Fat added to Cereal?	Specify:_____		********						
How many times a week do you eat porridge or breakfast cereals at any time of day (not only breakfast): _____ Now the Starches;										
STARCHES	Samp & Beans	Specify Ratio:_____	3402	1T = 50g 1SP = 125g ½C = 125g						
	Rice Brand:_____	White Brown	3247 3315							
	Stamped Wheat		3249	1T = 30g 1SP = 80g ½C = 80g						
	Pastas	Macaroni Spaghetti Plain	3262 3262	1SP = 70g ½C = 90g						
		Spaghetti & Tomato Sauce	3258	1T=45g; 1SP =80g; ½C=125g						
	Do you add any fat to these starches?	Other: Specify____								
		Yes_____No_____ If yes, specify____		******						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
BREADS AND SPREADS	How many times a week do you eat these starchy foods? _____ Next is bread & bread spreads:									
	Bread/ Bread rolls	White Brown Whole wheat	3210 3211 3212	Wh+Br 10mm=30g 20mm=60g 30mm=100g ½loaf=400g Ww 10mm=35g						
	Other Breads Specify:	Raisin Maize Meal Sweetcorn Rye Pumpernickel Other_____	3214 3278 3379 3213 3283	m/s=30g L/s=50g						
	How many times per week do you eat bread?									
	Dumpling	(Depends on area)		******						
	Vetkoek	(Depends on area)		8cm diam= 60g						
	Provita		3235	6g						
	Crackers	Cream Crackers Refined(e.g. Tuc) Wholewheat	3230 3331 3391	8g 4g 8g						
	Pizza	(Specify toppings) _____		*******						
	Hot Dogs	(Specify sausage) _____		*******						
	Hamburgers	(Specify Meat) _____		********						
	Do you use any of the following spreads on your bread? Fat spreads: (Tick off)	Butter Butro Animal Fat Hard Margarine Soft Margarine(PM) Soft Margarine(Med	3479 3523 3494 3484 3496 3531	1t = 5g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
BREADS AND SPREADS	Peanutbutter		3485	1t =12g						
	Sweet Spreads	Jam Syrup Honey	3985 3988 3984	1t = 15g						
	Marmite/Oxo	Marmite Oxo	4030 4029	thin =2g, medium =4g, thick =7g						
	Paste	Fish Paste Meat Paste	3109 2917	thin =5g, medium =7g, thick =10g						
	Cheese (Specify)	Cheddar Gouda	2722 2723	grated(med.) =10g, thick=15g cubes =30g slice =8g cheezi =20g						
		Cottage Lowfat Cream Cheese Other	2760 2725	thin =10g med. =20g thick =30g						
	Cheese Spreads (Specify)		2730	med. =12g thick =25g						
	Atchar		3117	1T =14g 1SP =60g						
	Other Spreads (Specify)									
	Next are protein foods:									
CHICKEN		Boiled with skin Boiled without skin Fried in batter Fried – not coated Roasted/grilled with skin Roasted/grilled without skin	2926 2963 3018 2925 2925 2950	Breast+skin =125g Thigh=80g Drumstick=42g Foot=30g Wing=30g						
	Chicken Stew	With Vegetables With Tom & Onion	3005 2985	1SP =90g ½C =125g						
	Chicken Offal	Giblets	2998	stomach=20g						
	Chicken Liver		2970	liver=30g						
	Chicken Feet		2997	foot=30g						
	Chicken Head		2999							
	Chicken Pie	Comm/homemade	2954	med=150g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
RED MEAT	Beef	Roasted with Fat Roasted, Fat trim Rump, Fried w fat Rump, fat trim Stewed w fat (Cab) Stewed w/o fat (Vegetables) Mince w Tom&On Other Prep. method	2944 2960 2908 2959 3006 2909 2987	120x60x5=35g 120x60x10=70g S/s 130x70x15=125g L/s 165x70x30=270g 1SP =105g ½C= 125g 1T= 40g 1SP= 85g ½C= 100g						
	Mutton	Fried/grilled w fat Fried/grilled w/o fat Stew: plain Stew: curry Stew: greenbean Other Prep. method	2927 2934 2974 2916 3039 3040	Loin chop=60g Rib chop=40g 1SP= 105g ½C= 125g						
	Pork	Fried/grilled w fat Fried/grilled w/o fat Roast w fat Roast w/o fat Other Prep. method	2930 2977 2958 2978	Chop115x80x20=100g Schnitzel 115x80x20=110g Roast 110x65x5=30g 1SP= 105g ½C= 125g						
	Offal	Velderm fried Liver/beef fried Liver/sheep fried Kidney/beef Kidney/sheep Tripe/beef, cooked in milk Heart/beef Heart/sheep Lung/beef	p0023 2920 0955 2923 2956 2951 2968 2969 3019	1SP= 105g ½C= 125g 80g 55g 85g 30g 1SP=105g ½C=125g 60g 60g 60g						
MEAT GENERAL	Sausage	Fried	2931	Thin 200mm=45g Thick 165mm=90g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
MEAT: GENERAL	Bacon	Fat Lean	2906 2915	1 rasher =10g						
	Cold Meats	Polony Ham Viennas Other	2919 2967 2936	slice 5mm=8g comm.slice=16g Med.slice =25g 100mm= 30g 150mm=40g						
	Canned Meats	Bully Beef Other (Specify)	2940	138x85x3 =20g ½C= 100g						
	Meat Pie	Bought(Steak&Kid) Other (Specify)	2957	120g						
	Legumes	Stew(Bean,pot&on)	3178	1T= 60g, 1SP=120g, ½C= 125g						
		Soup: comm. split pea lentil beef&veg beans	3165 3157 3153 3159 3145	½C=125g 1T= 35g 1SP= 80g ½C= 130g						
		Legume Salad		1T= 40g, 1SP= 105g ½C= 135g						
	Soya Products	Specify Brand & Flavour	3196	1SP= 85g ½C= 120g						
FISH	Fried Fish (Fresh/frozen, fried in s/oil	w batter/crumbs w/o batter/crumbs	3094 3084	Small 50x55x30= 60g Medium 100x55x30=120						
	Canned Fish Do you remove bones before eating it? [Y] [N]	Pilchards in Brine Pilchards in Tom.S Sardines in oil Sardines in Tom.S Tuna in oil Tuna in brine Other (Specify)	3055 3102 3104 3087 3093 3054	1 Pilchard=75g S/s = 7g L/s = 25g ¼C= 50g						
	Pickled/ Curried Fish		3076	1 SP= 95g ½C = 140g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
FISH	Fish Cakes	Fried: oil/butter/marg	3098	65x15mm =50g						
	Fish Fingers	Fried: oil/butter/marg	3081	85mm =35g						
EGGS	Eggs	Boiled/Poached	2867	1 egg= 50g						
		Scrambled in oil in butter in marg	2889 2886 2887	1T= 35g 1SP= 80g ½C= 115g						
		Fried in oil in butter in marg in bacon fat	2869 2868 2877 2870	1 egg= 52g						
		Curried	2092	1 egg+ sauce(1T)= 75g						
	How many times a week do you eat meat, beans, chicken, fish or eggs? _____ Now the vegetables:									
VEGETABLES	Cabbage	Boiled, plain Boiled with potato, onion & fat Fried, plain Boiled, then fried with potato, onion Other	3756 3813 3812 3815	1T=30g, 1SP=55g, ½C=80g 1T=35g, 1SP=75g, ½C=80g (Same as boiled, plain) (Same as boiled, w Pot,on & fat)						
	Spinach	Boiled, plain Boiled, fat added Boiled with Onion, Potato & fat Other	3980 3898 3901	1T=40g, 1SP=105g, ½C=90g (Same as boiled, plain) 1T=50g, 1SP=105g, ½C=105g						
	Tomato & Onion Gravy	Homemade w sugar Homemade w/o sugar Canned	3910 3925 4192	1T=35g, 1SP=75g, ½C=140g						
	Pumpkin (Specify type) _____ _____	Boiled, plain Cooked in fat & sugar Other	4164 3893	1T=45g, 1SP=85g, ½C=105g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
VEGETABLES	Carrots	Boiled, sugar & fat W Potato&On (HM) Raw, salad (sugar) Chakalaka	3818 3822 3721	1T=25g, 1SP=50g, ½C=85g 1T=35g, 1SP=70g, ½C=105g 1T= 25g						
	Mealies/ Sweet Corn	On Cob Sweetcorn, cream Whole kernel can Other	3726 3726 3942	1T=30g, 1SP=60g, ½C=95g 1T=55g, 1SP=125g, ½C=135g						
	Beetroot	Cooked no sugar with sugar	3698 3699	1T=40g, 1SP=70g, ½C=80g						
		Salad (grated)	3699	1T=25g, 1SP=65g						
	Potatoes	Boiled/baked with skin w/o skin	4155 3737	S/s=60g, m/s=90g						
		Mashed (W/M)	3876	1T=50g, 1SP=115g, ½C=125g						
		Roasted	3878	1 med=70g						
		French Fries	3740	½C=50g, med=80g						
		Salad	3928	1T=45g, 1SP=105g, ½C=120g						
		Other								
	Sweet Potatoes	Boiled/baked w skin W/o skin Mashed with sugar	3748 3903 3749	1T=50g, 1SP=110g, ½C=145g						
		Other								
	Green Beans	Fresh, frozen Cooked,pot&on/HM Other	4123 3792	1T=25g, 1SP=60g, ½C=80g 1T=40g, 1SP=75g, ½C=120g						
	Peas	Frozen, boiled Boiled w Sugar&butter	4146 3859	1T=30g, 1SP=65g, ½C=85g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
VEGETABLES	Green Peppers	Raw Cooked	3733 3775							
	Egg Plant	Cooked Fried in oil Stew (oil, onions, tomatoes)	3700 3802 3798	1 slice=20g (70mm) + batter=30g 1T=50g, 1SP=100g, ½C=130g						
	Mushrooms	Raw Sauteed in marg Sauteed in oil	3482 3839 3841	1T=30g, 1SP=65g, ½C=80g						
	Onions	Sauteed in oil	3730	1T=50g						
	Salad Vegetables	Raw tomato	3750	Med=120g, slice=15g						
		Lettuce	3723	1 med leaf=30g						
		Cucumber	3718	Med.slice=10g, thick=15g						
		Avocado Other	3656	¼avo=40g						
	Other Veggies: specify									
	If you fry veggies, or add fat, specify usual type used	Butter Butro Animal Fat Hard margarine Soft margarine	3479 3523 3494 3484 3496	1t=5g						
DRESSINGS	Mayonnaise/ Salad Dressing	Mayonnaise-bought –homemade	3488 3506	1t=10g, 1T=40g						
		Cooked salad dress Salad dress –low-oil Salad dress -french	3503 3505 3487	1t=5g, 1T=15g						
		Oil –Olive sunflower canola	3509 3507 4280	1t=5g, 1T=15g						
	How many times a week do you eat vegetables? _____ How many times will it be fresh _____ canned _____ frozen_____ Now comes Fruit.									

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
FRUIT	Apples	Fresh Canned, Pie, not sweet	3532 4216	1T=60g,½C=120g 1med=150g						
	Bananas		3540	1med=75g						
	Oranges/ Naartjies		3560	1med(7cm) =180g						
	Grapes		3550	Med. bunch =230g, ½C=90g						
	Peaches	Fresh Canned in syrup	3565 3567	1 med=150g (60x65)						
	Apricots	Fresh Canned in syrup	3534 3535	1 med=35g						
	Mangoes	Fresh	3556	135mm= 350g						
	Pawpaw		3563	Wedge 165x26x27= 90g						
	Pineapple	Raw Canned in syrup	3581 3648	1 slice (85x10mm) =40g						
	Guavas	Fresh Canned in syrup	3551 3553	Med (6cm) =95g						
	Pears	Fresh Canned in syrup	3582 3583	1 med (80x65mm) =165g						
	Berries Specify									
	Dried Fruit	Raisins Prunes Stewed Prunes(+S) Peaches Stewed Peaches(+S) Apples Dried Fruit sweets Other	4232 4230 3564 3568 3569 3600 3995	1handful=27g 1T=50g, ½C=110g, 1=12g 1med=150g (60x65) 1T=60g, ½C=120g						
	Other Fruit Specify									
	What sugar do you use over fruit?	White sugar Brown sugar Honey Sweetner	3989 4005 3988							

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
	How many times a week do you eat fruit? _____ How many times will it be fresh _____ canned _____ dried_____- Now for the drinks									
DRINKS	Tea	Ceylon Rooibos	4038 4054	Teacup=180ml, mug=250ml						
	Sugar per cup	White Brown sugar?	3989 4005	1t=6g						
	Milk per cup	Whole 2% Skim Whole milk powder Skim milk powder Milk blend Non-dairy creamer Condensed (whole) Condensed (skim) Evaporated (whole) Evaporated (skim)	2718 2772 2775 2831 2719 2771 2751 2714 2744 2715 2827	20ml tea in cup 35ml tea in mug 40ml coffee in cup 75ml coffee in mug 1t=4g (As with n milk) 1t=4g 1t=10g 1t=3g						
	Coffee		4037	Teacup=180ml, mug=250ml						
	Sugar per cup of coffee	White..... Brown sugar?	3989 4005	1t=6g						
	Milk per cup of coffee	Specify as above								
	Milk as such: what type of milk do you drink?	Specify as above		½C=125ml						
	Milk drinks: specify brands	Nestle Drinkingchoc Malted milk (eg. Milo)_____ Flavoured milk: _____ Other	4287 2735 2774	1t=5g 1t=5g Carton=250ml, S/s plastic =350ml						
	Do you add any sugar?									
	Yogurt	Drinking Thick:plain/fat-free plain/WM fruit, low fat	2756 2778 2757 2732	S/s=175ml, Yogisip=350ml, ½C=125g						

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
DRINKS	Squash	Sweeto, 11up Oros/Lecol Artif. sweetner Koolaid Other	3982 3982 3990 3982	Small glass=150ml, med glass=250ml large glass=500ml						
	Fruit juice	Fresh/Liquifruit /Ceres	2866	s/s= 250ml						
		Tropica (w milk)	2791	S/s= 350ml						
	Fruit Syrups		3865	1t= 5g						
	Fizzy Drinks	Sweetened Diet	3981 3990	S/s can=340ml, S/s bottle= 350ml, L/s bottle= 500ml						
	Other Drinks (specify)									
	Next comes the snacks:									
SNACKS	Potato Crisps		3417							
	Peanuts	Roasted Unsalted Roasted Salted	3452 3458							
	Cheese Curls (Nik Naks etc)		3267							
	Popcorn	Plain Sugar coated	3322 3359							
	Peanuts & Raisins	Roasted, salted								
	Chocolates	Specify	3992							
	Candies (Sugus, gums, hard sweets)	Specify	3986							
	Sweets (toffee, fudge, caramels)	Specify	3991							
	How many times a week do you eat snack food? _____									

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
CAKES, BISCUITS, COOKIES	Biscuits/ Cookies	Specify								
	Cakes&Tarts	Specify								
	Pancakes/ crumpets	Specify								
	Rusks	Specify								
	Scones	White, WM	3237	6cm diam= 35g, 8cm diam= 60g						
	Muffins	Plain Bran Other	3408 3407							
	Koeksisters		3231	100x35= 60g						
	Savouries	Sausage rolls Samoosas/meat Biscuits eg. Bacon Kips Other	2939 3355 3331	Rollx135mm= 165g S/s= 42g 4g/biscuit						
	How many times a week do you eat cakes/cookies? _____									
PUDDINGS	Jelly		3983	1T=35g, 1SP=75g, ½C=110g						
	Baked Puddings	Specify		Med. serving= 30g 30x65x65=50g						
	Instant Puddings	Specify		1T=45g, 1SP=95g, ½C=145g						
	Ice-cream	Comm. Regular Comm. Rich Soft Serve Sorbet Ice Lollies Chocolate coated lollies (Magnum)	3483 3519 3518 3491 3982	1scoop=40g, 1SP=65g, ½C=75g 135g 1scoop=40g, 1SP=65g, ½C=75g						
	Custard	Home made(W/M) (S/M)	2716 2717	1T=13g, 1SP=40g						
	Other Puddings	Specify								

	FOOD	DESCRIPTION	CODE	QUANTITY (g/ml)	USUAL AMOUNT EATEN (HHM)	USUAL AMOUNT EATEN (g)	P/D	D/W	P/M	SELDOM
	How many times a week do you eat pudding? _____									
SAUCES, GRAVY, CONDIMENTS	Tomato sauce		3139	1t=6g, 1T=25g						
	Worcester sauce									
	Chutney	Fruit Tomato	3168 3114	1t=14g, 1T=60g						
	Pickles		3866	1=10g						
	Packet soups		3165	½C=125g						
	Others									
	Please mention any other foods eaten by you more than once every 2 weeks that I have not mentioned.									

24-Hour RECALL Date of Recall: _____

Day of week of Recall: [Mo] [Tu] [We] [Th] [Fr] [Sa] [Su]

TIME	FOOD eaten	DESCRIPTION (Type and/or Preparation)	AMOUNT USUALLY EATEN (HHM)	AMOUNT USUALLY EATEN (g)	CODE

Anthropometric Data

Pre-pregnancy Weight/Weight < 12 weeks known: _____
Height: _____
BMI: _____

<16	16-16,9	17-18,49	18,5-24,9	25—29,9	30-39,9	>40

Weight 1: _____(_ _ weeks)
Weight 2: _____(_ _ weeks)
Weight 3: _____(_ _ weeks)

Weight gain Interpretation:
If Wt 1 end of 1st trimester, 1st trimester gain:_____/BMI value gain_____
If not;
Wt 2 – Wt 1 = _____ How much is that per week? _____ (__ trim.)
Interpret: _____
Wt 3 – Wt 2 = _____ How much is that per week? _____ (__ trim.)
Interpret: _____

Mid-upper arm circumference: _____
Percentile class.: _____

<5	5-10	10-25	25-75	75-90	90-95	>95

Biochemical Values:

Hb & HCT indicating ANAEMIA	**Smoking status**									
	0-9				**10-20**				**21-40**			
	Hb (g/l)		**HCT (%)**		**Hb (g/l)**		**HCT (%)**		**Hb (g/l)**		**HCT (%)**	
1st trimester	110		33		113		34		115		34.5	
2nd trimester	105		32		108		33		110		33.5	
3rd trimester	110		33		113		34		115		34.5	

	High Risk		**Moderate Risk**		**Low Risk**	
Zinc (μmol/l) 1st & 2nd trimesters	<7.9		7.9-10.5		>10.6	
Folate (nmol/l)	<6.8		6.8-13.5		13.6	
Vitamin A (μmol/l)	<0.35		0.35-1.04		>1.05	

Ferritin _____μg/l

Anaemia? [Yes] [No] If Y → Ferritin < 12μg/l? [Yes] [No] If Y, [**IDA**]
Iron deficiency if Ferritin < 20μg/l and if Hb > 105g/l, [Yes] [No]

Clinical Evaluation:

DEFICIENCIES/TOXICITY	CLINICAL SIGNS
MUSCLE WASTAGE	Thumb muscle [ ], fore arm [ ], upper arm [ ], face (temporal and orbital muscles) [ ], chest [ ], upper [ ] and lower legs [ ]
VITAMIN A TOXICITY	Palms: (very rare) Hypercarotenodermia [ ]
VITAMIN A	Fore arm and upper leg: Follicular Hyperkeratosis [ ] Eyes: Night blindness [ ], Xerosis [ ], Bitot's Spots[ ], Xeropthalmia [ ]
THIAMIN (VITAMIN B1)	Eyes: Nystagmus [ ] Lower leg: calf muscle tenderness [ ] Feet: "Foot drop" [ ]
PYRIDOXINE(VITAMIN B6)	Lower leg: calf muscle tenderness [ ] Eyes: redness & fissuring of eyelid corners [ ]
VITAMIN C	Scurvy (very rare): Follicular Hyperkeratosis [ ], swollen and inflamed gums [ ], loss of hair [ ], dry itchy skin [ ] Skin: bleeding, i.e. petechiae [ ], purpura [ ], ecchymoses [ ]
FOLIC ACID	Mouth: Glossitis [ ]
ZINC	Head: hair loss [ ] Skin: lesion (impaired wound healing) [ ]
IRON	Iron deficiency Anaemia: Spoon-shaped nails [ ] Anaemia (e.g. Iron deficiency): pale conjunctiva [ ]

New-Born Assessment

Baby's Name: _____
Mother's Name: _____

Termination of pregnancy, prematurity etc.				
Abortion .....weeks	Miscarriage/ Stillborn .....weeks	Premature .....weeks	Term .....weeks	Single/multiple birth .....

Anthropometric Data on.....weeks and .....days old							
Weight ..... W/A				Head Circ. ..... HC/A			
<5	5-50	50-95	>95	<5	5-50	50-95	>95

Birth Weight …….
LBW/VLBW/ELBW

FAS Diagnosis	
Full-blown FAS	
Partial FAS	
ARBD	
ARND	

Physical / Mental Defects
Delivery Problems

Severity Ranges on the Food Security Scale

(Analyses of Q's E.2.1-E.2.18.)

Questions	Food Security Status
None E.2.13 Worried food would run out E.2.14 Food bought didn't last	Food Secure
E.2.17 Child fed few low-cost foods E.2.3 Adult cut size or skipped meals E.2.15 Couldn't feed child balanced meals E.2.2 Adult eat less than felt they should	Food Insecure
E.2.4 Adult cut size or skipped meals, 3+ months E.2.16 Child not eating enough E.2.5 Adult hungry but didn't eat E.2.1 Respondent weight loss E.2.8 Cut size of child's meals	Food Insecure With Hunger Evident
E.2.6 Adult not eat whole day E.2.11 Child hungry E.2.7 Adult not eat whole day, 3+ months E.2.9 Child skipped meal E.2.10 Child skipped meal, 3+ months E.2.12 Child not eat for whole day	Food Insecure With Severe Hunger Evident

*****Modal households with no affirmatives are classified as food secure. To be classified in a given food security category, modal households must respond affirmatively to all questions associated with less severe categories, plus one or more of the questions associated with the category into which the household is classified. Other households (i.e., those not fitting the exact modal pattern) must give the same total number of affirmative responses as the modal households.

Appendix 4

PHYSICAL EXAMINATION

Name: _____ Number: _____

School's Name: _____

Date of Examination: / / Examiner: NK

D.O.B _____ Age: _____ years/months

Sex: _____

Ht _____	Ht % _____	☐ ExamHT
Wt _____	Wt % _____	☐ ExamWT
OFC _____	OFC % _____	☐ ExamOFC
ICD _____	ICD % _____	☐ ExamICD
IPD _____	IPD % _____	☐ ExamIPD
PFL _____	PFL % _____	☐ ExamPFL
Other Measurements _____		☐ OTHMEAS
Mental status/behaviour _____		☐ HYPERACT
Neurological _____		☐ FINEMOTR
Cranium _____		
Face: General _____		☐ HYPOFACE
Ears _____		☐ RREARS
Eyes _____		☐ PALPFISS
Nose _____		☐ STRABISM
Mouth _____		☐ PTOSIS
Neck _____		☐ EPICANTH
Thorax _____		☐ NALSBRDG
Heart _____		☐ ANTENARE
Arms _____		☐ LONGPHIL
Hands: General _____		☐ SMTHPHIL
Creases _____		☐ NRRWVRML
Dermal Patterns _____		☐ PROGNATH
Legs: _____		☐ HEARTMUR
Feet: _____		☐ SUPINATE
Skin: _____		☐ CLINDACT
Hair: _____		☐ CAMPDACT

Other/Comments _____ ☐ PALMCR

_____ ☐ HYPRTRIC

_____

Mother's Name _____

Address: _____

_____

Tel. No. _____

DIAGNOSIS

Abnormalities compatible with FAS (check ALL that apply)

- ☐ 1. Growth deficiency
- ☐ 2. Structural abnormality
- ☐ 3. Cognitive/behavioural abnormalities
- ☐ 4. No abnormalities compatible with FAS observed
- ☐ 5. No significant abnormalities of any kind observed

Does the Child have FAS?

- ☐ 1. No
- ☐ 2. Yes
- ☐ 3. Deferred

Does the Child have another diagnosis?

- ☐ 1. No
- ☐ 2. Yes (please specify)
  - a)_____
  - b)_____
  - c)_____
  - d)_____

Is special follow-up or testing recommended?

- ☐ 1. No
- ☐ 2. Yes (please specify)

_____
_____
_____
_____

Notes: _____
_____
_____
_____
_____
_____

Appendix 5

PILOT STUDY

The objectives of the pilot study were to determine:

- whether the methods decided upon were sufficiently mastered by the researcher,
- whether the pilot study population understood the questionnaires,
- the validity and reliability of the methodology, and to
- adapt the methodology if evaluation showed that it was not valid and/or reliable.

Validity refers to the degree to which a method actually measures what it is supposed to measure.[66] Reliability refers to the consistency of the results obtained.[4]

1. Area

The area selected for the pilot study was Bishop Lavis MOU. It also has a prenatal clinic, but unlike Hanover Park MOU it was only held on Friday mornings. The area was chosen because it is far away from Hanover Park MOU, the area chosen for the main study, thereby avoiding the risk of the study population gaining knowledge of the study and its methods from the pilot study population.

2. Approval

Approval for the pilot study was gained from the senior medical superintendent, community health services organization of PAWC, EW Michaels (*Appendix 6)*.

3. Sample and Time

The researcher visited Bishop Lavis MOU on 3 consecutive Friday mornings, (days when prenatal clinics were held) 27th July 2000, 4th August 2000 and 11th August 2000. Any pregnant woman who volunteered to take part was included in the pilot study.

Three women in total were used in the pilot study. The number was limited since an interview took about the time that the prenatal clinic lasted. No other women were willing to stay behind after clinic to partake in the pilot study.

4. Procedure

Since it was only possible due to time constraints to see the pilot study individuals on one occasion, a 24 hour recall, FFQ, questionnaire, anthropometric assessment and clinical assessment were done only once. The researcher only piloted the questionnaires, since she is not qualified to determine the individuals' alcohol consumption or do biochemical testing.

5. Results and Discussion

The results of the pilot study (*Appendix 7*) were scrutinized by the researcher and 2 of her study leaders. Everyone was happy with a majority of the methodology used and results gained. The exception was that a large difference between the averages of the FFQ and 24 hour recall of most nutrients was found in the population. A difference of 10–15% between FFQ and 24-hour recall values are acceptable according to the researcher's study leaders. An especially large intake of energy, protein, vitamin E, C, thiamin, niacin and vitamin B12 was found according to the FFQ, which was not confirmed by the 24-hour recall. A possible explanation for this could be that only one 24-hour recall was done with each individual, and that that might not be representative of the individual's usual dietary intake, as the FFQ represents.

The study leaders suggested that the researcher find a population where repeat visits is possible, to conduct 3 24-hour recalls, of which that average would be more representative of usual intake, to compare with a FFQ and see if these methods of dietary questionnaires are valid.

Appendix 6

Dear Mrs Klinger,

My deepest apologies for not replying when i promised to do so.

Permission is hereby granted for you to do your pilot study at Bishop Lavis as requested. You will of course arrange with the Sister in Charge regarding the times etc.

Sincerely

E.W.Michaels

SENIOR MEDICAL SUPERINTENDENT

COMMUNITY HEALTH SERVICES ORGANISATION

Appendix 7

Preliminary Results of Pilot Study:

The nutritional status of pregnant women in relation to alcohol consumption and pregnancy outcome.

Ingrid Klinger

The aim of the study is to define the nutritional status of pregnant women in relation to alcohol consumption during pregnancy, and pregnancy outcome.

In the pilot study alcohol consumption was not determined as to not bias the researcher. (In the main study alcohol consumption will be determined by experienced researchers of the Foundation for Alcohol related Research.)

Nutritional status was determined using the following parameters:

- Anthropometry,
- Diet analysis,
- Clinical evaluation, and
- Biochemical analysis (not used in pilot study).

Socio-demographic information (including systemic questioning) was gathered to determine factors that could influence nutritional status, and to have background on previous pregnancies.

The pilot study population consists out of 3 women.

Anthropometry:

1. Pre-pregnancy weight (PPW) is necessary to determine Body Mass Index (BMI), which determines how much weight is to be gained during pregnancy.

INDIVIDUALS	**PPW** (kg)	**HEIGHT** (cm)	**BMI**	**Interpretation**	**Weight gain**	**Rec. weight gain**	**Interpretation**
P1	49	1.56	20.13	Normal	7.15kg	1.6kg	>
P2	-	1.54	-	-	-	-	--
P3	55	1.49	24.77	Normal	13.8kg	5.56kg	>

Pre-pregnancy weight was not known in all cases.

In the cases where it was known, pre-pregnancy BMI was normal, but weight gain far exceeded the recommended amount.

2. Mid-upper arm circumference (MUAC) was determined as well.

INDIVIDUALS	**MUAC**(cm)	**MUAC Percentile value**	**Interpretation**
P1	25.5	10-25	Borderline Undernutrition
P2	27	25-50	Normal
P3	28.5	50-75	Normal

The values coincide with the BMI values, and indicate a "normal" nutritional status for P2 and P3. P1's pre-pregnancy BMI is in the lower range of normal; and therefore coincides with a MUAC value that indicates possible risk for undernutrition.

Diet analysis:

(Refer to graphs below.)

1. The results of the 24-hour recalls and Food Frequency questionnaire (FFQ) were compared individually and also as averages of the population.
2. It seems that in cases P1 and P3, where weight gain during pregnancy was more than recommended; that there average intake of energy (158% and 127% of RDA) and protein (191% and 166% of RDA) was high.
3. Intake of Iron was below 66.67% of the RDA in all 3 cases. This is worrisome since sufficient Iron intake is essential for both the foetus and the mother's health. Intake of Iodine was also below 66.67% of the RDA in all 3 cases. This could possibly be explained due to no salt being incorporated in the diet analysis.
4. Intake of vitamins B12 & Selenium were extremely high (for individuals and as averages of FFQ and 24 hour recalls). A possible explanation is that since meats, seafoods and organ meats are rich sources of B12 and Selenium, and the protein intakes of the individuals are very high (see above).
5. The values of the FFQ's was much higher in most cases than the 24 hour recall values. This has possibly skewed most average values of dietary intake.

Clinical Evaluation:

1. The only clinical observation that was made was in P3, who had pale conjunctiva (possible anaemia). This coincided with the fact that the doctor prescribed an Iron sulphate supplement for her during her visit to the clinic, and the fact that her Iron intake was below 66.67% of the RDA in both 24-hour recall and FFQ.

Socio-demographic information:

1. 100% of population is of Mixed ancestry (Coloured) race.
2. Range of ages: 22 – 31. Average age: 27.
3. P2 is unemployed; coincides with most cramped living conditions (2 persons/bedroom), having the most children (3), and being Food Insecure (Income/month R300 < Household subsistence level R772,47).
4. P1 and P3 are both employed, and are Food Secure.

Systemic questioning:

1. All individuals changed eating, drinking or smoking habits since they knew of their pregnancy;

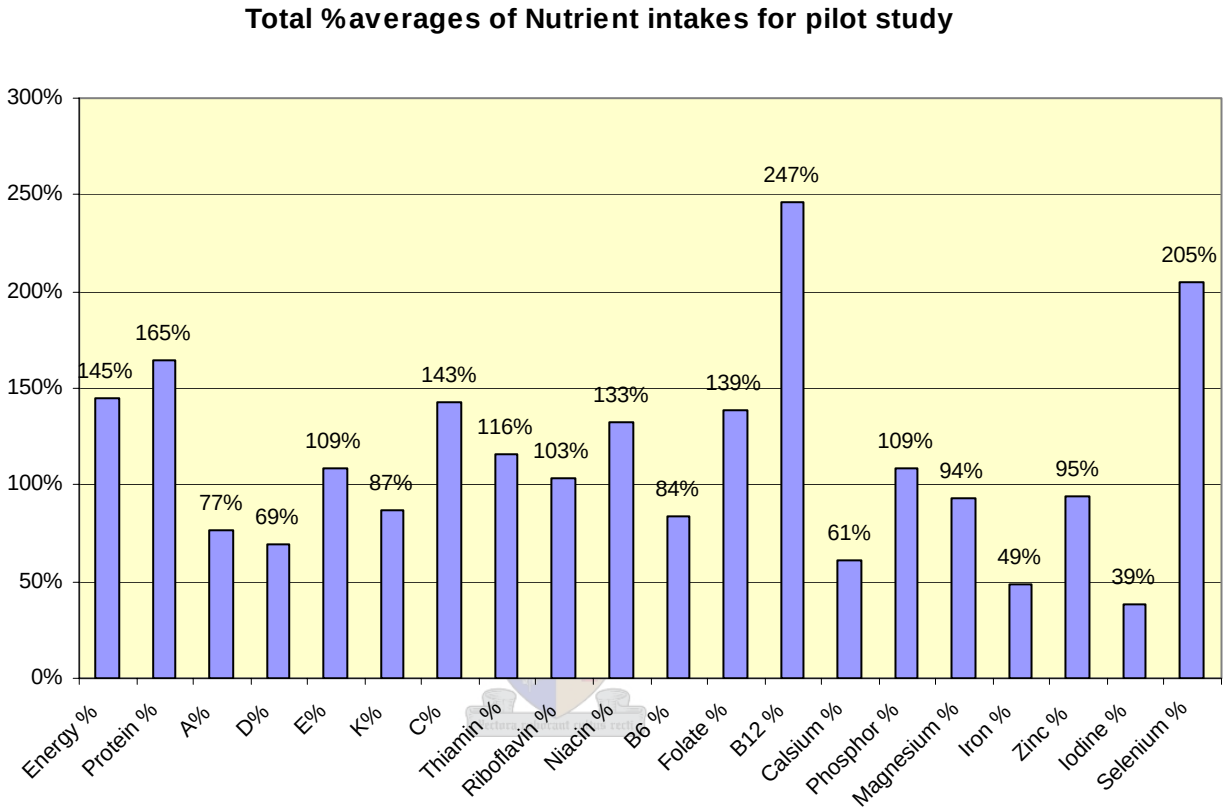
- P1: decreased smoking from 10 to 2 cigarettes; eats less as result of nausea,

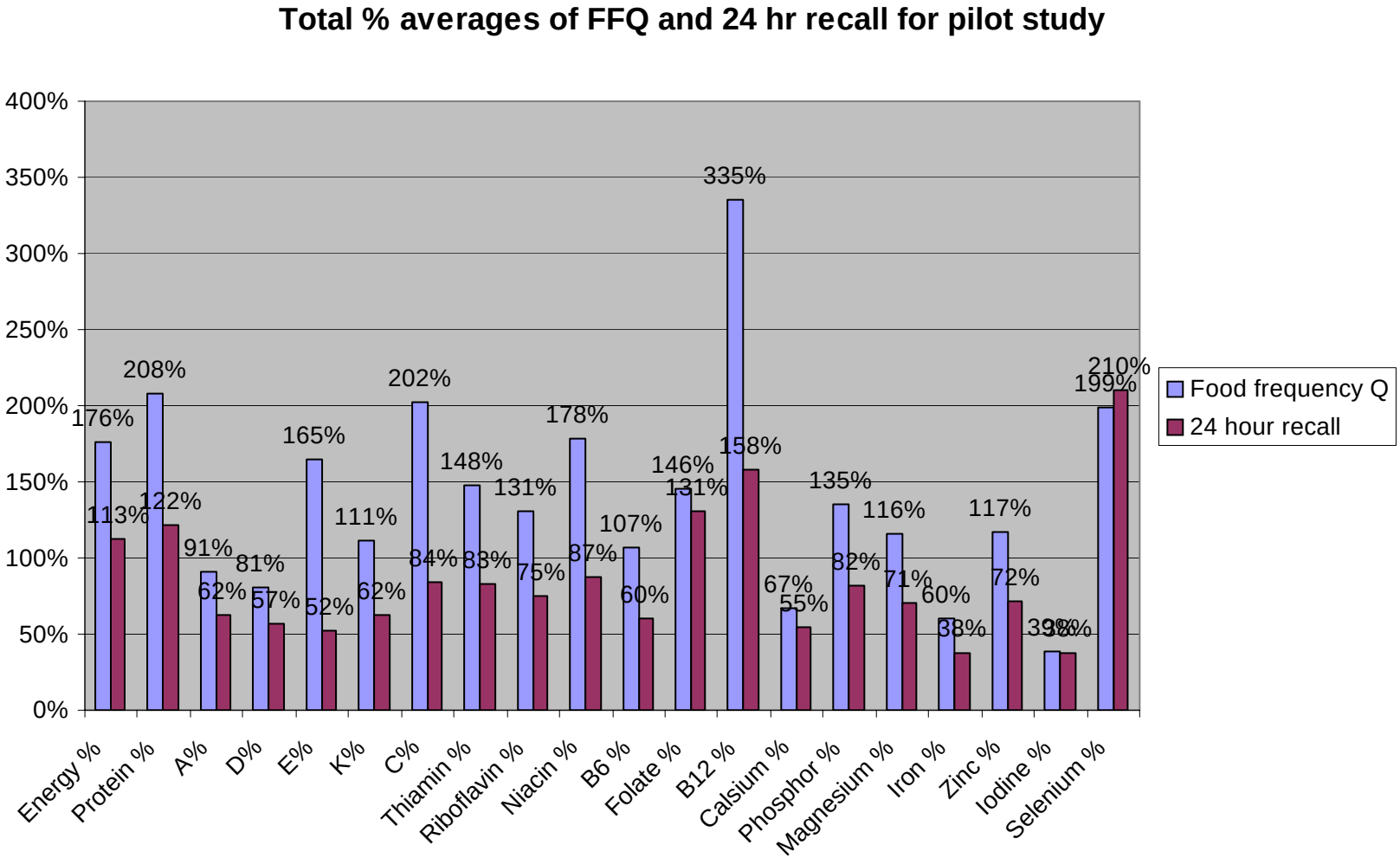
- P2: eats less as result of nausea,

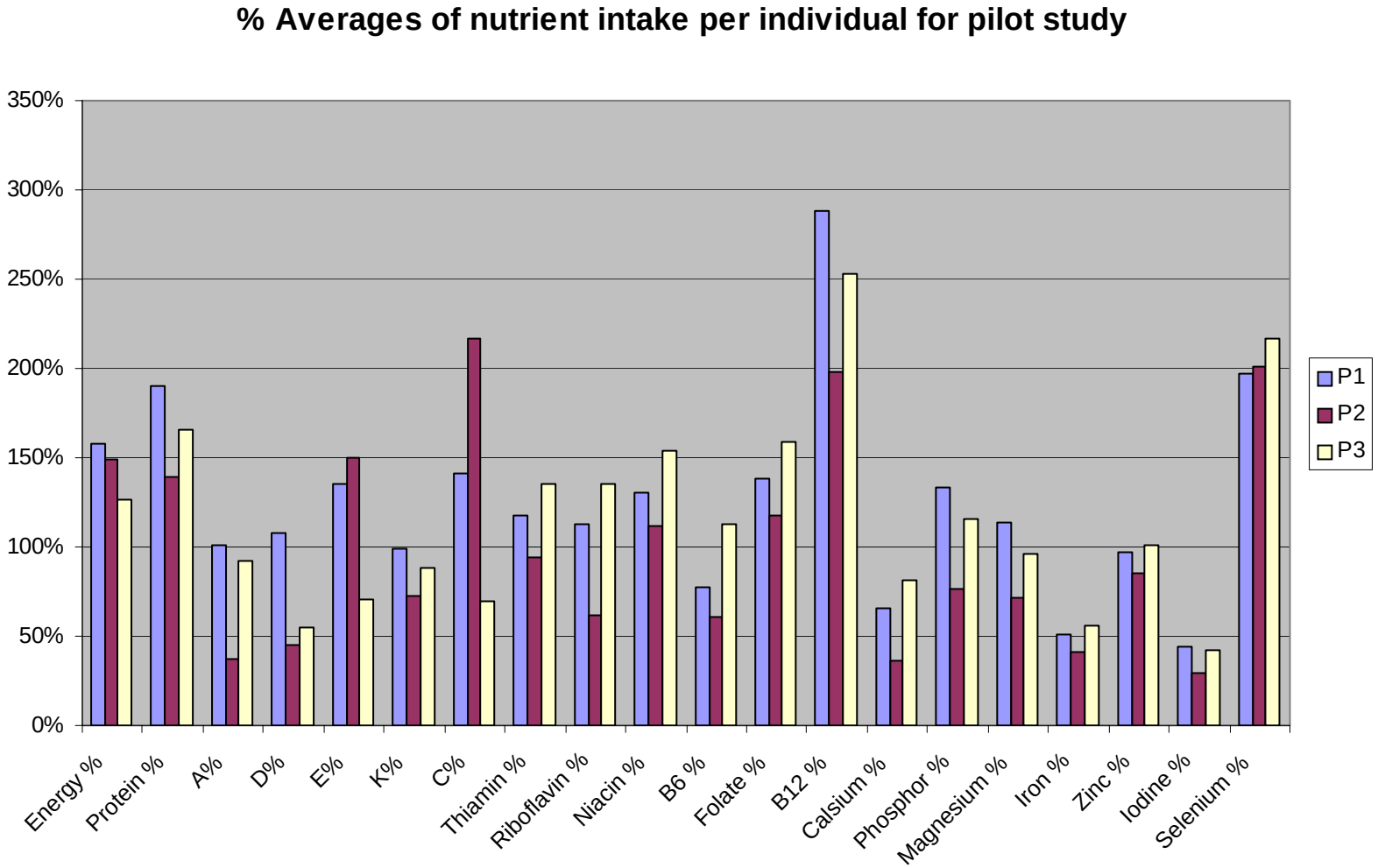
- P3: eats more.

2. P1 and P2 experienced a variety of gastro-intestinal symptoms, such as nausea, cramps, heartburn and diarrhoea. P3 experienced no symptoms, which could be attributed to her being in the later stage of the 2nd trimester, and the other 2 in the 1st trimester.
3. P1 and P2 have both developed an aversion to curry since being pregnant.
4. All of the women have developed "cravings" for certain foods since being pregnant (P1 and P2 for citrus fruits, and P3 for chocolates); but this could not be seen in their diet histories.
5. Only P1 admitted to smoking during pregnancy.
6. Only P1 admitted to skipping meals (lunch) every day of the week.
7. None knew of any previous obstetrical/gynaecological problems with previous pregnancy/ies.

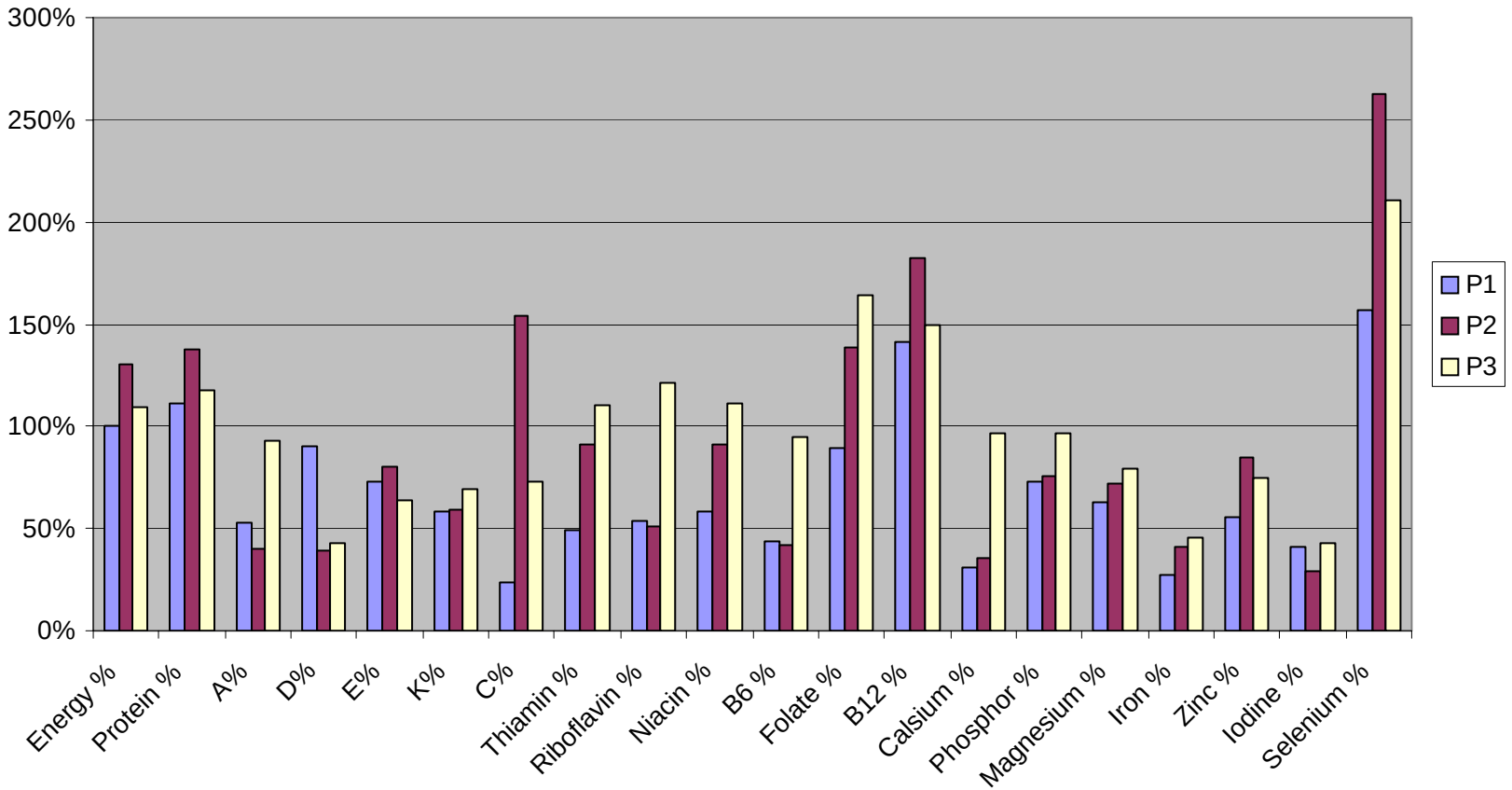
After delivery, within a month after delivery, anthropometric measurements will be done on the babies, and information regarding the delivery will be collected from the Road to Health Charts.


Total %averages of Nutrient intakes for pilot study
300%
250%
200%
150%
100%
50%
0%
145%
165%
77%
69%
109%
87%
143%
116%
103%
133%
84%
139%
247%
61%
109%
94%
49%
95%
39%
205%
Energy %
Protein %
A%
D%
E%
K%
C%
Thiamin %
Riboflavin %
Niacin %
B6 %
Folate %
B12 %
Calsium %
Phosphor %
Magnesium %
Iron %
Zinc %
Iodine %
Selenium %

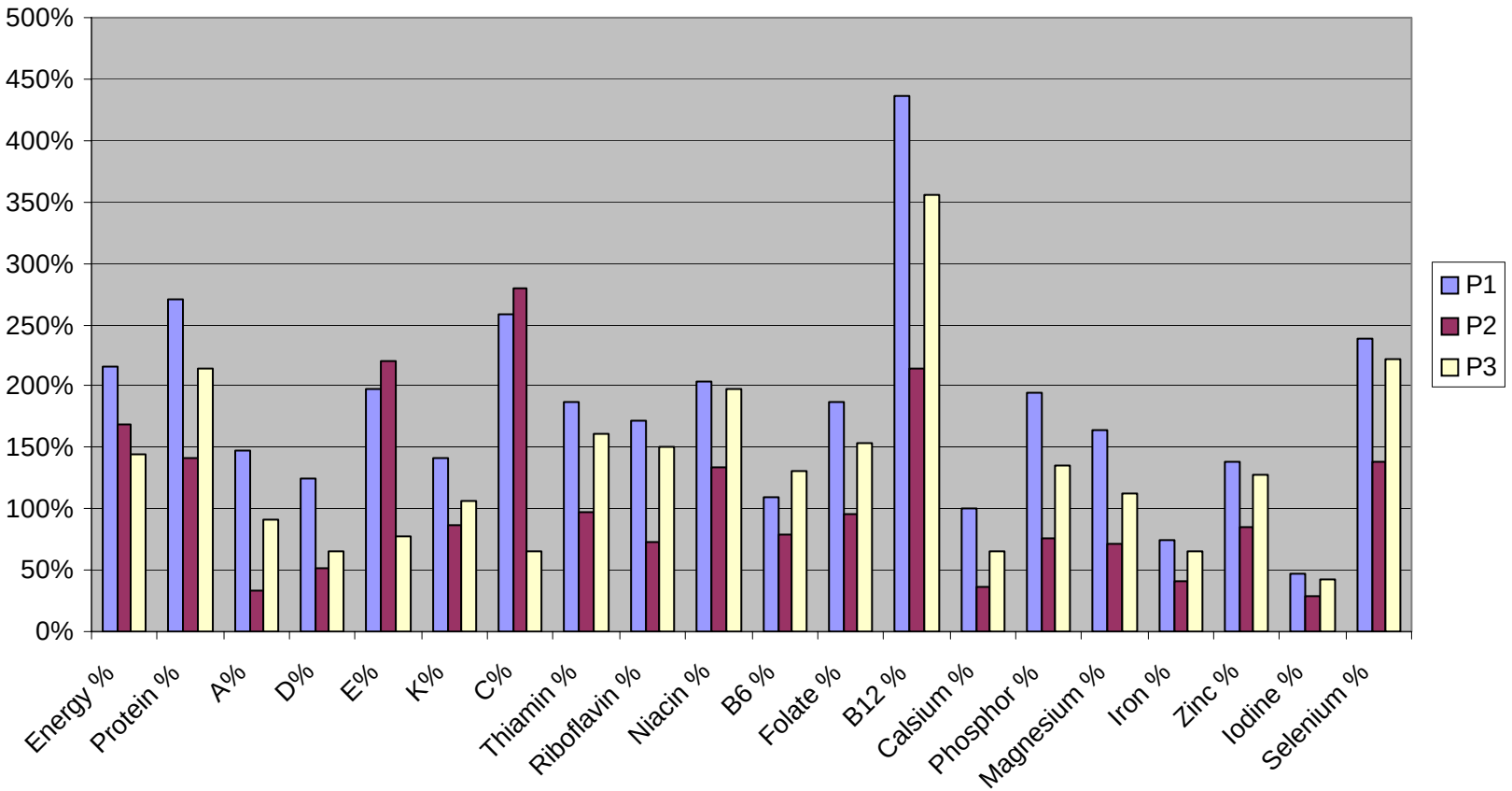

Total % averages of FFQ and 24 hr recall for pilot study
400%
350%
300%
250%
200%
150%
100%
50%
0%
176%
113%
208%
122%
91%
62%
81%
57%
165%
52%
111%
62%
202%
84%
148%
83%
131%
75%
178%
87%
107%
60%
146%
131%
335%
158%
67%
55%
135%
82%
116%
71%
60%
38%
117%
72%
199%
210%
Energy %
Protein %
A%
D%
E%
K%
C%
Thiamin %
Riboflavin %
Niacin %
B6 %
Folate %
B12 %
Calsium %
Phosphor %
Magnesium %
Iron %
Zinc %
Iodine %
Selenium %
Food frequency Q
24 hour recall


% Averages of nutrient intake per individual for pilot study
350%
300%
250%
200%
150%
100%
50%
0%
P1
P2
P3
Energy %
Protein %
A%
D%
E%
K%
C%
Thiamin %
Riboflavin %
Niacin %
B6 %
Folate %
B12 %
Calsium %
Phosphor %
Magnesium %
Iron %
Zinc %
Iodine %
Selenium %

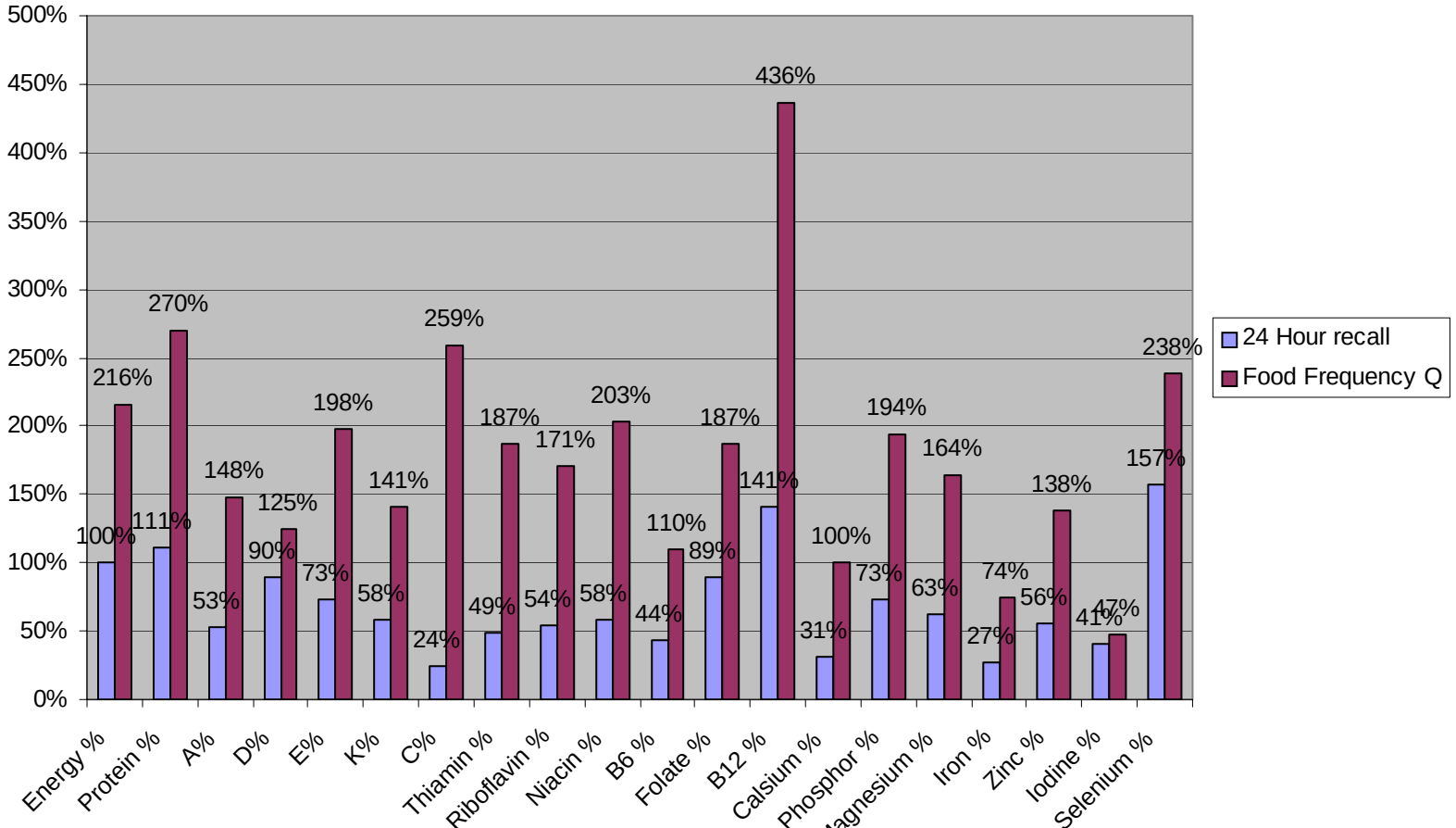
Comparison of nutrient intake of different individuals according to 24 hr recalls in % for pilot study

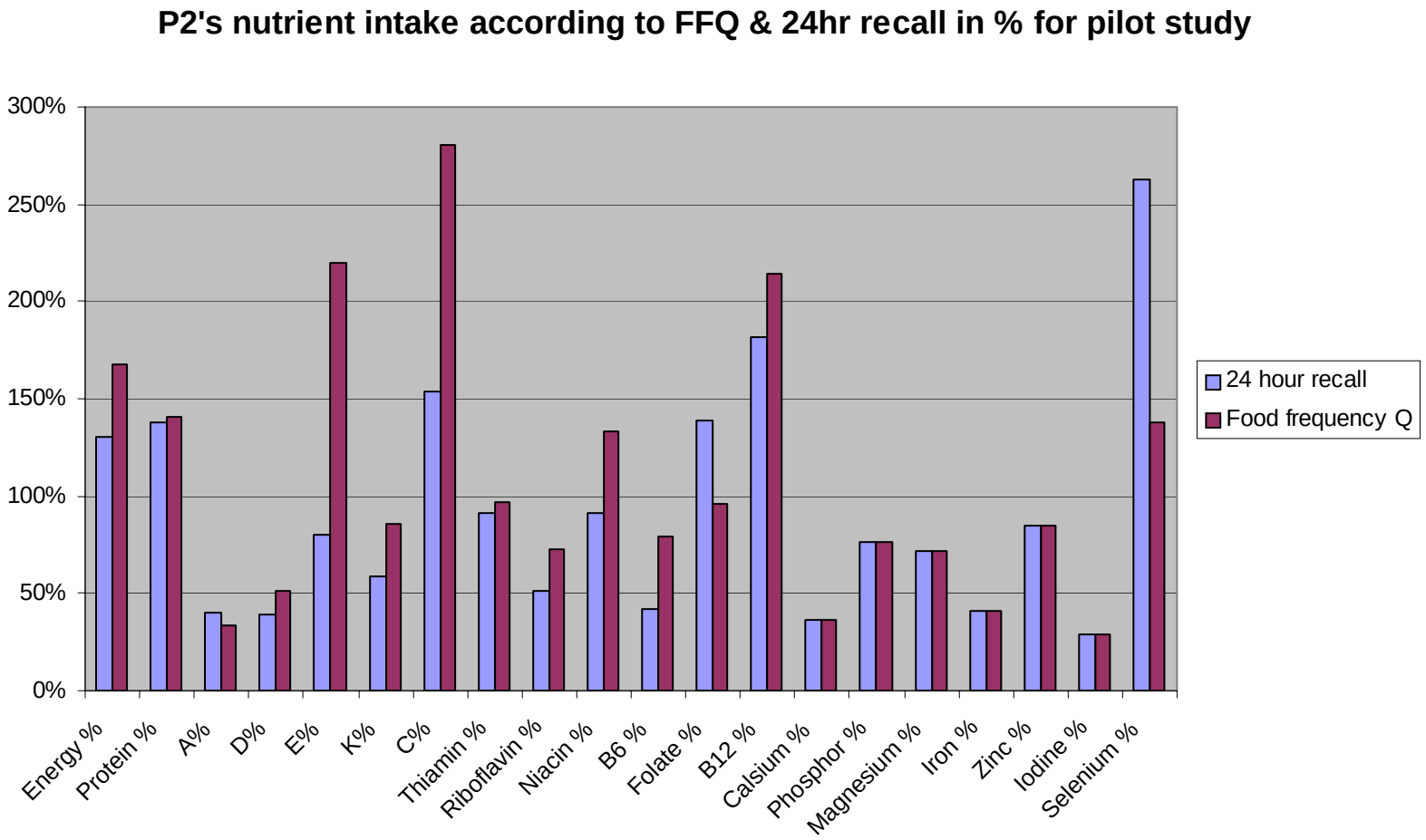


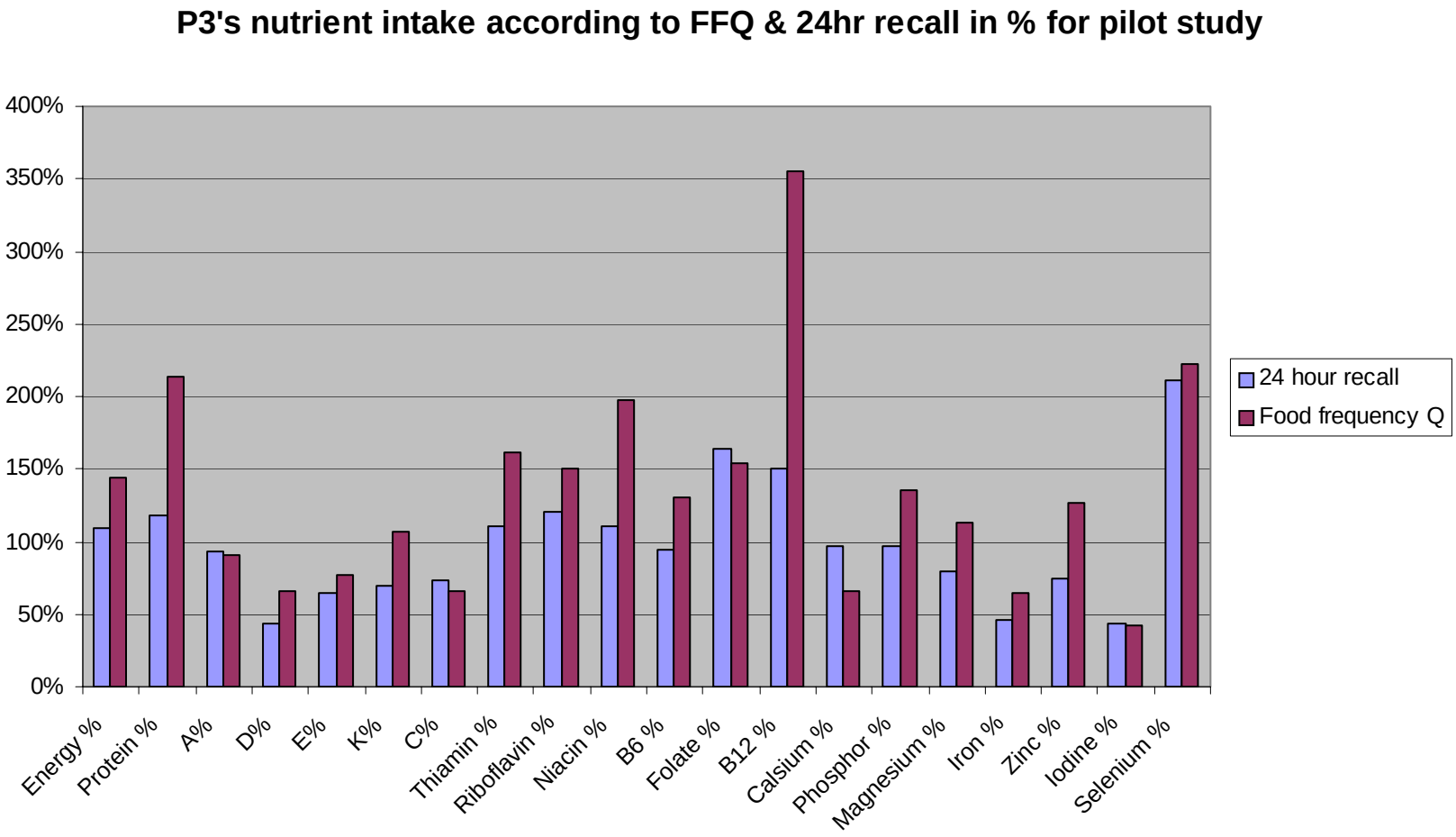
Comparison of nutrient intake of different individuals according to FFQ's in % for pilot study



P1's nutrient intake according to FFQ & 24hr recall in % for pilot study




P2's nutrient intake according to FFQ & 24hr recall in % for pilot study
300%
250%
200%
150%
100%
50%
0%
Energy %
Protein %
A%
D%
E%
K%
C%
Thiamin %
Riboflavin %
Niacin %
B6 %
Folate %
B12 %
Calsium %
Phosphor %
Magnesium %
Iron %
Zinc %
Iodine %
Selenium %
24 hour recall
Food frequency Q


P3's nutrient intake according to FFQ & 24hr recall in % for pilot study
400%
350%
300%
250%
200%
150%
100%
50%
0%
24 hour recall
Food frequency Q
Energy %
Protein %
A%
D%
E%
K%
C%
Thiamin %
Riboflavin %
Niacin %
B6 %
Folate %
B12 %
Calsium %
Phosphor %
Magnesium %
Iron %
Zinc %
Iodine %
Selenium %

Appendix 8

VALIDATION STUDY METHODOLOGY

1. Area

The researcher decided upon using a restaurant and guesthouse complex that she knew the proprietor of in Franschhoek as the area to gather another population out of to determine the validity and reliability of her dietary questionnaires.

2. Consent

The proprietor gave his consent that his staff be used in the validation study. He preferred that his establishment and staff remain anonymous.

3. Sample

After speaking with the staff of the establishment, 5 waitresses, 2 kitchen ladies and 2 laundromat ladies agreed to take part in the study. They were all of the mixed ancestry race, between the ages of 18 –40 years and all were not pregnant.

4. Procedure and Time

The researcher explained the methods to be used to the staff; three 24 hour recalls would be conducted with them, to gain information of dietary intake of 2 week days and 1 weekend day, and also conduct a FFQ to gain information of the intake of the week during which the 24-hour recalls will be conducted.

On 25th September 2000 (Monday), the day of receiving consent from the proprietor and the staff for participation in this study, the researcher conducted a 24-hour recall with each of them. She returned on Wednesday, 27 September 2000 and conducted the second 24-hour recall with each of them. On 30 September 2000 she conducted the third and last 24-hour

recall. Her last visit was on 2 October 2000 to conduct the FFQ, which was to cover the week period from the 25th September 2000 to the 1st October 2000 (Monday to Monday).

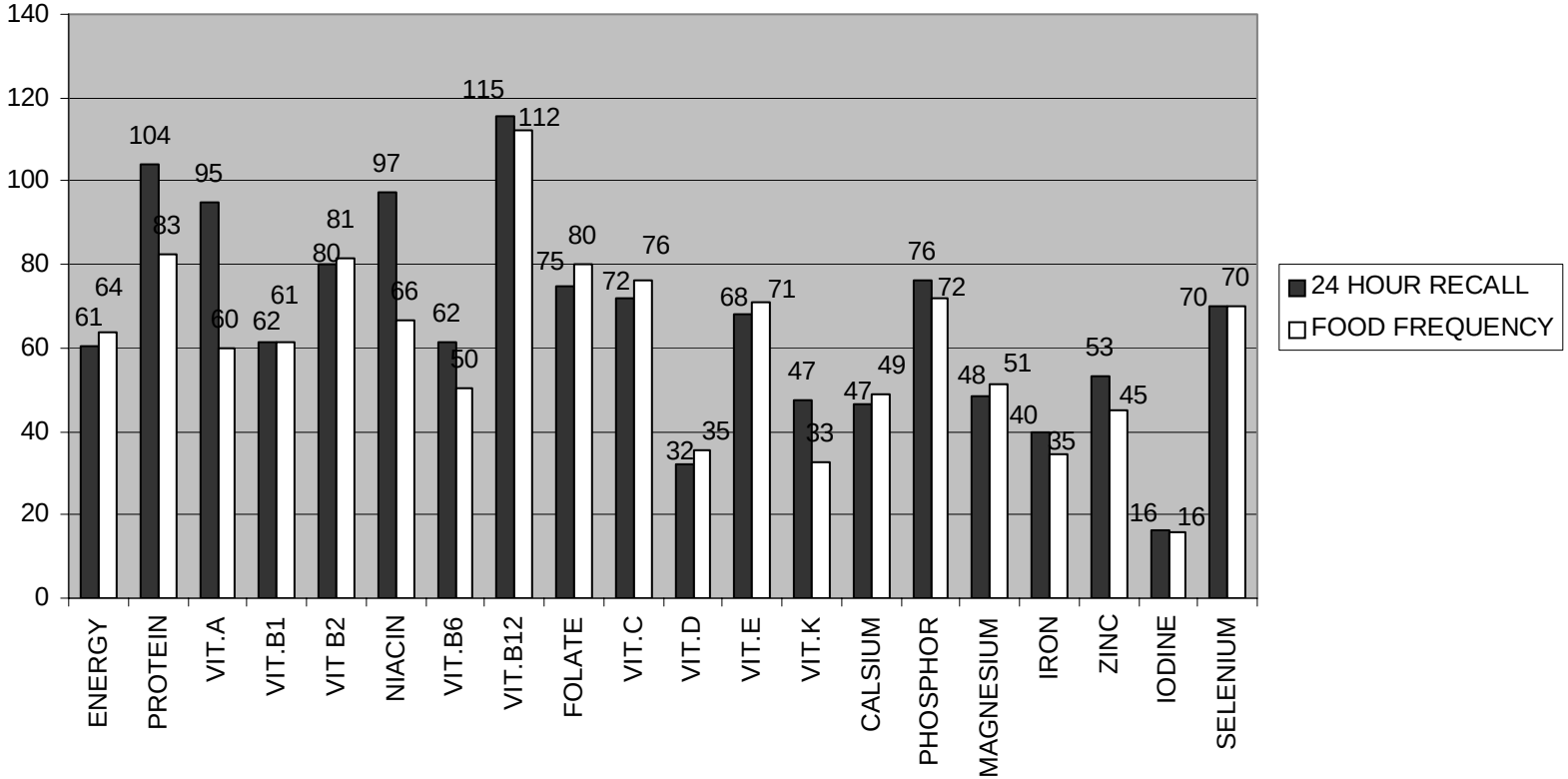
5. Results and Discussion

After comparing the averages of the three 24-hour recalls with that of the FFQ, it was found that the differences between most nutrients were acceptable and did not exceed 10 – 15%. (Protein, vitamin A and niacin did exceed 15%, but this could be explained by certain individuals' 24 hour recalls including large amounts of meat (protein and niacin rich) and pumpkin, sweet potato and liver (vitamin A rich). Therefore, the dietary methodology was valid and reliable, and approved for used in this study.

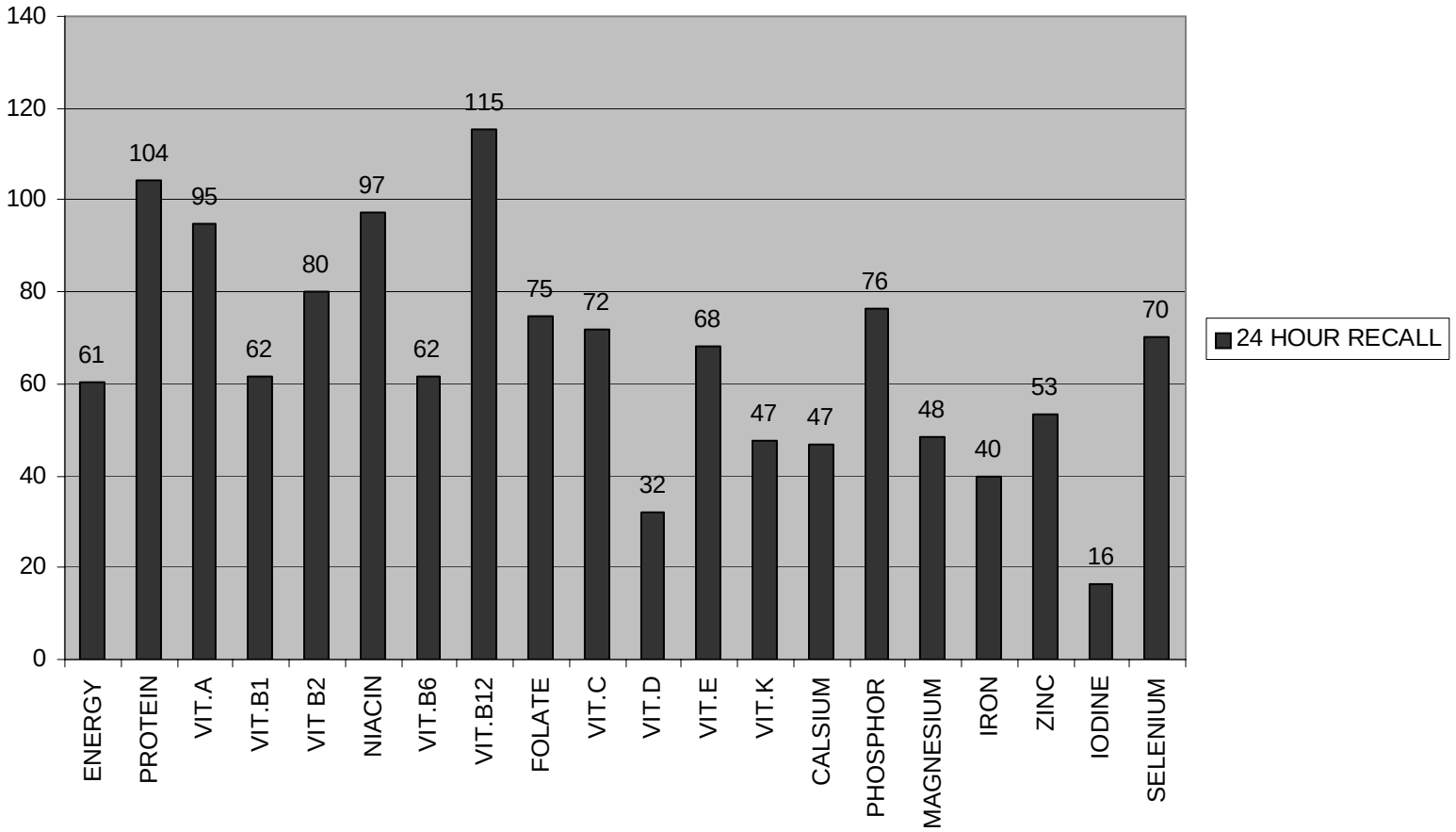
Appendix 9

VALIDATION STUDY GRAPHS

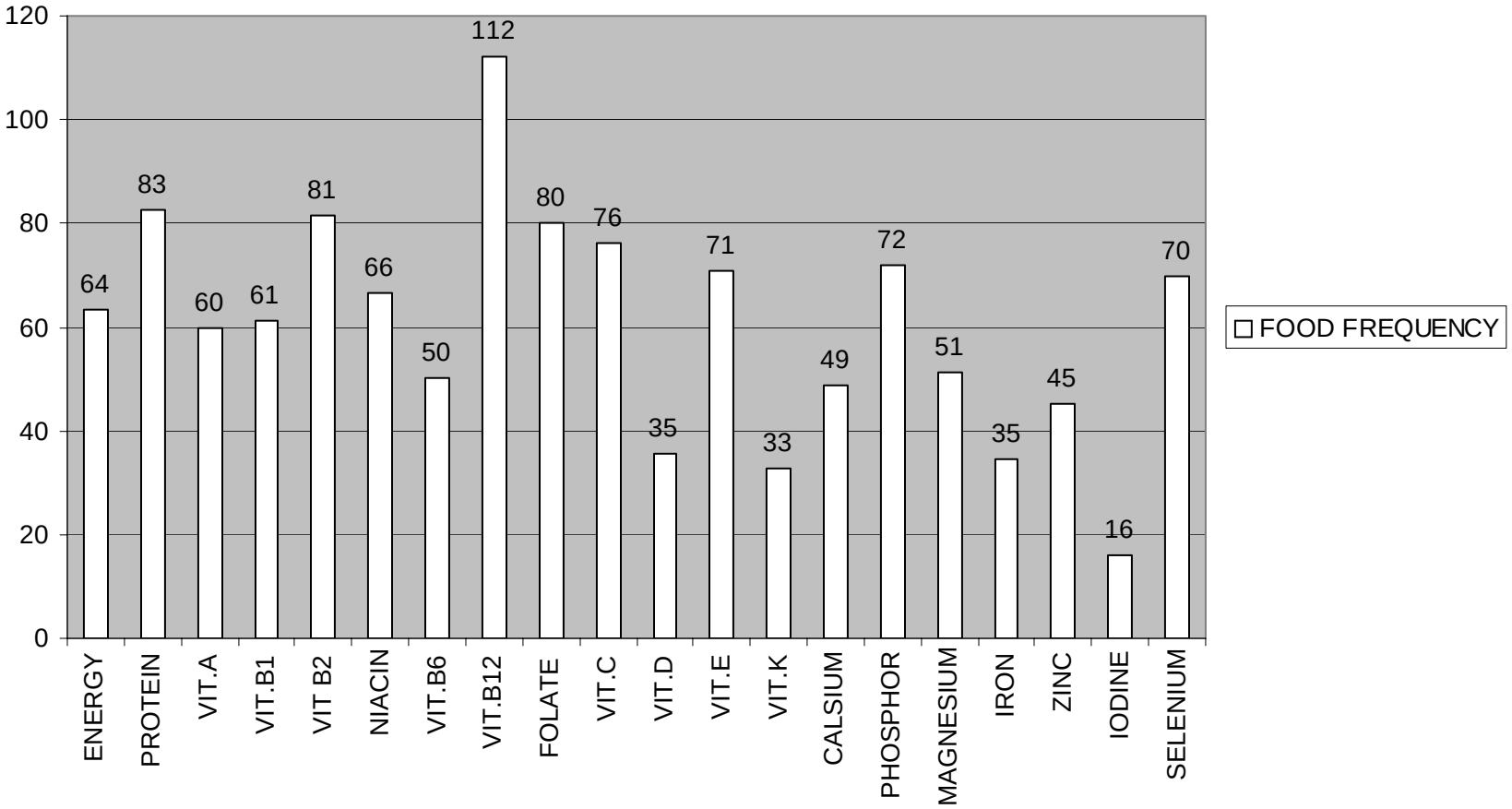
% AVERAGES OF NUTRIENT INTAKE ACCORDING TO 24 HR RECALLS & FFQ's FOR VALIDATION STUDY



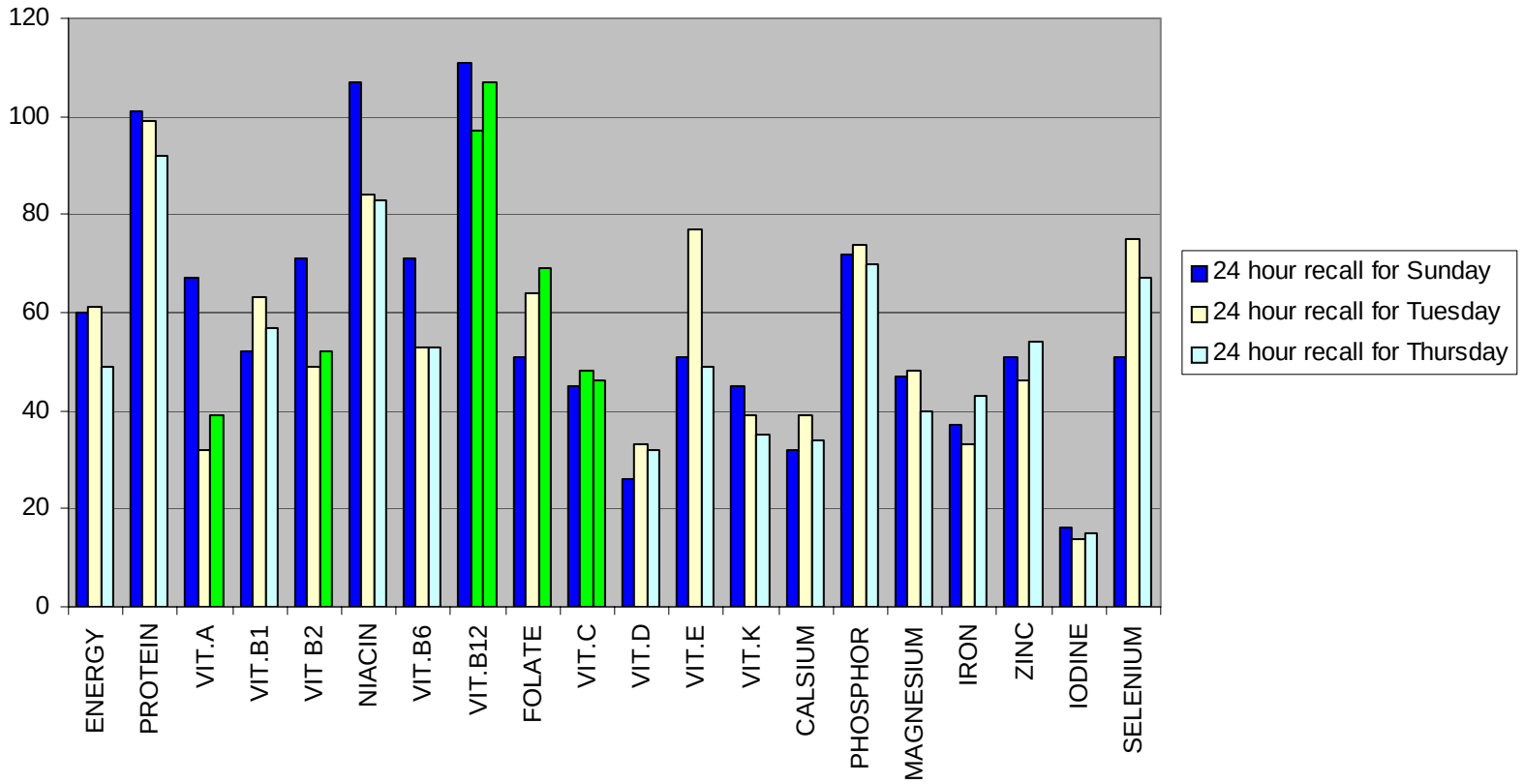
% AVERAGE OF NUTRIENT INTAKE ACCORDING TO 24 HR RECALLS FOR VALIDATION STUDY



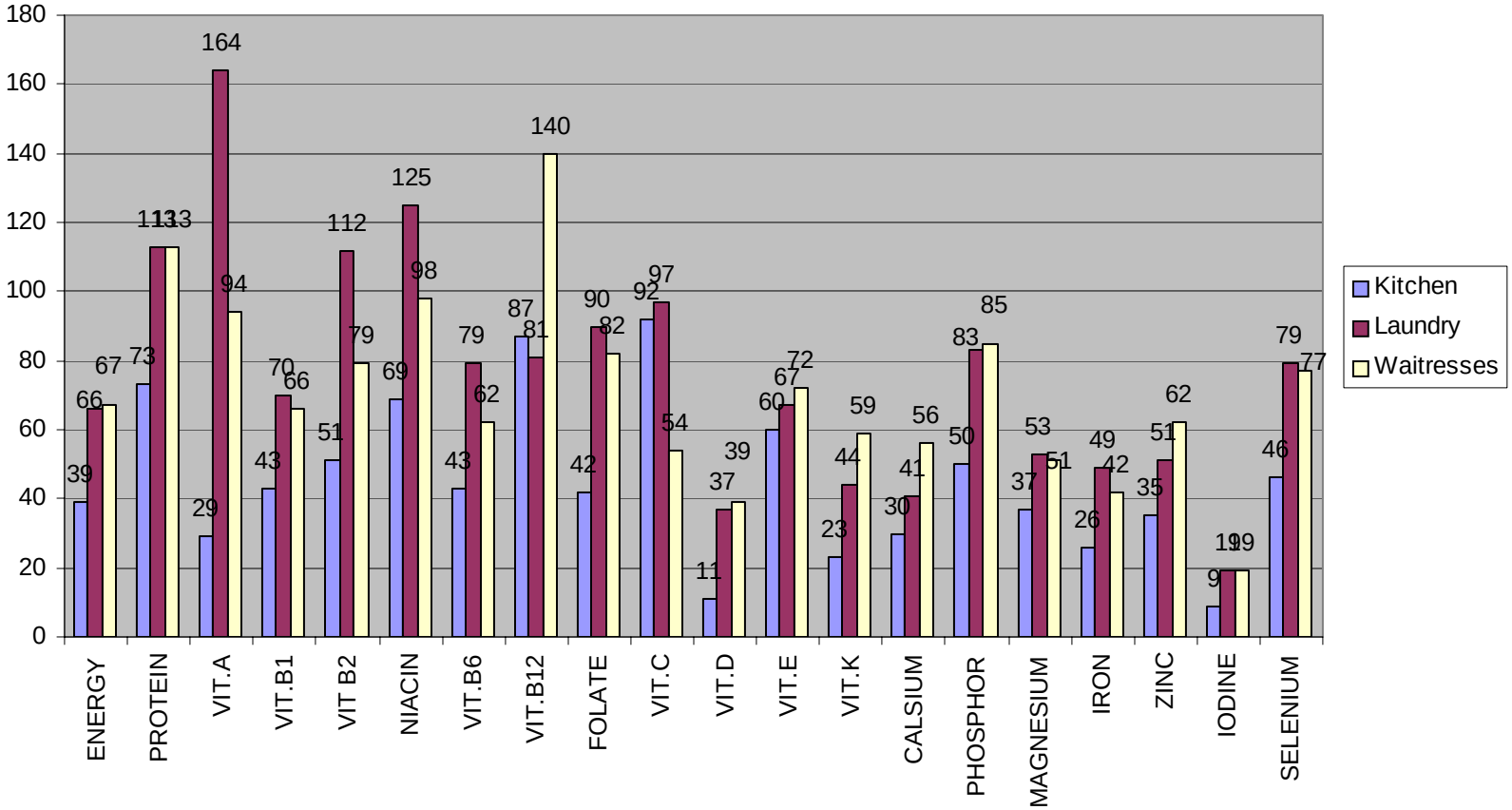
% AVERAGE OF NUTRIENT INTAKE ACCORDING TO FFQ's FOR VALIDATION STUDY



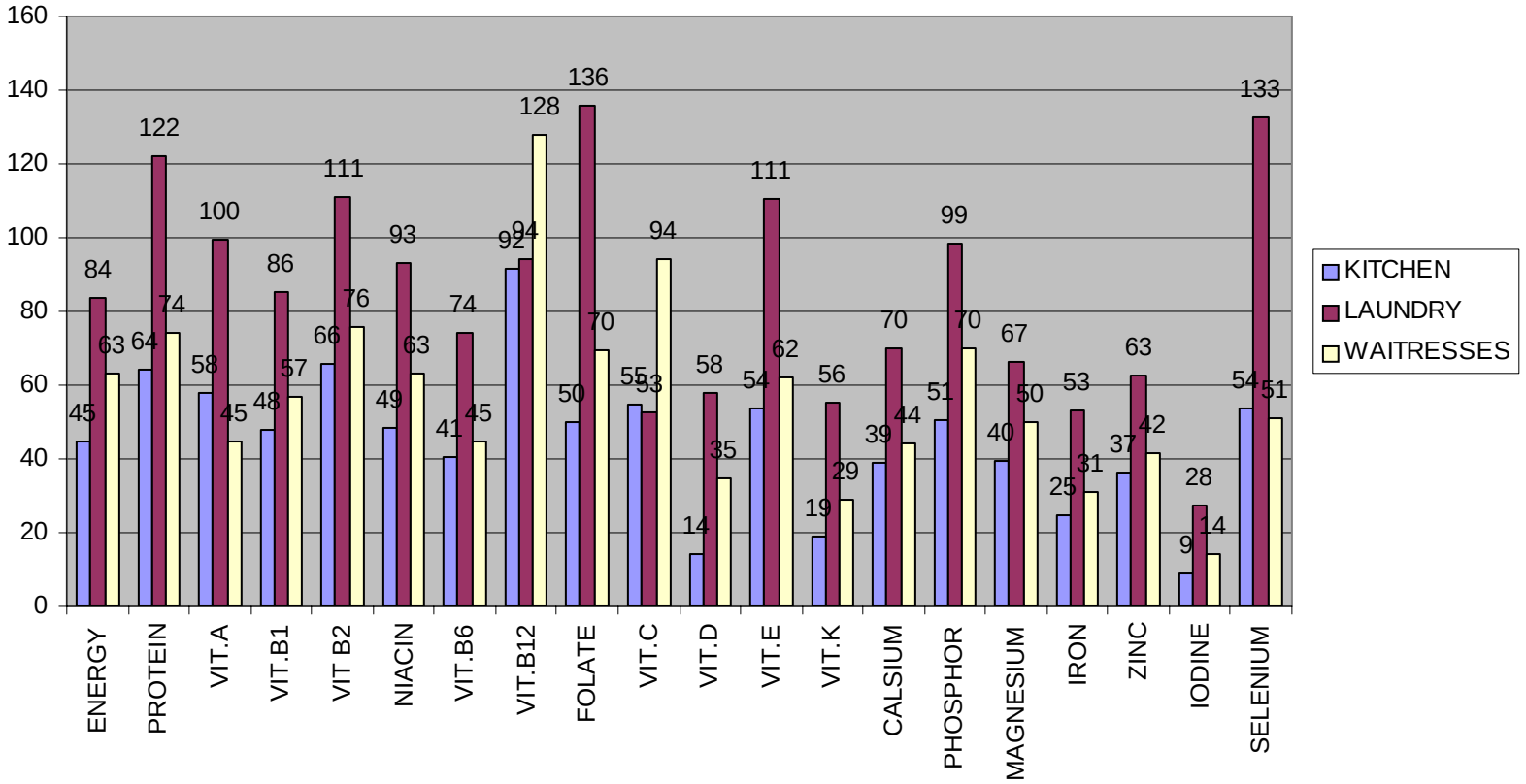
Comparison of % nutrient intake according to 24 hr recalls of different days of the week for validation study



Comparison of % nutrient intake according to 24 hr recalls among different professions for validation study



Comparison of% nutrient intake according to FFQ's among different professions for validation study



Appendix 10

RESEARCH STUDY METHODOLOGY

1. Recruitment

Each individual was approached by a FARR member at Hanover Park MOU and asked if she would like to participate in this study and a FARR study. After study methods were explained, she was questioned to determine whether she met the inclusion criteria for this study. If the mother met the criteria, a consent form was explained and signed. The researcher from FARR completed a questionnaire with the individual to attain demographic information (such as her age, obstetric history, living conditions, socio-economic status) and to determine her alcohol consumption status. An appointment was made for her first visit to FARR, at ±22 weeks gestational age of the individual's pregnancy.

2. First interview

Each individual was picked up at her home or another pre-arranged convenient location and taken to the FARR offices. If the woman was also taking part in FARR's ultrasound or maternal-infant study, she first completed her interview with a FARR researcher. (Ultrasound can accurately confirm gestational age of the pregnancy.)

After the mother had a break, the consent form (*Appendix 11*) for this study was read to each individual and she was given an opportunity to ask questions which the researcher answered. The consent form was signed and she received a copy and the researcher kept another. A FFQ was administered with the individual with the help of food models and serving utensils to ascertain usual intake since conception up until the first interview. A clinical examination was then completed. After, the woman was asked if she knew her usual pre-pregnancy weight as part of the anthropometric assessment. Her height, weight and mid-upper arm circumference were measured using standardised methods and equipment. Lastly a 24-hour recall was completed using food models and serving utensils as aids. Depending whether she gave her consent to have a blood sample drawn, a qualified nurse of FARR would then draw blood from the individual.

An appointment was made for her second visit to FARR at ±28 weeks gestational age. Each individual was offered coffee/tea and a sandwich during her visit. The FARR driver returned the mother and infant to her home or another convenient location.

3. Second interview

Each individual was picked up at her home or another pre-arranged convenient location and brought to the FARR office. If she was also taking part in FARR's ultrasound or maternal-infant study, she first completed her interview with a FARR researcher.

After the woman had a break, the general questionnaire for this study was completed with her, covering information such as her socio-demographic status, her past health and obstetric history, lifestyle and dietary evaluation regarding her present pregnancy. Another 24-hour recall was completed using food models and serving utensils as aids. A second weight appraisal and clinical examination was done.

An appointment was made for a third visit to FARR at ±36 weeks gestational age of her pregnancy. Each individual was offered coffee/tea and a sandwich during her visit. She was dropped off afterwards at her home or another convenient location.

4. Third interview

Every woman was picked up at her home or another pre-arranged location and brought to the FARR office. If the individual was also taking part in FARR's ultrasound or maternal-infant study, she first completed her interview with a FARR interviewer.

After she had a break, another FFQ was administered to determine usual intake of the time lapsed between the first and third interview. A last 24-hour recall was completed. Food models and serving utensils were used as aids with both food intake questionnaires. A third weight appraisal and clinical examination was done.

The mother was told that FARR would be in contact with Hanover Park MOU in and around the expected delivery date (EDD) of her pregnancy, to ascertain the exact delivery date. She would be contacted at home after the delivery and an appointment would be made for her and

her baby's postnatal visit and interview, when the baby was approximately 1 month old. Each individual was offered coffee/tea and a sandwich during her visit. The FARR driver returned the mother and infant to her home or another convenient location.

5. Birth information

With each delivery at Hanover Park MOU a birth summary is made. It contains information such as birth weight, birth head circumference, date of birth and any pregnancy and/or delivery complications. Because of the longstanding relationship between Hanover Park MOU and FARR, FARR had access to the birth summaries and it was made available to the investigator after the births.

6. Postnatal visit and interview

Each mother and baby were picked up at her home or another pre-arranged location and brought to the FARR office.

A paediatrician of FARR who is specialised in identifying alcohol related signs in the young, examined the baby and questioned the mother about her baby's health. The doctor did a complete anthropometric assessment and scrutinized the baby for any alcohol related or other medically significant signs. A summary of his findings was included on the baby's physical examination sheet (*Appendix 4).*

Each individual was offered coffee/tea and a sandwich during her visit. She was dropped off afterwards at her home or another convenient location.

Appendix 11

UNIVERSITEIT VAN STELLENBOSCH
UNIVERSITY OF STELLENBOSCH

Researcher: Ingrid Klinger
Study Leaders: Professor D. Labadarios
Ms D. Marais
Professor D. Viljoen

University of Stellenbosch

In Collaboration with the Foundation for Alcohol Related Research (FARR)

Informed Consent for Masters in Nutrition Study:

THE NUTRITIONAL STATUS OF PREGNANT WOMEN IN RELATION TO ALCOHOL CONSUMPTION DURING PREGNANCY AND PREGNANCY OUTCOME.

I, the undersigned, ...

[ID.....], of .....

.....(address)

A. Declare that:

1. I have been invited to participate in above mentioned study by the University of Stellenbosch in collaboration with the Foundation for Alcohol Related Research (FARR).

2. It was explained to me that:

2.1 The ***purpose of this study*** is to determine the nutritional status of heavy drinking and light drinking (or abstaining) women during pregnancy, and the outcome of their pregnancy. The "outcome of their pregnancy" refers to the following: after birth the baby's nutritional status will be compared to that of the mother; and the mother's alcohol use will be related to the presence of any alcohol-related abnormalities in the baby.

2.2 The following ***procedures*** will be done at the following ***places*** at the specified ***time***:

Each interview will be conducted in private by the researcher who is a qualified dietician.

I will be interviewed on 3 occasions by the researcher. During the 1st interview, my weight will be measured and I will be questioned about my food intake. I will then be asked to give a blood sample (3 tablespoons of blood in total) for vitamin and mineral analysis. It will be drawn by a qualified and experienced nurse, and it should cause me no major discomfort. I was told that I am not obliged to give the blood sample, and by not giving it will not mean a penalty or loss of benefits for me.

Fakulteit Geneeskunde • Faculty of Medicine
Departement Menslike Voeding • Department of Human Nutrition
Posbus / P O Box 19063, 7505 Tygerberg, Suid-Afrika / South Africa
Tel. (27) 021 - 9389259, Fax: (27) 021 - 9332991

During the 2nd interview, I will be required to complete a questionnaire with researcher that will cover basic information about my living conditions, income, health and food intake. Another 24-hour recall, as was done at the 1st interview will be completed. If any of these questions make me feel uncomfortable, I am not obliged to answer.

After the questionnaire, the researcher will take my weight, height and mid-upper arm circumference and have a look at my skin, hair, eyes and mouth to help determine my nutritional status and if I have any nutrient deficiencies.

At the 3rd and last interview, I will be weighed again and another questionnaire regarding my food intake will be completed with me.

I will be notified in advance when the interviews will take place, and I have the right to say no if the time doesn't suit me or if the interviews make me uncomfortable.

When my child is approximately one month old, I give consent that my baby may be weighed and it's head circumference be taken. This will be done to determine whether my baby is growing well. My baby will also be examined by a doctor from FARR. I can be with my baby at all times during the examination and measurement taking. Additional information such as birth weight, birth head circumference, gestational age (how many weeks old my baby was at birth) and details about the birth such as apgar count will be taken from my baby's Road to Health Chart.

Certain information gathered from the FARR studies will be shared with this study. Under all circumstances will such information be treated confidentially. Specifically, the researcher will not know information regarding my alcohol intake until the data collection is completed.

I will be transported to UCT Medical School at no cost to me, in a vehicle provided by FARR, driven by a licensed driver, and returned to my home, or alternatively to a place which is convenient to everyone.

2.3 The study aims to have a total of 100 women taking part. I will be required to sit in for 3 interviews to complete questionnaires, have clinical assessment and anthropometric measurements done on me; and a blood sample taken. My baby's weight and head circumference will only be measured once by the researcher.

3. I have been told that the methods used in this study should not cause me any major ***discomfort***. No ***risks*** are anticipated during the study either.

4. I understand that the potential ***benefits*** of the study include the assessment of my and my baby's nutritional status, i.e. if I am interested, the researcher can inform me whether my or my baby's weight is normal, too much or too little.

   If, at any time, I need nutritional advice, I can contact the researcher for informal counselling with no payment required. Such information can improve my health, the health of my baby and that of any children I may have in the future.

   The information gathered during the study will be used to identify the risks involved with heavy or light drinking during pregnancy. Information regarding nutritional status, living conditions, income, health and dietary intake will be used to describe the population of women who attend Hanover Park MOU, and identify potential risks for women's health or that of their pregnancies. Possible intervention or support programmes can be initiated to improve the health of the population.

5. I was told that all information received about me, my baby, and my family will be kept strictly ***confidential*** and will not be used to reveal my identity or to compromise me or my family's well being. No names or other form of identification will be used in reporting the research results in the form of a Masters Study thesis.

6. After completion of the study, there are no plans to provide ***feedback*** to me, as this is general research to identify risk factors in an entire population.

7. I understand that my participation is ***voluntary*** and I may ***withdraw*** from the study at any time. If I do not participate, or if I stop my participation during the study, there will be no penalty or loss of benefits or medical care to which I am otherwise entitled. If I have any questions about my rights as a subject all relating to the study itself, I may contact the researcher or any of the following people:

Ingrid Klinger Tel. no. (021) 406-6228

Professor Demetre Labadarios Tel. no. (021) 938-9259

Ms Debbi Marias Tel. no. (021) 938-9473

Professor Denis Viljoen Tel. no. (011) 489-9239

I also understand that the researcher may withdraw me from the project at any time if the researcher feels that it is in my best interest.

8. This letter is available in English and Afrikaans and an ***interpreter*** can be made available, if requested. The procedures of the study have been explained to me verbally and I have had a chance to ask questions, and I am satisfied with the answers I received.

9. I have not been forced to participate in this study and I am reminded again that I may withdraw at any time without penalty.

10. Participation in this study will not cost me anything.

B I voluntarily agree to take part in the study and acknowledge that I have been given a copy of this letter. I am not giving up my legal rights.

Signed at ..... on .....2000.

....

Signature of subject Witness

Declaration by Researcher

I, ..., declare that I:

1. The information within this document was explained to the subject named above;
2. I enquired if the subject had any questions and I cleared up any uncertainties;
3. That this conversation took place in Afrikaans, English, Xhosa, other(.....) and that no interpreter was used/ an interpreter was used, namely

Dr/Mr/Ms .....

Signed at ..... on .....2000.

....

Researcher Witness

Declaration by Interpreter

I, _____ declare that:

1. I translated the above document from Afrikaans/English to the patient or patients' representative as well as all the answers asked by the patient/patients' representative addressed to the researcher/researchers' representative and that I translated this person's answers.

2. The information that I gave was a virtually correct replica of what was told to me.

Signed in _____ on _____

_____ _____

Interpreter's signature Witness's signature

UNIVERSITEIT VAN STELLENBOSCH
UNIVERSITY OF STELLENBOSCH

Navorser: Ingrid Klinger
Studieleiers: Professor D. Labadarios
Me D. Marais
Professor D. Viljoen

Universiteit van Stellenbosch
In Samewerking met die Stigting vir Alkohol-verwante Navorsing (SAVN)

Ingeligte Toestemming vir Meestersstudie in Voeding:

DIE VOEDINGSTATUS VAN SWANGER VROUE IN VERWANTSKAP MET ALKOHOLINNAME TYDENS SWANGERSKAP EN SWANGERSKAPSUITKOMS.

Ek, die ondergetekende, ...

[ID.....], van .....

.....(adres)

A. Bevestig dat:

1. Ek genooi is om deel te wees van bogenoemde studie van die Universiteit van Stellenbosch, in samewerking met die Stigting vir Alkohol-verwante Navorsing (SAVN).

2. Dit is aan my verduidelik dat:

2.1 Die ***doel van die studie*** is om die voedingstatus van swaar drinkende en lig drinkende (of geheelonthoudende) vroue tydens swangerskap, en die uitkoms van hulle swangerskappe te bepaal. Die "swangerskapsuitkoms" verwys na die volgende: na geboorte sal die baba se voedingstatus vergelyk word met die ma s'n; en die moeder se alkohol gebruik word vergelyk met die teenwoordigheid van enige alkohol-verwante abnormaliteite van die baba.

2.2 Die volgende ***prosedures*** gevolg sal word by die volgende ***plekke*** en op gespesifiseerde ***tye***:

Elke onderhoud word in privaatheid gedoen deur die navorser wat 'n gekwalifiseerde dieetkundige is.

Daar sal 3 onderhoude met my gevoer word deur die navorser. Gedurende die 1ste onderhoud, sal my gewig geneem word en ek sal gevra word oor my voedselinname. Ek sal dan gevra word om 'n bloedmonster te gee (3 eetlepels bloed in totaal) vir vitamien- en mineraalanalise. Dit sal getrek word deur 'n gekwalifiseerde verpleegster met baie ondervinding in bloedtrek, en behoort my nie baie ongemak te veroorsaak nie. Ek is gesê dat ek nie verplig is om 'n bloedmonster te gee nie, en deur dit te weier ek nie gepenaliseer sal word nie.

Tydens die 2de onderhoud, sal daar van my verwag word om 'n vraelys te voltooi wat basiese inligting rakende my lewensomstandighede, inkomste, gesondheid en voedselinname behels. As enige van die vrae my ongemaklik maak, is ek nie verplig om dit te antwoord nie.

Na die vraelys, sal die navorser my gewig, lengte en bo-armomtrek vat en na my vel, hare, oë en mond kyk om my voedingstatus te bepaal en te kyk of ek enige nutriënttekorte het.

By die 3de en laaste onderhoud, sal ek weer geweeg word en nog 'n vraelys rakende my voedselinname sal met my voltooi word.

Ek sal vroegtydig in kennis gestel word wanneer die onderhoude sal plaasvind, en ek het die reg om te nee te sê as die onderhoude vir my op 'n ongeleë tyd is of my ongemaklik laat voel.

Wanneer my baba ongeveer een maand oud is; gee ek toestemming dat my baba geweeg word en hy/sy kopomtrek geneem word. Dit sal gedoen word om te bepaal of my baba goed groei. 'n Dokter van SAVN sal ook my baba ondersoek. Ek kan te alle tye by my baba wees tydens die ondersoek en metings. Addisionele inligting soos geboortegewig, geboortekopomtrek, gestasie-ouderdom (hoeveel weke my baba oud was by geboorte) en details van die geboorte soos apgartelling sal verkry word van my baba se "Road to Health Chart".

Sekere inligting wat deur die SAVN studies ingesamel sal word, sal met hierdie studie gedeel word. Onder alle omstandighede sal sulke inligting streng vertroulik hanteer word. Spesifiek, sal die inligting van my alkohol inname nie bekend wees aan die navorser nie, totdat die data-versameling van die studie voltooi is.

Ek sal vervoer word na UK Mediese Skool met geen onkoste vir my nie, in 'n voertuig wat deur SAVN verskaf word, wat deur 'n gelisensieërde bestuurder bestuur sal word; en weer teruggeneem word na my huis toe, of op 'n alternatiewe plek afgelaai word wat vir almal gemaklik is.

2.3 Die studie sal poog om 'n totaal van 100 vroue in te sluit. Daar word van my gevra om vir 3 onderhoude te kom om vraelyste te voltooi, kliniese evaluering en antropometriese metings te laat doen; en 'n bloedmonster te neem. My baba se gewig en kopomtrek sal ook slegs eenmaal geneem word deur die navorser.

3. Daar is aan my verduidelik dat die metodes wat gebruik sal word my nie baie ***ongemak*** sal veroorsaak nie. Geen ***risiko's*** word voorsien tydens die studie nie.

4. Die moontlike ***voordele*** van die studie sluit in die bepaling van my en my baba se voedingstatus, met ander woorde, as ek belangstel, kan die navorser my meedeel of my of my baba se gewig normaal, te veel of te min is.

   As, te enige tyd, ek voedingsadvies benodig, kan ek die navorser kontak vir informele raadgewing sonder enige koste. Sulke inligting kan my gesondheid, my baba se gesondheid en die van enige ander kinders wat ek mag hê in die toekoms, verbeter.

   Die inligting wat tydens die studie versamel sal word, gaan gebruik word om moontlike risiko's verbonde aan swaar of ligte drink tydens swangerskap te identifiseer. Inligting rakende voedingstatus, lewensomstandighede, inkomste, gesondheid en dieetinname sal gebruik word om die populasie van swanger vroue wat Hanover Park MOU besoek, te beskryf, en om moontlike risiko's vir die vroue se gesondheid en swangerskappe te identifiseer. Moontlike intervensie of ondersteuningsprogramme kan begin word om die gesondheid van die populasie te verbeter.

5. Ek is vertel dat alle inligting wat verkry is oor my, my baba, en my familie sal as streng ***vertroulik*** hanteer word en sal op geen manier gebruik word om my identiteit bekend te maak of ek en my familie te benadeel nie. Geen name of ander vorm van identifikasie sal gebruik word tydens rapportering van navorsingsresultate nie.

6. Na die voltooiing van die studie, is daar geen planne om aan my ***terugvoer*** te gee nie, omdat dié algemene navorsing is wat risikofaktore identifiseer in 'n hele populasie.

7. Ek verstaan dat my deelname aan die studie ***vrywillig*** is en ek kan te enige tye ***onttrek*** aan die studie as ek dit so verkies. As ek nie deelneem nie, sal daar geen straf of verlies van voordele of mediese sorg wees nie wat my andersins toekom nie. As ek enige vrae het oor my regte as individu wat die studie betref, kan ek die navorser of enige van die volgende persone kontak:

Ingrid Klinger Tel. no. (021) 406-6228

Professor Demetre Labadarios Tel. no. (021) 938-9259

Me Debbi Marias Tel. no. (021) 938-9473

Professor Denis Viljoen Tel. no. (011) 489-9239

Ek verstaan ook dat die navorser my enige tyd kan onttrek aan die studie as die navorser voel dat dit tot my voordeel is.

8. Die brief is beskikbaar in Engels en Afrikaans en 'n ***tolk*** is beskikbaar indien ek een wou aanvra. Die prosedures van die studie is mondeliks aan my verduidelik en ek het die geleentheid gehad om ***vrae*** te vra; en ek is tevrede met die antwoorde wat ek gekry het.

9. Ek is nie forseer om deel te neem nie en ek word weer daaraan herinner dat ek enige tyd kan onttrek aan die studie sonder enige penilisasie.

10. Deelname aan dié studie kos my niks geld nie.

B. Ek gee hiermee vrywillig toestemming om deel te neem en ek bevestig dat ek 'n kopie van dié toestemmingsbrief gekry het. Ek gee nie enige van my regte op nie.

Geteken te ..... op .....2000.

....

Handtekening van Individu Getuie

Verklaring deur Navorser

Ek, ..., verklaar dat ek:

Die inligting vervat in hierdie dokument aan die individu (bogenoem) verduidelik het;

Sy het die geleentheid gekry om vrae te vra en ek het enige onduidelikhede opgeklaar;

Hierdie gesprek het in Afrikaans, Engels, Xhosa, ander (.....) plaasgevind en dat 'n tolk nie gebruik is/ wel gebruik is, naamlik

Dr/Mnr/Me .....

Geteken te ..... Op ..... 2000.

....

Navorser Getuie

Verklaring deur tolk

Ek, _____ verklaar dat:

1. Ek die dokument vertaal het van Afrikaans/Engels na 'n taal wat die pasiënt en/of sy verteenwoordiger verstaan het. Ek het ook al die vrae wat deur die pasiënt of sy verteenwoordiger gevra is, en die antwoorde van die narvorsers vertaal.
2. Die inligting wat ek gegee het was 'n korrekte weergawe van wat ek vertel was.

Handtekening _____in _____ op_____

_____ _____

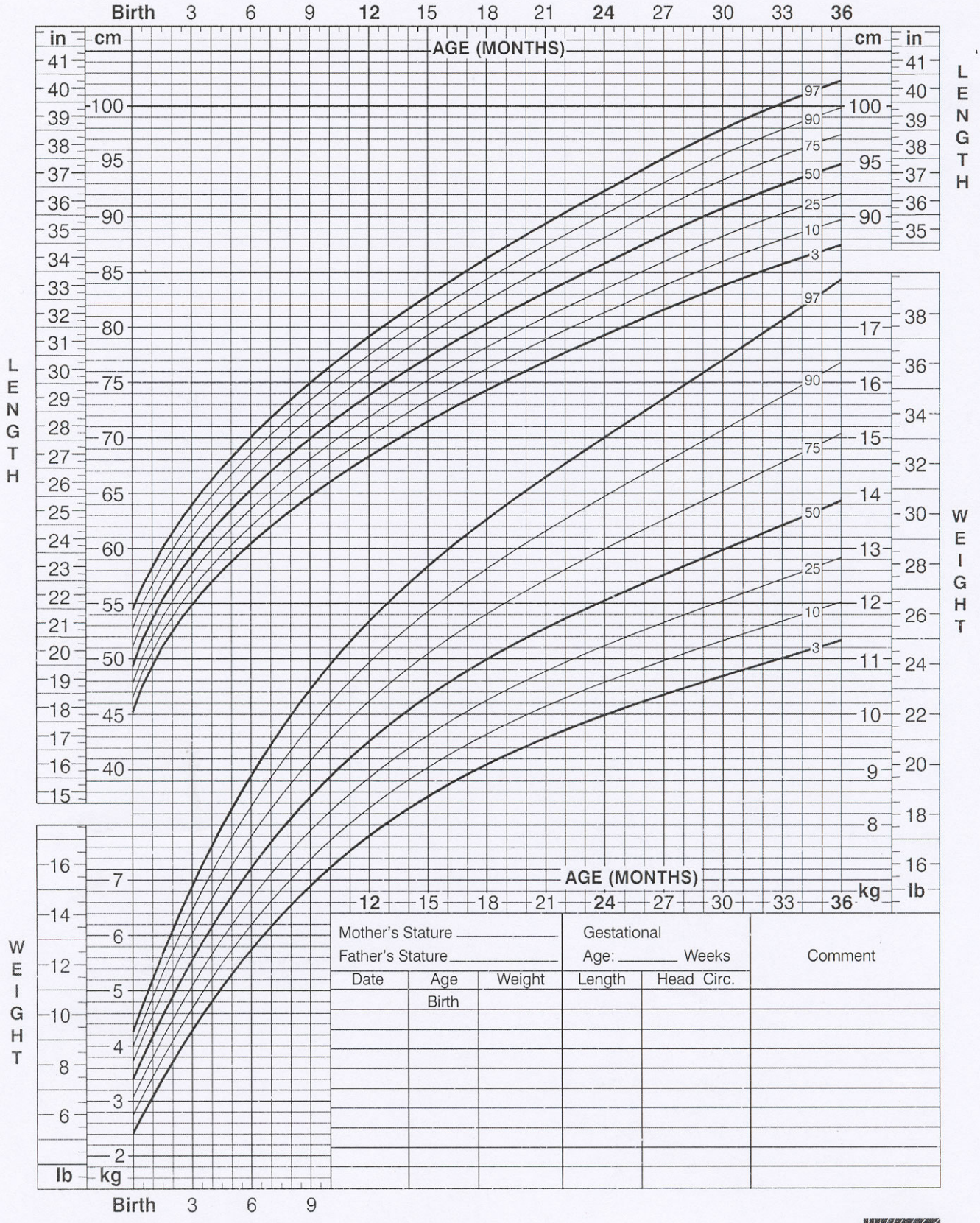
Tolk Getuie

Appendix 12

Birth to 36 months: Girls
Length-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Mother's Stature _____ Father's Stature _____			Gestational Age: _____ Weeks		Comment
Date	Age	Weight	Length	Head Circ.	
	Birth				

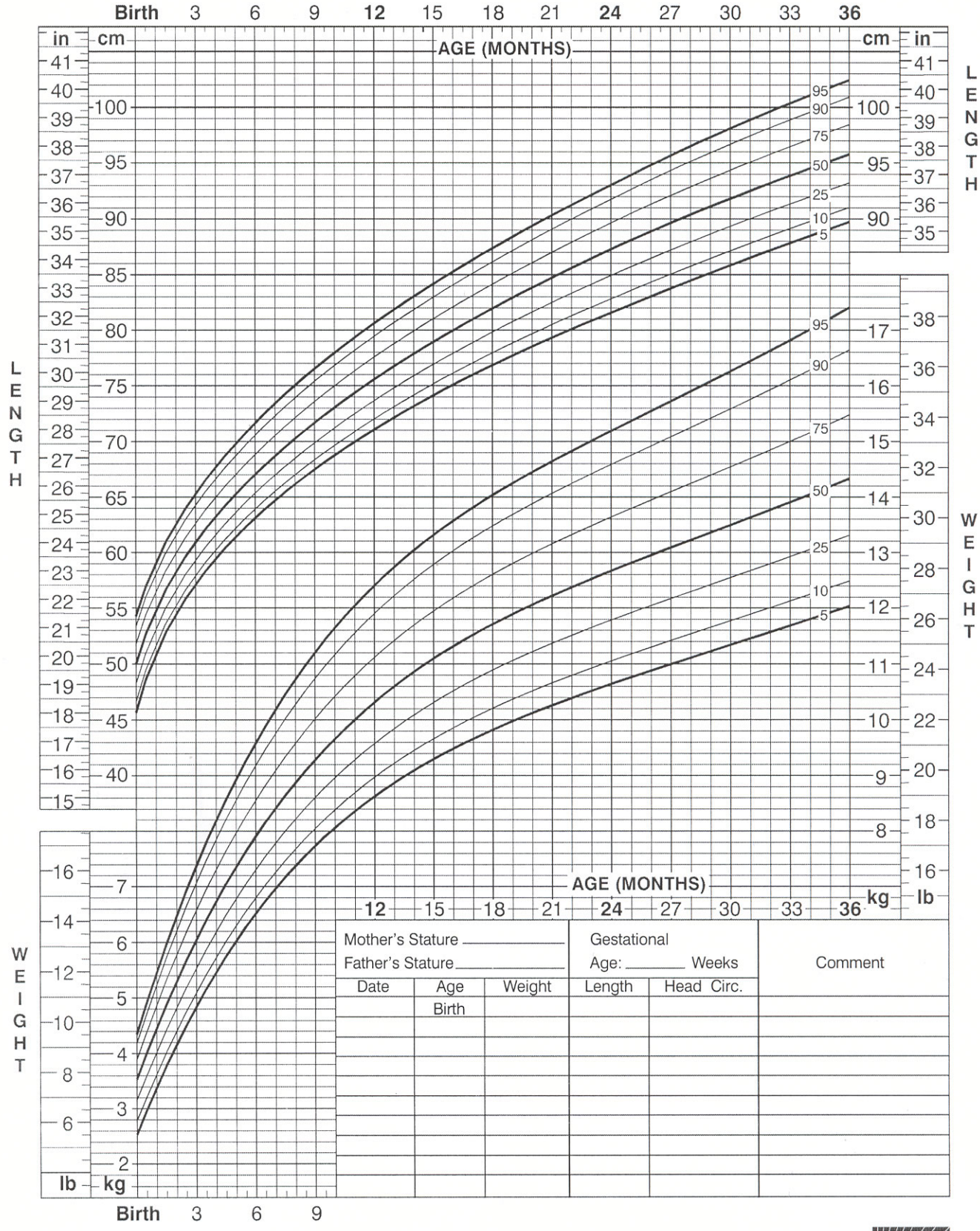
Published May 30, 2000 (modified 4/20/01).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

CDC
SAFER · HEALTHIER · PEOPLE™

Birth to 36 months: Boys
Length-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 4/20/01).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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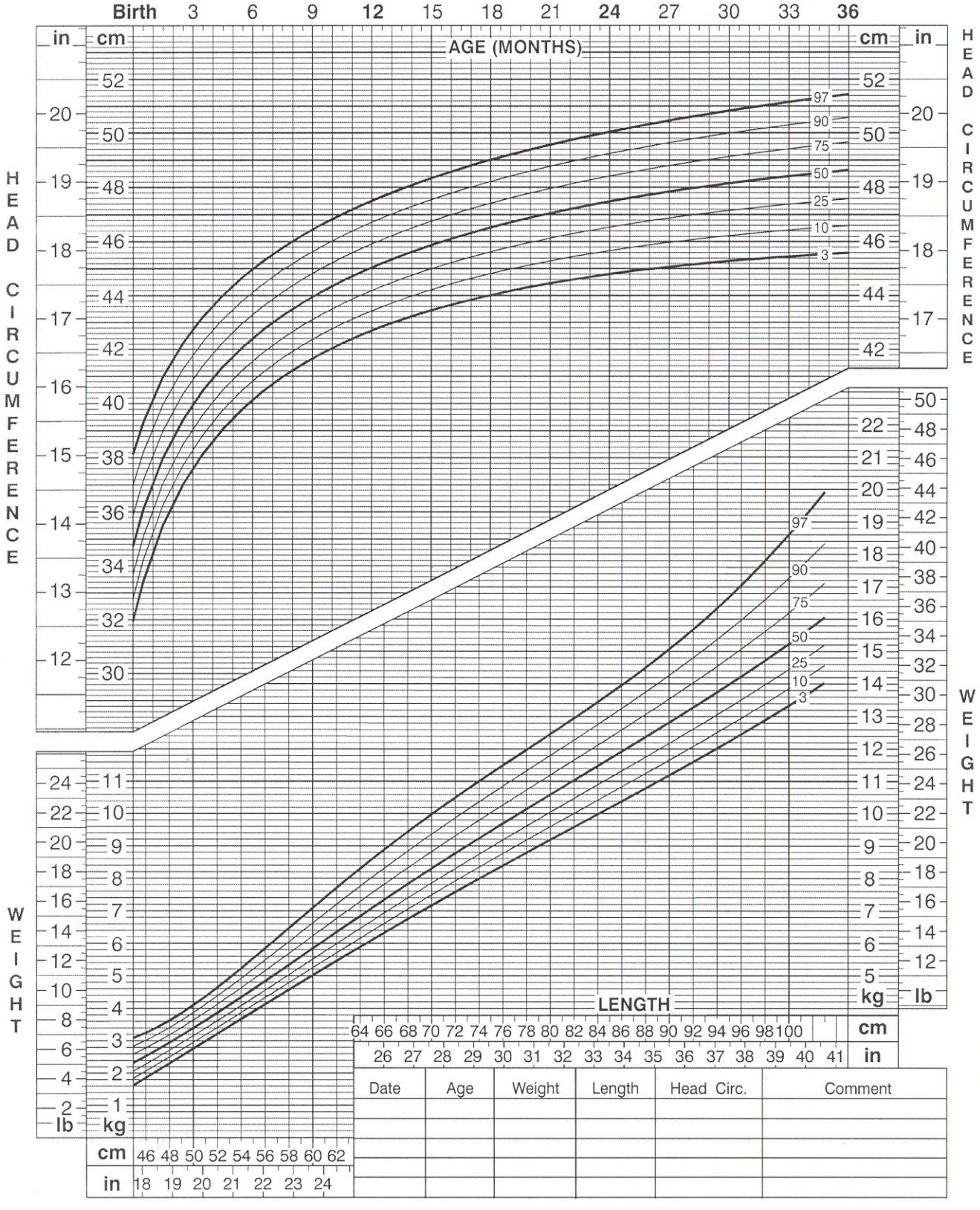
Appendix 13

Birth to 36 months: Girls
Head circumference-for-age and
Weight-for-length percentiles

NAME _____

RECORD # _____



Date	Age	Weight	Length	Head Circ.	Comment

Published May 30, 2000 (modified 10/16/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

CDC
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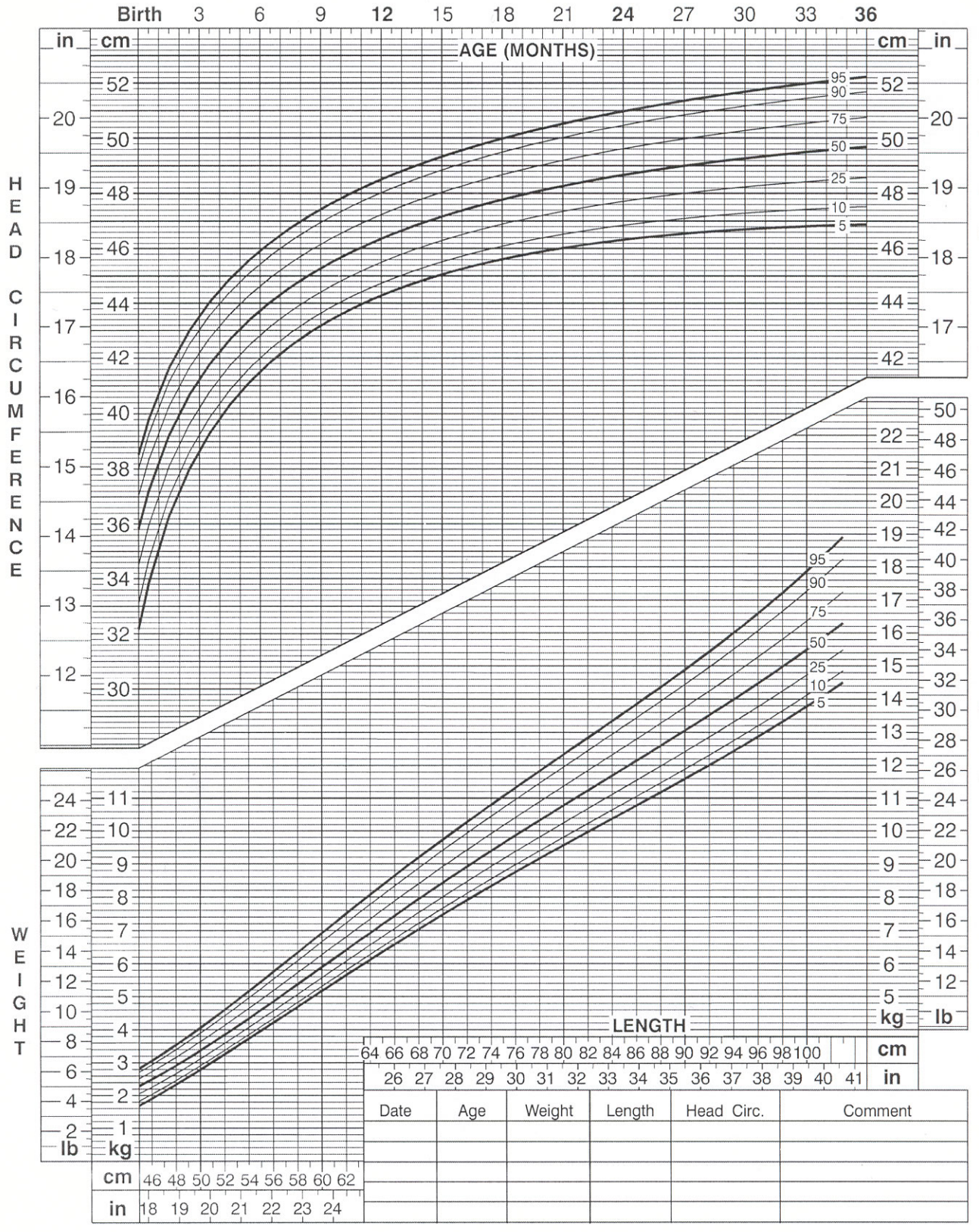
Birth to 36 months: Boys
Head circumference-for-age and
Weight-for-length percentiles

NAME _____

RECORD # _____



Date	Age	Weight	Length	Head Circ.	Comment

Published May 30, 2000 (modified 10/16/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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Appendix 14

Unmatched individual's parity and gestational age at recruitment

Individual's number	Parity	Gestational age
1	0	24
6	2	7
7	2	15
10	1	10
12	0	9
17	3	8
20	0	10
26	1	22
27	5	14
28	2	10
33	2	21
35 TERMINATION	0	9
36	1	16
38	1	19
41	1	9
43	1	22
45	0	20

Appendix 15

RESULTS

1. Sample

From a population of 47 individuals, 15 matched pairs of subjects and controls were made. The individuals were matched according to their race, gestational age at the onset of their participation in the study, and their parity. As shown in *Table 1,* it was only necessary to match the individuals according to gestational age and parity, since they were all of the mixed ancestry population.

Table 1: Matched pairs of subjects and controls according to their gestational age and parity.

SUBJECT NUMBER	Parity	Gestational age	CONTROL NUMBER	Parity	Gestational age
3	0	16	5	0	15
4	0	21	42	0	21
8	0	18	30	0	18
15	0	18	37	0	18
16	0	19	11	0	19
21	1	8	2	1	12
22	1	12	9	1	14
23	1	21	14	1	18
24	2	19	18	2	20
25	1	20	40	1	18
31	0	24	19	0	21
34	1	19	29	1	18
39	0	19	44	0	17
46	0	18	32	0	19
47	3	20	13	3	20

Results were derived from the subject and control populations (n=15) and the total population (n=47).

According to the title of the thesis, the aspects which influence the nutritional status of pregnant women will be analysed first, namely the dietary intake, anthropometry, biochemical analysis, and questionnaire information which covers socio-demographics, health history and lifestyle factors that could affect the mother's nutritional status and obstetric health. The results regarding the outcome of the pregnancy and the possible effect of alcohol on pregnancy outcome will be discussed thereafter.

2. Questionnaire

See *Appendix 3* for the contents of the questionnaire which 46 of the total population completed. Here follows the breakdown of all the relevant data.

2.1 Socio-demographics

- 100% of the individuals are of the Mixed Ancestry race (n=47).

- Ages of individuals at the onset of their participation in the study:

Table 2: Values regarding age of the different population groups.

	Subjects (n=15)	Controls (n=15)	Total (n=46)
Mean	24.93	25.07	25.23
Standard Deviation	4.43	6.70	5.89
Minimum - Maximum	19.00-33.00	18.00-40.00	18.00-40.00
Median	26.00	24.00	24.00
Interquartile range	21.00-28.00	19.00-30.00	20.00-29.00

- Living conditions: the amount of people who live in the house of the individual who took part in the study.

Table 3: Values regarding number of people per house of the different population groups.

	Subjects (n=15)	Controls (n=15)	Total (n=46)
Mean	5.80	5.40	5.72
Standard Deviation	3.14	3.68	3.51
Minimum - Maximum	2.00-13.00	2.00-15.00	2.00-16.00
Median	5.00	5.00	5.00
Interquartile range	3.00-8.00	3.00-7.00	3.00-7.00

- Living conditions: the average living space of the individuals who took part in the study. It is derived from the total number of people staying in the house compared to the number of bedrooms in the house, i.e. number of people/ number of bedrooms.

Table 4: Values regarding the number of people per bedroom of the different population groups.

	Subjects (n=15)	Controls (n=15)	Total (n=46)
Mean	3.80	3.07	3.28
Standard Deviation	2.70	1.75	1.96
Minimum - Maximum	1.00-13.00	1.00-7.00	1.00-13.00
Median	3.00	3.00	3.00
Interquartile range	3.00-4.00	2.00-4.00	2.00-4.00

- How many women were working during their pregnancy? Of the total population (n=46), 21 were working, i.e. **45.65%**. Of the subject population (n=15) 33.33% were working and 46.67% of the control population (n=15). The income of the women are described as follows:

Table 5: Values regarding the income of individuals of the different population groups.

In Rands:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Mean	474.00	1094.57	643.87
Standard Deviation	966.60	1379.92	1049.63
Minimum- maximum range	0.00-3520.00	0.00-4000.00	0.00-4000.00
Median	0.00	488.00	0.00
Interquartile range	0.00-600.00	0.00-1496.00	0.00-900.00

There was no statistical significance to the above socio-demographic data.

2.2 Health history

- Chronic illnesses: **82.61%** of the total population (n=46) has never had any chronic illnesses. Of the subject population (n=15), four individuals had problems with asthma, hypertension, or T.B. Of the control population (n=15), one individual had asthma and anaemia.

- Acute illnesses: **84.78%** of the total population (n=46) hasn't had any serious acute illnesses. Of the subject population (n=15), two individuals had problems with German Measles or idiopathic vaginal bleeding. Of the control population (n=15), two individuals had problems with meningitis or pneumonia.

- Hospitalisation: Of the total population (n=46) **28.26%** reported to have been hospitalised for an undetermined period of time. Of the subject population (n=15), five have hopitalised compared to four of the control population (n=15).

There is no statistical significance of the data compared between the subject and control populations according to the Fischer's Exact test.

2.3 Obstetric history

- Parity: That refers to the amount of successful pregnancies the individual has had, i.e. how many children does she have, excluding the one she's bearing in the present pregnancy. Of the total population (n=46), **45.64%** was expecting their first child at the time of the study. The parity of the remaining women ranged from one to five. Within the different populations, the incidence of the different parities were as follows:

Table 6: The incidence of different parities of the population groups.

Parity:	0	1	2	3	5
Subjects (n=15)	9	4	1	1	0
Controls (n=15)	8	5	1	1	0
Total (n=46)	21	15	6	3	1

There was no statistical significance to the different incidences of parities between the subject and control populations according to the Fischer's Exact test.

The researcher wanted to determine what the age distribution of the individuals were in terms of their parity, and if there was a possible trend.

Table 7: The distribution of age compared with the individual's parity.

Age (in years):	PARITY (n=46)				
	0	1	2	3	5
18-19	8	2	0	0	0
20-24	9	4	0	0	0
25-29	2	5	3	2	0
30-34	2	4	2	0	0
35-39	0	0	1	0	1
40	0	0	0	1	0

2.4 Lifestyle

- Exercise: Individuals were asked how many took part in an organized exercise routine before and during their pregnancy. The results were:

Table 8: Incidence of individuals exercising of the different population groups.

	Subjects (n=15)	Controls (n=15)	Total (n=46)
Yes	2	1	3
No	13	14	43

There was no statistical significance between the number of individuals who exercise between the subject and control populations according to the Fischer's Exact test.

- Supplementation: It was deemed necessary to ask who took any form of vitamin, mineral or a combined supplement during pregnancy, since it may have an affect on the individual's nutritional status.[63] The results were as following:

Table 9: The incidence of different supplement taking of individuals in the population groups.

Supplement:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Vitamin	1	1	3
Mineral	2	4	9
Combined vitamin-mineral	0	2	5
Vitamin and Mineral	0	1	1
None	12	7	28

There is no statistical significance to the difference in incidence of supplement taking between the subject and control populations according to the Fischer's Exact test.

- Smoking: As seen in **1.4**, smoking has a significant effect on the pregnancy outcome. Also, a large number of women admitted to smoking during pregnancy in another study conducted in the same area among the same population group. It is quite shocking to see a similar high incidence of smoking during pregnancy in this study:

Table 10: The incidence of smoking and the amount smoked per day of individuals of the population groups.

Number of cigarettes/day:	Subjects (n=15)	Controls (n=15)	Total (n=46)
1-4	6	4	15
5-10	7	1	17
Non-smoker	2	10	15

There was no statistical significance to the difference of number of individuals smoking between the subject and control populations according to the Fischer's Exact test.

2.5 Food security

- Facilities: The availability of certain facilities as specified below, influence the household's ability to safely store, prepare and consume food. The availability of these facilities were as follows:

Table 11: The incidence of facilities among the different population groups.

Facilities	Subjects (n=15)		Controls (n=15)		Total (n=46)	
	Yes	No	Yes	No	Yes	No
Running water	11	4	14	1	37	9
Electricity	13	2	15	0	41	5
Stove (electrical or gas)	14	1	15	0	45	1
Fridge	11	4	15	0	38	8
Toilet	11	4	10	5	33	13

There is no statistical significant difference between the incidences of facilities between the subject and control populations according to the Fischer's Exact test.

The researcher wanted to determine whether there was a connection between unemployment and the availability of facilities. The following table will only show whether the individuals were employed or not, and will not go into the specific income of the individuals.

Table 12: The availability of facilities in the individual's household compared with employment.

Facilities:		Employed?	
		"Yes"	"No"
Running water	"Yes"	18	19
	"No"	3	6
Electricity	"Yes"	19	22
	"No"	2	3
Stove	"Yes"	21	24
	"No"	0	1
Fridge	"Yes"	17	21
	"No"	4	4
Toilet	"Yes"	16	17
	"No"	5	8

- Food security: Through a questionnaire (*see Appendix 3, section E.2*), it was possible to determine the individual's food security status. Results were as follows:

Table 13: The comparison of different food security statuses of individuals in the population groups.

Food security status:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Food secure	7	14	32
Food insecure	4	0	8
Food insecure with hunger evident	3	1	5
Food insecure with severe hunger evident	1	0	1

There is a statistical significant difference of the incidences of the different food security statuses between the subject and control populations according to the Fischer's Exact test ($p<0.0233$) and the Chi-square test ($p<0.0396$).

The researcher wanted to determine whether there was a trend between the individual's food security status and employment. The following table will only concentrate on whether the individual is employed or not and not how much her income was.

Table 14: The comparison of individual's food security status and employment.

Food security status:	Employed?	
	"Yes"	"No"
Food secure	16	15
Food insecure	4	4
Food insecure with hunger evident	1	4
Food insecure with severe hunger evident	0	1

Furthermore, the researcher wanted to determine whether there was any connection between an individual's food security status and her BMI classification. The BMI classification below indicates undernourished (BMI < 18.5), normally nourished (BMI 18.5 – 25) and over nourished (BMI > 25).

Table 15: The comparison of food security status with nutritional status according to BMI.

Food security status (n=46):	BMI classifications:		
	< 18.5	18.5 - 25	> 25
Food secure	2	24	6
Food insecure	2	4	2
Food insecure with hunger evident	0	1	4
Food insecure with severe hunger evident	1	0	0

There was no statistical significance to these comparisons.

2.6 Eating habits influenced by pregnancy

- Quantity of food intake changed: Individuals were asked whether they thought they consumed more or less on average during their pregnancy, compared to before their pregnancy. The results were as follows:

Table 16: Incidence of changes of quantity eaten by individuals in the population groups.

Quantity changes:	Subjects (n=15)	Controls (n=14)	Total (n=45)
Eating more	7	7	21
Eating less	7	2	16
No change	1	5	8

There was no statistical significance to the difference in food intake quantity changes between the subject and control populations according to the Fischer's Exact test.

- Gastro-intestinal symptoms: The individuals were asked if they experienced any gastro-intestinal symptoms regularly that could influence their food intake. (Since a couple of individuals experienced more than one type of gastro-intestinal symptom, the symptoms will be expressed as percentage of the total of individuals per group.) The results were as follows:

Table 17: The incidence of gastro-intestinal symptoms among individuals of the population groups.

GI symptoms as %:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Anorexia	0.00	0.00	2.17
Nausea	60.00	40.00	47.83
Vomiting	13.33	26.67	19.57
Diarrrhoea	6.67	6.67	4.35
Constipation	33.33	53.33	47.83
Heartburn	93.33	26.67	65.22
No symptoms	0.00	6.67	2.17

There is a statistical significant difference of the incidences of gastro-intestinal symptoms between the subject and control populations according to the Fischer's Exact test ($p<0.0019$) and the Chi-square test ($p<0.04$).

- Skipping meals: The individuals were asked if they skipped meals on a regular basis since they've been pregnant. Since a couple of individuals skipped more than one type of meal on a regular basis during pregnancy, the meal-type skipped will be expressed as a percentage of the total number of people per group; and will thus not necessarily add up to the sum of people in a group. The results were as follows:

Table 18: The incidence of meal skipping among individuals of the population groups.

Meals skipped as %:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Breakfast	6.67	26.67	17.39
Lunch	26.67	0.00	15.22
Supper	6.67	0.00	10.87
None skipped	60.00	73.33	63.04

There was no statistical significance to the difference of meal skipping between the subject and control populations according to the Fischer's Exact test.

- Avoiding certain foods: The individuals were asked if they avoided certain foods since they've been pregnant, and the reason for them avoiding it. Here follows the reasons for avoiding:

Table 19: The incidence of avoiding certain foods among individuals of the population groups.

Reasons for avoiding certain foods:	Subjects (n=15)	Controls (n=15)	Total (n=46)
Dislike	12	6	26
Allergy	0	0	0
Religious reasons	0	0	0
Doctor's advice	0	1	1
No avoiding	3	8	19

There is no statistical significant difference to the incidence of avoiding certain foods between the subject and control populations according to the Fischer's Exact test.

The one individual whose doctor advised her to avoid certain foods specified soft cheeses and soft boiled eggs. A list follows to describe the foods that individuals disliked during their pregnancy in order of most reported to least:

- spicy foods (curry, biryani),
- meat (pork, chicken, beef),
- fish,
- vegetables,

- coffee, tea,
- fatty foods,
- porridge,
- spaghetti, macaroni and cheese,
- bread,
- peanutbutter, and
- eggs.

• Craving certain foods: Individuals were asked if they started craving certain foods or non-food items (pica) during their pregnancy. None of the individuals indicated that they craved any non-food items. The number of individuals who developing food cravings during pregnancy are as follows:

Table 20: The incidence of food cravings among individuals of the population groups.

	Subjects (n=15)	Controls (n=15)	Total (n=46)
Developed cravings	14	13	40
No cravings	1	2	6

There is no statistical significance between the differences of individuals craving certain foods or not between the subject and control populations according to the Fischer's Exact test.

A list follows to describe the foods that was craved by the women in the study in order of most reported to least:

- vegetables, salad,
- chocolate, sweets,
- yoghurt, milk,
- fruit,
- spaghetti, macaroni and cheese,
- meat (chicken, beef)
- curry, stews, biryani,
- pies, pizza,
- seafood, fish,
- ice-cream,
- eggs,

- potato chips,
- dressing, mayonnaise,
- cake, biscuits,
- offal, and
- soup.

3. Dietary intake

Dietary intake during pregnancy was determined by two types of dietary questionnaires, namely the 24 hour recall and food frequency questionnaire (FFQ). The time when the questionnaires were done and the period of time which these questionnaires covered, were discussed in **2.5.1**.

Ideally, three 24 hour recalls and two FFQ's were completed with each individual. Of the subject population (n=15), all completed the first two 24 hour recalls, and fourteen completed the third. All individuals of the control population completed all three 24 hour recalls. All subjects and controls (n=30) completed both FFQ's.

As shown in *Table 22*, the mean, standard deviation, median and the interquartile range (q1-q3) were calculated of the specified nutrients for the subject population, the control population and the total population. The non-parametric sign test was used to determine if there was any significant difference between the subject and control populations' nutrients' mean values. It was found that there was a significant difference with vitamin D according to FFQ 1, and also for selenium according to FFQ 2.

The mean, standard deviation, median and the interquartile range (q1-q3) were also calculated of the specified nutrients for the average of the three 24 hour recalls and the two FFQ's of the subject, control and total population. No significant difference was found however.

Please note since everyone did not complete all three 24 hour recalls and the two food frequency questionnaires, the n values may differ with each questionnaire, as shown in *Table 21*:

Table 21: n values of different dietary questionnaires of the different population groups.

Dietary questionnaires:	Subjects	Controls	Total
24 hour recall no. 1	15	15	47
24 hour recall no. 2	15	15	46
24 hour recall no. 3	14	15	42
24 hour recalls combined	14	15	42
FFQ no. 1	15	15	46
FFQ no. 2	15	15	43
FFQ's combined	15	15	43

Table 22: The mean, standard deviation, median and interquartile range (q1-q3) values of the specified nutrients according to the individual and averages of three 24 hour recalls and two FFQ's for the subject, control and total populations.

Variable	Subjects		Controls		All	
Energy (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	103.14 (45.40)	106.77 (75.79-140.81)	82.72 (24.28)	85.28 (72.70-93.31)	92.19 (35.54)	90.60 (68.35-112.81)
24 hr no2	92.55 (37.36)	104.38 (69.82-123.20)	88.08 (34.85)	80.70 (66.00-101.50)	88.85 (34.58)	81.96 (67.86-119.47)
24 hr no 3	107.18 (52.91)	94.64 (70.59-129.90)	101.29 (44.68)	97.39 (68.21-129.26)	100.13 (45.64)	95.88 (68.21-128.30)
Combined	101.58 (39.02)	90.52 (69.23-128.83)	90.69 (30.58)	93.90 (70.31-101.54)	102.59 (66.78)	92.27 (71.04-109.52)
FFQ no 1	173.47 (80.36)	170.00 (121.34-194.77)	147.63 (32.41)	149.90 (113.45-166.11)	158.34 (52.26)	152.44 (129.91-172.27)
FFQ no 2	165.64 (116.25)	121.12 (107.51-190.40)	118.07 (26.67)	112.16 (98.20-141.88)	136.95 (75.71)	115.55 (99.64-144.40)
Combined	169.55 (94.58)	146.69 (113.85-206.90)	132.85 (22.28)	127.67 (118.33-152.10)	161.57 (113.46)	132.63 (113.85-164.67)

Variable	Subjects		Controls		All	
Protein (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	143.57 (62.27)	158.80 (97.50-182.50)	114.78 (33.58)	117.30 (101.80-127.70)	129.89 (52.13)	120.20 (94.40-168.30)
24 hr no2	124.09 (37.82)	121.30 (112.50-143.10)	140.81 (51.80)	134.10 (111.70-173.10)	133.03 (57.30)	121.12 (97.00-165.70)
24 hr no 3	147.05 (83.16)	133.25 (91.60-196.10)	155.83 (71.55)	142.40 (114.70-196.20)	146.24 (73.05)	136.00 (82.20-194.30)
Combined	140.01 (45.29)	135.32 (107.80-160.97)	137.14 (43.25)	133.70 (111.63-158.00)	137.35 (42.58)	137.47 (111.62-159.72)
FFQ no 1	208.27 (86.55)	191.70 (168.00-253.20)	182.53 (32.06)	175.60 (159.20-200.00)	191.17 (58.44)	187.70 (159.20-217.40)
FFQ no 2	190.30 (132.33)	158.00 (124.70-228.40)	144.71 (35.17)	129.20 (115.50-175.30)	151.07 (91.41)	133.50 (115.50-166.20)
Combined	199.29 (103.17)	177.05 (139.95-219.95)	163.62 (28.54)	163.70 (139.55-175.45)	167.47 (70.66)	161.04 (134.05-195.05)

Variable	Subjects		Controls		All	
Vitamin A (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	57.36 (50.38)	49.90 (26.80-73.90)	104.51 (117.86)	58.70 (32.80-112.00)	76.49 (78.32)	56.00 (30.00-86.40)
24 hr no2	83.29 (143.20)	43.10 (32.90-87.40)	80.50 (54.69)	63.20 (55.50-95.50)	77.21 (92.79)	57.20 (33.30-82.90)
24 hr no 3	150.96 (231.62)	49.20 (34.80-128.50)	94.65 (107.23)	67.90 (30.30-124.10)	102.92 (150.91)	55.05 (34.40-96.40)
Combined	98.30 (99.85)	50.88 (32.00-113.03)	93.22 (67.63)	62.77 (50.50-105.33)	85.00 (72.08)	60.91 (43.40-93.20)
FFQ no 1	319.00 (357.54)	220.10 (115.00-316.10)	204.48 (120.26)	173.10 (97.10-312.80)	250.37 (246.03)	186.55 (110.90-272.30)
FFQ no 2	193.80 (138.46)	141.60 (90.60-227.20)	192.44 (131.83)	138.20 (83.40-265.80)	165.94 (131.29)	104.30 (81.90-227.20)
Combined	256.40 (216.54)	191.55 (136.55-269.75)	198.46 (112.65)	166.80 (94.10-302.40)	203.72 (158.17)	151.72 (96.05-269.75)

Variable	Subjects		Controls		All	
Vitamin D (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	46.26 (51.67)	34.90 (19.30-62.00)	25.80 (24.64)	20.90 (9.30-35.10)	46.49 (58.19)	28.00 (14.60-57.30)
24 hr no2	43.97 (37.12)	23.10 (10.10-81.80)	34.57 (24.26)	28.80 (19.80-43.60)	36.85 (32.07)	27.85 (15.00-43.60)
24 hr no 3	47.54 (46.54)	29.15 (16.80-95.70)	42.41 (29.12)	33.40 (17.10-69.30)	45.99 (37.32)	32.90 (16.80-69.30)
Combined	46.14 (37.17)	35.53 (20.27-48.43)	34.26 (16.22)	31.37 (24.37-46.70)	41.25 (27.22)	34.83 (24.37-48.73)
FFQ no 1	93.33* (44.473)	96.50 (64.20-110.00)	53.33* (22.31)	45.30 (35.60-77.90)	80.62 (47.83)	72.25 (44.40-102.00)
FFQ no 2	78.15 (44.69)	83.50 (40.40-108.50)	52.37 (30.59)	43.40 (33.50-69.20)	61.17 (39.19)	60.00 (34.20-83.50)
Combined	85.74 (40.71)	85.75 (45.10-106.20)	52.84 (20.71)	53.10 (31.80-74.20)	69.38 (36.28)	69.78 (40.59-94.09)

* Significant difference between subject and control population; $p < 0.001$ (determined through non-parametric sign test).

Variable	Subjects		Controls		All	
Vitamin E (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	85.12 (49.47)	84.50 (25.30-119.05)	83.69 (59.55)	76.20 (31.90-106.90)	97.28 (66.72)	84.50 (45.90-125.00)
24 hr no2	77.09 (50.81)	80.40 (39.90-110.40)	106.08 (126.22)	71.10 (41.80-134.50)	98.10 (88.93)	75.75 (41.80-134.50)
24 hr no 3	91.27 (44.21)	88.85 (51.80-121.80)	135.81 (138.21)	100.90 (56.90-168.10)	114.65 (95.41)	88.85 (56.90-154.60)
Combined	84.95 (32.15)	88.68 (53.93-112.27)	108.53 (98.27)	82.73 (67.10-114.73)	102.59 (67.33)	91.56 (66.53-119.37)
FFQ no 1	217.16 (91.16)	208.30 (142.10-327.20)	162.21 (59.75)	164.80 (100.80-208.50)	189.58 (71.89)	184.25 (142.10-216.40)
FFQ no 2	180.85 (140.09)	131.20 (102.90-201.60)	147.63 (93.64)	121.50 (72.70-178.80)	141.33 (105.70)	117.40 (82.10-161.10)
Combined	199.01 (100.89)	169.75 (130.20-244.85)	154.92 (66.14)	139.80 (94.05-201.65)	161.94 (77.18)	154.73 (114.25-193.68)

Variable	Subjects		Controls		All	
Vitamin K (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	78.11 (74.55)	48.20 (30.50-112.60)	51.74 (53.29)	38.00 (18.00-68.70)	68.62 (63.78)	40.80 (26.40-94.90)
24 hr no2	54.42 (73.74)	23.20 (17.30-59.80)	90.31 (75.64)	60.70 (28.00-155.20)	72.12 (78.59)	41.25 (17.80-101.50)
24 hr no 3	78.30 (85.69)	42.00 (28.90-93.80)	63.33 (72.93)	39.50 (24.90-64.90)	66.12 (70.61)	40.75 (26.90-71.60)
Combined	72.81 (48.36)	45.80 (34.63-115.83)	68.46 (38.86)	62.73 (35.17-102.77)	68.92 (42.45)	52.21 (34.74-107.11)
FFQ no 1	109.12 (43.89)	101.90 (77.80-144.90)	172.02 (159.74)	128.60 (97.00-174.40)	140.55 (101.49)	128.45 (88.70-171.80)
FFQ no 2	121.07 (87.65)	104.50 (70.80-132.20)	111.31 (74.51)	86.50 (63.50-126.40)	104.63 (74.18)	89.30 (65.90-122.60)
Combined	115.09 (58.99)	89.25-119.80)	141.66 (112.83)	106.85 (89.80-130.95)	119.98 (77.50)	103.40 (85.88-130.95)

Variable	Subjects		Controls		All	
Vitamin C (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	223.57 (360.30)	71.40 (37.60-201.60)	243.24 (331.76)	88.90 (45.00-349.00)	178.27 (282.68)	78.70 (37.60-193.00)
24 hr no2	210.17 (411.70)	75.80 (19.5-193.10)	194.22 (143.07)	202.90 (73.00-282.50)	182.08 (264.24)	95.20 (23.60-210.20)
24 hr no 3	138.69 (173.63)	71.55 (31.20-157.90)	182.70 (240.57)	103.60 (58.30-169.80)	149.28 (192.11)	76.10 (40.40-169.80)
Combined	183.87 (269.77)	75.40 (44.87-226.37)	206.72 (169.94)	148.93 (79.50-351.63)	166.77 (194.09)	105.47 (48.89-207.97)
FFQ no 1	426.79 (342.90)	304.10 (138.90-549.80)	389.99 (302.01)	303.10 (166.60-495.00)	363.71 (276.55)	282.40 (166.60-457.60)
FFQ no 2	301.19 (260.73)	199.80 (95.80-373.90)	397.89 (542.32)	238.30 (168.30-367.90)	280.73 (359.37)	189.90 (105.50-348.00)
Combined	363.99 (268.97)	234.95 (127.60-609.30)	393.94 (311.30)	272.80 (159.65-511.30)	315.39 (253.95)	229.15 (135.82-415.77)

Variable	Subjects		Controls		All	
Thiamin (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	84.09 (53.77)	74.10 (51.90-109.30)	71.83 (37.07)	64.20 (46.60-92.00)	72.05 (41.82)	60.30 (46.60-94.80)
24 hr no2	62.49 (40.22)	56.00 (43.20-63.00)	72.93 (32.89)	65.20 (52.30-90.80)	61.98 (34.37)	54.90 (39.30-77.60)
24 hr no 3	77.54 (58.55)	57.80 (41.80-88.10)	85.79 (48.18)	67.50 (43.60-116.60)	74.38 (47.61)	61.00 (41.80-99.00)
Combined	73.39 (41.91)	59.48 (45.17-88.77)	76.85 (32.69)	69.93 (56.27-92.17)	69.53 (33.20)	63.55 (46.87-77.40)
FFQ no 1	130.28 (67.82)	120.50 (87.70-145.50)	121.70 (31.73)	115.30 (104.10-126.20)	123.76 (48.22)	115.45 (95.80-126.20)
FFQ no 2	118.13 (74.28)	76.20 (67.10-152.60)	105.81 (38.78)	90.80 (74.90-136.60)	98.23 (56.66)	84.85 67.10-125.00)
Combined	124.20 (66.06)	98.15 (81.95-166.10)	113.75 (29.81)	106.80 (93.95-127.15)	108.60 (48.76)	98.10 (83.07-122.83)

Variable	Subjects		Controls		All	
Riboflavin (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	102.21 (107.76)	50.00 (44.20-156.30)	103.19 (101.72)	49.40 (43.40-114.00)	96.82 (91.66)	60.30 (43.30-116.50)
24 hr no2	71.25 (41.88)	64.30 (36.80-102.80)	94.59 (52.95)	91.10 (65.50-113.80)	76.15 (43.12)	69.60 (40.10-99.40)
24 hr no 3	85.59 (73.11)	60.90 (36.70-127.10)	98.16 (52.20)	100.70 (48.20-152.20)	87.95 (62.56)	66.70 (39.60-127.90)
Combined	87.98 (64.44)	70.18 (42.47-104.47)	98.65 (57.37)	84.40 (61.90-109.63)	88.94 (56.67)	79.13 (50.31-105.00)
FFQ no 1	196.39 (108.25)	166.40 (131.90-225.90)	180.82 (63.86)	162.70 (128.20-222.50)	184.46 (85.93)	163.55 (131.90-201.40)
FFQ no 2	172.26 (118.87)	127.90 (101.00-207.80)	138.97 (54.79)	127.80 (102.10-196.00)	138.96 (91.24)	123.70 (88.30-184.00)
Combined	184.33 (95.61)	164.30 (126.85-211.65)	159.90 (50.64)	156.85 (113.95-201.05)	158.25 (74.61)	135.56 (113.97-187.41)

Variable	Subjects		Controls		All	
Niacin (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	140.38 (70.36)	131.00 (89.00-177.20)	112.42 (41.88)	110.80 (93.50-143.80)	120.32 (55.56)	110.80 (88.70-162.90)
24 hr no2	97.81 (40.02)	93.20 (80.70-110.60)	130.55 (53.58)	124.80 (93.10-159.50)	111.29 (55.39)	100.15 (72.50-130.00)
24 hr no 3	149.27 (103.10)	120.20 (76.70-156.90)	143.79 (73.17)	125.20 (82.00-211.60)	135.88 (80.30)	125.20 (76.70-184.10)
Combined	130.51 (53.61)	112.37 (100.57-158.43)	128.92 (38.19)	134.37 (105.60-154.33)	123.76 (45.16)	115.18 (99.26-154.35)
FFQ no 1	173.35 (76.71)	175.70 (128.40-193.60)	170.80 (36.50)	178.40 (143.80-194.40)	166.07 (53.42)	169.30 (128.40-193.50)
FFQ no 2	156.89 (120.61)	111.40 (92.00-189.60)	126.70 (34.26)	109.30 (99.00-150.90)	126.23 (81.49)	109.10 (94.00-146.90)
Combined	165.12 (91.81)	139.75 (112.55-191.60)	148.76 (27.00)	143.95 (134.00-161.30)	143.04 (62.37)	134.47 (113.26-161.28)

Variable	Subjects		Controls		All	
Vitamin B6 (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	95.45 (68.72)	75.80 (61.20-133.00)	79.95 (36.51)	83.90 (60.80-99.70)	84.66 (55.25)	75.80 (44.00-101.40)
24 hr no2	54.56 (24.17)	50.30 (35.30-70.70)	67.85 (32.94)	62.20 (41.70-82.30)	59.46 (30.94)	53.30 (38.30-72.20)
24 hr no 3	89.61 (68.71)	65.60 (47.30-112.30)	90.36 (66.13)	67.20 (44.90-137.00)	82.09 (61.21)	62.55 (38.70-112.30)
Combined	81.63 (46.35)	70.02 (55.73-85.70)	79.39 (35.45)	76.70 (53.53-105.80)	75.36 (37.22)	71.23 (52.00-89.68)
FFQ no 1	131.73 (68.67)	112.10 (96.20-155.30)	119.87 (28.38)	111.40 (102.30-145.60)	124.35 (48.71)	111.70 (96.20-145.60)
FFQ no 2	117.77 (76.13)	86.40 (69.00-158.20)	101.46 (28.25)	99.70 (74.80-124.20)	95.70 (55.37)	83.65 (69.00-114.40)
Combined	124.75 (68.58)	92.75 (81.55-154.65)	110.67 (21.96)	108.80 (93.85-127.05)	107.68 (48.31)	95.16 (79.55-127.05)

Variable	Subjects		Controls		All	
Folate (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	68.44 (43.32)	63.00 (32.30-95.20)	63.95 (44.98)	51.00 (38.60-87.60)	61.90 (38.08)	52.40 (35.30-86.00)
24 hr no2	46.21 (25.14)	38.00 (30.40-65.90)	67.97 (43.29)	54.30 (40.90-84.40)	52.41 (35.49)	45.05 (29.50-69.20)
24 hr no 3	63.27 (46.42)	50.40 (28.40-81.70)	55.29 (21.63)	51.70 (42.60-71.50)	58.62 (34.96)	50.70 (33.50-81.00)
Combined	59.09 (27.32)	54.85 (45.17-65.97)	62.40 (30.53)	52.97 (43.97-74.97)	57.96 (27.22)	54.85 (43.97-69.05)
FFQ no 1	128.03 (74.64)	109.30 (82.10-145.70)	107.29 (41.04)	98.50 (82.80-112.20)	115.01 (51.69)	105.35 (82.80-138.20)
FFQ no 2	113.44 (81.63)	91.90 (70.60-126.00)	108.54 (87.27)	78.60 (63.10-100.70)	94.03 (73.94)	76.05 (57.10-100.70)
Combined	120.73 (69.71)	101.40 (85.80-119.30)	107.91 (50.80)	85.95 (79.65-113.25)	102.29 (53.55)	89.01 (76.88-113.25)

Variable	Subjects		Controls		All	
Vitamin B12 (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	235.23 (232.09)	178.00 (137.00-319.80)	146.88 (82.94)	125.90 (95.00-222.50)	190.09 (169.18)	147.60 (77.70-231.10)
24 hr no2	160.17 (97.31)	147.30 (79.10-217.10)	184.18 (111.61)	169.30 (123.60-278.10)	176.72 (109.50)	158.20 (84.50-237.70)
24 hr no 3	179.08 (122.95)	175.10 (107.60-218.20)	192.12 (99.93)	200.50 (130.10-265.50)	187.69 (115.01)	159.65 (107.60-265.50)
Combined	196.83 (123.37)	161.52 (108.73-268.60)	174.39 (75.27)	159.43 (129.27-229.23)	187.59 (94.39)	169.84 (124.50-232.73)
FFQ no 1	431.47 (194.48)	392.60 (311.11-590.10)	369.29 (205.56)	263.50 (217.00-460.90)	411.68 (208.85)	379.70 (229.60-532.50)
FFQ no 2	341.39 (237.87)	317.30 (222.10-403.30)	234.99 (80.53)	232.80 (181.70-270.70)	272.00 (185.31)	240.35 (172.60-336.60)
Combined	386.43 (193.17)	342.05 (243.35-464.55)	302.14 (121.51)	260.85 (214.10-358.85)	334.59 (161.77)	315.91 (216.98-423.64)

Variable	Subjects		Controls		All	
Calcium (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	56.33 (52.18)	37.40 (18.10-82.80)	52.38 (47.89)	35.60 (23.90-58.00)	48.64 (41.56)	37.40 (22.60-60.00)
24 hr no2	42.19 (34.03)	33.10 (16.50-58.60)	63.99 (37.17)	59.80 (34.10-88.40)	48.56 (32.09)	39.15 (27.20-62.90)
24 hr no 3	40.92 (30.58)	29.50 (20.10-42.90)	69.67 (51.11)	49.50 (36.90-108.90)	53.61 (46.47)	39.50 (22.40-68.80)
Combined	47.68 (32.90)	37.10 (22.80-62.90)	62.01 (42.47)	48.67 (29.37-72.63)	51.22 (34.55)	39.44 (29.38-62.90)
FFQ no 1	102.17 (73.42)	79.90 (55.90-115.40)	115.65 (34.56)	100.60 (89.70-146.30)	101.82 (48.83)	90.35 (75.00-119.80)
FFQ no 2	106.64 (98.12)	78.10 (49.70-128.20)	88.07 (33.96)	81.20 (64.70-102.50)	84.86 (64.97)	71.40 (49.70-103.00)
Combined	104.40 (81.31)	95.70 (62.00-108.80)	101.86 (30.25)	89.70 (81.15-115.20)	91.36 (53.26)	83.54 (62.75-106.47)

Variable	Subjects		Controls		All	
Phosphorous (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	98.86 (48.83)	104.30 (77.80-119.60)	87.75 (38.59)	78.50 (70.60-91.80)	91.35 (39.87)	83.90 (70.60-112.40)
24 hr no2	84.01 (32.86)	79.90 (61.10-106.20)	98.36 (36.60)	99.90 (74.60-110.00)	87.09 (34.87)	82.50 (63.80-108.20)
24 hr no 3	95.60 (59.14)	80.65 (68.30-109.80)	111.49 (52.66)	103.50 (61.00-160.30)	99.39 (54.67)	88.35 (61.00-120.00)
Combined	93.99 (38.93)	81.93 (64.17-106.97)	99.20 (38.89)	94.03 (72.07-116.80)	93.25 (36.44)	86.44 (67.63-106.97)
FFQ no 1	166.16 (89.12)	151.00 (113.20-197.30)	161.87 (35.00)	150.50 (141.60-187.80)	159.02 (56.79)	148.80 (131.80-180.00)
FFQ no 2	163.63 (135.44)	131.30 (94.40-203.80)	125.88 (40.62)	111.70 (94.30-155.00)	127.58 (90.16)	109.40 (90.60-150.80)
Combined	164.90 (107.87)	144.45 (104.90-178.65)	143.87 (33.36)	136.10 (120.65-155.65)	140.25 (70.28)	132.75 (105.17-155.67)

Variable	Subjects		Controls		All	
Magnesium (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	87.46 (37.88)	86.40 (70.40-114.80)	82.32 (33.23)	78.90 (68.20-91.00)	83.44 (34.25)	80.60 (65.60-106.70)
24 hr no2	76.22 (31.93)	77.20 (46.90-97.10)	85.87 (30.78)	84.10 (66.20-93.70)	76.68 (30.53)	76.50 (54.90-90.60)
24 hr no 3	89.45 (55.34)	72.85 (62.10-97.60)	91.69 (39.52)	85.00 (65.70-127.90)	87.18 (45.90)	77.15 (61.10-103.60)
Combined	85.33 (34.58)	76.38 (59.13-99.10)	86.63 (30.66)	84.83 (64.60-99.60)	82.95 (30.96)	79.14 (62.27-99.10)
FFQ no 1	156.41 (91.28)	144.80 (98.10-174.80)	153.14 (41.24)	151.70 (118.30-176.90)	147.61 (61.06)	142.20 (114.40-160.50)
FFQ no 2	157.67 (134.91)	110.10 (92.50-175.70)	121.21 (46.61)	103.60 (84.50-143.30)	122.32 (90.11)	102.55 (84.50-142.40)
Combined	157.04 (107.04)	126.45 (96.25-181.35)	137.18 (38.96)	133.05 (110.95-148.40)	132.09 (71.75)	124.27 (95.64-144.37)

Variable	Subjects		Controls		All	
Iron (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	41.48 (25.98)	34.60 (29.40-51.30)	35.56 (20.86)	31.30 (22.30-45.50)	35.68 (20.47)	31.30 (23.90-47.40)
24 hr no2	29.97 (12.25)	27.50 (19.80-39.40)	37.88 (21.30)	30.70 (26.20-49.50)	33.03 (16.07)	30.15 (23.00-38.80)
24 hr no 3	39.42 (27.09)	28.95 (20.90-49.50)	38.63 (16.20)	40.70 (27.90-49.50)	36.77 (19.35)	31.75 (22.50-47.50)
Combined	37.56 (19.33)	33.32 (24.13-46.23)	37.36 (15.48)	34.60 (29.60-43.47)	35.47 (15.25)	33.54 (24.97-40.70)
FFQ no 1	68.86 (34.99)	64.00 (50.90-80.60)	67.10 (32.84)	53.80 (50.00-73.70)	63.97 (28.40)	57.10 (50.00-70.20)
FFQ no 2	59.74 (36.09)	44.60 (36.00-71.60)	50.85 (22.91)	44.80 (36.30-60.90)	48.15 (28.92)	39.95 (35.10-55.10)
Combined	64.30 (32.88)	50.70 (44.90-76.10)	58.97 (22.39)	48.90 (44.50-66.20)	54.87 (25.66)	47.79 (41.00-65.60)

Variable	Subjects		Controls		All	
Zinc (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	71.37 (39.71)	64.60 (49.00-93.50)	65.45 (23.08)	64.60 (47.30-87.70)	66.92 (33.25)	60.40 (44.90-87.70)
24 hr no2	72.13 (33.98)	56.80 (43.30-101.60)	89.19 (44.99)	74.40 (58.40-127.70)	75.78 (36.34)	68.35 (50.10-98.80)
24 hr no 3	78.99 (48.53)	60.35 (50.80-111.60)	78.27 (35.83)	72.80 (48.80-104.20)	75.06 (39.98)	66.70 (48.10-104.20)
Combined	75.72 (32.55)	66.15 (53.00-89.40)	77.64 (24.01)	76.87 (61.50-89.60)	73.63 (25.61)	66.29 (56.87-82.54)
FFQ no 1	117.60 (53.62)	108.70 (87.00-144.00)	108.17 (29.88)	100.30 (87.40-128.00)	109.54 (37.32)	108.00 (87.40-128.00)
FFQ no 2	112.23 (86.45)	89.30 (70.70-125.80)	86.18 (22.48)	82.40 (67.90-100.90)	89.11 (57.68)	82.85 (68.00-100.30)
Combined	114.92 (66.17)	97.95 (80.00-127.10)	97.18 (22.72)	95.05 (78.25-114.55)	97.20 (44.46)	92.99 (75.79-113.85)

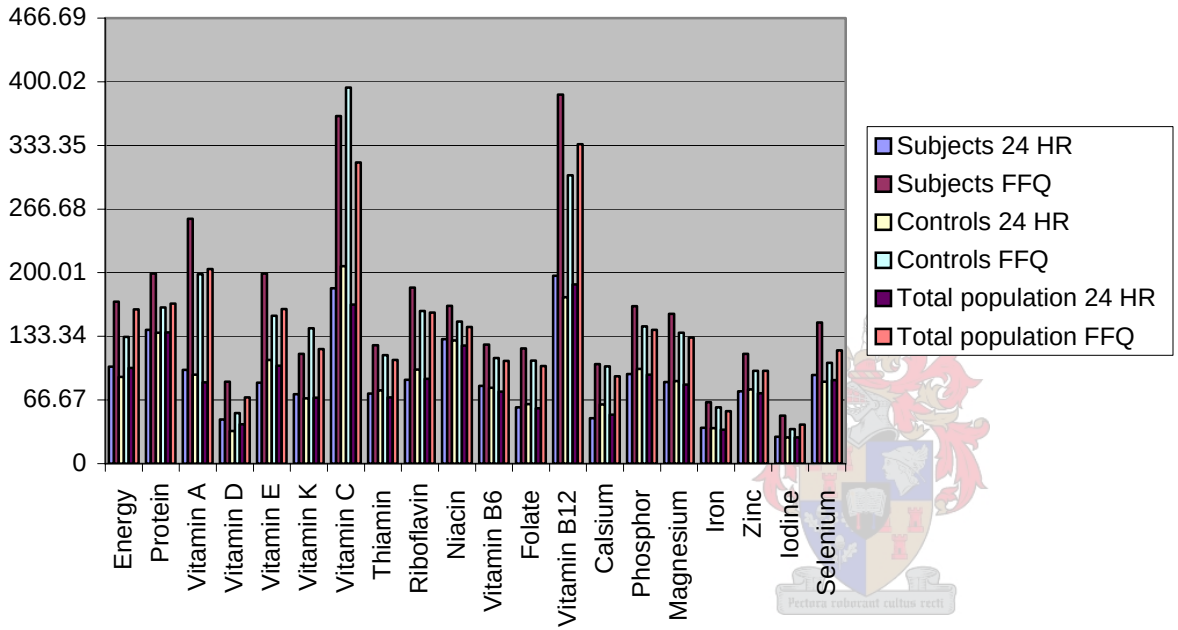
Variable	Subjects		Controls		All	
Iodine (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	27.64 (14.95)	28.60 (15.90-38.50)	24.49 (14.48)	22.40 (16.70-28.30)	31.06 (34.21)	23.40 (12.80-37.40)
24 hr no2	25.53 (13.28)	20.20 (16.10-38.90)	28.39 (16.83)	22.80 (16.00-46.30)	23.67 (14.43)	19.25 (14.70-32.80)
24 hr no 3	30.56 (21.45)	25.80 (14.60-48.30)	29.29 (19.90)	24.30 (14.30-47.00)	29.20 (18.84)	25.00 (14.60-44.10)
Combined	28.29 (11.49)	26.72 (21.97-34.13)	27.39 (13.03)	22.67 (20.20-38.67)	27.63 (14.81)	23.48 (19.57-34.16)
FFQ no 1	48.41 (26.67)	46.70 (24.20-58.00)	38.71 (12.89)	36.20 (29.00-46.90)	45.55 (22.29)	40.30 (31.50-52.80)
FFQ no 2	52.43 (34.41)	42.70 (33.90-62.70)	33.81 (14.11)	28.70 (24.50-35.40)	38.33 (25.62)	34.25 (25.40-46.80)
Combined	50.42 (29.37)	46.80 (36.75-54.70)	36.26 (12.21)	35.55 (26.45-42.45)	41.05 (21.27)	37.86 (26.46-49.99)

Variable	Subjects		Controls		All	
Selenium (%)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
24 hr no 1	95.83 (48.64)	100.60 (62.80-116.00)	74.29 (28.96)	82.40 (52.30-101.00)	86.64 (50.26)	82.40 (53.80-111.40)
24 hr no2	81.23 (44.35)	69.40 (48.50-106.70)	92.29 (46.95)	86.20 (54.10-130.10)	80.03 (47.06)	74.20 (46.10-106.60)
24 hr no 3	97.59 (58.80)	114.00 (59.00-133.30)	91.25 (50.55)	80.70 (62.80-136.10)	97.67 (54.12)	87.40 (62.80-133.30)
Combined	92.73 (30.78)	85.53 (78.23-109.23)	85.94 (26.92)	83.10 (66.50-110.77)	87.40 (30.56)	85.55 (69.41-109.23)
FFQ no 1	152.93 (52.93)	156.50 (115.10-166.00)	120.00 (48.97)	108.70 (88.30-138.90)	135.67 (49.05)	126.20 (102.70-157.00)
FFQ no 2	142.47* (88.60)	111.50 (94.80-156.80)	90.93* (30.41)	84.00 (67.60-112.80)	106.59 (66.51)	102.15 (69.20-121.20)
Combined	147.70 (65.56)	130.25 (110.65-155.30)	105.46 (34.36)	98.75 (86.90-120.50)	118.55 (51.97)	114.65 (88.12-137.80)

* Significant difference between subject and control population; $p < 0.0352$ (determined through the non-parametric sign test).

A visual representation of the dietary intake is shown in *Figure 1*.

Figure 1: The % nutrient intake according to 24 hr recalls and FFQ's for the different population groups



Ideally, the average of the three 24 hour recalls and the first FFQ should be representative of usual intake of during pregnancy. However, the difference in mean and median of the two different questionnaires is quite large for most nutrients as can be seen above; and to combine them would skew the data and would not necessarily be representative of the usual intake.

Nutrient intake was expressed as a percentage value of the Recommended Daily Allowance (RDA). Adequate daily intake is 66.67 – 133.33% RDA, inadequate intake is < 66.67% RDA and > 133.33% RDA. Following in *Table 14* is the distribution of the number of individuals in the subject, control and the total population in the different RDA groups of adequate and inadequate. There was only a significant difference between the subject and control population's calcium intake according to the FFQ. The n values are the same as specified in *Table 21*.

Table 23: Distribution of individuals having adequate or inadequate intake of nutrients within the different population groups.

Energy	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	3	1	8	4	3	10
Control population	2	0	12	10	1	5
Total population	8	1	30	22	4	20

Protein	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	0	1	6	2	8	12
Control population	1	0	6	1	8	14
Total population	2	1	16	8	24	37

Vitamin A	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	8	0	3	3	3	12
Control population	8	1	4	5	3	9
Total population	25	3	11	14	6	29

Vitamin D	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	12	5	1	8	1	2
Control population	15	11	0	4	0	0
Total population	36	22	5	21	1	3

Vitamin E	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	5	0	9	4	0	11
Control population	3	0	10	7	2	8
Total population	11	0	25	16	6	30

Vitamin K	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	8	1	4	11	2	3
Control population	8	2	7	10	0	3
Total population	23	5	16	31	3	10

Vitamin C	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	7	1	2	3	5	11
Control population	3	0	4	1	8	14
Total population	16	1	9	8	17	37

Thiamin	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	8	1	5	9	1	5
Control population	5	0	9	12	1	3
Total population	23	3	17	11	2	2

Riboflavin	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	6	0	6	5	2	10
Control population	4	0	8	6	3	9
Total population	17	0	9	21	6	25

Niacin	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	1	1	8	5	5	9
Control population	1	0	6	3	8	12
Total population	4	2	21	18	17	26

Vitamin B6	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	6	1	6	9	2	5
Control population	7	0	7	12	1	3
Total population	19	3	20	32	3	11

Folate	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	11	2	2	11	1	2
Control population	8	1	6	11	1	3
Total population	28	6	12	35	2	5

Vitamin B12	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	0	0	6	11	8	14
Control population	1	0	3	11	11	15
Total population	1	0	14	35	27	45

Calcium	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	11	6*	2	7*	1	2*
Control population	10	0*	4	13*	1	2*
Total population	33	13	7	29	2	4

* Statistical significant difference according to the Fischer's Exact test $p < 0.0134$.

Phosphorous	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	5	1	6	6	3	8
Control population	2	0	11	5	2	10
Total population	10	1	26	22	6	23

Magnesium	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	5	1	7	8	2	6
Control population	5	0	9	7	1	8
Total population	15	2	24	27	3	17

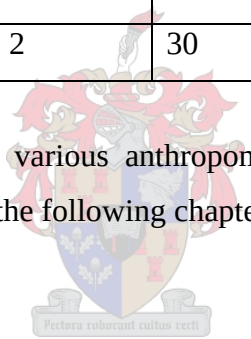
Iron	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	13	10	1	4	0	1
Control population	14	12	1	3	0	0
Total population	40	38	2	7	0	1

Zinc	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	8	1	5	11	1	3
Control population	7	1	7	13	1	1
Total population	22	5	18	37	2	4

Iodine	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	14	12	0	2	0	1
Control population	15	14	0	1	0	0
Total population	41	41	1	4	0	1

Selenium	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Subject population	2	0	11	8	1	7
Control population	4	1	10	12	1	2
Total population	10	2	30	31	2	13

Dietary intake was compared to various anthropometric, biochemical and newborn data. These results will be discussed in the following chapters.



4. Anthropometric results

In **2.5.2** the type and number of anthropometric measurements taken of each individual is discussed in full.

Body Mass Index (BMI), can only be used as an indice for nutritional status if the pregnant mother knew what her pre-pregnancy weight (PPW) was or if her weight was taken within the 1st trimester of the pregnancy.[67] As shown in *Table 24*, the following individuals did or did not know their PPW or had a weight appraisal in the 1st trimester.

Table 24: The number of individuals of whom the PPW or a 1st trimester weight appraisal value was known of in the different population groups.

PPW or weight in the 1st trimester?	Subjects (n=15)	Controls (n=15)	Total (n=46)
"Yes"	6	8	25
"No"	9	7	21

As seen above, almost half of the population did not know their PPW, or they only had their weight appraised after the 1st trimester of pregnancy. This was quite problematic for the researcher, since it was important to determine the mothers' nutritional status to determine whether their weight gain during pregnancy was appropriate or not, according to their pre-pregnancy nutritional status.

Nutritional status distribution according to anthropometric measurements

The nutritional status distribution according to BMI, < 18.5 being undernourished, 18.5 – 25 normally nourished, and > 25 overnourished, for the subject and control populations are:

Table 25: Nutritional status distribution according to BMI of the different population groups.

BMI classifications:	Subjects (n=6)	Controls (n=8)	Total (n=25)
< 18.5	2	0	3
18.5 – 25	3	6	16
> 25	1	2	6

Figure 2 shows the BMI distribution visually in the different population groups.

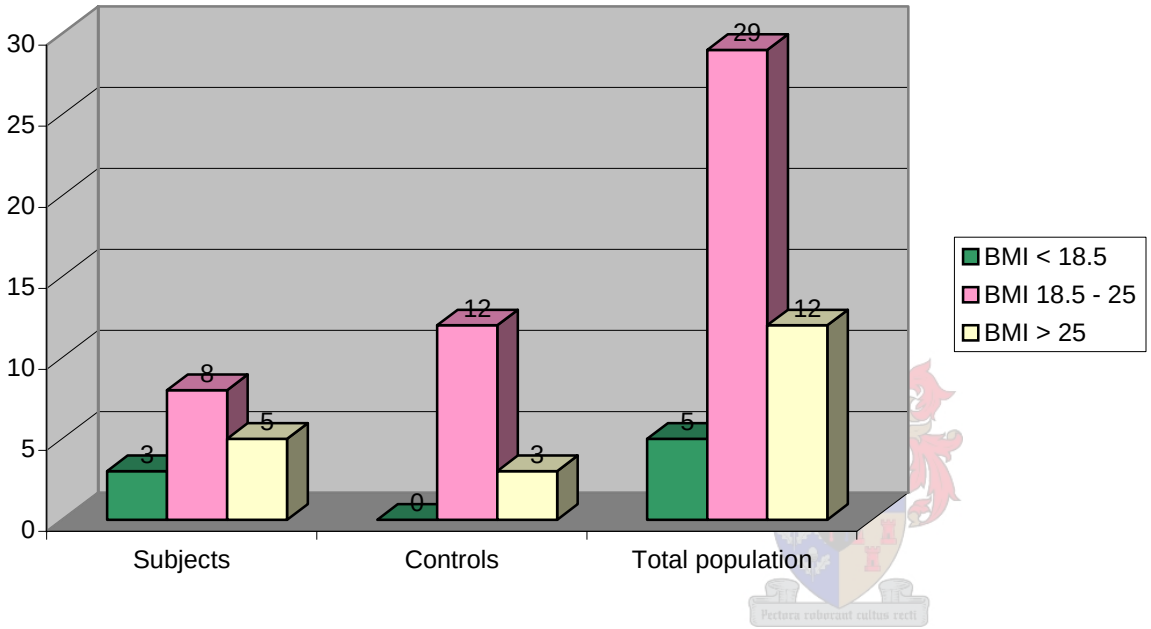
When the complete classification system of BMI is used, there was no significance; probably because the numbers per classification in the different population groups were too small.

Table 26: Nutritional status distribution according to BMI of the different population groups.

BMI classifications		Subjects (n=15)	Controls (n=15)	Total (n=46)
Undernourished	Severe: < 16	1	0	1
	Mild: 16 – 16.9	1	0	1
	Slight: 17 – 18.49	1	0	3
Normal	18.5 – 24.9	8	12	29
Overnourished	Grade 1: 25- 29.9	4	3	10
	Grade 2: 30 – 39.9	1	0	2
	Grade 3: > 40	0	0	0

The other method of nutritional status assessment was measuring the MUAC. The interpretation of the MUAC indices is shown in the Methodology chapter:

Figure 2: Nutritional status distribution according to BMI of the different population groups



The distribution of the subject and control populations are as follows:

Table 27: The distribution of individuals according to nutritional status expressed as MUAC or the different population groups.

MUAC percentile groups	Subjects (n=15)	Controls (n=15)	Total (n=46)
< 5	3	0	4
5 – 10	0	0	3
10 – 25	4	3	8
25 – 75	6	9	21
75 – 90	2 *	3	7
90 – 95	1	0	3 *
> 95	0	0	0

* Two individuals with BMI's over 30, which could make their MUAC's inaccurate.

There was no statistical significance to the difference in distribution.

Pre-pregnancy BMI is used to determine how much weight an individual must gain in the different trimesters of pregnancy (see *Table 5*). Three weight appraisals were done by the researcher on each individual. The difference between the three appraisals was then interpreted according to which BMI grouping and trimester of pregnancy. For the sake of convenience, the researcher decided to name the different results of weight gain, for easier understanding:

- "correct" being weight gain closest to the recommended weight gain,
- "positive" being that weight is gained during pregnancy, but not enough according to the recommended amount of weight gain,
- "fast" being that more weight than is recommended is being gained, and
- "negative" being that no weight is gained, or that weight is actually being lost.

The distribution of the subject and control populations are as follows:

Table 28: The distribution of individuals in the different population groups according to weight gain interpretation groups.

Weight gain interpretation:	Subjects (n=15)	Controls (n=15)	Total (n=46)
"Correct"	2	1	5
"Fast"	5	1	88
"Negative"	5	0	8
"Positive"	7	9	24
"Positive", but individual obese	0	0	1

* The statistical significance according to Chi-Square test is $p < 0.0411$.

The researcher wanted to know if there was a trend between the different weight gain groups and nutritional status classifications, using BMI (< 18.5 being undernourished, 18.5 – 25 normally nourished, and > 25 overnourished). The distribution were as follows:

Table 29: Distribution of the population between different weight gain interpretation groups compared to the BMI classification groups.

BMI classification	"Correct" weight gain (n=5)	"Fast" weight gain (n=8)	"Positive" weight gain (n=25)	"Negative" weight gain (n=8)
< 18.5	0	0	2	3
18.5 – 25	4	6	17	2
> 25	1	2	6	3

There was no statistical significance to the distribution.

Anthropometric measurements compared with dietary intake

Nutritional status according to the BMI classification (< 18.5 being undernourished, 18.5 – 25 normally nourished, and > 25 overnourished), have been compared to the average dietary intake of the three 24 hour recalls and the two FFQ's. The results are as follows:

Table 30: Mean nutrient intake of the different BMI classification groups according to the average of three 24 hour recalls and two FFQ's.

NUTRIENTS Mean values in percentage	**BMI < 18.5** (n=9)		**BMI 18.5 – 25** (n=29)		**BMI > 25** (n=9)	
	24 hr recalls	**Food frequency**	**24 hr recalls**	**Food frequency**	**24 hr recalls**	**Food frequency**
Energy	103.63	145.93	88.04	133.60	105.79	188.83
Protein	137.96	164.71	130.10	158.08	161.21	218.80
Vitamin A	72.12	266.93	82.46	192.26	106.27	207.11
Vitamin D	70.65	74.84	32.49	62.18	45.09	95.48
Vitamin E	120.74	159.10	99.59	154.04	96.83	207.89
Vitamin K	74.90	94.84	71.45	128.46	55.16	128.35
Vitamin C	209.06	286.72	167.85	329.35	126.13	330.82
Thiamin	79.89	105.68	64.21	103.13	78.32	141.07
Riboflavin	87.85	155.88	84.50	149.50	104.99	206.23
Niacin	132.56	136.58	118.32	136.61	134.46	185.41
Vitamin B6	82.56	104.49	67.45	101.54	95.81	142.28
Folate	69.23	108.51	54.62	99.53	59.36	117.04
Vitamin B12	217.60	362.02	166.24	313.25	233.54	416.04
Calcium	38.57	77.75	51.74	87.66	60.55	125.51
Phosphorous	88.89	131.18	89.44	132.66	109.97	188.37
Magnesium	85.66	125.89	78.70	125.98	94.91	171.97
Iron	39.63	53.54	33.52	53.03	38.43	68.07
Zinc	74.66	95.74	68.48	92.49	90.10	124.53
Iodine	30.57	40.23	24.40	36.57	35.97	60.78
Selenium	105.02	137.84	78.84	108.11	100.88	148.22

There was no statistical significance when the mean values of different nutrients were compared among the different BMI classification groups.

The different weight gain interpretations ("correct", "fast", "positive" and "negative") were compared to the average dietary intake of the three 24 hour recalls and the two food frequency questionnaires. The results are as follows:

Table 31: Mean nutrient values of the different weight gain interpretation groups.

NUTRIENTS' mean values expressed as %	**CORRECT WEIGHT GAIN** (n=5)		**FAST WEIGHT GAIN** (n=8)		**POSITIVE WEIGHT GAIN** (n=25)		**NEGATIVE WEIGHT GAIN** (n=8)	
	24 hour recall	**FFQ**	**24 hour recall**	**FFQ**	**24 hour recall**	**FFQ**	**24 hour recall**	**FFQ**
Energy	93.65	135.56	103.96	181.38	91.52	136.27	90.52	154.81
Protein	123.86	165.21	152.10	212.04	136.25	161.03	132.45	169.15
Vitamin A	70.75	211.19	93.95	193.71	92.57	219.15	48.65	196.59
Vitamin D	43.57	79.55	35.45	78.91	44.95	62.35	30.51	78.95
Vitamin E	91.16	167.31	111.08	196.46	108.83	152.48	70.50	173.60
Vitamin K	58.71	97.69	71.41	123.07	75.43	136.23	43.95	96.36
Vitamin C	107.63	268.58	169.05	301.89	200.45	334.56	60.59	366.57
Thiamin	56.00	103.39	70.42	135.89	73.39	106.95	62.91	107.46
Riboflavin	67.89	134.77	81.84	211.38	96.21	154.76	86.63	158.82
Niacin	115.71	129.73	139.53	180.79	123.83	141.80	106.31	137.94
Vitamin B6	56.83	97.52	82.93	143.51	78.00	104.73	69.17	104.31
Folate	49.75	79.81	57.53	119.94	62.45	108.31	45.31	95.65
Vitamin B12	145.11	359.54	191.30	351.84	194.86	313.64	189.44	406.81
Calcium	39.76	77.14	48.68	122.36	54.50	90.84	51.01	87.14
Phosphorous	80.88	135.96	102.06	185.24	94.25	136.21	86.77	131.72
Magnesium	71.28	113.65	93.73	176.44	83.97	132.82	72.49	118.86
Iron	29.88	49.18	39.00	63.23	36.28	55.45	31.57	57.14
Zinc	65.15	92.57	80.82	125.26	72.62	94.13	75.41	97.55
Iodine	24.68	44.60	24.38	51.41	30.77	38.05	20.76	41.99
Selenium	87.99	135.07	89.73	139.48	89.90	114.45	71.12	114.99

There was no statistical significance when the mean values of different nutrients were compared among the different weight gain groupings.

Anthropometric measurements compared with newborn data

The mothers' BMI, MUAC and weight gain was compared with the newborns' birth weight, gestational age at birth, weight for age and head circumference for age taken at one month, for any significance. The results for the total population (n=45) were as follows:

Table 32: The newborn's weight for age classification compared with the mother's BMI classification.

BMI classification	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
< 18.5	1	4	4	0
18.5 – 25	3	12	12	0
> 25	0	6	2	1

Table 33: The newborn's head circumference for age classification compared with the mother's BMI classification.

BMI classification	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
< 18.5	0	6	3	0
18.5 – 25	2	9	15	1
> 25	0	3	6	0

Table 34: The newborn's birth weight compared with the mother's BMI classification.

BMI classification	Normal birth weight (> 2500g)	Low birth weight (< 2500g)
< 18.5	3	2
18.5 – 25	20	8
> 25	11	1

Table 35: The newborn's gestational age at birth compared with the mother's BMI classification.

BMI classification	Term birth (38 – 42 weeks)	Premature birth (< 38 weeks)
< 18.5	3	2
18.5 – 25	25	4
> 25	12	0

Table 36: The newborn's weight for age classification compared with the mother's MUAC classification.

MUAC classification (in percentiles)	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
< 25	2	8	5	0
25 – 75	2	10	8	0
> 75	0	4	5	1

Table 37: The newborn's head circumference for age classification compared with the mother's MUAC classification.

MUAC classification (in percentiles)	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
< 25	0	8	6	1
25 – 75	2	7	11	0
> 75	0	3	7	0

Table 38: The newborn's birth weight compared with the mother's MUAC classification.

MUAC classification (in percentiles)	Normal birth weight (> 2500g)	Low birth weight (< 2500g)
< 25	10	5
25 – 75	14	5
> 75	10	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0449$.

Table 39: The newborn's gestational age at birth compared with the mother's MUAC classification.

MUAC classification (in percentiles)	Term birth (38 – 42 weeks)	Premature birth (< 38 weeks)
< 25	10	4
25 – 75	19	2
> 75	10	0

Table 40: The newborn's weight for age classification compared with the mother's weight gain interpretation groups.

Weight gain	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
"Correct"	0	0	5	0
"Fast"	0	5	3	0
"Negative"	0	3	5	0
"Positive"	4	14	5	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0056$.

Table 41: The newborn's head circumference for age classification compared with the mother's weight gain interpretation groups.

Weight gain	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
"Correct"	0	2	3	0
"Fast"	0	2	6	0
"Negative"	0	2	6	0
"Positive"	2	12	9	1

Table 42: The newborn's birth weight compared with the mother's weight gain interpretation groups.

Weight gain	Normal birth weight (> 2500g)	Low birth weight (< 2500g)
"Correct"	4	1
"Fast"	8	0
"Negative"	8	0
"Positive"	14	10

Table 43: The newborn's gestational age at birth compared with the mother's weight gain interpretation groups.

Weight gain	Term birth (38 – 42 weeks)	Premature birth (< 38 weeks)
"Correct"	5	0
"Fast"	8	0
"Negative"	6	2
"Positive"	20	4

Other than those indicated, none of the above had any statistical significance.

The researcher wanted to determine whether there was any link between the incidence of FAS or "deferred" status of the newborn, and the mothers being undernourished or not having gained sufficient weight during pregnancy. The results were as follows:

Table 44: The newborn's FAS status compared to the mother's BMI classification.

BMI classification	Normal	"deferred"	FAS
< 18.5	4	1	0
18.5 – 25	21	5	2
> 25	6	5	1

When the researcher subdivided the BMI classification to differentiate between subjects and controls (n=30), the results were non-significant as well.

Table 45: The newborn's FAS status compared to the mother's BMI classification, subdivided as subjects and controls.

BMI classification		Normal	"deferred"	FAS
< 18.5	Subjects	2	1	0
	Controls	0	0	0
18.5 – 25	Subjects	4	3	0
	Controls	11	1	0
> 25	Subjects	2	2	1
	Controls	2	1	0

Table 46: The newborn's FAS status compared to the mother's weight gain interpretation groups.

Weight gain	Normal	"deferred"	FAS
"Correct"	2	3	0
"Fast"	6	2	0
"Negative"	7	0	1
"Positive"	16	6	2

There was no statistical significance found for the above data and of that between the newborn's FAS-status and MUAC classification of the mother.

5. Biochemical data

The researcher decided to investigate serum folate, vitamin A, ferritin, plasma zinc and haemoglobin and haematocrit.

The individuals who took part in the researcher's study was asked to volunteer a blood sample as not to be under any pressure to do so and perhaps consequently withdraw from the study. The results of individuals who did or did not volunteer a blood sample are as follows:

Table 47: The number of individuals of the different population groups who volunteered a blood sample.

Volunteer?	Subjects (n=15)	Controls (n=15)	Total (n=47)
"Yes"	11	10	31
"No"	4	5	16

Biochemical data distribution

The mean, standard deviation, median and interquartile range (q1-q3) of zinc, folate, vitamin A and ferritin was determined of the subject, control and total population groups:

Table 48: Zinc's values of the different population groups.

Zinc:	Subjects (n=8)		Controls (n=10)		Total (n=25)	
	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
	14.70 (1.88)	14.80 (13.20-16.00)	15.14 (2.53)	14.80 (14.00-18.00)	14.25 (2.24)	14.10 (12.30-15.80)

Table 49: Folate's values of the different population groups.

Folate:	Subjects (n=11)		Controls (n=10)		Total (n=31)	
	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
	17.06 (18.55)	11.10 (7.48-19.27)	12.19 (7.85)	9.97 (8.16-18.10)	15.14 (12.98)	11.21 (8.16-18.10)

Table 50: Vitamin A's values of the different population groups.

Vitamin A:	Subjects (n=11)		Controls (n=10)		Total (n=31)	
	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
	5.72 (13.27)*	1.58 (1.51- 2.54)	1.20 (0.31)*	1.04 (0.98- 1.30)	3.24 (8.24)	1.47 (1.06- 1.76)

* The statistical significance according to Kruskal-Wallis test is $p < 0.0097$.

Table 51: Ferritin's values of the different population groups.

Ferritin:	Subjects (n=11)		Controls (n=9)		Total (n=30)	
	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)	Mean (Std Dev)	Median (Inter-quartile range)
	24.92 (16.73)	22.80 (14.40-29.40)	23.83 (12.37)	25.70 (20.90-29.90)	24.00 (15.34)	22.10 (14.40-29.40)

According to literature, folate, vitamin A and zinc could be classified as "low risk", "moderate risk" and "high risk" values.[69] The distribution of values between the subject and control populations were as follows:

Table 52: The distribution of individuals among the different population groups according to their risk value grouping of zinc.

Zinc's classification:	Subjects (n=8)	Controls (n=9)	Total (n=25)
Low Risk	8	7	25
Moderate Risk	0	0	0
High Risk	0	0	0

Table 53: The distribution of individuals among the different population groups according to their risk value grouping of folate.

Folate's classification:	Subjects (n=11)	Controls (n=10)	Total (n=31)
Low Risk	3	3	11
Moderate Risk	5	3	15
High Risk	2	1	4

Table 54: The distribution of individuals among the different population groups according to their risk value grouping of vitamin A.

Vitamin A 's classification:	Subjects (n=11)	Controls (n=10)	Total (n=31)
Low Risk	11	4	25
Moderate Risk	0	4	6
High Risk	0	0	0

There was no statistical significance found.

According to literature, ferritin is a good measure of indicating iron deficiency in conjunction with haemoglobin, which can be detrimental to the mother and growing fetus' health. The cut-off points indicating iron deficiency, are ferritin < 20μg/l and haemoglobin > 105g/l.[63] The distribution of the population groups were as follows:

Table 55: The distribution of iron deficiency among the different population groups.

Iron deficiency?	Subjects (n=10)	Controls (n=9)	Total (n=30)
"Yes"	4	2	13
"No"	6	7	17

Anaemia, according to literature, is defined by the haemoglobin and haematocrit values of the individual. During the different trimesters of pregnancy, the cut-off values for anaemia are different. Also, the cut-off values are influenced by the amount of cigarettes smoked per day and at what height above sea level the individuals live when the blood samples are taken. As the study was conducted in Cape Town, which is right at the sea, last mentioned would not influence the cut-off values for anaemia. See the methodology chapter for the specific cut-off values for anaemia during pregnancy depending on the amount of cigarettes that were smoked per day. Each individual's haemoglobin and haematocrit values were interpreted to determine whether they had anaemia or not; the results were as follows:

Table 56: The distribution of individuals with anaemia among the different population groups.

Anaemia?	Subjects (n=15)	Controls (n=14)	Total (n=46)
"Yes"	3	0	3
"No"	12	14	43

If anaemia coincides with a ferritin value < 12μg/l, then the individual has an iron deficiency anaemia.[26] Only one of the three subjects who had anaemia had the appropriate ferritin value to have iron deficiency anaemia.

None of the above distributions had any statistical significance.

Biochemical data compared with dietary intake.

Zinc, folate and vitamin A's groupings of biochemical values were compared to the average distribution of the appropriate nutrient in groupings of < 66.67% (low intake), 66.67 – 133.33% (adequate intake) and > 133.33% (high intake) of the RDA. Both 24 hour recall and FFQ data will be showed:

Table 57: Comparison of zinc's values according to dietary intake and biochemical analysis.

Zinc (n=30)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Low risk"	7	1	6	13	2	1
"Moderate risk"	-	-	-	-	-	-
"High risk"	-	-	-	-	-	-

Table 58: Comparison of folate's values according to dietary intake and biochemical analysis.

Folate (n=30)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Low risk"	5	1	3	5	1	3
"Moderate risk"	5	1	3	7	0	0
"High risk"	2	0	0	2	1	1

Table 59: Comparison of vitamin A's values according to dietary intake and biochemical analysis.

Vitamin A (n=30)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Low risk"	9	0	2	4	4	11
"Moderate risk"	2	1	1	0	2	4
"High risk"	-	-	-	-	-	-

The presence or absence of iron deficiency, anaemia and iron deficiency anaemia was compared to the average distribution of the dietary intake of iron in groupings of < 66.67% (low intake), 66.67 – 133.33% (adequate intake) and > 133.33% (high intake) of the RDA. Both 24 hour recall and FFQ data will be showed:

Table 60: Comparison of the incidence of iron deficiency to the iron intake of individuals.

Iron deficiency? (n=30)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Yes"	6	5	0	1	-	0
"No"	20	16	2	6	-	1

Table 61: Comparison of the incidence of anaemia to the iron intake of individuals.

Anaemia? (n=30)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Yes"	2	2	1	1	-	0
"No"	16	14	1	2	-	1

Table 62: Comparison of the incidence of iron deficiency anaemia to the iron intake of individuals.

Iron deficiency anaemia? (n=30)	< 66.67% RDA		66.67 – 133.33% RDA		> 133.33% RDA	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
"Yes"	1	1	0	0	-	0
"No"	17	15	2	3	-	1

There is no statistical significance to any of the above.

Biochemical data compared to clinical signs.

The coincidence of low biochemical values and the presence of clinical deficiency signs are discussed fully in **6.1**. However, no statistical significant relationships were found.

Biochemical data compared to anthropometric data.

According to literature, the only biochemical markers that coincides with an anthropometric finding, is the relationship between anaemia, iron deficiency anaemia and undernutrition.[4] The presence of anaemia and iron deficiency anaemia was compared to the distribution of nutritional status according to BMI and MUAC, and the rates of weight gain. The only statistically significant relationship was found between BMI and anaemia:

Table 63: The incidence of anaemia of individuals compared with their BMI classification.

BMI classification	Anaemia?	
	"Yes"	"No"
< 18.5 (undernourished)	2	2
18.5 – 25 (normally nourished)	0	11
> 25 (overnourished)	1	4

* A statistical significance according to the Fischer's Exact test of $p < 0.0386$ was found.

Biochemical values compared to newborn findings

According to literature, an increased risk for LBW and prematurity was associated with low biochemical values of zinc and folate.[3,20] The same association was found between pregnancy induced hypertension and zinc;[20] iron deficiency anaemia, anaemia and prematurity and LBW.[21] The researcher determined that there was no statistical significance between these associations in this particular study.

Literature also linked heavy alcohol consumption with impaired absorption, utilization or excretion of folate, zinc and vitamin A.[43,45] The researcher tried to find an association between the incidence of FAS or the "deferred" status and low biochemical values of folate, zinc and vitamin A. No statistical significant associations were found.

6. Clinical signs

Individuals were examined for clinical signs of nutrient deficiencies and/or toxicities that were relevant to this study. These nutrient deficiencies and toxicities are: energy and protein deficiency, vitamin A deficiency and toxicity, thiamin deficiency, vitamin B6/ pyridoxine deficiency, vitamin C deficiency, folate deficiency, zinc deficiency and iron deficiency. The total, subject and control population's incidence of clinical deficiency signs are as follows:

Table 64: The incidence of different clinical signs among the population groups.

DEFICIENCIES/ TOXICITY	CLINICAL SIGNS		Total (n=47)	Subject (n=22)	Control (n=25)
MUSCLE WASTAGE (ENERGY AND PROTEIN DEFICIENCY)	Thumb muscle		0	0	0
	Fore arm		4	3	1
	Upper arm		3	2	1
	Face		0	0	0
	Chest		0	0	0
	Upper leg		2	2	0
	Lower leg		4	3	1
VITAMIN A TOXICITY	Palms: Hypercarotenodermia		0	0	0
VITAMIN A DEFICIENCY	Fore arm and upper leg: Follicular Hyperkeratosis		0	0	0
	Eyes	Night blindness	2	2	0
		Xerosis	0	0	0
		Bitot's Spots	0	0	0
		Xeropthalmia	0	0	0
THIAMIN (VITAMIN B1) DEFICIENCY	Eyes: Nystagmus		0	0	0
	Lower leg: calf muscle tenderness		0	0	0
	Feet: "Foot drop"		0	0	0
PYRIDOXINE (VITAMIN B6) DEFICIENCY	Lower leg: calf muscle tenderness		0	0	0
	Eyes: redness & fissuring of eyelid corners		0	0	0
VITAMIN C DEFICIENCY	Scurvy	Follicular hyper-keratosis	0	0	0
		Dry itchy skin	3	2	1
		Swollen and inflamed gums	0	0	0
		Loss of hair	1	1	0
	Skin: bleeding		0	0	0
FOLATE DEFICIENCY	Mouth: Glossitis		0	0	0
ZINC DEFICIENCY	Head: hair loss		1	1	0
	Skin: impaired wound healing		0	0	0
IRON DEFICIENCY	Iron deficiency anaemia: spoon-shaped nails		1	0	1
	Anaemia (e.g. iron deficiency): pale conjunctiva		12	5	7

Other clinical signs which were observed, but that did not have any significance to this study were eczema (three times), opaque nails (four times) and pellagra on one occasion.

Because of the low incidences of clinical signs, significance could not be tested.

6.1 Clinical signs corroborated by biochemical deficiencies

A clinical sign is only a possible indication of a certain nutrient's deficiency and/or toxicity. Since clinical signs could have other causes than nutrient deficiencies and/or toxicities, a biochemical test should be done to confirm whether the clinical sign's cause is a nutrient deficiency and/or toxicity.[4] In this study vitamin A, folate, zinc and ferritin were tested for.

The clinical sign that appeared most frequent was the presence of pale conjunctiva, which could be an indication of anaemia, e.g. an iron deficiency anaemia. The clinical sign on its own does not confirm anaemia, and must corroborated by low haemoglobin, haematocrit; and in the case of iron deficiency anaemia, a low ferritin value as well. Of the subject population, only two individuals had the biochemical markers of anaemia and the clinical signs, of which one individual had low ferritin as well. In the control population, only one individual had a low ferritin value but no anaemia according to her biochemical values. Using Fischer's Exact test, no significance was found between the clinical sign for anaemia and iron deficiency anaemia, and the biochemical markers indicating anaemia, iron deficiency anaemia or iron deficiency.

The incidence of vitamin A deficiency's clinical signs (two cases of night blindness) did not coincide with a high-risk value of vitamin A according to biochemical tests; therefore no significance was found.

There was no incidence of zinc and folate deficiencies' clinical signs to compare with biochemical values.

6.2 Clinical signs corroborated by inadequate dietary intakes

All the nutrients that could possibly be involved with the presence of clinical signs noted in this study were measured through dietary recall questionnaires. Significance testing was done on the incidence of clinical signs compared to the average intake of the group falling within <66.67% (low intake), 66.67 - 133.33% (adequate intake), and the >133.33% (high intake) of the RDA intake groups. No significance was found.

As another option, the mean intake of the group having clinical signs, compared to those not having clinical signs, were compared. Here follows an example of the clinical signs of muscle wastage.

Table 65: Comparison of energy and protein intake between individuals having clinical signs of muscle wastage and those who do not.

Muscle wastage area (number who had signs)	Nutrient concerned	No clinical signs observed		Clinical signs observed	
		Mean value as % of 24 hour recall	Mean value as % of FFQ	Mean value as % of 24 hour recall	Mean value as % of FFQ
Fore arm (4)	Protein	140.5	174.7	96.3	133.8
	Energy	105.7	149.1	62.3	121.9
Upper arm (3)	Protein	138.8	174.4	107.8	123.9
	Energy	104.4	148.7	66.4	116.2
Upper leg (2)	Protein	138.8	172.7	107.8	135.5
	Energy	104.4	148.7	66.4	116.2
Lower leg (4)	Protein	140.5	96.3	174.7	133.8
	Energy	105.7	149.1	62.3	121.9

Although there seems to be a difference between the mean nutrient intake of two groups, the sample size was too small to do any significance testing on.

6.3 Clinical signs corroborated by anthropometric measures

The focus here is on the coincidence of clinical signs (especially muscle wastage) with low BMI, MUAC (see Methodology chapter classifications) and weight gain groupings. Fischer's Exact test were performed and the following significant relations were found:

Table 66: Comparison of different muscle wastage areas to the BMI classifications of individuals.

BMI classification	Fore arm muscle wastage*		Upper leg muscle wastage*		Lower leg muscle wastage*	
	No's of "yes"	No's of "no"	No's of "yes"	No's of "no"	No's of "yes"	No's of "no"
<16	0	0	0	0	0	0
16-17	1	2	1	0	1	0
17-18.5	2	2	1	3	2	2
18.5-25	1	27	0	28	1	27
25-30	0	11	0	11	0	11
30-40	0	3	0	3	0	3
>40	0	0	0	0	0	0

* Significance according to Fischer's Exact tests: fore arm muscle wastage p <0.0077, upper leg muscle wastage p <0.0093, and lower leg muscle wastage p <0.0077.

Table 67: Comparison of upper leg muscle wastage to the MUAC classifications of individuals.

MUAC classification	Upper leg muscle wastage*	
	No's of "yes"	No's of "no"
<5	2	2
5-10	0	3
10-25	0	8
25-75	0	22
75-90	0	7
90-95	0	3
>95	0	0

* Significance according to Fischer's Exact test: p <0.0111

Inappropriate weight gain had a significant relationship with the presence of pale conjunctiva (p <0.0334) only.

6.4 Clinical signs corroborated by newborn findings

The aim was to determine a relationship between clinical signs of the mother and the newborn's anthropometric data and FAS status.

The only finding in the newborn's anthropometric data was that there was a significant relationship between the newborn's weight for age grouping and the presence of night blindness in the mother ($p < 0.0101$).

Regarding the newborns who had a FAS or "deferred" status, the researcher wanted to determine whether the affected newborn's mothers had more clinical signs compared to those mothers of unaffected newborns.

Table 68: Incidence of clinical signs between the different FAS status groups

Clinical sign		Number of "deferred" status newborns	Number of FAS status newborns	Number of normal newborns
Fore arm muscle wastage	"Yes"	1	1	2
	"No"	10	2	29
Upper arm muscle wastage	"Yes"	1	0	2
	"No"	10	3	29
Upper leg muscle wastage	"Yes"	1	0	1
	"No"	10	3	30
Lower leg muscle wastage	"Yes"	1	1	2
	"No"	10	2	29
Night blindness	"Yes"	1	0	1
	"No"	10	3	30
Hair loss	"Yes"	1	0	0
	"No"	10	3	31
Dry itchy skin	"Yes"	1	0	2
	"No"	10	3	29
Spoon-shaped nails	"Yes"	0	1	0
	"No"	11	2	31
Pale conjunctiva	"Yes"	2	2	8
	"No"	9	1	23

There was no significance between the relationship of FAS or "deferred" status of the newborns compared to the presence of clinical signs in the mother.

7. Newborn data

Upon visiting the FARR offices with their newborns (at approximately one month of age), forty-five mothers had their babies examined by an experienced paediatric doctor who has experience in identifying alcohol related signs in children.

7.1 Newborn statistics

The sex distribution of the babies were as follows:

Table 69: The sex distribution of the newborns in the population groups.

SEX	Subjects (n=15)	Controls (n=15)	Total (n=45)
Female	6	10	24
Male	9	5	21

After each birth at Hanover Park MOU, a "birth summary" is made, which gives details regarding the birth, the babies' birth weight, head circumference (HC) and length, and any other relevant data that could influence the health of the mother or the baby. The researcher was interested in the birth weight, which was classified as normal birth weight and different classifications of low birth weight (LBW). The distribution was as follows:

Table 70: The distribution of birth weights between the different population groups.

Weight classifications	Subjects (n=15)	Controls (n=15)	Total (n=45)
Normal birth weight (> 2500g)	10	12	34
Low birth weight (< 2500g)	5	3	11

The paediatric doctor did a full anthropometric assessment on the babies. The researcher concentrated on their weights and head circumferences, and they were plotted on percentile tables appropriate for age. The following table divides the weights and HC's into < 5th

percentile (low weight or small), 5 – 95th percentile (normal) and > 95th percentile (over weight or large) for the subject and control populations.

Table 71: The distribution of newborns according to their weight and head circumference in the different population groups.

Anthropometric measurement and classification		Subjects (n=14)	Controls (n=15)	Total (n=45)
Weight for age	< 5	1	0	3
	5-50	7	11	23
	50-95	6	4	18
	> 95	0	0	1
HC for age	< 5	0	0	2
	5-50	7	6	18
	50-95	7	9	24
	>95	0	0	1

Using the estimated date of delivery (EDD) determined by the ultrasound examination each mother underwent, the researcher determined whether the babies were born prematurely (before 38 weeks gestational age) or term (between 38 and 42 weeks gestational age). The distribution were as follows:

Table 72: The distribution of newborns being born term or prematurely in the different population groups.

Gestational age	Subjects (n=15)	Controls (n=15)	Total (n=45)
Term	13	14	39
Premature	2	1	6

The paediatric doctor, who is spesialised in identifying alcohol related signs in babies and children, did a thorough examination of each newborn checking for any alcohol related signs. After this, he would make a diagnosis of the newborn either having Fetal Alcohol Syndrome (FAS), being normal or state the following: “deferred”. Defer, in this context, means to delay until a later date. It is very difficult to make an accurate diagnosis of FAS in infancy, and that a re-examination from three years of age would make for a better judgment. Nevertheless, here follows the doctor’s findings shown in *Table 73*:

Table 73: The distribution of the newborns according to their "FAS status" in the different population groups.

Diagnosis	Subjects (n=15)	Controls (n=15)	Total (n=45)
Normal	8	14	31
FAS	1	0	3
"deferred"	6	2	11

There was no statistical significance to the above data.

In the birth summary, any complications that were experienced during delivery or the pregnancy were noted. The researcher was interested in the incidence of complications in the subject and control populations, and the difference thereof. However, no statistical significance was found between the difference. Here follows the findings:

Table 74: The presence of pregnancy or delivery complications among the individuals and their newborns of the different population groups.

Pregnancy or delivery complications *	Subjects (n=15)	Controls (n=15)	Total (n=45)
Yes	6	4	12
No	9	11	33

*The complications experienced were: emergency caesarian sections, fetal distress, pregnancy induced hypertension, intra uterine growth retardation (IUGR), cord around neck, gestational diabetes mellitus.

The paediatric doctor also made mention in his report if he found a physical or mental problem which he thought needed attention, which was not necessarily related to alcohol. The researcher tested whether the difference in incidence of such problems between the subject and control populations were statistically significant; unfortunately they were not. Here follows the findings:

Table 75: The presence of physical defects among the newborns of the different population groups.

Physical defects *	Subjects (n=15)	Controls (n=15)	Total (n=45)
Yes	4	5	13
No	11	10	32

* The physical defects found were: heart murmurs, hirsutism, strabismus, oral thrush, seborrhoeus dermatitis, bulbous impetigo, peripheral pulmonary artery stenosis, torticollis.

Other findings that the researcher thought necessary to mention were:

Table 76: Other findings found among the newborns of the different population groups.

Findings:	Subjects (n=15)	Controls (n=15)	Total (n=45)
Alcohol related signs present, but no FAS or "deferred" diagnosis	3	5	11
Growth deficiency	0	1	4
Concern regarding neurodevelopmental progress *	1	0	1

* This newborn was diagnosed with FAS and was < 3rd percentile weight for age.

One mother had a termination when an ultrasound investigation showed a case of possible spina bifida. A full investigation was done, which confirmed this.

The researcher wanted to determine whether there was any statistical significance between the above-mentioned data of the newborn. Of the total population (n=45), the following results were found:

Table 77: Comparison of weight for age classification to the birth weight classification of the newborns.

Birth weight classification	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
LBW	3	7	1	0
Normal	1	15	17	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0127$.

Table 78: Comparison of weight for age classification to the gestational age at birth of the newborns.

Gestational age at birth (in weeks)	Weight for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
31	2	0	0	0
32	0	0	1	0
34	0	1	1	0
36	1	0	0	0
Term	1	21	16	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0062$.

Table 79: Comparison of weight for age classification to the "FAS status" of the newborns.

FAS status	Weight for age (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
"deferred"	1	6	4	0
FAS	2	1	0	0
Normal	1	15	14	1

Table 80: Comparison of head circumference for age classification to the birth weight classification of the newborns.

Birth weight classification	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
LBW	2	7	2	0
Normal	0	11	22	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0041$.

Table 81: Comparison of head circumference for age classification to the gestational age at birth of the newborns.

Gestational age at birth (in weeks)	Head circumference for age classification (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
31	2	0	0	0
32	0	1	0	0
34	0	0	2	0
36	0	1	0	0
Term	0	16	22	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0030$.

Table 82: Comparison of head circumference for age classification to the "FAS status" of the newborns.

FAS status	Head circumference for age (in percentiles)			
	< 5	5 - 50	50 - 95	> 95
"deferred"	0	7	4	0
FAS	1	2	0	0
Normal	1	9	20	1

* The statistical significance according to the Fischer's Exact test is $p < 0.0471$.

Table 83: Comparison of newborns' FAS status with birth weight

FAS status	Birth weight	
	Normal birth weight	Low birth weight
"deferred"	6	5
FAS	2	1
Normal	26	5

Table 84: Comparison of newborns' birth weight with their gestational age at birth

Gestational age at birth:	Birth weight	
	Normal birth weight	Low birth weight
Term	32	7
Premature	2	4

Table 85: Comparison of newborns' gestational age at birth with their FAS status

FAS status	Gestational age at birth	
	Term	Premature
"deferred"	10	1
FAS	2	1
Normal	27	4

7.2 Comparison of newborn and mother's data

All anthropometric data and birth related data of the newborn was compared with the mother's relevant nutrititional status parameters.

7.2.1 Newborn data compared to the mother's dietary intake

The mothers' dietary intake was divided into < 66.67% RDA (low intake), 66.67 – 133.33% RDA (adequate intake) and > 133.33% RDA (high intake) groups of the average of the three 24 hour recalls, and the average of the two FFQ's. When comparing the newborns' weight for age, HC for age and birth weight with the mothers' dietary intake, no statistical significance was found according to the Fischer's Exact test.

When comparing the newborns' gestational age at birth with the mothers' dietary intake, a statistical significance was found with energy of the 24 hour recalls.

Table 86: Comparison of the mother's energy intake to the newborn's gestational age at birth.

Gestational age at birth in weeks	Energy (< 66.67% RDA)	Energy (66.67 – 133.33% RDA)	Energy (> 133.33% RDA)
31	1	0	0
32	0	0	0
34	0	0	2
36	0	1	0
Term (> 38 weeks)	7	28	2

* Statistical significance according to Fischer's Exact test is $p<0.0083$.

When comparing the newborns' alcohol related signs status (FAS, "deferred", normal) to the mothers' dietary intake, no statistical significance was found.

Table 87: Comparison of the nutrient intake of the mothers of newborns with different "FAS statusses"

NUTRIENTS' mean values in %	**Normal offspring** (n=31)		**"deferred" offspring** (n=11)		**FAS-affected offspring** (n=3)	
	24 hour recall	FFQ	24 hour recall	FFQ	24 hour recall	FFQ
Energy	95.80	142.32	94.83	168.59	63.90	130.31
Protein	132.99	162.39	131.17	197.46	97.97	167.47
Vitamin A	70.67	201.22	122.87	197.65	44.79	320.50
Vitamin D	34.32	63.04	58.21	82.06	54.09	113.65
Vitamin E	103.59	156.88	94.15	190.47	56.10	165.17
Vitamin K	67.01	121.90	71.59	116.79	31.47	151.11
Vitamin C	191.22	344.04	109.88	298.77	38.89	175.57
Thiamin	70.61	109.09	61.63	119.82	37.88	98.91
Riboflavin	90.47	156.79	71.89	176.56	47.42	159.77
Niacin	122.77	141.66	111.38	160.76	80.03	140.55
Vitamin B6	75.21	107.63	70.84	120.46	42.74	97.25
Folate	57.48	102.97	53.89	107.39	33.82	110.54
Vitamin B12	174.68	305.74	199.26	407.09	116.42	487.66
Calcium	51.19	90.11	43.34	105.22	31.65	84.29
Phosphorous	90.96	135.23	87.81	168.92	64.46	135.43
Magnesium	80.61	129.61	78.81	152.64	59.05	127.19
Iron	35.41	55.55	31.41	57.62	21.53	35.75
Zinc	71.31	94.59	67.64	115.02	53.16	92.11
Iodine	25.96	37.48	29.36	52.21	25.89	51.86
Selenium	81.70	115.03	94.49	140.44	72.64	115.36

The researcher was also interested in the comparison of the mean dietary intake of mothers whose newborns were normal weight compared to those whose newborns were of LBW. As seen below, there was no significance between the difference of most mean dietary values, except in the case of vitamin D, according to the Kruskal-Wallis test.

Table 88: Comparison of mothers' nutrient intake of newborns of different birth weights.

NUTRIENTS' mean values expressed as %	**LOW BIRTH WEIGHT** (<2500 g) (n=11)		**NORMAL BIRTH WEIGHT** (>2500g) (n=34)	
	24 hour recall	**FFQ**	**24 hour recall**	**FFQ**
Energy	96.05	135.69	93.51	150.77
Protein	134.73	158.92	138.56	176.49
Vitamin A	115.08	233.81	77.02	202.94
Vitamin D	56.43*	73.27	37.10*	69.43
Vitamin E	133.39	160.29	94.05	166.74
Vitamin K	72.96	156.42	68.79	113.43
Vitamin C	196.97	325.71	159.12	329.39
Thiamin	66.49	99.12	70.44	116.65
Riboflavin	101.03	144.80	86.63	170.36
Niacin	122.46	126.26	124.72	153.78
Vitamin B6	7.45	97.16	77.02	115.92
Folate	64.78	102.39	55.95	105.96
Vitamin B12	209.15	332.08	179.91	346.40
Calcium	61.63	87.14	48.55	97.72
Phosphorous	99.56	133.36	91.72	148.63
Magnesium	89.29	126.79	81.11	139.71
Iron	35.89	54.06	35.44	57.35
Zinc	73.99	92.19	73.82	102.92
Iodine	34.28	44.95	25.85	41.36
Selenium	91.62	111.37	86.24	124.89

* There is a statistical significance according to Kruskal-Wallis test ($p<0.0288$).

The mother who had a termination because her fetus was diagnosed with spina bifida, had a normal to high intake of folate according to the one 24 hour recall (81% RDA) and food frequency questionnaire (144%).

7.2.2 Newborn data compared to the mother's anthropometric data

The mothers' anthropometric assessment (includes BMI classification, MUAC classification and weight gain during pregnancy) was compared with those of the newborns for any possible significance. There was a significant relationship between the mothers' weight gain and the newborns' weight for age classification. The full results are given in **3.4**.

7.2.3 Newborn data compared to the mother's clinical data

As discussed in **6.4**, there was no interesting significant relationship between the newborns' anthropometric data and FAS status, and the mothers' clinical data.

7.2.4 Newborn data compared to the mother's biochemical data

There are several nutrients which when inadequately consumed during pregnancy, can adversely affect the pregnancy and pregnancy outcome. Of the larger scope of nutrients investigated in the dietary assessment of the mothers, four nutrients were singled out to be biochemically investigated. Folate, zinc, ferritin and vitamin A were chosen since there is so much supporting literature suggesting that inadequate intake (leading to low biochemical values) of them could lead to adverse pregnancy and pregnancy outcomes such as LBW, prematurity and PIH.

However, there was no statistical significance between the above mentioned biochemical values and any of the newborn statistics.

7.2.5 Newborn data compared to the mother's questionnaire data

The researcher was interested in the interaction of smoking by the mother (see *Table 65*) and the effect of it on the newborn. Smoking (and how much) versus non-smoking will be compared with the incidence of LBW and the newborn's FAS status.

Table 89: The incidence of low birth weight and normal birth weight of the newborn compared with the smoking status of the newborn's mother.

Birth weight:	Smoking status:		
	Non-smoker	1-4 cigarettes/day	5-10 cigarettes/day
Low birth weight	1	6	4
Normal birth weight	13	8	13

Table 90: The smoking status of the mother compared with the FAS status of her newborn.

FAS status:	Smoking status:		
	Non-smoker	1-4 cigarettes/day	5-10 cigarettes/day
Normal	13	7	11
"deferred"	1	6	4
FAS	0	1	3

There was no significance to the above data.