DIETARY ADEQUACY, VARIETY AND DIVERSITY AND ASSOCIATED FACTORS (ANTHROPOMETRY AND SOCIO-ECONOMIC STATUS) IN PREGNANT WOMEN ATTENDING THE BISHOP LAVIS MOU IN CAPE TOWN

By

Celeste Marinda de Bruyn, BSc Dietetics

DBRCEL001

SUBMITTED TO THE UNIVERSITY OF CAPE TOWN

In fulfilment of the requirements for the degree

MSc (Med) in Dietetics

Faculty of Health Sciences
UNIVERSITY OF CAPE TOWN

Date of submission: August 2015

Supervisor:
Dr Janetta Harbron, Division of Human Nutrition, Department Human Biology, UCT

Co-supervisors:
Prof Marjanne Senekal, Division of Human Nutrition, Department of Human Biology, UCT
Prof Hein J Odendaal, Department of Obstetrics and Gynaecology, Stellenbosh University

DECLARATION

I, *Celeste Marinda de Bruyn*, hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

I empower the university to reproduce for the purpose of research either the whole or any portion of the contents in any manner whatsoever.

Signature: Signed by candidate .....

Date: *16 August 2015*

ABSTRACT

Aim: The aim of this study was to determine the adequacy of the dietary intake of pregnant women attending Bishop Lavis MOU, in the Tygerberg area of Cape Town.

Methods: One hundred and fifty-two women between 12 and 20 weeks' gestational age participating in the Main PASS study were recruited. They completed three interviewer-administered 24-hour dietary recall assessments on three different days, each approximately two weeks apart. Dietary reference values for adequate nutritional intake during pregnancy and the South African food based dietary guidelines and NARs and MAR were used to assess the nutritional adequacy. Anthropometric and socio-demographic information was also collected.

Results: The results indicate that just over a quarter of the sample were classified as teenage pregnancies. The majority had between grade eight and ten, and had a monthly household income between R500 and R5 000. With a mean energy intake of 10 168.4kJ, majority (79.5%) of the study sample did not meet the energy DRI. Close to half (42.8%) of the study sample did not meet the DRI for protein intake. All participants met the carbohydrate EAR, and many exceeded the recommended fat intake. The intake of sugar and saturated fats exceeded recommendations with sugar contributing to almost half of the total energy from carbohydrates. The intakes of vitamin A, D and E, pantothenate, biotin, folate, calcium, iron, magnesium, potassium, and manganese fell below the recommendations. Sugar was the most commonly consumed food item, followed by potato, chicken, milk, and white bread. Apples were the most commonly consumed fruit. When compared to the FBDG, the study sample consumed double the recommended portions of starch, half the recommended daily fruit and vegetables, and half the recommended legumes.

Conclusion: The high intake of refined carbohydrates, especially sugar, and the high intake of foods high in saturated fats needs to be addressed. Micronutrient intake is generally poor, especially with nutrients that are vital to proper growth and development of the foetus. Education on appropriate dietary changes, as well as suggestions to make implementation of such changes affordable would be invaluable, and may contribute towards decreasing the incidence of adverse pregnancy outcomes.

ACKNOWLEDGEMENTS

To all the amazing people who have supported me through this journey; all my family, friends and supervisors, the UCT administration team, my employers, and strangers that I met along the way – a sincere and heartfelt thank you for you support, encouragement, patience, assistance and friendly smiles.

Each one of you played a special part in helping me reach this end.

"I pass with relief from the tossing sea of Cause and Theory
to the firm ground of Result and Fact."

— Winston S. Churchill, The Story of the Malakand Field Force

CONTENTS

LIST OF TABLES AND FIGURES

LIST OF ABBREVIATIONS

ADA	American dietetics association
ADH	Alcohol dehydrogenase
ADHD	Attention deficit hyperactivity disorder
AFA	Arm fat area
AI	Adequate intake
AMA	Arm muscle area
AMC	Arm muscle circumference
AMDR	Acceptable macronutrient distribution range
ATP	Adenosine triphosphate
BMI	Body mass index
cAMA	Corrected arm muscle area
CDC	Centre for disease control
CP	Cerebral palsy
CRIBSA	Cardiovascular risk in black South Africans
CRP	C-reactive protein
CVD	Cardiovascular disease
DAEK	Dietary assessment and education kit
DDS	Dietary diversity score
DHA	Docosahexaenoic acid
DHIS	District health information software
DLW	Douby labelled water
DM	Diabetes mellitus
DNA	Deoxyribonucleic acid
DoH	Department of health
DRI	Dietary reference intakes
EAR	Estimated average requirements
EER	Estimated energy requirements
EI	Energy intake
ELBW	Extremely low birth weight (birth weight <100g)
FAD	Flavin adenine dinucleotide
FAS	Foetal alcohol syndrome
FASD	Foetal alcohol spectrum disorder
FBC	Full blood count
FFQ	Food frequency questionnaire
FMN	Flavin adenine mononucleotide

GDM	Gestational diabetes mellitus
GGT	Gamma-glutamyl transferase
GI	Glycaemic index
Hb	Haemoglobin
hCS	Human chorionic somatotropin
IBD	Irritable bowel disease
IDA	Iron deficiency anaemia
IGF-1	Insulin-like growth factor-1
IQR	Interquartile range
IU	International unit
IUGR	Intr-uterine growth restriction
IVH	Intraventricular haemorrhage
kg	Kilogram
kJ	Kilojoule
LBW	Low birth weight (birth weight <2 500g)
LC-PUFA	Long chain polyunsaturated fatty acids
LDL	Low density lipoprotein cholesterol
LGA	Large for gestational age (birth weight >90th percentile
MAR	Mean adequacy ratio
MEOS	Microsomal ethanol oxidising system
MOU	Maternity and obstetrics unit
MRC	Medical research council
MUAC	Mid-upper arm circumference
MUFA	Monounsaturated fatty acids
n-3	Omega-3
n-6	Omega-6
NAD	Nictotinamide adenine dinucleotide
NADPH	Nictotinamide adenine dinucleotide phosphate
NAR	Nutrient adequacy ratio
NCD	Non-communicable disease
ND	Not determinable
NEC	Necrotising enterocolistis
NFCS	National food consumption survey
NHMRC	National health and medical research council
NRC	National research council
NS	Not significant
NTD	Neural tube defects

NVP	Nausea and vomiting of pregnancy
P REE	Pregnancy resting energy expenditure
PA	Physical activity
PAL	Physical activity level
PASS	Prenatal alcohol in sudden infant death syndrome and stillbirth
PIH	Pregnancy induced hypertension
PSEA	Portion size estimation aid
PUFA	Polyunsaturated fatty acids
QFFQ	Quantitative food frequency questionnaire
RCF	Red cell folate
RDA	Recommended dietary allowances
RDS	Respiratory distress syndrome
REE	Resting energy expenditure
RNA	Ribonucleic acid
SADHS	South Africa demographic and health survey
SAFBDG	South African food based dietary guidelines
SANHANES	South African national health and nutrition examination survey
SD	Standard deviation
SES	Socio-economic status
SF	Serum folate
SFA	Saturated fatty acid
SGA	Small for gestational age (birth weight <10th percentile)
SIDS	Sudden infant death syndrome
SSB	Sugar sweetened beverage
TC	Total carbohydrates
TE	Total energy
TEE	Total energy expenditure
TF	Total fat
TFA	Trans fatty acids
TFib	Total fibre
TP	Total protein
TS	Total sugar
TSF	Triceps skinfold
UK	United Kingdom
UL	Upper limit
UNICEF	United Nations children's fund
USDA	United States department of agriculture

VAD	Vitamin A deficiency
VB12	Vitamin B12
VLBW	Very low birth weight (birth weight <1500g)
WHO	World health organisation

CHAPTER ONE

INTRODUCTION

1.1 Motivation for study

Maternal mortality remains a major health problem, particularly in the developing world (WHO 2014; Brabin et al., 2001). The maternal mortality rate in many parts of Africa is as high as 510 per 100 000 live births, while the rate in South Africa was reported to be 154 per 100 000 live births in 2013, and 79 per 100 000 in the Western Cape for the same year (Saving mothers, 2014). Developed countries have lower maternal mortality rates, for example 18.5 deaths per 100 000 live births as reported for the United States (UNICEF, 2014). It has been indicated that the following factors may increase risk for maternal mortality: abortion, hypertensive disorders, haemorrhage, sepsis, maternal infectious diseases, other maternal diseases, and labour complications (Bomela, 2015). While malnutrition is not classified as a direct predictor of maternal mortality, it does increase the risk for several of the causes. In South Africa, 8% of live births were born preterm, compared to the world wide rates of more than 10% (March of Dimes, 2012). Rates of low birth weight (LBW) in South Africa were reported at 13.5% (DHIS, 2012) while world rates were around 14% (UNICEF, 2009).

Various factors lead to poor pregnancy outcome including maternal age at time of pregnancy, inadequate prenatal care, lifestyle factors, physical insults to the mother, genetic predispositions to various diseases or disorders, and malnutrition either before or during the pregnancy (Wardlaw & Kessel, 2002). It is undisputed that adequate nutritional intake during pregnancy is important for optimal health outcomes for mother and infant (NHMRC, 2013; Wu et al., 2004). For example, insufficient or excessive dietary intake during pregnancy may lead to inadequate weight gain which can increase risks for several adverse outcomes including gestational and type II diabetes, pregnancy induced hypertension (PIH) and miscarriage (Margerison Zilko et al., 2010; Artal et al., 2010) in the mother, and small for gestational age (SGA) and LBW in the infant, and asthma and obesity in their later life (Mamun et al., 2014; Forno et al., 2014; Ohkawa et al., 2010; Han et al., 2010). Furthermore, inadequate intake of specific nutrients can cause several complications including maternal mortality (WHO, 2011), spontaneous abortions (Kotha & De Souza, 2014) and preeclampsia (Erick, 2012) in the mother, and neural tube defects (NTD) (Gallagher, 2012; Shaw et al., 2009), poor foetal brain development (Zeisel, 2013) and preterm delivery (Molloy et al., 2008) in the infant. A common example includes the inadequate intake of iron or folate that can lead to iron deficiency anaemia in the mother or poor growth and development of the foetus; or excessive or inadequate weight gain that has numerous effects on the infant and the mother (Thompson & Manore, 2005; Erick, 2008; Scholl & Reilly, 2000).

No studies on the dietary intake of pregnant women in South Africa from a national representative sample have been conducted. Futhermore, a limited number of studies could be traced that investigated the dietary intake and nutritional status of pregnant women in smaller, select samples in various regions in South Africa (Bopape et al., 2008; Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Tshitaudzi, 2003; Klinger, 2004; Mamabolo et al., 2004). These studies point out that the dietary intakes of iron, folate, vitamin C and B12, as well as meat and vegetable food groups in pregnant women may be inadequate, with possible further concerns of inadequate calcium and zinc. The majority of these studies used the RDA as their reference range, which has been shown to seriously overestimate the risk of inadequacy of group intakes (Murphy & Poos, 2002). It should be noted that four of the studies were done in various areas in Limpopo (Bopape et al., 2004; Mostert et al., 2005; Tshitaudzi, 2003; Mamabolo et al., 2004), while only two studies were conducted on women in the Western Cape attending the Paarl hospital (Jaffer et al., 2008), and Hanover Park maternal and obstetrics unit (MOU) (Klinger, 2004). Furthermore, the study by Klinger (2004) focussed on the effects of alcohol intake on pregnancy and not specifically on dietary intake. Therefore, more data is needed on overall dietary intake of pregnant women in the Cape Town area that uses the most recent reference ranges and recommendations. It is important to investigate and understand the current dietary intake of a target population in order to develop and successfully implement intervention strategies to address identified problem areas.

The Prenatal Alcohol in SIDS and Stillbirth (PASS) study is a multidisciplinary prospective study investigating various environmental, genetic, autonomic, neurobehavioral, maternal and placental factors associated with foetal and infant mortality related to sudden infant death syndrome (SIDS) and stillbirth as well as poor pregnancy outcomes such as foetal alcohol syndrome (FAS). The study is overseen by the PASS Network and aims to compare data from pregnant women in the Northern Plains area of the United States with pregnant women attending the Bishop Lavis MOU in Cape Town, South Africa. Data has been collected in these two regions over the past seven and a half years. Further details on this study are elaborated on in other papers (Dukes et al., 2014). As nutritional status may influence the association between the above-mentioned variables and pregnancy outcome it was deemed important to assess the dietary intake of the pregnant women. The PASS study thus provided an excellent opportunity to further explore the dietary intake of a sub-sample of women that participated in the main PASS study.

1.2 Aims and objectives

The **overarching aim** of this research is to investigate the dietary adequacy, variety and diversity and associated factors (anthropometry and socio-economic status) in pregnant women attending the Bishop Lavis MOU in Cape Town.

For these purposes three specific aims and several objectives for each aim have been formulated as outlined below.

Aim 1:

To assess and describe the following **socio-demographic and anthropometric** data for pregnant women in the total sample and for adolescent and adult participants:

The specific objectives that were formulated for Aim 1 include:

- To assess and describe socio-demographic data including maternal age, years of formal education, monthly household income and number of people supported by income;
- To measure and describe anthropometric data at recruitment including weight, height, Triceps skinfold measures (TSF) and mid-upper arm circumference (MUAC);
- To obtain self-reported pre-pregnancy weight;
- To calculate the arm fat area (AFA) and corrected arm muscle area (cAMA);
- To calculate and describe the pre-pregnancy BMI, recruitment BMI and weight change from pre-pregnancy to recruitment;
- To investigate associations between socio-demographic and anthropometric data.
- To compare the socio-demographic and anthropometric data of adolescent and adult participants in the sample.

Aim 2:

To investigate the **adequacy** of dietary intake of pregnant women in the total sample as well as for adolescent and adult participants using the average of three 24-hour recalls:

The specific objectives that were formulated for Aim 2 include:

- To assess and describe the dietary intake of energy, macro- and micronutrients.
- To compare actual energy intake with current dietary standards for pregnant women e.g. the estimated energy requirements (EERs);
- To compare actual intakes of carbohydrates, proteins, vitamins and minerals with current dietary standards for pregnant women e.g. estimated average requirements (EARs) and adequate intakes (AIs);

- To calculate and describe the macronutrient distribution as a percentage of total energy intake;
- To compare the macronutrient distribution as a percentage of total energy intake with the recommended macronutrient distribution ranges;
- To compare the intake in number of portions of different food groups with the recommendations outlined by the South African Food Based Dietary Guidelines (SAFBDG);
- To calculate and describe the nutrient adequacy ratio (NAR) of each nutrient consumed and the mean adequacy ratio (MAR) of intakes;
- To compare all above-mentioned dietary intake variables between adolescent and adult participants in the study sample;
- To investigate the association between the different dietary intake variables;
- To investigate associations between dietary intake variables (energy, macronutrients, micronutrients, NAR and MAR) and socio-demographic variables;
- To investigate associations between dietary intake variables (energy, macronutrients, micronutrients, NAR and MAR) and anthropometric variables.

Aim 3:

To investigate and describe the **variety** of dietary intake of pregnant women in the total study sample and for adolescent and adult participants using the average of three 24-hour recalls.

The specific objectives that were formulated for Aim 3 include:

- To compile three different lists of food items with each list consisting of the top 20 most commonly consumed food items, fruits, and vegetables respectively;
- To compile six different lists of food items with each list consisting of the top ten foods contributing to intakes of total energy, protein, carbohydrates, fiber, fat and added sugar respectively;
- To calculate and describe the dietary diversity score (DDS).

1.3 Outline of thesis

Chapter two of this thesis contains a review of literature covering various aspects of pregnancy, including foetal development and the accompanied nutritional needs, and the assessment of nutritional status. Chapter three covers the methods used throughout the sub-study. Chapters four, five, and six each include a brief introduction and cover the results and discussion of socio-demographic and anthropometric findings, dietary adequacy, and dietary variety. These chapters do

not expand again on methodologies used. The final chapter summarises these findings, and presents possible workable solutions to the imbalances in nutritional intake found by the study.

1.4 Candidate's contribution

The thesis candidate was responsible for:

- Conducting all 480 interviews including three separate 24-hour recalls, and between two and three food frequency questionnaires (FFQs) for each participant
- Arranging all follow-up visits for participants of the sub-study
- Checking through data captured and exported to Microsoft Excel spreadsheets, and highlighting and fixing errors
- Assisting MRC statistician with running statistical analyses of raw data
- Running statistical tests and comparisons on analysed data and compiling and analysing all tables generated from these assessments
- Compiling all chapters of the thesis

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

This literature review covers various dietary and nutrition related aspect of pregnancy, starting with a discussion on the physiological aspects of pregnancy, including metabolism of various nutrients and alterations in metabolism that take place, as well as other physiological changes that occur in a pregnant woman's body. The energy and nutrient requirements of pregnant women are then discussed, alluding briefly to supplementation use. This is followed by an in-depth critical discussion of studies that have investigated dietary intake in pregnant women. An overview of the nutrition related complications during pregnancy and nutrition related pregnancy outcomes are also presented. Methodologies for nutrition status assessment and interpretation of dietary intake data including dietary adequacy, diversity and variety are also discussed.

2.2 Physiological changes in pregnancy

2.2.1 Non-metabolic changes in pregnancy

The physiological changes that occur in the mother during pregnancy (Table 2.1) can be divided into two stages with the first being a "maternal anabolic phase" and the second being a "maternal catabolic phase". The anabolic phase is geared towards preparing the mother's body to be able to deliver increased quantities of blood, oxygen and nutrients to the foetus in the second half of pregnancy, while the "catabolic phase" is aimed at delivering the energy and nutrients stored in the first half of pregnancy to the foetus (Brown, 2008; Lof et al., 2005). These changes are essential for preparing the mother for birth and for supporting and protecting the foetus (Carlin & Alfirevic, 2008). The sequence of events is crucial, with each step being dependent on the successful completion of the one before it (Brown, 2008). Poor nutrition at any one point can have severe repercussions for the foetus (Christian & Stewart, 2010).

Several hormonal changes take place throughout pregnancy as the mother's body adapts to the growing foetus. The placenta is the major site for production of these hormones (Erick, 2012). Increased secretion of progesterone and oestrogen promote insulin production and conversion of glucose to glycogen and fat to be stored for use by the mother in the second, more catabolic phase of pregnancy, and during breastfeeding (Brown, 2008; Lof et al., 2005). Progesterone also affects the muscles of the intestine resulting in increased transit times and increased constipation and bloating (Erick, 2012; Prather, 2004) while also being responsible for the decreased efficiency of gall bladder emptying (Erick, 2012; Portincasa et al., 2008). The systematic secretion of hormones ensures a suitable environment for foetal development by adequately preparing the mother's body.

TABLE 2.1 *Summary of key physiological changes that take place in a pregnant woman*

	CHANGE	REASON AND/OR EFFECT	REFERENCE
Cardiovascular system			
Cardiac output	↑ by 20-50%	↑, peaking by end of 2nd trimester, ↓ end of 3rd trimester	Liu & Arany, 2014; Carlin & Alfirevic, 2008; Brown, 2008
Blood and plasma volume	20% and 50% expansion respectively	↓ haemoglobin, water-soluble vitamin and serum protein concentrations ↑ fat-soluble vitamins, triglycerides, cholesterol and free fatty acid blood concentrations	Liu & Arany, 2014; Erick, 2012; Brown, 2008
Heart rate	↑ 15-30% by 3rd trimester	↑ as systemic vascular resistance ↓ with peripheral vasodilation	Liu & Arany, 2014; Moertl et al., 2009; Carlin & Alfirevic, 2008
Blood pressure	Diastolic ↓10% in the first eight weeks; ↑ in 3rd trimester	Secondary to peripheral vasodilation; returns to pre-pregnancy levels by term	Liu & Arany, 2014; Brown, 2008; Carlin & Alfirevic, 2008; Moertl et al., 2009
Respiratory system			
Oxygen requirements	↑	Maternal threshold of PCO_2 ↓ in relation to that of the foetus (possibly due to ↑ progesterone) which creates a more effective transfer of oxygen to the foetal blood system	Erick, 2012 ; Carlin & Alfirevic, 2008; Alaily & Carrol, 1978; Wilbrand & Porath, 1952
Oxygen consumption	↑ 30-50 mL/min		
	Dyspnoea	In later pregnancy the growing uterus pushes the diaphragm upwards causing dyspnoea	Carlin & Alfirevic, 2008; Erick, 2012
Renal changes			
Glomerular Filtration Rate	↑ 40-50% by end of 1st trimester	Compensates for ↑ blood volume resulting in ↓ serum urea and creatinine levels, and ↑ urinary glucose levels (↑ risk of urinary tract infections)	Carlin & Alfirevic, 2008; Erick, 2012; Brown, 2008
	Sodium and water retention	Caused by angiotensin II resistance and ↓ renal tubular absorption resulting in ↑extracellular water volume	Lindheimer & Barron, 1998
Gastrointestinal changes			
	Nausea and vomiting	Occurs in 70% and 40% of pregnancies respectively in 1st trimester	Nulman et al., 2009; Brown, 2008
	Reflux and heartburn	↑ uterus size and relaxing of sooth muscles caused by ↑ progesterone	DallAlba et al., 2010; Carlin & Alfirevic, 2008
	Constipation	↓ gastric motility due to ↑ progesterone levels	Cullen & O'Donoghue, 2007

↑ = Increase ↓ = Decrease

2.2.2 Physiology of foetal development

Conception and foetal development

A full term pregnancy lasts between 38 and 42 weeks (or an average of 280 days) and is measured from the first day of the last menstrual bleeding (menstrual age) (Brown, 2008). The process of foetal growth and development involves hyperplasia (increased cell multiplication), hypertrophy (growth of cells) and maturation (stabalisation of cell numbers and sizes) (Brown, 2008). The periods of hyperplasia occur mostly in the first two months of pregnancy when the majority of organogenesis takes place (Brown, 2008; Moore & Persaud, 1998; Rozovski & Winick, 1979). These are the critical periods in which major abnormalities can occur if there are any adverse

exposures, including under or over nutrition (Godfrey & Barker, 2000; Moore & Persaud, 1998) with possible lifelong implications. Figure 2.1 summarises the critical stages in foetal development.

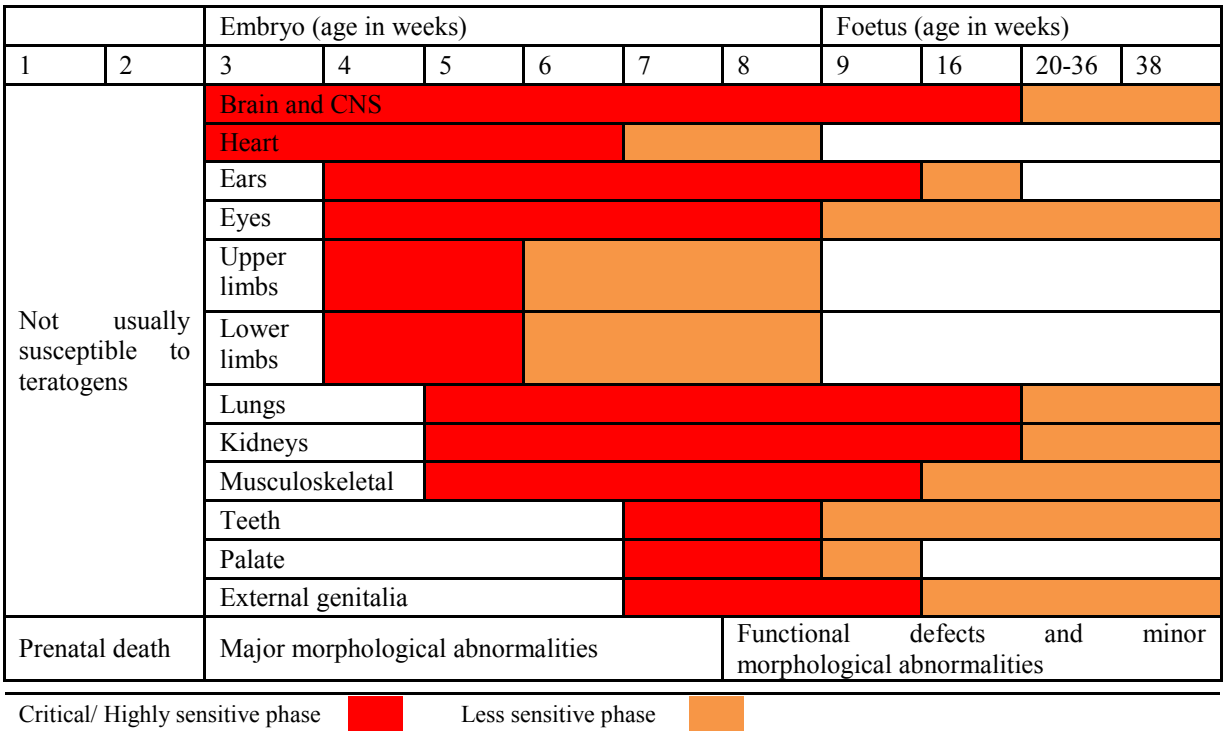

FIGURE 2.1 *Critical stages of development in the foetus* (Adapted from Moore & Persaud, 1998)

Throughout foetal development the brain and central nervous system take priority in terms of nutrient and oxygen supply, followed by the heart and adrenal glands (Gluckman, 2003). The cells of the central nervous system continue to multiply for about two years after birth with a further growth spurt during the adolescent growth spurt, making the cells susceptible to damage from poor diet into early childhood (Brown, 2008).

Any adverse effects during the hypertrophic period are associated with decreases in organ and tissue function. Unlike the consequences of adverse effects during critical periods of hyperplasia, these functional changes can be reduced or reversed if proper nutrition is attained (Rozovski & Winick, 1979).

Healthy foetal growth and weight gain

The majority of foetal growth occurs in the third trimester during the maternal catabolic phase (Brown, 2008; Erick, 2012). In the last three months of pregnancy the foetus should double in

length and gain close to three quarters of its own body weight at birth (Erick, 2012). The recommended rate of weight gain and growth per trimester are shown in Table 2.2.

TABLE 2.2 *Average weight and height of a healthy foetus by trimester* (Adapted from Brown, 2008; Gilbert-Barness & Debich-Spicer, 2005)

TRIMESTER	WEEKS	DAYS	WEIGHT (kg)	LENGTH (cm)
End of first	0-13	0-93	0.68-1.00	7.60-10.50
End of second	14-27	94-187	0.91-1.60	26.00-30.00
End of third	28-40	189-280	3.40-3.50	45.72– 55.88

Foetal composition

The majority of the weight gained in the foetus can be attributed to fat and proteins (Waters et al., 2012; Brown, 2008; Ziegler et al., 1976). Fat, vitamin, and mineral deposition in the foetus occurs mostly in the last trimester and is associated with maternal weight gain (Ziegler et al., 1976). Protein deposition occurs throughout pregnancy while the distinct increase in calcium concentration from week 32 to 40 indicates the growing bone mass and density of the foetus which occurs mainly in the third trimester (Ziegler et al. 1976).

2.2.3 Recommended weight gain for pregnant women

Weight gain during pregnancy is an important determinant of infant health and their health status later in life (Ehrenthal et al., 2014). Recommended maternal weight gain during pregnancy differs depending on pre-conception weight and nutritional status, as well as the number of foetuses carried (Table 2.3) (Ehrenthal et al., 2014; IOM, 2009). It is recommended that women be within their normal weight range before conception, and that weight loss should not be attempted during pregnancy (IOM, 2009).

TABLE 2.3 *Recommended weight gain for pregnant women based on pre-conception weight* (Institute of Medicine, 2009)

PRE-CONCEPTION BMI (kg/m^2)	**1st TRIMESTER (kg)**	**2nd AND 3rd TRIMESTER (kg/ week)**	**TOTAL WEIGHT GAIN (kg)**
<18.5 (Underweight)	0.5 – 2	0.45 – 0.59	12.7 – 18.2
18.5-24.9 (Normal weight)	0.5 – 2	0.36 – 0.45	11.4 – 15.9
25.0-29.9 (Overweight)	0.5 – 2	0.23 – 0.32	6.8 – 11.4
≥30 (Obese)	0.5 – 2	0.18 – 0.27	6.8 – 9
Twins			15.9– 20.5

Abbreviations: BMI = Body mass index

The total recommended weight gain during pregnancy is attributable to various components (Table 2.4). The weight of the foetus comprises the largest component of maternal weight gain, while additional fat and protein stores are the second largest contributor. Failure of either of these components to increase sufficiently can have serious consequences for both mother and foetus (Calvo & Lopez, 2012).

TABLE 2.4 *Components of maternal weight gain* (Adapted from Calvo & Lopez, 2012; Erick, 2012)

COMPONENT	**WEIGHT (kg)**			
	10 weeks	**20 weeks**	**30 weeks**	**40 weeks**
Foetus	0.005	0.30	1.50	2.50 – 3.90
Amniotic fluid	0.03	0.35	0.75	0.80 – 1.00
Placenta and umbilical chord	0.02	0.17	0.43	0.67 – 0.70
Uterus	0.14	0.32	0.60	0.90 – 1.12
Blood	0.01	0.06	1.30	1.20 – 1.80
Extracellular fluids	0.00	0.27	0.80	1.20 – 3.20
Fat and protein stores	0.32	2.14	3.64	3.40 – 3.50
Breast tissue	0.05	0.18	0.36	0.45 – 0.50
TOTAL	**0.575**	**3.79**	**9.38**	**12.16 – 15.62**

2.3 Nutrition and pregnancy

2.3.1 Daily nutrient requirements before and during pregnancy

Maternal nutritional requirements of macro- and some micronutrients increase during pregnancy. Meeting these requirements is crucial for proper growth and development of the foetus.

2.3.2 Energy, macronutrients and fluids

The energy and macronutrient dietary reference intakes (DRIs) for pregnant and non-pregnant women are summarised in Table 2.5 and discussed in the following sections.

TABLE 2.5 *Daily macronutrient Dietary reference intakess* (FNB of the IOM, 2005; 2002)

			NON-PREGNANT	1ST TRIMESTER	2ND TRIMESTER	3RD TRIMESTER
Energy - Adolescents	**kcal**	EER	±2368	±2368	±2708	±2820
- Adults			±2403	±2403	±2743	±2855
Protein	**g**	RDA	46	71		
	g/kg	EAR	0.66	0.88		
Carbohydrates	**g**	RDA	130	175		
	g	EAR	100	135		
Fibre	**g**	AI	25	28		
Fat – n-6	**g**	AI	12	13		
n-3	**g**	AI	1.1	1.4		
Total water	**L**	AI	2.7	3.0		

Abbreviations: EER = Estimated Energy Requirements AI = Adequate Intake RDA = Recommended Dietary Allowance EAR = Estimated Average Requirements

Energy expenditure: subtract 7kcal/day for each year of age above 19

Energy

Energy requirements for the first trimester are the same as for non-pregnant women but increase from the second trimester. This increase is attributed to new tissue creation (foetus, placenta, amniotic fluid), growth of existing maternal tissue, and extra fat deposition (Williamson, 2006). Energy expenditure also rises due to the 15% increase in metabolic rate that is typical of this trimester (Erick, 2012). Total energy requirements depend on the mother's pre-pregnancy BMI, her age, as well as the rate of weight gain during pregnancy (Kaiser & Allen, 2002).

Equations and tables have been compiled to calculate energy requirements during pregnancy (Hronek et al., 2009; IOM, 2005). Hronek et al. (2009) adapted the Harris Benedict equation to produce the resting energy expenditure during pregnancy (Pregnancy resting energy expenditure (P REE), see box below), but this was done only on women with normal pre-pregnancy BMI.

P REE = 346.43943 + 13.9625643 x W + 2.7004163 x H + 6.8263763 x A

W, weight (kg); H, height (cm); A, age (years)

The equation developed by the Institute of Medicine (2005) is based on the total energy expenditure (TEE) as determined by the doubly labelled water (DLW) technique on pregnant women. This equation also takes into account activity level and pre-pregnancy BMI as well as median change in TEE per trimester to account for increased fat and tissue deposition (Ritchie & King, 2008). Table 2.6 shows the steps involved in the energy calculation, while Table 2.7 shows pre-calculated estimates for women 30 years of age, with pre-pregnancy BMIs of 18.5 and 24.99kg/m^2.

TABLE 2.6 *Calculating EER for pregnant females* (Widen & Siega-Riz, 2010)

Assess PAL (kcal/day)	**Sedentary**	**Low activity**	**Active**	**Very active**
Girls 3 – 18 years	1.0	1.16	1.31	1.56
Women ≥19 years	1.0	1.12	1.27	1.45
Calculate non-pregnant EER				
9 -18 years	135.3 – (30.8 x age) + PAL x (10 x weight + 934 x height) + 25			
Women ≥19 years	354 – (6.91 x age) + PAL x (9.36 x weight + 726 x height)			
Calculate pregnant EER	**1st trimester**	**2nd trimester**	**3rd trimester**	
	Non-pregnant EER + 0	Non-pregnant EER + 340	Non-pregnant EER + 452	
Age in years, weight in kg, height in m				

Abbreviations: EER = Estimated Energy Requirements PAL = Physical Activity Level

TABLE 2.7 *Pre-calculated EER for pregnant women 30 years or older* (Ritchie & King, 2008, adapted from IOM 2005)

Height m (in)	PAL	**EER (kcal/day)** Pre-pregnancy BMI = 18.5 kg/m^2	Pre-pregnancy BMI = 24.99 kg/m^2
FIRST TRIMESTER			
1.50 (59)	Sedentary	1625	1762
	Low activity	1803	1956
	Active	2025	2198
	Very active	2291	2489
1.65 (65)	Sedentary	1816	1982
	Low activity	2016	2202
	Active	2267	2477
	Very active	2567	2807
1.80 (71)	Sedentary	2015	2211

Height m (in)	PAL	EER (kcal/day) Pre-pregnancy BMI = 18.5 kg/m^2	Pre-pregnancy BMI = 24.99 kg/m^2
	Low activity	2239	2459
	Active	2519	2769
	Very active	2855	3141
SECOND TRIMESTER			
1.50 (59)	Sedentary	1965	2102
	Low activity	2143	2296
	Active	2365	2538
	Very active	2631	2829
1.65 (65)	Sedentary	2156	2322
	Low activity	2356	2542
	Active	2607	2817
	Very active	2907	3147
1.80 (71)	Sedentary	2355	2551
	Low activity	2579	2799
	Active	2859	3109
	Very active	3195	3481
THIRD TRIMESTER			
1.50 (59)	Sedentary	2075	2212
	Low activity	2253	2406
	Active	2475	2648
	Very active	2741	2939
1.65 (65)	Sedentary	2266	2432
	Low activity	2466	2652
	Active	2717	2927
	Very active	3017	3257
1.80 (71)	Sedentary	2465	2661
	Low activity	2689	2909
	Active	2969	3219
	Very active	3305	3591
Add 7kcal/day for each year below 30			

Abbreviations: EER = Estimated Energy Requirements PAL = Physical Activity Level BMI = Body Mass Index

Protein

Substantial increases in protein requirements during pregnancy result from the increase in new tissue and muscle formation in both mother and foetus (Erick, 2008; Brown, 2008). The requirements increase as the pregnancy progresses (Williamson, 2006) from 0.66g/kg/day in the first trimester to 1.1g/kg/day, or 71g/day, for the second half of pregnancy (Otten et al., 2006; IOM, 2002). Additional protein is also required for multiple pregnancies (25g/day additional) (Erick, 2008; IOM, 2005). Current protein recommendations are based on the factorial method whereby protein needs are calculated from nitrogen balance data obtained from non-pregnant adults (Stephens et al., 2015; Pencharz et al., 2014). Stephens et al. (2015) concludes that these requirements are inadequate, and suggested an EAR of 1.22 and 1.52g/kg/day for early and late gestation respectively. These authors used the indicator amino oxidation method in healthy pregnant

women during either early or late pregnancy to determine protein needs. However, the study sample was relatively small (29 healthy women) and the suggestions by Stephens et al. (2015) need to be further tested before they can be considered for implementation. When considering protein requirements, it needs to be noted that physiological adaptation during pregnancy decreases nitrogen excretion and increases the conservation of amino acids (Brown, 2008).

All amino acids that are essential to the mother are also essential to the foetus. Several amino acids are neurotransmitters or precursors thereof, and an inadequacy or deficiency of a single amino acid can adversely affect the developing brain, and hinder protein synthesis in the foetus (Antonow-Schlorke et al., 2011; Fugelstad et al, 2008; Morgane et al., 2002). While all nutrients play some role in brain maturation, protein seems to be the most critical to development of neurological functions including motor and cognitive development (Antonow-Schlorke et al., 2011; Tolsa et al., 2004; Morgane et al., 2002), with the extent of the impact on behaviour and learning capabilities depending on the timing of the deficiency, and the type and severity of the deprivation (Morgane et al., 1993). Inadequate maternal protein intake can also affect embryo, foetal and DNA development negatively which may lead to insulin resistance and hypertension in later life (Sinclair & Watkins, 2013), as well a large number of other adverse outcomes (Imdad & Bhuta, 2012b). Inadequate protein intake has also been associated with intrauterine growth restriction (IUGR) and LBW (Sinclair & Watkins, 2013; Imdad & Bhutta, 2011; Tolsa et al., 2004).

Carbohydrates

An increase in carbohydrate intake from 130g to 175g per day is the minimum required to maintain blood glucose levels and to prevent ketosis in pregnancy (Erick, 2012; Kaiser & Allen; 2008). Ketones pass through the placenta and can cause foetal lactic acidosis and hypoxemia (Parker & Conway, 2007) and can lead to neurological impairment and decreased intellect (IQ) in later life (Parker & Conway, 2007; Churchill et al., 1969).

Carbohydrate containing food choices should focus on unrefined cereals, legumes and a limited amount of sugar and sugar containing items. This will contribute to micronutrient intake and ensure an adequate fibre intake that is specifically important during pregnancy and may help aid in alleviating constipation caused by the hormonal changes that occur in pregnancy (Rizotto et al., 2010; Williamson, 2006). Soluble fibre also helps in the control of postprandial blood glucose levels by slowing the absorption of nutrients (Rizotto et al., 2010). This can aid in counteracting the insulin resistance caused by the increase in human Chorionic Somatotropin (hCS) levels during

pregnancy (Brown, 2008). Counteracting insulin resistance is important as high blood glucose levels during pregnancy are known to contribute to excessive foetal size (Scholl et al., 2001). Consideration of the effect of carbohydrate sources on the blood glucose levels subsequent to consumption (the glycaemic index (GI) of a food) may also be helpful in controlling blood glucose levels (Louie et al., 2011).

High intake of sugar and sugar containing items may greatly increase the risk for excessive weight gain (Temple & Steyn, 2013). A large (121 000 men and women) prospective study done in the USA as well as a meta-analysis published in 2012 showed strong associations between weight gain and the intake of sugar sweetened beverages (SSBs), as well as the intake of added sugar (Te Morenga et al., 2012; Mozaffarian et al., 2011). Another meta-analysis highlights the role of SSBs in the aetiology of the metabolic syndrome as well as type 2 diabetes (Malik et al., 2010) emphasising the effect of sugar on glucose tolerance (Salas-Salvado et al., 2011), which further contributes to excessive weight gain (Mann, 2007).

Lipids

There is no evidence that the dietary fat requirements during pregnancy differ from that of non-pregnant women (Koletzko et al., 2007). According to Brown (2008) any increased requirements are met by the changes in fat metabolism that promote fat accumulation. The recommendation of 20-35% of daily energy requirements for non-pregnant women is thus applicable to pregnant women to prevent excessive weight gain, provide essential fatty acids and ensure adequate absorption of fat-soluble vitamins (Rizotto et al., 2010). The recommendations for cholesterol (<300mg), saturated fatty acids (SFA) (<10%TE) and trans-fatty acids (TFA) (<1% TE) (Smuts & Wolmarans, 2013) also remain the same.

The DRIs for omega-3 and omega-6 fatty acids for pregnant women are AIs of 1.4g/day and 13g/day respectively, with the AIs for non-pregnant women being 1.1g/day and 12g/day respectively (FNB of the IOM, 2005). The intake of the essential fatty acids omega-6 linoleic acid and omega-3 alpha-linolenic acid is important during pregnancy due to their likely role in the prevention of cancer, diabetes, cardiovascular disorders, obesity and steatosis in the adult offspring (Mennitti et al., 2015). Furthermore, the intake of omega-3 long-chain polyunsaturated fatty acids (LC-PUFA) is important for optimal brain and central nervous system development (Koletzko et al. 2014; Gould et al., 2013; Campoy et al., 2012; Schuchardt et al., 2010; Kris-Etherton & Innis, 2007; Hadders-Algra et al., 2007; Uauy & Dangour, 2006; Hibbeln et al., 2006; McCann & Ames, 2005;

Heird & Lapillonne, 2005). A dietary intake of n-3 LC-PUFA that provides at least 200mg/d of DHA is recommended during pregnancy (Koletzko et al., 2014), while intakes of up to 1 g/d DHA or 2.7g/d n-3 LC-PUFA have been shown to have no significant adverse effects in pregnant women (Koletzko et al., 2007). Maternal deficiency of LC-PUFAs increases the risk of impaired cognitive and behavioural performance (Innis, 2009). Although not yet conclusive, research has suggested a possible association between maternal omega-3 and -6 levels and the development of fat cells in the foetus where low maternal stores possibly increase the risk of childhood obesity (Muhlhausler et al., 2010; Ailhaud et al., 2006). In the mother, intakes of omega-6 linoleic acid and omega-3 alpha-linolenic acid have also been shown to regulate blood pressure, thrombotic aggregation, and inflammatory responses (Smuts & Wolmarans, 2013). The decline in PUFA concentration levels during pregnancy has been thought to contribute to the aetiology of depression in pregnancy (Horrobin & Bennett, 1999). The use of omega-3 supplements is discussed in section 2.3.6.

Blood lipid profiles of pregnant women change as pregnancy progresses with substantial increases in total cholesterol and triglyceride levels (Brown, 2008). The increased cholesterol supply allows for steroid hormonal synthesis by the placenta and nerve and membrane formation by the foetus (Butte, 2000).

Fluids

Fluid recommendations for a person should take into consideration the body size of the individual (Erick, 2008). The AI for pregnant women increases from the 2.7L/d recommended for non-pregnant women to 3.0L/d for total water (including fluid from foods) and from 2.2L/d to 2.3 L/d for drinking fluids (IOM, 2005; Procter & Campbell, 2014). Increased fluid intake is essential for the increase in blood volume that takes place, temperature regulation, metabolic processes, maintaining the amniotic fluid surrounding the foetus, preventing constipation and urinary tract infections (Brown, 2008; Erick, 2008). Inadequate fluid intake increases the risks of premature contractions (Stan et al., 2002) and reduced amniotic fluid volume (Margann, 2003). Slight oedema in pregnancy generally reflects healthy plasma volume expansion as long as it is not accompanied by hypertension (Brown, 2008).

2.3.3 Vitamins

The requirements for certain vitamins are increased during pregnancy to obtain an optimal pregnancy outcome (Erick, 2012; Trumbo et al., 2002). Because some vitamin and mineral requirements increase to a much greater extent than the overall energy requirements, especially for

the first trimester, the approach to dietary intake should be one of high nutrient density, rather than energy density (Cox & Phelan, 2008). Table 2.8 summarises the increases in vitamin requirements in pregnant women compared to non-pregnant women. A discussion on the vitamins that are important during pregnancy follow in the next sections.

TABLE 2.8 RDAs*, AIs, EARs and ULs of Vitamins for non-pregnant and pregnant women* (Gallagher, 2012; FNB of the IOM, 2006; Trumbo et al., 2002)

	Vitamin A (mcg RE)	**Vitamin C (mg)**	**Vitamin D (ug)**	**Vitamin E (mg)**	**Vitamin K (ug)**	**Thiamin (mg)**	**Riboflavin (mg)**	**Niacin (mg)**	**Vitamin B6 (mg)**	**Folate (ug)**	**Vitamin B 12 (mcg)**	**Pantothenic acid (mg)**	**Biotin (mcg)**	**Choline (mg)**
Pre-pregnancy (19 – 30 years of age)														
RDA/ AI	700	75	15	15	90*	1.1	1.1	14	1.3	400	2.4	5*	30*	425*
EAR	500	60	10	12	-	0.9	0.9	11	1.1	320	2.0	-	-	-
UL	3000	2000	100	1000	ND	ND	ND	35	100	1000	ND	ND	ND	3.5
During pregnancy (14 – 18 years of age)														
RDA/ AI	750	80	15	15	75*	1.4	1.4	18	1.9	600	2.6	6*	30*	450*
EAR	530	66	10	12	-	1.2	1.2	14	1.6	520	2.2	-	-	-
UL	2800	1800	50	800	ND	ND	ND	30	80	800	ND	ND	ND	3000
During pregnancy (19-30 years of age)														
RDA/ AI	770	85	15	15	90*	1.4	1.4	18	1.9	600	2.6	6*	30*	450*
EAR	550	70	10	12	-	1.2	1.2	14	1.6	520	2.2	-	-	-
UL	3000	2000	100	1000	ND	ND	ND	35	100	800	ND	ND	ND	3500

Abbreviations: AI = Adequate Intake RDA = Recommended Dietary Allowance EAR = Estimated Average Requirements UL = Upper Limit ND = Not determinable

AIs are marked with an asterisk *

Vitamin A

The requirements for vitamin A increase from 700mcg to 770mcg (from ≈2300 to 2500 IU) in pregnancy. Amounts exceeding 7 500mcg (25 000IU) per day have been shown to have teratogenic effects on the foetus (Gutierrez-Mazariegos et al., 2011; US Teratology Society, 1987). The absorption of fat-soluble vitamins depends on a minimum amount of dietary fat and on adequate bile salts and pancreatic juices (Gallagher, 2012).

Vitamin A plays a vital role in visual function as well as in growth, reproduction, normal immune function modulation and epithelial tissue maintenance. Its function in cell proliferation and organogenesis makes it important during pregnancy and early childhood when rapid proliferation occurs (Gutierrez-Mazariegos et al., 2011; Azaïs-Braesco & Pascal, 2000). Vitamin A also plays a role in immune function, and beta-carotene can act as an anti-oxidant (Gallagher, 2012; Stephenson, 2001). Foetal and breast milk stores of vitamin A are only accumulated in the third trimester. As a result, because vitamin A is also essential for lung maturation, infants born premature are at high risk for bronchopulmonary dysplasia due to insufficient vitamin A stores (Gutierrez-Mazariegos et al., 2011). Vitamin A deficiency also increases the risk of mortality from common childhood infections (Benn et al., 2015; Underwood, 1994).

A deficiency of Vitamin A in the mother can cause gestational night-blindness (in the mother), and may increase the risk of maternal mortality (WHO, 2011; WHO, 2009). It needs to be noted that Vitamin A Deficiency (VAD) in the mother does not necessarily lead to VAD in the infant, as preference for vitamin A is given to the foetus (Gutierrez-Mazariegos et al., 2011; Venkatachalam et al., 1962). However, where the space between pregnancies is very short, and in the case of multiple pregnancies, the risk of VAD in the infant is greater (Gutierrez-Mazariegos et al., 2011). VAD in children has been classified by the World Health Organisation as a moderate health problem in developing countries and is the leading cause of childhood blindness (WHO, 2009).

B Vitamins

Thiamin, riboflavin and niacin act as coenzymes in energy metabolism (Gallagher, 2012; Williamson, 2006) and thus their requirements increase towards the third trimester in line with the increase in energy requirements (Williamson, 2006). Thiamin is required for carbohydrate and energy metabolism and for neural function (Guilland, 2013; Gallagher, 2012). Thiamin deficiency in pregnancy may occur as a result of hyperemesis gravidarum, placing the mother at risk of Wernicke's encephalopathy (Saab et al., 2014) and could increase the risk of spontaneous abortion (Kotha & De Souza, 2014). Riboflavin is essential in energy production and metabolism through its coenzyme role in the production of flavin adenine mononucleotide (FMN) and flavin adenine dinucleotide (FAD) (Gallagher 2012), and a deficiency of riboflavin during pregnancy may have adverse effects on growth and development (Powers et al., 2011). Niacin is essential for energy metabolism and production at a cellular level through its function as a component of nictotinamide

adenine dinucleotide (NAD) and nictotinamide adenine dinucleotide phosphate (NADPH) (Gallagher, 2012).

The recommendation for vitamin B6 increases from 1.3mg to 1.9mg per day. Extra vitamin B6 is required for the increase in production of nonessential amino acids for growth in pregnancy (Erick, 2012). There is limited evidence that vitamin B6 aids in cases of severe nausea and vomiting (Matthews et al., 2010; Gallagher, 2012; Aikins, 1998).

Folate is well known for its role in lowering the risk of NTDs in those who are genetically susceptible to this condition (Cox & Phelan, 2008). The RDA for folate increases substantially from 400mcg to 600mcg per day as a result of the increased needs for DNA synthesis and cell replication (placental growth, neural tube development) (Gallagher, 2012; Brown, 2008, Tamura & Picciano, 2006). The first 28 days post conception are the most critical as it is within this time period that the neural tube forms and closes (Erick, 2012). Without supplementation, the blood folate concentrations have been shown to decline rapidly in pregnant women (Tamura & Picciano, 2006). Folate deficiency during pregnancy may also lead to anencephaly, low birth weight, preterm birth, growth faltering, preeclampsia and macrocytic anaemia in the mother (Erick, 2012; Scholtz et al., 2010; Brown, 2008; Tamura & Picciano, 2006). Folate depletion caused by short inter-pregnancy intervals also increases the risk of IUGR and LBW (van Eijsden et al., 2008).

Vitamin B12 requirements increase slightly during pregnancy from 2.4mcg to 2.6mcg daily. Its role in regeneration of the active form of folate aids in decreasing the risk of NTDs (Gallagher, 2012; Molloy et al., 2008). Cobalamin is also essential for normal metabolism in cells, and in maintaining normal homocysteine levels. During pregnancy it helps prevent macrocytic anaemia (Gallagher, 2012). A vitamin B12 deficiency may cause recurrent spontaneous abortions, as well as increasing the risks of preterm delivery (Molloy et al., 2008).

Pantothenic acid is an integral part of coenzyme-A which plays a critical role in metabolism (Gallagher, 2012). Pantothenic acid may also play a role alongside folate in decreasing the risk of NTDs (Dawson et al., 2006). Deficiencies of pantothenate are rare as this vitamin is widely distributed in foods (Gallagher, 2012).

Biotin is a carboxyl carrier that plays a role in the metabolism of folate, pantothenate, and vitamin B12 (Gallagher, 2012; Perry & Caudill, 2012). Cases of symptomatic biotin deficiency are rare

because of its widespread availability in food, with deficiencies being reported in only very specific cases of chronic ingestion of un-denatured egg whites, in incomplete parenteral nutrition, and with inborn errors of metabolism (Mock, 2013; Zempleni, 2012). Biotin deficiency has shown to be teratogenic in several animal studies (Mock, 2014). Spontaneous marginal biotin deficiency has been observed in cohort studies on pregnant women (Perry & Caudill, 2012; Mock, 2009; Mock et al., 1997). This has been found to be highly teratogenic in mice and may have similar impacts on human pregnancy (Mock, 2009). Perry et al. (2014) suggest that the current AI of 30ug/day for pregnant women may be insufficient, but no studies currently indicate adverse pregnancy outcomes related to insufficient biotin intake (Mock, 2014).

Choline

Choline requirements increase from 400mg/day and 425mg/day in adolescent and adult participants respectively, to 450mg/day during pregnancy. Despite an enhanced ability to synthesise choline during pregnancy, it remains an essential nutrient as its synthesis remains insufficient to meet requirements (Gallagher, 2012; Zeisel & Da Costa, 2009). During pregnancy, choline is required for structural integrity of cell membranes, cell signalling, nerve impulse transmission (Gallagher, 2012), maternal liver and placental function (Zeisel, 2013). A possible association was reported between choline deficiency and risk for NTDs (Shaw et al., 2009), as well as with foetal brain development and cognition (Zeisel, 2013).

Vitamin C

Vitamin C requirements increase from 75mg to 85mg per day during pregnancy. Vitamin C acts as a reducing agent essential for collagen synthesis (Gallagher, 2012) which is important for foetal bone development. Vitamin C works as an antioxidant facilitating immune function, and may help in preventing preeclampsia and premature membrane ruptures (Erick, 2012; Klemmensen et al., 2009; Hassan & Onu, 2006). However, Roberts et al., (2010) did not find an association between preeclampsia and vitamin C intake. As an antioxidant, it can also help to compensate for the pro-oxidative effect that pregnancy can have on the mother (Brown, 2008; Casaneuva & Viteri, 2003). Vitamin C also assists in iron absorption which is crucial during pregnancy (Gallagher, 2012).

Vitamin D

Although the RDA for vitamin D does not increase during pregnancy, adequate intake is essential as this plays various essential roles in maintaining maternal calcium balance and aiding in foetal skeletal calcification (Gallagher, 2012; Williamson, 2006). Deficiency during pregnancy can cause

neonatal hypocalcaemia and has been associated with preeclampsia and recurrent pregnancy loss (Achkar et al., 2014; Aghajafari et al., 2013; Erick, 2008; Hypponen, 2005), gestational diabetes and Small-for-Gestational-Age (SGA) (Aghajafari et al., 2013). In-utero bone mineral accrual, and maternal 25(OH)-vitamin D concentration specifically, may play a substantial role in bone health in later life, especially on osteoporosis risk (Curtis et al., 2014; Hart et al., 2014; Zhu et al., 2013). A birth cohort study done by Hart et al. (2014) also found maternal vitamin D deficiency to be associated with impaired lung development and neuro-cognitive difficulties in children, and an increased risk of eating disorders in adolescence.

Vitamin E

Vitamin E requirements do not increase during pregnancy, but it plays an important role as a lipophilic antioxidant (Gallagher, 2012). Vitamin E deficiency has been speculated to cause miscarriage, IUGR and preterm birth (Gagne et al., 2009) and a low vitamin E intake in the second trimester has been associated with increased risk of hyperglycaemia and insulin resistance later in pregnancy (Ley et al., 2013). However, a Cochrane review indicated that current results regarding supplementation with vitamin E during pregnancy are still inconclusive (Rumbold & Crowther, 2014).

Vitamin K

Vitamin K requirements are higher for adolescents than for adults, but these requirements stay the same during pregnancy. The role of vitamin K in bone health and coagulation homeostasis makes the vitamin vital in pregnancy (Gallagher, 2012). A deficiency of vitamin K during pregnancy has been reported in women that presented with hyperemesis gravidarum, Crohn's disease, and that have had bariatric surgery (Brunetti-Pierri et al., 2007). A deficiency may increase the risks of haemorrhage in the newborn (Shearer, 2009; Shills, 2006).

2.3.4 Minerals

As with vitamin requirements, some mineral requirements increase substantially during pregnancy to allow for healthy bone growth and for adequate blood volume expansion. The DRIs for minerals are summarised in Table 2.9.

TABLE 2.9 *RDAs, AIs, EARs and ULs of Minerals for non-pregnant and pregnant women* (Food and Nutrition board of the IOM, 2010; 2006)

	Calcium (mg)	Chromium (ug)	Copper (ug)	Fluoride (mg)	Iodine (ug)	Iron (mg)	Magnesium (mg)	Manganese (mg)	Phophorous (mg)	Selenium (ug)	Zinc (mg)	Potassium (g)	Sodium (g)	Chloride (g)
Non-pregnancy (19 – 30 years of age)														
RDA/ AI	1000	25*	900	3*	150	18	310	1.8*	700	55	8	4.7*	1.5*	2.3*
EAR	800	-	700	-	95	8.1	265	-	580	45	6.8	-	-	-
UL	2500	ND	10000	10	1100	45	350	11	4000	400	40	ND	2.3	3.6
During pregnancy (14 – 18 years of age)														
RDA/ AI	1300	29*	1000	3*	220	27	400	2.0*	1250	60	12	4.7*	1.5*	2.3*
EAR	1000	-	785	-	160	23	335	-	1055	49	10.5	-	-	-
UL	2500	ND	8000	10	900	45	350	9	3500	400	34	ND	2.3	3.6
During pregnancy (19 – 30 years of age)														
RDA/ AI	1000	30*	1000	3*	220	27	350	2.0*	700	60	11	4.7*	1.5*	2.3*
EAR	800	-	800	-	160	22	290	-	580	49	9.5	-	-	-
UL	2500	ND	10000	10	1100	45	350	11	3500	400	40	ND	2.3	3.6

Abbreviations: AI = Adequate Intake RDA = Recommended Dietary Allowance EAR = Estimated Average Requirements UL = Upper Limit ND = Not determinable

AIs are marked with an asterisk *

Calcium

Daily calcium requirements for pregnant adolescents increase from 1 000mg to 1 300mg to meet both maternal and foetal skeletal needs (IOM, 2010; Cox & Phelan, 2008). Requirements for adult pregnant women do not increase because of the hormonal changes that increase the efficiency of calcium absorption and resorption (Erick, 2012; Williamson, 2006). Women with long term calcium inadequacy may be at increased risk for bone loss during pregnancy (Hacker et al., 2012). Short term deficiencies may also lead to osteoporosis (Gallager, 2012). Miyake et al. (2014) reported that depressive symptoms during pregnancy decreased with an increased intake of yoghurt and calcium. An association has also been found between calcium deficiency and increased risk for preeclampsia (Hofmeyer et al., 2014; Imdad & Bhutta, 2012). Calcium intake in the mother has been positively associated with bone outcomes in the offspring (Ganpule et al., 2006; Chang et al., 2003).

Iodine

Recommendations for iodine increase from 150mg to 220mg per day in both adolescent and adult participants. Iodine is an essential trace element required for the production of thyroid hormones in humans (Delange, 1994; Laurberg, 2012) that regulate basal metabolic rate, cellular oxidation and resting energy expenditure, and are involved in carbohydrate, protein and fat metabolism (Sherwood, 2004). Thyroid hormones also play a crucial role in normal brain and nervous system development, and are essential for growth. Iodine deficiency during infancy is now accepted as the most common preventable cause of brain disorders in children in the world today, including impaired cognitive development and mental retardation (Ohara et al., 2004; Black, 2003, Gallagher, 2012, Roman, 2012). Maternal iodine deficiency during pregnancy decreases foetal iodine availability and may irreversibly impair development and increase mortality (Andersson et al., 2010). The effects range from small neurological changes to impaired learning ability and performance in school. Bath et al. (2013) found that children of mothers with even mild iodine deficiency during pregnancy had significantly decreased IQ scores, while a meta-analysis of Chinese studies by Qian et al. (2005) reported an approximate difference of 10 IQ points between moderate to severely iodine-deficient and iodine-sufficient or iodine-supplemented populations.

Iron

The daily requirements for iron during pregnancy are 27mg for both adolescent and adult participants, compared to 18mg for non-pregnant women. This increase in requirements is due to the expansion of maternal plasma volume for sustaining foetal growth and nutrient transport. A marked increase in iron requirements in the third trimester coincides with foetal storage of iron during this time. Approximately 1g of iron needs to be stored throughout pregnancy: 300mg for the foetus and placenta, 450mg to increase red blood cell mass, and 250mg that is lost during delivery (Scholl, 2011).

Mothers that enter pregnancy with iron deficiency or inadequate iron stores are at risk for developing maternal iron deficiency (McArdle et al., 2014; Scholl, 2011) and inadequate weight gain (Scholl, 2005). Iron deficiency during pregnancy reduces foetal iron stores into the first year of life and is associated with a two-fold increase in the risk for LBW, preterm delivery and stillbirth (McArdle et al., 2014; Gallagher, 2012; Scholl, 2011; Allen, 2005; Ronnenberg et al., 2004). Maternal iron deficiency increases the infant's risk of becoming anaemic and has a negative impact on infant growth, cognitive and behavioural development (McArdle et al., 2014; Herna´ndez-Martı´nez et al., 2011; Lozoff et al., 2006; Allen, 2005). Furthermore, it has also been found that

excessive maternal weight gain during pregnancy and maternal obesity are also independent risk factors for iron deficiency in the newborn (Phillips et al., 2014; Jones & Lozoff, 2014).

Iron deficiency in the third trimester reflects the normal physiological expansion of maternal plasma volume (Scholl, 2011; Pena-Rosas & Viteri, 2009; Allen, 2005; Casanueva & Viteri, 2003). High haemoglobin levels in late pregnancy (indicating an inadequate blood volume expansion) have been shown to increase the risk for preeclampsia, maternal diabetes, low birth weight and preterm delivery (Pena-Rosas & Viteri, 2009; Steer et al., 1995).

Iron deficiency anaemia (IDA) has been shown to contribute to maternal morbidity and mortality rates by increasing susceptibility to infections (Scholl, 2011; Brabin et al., 2001) because of the role of iron in humeral and cellular immunity (Gallagher, 2012). Brabin et al., (2001) showed that overall deaths due to anaemia in developed regions was below 1 000 in both age categories (15-29 years and 30-44 years) while the rates in developing countries was 9 000 in 15-29 year olds and 7 100 in 30-44 year olds.

Magnesium

The requirements for magnesium increase during pregnancy from 310mg/d to 350 mg/day and 400mg/day for adolescent and adult participants respectively. Magnesium plays an important role in neuromuscular transmission (Gallagher, 2012) and blood pressure regulation (Kass et al., 2012; Zhang et al., 2012). A deficiency during pregnancy has been associated with preterm labour (Rylander, 2014; Hantoushzadeh et al., 2007; Durlach, 2004), hypertension, IUGR and LBW (Rylander, 2014; Jain et al., 2010; Cooke & Mimouni, 1997). Magnesium depletion may play a role in the aetiology of sudden infant death syndrome by affecting thermoregulation mechanisms (Durlach, 2004).

Phosphorous

Phosphate requirements increase from 700mg to 1250mg per day during pregnancy. Absorption is increased, while excretion is decreased to aid in meeting these requirements (Abrams, 2007). Phosphorous is the main component of ATP and also forms the basis of DNA and RNA (Gallagher, 2008), and is one of the major minerals involved in bone formation (Garza-Gisholt et al., 2012). Low levels of phosphorous in pregnancy may be caused by severe vomiting and can be life threatening if levels are not restored promptly (Stanga et al., 2008). A significant positive

association has been reported between birth length and cord phosphorous blood levels (Colak et al., 2014).

Zinc

The RDA for zinc increases during pregnancy from 8mg per day to 11mg for adolescents and 12mg for adults. Zinc functions as a cofactor in protein synthesis and many other enzymatic reactions, including translation of the insulin-like growth factor 2 gene (Gallagher, 2012; Izquierdo-Alvarez et al., 2007). It thus plays a very important role in normal growth and development of the foetus and the production of pregnancy related "tissues" in the mother (Erick, 2012; Izquierdo-Alvarez et al., 2007). Intestinal zinc absorption is enhanced during pregnancy to help meet requirements (King, 2000). Deficiencies of zinc in pregnancy have been associated with preterm birth, intrapartum haemorrhage, infections, (Brown, 2008), LBW, congenital defects and abnormal foetal brain development (Erick, 2012). The possible role of zinc deficiency in the onset of preeclampsia is being investigated (Al-Jameil et al., 2014; Lambe et al. 2014).

Sodium

Sodium requirements remain the same during pregnancy, although hormonal changes affect sodium metabolism and glomerular filtration thereof (Erick, 2013). It is an important mineral to monitor because of its role in heart health, and specifically in pre-eclampsia and eclampsia (Steffen, 2014; Rakova et al., 2014). Excessive sodium intake can lead to eclampsia (Rakova et al., 2014), while insufficient intake or excessive loss of sodium (as with NVP or Hyperemesis Gravidarum) can lead to electrolyte imbalances and possible hospitalisation for rehydration and nutritional support (Matthews et al., 2014; Erick, 2012). Thus, aggressive dietary sodium restriction is unwarranted in pregnancy, and intake should remain above 2 g/day (Erick, 2013).

2.3.5 Food portion intake recommendations to meet energy and nutrient requirements during pregnancy

With the exception of protein, requirements of micronutrients increase to a larger extent than that of macronutrients. It is thus recommended that the nutrient-density of the diet of a pregnant woman should be increased more so than the energy density thereof. Currently there are no general South African portion size recommendations for pregnant women. In December 2011 the USDA launched a free interactive website, "Super Tracker", to help the general population to calculate the number of portions they require from the different food groups and thus adhere to their personal dietary recommendations according to the most recent research (McGuire, 2011; USDA, 2011). Portion

recommendations (number per food group) for pregnancy from the "Super Tracker" and from the Canada's Food Guide (2011) are summarised in Table 2.10 below.

TABLE 2.10 *Comparison of food portion intake recommendations for non-pregnant* (Daily Food Guide, 11-50 year olds; ADA 2002) *and pregnant women* (USDA, 2011; McGuire, 2011)

FOOD GROUP	**Number of food portions recommended from food groups**		
	NON-PREGNANT	**PREGNANT OR LACTATING**	
Reference	Erick (2012); ADA 2002	USDA (2011)	Canada's Food Guide (2011)
Proteins *	5 (3 per week of vegetable protein)	2 – 3	2
Dairy	2 – 3	3	2
Whole-grains	7	6 – 11	6 – 7
Vegetables	3	3 – 5	7 – 8
Fruits	2	3 – 5	
Unsaturated fats	3	5 – 7 tsp (25 – 35 mL)	6 – 9 tsp (30 – 45mL)

* Canada's Food Guide uses 75g as an average meat portion as opposed to 30g used by the ADA

In addition to the recommended servings shown in Table 2.8, Canada's Food Guide allows for an extra two to three servings from any group except fats to make up the daily dietary requirements. According to the USDA (2011), portion recommendations increase for most food groups (except fats) during pregnancy and mostly from the second half of pregnancy when energy requirements increase greatly. The USDA "Super Tracker" adjusts requirements and recommendations according to the specific gestational period. For example, recommendations for the number of portions of fruits and vegetables required increases between the first and second trimester, recommendations for grains increases by one portion per trimester, and for proteins increases by a half a portion per trimester (USDA, 2011).

2.3.6 Quality based food recommendations

Following recommended food group guidelines assists an individual in attaining a diet that incorporates a variety of foods and possibly covers most macronutrient requirements. Making healthy food choices from each food group allows for a wide variety of intake, meeting all macro- and micronutrient requirements. With the rising prevalence of obesity and several non-

communicable diseases (NCDs) across the globe, making healthier food choices is becoming increasingly important (Epping-Jordan et al., 2005).

Foods from the meat and meat product group are generally rich in iron, vitamin A and the B vitamins (Schonfeldt et al., 2013). Choosing lean prudent portions will add variety and necessary nutrients, without adding excessive total fat, SFA and cholesterol (Smuts & Wolmarans, 2013; Schonfeldt et al., 2013; Canada's Food Guide, 2011). It is also recommended to remove the visible fats from these products, and to avoid certain preparation methods like frying, deep frying, or roasting in excessive fats (Canada's Food Guide, 2011; Smuts & Wolmarans, 2013). The intake of sausages and processed meats should be limited to low sodium varieties (Canada's Food Guide, 2011; Smuts & Wolmarans, 2013). At least two 75g portions of fish should be consumed per week to contribute to the intakes of omega-3 alpha-linolenic acid (DHA and EPA) (Canada's Food Guide, 2011; Mennitti et al., 2015).

While starchy foods should be a part of most meals, the emphasis should be on whole grain, unrefined choices (Vorster, 2013). These include whole wheat bread and pasta, brown rice and oats, as well as legumes and roots (Canada's Food Guide, 2011; Vorster, 2013; Venter et al., 2013). Beneficial effects accompanying the intake of minimally processed starchy foods include a higher intake of micronutrients and dietary fibre (Venter et al., 2013). The intake of sugar, sugar-sweetened beverages, and other food items high in sugar and fat should be minimal (Canada's Food Guide, 2011; Vorster, 2013).

Fats should be used sparingly, with a preference for vegetable oils, PUFAs and MUFAs. Fried and processed foods, as well as other foods high in SFA and industrially produced TFA should be avoided (Smuts & Wolmarans, 2013).

Fruits and vegetables are rich in vitamins and minerals and should be eaten every day (Naude, 2013). Eating whole fruits and vegetables is preferable to having juices, and fruit juices should be limited or avoided (Naude, 2013). Shorter cooking times for the majority of vegetables is favourable as excessive heat and cooking may result in increased nutrient and fibre loss. Certain cooking methods (steaming, microwaving or pressure-cooking) aid in retaining more nutrients in the majority of vegetables. Carrots are the biggest exception to this where boiling improves the nutrient availability (Bureau et al., 2015; Palermo et al., 2014). Sugar and fat added during or after the preparation of vegetables should be limited or avoided.

2.3.7 Need for nutrient supplementation during pregnancy

It is currently recommended that when an adequate and varied diet is being followed, multivitamin and mineral supplements are not necessary in pregnancy (Kaiser & Allen, 2002) with the exception of folic acid supplementation. However, if dietary intake is inadequate and/or a pregnant adolescent/woman has additional needs for one or more reasons, supplementation should be considered. Females (adolescent and adult) with poor nutritional status at the onset of pregnancy, vegans, females with histories of substance abuse, multiparous pregnancies, closely spaced pregnancies or those unable to reach their daily dietary requirements for other reasons, and adolescents per se, may benefit from supplementation (Erick, 2012; Wendt et al., 2012; Dewey & Cohen, 2007; Kaiser & Allen, 2002). Research shows that the more compromised the nutritional status of the woman before and during pregnancy, the greater the benefit of correct supplementation will be on pregnancy outcome (Erick, 2012).

Iron

Several studies support prophylactic supplementation with 48-60 mg/day iron throughout pregnancy because of the high risk of IDA in pregnant women (Ribot et al., 2012; Scholl, 2011; Black et al., 2011). The WHO recommends starting with supplementation with 60mg early in the pregnancy (WHO 2001, 2007) to prevent maternal IDA, preterm labour and LBW (Scholl, 2011; Ribot, 2012; Siega-Riz et al., 2006; Cogswell et al., 2003). Iron supplements have been found to result in a number of side-effects such as constipation, nausea and epigastric pain that may reduce compliance (Lutsey et al., 2008). Strategies that have been suggested and implemented to address compliance problems include weekly taking of supplements instead of daily, fortification of staples with iron and food diversification (Habib et al., 2009; UNICEF, 2007). It needs to be noted that iron supplements may have been reported to result in negative health outcomes, including increased risk for bacterial infections (Fishbane, 1999), increased risk for oxidative stress in the event of high free iron levels (Bullen et al., 2005) and possibly increased risk for celiac disease in the child (Norwegian Mother and Child cohort study) (Strodal et al., 2014). It is also recommended that iron and zinc should not be taken together as iron inhibits zinc absorption (Gallagher, 2012). Furthermore, excessive iron intake also inhibits absorption of copper (Gallagher, 2008).

Zinc

Zinc supplementation of 25mg dose should be considered if the mother smokes, drinks excessive amounts of alcohol, has poor gastrointestinal function or in the case of trauma or acute stress

response (King, 2000). Zinc supplementation may decrease prevalence of preterm birth in women with low socioeconomic status (Mori et al., 2012).

Calcium and Vitamin D

Risk of preeclampsia and other hypertensive disorders have been shown to decrease with calcium supplementation of between 500mg/day up to 2g/day during pregnancy (Hofmeyer et al., 2014; Imdad & Bhutta, 2012; Kumar et al., 2009; Villar & Belizan, 2000). Vitamin D supplementation is recommended for women that do not get sufficient sun light (Kaiser & Allen, 2008). Calcium and vitamin D supplements may be needed in women that do not consume dairy products due to allergies, intolerances or dietary preferences (Kaiser & Allen, 2002). It needs to be borne in mind that some studies show that excessive/ unnecessary calcium supplementation may increase the risk for cardiovascular disease later in life (Wange et al., 2012; Li et al., 2012).

Folic acid

Supplementation with 400ug/d of folic acid preconceptionally through to the twelfth week of pregnancy is recommended to reduce the risk of NTDs (Erick, 2012; De-Regil et al., 2010). If the child from a previous pregnancy had a neural tube defect, the CDC recommends consulting a doctor on the possibility of taking a 4 000ug folate supplement before and during subsequent pregnancies (CDC, 2014; Procter & Campbell, 2014). Supplementation in later pregnancy may decrease the risk of other birth defects (Butte & King 2005), though this needs further investigation (De-Regil et al., 2010). Supplementation from four weeks prior to pregnancy to eight weeks post-conception has been associated with a reduced risk of language delay (Roth et al., 2011). Some studies have linked folate supplementation with a decreased risk of colorectal cancer in the offspring (Sie et al., 2011; Kennedy et al., 2011). However, a cohort study of over 66 000 women also found an increase in the risk of preterm delivery when folate supplementation was started more than eight weeks prior to conception (Sengpiel et al., 2014). It should also be noted that folate supplementation has been associated with increased risk of breast (Larsson et al., 2010), colon and overall cancer (Wien et al., 2012) in the mother and a slight increase in risk of wheezing and lower respiratory tract infections in the offspring (Bekkers et al., 2012; Haberg et al., 2009).

Omega-3 supplementation

Despite extensive research, evidence to support omega-3 supplementation during pregnancy to promote neurodevelopment, visual acuity, and infant growth, is still inconsistent and inconclusive (Haggarty, 2014; Campoy et al. 2012). Typical amounts given during the studies ranged from 240

to 3 300mg/d of LC-PUFAs, and 200 – 2 200mg/d of DHA (Gould et al., 2013). Some evidence suggests that LC-PUFA supplementation decreases the risk of preterm delivery (Koletzko et al., 2014; Haggarty, 2014) and of respiratory symptoms (Escamilla-Nuñez et al., 2014). Omega-3 supplements taken during pregnancy have been shown to decrease existing maternal depressive disorders (Appleton et al., 2010; Golding et al., 2009; Su et al., 2008). These disorders have been shown to increase the risk of LBW and preterm birth (Grote et al., 2010; Hedegaard et al., 1993). At this point in time, however, larger studies standardised for genetic factors are needed to provide better insights in the need for supplementation, as well as the safety thereof.

Multiple micronutrient supplementation

According to West et al. (2014) there is inadequate evidence to recommend multiple micronutrient supplementation other than only folic acid and iron supplementation. However, research does show that multiple micronutrient supplementation may have a greater beneficial impact on birth weight and gestational age when compared with two or less micronutrients, no supplementation, or a placebo (Haider & Bhutta, 2012; Kawai et al., 2011; Haider et al., 2011; Fall et al., 2009). Among the concerns of multiple micronutrient supplementation during pregnancy is the possible increased risk of early infant mortality rates. This was reported in a Nepalese study where women receiving multi-micronutrient supplements at RDA levels had a greater risk of perinatal morbidity and mortality compared to women receiving only iron-folate supplements (Osrin et al., 2005). Clearly further research is essential to clarify the benefits and potential dangers of supplementation of multiple nutrients versus single or specific combinations of nutrients during pregnancy.

Within the South African context, the integrated nutritional supplementation/ therapeutic programme (NSP/ NTP) was initiated in 1995, focuses specifically on maternal and child health, and offered food and/ or micronutrient supplementation to women that met certain criteria (Grundlingh et al., 2013). The South African National Food Consumption Programme was also initiated to provide nutrients found to be consumed below recommendations. This programme entails compulsory fortification of maize and wheat foodstuffs with folic acid, iron, vitamin A, thiamine, riboflavin, niacin, pyridoxine and zinc, and was implemented in October 2003 to improve the nutritional composition of the average dietary intake of South Africans (UNICEF, 2007). Modjadji et al. (2007) reported that this programme had some positive effects on folate status in non-pregnant women in a small rural area in the Limpopo province.

2.3.8 Adequacy of dietary intake in pregnant and non-pregnant women

Seven studies could be traced that investigated the adequacy of dietary intake of pregnant women in various regions of South Africa (Table 2.11). Three additional studies assessing the dietary adequacy of non-pregnant South African women of child-bearing age were summarised in Table 2.12. A critical discussion of all these studies follows after the tables. The quality of the research, population demographics, analysis of reference the data used, and the results of dietary assessments are covered in this discussion.

Study design

It is important to consider the study design, population and sample size when comparing the results of different studies. Generally, the larger the sample size, the better the quality of observational research (Gibson, 2005). The sample sizes used in the studies summarised in Tables 2.11 and 2.12 range from 15 (Klinger, 2004) to 431 (Kesa & Oldewage-Theron, 2005) in the pregnant women, and 80 (Modjadji et al., 2007) to 1015 (Serfontein et al., 2010) in the non-pregnant women.

The sampling methods used directly affect data quality and the extent to which the data can be used to describe a general population. All the studies on pregnant women, except for one (Kesa & Oldewage-Theron, 2005) used convenience sampling which may not accurately represent the overall population. Some studies either implemented supplementation (Mamabolo et al., 2004) or fortification (Modjadji et al., 2007) programmes, or stated that the participants were on such programmes (Bopape et al., 2008). The effect of these programmes, if properly implemented could explain disparities in the nutrient results between studies. Of the studies done on pregnant women, three were longitudinal (Bopape et al., 2008; Mostert et al., 2005; Klinger, 2005), three were cross-sectional (Jaffer et al., 2008; Kesa & Oldewage-Theon, 2005; Mamabolo et al., 2004), and one was a case-control study (Tshitaudzi, 2003). Two of the studies done on non-pregnant women were cross-sectional (Hattingh et al., 2008; Serfontein et al. 2010) and one was experimental in design (Modjadji et al., 2007).

Population demographics

The demographic profile of participants may impact on data quality, be it the literacy level (affecting the quality of the captured data) or the Socio-Economic Status (SES) (affecting actual dietary quality and food availability). Four out of the seven studies done on pregnant women (Bopape et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al. 2005; Tshitauddzi, 2003; Mamabolo et al., 2004), and one of the studies on non-pregnant women (Modjadji et al. 2007) were

carried out in the Limpopo Province. Two studies, both on pregnant women, were done in the Western Cape (Jaffer et al., 2008; Klinger, 2004). Kesa and Oldewage-Theron (2005) conducted their study in Gauteng, Hattingh et al. (2008) used a sample from Bloemfontein, and Serfontein et al. (2010) used the Northwest Province. In general, all the populations in the studies included in Table 2.11 and Table 2.12 fell into poor SES categories, making the data comparable in this regard.

Effect of different ages

The ages of the pregnant women across the studies in Table 2.11 range from 12 years (Bopape et al., 2008) to 40 years (Mostert et al., 2005; Klinger, 2004). All the studies on pregnant women included adolescents, with Tshitaudzi's (2003) sample including only adolescents. Klinger (2004) used 18 years as a minimum inclusion criterion, while the other studies included younger adolescents. Hattingh et al. (2008) was the only study that included only adults, while Modjadji et al. (2007) also used 18 years as a minimum inclusion criterion. Similarly, the age of non-pregnant women depicted in Table 2.12 ranged from 15 (Serfontein et al., 2010) to 44 years (Hattingh et al., 2008; Modjadji et al., 2007). The broad age range covered in these studies results in the need to use a range of dietary standards references for interpretation of adequacy of intake, which complicates comparisons between studies.

Methodology

Every method of dietary data collection has advantages and disadvantages (see section 2.6.1 *Overview of dietary intake assessment methods*) and may not always yield the same results. Out of all ten studies (those on pregnant and non-pregnant women) seven studies used FFQs of varying lengths (Bopape et al., 2008; Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005; Tshitaudzi, 2003; Klinger, 2004; Hattingh et al., 2008; Serfontein et al., 2010), three studies used repeated 24-hour recalls combined with (Bopape et al., 2008; Klinger, 2004) or without (Mostert et al., 2005) FFQs and two studies used other recall methods combined with the FFQ (Kesa & Oldewage-Theron, 2005; Serfontein et al., 2010). Where 24-hour recalls were done, either two (Mostert et al., 2005; Bopape et al., 2008) or three (Klinger, 2004) days of recall were collected, rendering these studies comparable in this regard. Eight studies used blood and serum samples to assess nutritional status in addition to dietary intake assessments (Bopape et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Tshitaudzi, 2003; Klinger, 2004; Mamabolo et al., 2004; Serfontein et al., 2010; Modjadji et al., 2007).

More specific details of the methodology used that need to be considered are as follows: Kesa and Oldewage-Theron (2005) used a QFFQ to gather information on food consumption patterns. A single-day 'food diary' consisting of a list of iron-rich foods was used to cross-reference with the QFFQ. The food-diary method usually requires good participant literacy and higher participant burden, but using a list of foods may help to partly overcome these barriers. However, a food list could also have impacted on actual food choices and behaviours for the day that the diary was being kept. Serfontein et al. (2010) used a 7-day weighed food record against which to validate their QFFQ. Not all the studies that used FFQs stated the number of items included in the food list (Hattingh et al., 2008; Serfontein et al., 2010). Jaffer et al. (2008) used a 14-item FFQ and Klinger (2004) used a 144 item questionnaire. Bopape et al. (2008) used a FFQ that focussed on the assessment of intake of specific nutrients, namely iron, folate and vitamin B12. The length of a FFQ may influence the quality and accuracy of data, with longer, more in-depth questionnaires providing more detailed and more accurate data (Cade et al., 2002). Modjadji et al. (2007) did not include a dietary analysis, but rather used only biochemical data for specific nutrients.

Conclusions drawn regarding the adequacy of the dietary intake of pregnant and non-pregnant women based on the data presented in Tables 2.11 and 2.12 needs to be considered bearing in mind the differences in and limitations of the dietary intake assessment methodology used.

Analysis and reference data

It is recommended by the Food and Nutrition Board of the IOM to use EARs when evaluating group intake for dietary adequacy (Murphy & Poos, 2002), since the RDA, by definition, is likely to seriously overestimate the proportion of the group at risk for inadequacy (Murphy & Poos, 2002).

The majority of the studies summarized in Tables 2.11 and 2.12 used the RDAs as the reference points, with the exception of Bopape et al. (2008) and Serfontein et al. (2010) who used the EAR cut-point method. Interpretation of RDA was either based on actual % RDA (Kesa & Oldewage-Theron, 2005; Klinger, 2004) or proportion that consumed <67% RDA (Mostert et al., 2005; Tshitaudzi, 2003; Hattingh et al., 2008). Comparison between these studies in terms of adequacy of intake should thus be done with care to accommodate the differences in interpretation criteria used.

Dietary Assessment

A variety of nutrients were assessed and where possible, comparisons are made in the following sections, bearing in mind the points made above regarding study design, population demographics

and reference data used. A lack of detailed description of the diets analysed in these studies should be considered as many foods which are good sources of some of these nutrients are not mentioned. Whether maize meal mentioned in the studies has been fortified or not is also not clarified, and this would affect the dietary adequacy of the women's diets and comparison with blood results.

Iron

Several of the authors reported inadequate dietary iron intake in pregnant women (Bopape et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Tshitaudzi, 2003; Klinger, 2004; and Mamabolo et al., 2004) and non-pregnant women (Hattingh et al., 2008). The extent of inadequate intake using the <67% of the RDA cut-off was reported to be 36% (Kesa & Oldewage-Theron, 2005), 49.1% (Hattingh et al., 2008), 50% (Klinger, 2004), 50.9% (Mamabolo et al., 2004), 55% (Tshitaudzi, 2003) and 73.9% (Mostert et al., 2005). Using the EAR cut point method, Bopape et al. (2008) found 98% of pregnant women to have an inadequate iron intake. In studies done on non-pregnant women, Hattingh et al. (2008) found that 49.1% of the study population did not meet 67% of the iron RDA. However, Modjadji et al. (2007) found that only 7.5% of the non-pregnant women included in their study were actually iron deficient (from blood results), compared to the combined average of 74% of pregnant women who are at risk for inadequate iron intake as mentioned by studies above. As most of the iron intake comes from plant sources (Kesa & Oldewage-Theron, 2005; Tshitaudzi, 2003) it is important that adequate amounts of vitamin C are consumed to aid the absorption of non-haeme iron.

All the reviewed studies (Tables 2.11 and 2.12) illustrate that both pregnant and non-pregnant women may be at risk of having or developing iron deficiency as a result of inadequate dietary intake. The work by Jaffer et al. (2008), Kesa & Oldewage-Theron (2005) and Mostert et al. (2005) on habitual food consumption supports this notion. They found that intakes of foods generally rich in iron were not very high. Over and above low intakes, Bopape et al. (2008) also found that the intake of phytate-rich foods that reduce the bioavailability of iron in the diet, was very common.

The prevalence of iron deficiency anaemia (IDA) reported in the studies that collected appropriate blood samples was 79.1% (Mamabolo et al., 2004), 57% (Bopape et al., 2008), 50% (Kesa & Oldewage-Theron, 2005) and 30% (Tshitaudzi, 2003) of the pregnant women, using the diagnostic criteria of an Hb <11g/dL or <10.5g/dL and/or ferritin levels of <12µg/L. IDA is clearly a serious concern among pregnant women in the investigated populations and the poor iron intake, as well as consumption of inhibitors of iron absorption, may be important causes of the problem. Bopape et

al. (2008) mention that hookworm prevalence, which would cause excessive blood loss and poor adherence to iron supplementation programmes, are also important potential causes to consider, while the use of injectable contraceptives is a protective factor.

Folate

The range of inadequate dietary folate intake is wide, with percentage of study samples below the RDA being 93% (Bopape et al., 2008) and 100% (Mostert et al., 2005) for adults and 5% (Tshitaudzi, 2003) and 100% for adolescents (Bopape et al., 2008). Klinger (2004) reported that their sample only consumed 53% of the required RDA. In the non-pregnant women, folate intake was found to be insufficient by Hattingh et al. (2008) (56.6% <67% RDA), Serfontein et al. (2010) (48%) and Modjadji et al. (2007) (27.6%). On average approximately 53% of the pregnant women were at risk of folate deficiency as a result of a folate intake below the RDA; this proportion was on average approximately 42% in non-pregnant women and illustrates the possibility that pregnant women may have an increased risk for folate deficiency than non-pregnant women. However, it must be borne in mind that this risk may be overestimated in both groups as a result of using the % RDA for interpretation of adequacy of intake.

Two different tests were used in the studies presented in Tables 2.11 and 2.12 to assess blood folate levels, namely Red Cell Folate (RCF) and Serum Folate (SF) levels. SF has been shown to be more sensitive to acute changes in serum folate levels and fluctuates with recent changes in folate intake. SF values decline rapidly with inadequate folate intake, but would stabalise at around 7nmol/L after about 2-3 weeks (Cooper & Lowenstein, 1964). When using RCF to determine folate status, an additional test should be done to determine vitamin B12 status as they both present with very similar abnormal morphological changes when intakes are deficient (O'Connor, 1994). Green (2008) states that RCF is a more reliable indicator of longer term folate status. Folate enters the red blood cell in the marrow during the development thereof and is then mostly retained in the cell, providing, an indication of folate status over the past 120 days as this is the average red blood cell lifespan. RCF tests would thus pick up a long-standing, chronic folate deficiency of between two and four months (O'Connor, 1994). A single measure of SF alone cannot distinguish between a transient drop in folate intake and a long-standing chronic deficiency (Green, 2008).

Bopape et al. (2008) and Klinger (2004) analysed only SF while Mamabolo et al. (2004) and Tshitaudzi (2004) analysed both SF and RCF, making their results more reliable indicators of folate status. A further challenge in the comparison of prevalence of actual deficiency between studies is

that different cut-offs ranging from 3 to 4.1ng/mL and 140 to 157ng/mL were used for both SF and RCF. Folate deficiencies were found in 9.8% (SF) (Bopape et al., 2008), 5% (SF) and 2.5% (RCF) (Tshitaudzi, 2003), and 10.3% (SF) and 4.6% (RCF) (Mamabolo et al., 2004) of the pregnant women. The results from both Mamabolo et al., (2004) and Tshitaudzi (2003) show that the values derived from the short term (current) indicator of folate levels (SF) were about double the values derived from the longer term indicator of folate levels (RBC), possibly reflecting an effect of routine supplementation during pregnancy, or an increased dietary intake. However, the dietary intake results discussed above do not support the latter possibility.

Assessment of folate levels in non-pregnant women showed that 27.6% (SF) and 26.4% (RCF) of the population were folate deficient (Modjadji et al., 2007). The RCF and SF results from Modjadji et al. (2007) showed the same discrepancy between current and long-term folate status as found in the pregnant women, but the difference was not as significant.

Vitamin C

Inadequate vitamin C intake in pregnant women was reported in three of the four studies that measured dietary vitamin C. Percentage intakes <67% of the RDA were reported to be 75% for adults and 94% for adolescents (Bopape et al., 2008), 73.9% (Mostert et al., 2005) and 25% (Tshitaudzi, 2003). In the non-pregnant women, vitamin C intake was found to be deficient by Hattingh et al. (2008) (46.2% <67 %RDA) and Serfontein et al. (2010) (52%). As most of the iron intake comes from plant sources (Kesa & Oldewage-Theron, 2005; Tshitaudzi, 2003) it is important that adequate amounts of vitamin C are consumed to aid the absorption of non-haeme iron. The high risk for inadequate vitamin C intake in the females investigated in the studies presented in Tables 2.9 and 2.10 may thus increase the risk for iron deficiency and IDA.

Vitamin B12

Vitamin B12 intake was reported to be adequate in the majority of studies (Tshitaudzi, 2003; Klinger, 2004; Modjadji et al., 2007), while inadequate intakes were reported for 55% of adults and 66% of adolescents (% <EAR) by Bopape et al. (2008). Serfontein et al. (2010) also reported that the mean vitamin B12 intake met only 50% of the EAR in non-pregnant women. These results are in line with the findings that milk intake is inadequate among many pregnant women (Jaffer et al., 2008) since the main food sources of vitamin B12 is foods like liver, milk, eggs, fish and cheese.

Vitamin A

Tshitaudzi (2003) and Mostert et al. (2005) reported inadequate vitamin A intakes of 52.5% and 76.1% (<67% RDA) respectively in pregnant women. This is supported by results from actual food group intake indicating a low or inadequate intake of liver, eggs and dark green and yellow vegetables (Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005; Tshitaudzi, 2003).

Vitamin B6

In the three studies on pregnant women that reported on vitamin B6 intake, inadequate intake was found in 42.5% (<67% RDA) (Tshitaudzi, 2003) and 97.8% (Mostert et al., 2005). Low intakes of meat, whole grains, vegetables and nuts as reported by Jaffer et al. (2008), Mostert et al. (2005) and Tshitaudzi (2003) may explain these findings.

Calcium and Zinc

Tshitaudzi (2003) and Mostert et al. (2005) found calcium (25% and 89.1%) and zinc (62.5% and 93.5%) intakes below 67% RDA. This could be explained by low intakes of red meat, poultry, dairy products and dark green leafy vegetables reported by Jaffer et al. (2008), Kesa and Oldewage-Theron (2005) and Mostert et al. (2005).

Food groups

Jaffer et al. (2008) and Mostert et al. (2005) reported that the most commonly consumed foods in their study samples were starches. Maize was one of the top ten most frequently consumed foods in the study by Kesa and Oldewage-Theron (2005), and is mentioned as a staple in the study sample by Tshitaudzi (2003). Kesa and Oldewage-Theron (2005) state that milk was the most commonly consumed food item, while Jaffer et al. (2008) found milk intake to be inadequate with 55% of the pregnant women not consuming milk products daily. Meat intake was also not regularly consumed, and intake was found to be inadequate (Mostert et al., 2005; Jaffer et al., 2008; Bopape et al., 2008; Kesa & Oldewage-Theron, 2005). Tshitaudzi (2003) found that plant protein intake was greater than animal protein intake in his study population which may increase risk for protein inadequacy if protein complementation is not implemented. Vegetable intake was found to be inadequate (Jaffer et al., 2008; Mostert et al., 2005), and no vegetable was among the top ten foods reported by Kesa and Oldewage-Theron (2005). These authors reported fruit juices (6th) to be among the top ten most commonly consumed food items (Kesa and Oldewage-Theron, 2005).

In conclusion, when compared to the respective reference values, the majority of the studies reported concerning levels of inadequate dietary intakes for iron, folate, vitamin A, vitamin C, calcium and zinc. Diets were found to be very low in vegetables and especially fruits, with varying intakes of meat and dairy products. The fact that dietary intake assessment methodology and interpretation criteria for adequacy varied substantially across studies makes it challenging to provide an overall picture of the dietary adequacy of pregnant and non-pregnant women. Bearing these limitations in mind, conclusions of the critical review of the information provided in Tables 2.11 and 2.12 are that the dietary intakes of iron, folate, vitamin C and B12, as well as meat and vegetable food groups in pregnant women and intakes of folate and vitamin B12 in non-pregnant women may be inadequate. Further research is clearly needed to better assess the dietary adequacy of pregnant women.

TABLE 2.11 *Studies on the adequacy of dietary intake in pregnant women in South Africa*

REFERENCE	AIM AND LOCATION	STUDY DESIGN AND SAMPLE SIZE	POPULATION (AGE, RACE, SES)	ASSESSMENT METHOD	RESULTS	CONCLUSION
Bopape et al. (2008)	Determine iron, folate and vitamin B12 status of pregnant teenagers attending four clinics in Limpopo Province	Longitudinal, descriptive analytical study Convenience sampling n=123	12-21 years age • 38% 12-18 years • 62% 19-21 years 5% 1st trimester 34% 2nd trimester 61% 3rd trimester Mostly rural areas	Two 24-hour recalls (1 weekday, 1 Sunday, 2 or 4 weeks apart) One FFQ (containing folate, iron, VB12 and C, phytate and tannin-rich foods) Food models and household measures Blood samples: SF, ferritin, VB12 and FBC	24-hr recalls: Intake <67% EAR [<67% RDA] classified as deficient: <u>≤18 years</u>: iron 100% [100%], folate 100% [100%], VB12 66% [66%], Vitamin C 87% [94%] <u>19-21 years</u>: iron 96% [96%], folate 93% [98%], VB12 50% [55%], Vitamin C 66% [75%] FFQ: Poor intake of iron, folate and vitamin C 29% and 39% reported weekly red meat and chicken intake, High intakes of phytate-rich foods (bread, maize meal and indigenous vegetables) Biochemical: 36% anaemic, 57% IDA, 9% folate deficiency anaemia, 7% vitamin B12 deficiency anaemia	Majority of participants are anaemic, with poor dietary intake of folate, vitamin C and iron, and high intake of phytate-rich foods. This could explain high prevalence of anaemia. Nutritional education could be beneficial especially during a critical period such as pregnancy.
Jaffer et al. (2008)	Assessing dietary intake of pregnant women at Paarl Hospital in West Coast/ Winelands region and its association with LBW	Case-control Cases n=198 (birth weight ≤2500g) Non-matched controls n=202 (birth weight >2500g)	Case [Controls] age 16.2% [13.4%] ≤19yr 68.7% [76.2%] 20-34yr 15.2% [10.4%] ≥35 yr Postpartum (within 24 hours) women Low SES farm workers	14-item non-quantified FFQ recording previous month's intake	Results for case and controls respectively: Starches biggest intake group in case and control, inadequate vegetable (3 and 4 times per week), fruit (11.7 and 12.4 times per week) and milk product intake (55% case and 52% control did not consume milk products daily). Meat group (4 times per week for case and control), legumes (2 per week for case and control), sugar (8 and 10 times per week), and fats adequate. Sugar, maize and yellow/ orange vegetables consumed in significantly greater amounts in control mothers	Women with greater variety in intake seem to have decreased risk of LBW birth (though not statistically significant in this report). Maternal dietary intake affects LBW risk.
Kesa & Oldewage-Theron (2005)	Determine anthropometry and nutritional intake of pregnant and lactating women in Vaal Triangle	Cross-sectional Stratified random sampling n=431 • pregnant n=315 • lactating n=116)	16-35 years age Monthly household income <R1 000 Majority BMI >25kg/m^2 All stages of pregnancy	Validated QFFQ with food models, cross-referenced with 1-day food diary (included a list of foods with very high iron content) Fasting blood: FBC, transferrin and ferritin	10 items consumed most frequently in descending order: fresh milk; tea; coffee; cold drinks; maize meal; fruit juice; bread; magou; rice and sugar. Mean daily intakes: 8425.71 ± 2279 kJ (75% RDA), 73.18 ± 23 g protein (103% RDA), 62.29 ± 23.7 g fat (200% AMDR), 292.45 ± 72.2g carbohydrate (167% RDA) and 9.74 ± 3.8 mg iron (36% RDA). 50% IDA.	Diets consisted primarily of plant-based foods; animal foods were scarce except for milk. Most of the items consumed were low in iron. ≈80% of pregnant women have BMI >25kg/m^2
Mostert et al. (2005)	Determine dietary intake of pregnant (and lactating) women visiting Dikgale primary health-care clinic in poor rural area in Limpopo	Longitudinal Random convenience sampling n=46 with follow-up at 6 months	13 - 40 years age Severely poor rural area Average BMI 24kg/m^2 Second trimester	Two 24-hour recalls (1 weekday, 1 weekend day) Food models and measuring cups used Blood samples: from 20 consenting participants; retinal and α-tocopherol	Diets consisted mainly of maize, brown bread, sweetened beverages (cold drink and tea), and small amounts of vegetables and chicken. Percentage of pregnant women with dietary intake <67% RDA: protein 23.9%, energy 26.1%, dietary fibre 43.5% , calcium 89.1%, iron 73.9%, zinc 93.5%, niacin 52.2%, folate 100%, and vitamins A 76.1%, C 73.9%, E 69.6%, and B6 97.8%	Pregnant and lactating women had diets low in energy and micronutrients as reflected by high prevalence of underweight at birth

REFERENCE	AIM AND LOCATION	STUDY DESIGN AND SAMPLE SIZE	POPULATION (AGE, RACE, SES)	ASSESSMENT METHOD	RESULTS	CONCLUSION
Tshitaudzi (2003)	Assess nutritional status, with emphasis on iron and folate, and its relationship with pregnancy outcome and the newborn baby, of pregnant teenagers attending Siloam antenatal clinic in the Limpopo Province	Case-control study n=40 pregnant teenagers matched with non-pregnant, non-lactating controls from schools in the area n=40	14-19 years age Rural with primarily low SES	Pre-tested QFFQ covering the previous six months Standard household measures, empty containers, and actual food items used. Blood samples: during and after pregnancy in case participants; one sample from controls. FBC, haematocrit, serum iron concentration, SF, RCF, serum ferritin, serum transferrin, transferrin saturation, vitamin B12.	Maize meal a staple food, mostly unfortified. Daily plant protein intake (38.1g) > animal proteins (22.6g); protein intake lower in pregnant teenagers. Mean intakes of pregnant teenagers (%RDA): Energy 9123kJ (81%), total protein 60.9g (86%), total fat 48.4g (172%), carbohydrates 344.7g (197%), fibre 25.6g (91%), Ca 346.6mg (35%), Fe 10.4mg (39%), Mg 375.7mg (107%), Ph 1002.3mg (143%), Se 9.5g (16%), Zn 7.7mg (70%), I 16.4mg (74%), Vit A 720 RE (94%), B1 0.8mg (57%), B2 0.8mg (57%), B3 12.3mg (68%), B6 1.2mg (63%), Folate 215.5µg (36%), B12 4.4mg (170%), C 99.9mg (118%), D 4.1mg (82%), E 9.5mg (63%) Percentages <67% RDA: Energy 5%, total protein 2.5%, Ca 25%, Fe 55%, Se 65%, Zn 62.5%, Vit A 52.5%, B6 42.5%, Folate 5%, VB12 15%, Vit C 25%. % Pregnant population below cut-offs: SF 5%, RCF 2.5%, serum B12 10%, serum ferritin 30%, serum iron 22.5%, Hb 72.5%, serum transferrin 0%, Haematocrit 57.5%, MCV 22.5%, MCH 42.5%, MCHC 65% 45% of pregnant cases iron deficient, 30% IDA	Growth demands of pregnancy have detrimental effect on iron and folate status of teenagers. Teenagers could benefit from nutrition education programmes and periodic anaemia screening
Klinger (2004)	Determine nutritional status and alcohol consumption in pregnancy in relation to pregnancy outcome in women attending Hanover Park Midwife and Obstetrics unit	Prospective, longitudinal, cohort study Matched case (heavy drinkers) n=15 and controls (light or non-drinkers) n=15	18-40 years age Mostly coloured race group Low SES	Two FFQs covering 1st and 2nd halves of pregnancy respectively Three 24-hour recalls (one weekend day not always included) covering the second half of pregnancy Blood samples: FBC, plasma vitamin A, SF and zinc	Mean intakes for participants (case and controls combined) (% RDA): Folate (53%), iron (50%), iodine (27%), vitamin E (87%), calcium (86%), and vitamin K (82%); CHO (200%), fat (290%), vitamin A (150%), vitamin C (199%), riboflavin (140%), vitamin B12 (212%), phosphorous (200%), magnesium (172%)	Though good nutritional status did have a few statistically significant effects on pregnancy, it did not always protect offspring against teratogenic effects of alcohol.
Mamabolo et al. (2004)	Evaluate the effectiveness of iron and folate supplementation in antenatal clinics in a rural Limpopo village	Cross-sectional analytical study Convenience sampling n=262	25.67 ± 7.02 years age 3rd trimester pregnant women Average BMI 27kg/m^2	Fasting serum and plasma samples: FBC, ferritin, VB12, SF and RCF	Iron depletion (50.9%) diagnosed with ferritin 12- 20 µg/ml (moderate iron depletion) (24.5%), <12 µg/ml (severe iron depletion) (26.4%). IDA: low Hb and severe iron depletion (79.1%). Vitamin B12: <145 pg/ml deficiency (16.4%), SF and RCF <3 ng/ml (10.3%) and <157 ng/ml (4.6%) respectively indicated folate deficiency.	Iron deficiency and iron deficiency anaemia prevalent despite supplements offered at antenatal clinics

Abbreviations: SF = serum folate RCF = Red Cell Folate VB12 = Vitamin B12 FBC = Full Blood Count FFQ = Food Frequency Questionnaire QFFQ = Quantified Food Frequency Questionnaire GGT = gamma-glutamyl transferase Hb = Haemoglobin IDA = Iron Deficiency Anaemia LBW = Low birth weight BMI = Body Mass Index EAR = Estimated average requirement AMDR = Acceptable Macronutrient Distribution Range SES = Socio-economic Status

TABLE 2.12 *Studies on adequacy of dietary intake in non-pregnant women of child-bearing age in South Africa*

REFERENCE	AIM AND LOCATION	STUDY DESIGN AND SAMPLE SIZE	POPULATION (RACE, SES, AGE)	ASSESSMENT METHOD	RESULTS	CONCLUSION
Hattingh et al. (2008)	Assess micronutrient intake of black women from four settlements (formal and informal) living in Manguang, Bloemfontein	Cross-sectional Proportionate random sampling n=496	25 to 44 years age Pre-menopausal black women aged	QFFQ using household measuring cups, spoons, empty labelled food containers and real snack foods Carried out by five trained fieldworkers	Less than 67% RDA: total iron (>49.1%), selenium (50%), folate (>56%), vitamin C (>46%) and iodine (>94.6%). Below AI: median calcium and vitamin D intakes; Exceeded AI: median chromium intake Exceeded RDA: potassium, manganese, copper, magnesium, zinc and phosphorous	Generally adequate intake, attention should be given to those nutrients below 67% RDA, and those not meeting AI
Serfontein et al. (2010)	Investigate possibility of micronutrient dilution by alcohol in diets of adult population in nutrition transition from 37 randomly selected rural and urban sites in SA	Cross-sectional, comparative, population-based study n=1015 women Mean BMI = 27kg/m^2	15 years and older 1757 total subjects aged	QFFQ validated against 7-day weighed food record Alcohol consumption was divided into three levels of intake (non, light and heavy) Blood samples: GGT.	Mean vitamin intakes below EAR (%EAR): B5 3.5g (70%), B12 1g (42%), C 38.9g (52%), D 4.5g (90%), E 10.2g (68%), biotin 21.7g (72%), VB12 1ug (50%), folate 193.2g (48%), calcium (AI) 409.8g (32%), magnesium 290.3g (94%), zinc 8.0g (89%) and fibre 16.4g (64%). Mean macronutrient intakes: protein 57.7g (126%), fat 54.3g (174%), CHO 296.0g (228%), energy 8005kJ (71%).	Respondents who can buy alcoholic drinks can possibly also afford more nutritious foods
Modjadji et al. (2007)	Assess effect of fortification of staple foods on folate and iron status of women of childbearing age living in Dikgale DSS in the Limpopo province.	Prospective cohort study n=80 women	18 - 44 years age Non-pregnant, non-lactating women Poor rural, low SES	Blood samples taken before and 9 months after food fortification: FBC, serum ferritin, VB12 and SF and RCF.	27.6% had low SF, 26.4% low RCF, 6.3% were VB12 deficient, 7.5% iron deficient	Folate deficiency greatly improved with food fortification, but iron status did not improve

Abbreviations: SF = serum folate RCF = Red Cell Folate VB12 = Vitamin B12 FBC = Full Blood Count FFQ = Food Frequency Questionnaire QFFQ = Quantified Food Frequency Questionnaire GGT = gamma-glutamyl transferase Hb = Haemoglobin IDA = Iron Deficiency Anaemia BMI = Body Mass Index EAR = Estimated average requirement AMDR = Acceptable Macronutrient Distribution Range SES = Socio-economic Status

2.4 Nutrition-related complications in pregnancy

2.4.1 Unhealthy weight and weight gain

Pre-conception BMI

Maternal pre-conception obesity increases the risk of preterm and very preterm birth (<32 weeks), cardiac defects, NTD, macrosomia (Calvo & Lopez, 2012; McDonald et al., 2010; Artal et al., 2010; Callaway et al., 2009; Frederick et al., 2008; ACOG, 2005), childhood asthma (Forno et al., 2014), and autism (Moss & Chugani, 2014) in the infant. Being overweight or obese before pregnancy also increases the mother's risk of pregnancy-induced hypertension (PIH), gestational diabetes and increased risk of caesarean sections (Tanaka et al., 2014; Calvo & Lopez, 2012; Guelinckx et al., 2008; Sebire et al., 2001; Durnwald et al., 2004). It is thus concerning that obesity prevalence in South Africa is on the increase. According to the 2003 South Africa Demographic and Health Survey (SADHS), 27.5% of South African women are overweight, and 27.4% are classified as obese. The recent SANHANES found 25% of women to be overweight, and 40.1% to be obese (Shisana et al., 2013).

A low pre-pregnancy BMI has been associated with significantly lower foetal birth weight and height as compared to those born to mothers with BMIs in the normal range before pregnancy (Jeric et al., 2012; Calvo & Lopez, 2012). Pre-pregnancy underweight has also been associated with preterm birth (Hoellen et al., 2014; Han et al., 2011; Khashan & Kenny, 2009), increased risk of spontaneous abortions (Helgstrand & Andersen 2005), placental abruption (Deutsch et al., 2010), and autism (Moss & Chugani, 2014). The prevalence of underweight has been reported as 6.2% in the 2003 SADHS (SADHS, 2007) and 4.0% in 2012 (Shisana et al., 2013).

In light of these complications it is therefore recommended that women be at their optimal weight before becoming pregnant to avoid an increase in pregnancy complications and adverse outcomes for the child (Moss & Chugani, 2014; Tanaka et al., 2014; Calvo & Lopez, 2012; IOM, 2009).

Insufficient weight gain during pregnancy

Inadequate maternal weight gain during pregnancy increases the risk of preterm birth, LBW, SGA (Han et al., 2010; Margerison Zilko et al., 2010; Ricci et al., 2010; Wolfe et al., 1991), and infant mortality (Davis et al., 2014). For women who were normal weight pre-pregnancy,

the risk of having a SGA infant is doubled if maternal weight gain during the second trimester is below 0.25kg per week (Carmichael & Abrams, 1997).

Under-nutrition during pregnancy, which has been associated with inadequate weight gain, is associated with a lower Insulin-like growth factor-1 (IGF-1) concentration. IGF-1 plays an essential role in placental and foetal growth by promoting foetal nutrient uptake and inhibiting foetal tissue breakdown. Therefore, lower levels of IGF-1 may cause SGA and low birth weight as a result of depleted foetal muscle and skeletal mass (Iniguez et al., 2006; Ohkawa et al., 2010). Under nutrition during pregnancy may also result in foetal programming for chronic disease in later life (see section 2.5.3 *Foetal programming*)

Excessive weight gain during pregnancy

Excessive weight gain (>0.7kg/week in third trimester) during pregnancy could lead to a large-for-gestational-age (LGA) baby. Infants born LGA are at greater risk of being overweight at age three and beyond (Mamun et al., 2014; Crozier et al., 2010; Olson et al., 2009; Moreira et al., 2007), and of developing childhood asthma (Forno et al., 2014). An LGA birth also increases the risk of labour difficulties (Mamun et al., 2010). For the mother, excessive weight gain can cause an increase in weight retention after delivery and increases maternal risk of type II diabetes (Margerison Zilko et al., 2010; Mamun et al., 2010; Olson et al., 2009; IOM, 2009). Obesity during pregnancy increases the risk of gestational diabetes, PIH, need for caesarean section, late-pregnancy (>42 weeks gestation) and miscarriage (Artal et al., 2010; Mamun et al., 2010; Callaway et al., 2009; ACOG, 2005).

2.4.2 Dieting

Cohen & Kim (2009) reported that 8.1% of pregnant women in the USA try to lose weight during pregnancy. These women were characterised with being obese, consuming excessive amounts of alcohol, Hispanic ethnicity and being older between 35 and 44 years.

Weight loss during pregnancy has been associated with an increased risk of infant mortality (Davis et al., 2014), LBW, NTDs, preterm birth, and diabetes in later life (NICE, 2010; Johnson et al., 2006; Stocker et al., 2005; Dewey & McCrory, 2004; Carmichael et al., 2003; Mann & Truswell, 2002; Anderson, 2001). Weight loss is typically associated with low energy and micronutrient intake (Carmichael et al., 2003), which in itself can be harmful to pregnancy outcomes. Total fasting and carbohydrate restriction, which fad diets often

promote, cause the production and release of ketones via lipolysis. The ketones pass through the placenta and are readily taken up and metabolised by the foetal brain and adversely affects foetal neuro-cognitive development (Sibai & Viteri, 2014; Erick, 2012; NICE, 2010).

Rather than losing weight during pregnancy, it is recommended that overweight or obese women should gain less weight during pregnancy compared to women that are at their ideal weight, or underweight at the time of conception (see Table 2.5) (IOM, 2009).

2.4.3 Nausea and vomiting

Nausea and vomiting of Pregnancy (NVP) affects 50 - 90% of pregnant women and is thought to be caused by the secretion of the hormones hCG and thyroxin (Matthews et al., 2014; Ding et al., 2013; Nulman et al., 2009). The onset is usually within four weeks after the first missed menstrual cycle and dissipates by 17 weeks. However, 5 – 10% of women may experience NVP throughout their entire pregnancy (Erick, 2012; Niebyl, 2010). NVP can cause aversion to certain foods (Furneaux et al., 2001; Crystal et al., 1999) and under nutrition due to increased losses and decreased intake (Robinson et al., 2004; Broussard & Richter 1998). One to two percent of women experience the most severe form of NVP namely Hyperemesis Gravidarum. This condition is characterised by excessive vomiting in early pregnancy and causes dehydration, decreased dietary intake, electrolyte imbalance, ketonuria and inadequate weight gain. Hospitalisation is usually required for rehydration and nutritional support (Matthews et al., 2014; Erick, 2012; Tan et al., 2007).

Along with medications that can be taken in severe cases of NVP some dietary adjustments may help to lessen the symptoms. Having small frequent meals and consuming liquids 20 minutes after eating (as opposed to with meals) may help reduce nausea. Cold fluids may also be better tolerated. Multivitamins containing iron may exacerbate nausea (Einarson et al., 2007). Vitamin B6 supplementation has shown varying and inconclusive results of effectiveness against nausea (Matthews et al., 2014; Thaver et al., 2006; Shrim et al., 2006). The use of ginger to lessen nausea is similarly inconclusive (Viljoen et al., 2014; Ding et al., 2013).

2.4.4 Heartburn

Heartburn is common during pregnancy affecting up to 80% of women (DallAlba et al., 2010; Dowswell & Neilson, 2008). During the first and second trimester heartburn is usually

the result of hormones (mostly progesterone) which relaxes the oesophageal sphincter and decreases gastric motility. During the second half of pregnancy the pressure of the enlarged uterus on the stomach and intestines may cause acid reflux and heartburn (White, 2014; Vazquez, 2010; Dowswell & Neilson, 2008).

While heartburn in itself is not threatening to the pregnancy it may be distressing to the mother (Quartarone, 2013; Gill et al., 2009). Eating smaller and more frequent meals and avoiding trigger foods may help to reduce heartburn (Erick, 2012), as might raising the head of the bed or sleeping on a wedge (White, 2014). Decreasing the intake of fatty acids in the third trimester may aid in decreasing the symptoms of heartburn (Dall'Alba et al., 2010) with the aim of maintaining the recommended level of intake. The effects of antacid use during pregnancy has not been researched thoroughly enough and these products should thus be taken with care (Dowswell & Neilson, 2008).

2.4.5 Pica

Pica refers to the "craving and purposive consumption of substances that the consumer does not define as food" (Young et al., 2010). Studies on pica in pregnancy indicate higher prevalence in Africa compared to Europe and the United States (Young et al., 2010) and greater frequencies among urban and rural African women (38.3% and 44.0% respectively) compared to Indian, Coloured and White women (22%, 4.4% and 1.6% respectively) (Walker et al, 1985).

Food cravings and aversions during pregnancy are probably caused by fluctuating hormone levels or other physiological changes that affect taste and smell (Erick, 2012; Brown, 2008). Hypotheses suggest that pica may result from vitamin and mineral deficiencies, especially that of iron (Miao et al., 2014; Simpson et al., 2000). However, research is inconclusive as to whether pica causes iron deficiency, or if iron deficiency increases the risk for pica eating behaviours (Lumish et al., 2014; Miao et al., 2014; Young et al., 2010; Simpson et al., 2000; Geissler et al., 1998). Generally, cravings and aversions are not detrimental to the mother or foetus. However, in the case of pica where non-food items are consumed, there may be a risk of inadequate nutrition if the craved food offers no nutritional value, interferes with absorption of nutrients, causes intestinal blockage or causes excessive weight gain (as occurs when clay or starch are consumed) (Miao et al., 2014; Khan & Tisman, 2010; Olds, 2003).

2.4.6 Constipation

The prevalence of constipation during pregnancy ranges from 9% to 39% (Bradley et al., 2007). The Rome III criteria define constipation as stool frequency less than three times in a week, with hard stools or difficulty on evacuation (Drossman & Dumitrascu, 2006). Constipation may be caused or exacerbated by dietary changes, insufficient fibre and fluid intake, decreased physical activity and by the physiological effects of pregnancy hormones on gastrointestinal motility (White, 2014; Bradley et al., 2007; Cullen & O'Donoghue, 2007; Williamson, 2006). Certain antiemetic medications and iron supplements may worsen the symptoms (Erick, 2012; Williamson, 2006). Physical examination and detailed history should be done to rule out the possibility of more severe gastrointestinal pathologies (Cullen & O'Donoghue, 2007). Various medications are available to alleviate constipation (White, 2014) but care must be taken to avoid teratogenicity. Deferring treatment until after pregnancy is recommended unless if the cause of the constipation is IBD (Mahadevana & Kaneb, 2006). Both constipation and pregnancy may also increase the risk for haemorrhoids and haemorrhagic haemorrhoids (Hibberts & Schizas, 2010).

2.4.7 Gestational diabetes mellitus

Gestational diabetes mellitus (GDM) is defined as glucose intolerance diagnosed during the second or third trimester that is not overt diabetes (Roglic & Colagiuri, 2014; WHO, 2013). GDM is now regarded as a condition other than diabetes in pregnancy (hyperglycaemia diagnosed in the first trimester), because of the growing diabetes epidemic (Roglic & Colagiuri, 2014; WHO, 2013). A South African study conducted in Limpopo found that 8.8% of pregnant women are affected by gestational impaired glucose tolerance (7.3%) and GDM (1.5%) (Mamabolo et al., 2007).

Uncontrolled GDM can cause preeclampsia, caesarean delivery, LGA infants and consequent preterm birth and birthing trauma (Ngai et al., 2015; Landon et al., 2009). Both the foetus and mother may be at increased risk for developing cardiovascular disease and type II diabetes mellitus later on (Bellamy et al., 2009; Williamson, 2006). Blood glucose levels need to be strictly controlled to prevent any adverse effects to the mother or foetus, and this can be accomplished through proper diet and medication where necessary (Landon et al., 2009). Low to moderate intensity exercise can also help control blood glucose levels (Metzger et al., 2007).

2.4.8 Pregnancy induced hypertension

Pregnancy induced hypertension (PIH) incorporates a group of blood pressure disorders originating in pregnancy and include pre-eclampsia, eclampsia and gestational hypertension. The major difference between these is the lack of oedema and proteinuria in gestational hypertension, which is present in pre-eclampsia and eclampsia (Nawaz et al., 2014; Watanabe et al., 2013; Erick, 2012; Sahu et al., 2009).

Five to 11% of all pregnancies are complicated by hypertension (Villar et al., 2004). The South African Saving Mothers Report of 2005-2007 reported 622 maternal deaths due to hypertensive disorders (National Committee on Confidential Enquiries, 2009). Globally up to 40 000 maternal deaths are caused by hypertension (Moodley, 2011; Villar et al., 2004) making up 12-20% of all cases of maternal mortality (Judith et al., 2003).

Although not fully understood, according to current consensus, PIH is believed to be caused by insufficient placental flow (Heimrath et al., 2014). Certain factors may increase the risk of PIH, including low levels of vitamin D, high pregnancy weight gain and environmental exposure to lead (Yazbeck et al., 2009; Getahun et al., 2007). Multiple foetuses, diabetes, chronic hypertension, being of African American descent, and advanced maternal age have also been associated with an increased risk of preeclampsia (Getahun et al., 2007; Ananth & Basso, 2010).

Treatment of PIH depends on the severity of the hypertension, and controversy exists around which drug is the best to use (Magee et al., 2010). Magnesium sulfate has been used to prevent eclamptic seizures and to treat preeclampsia and eclampsia (Euser & Cipolla, 2009; Erick, 2008). Moderate restrictions of sodium intake, as well as an increase in the intake of calcium and magnesium rich foods (e.g. milk, soybean products, leafy vegetables and cheese) may reduce the risk of PIH (Indumati et al., 2011; Hofmeyer et al., 2010).

2.4.9 Maternal age

Pregnancy in adolescence

The 2003 South Africa Demographic and Health Survey (SADHS) reports that during the study period, 27% of women younger than 19 years of age gave birth. Like pregnancy, adolescence is a period of rapid development and tissue and bone growth leading to increased nutritional requirements (Williamson, 2006). Furthermore, adolescents in general follow diets

that are micronutrient-poor (Northstone et al., 2013) and lacking especially in folate, iron, calcium and vitamin D (Erick, 2012; Moran, 2007). Adolescent pregnancies are more likely to result in preterm delivery, LBW and SGA (Calvo & Lopez, 2012; Baker et al., 2009; Chen et al., 2007). Regular prenatal care, adequate dietary guidance, and avoidance of alcohol can help decrease the risks of these adverse effects (Erick, 2012).

Pregnancy in older women

Of all the babies born in South Africa between 1998 and 2003, approximately 15% were born to mothers over the age of 35 (SADHS, 2003). Older women (≥35 years of age) may be at increased risk for hypertension, miscarriage, ectopic pregnancy (Mills & Lavendar, 2010; Yogev et al., 2010; Huang et al., 2008), still birth, pre-term delivery, caesarean delivery (Laopaiboon et al., 2014; Flenady et al., 2011; Aliyu et al., 2008; Delbaere et al., 2007; Joseph et al., 2005), macrosomia (Kenny et al., 2013), and LBW (Aliyu et al., 2008; Joseph et al., 2005). Advanced maternal age is also strongly linked with chromosomal birth defects, including trisomies 13, 18, and 21 (Gill et al., 2012; Hagen et al., 2011; Cocchi et al. 2010; Loane et al., 2009).

2.4.10 Vegetarianism

Vegan and vegetarian diets exclude foods from animal origins, but to varying degrees. Vegetarian diets can be either lacto-ovo- (allowing milk and eggs but no other foods derived from animals) or ovo-vegetarian (including eggs but no milk or other foods derived from animals), while a vegan diet excludes all foods derived from animals (Craig & Mangels, 2009). According to the American Dietetic Association position statement of 2009, Lacto-ovo vegetarians following a well-balanced and varied diet have no additional nutritional concerns compared to their non-vegetarian counterparts. Vegetarian (lacto-ovo and vegan) diets have also been associated with a lowered risk of cardiovascular disease, some forms of cancer, type 2 diabetes and hypertension (Roman et al., 2014; Craig, 2010; Craig & Mangels, 2009; Mangels, 2008). Following a vegetarian diet during the first trimester has also been shown to decrease the risk of excessive weight gain (Craig, 2010; Stuebe et al., 2009).

However, vegetarian diets may be low in bioavailable iron and are likely to be deficient in vitamin D, vitamin B12, calcium, zinc, omega-3 fatty acids, and riboflavin because of the exclusion of meat, fish and other animal products, especially vegan diets. These nutrients may need to be supplemented, especially during pregnancy (Craig, 2010; Penney & Miller,

2008; Mangel, 2008; Williamson, 2006; Hronek & Kudlácková, 2005), and especially in women adhering to vegan diets (Koletzko et al., 2012). Supplementation with iron is recommended in these women (Craig, 2010; Cox, 2008) because of their increased risk of developing iron deficiency anaemia (Hronek & Kudlácková, 2005; Hercberg et al., 2001), as is fatty acid supplementation to counter the lack of oily fish intake (Craig, 2010; Cox, 2008).

A vitamin B12 deficiency during pregnancy can cause neurological impairment and growth failure in the infant (Bhate, 2012; Veena et al., 2010; Brown, 2008; Hronek & Kudlácková, 2005). Hronek & Kudlácková (2005) also confirmed an increased risk of abortion, hypoproteinaemia and oedema in the infant born to vegetarian mothers. Some (Hronek & Kudlácková, 2005, Sander, 1994), but not all studies show that vegetarian and vegan diets lead to LBW deliveries (Roman et al., 2014; Drake et al., 1998; Lakin et al., 1998).

Individual assessment and counselling is crucial in pregnant women following vegetarian and vegan diets to ensure adequate nutritional intake (Roman et al., 2014; Koletzko et al., 2012; Penney & Miller, 2008; Cox, 2008; ADA, 2003; Sanders, 1994).

2.4.11 Caffeine consumption

Caffeine acts as a central nervous system stimulant and can reach the foetal brain by crossing through the placenta and foetal blood-brain barrier (Greenwoode et al., 2014; Mose et al., 2008). A high intake of caffeine has been associated with LBW (Vlanjinac et al., 1997), miscarriage (Greenwood et al., 2014; 2010; Weng et al., 2008; Wen et al., 2001) and behavioural problems (Bekkhus et al., 2010) by some, but not by others (Loomans et al., 2012; Clausson et al., 2002; Santos et al., 1998).

Although studies on the effect of caffeine on the foetus are inconclusive and contradictory, current recommendations are to consume no more than 300mg of caffeine a day (Greenwood et al., 2014; ADA, 2008; COT, 2001). Table 2.13 shows the average estimated caffeine content in various foods and drinks. Variation amongst different brands can be extensive and checking labels is thus very important (Greenwood et al., 2014; Williamson, 2006). It is also important to bear in mind that various drugs and over-the-counter medicines may also contain caffeine (COT, 2001; Carrillo & Benitez, 1996).

TABLE 2.13 *Caffeine content of commonly consumed foods and drinks* (Williamson, 2006; COT, 2001)

ITEM	SERVING SIZE	CAFFEINE CONTENT (mg)
Instant coffee	190mL	± 75
Brewed coffee	190mL	± 100-115
Decaffeinated coffee	190mL	± 4
Tea	190mL	± 50
Green tea	190mL	± 24
Drinking chocolate	200mL	1.1 – 8.2
Energy drinks with added caffeine	250mL	28 – 87
Cola	330mL	11 – 70
Chocolate	50g	5.5 – 35.5

2.5 Nutrition related pregnancy outcomes

2.5.1 Infant birth weight

Infant birth weight may impact on the risk for various complications or diseases in later life (Anderson, 2008). Premature, SGA (birth weight <10th percentile), LBW (<2500g), very low birth weight (VLBW) (<1500g) and extremely low birth weight (ELBW) (<100g) infants have greater morbidity and mortality rates than those born at full term, and at appropriate birth weights (Pulver et al., 2009; Khashu et al., 2009; Anderson, 2008; Rosenberg, 2008). It has been shown that normal weight infants have better mental development and an overall better health status with a lower risk of developing heart disease, diabetes, lung disease, end-stage renal disease and hypertension in later life (Christian et al., 2014; Conen et al., 2010; Evansen et al., 2009; Aarnoudse-Moens et al., 2009; Vikse et al., 2008; Whincup et al., 2008; Eichenwald & Stark, 2008; Rich-Edwards et al., 2005).

Most infants born SGA have experienced some form of intra-uterine growth restriction (IUGR) where suboptimal uterine conditions or poor nutrition during critical stages of development cause prioritisation of nutrients to vital organs rather than to growth (Cianfarani et al., 2012; Cetin & Alvino, 2009; Rosenberg, 2008; Hales & Barker, 2001). IUGR manifests symmetrically with both head circumference and length below the 10th percentile, or asymmetrical with length and head circumference between the 10th and 90th percentiles (Anderson, 2012; Cox & Marton, 2009). Symmetrical IUGR indicates early and chronic

intrauterine deficits and can also be referred to as 'impaired growth potential' because all organs are equally adversely affected (Cox & Marton, 2009). Symmetrical IUGR is generally associated with a greater risk for later growth and developmental problems (Anderson, 2012). Asymmetrical IUGR can be viewed as 'impaired foetal nutrition' and is often caused by some pathology of the placenta (Cox & Marton, 2009). IUGR infants are at increased risk for perinatal asphyxia and chronic lung disease, hypocalcaemia, hypoglycaemia, hypothermia, heart disease, NAFDL, type 2 Diabetes, obesity and hypertension (Crume et al., 2014; Anderson, 2012; Cianfarani et al., 2012; Cetin & Alvino, 2009; Brown, 2008; Rosenberg, 2008).

Large for gestational age (birth weight >90th percentile) (LGA) is associated with pre-pregnancy obesity, uncontrolled maternal diabetes and excessive maternal weight gain during pregnancy (Jansson et al., 2006; Ehrenberg et al., 2003; Lampl & Jeanty, 2002; Kramer et al., 2002). Several studies show that infants born LGA are at an increased risk for becoming overweight or obese adults (Mehta et al., 2011; Lawlor et al., 2006; Whitaker, 2004; Catalano et al., 2003; Martorell et al., 2001). Furthermore, the risk of asphyxia and shoulder dystocia during birth is also increased in LGA infants (Zhang et al., 2008; Jones, 2001). Although the rates of emergencies in labour with LGA infants are greater (Heslehurst et al., 2008; Jansson et al., 2006), their risk of illness, infant death rate and general health problems are lower than their SGA counterparts (Brown, 2008).

2.5.2 Perinatal mortality

Perinatal mortality refers to the number of infant deaths occurring between 28 weeks gestation and four weeks postpartum (Erick, 2012). Nutritional status of women before or during pregnancy may influence perinatal mortality. Risk factors for perinatal mortality relating to nutritional status include LBW and especially VLBW (Carlo et al., 2010; Hoyert et al., 2006). However, this association follows an inverted J-shaped curve; mortality decreases with increasing birth weight up to a point, after which the slope reverses and mortality rate increases with increasing birth weight (Graafmans et al., 2002; Wilcox, 2001). A high or low maternal pre-pregnancy BMI, inappropriate weight gain during pregnancy, development of gestational diabetes (Gallagher, 2008; Erick, 2012), multiple deliveries, premature births and teenage pregnancies also contribute to perinatal mortality (MacDorman & Kirmeyer, 2009; Anderson, 2008; Carlo et al., 2010).

Other non-nutrition-related risk factors for perinatal mortality include necrotising enterocolistis (NEC), cerebral palsy (CP), respiratory distress syndrome (RDS), intraventricular haemorrhage (IVH), developmental delays and learning disorders (including attention deficit hyperactivity disorder (ADHD)) (Gallagher, 2008; Erick, 2008).

2.5.3 Foetal programming

Foetal programming refers to any insult or stimulus that affects some aspect of foetal development during a sensitive or critical period (Langley-Evans, 2014; Barker, 2002). The intrauterine environment, including availability of energy and nutrients during foetal development, may program and permanently alter the structure and functionality of cells and genes, and thus the metabolism of the body (Barker & Thornburg, 2013; Drake & Walker, 2004; Fleming et al., 2004). Physiologically, maternal under nutrition causes a decreased blood supply to the placenta, which impairs placental development and disrupts the flow of nutrients from mother to foetus (Hanson et al., 2011; Erick, 2008; Lederman, 1985). Inadequate nourishment during pregnancy affects the foetus proportionately more than the mother as her requirements will be met before those of the placenta and foetus respectively (Brown, 2008).

Children born to mothers that were affected by famine during their pregnancy suffered adverse effects from the dietary restriction (Erick, 2012; Barker 2006; Hulshoff et al., 2000). For instance, the offspring of women exposed to the Dutch Famine during the first trimester of their pregnancy had stunted foetal brain development (Bale et al., 2010; Roseboom et al., 2006). Furthermore, increased risk of spontaneous abortion, stillbirths, congenital defects, LBW and SGA births was found (Erick, 2012; Langley-Evans, 2014).

Foetal programming may influence the development of chronic diseases later in life as reported by studies conducted in developed (Langley-Evans, 2014; Barker & Thornburg, 2013; Roseboom et al., 2006; Kaiser & Allen, 2002; Barker, 1990) and developing (Victoria et al., 2008) countries. It has been shown that individuals that were born with a LBW have a greater tendency to increased stores of abdominal fat, greater risk for developing hypertension, glucose intolerance (and associated DM), increased risk of CVD, DM, hypertension, hypercholesterolemia and hypertriglyceridaemia (Langley-Evans, 2014; Victoria et al., 2008; Symonds et al., 2007; Barker, 2006; McMillen & Robinson, 2005; Kajantie et al., 2005; Syddall et al., 2005).

2.5.4 Effects of alcohol consumption during pregnancy

Alcohol is metabolised in the liver by two pathways. When consumed in moderate amounts and/ or only occasionally, it is metabolised by Alcohol dehydrogenase (ADH) to acetaldehyde in the liver with the transfer of hydrogen to NAD. Acetaldehyde is then converted to acetate by aldehyde dehydrogenase and released into the blood. Acetate is broken down to acetyl CoA in the muscle tissue and stored as fat or used in the TCA cycle (Hasse & Matarese, 2012; Gallagher, 2012; Edenberg, 2007; Shankar et al., 2007; Lieber, 2003). Thiamine and niacin are needed for the proper functioning of ADH. Excessive alcohol intake or insufficient NAD activates the Microsomal Ethanol Oxidising System (MEOS) which also converts alcohol to acetaldehyde in the liver (Gallagher, 2012; Lieber, 2003). The acetaldehyde product of both these alcohol-metabolising systems can itself be toxic and lead to liver cirrhosis (Gallagher, 2008; Edenberg, 2007; Lieber, 2003).

Numerous studies have shown that alcohol consumption during pregnancy results in adverse effects for both mother and foetus. It is generally known that alcohol consumption during pregnancy may be teratogenic as it easily crosses the placenta and accumulates in the foetal blood stream (Kodituwakku & Kodituwakku, 2013; May et al., 2013; Warren et al., 2004; Warren & Hewitt, 2009). Because the foetal organs are not yet fully developed, the liver is not able to metabolise alcohol (Smith et al., 2014; Gallagher, 2012). The series of adverse effects that can occur in infants born to mothers that consumed alcohol during pregnancy are encompassed in the term Foetal Alcohol Spectrum Disorder (FASD) (May et al., 2013; Manning & Hoyme, 2007; Riley & McGee, 2005). Foetal alcohol syndrome is the most severe form of FASD and is the result of chronic consumption of high doses of alcohol (Kodituwakku, 2009; Bertrand et al., 2004). South Africa has the highest prevalence of FAS and FASD in the world, with the Western Cape (rates between 40.5 and 89.2 per 1,000 children) (May et al., 2013b; May et al., 2007; Viljoen et al., 2005; May et al., 2000) and the Northern Cape Province (rates between 119.4 and 74.7 per 1,000 children) (Urban et al., 2008) being specifically afflicted. The prevalence of FAS in the USA is estimated at 0.2 to 1.5 cases per 1,000 (CDC, 1993).

Occasional social or binge drinking also increases the risk of spontaneous abortions, preterm birth, LBW delivery, traumatic labour, developmental delays, mental retardation and cognitive compromise (Erick, 2012).

Besides the teratogenic effect, alcohol consumption also affects nutritional status. Primary malnutrition occurs when alcohol replaces intake of other nutrients. While alcohol contributes 7kcal/g (30kJ/g) of energy, it provides very few other nutrients, and could result in pregnant women not meeting their increased nutrient requirements (Gallagher, 2012). Alcohol can also result in secondary malnutrition, which refers to nutritional deficiency caused by the altered efficiency or ability of the body to break down and absorb nutrients (Lieber, 2003). Alcohol has been shown to suppress bone formation (Maddalozzo et al., 2009; Turner, 2000) and impairs the transport of folate to the foetus (Hutson et al., 2012). The uptake of essential amino acids and the production of protein in the liver is also decreased (Erick, 2012; Keen et al., 2010; Adibi et al., 1992), which directly compromises meeting the the increased protein requirements during pregnancy. Alcohol also affects the absorption, metabolism and excretion of nutrients that are essential in blastogenesis and cell differentiation, including vitamins B1, B3, B6, magnesium and zinc (Erick, 2012; Keen et al., 2010; May et al., 2004; Tamura et al., 2004).

As no safe level and safe time to consume alcohol during pregnancy are known (Cheng et al., 2011; Gray et al., 2009) it is recommended that pregnant women avoid alcohol consumption completely (Surgeon General, 2005).

2.6 Nutritional status assessment in pregnant women

Assessment of anthropometry, biochemistry, clinical signs and symptoms, and dietary intake is used in clinical settings to provide a comprehensive picture of nutritional status of individuals or groups. In a community setting, dietary assessment forms an important part of nutritional science, being used to identify public health nutrition problems and associations between dietary intake, health and disease (Adamson & Baranowski, 2014; Biltoft-Jensen et al., 2009; Ma et al., 2009). Various dietary intake assessment methods are available (see section 2.6.1 and Table 2.12) and an appropriate method should be selected based on the characteristics of the community and the aims of the study. Recent developments in this area aim to increase ease of use and improve validity and reliability of dietary intake data, by employing various technologies (Adamson & Baranowski, 2014; Baranowski et al., 2014; Lewis et al., 2014; Kirkpatrick et al., 2014).

2.6.1 Overview of dietary intake assessment methods

Several dietary intake assessment methods are discussed below and a summary of the advantages and disadvantages of each method is summarised in Table 2.14.

Daily food diary (prospective recording)

When using this method, the patient is required to record what and how much is eaten as it occurs throughout the day in a food diary. Patients may also be required to indicate the place, time and reason for eating. Quantities are recorded either in common household measures or by actual weighing of portions (Lee & Nieman, 2010; Gibson, 2005b; de Castro, 1999). The desired period to record food intake in a food diary is three to seven days (Hammond, 2012; Biltoft-Jensen et al., 2009). Food records can also be used to determine eating patterns and identify problem areas in the diet. The quality of the food diary data is dependent on the individual's honesty and accuracy of recording (Hammond, 2008). Furthermore, factors such as the 'experimenter' and 'fatigue' effects may impact negatively on results and thus need to be considered (Biltoft-Jensen et al., 2009).

Food Frequency Questionnaires (recall of past intake)

Food Frequency Questionnaires (FFQ) involve retrospective recall of the frequency of intake of certain foods. The food items are presented in a list and subjects are required to indicate how often they consumed each item per week, month, or year period (Hammond, 2012; Lee & Nieman, 2010; Gibson, 2005b). The questionnaire can be interviewer- or self-administered. FFQs can be simple or non-quantitative (no portion size included, only frequency recorded), semi-quantitative (using estimates of standardised portions such as small, medium or large), or quantitative (includes specific portion sizes including weight, volume or household measures: QFFQ) (Lee & Nieman, 2010; Gibson, 2005; Thompson & Byers, 1997). The items in the FFQ food list should be aligned with the aim of the dietary intake assessment component of the research strongly impacts on to ensure that the research question can be answered It needs to be noted that a small number of items may provide a less clear/valid estimate of intake, while a comprehensive QFFQ could give more detailed information and a more valid estimation of usual total energy and nutrient intakes (Cade et al., 2004, 2002). FFQs focussing on assessing specific nutrients would generally include less items than those aiming to determine overall usual dietary intake (Cade, 2001). However, it has been proposed that very long questionnaires offer little extra value and increase respondent burden (Willett, 1998). It has been recommended that a FFQ should be developed

and validated for specific target groups to account for the possible differences in culture, environment, availability of foods (Torheim et al., 2001), race/ethnicity and education (Kristal et al., 1997).

24-Hour recall (recall of past intake)

This method is usually interviewer-administered and relies on the individual to remember the detail of the foods consumed in the past 24 hours, as well as the amount (portion size) of each food consumed (Hammond, 2012; Lee & Nieman, 2010; Gibson, 2005b). The interviewer can assist the respondent to remember foods consumed by asking questions about activities or events that took place on the day in question. Brand names, preparation methods and set serving sizes/units should be specified to increase accuracy of analysis. As a single 24-hour recall does not provide a valid reflection of usual, it is recommended that the recall be combined with a FFQ (Hammond, 2012), or that repeated 24-hour recalls (two to five) be conducted (Cade et al., 2002).

TABLE 2.14 A*dvantages and disadvantages of food records, FFQs and 24-recall(s) for dietary intake assessment*

METHOD	ADVANTAGES	DISADVANTAGES
Food diary/ Estimated / Weighed food record	- Does not rely on memory of participant - Greater reproducibility (weighed food record) (Gibson, 2005) - Least correlated errors with FFQ (Cade et al., 2002) - Reflection of usual intake if recorded over four (Smithers et al, 1998) to seven days (Carlsen et al., 2010; Bingham et al., 1994)	- Demanding method requiring literate respondents (Gibson, 2005; Rockett et al., 1997) - Respondents may fail to quantify portions correctly (in estimated food records) (Gibson, 2005) - Increased costs if scales are used - Greater participant motivation needed (Gibson, 2005; Rockett et al., 1997) - Participants may adapt eating habits to make recording easier (Gibson, 2005; Trabulsi & Schoeller, 2001; Jacques et al., 1993) - Significant under-reporting may occur (Gibson, 2005; Cade et al., 2002)
Food Frequency Questionnaire	- Relatively lower administrative costs and less time consuming on the participant (Sevak et al., 2004; Mayer-Davis et al., 1999; Resnicow et al., 1997; Jacques et al., 1993)	- Relies on memory (Adamson & Baranowski, 2014; Cade et al., 2002; Trabulsi & Schoeller, 2001) - Requires conceptualisation of portion sizes (Adamson & Baranowski, 2014; Cade et al.,

METHOD	ADVANTAGES	DISADVANTAGES
	- Ability to assess usual intake, and intake over a longer period of time (Gibson, 2005; Black et al., 1993; Mayer-Davis et al., 1999; Resnicow et al., 1997) - Data is easy to collect and process (Gibson, 2005; Sevak et al., 2004) - Can be self-administered and used in large populations (Sevak et al., 2004; Black et al., 1999)	2002; Trabulsi & Schoeller, 2001) - Limited ability to quantify absolute intake (Torheim et al., 2001; Resnicow et al., 1997) - Over- (Sevak et al., 2004; Torheim et al., 2001) and under- (Subar et al., 2003) estimation has been found - Validity partly dependent on completeness of lists (Mayer-Davis et al., 1999)
24-Hour recall	- Inexpensive (Lee & Nieman, 2010) - Requires only short-term memory (Lee & Nieman, 2010) - Can be repeated during different seasons to estimate average food intake over a longer time (Gibson, 2005) - Lower respondent burden compared to weighed food record (Lee & Nieman, 2010; Cade et al., 2002; Black et al., 1999) - Less likely to influence actual diet of subjects (Cade et al., 2002) - Better suited to populations with less literate participants (interviewer administered) (Cade et al., 2002) - Reflection of usual intake with three recalls (Burrows et al., 2010; Ma et al., 2009)	- Gives information only on previous day's dietary intake (Rockett et al., 1995), thus single recall may not be representative of usual intake - Exaggerates estimate of individual variability (Rockett et al., 1995) - Relies on memory and conceptualisation of portion sizes (Cade et al., 2002; Trabulsi & Schoeller, 2001) - Requires an experienced interviewer - Prone to under-reporting (Trabulsi & Schoeller, 2001) especially of binge eating behaviours, alcohol, and 'unhealthful' foods (Lee & Nieman, 2010) - Prone to over-reporting of expensive or healthful foods (Feskanich & Willett, 1993) - More administration time may be needed to do multiple assessments (Resnicow et al., 1997)

Portion size estimation

The estimation of portion sizes can be challenging but is an important determinant of the accuracy of dietary intake data. It has been indicated that inaccurate portion size estimation is one of the major sources of reporting error in recall methods (Rumpler et al., 2008). In order to assist with portion size estimation, numerous portion size estimation aids (PSEA) have been developed (Subar et al., 2010; Biro et al., 2002). These include two-dimensional (food photographs, drawings of food or household measures/ abstract shapes, package labels, or computer graphics) or three-dimensional (actual food samples, food replicas or models, household measures) aids (Biro et al., 2002; Cypel, 1997).

Factors affecting portion size estimation include perception, conceptualisation, and memory (Biro et al., 2002; Nelson et al., 1994). Studies assessing these factors showed that age, type of PSEA used and the type of food being estimated affected the participant's perception of the portion size (Steyn et al., 2006; Hernandez et al., 2006; Byrd-Bredbenner & Schwartz, 2004), the accuracy of conceptualisation and ability to recall meals (Foster et al., 2008; Hernandez et al., 2006; Chambers et al., 2000; Nelson et al., 1994; Faggiano et al., 1992). Lower literacy levels have been linked with less accuracy in portion size estimation (Huizinga et al., 2009). Actual portion sizes consumed inversely impact the reported estimation where larger portions tend to be underestimated and smaller ones overestimated (Harnack et al., 2004; Faggiano et al., 1992)

Food photographs have been found to be cheap, reproducible, transportable, and easily adaptable to different communities (Huybregts et al., 2007). Furthermore, it is suitable for groups and in rural settings (Huizinga et al., 2009). Images of household measures or of different portion-sized line drawings have been reported to be as accurate as images of actual food, making it more cost-effective (Subar et al., 2010). It has been found that the addition of photographs to a traditional food diary and self-administered 24-hour recall enhanced the validity and reliability of dietary intake recording (Small et al., 2009; Subar et al., 2010). Subar et al. (2010) and Nelson et al. (1994) also found that the use of more images (eight vs. four images and four vs. one respectively) attained more accurate data. However, some studies assessing the validity of food photographs have shown significant portion size estimation errors when compared to plated food weighed before and after the meal in question (Robson & Livingstone, 2000; Faggiano et al., 1992) and also inter-individual variability depending on the portion size depicted in the photograph (Ovaskainen et al., 2008; Nelson et al., 1994).

Several studies have shown that a variety of PSEAs used together achieve more accurate estimates of intake (Subar et al., 2010; Byrd-Bredbenner & Schwartz, 2004; Faggiano et al., 1992; Hankin et al., 1991).

2.6.2 Interpretation of dietary intake data

There are several ways in which dietary intake data can be interpreted, including consideration of dietary adequacy and dietary diversity.

Dietary adequacy

Comparison of mean energy and nutrient intakes to respective DRIs and to the FBDG are commonly used to assess the adequacy of dietary intake of individuals/groups. Two ratios, namely the nutrient adequacy ratio (NAR) and the mean adequacy ratio (MAR) can also be used to gauge the adequacy of dietary intake. The NAR is the ratio of observed intake of a nutrient to the recommended intake for that nutrient (FAO/ WHO 2002). The MAR is a measure of overall dietary adequacy and is calculated as the sum of the NAR for each nutrient (except protein and energy) divided by the number of nutrients (Steyn et al., 2005; Hartloy et al., 1998). Both ratios are expressed as a percentage, and an ideal score of 100% would indicate an intake that is in line with requirements (Steyn et al., 2005). Neither the NAR nor the MAR has been used for the assessment of the adequacy of the dietary intake of pregnant women in South Africa.

Dietary diversity

Dietary diversity scores (DDSs) have been developed to provide insights into the variety of foods consumed by individuals/groups. DDSs are generally defined as the number of food groups consumed over a 24-hour period (Sealy-Potts & Potts, 2014; Steyn et al., 2005; Hatloy et al., 1998). A number of national and international research groups have developed DDSs (see Table 2.15 for examples of DDSs developed for developing countries). DDSs developed by different groups may have elements in common, but may differ depending on the aim of a particular study (Hatloy et al., 2000). There is no international consensus on which food groups should be included in the calculation of the DDS (Kennedy et al., 2012).

TABLE 2.15 *Examples of Dietary Diversity Scores (DDSs) applied in studies in developing countries*

	1	2	3	4	5	6	7	8
Household vs. Individual DDS	Individual	Household	Household	Household	Individual	Individual	Household	Individual
Sample age	Pre-schoolers 2-4 years	Average 56.1± 15.7 years	Average 33.5 years	16 years and older	Female household heads	Children 1-8 years	Children 6 – 59 months	Children 13 – 58 months
Country	Republic of Trinidad, South America	Limpopo province, South Africa	Johannesburg, South Africa	Nine provinces of South Africa	Vaal region, South Africa	Nationally representative sample of South Africa	Koutiala, Mali	Koutiala, Mali
Dietary intake method used	Single 24-hour recall	Non-quantitative food group questionnaire	Single non-quantitative 24-hour recall	Single 24-hour recall	7-day QFFQ	24-hour recall	Single FFQ	Three consecutive weighed food records
Cut-off for inadequate DDS	≤ 3		< 4	< 4	≤ 4	≤ 4	2 – 5	< 6
Mean DDS	4.19 ± 0.83	4.57 ± 1.96	4.1 (3.2 and 4.8 informal vs. formal settlements)	4.02	2.82 ± 0.99	3.6 ± 1.4		5.8 ± 1.1
% below cut-off	19.15%		36.8% (68.1% in informal and 15.4% in formal settlements)	Province varied; Lowest Western Cape: 15.7% Highest Eastern Cape: 59.6%	79.5%			

	1	2	3	4	5	6	7	8
Food groups:								
- Staples (grains, cereals, roots, tubers)	X						X	X
- Cereals, roots and tubers		X	X	X	X	X		
- Dairy (milk, yoghurt, cheese)	X	X	X	X	X	X		X
- Legumes and nuts	X			X		X	X	
- Legumes		X	X		X			
- Animal/ flesh foods (eggs, meat, fish, poultry, liver/ organ meat)	X							
- Meat, poultry and fish		X	X	X	X	X	X	
- Eggs		X	X	X	X	X	X	X
- Meat								X
- Fish								X
- Leaves/ gathered foods							X	X
- Vitamin A-rich fruits and vegetables	X	X	X	X	X	X		
- Other fruits and vegetables	X	X						

	1	2	3	4	5	6	7	8
- Other fruits			X	X	X	X	X	X
- Other vegetables			X	X	X	X	X	X
- Fats and oils		X	X	X		X		
- Oil/ Sugar							X	
- Sugar		X						
- Beverages		X						

Abbreviations: FFQ = Food frequency questionnaire QFFQ = Quantitative food frequency questionnaire

1. Sealey-Potts & Potts, 2014 2. De Cock et al., 2013 3. Drimie et al., 2013 4. Labadarios et al., 2011 5. Oldewage-Theron & Kruger, 2011
6. Steyn et al., 2006 7. Hatloy et al., 2000 8. Hatloy et al, 1998

Four of the studies included in Table 2.15 were done in children between the ages of six months and eight years (Sealey-Potts & Potts, 2014; Steyn et al., 2006; Hatloy et al., 2000; Hatloy et al., 1998), while the rest of the studies were done in adults or adolescents of 16 years or older (De Cocl et al., 2013; Drimie et al., 2013; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011). Five of the studies were done in various provinces in South Africa (De Cock et al., 2013; Drimie et al., 2013; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011; Steyn et al., 2006) and the remaining three in other developing countries. Different dietary intake methods were used in the studies to assess the dietary intake data from which the DDS was derived. The most commonly used method was a single 24-hour recall (four studies) (Sealey-Potts & Potts, 2014; Drimie et al., 2013; Steyn et al., 2011; Steyn et al., 2006) followed by a the FFQ method (two studies) (Oldewage-Theron & Kruger, 2011; Hatloy et al., 2000).

The food groups used in the studies were not necessarily aligned. For example, one study did not include dairy products, (Hatloy et al., 2000); three studies combined legumes and nuts (Sealey-Potts & Potts, 2014; Steyn et al., 2006; Hatloy et al., 2000), while four studies included legumes only (De Cock et al., 2013; Drimie et al., 2013; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011); and one study included neither legumes nor nuts (Hatloy et al., 1998). Half of the studies included fats and oils as a food group (De Cock et al., 2013; Drimie et al., 2013; Labadarios et al., 2011; Steyn et al., 2006), one study combined oil and sugar (Hatloy et al., 2000), while the remaining three studies did not include fats or sugars (Sealey-Potts & Potts, 2014; Oldewage-Theron & Kruger, 2011; Hatloy et al., 1998). These variations are in line with recommendations by the FAO where fats and oils are excluded from assessment when analysis of individual DDS is done (Kennedy et al., 2012) except for the study by Steyn et al. (2006) where individual DDS analysis was done with the inclusion of fats and oils as a food group. Only one study classified sugar as a food group. The same study was also the only one to classify beverages as a food group (De Cock et al., 2013). One study included a combined food group containing meat, poultry and fish, along with eggs (Sealey-Potts & Potts, 2014), while six studies separated eggs and animal flesh into two different food groups (De Cock et al., 2013; Drimie et al., 2013; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011; Steyn et al., 2006; Hatloy et al., 2000). Hatloy et al. (1998) divided these foods into separate groups for meat, fish and eggs. Two studies included a food group for leaves and gathered foods (Hatloy et al., 2000; Hatloy et al., 1998). Six studies included a food group for Vitamin A-rich fruits and vegetables (Sealey-Potts & Potts,

2014; De Cock et al., 2013; Drimie et al., 2013; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011; Steyn et al., 2006). Other fruits and vegetables were combined into a single category in two of the studies (Sealey-Potts & Potts, 2014; De Cock et al., 2013), and separated into two categories in the other six studies (Drimie et al., 2013; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011; Steyn et al., 2006; Hatloy et al., 2000; Hatloy et al., 1998).

Studies that investigated the association between dietary diversity and micronutrient intake have found positive correlations (Narmaki et al., 2015; Steyn et al., 2005; Foote et al., 2004; Hatloy et al., 1998). Associations were also found to be more likely to be significant in rural when compared to urban areas (Amugsi et al., 2014; Drimie et al., 2013; Ruel, 2003). Dietary diversity has also been linked to food security (Hillbruner & Egan, 2008; Swindale & Bilinsky, 2005) with a score below four indicating poor dietary diversity, and poor food security (Labadarios et al., 2011). In general, lower DDSs were found in those living in informal areas (Drimie et al., 2013; De Cock et al., 2013; Labadarios et al., 2011; Steyn et al., 2011; Oldewage-Theron & Kruger, 2011). As stated by the FAO, no established cut-off points are available (Kennedy et al., 2012), but the majority of studies summarised used a score below four as being indicative of poor dietary diversity, and poor food security (Drimie et al., 2013; Labadarios et al., 2011; Oldewage-Theron & Kruger, 2011; Steyn et al., 2006; Hatloy et al., 2000).

2.6.3 Anthropometric assessments

Anthropometry refers to physical measurements that are obtained from an individual that can be compared to standards to analyse and monitor the growth and development of the individual (Hammond & Litchford, 2012). In pregnant women, BMI and weight monitoring gives an indication of foetal growth and health (Calvo & Lopez, 2012; Lopez et al., 2011) and may predict longer term health in both the mother and child. Specifically, weight and BMI monitoring throughout pregnancy has been shown to help in preventing high blood pressure in the offspring during adolescence (Laura et al., 2010). However, weight and BMI values are only effective monitoring tools if pre-pregnancy weight and BMI are known, which is often not available in developing communities (Calvo & Lopez, 2012).

Mid upper arm circumference (MUAC) has been shown to be a suitable substitute for BMI, because of the strong correlation thereof with body weight (Ogbonna et al., 2007; Thame et

al., 2007; Kelly et al., 1996; Pelletier et al., 1995; James et al., 1994). This has been shown to hold true in pregnancy (Calvo & Lopez, 2012; Lopez et al., 2011), with MUAC during early pregnancy being a better predictor of prematurity (Liljestrand & Bergström, 1991) and LBW (Lopez et al., 2011; Ojha & Malla, 2007; Kruger, 2005) than maternal weight gain or BMI. However, more research needs to be done to evaluate the variation of MUAC throughout pregnancy (Araújo et al., 2009, Licitra et al., 1998; Zekan et al., 1998). Table 2.16 shows the MUAC cut-offs suggested by Lopez et al. (2011) to detect LBW pregnancies, while Table 2.17 shows values recommended by a study done on South African women to indicate risk of malnutrition and adverse pregnancy outcomes based on MUAC readings (Kruger, 2005).

TABLE 2.16 *Proposed MUAC indices to detect low birth weight pregnancies* (Lopez et al., 2011)

GESTATION (weeks)	**MUAC (cm)**
16	<24.5
28	<25.5
36	<26.5

Abbreviations: MUAC = Mid-upper arm circumference

TABLE 2.17 *MUAC cut-offs as indicators of malnutrition during pregnancy* (Kruger, 2005)

MUAC (cm)	**INDICATOR OF MALNUTRITION**
<25	Warning
<23	Strong indication
<22	High risk of adverse pregnancy outcomes High risk of LBW

Abbreviations: MUAC = Mid-upper arm circumference LBW = Low birth weight

Skin fold measurements reflect subcutaneous fat stores (Calvo & Lopez, 2012; Lopez et al., 2011). These measurements can be used in combination with weight to give an indication of body composition (subcutaneous fat stores versus muscle mass) (Widen & Gallagher, 2014; Lee & Nieman, 2010). Skinfold measures could give an indication of maternal nutritional status (Widen & Gallagher, 2014; Lopez et al., 2011), although more research is needed to identify changes in these measurements throughout pregnancy (Widen & Gallagher, 2014; Calvo & Lopez, 2012).

2.6.4 Biochemical assessments

Biochemical markers are objective and thus not subject to the same risk of false reporting as other methods of nutritional assessment, but may be more expensive, invasive and very nutrient specific (Litchford, 2012; Lee & Nieman, 2010; Cade et al 2002). Single-test values should be used only for screening purposes or to confirm other assessments, while comparison to previous results is preferable and more accurate (Litchford, 2012). Interpretation of biochemical results for pregnant women should be done with care because of the influence pregnancy may have on these markers. For example, plasma vitamin B12 levels drop during pregnancy due to haemodilution (Ball & Giles, 1964) and alterations in relevant binding and transport proteins (Morkbak et al., 2007). However, holotranscobalamin levels have been shown to remain unchanged during pregnancy and may be a good marker of vitamin B12 deficiency in pregnancy (Morkbak et al., 2007).

Several biochemical markers have been associated with negative outcomes in pregnancy, especially with preterm birth. Serum folate is possibly the best known marker in pregnancy, with a low value being significantly correlated with increased risk of neural tube defects. Low serum folate has also been associated with preterm birth and LBW (Refsum, 2001).

Increased plasma or serum total homocysteine concentrations have been associated with an increased risk for preeclampsia, preterm birth, IUGR, LBW and perinatal death (Vollset et al., 2000; Burke et al., 1998; Leeds et al., 1998)

Decreased serum vitamin D levels during pregnancy have been shown to affect foetal femoral development (Thomas & Demay, 2011; Mahon et al., 2010; Millen & Bodnar, 2008), increase the risk of preeclampsia (Bodnar et al., 2007), increase caesarean section births (Merewood et al., 2009), and have been associated with preterm birth (Shibata et al., 2011; Gaunekar & Crowther, 2004).

Other biochemical markers that may be associated with preterm delivery include elevated CRP (C-reactive protein) (Pannacciulli et al., 2001), low serum magnesium (Wojcicka-Jagodzinska et al., 1998; Kurzel, 1991), low total serum calcium and phosphorous (Wojcicka-Jagodzinska et al., 1998), and a low white blood cell count (Hsu et al., 2013). Both high and low haemoglobin concentrations in early pregnancy (Goel et al., 2003; Steer, 2000), as well

as elevated serum ferritin concentrations (>40µg/dL) at 34 weeks have also been associated with increased risk for preterm delivery (Hsu et al., 2013; Scholl, 1998).

2.7 Concluding remarks

Several physiological changes occur throughout pregnancy enabling the growth and development of a healthy foetus. Nutrition-related factors that influence pregnancy outcome highlighted the importance of pre-pregnancy weight status and adequate weight gain throughout pregnancy to lower the risk of complications. Dieting, caffeine and alcohol consumption and being adolescent or of advanced maternal age during pregnancy further increases the risk of adverse pregnancy outcomes, while vegetarians need to be vigilant to ensure adequate intake of all nutrients. Maternal requirements of energy, protein, carbohydrates, and several nutrients increases during pregnancy. Most notably, vitamin A, folate, calcium, iodine, iron and phosphorous. Dietary guidelines suggest incorporating more nutrient-dense foods into the diet while also restricting refined foods, and foods high in saturated fats and sugar. The need for supplementation should be assessed individually and recommended only as required. The use of multiple micronutrient supplements is not supported by adequate evidence.

A review of South African studies (n =7) assessing the dietary intake of pregnant women showed that dietary intake levels of vitamin C, vitamin B12, iron and folate were typically inadequate, with low intakes of fruits, vegetables, and meat. Dietary intake assessment methodology and interpretation criteria for adequacy varied substantially across these studies, making it challenging to provide an overall picture of the dietary adequacy of pregnant women. The sample sizes of these studies were also relatively small, and thus are not representative of all pregnant women in South Africa. For these reasons, more studies investigating the dietary intake of pregnant women in South Africa are needed. Various methods are available to assess the nutritional, anthropometric and biochemical status of pregnant women and to aid in monitoring these as the pregnancy progresses. The advantages and disadvantages of each method must be considered when analysing results from any method.

CHAPTER THREE

METHODS AND PROCEDURES

3.1 Study design

A cross-sectional study design was used to assess the dietary intake, adequacy and variety as well as anthropometric and socio-demographic information of pregnant women attending the Bishop Lavis MOU in Cape Town. For the purposes of this study data on socio-demographic and anthropometric variables that were collected after recruitment at baseline and three 24-hour recalls that were collected over a one-month period (for the validation of a food screener in the main PASS study) were included. The 24-hour recalls were collected on three different days of the week, each one approximately two weeks apart. One of the recall days for each participant was a Sunday.

3.2 Sampling and recruitment

3.2.1 Study population and sampling

The study population included all pregnant women who attended the Bishop Lavis MOU for their first antenatal appointment and agreed to participate in the main Safe Passage (PASS) study. The PASS study is a large multidisciplinary prospective study investigating various factors associated with foetal and infant mortality related to sudden infant death syndrome (SIDS) and still birth as well as poor pregnancy outcomes such as Foetal alcohol syndrome (FAS). From this population a subsample of pregnant women was included in the dietary assessments sub-study using a convenience sampling method (see section 3.2.3).

3.2.2 Inclusion and exclusion criteria

To be included, participants had to understand and speak either English or Afrikaans, be enrolled as a participant in the main PASS study, and be between 12 and 20 weeks (84 to 140 days) gestational age at the time of recruitment. The latter criteria on gestational age was included to minimise the impact of differing stages of pregnancy, for example the increased requirements of the second and third trimesters, and the increased risk of NVP during the first trimester, on dietary intake.

3.2.3 Recruitment process

Pregnant women attending the Bishop Lavis MOU on Monday, Tuesday, Thursday and Friday mornings were recruited for the main PASS study. All pregnant women in the waiting room at Bishop Lavis MOU, who attended the clinic for their first antenatal appointment, were invited into a private room for explanation of the main PASS study by the PASS study fieldworkers. They were subsequently offered the opportunity to sign up for the main study.

Recruitment for the dietary sub-study took place between August 2011 and August 2012. During this period the main PASS study explanation also covered the dietary component and women who were between 12 and 20 weeks' gestational age at the time of recruitment were also offered the opportunity to participate in the sub-study. Those who consented to both the main and sub-study were requested to stay for a further interview with the dietitian after they had completed the recruitment interview process with PASS study fieldworkers. The main PASS study interview was carried out by PASS study fieldworkers and lasted approximately one and a half hours.

In the recruitment interview with the dietitian, the sub-study itself and what was expected of the participant was explained in more detail. The dietitian then provided eligible participants with the opportunity to ask questions regarding the sub-study. Consent forms were available in both English and Afrikaans, as this catered to the vast majority of the population being targeted. This population was largely homogenous, with 99.6% of the South African Main PASS study sample being of mixed ancestory, and only 0.4% and 0.001% classified as "black" and "white" respectively. These were signed by those who volunteered for participation and were willing to commit to three sessions (one on the same day and two follow-up sessions) for collection of dietary intake information. A copy of the signed consent form was given to each participant. An appointment was then set for the first follow-up visit, which had to take place within two weeks of the recruitment interview on a different day of the week than the first interview. It was necessary for participants to be able to attend both follow-up sessions between two and four weeks apart. Participants that failed to arrive for a scheduled interview were contacted by the interviewer and, where possible, another interview was scheduled. If the participant could not be contacted, was unwilling to reschedule the appointment, or did not attend the rescheduled interview, they were classified as a 'drop-out' and another participant was recruited in their place.

3.2.4 Final sample

OpenEpi was used to calculate the sample size required for this study. The results of South African studies that investigated adequacy of dietary intake of pregnant women were used for these calculations. These studies showed that when intake was inadequate for an investigated nutrient it was mostly in the range of 20% or more of subjects (Bopape et al. 2008, Mostert et al. 2005, Tshitaudzi et al. 2003). For the final sample size calculation, a population size of 1 million, confidence level of 95%, precision/confidence limit of 6% and an anticipated

frequency of 20% were used and a sample of 139 participants was indicated. A total of 170 participants were recruited to accommodate possible drop out. Eighteen participants failed to return for follow-up appointments of which 15 did not arrive for the first follow-up appointment, while three others did not arrive for the second follow-up appointment.

Reasons for not attending the first and/ or second follow-up appointments included illness (n=1), poor perceived memory (n=3), misunderstanding of transport services offered (n=1) and change in work (n=1), boss (n=1), work hours (n=2), and work or home location (n=2). Attempts to contact these subjects to make new appointments were not always successful as a result of incorrect contact details given, or because of changed contact details that were not relayed to the dietitian or the main study administrative offices. Two participants, not included in these numbers, did not complete the recruitment process due to time constraints and administrative error. One other subject was excluded from the sub-study at the first follow-up as a review of her clinical records showed that she was over twenty weeks gestational age at recruitment, and she could not be contacted prior to the appointment. The 18 participants that dropped-out were excluded from data analyses and the final sample included is 152 participants who attended all data collection appointments. In total, 477 interviews were carried out (including drop-outs), and 456 of the 24-hour dietary recalls were included in the final analysis.

3.3 Measures and questionnaires

3.3.1 Anthropometric measures

Execution of measures

Anthropometric measures were done by the main PASS study fieldworkers during the recruitment interview. The fieldworkers were all trained nurses, and as such, had previous training in anthropometric assessments. A manual that included the maternal and infant protocol for anthropometric measurements, based on the NHANES anthropometry procedures manual included detailed guidelines and instructions for all anthropometrical measurements and calibration of equipment (CDC, 2004). All fieldworkers were trained according to the protocol. Laminated copies of the instructions were available in each room used for the recruitment interviews to allow fieldworkers to easily verify each step. The protocols were strictly followed, and annual site checks and re-training was carried out to ensure a maintained standard of quality.

The following anthropometric measures were obtained and analysed in this sub-study: height, weight, triceps skinfold (TSF), and mid-upper arm circumference (MUAC).

Height was taken to the nearest 0.1cm without shoes, socks or any other clothing that may hinder accurate measurements, using a wall-mounted stadiometer with European Union quality standard. The participant's head, back and buttocks touched the stadiometer and the head was held in the Frankfort horizontal plane when the measurement was taken. Weight was measured to the nearest 0.1kg with an electronic scale (Mellerware®) which was calibrated every six months, using standard test weights, according to specified protocols. Because of the stringent calibration protocols, the same scales were able to be used for the duration of the study. Participants wore light clothing, no shoes, and stood in the middle of the scale, with no other body part touching anything else. Triceps skinfold (TSF) measures were taken with a Harpenden calliper on the left arm, at the crossing between the midpoint of the acromium and olecranum processes, and at the posterior midline of the upper arm. The arm was hanging loosely by their side, and measurements were recorded to the nearest 0.2mm (Lee & Nieman, 2010; Hammond & Litchford 2012).

MUAC was measured using a non-stretchable measuring tape at the same midway line as used for the TSF measurement, ensuring no skin indentation, and was recorded to the nearest 0.1mm (Lee & Nieman, 2010; Hammond & Litchford 2012). All measurements were taken twice, and the average between them was used in statistical analyses. Participants were also asked to report their pre-pregnancy weight.

Interpretation of measures

The estimated pre-pregnancy BMI and current BMI were calculated by dividing estimated weight and current weight in kilograms by height in square metres. It is important to note that different interpretation criteria for pre-pregnancy BMI are applicable for adult and for adolescent females. For adult participants, pre-pregnancy BMI was interpreted using the following categories: underweight (BMI <18.5kg/m^2), normal weight (BMI between 18.5 and 24.9 kg/m^2), overweight (BMI between 25.0 and 29.9 kg/m^2), and obese (BMI ≥30 kg/m^2) (WHO, 2006b). The WHO BMI-for-age chart (z-scores) for females between the ages of 5

and 19 years was used for interpretation of the pre-pregnancy BMI of adolescent participants. The BMI of each of these participants was plotted and the corresponding category was noted for each adolescent: Severe thinness (below -3 SD), thinness (below -2 SD), normal weight, overweight (above +1 SD), and obese (above the +2 SD) (WHO, 2015). The BMI of adolescent participants was also classified using the tables compiled by Cole et al. (2000) (categorisation as overweight or obese) to render categorised results for adolescents that are comparable to adult participants. The current BMI of participants was not classified according to BMI categories as the meaningful interpretation thereof is not feasible. Current BMI was used to investigate associations with other variables.

Estimated weight change was calculated as current weight minus estimated pre-pregnancy weight. The resulting value was interpreted using the IOM (2009) recommendations for adequate weight gain in pregnant women (Table 2.3) based on pre-pregnancy BMI and gestational age at recruitment.

Recommended MUAC cut-offs as suggested by Kruger (2005) were used in this study sample. A measurement below 25cm was interpreted as a warning of malnutrition, a measurement below 23cm was a strong indication of the same, and a measurement below 22cm indicated a high risk of adverse pregnancy outcomes and LBW. No specific recommendations are available for the interpretation of TSF measures in pregnancy, but their use in calculating and assessing body composition is well established (Widen & Gallagher, 2014; Lee & Nieman, 2010). The MUAC and TSF were used to calculate arm muscle circumference (AMC), corrected arm muscle area (cAMA) and arm fat area (AFA) for women using the following equations (Lee & Nieman, 2010).

$$AMC = MUAC\ (cm) - (\pi \times TSF\ (mm)$$

$$cAMA\ (cm^2) = AMC\ (cm^2) / 4\ \pi - 6.5$$

$$AFA = [(TSF\ (cm) \times MUAC\ (cm)) /2] - [(\pi \times TSF^2\ (cm)) /4]$$

Arm fat area values less than 20cm^2 were classified as greater risk for poor pregnancy outcome (Friis et al., 2004).

3.3.2 Socio-demographic questionnaire

The following socio-demographic data were collected with an interview-administered questionnaire by the main PASS study fieldworkers: maternal and gestational age, ethnicity, marital status [married; partnered (boyfriend or girlfriend), living together; partnered (boyfriend or girlfriend), not living together; separated; divorced; single; widowed], years of formal education completed, monthly household income ("Don't know", "≤R500", "R500 – R1 000", R1 001 – R2000" "R2 001 – R3 000", R3 001 – R4 000", "R4 001 – R5 000", and "≥R5 001"), and number of people supported by this income.

Information recorded by the main PASS study fieldworkers was captured in the main study data base and then exported to an Excel spreadsheet. This spread sheet was subsequently merged with the dietary data (see section 3.3.3 *Data capturing* and *Cleaning of the 24-hour recall data*) for statistical analyses.

3.3.3 24-Hour recall

Administration of the 24-Hour recall

The 24-hour recall was administered by the primary researcher using the multiple-pass method. This method entails following three steps to attain as accurate a recall as possible (Wrieden et al., 2003). In the first step a quick list of foods consumed in the previous 24 hours was recorded. The second pass allowed for elicitation of more detailed information about each food item, drink, meal and snack that had been recalled, while the third pass prompted for foods/drinks that may have been forgotten. Foods that are known to be easily forgotten such as fruit, juices or other drinks, crisps, and sweets or other snacks were asked specifically. A final review was used to glean any additional information about the previous day's intake. The method allows for probing questions to assist the participant in recalling the full day's intake. Time of meals and snacks was asked and recorded to help the participant methodically remember the events of the day and foods consumed. Preparation methods and condiment use were included in the questioning specifically during the second pass.

Portion sizes were estimated during the second pass with the help of simplified line drawings of household measures (heaped and level spoons, glasses and cups), matchboxes and portion

sizes of amorphous foods as depicted in the Dietary Assessment and Education Kit (DAEK) (Steyn & Senekal, 2004).

Training of fieldworkers

The dietitian (primary researcher) received training in the administration of the multiple pass 24-hour recall method prior to commencement of the sub-study to ensure that correct and consistent procedures and prompting techniques were used. For these purposes, four test 24-hour recalls were conducted with women attending the clinic which were then checked by the trainer (an expert in dietary intake methodology). All 477 interviews were conducted by the candidate.

Data collection timeline and implementation

The interviews took place over a period of one year between August 2011 and August 2012. Table 3.1 shows the distribution between spring/ summer and autumn/ winter interviews, and between the data used for analysis ("Complete") and all the data collected, including incomplete data sets (where participants dropped out of the study). The current study was done on an urban population where seasonal variation in intake of fruits and vegetables is not expected to affect dietary intake results as these are available at supermarkets throughout the year. We have also found no significant difference (p=0.6852) between seasonal intake in fruits and vegetables (Table 3.2).

TABLE 3.1 *Distribution of interviews across the seasons*

	SPRING/ SUMMER	**AUTUMN/ WINTER**	**TOTAL**
Complete	244 53.5%	212 46.5%	456
Incomplete	10 47.6%	11 52.4%	21

TABLE 3.2 *Distribution of fruit and vegetable intake across the seasons*

	SPRING/ SUMMER	AUTUM/ WINTER
Fruits	67 44.1%	60 39.5%
Vegetables	50 54.6%	40 45.4%
Combined	117 77.0%	100 65.8%

Interviews were conducted in a private room at the Bishop Lavis MOU. The recruitment interview with the dietitian included the first 24-hour dietary recall that also lasted about 15 to 20 minutes. During the first follow-up appointment a second 24-hour recall was administered. An appointment for the third interview was arranged, usually within two weeks, and on a day of the week that had not yet been covered by the other two interviews. During the second follow-up appointment, the third 24-hour recall was collected. The second and third follow-up interviews lasted about 15 to 20 minutes. Each participant received a R100 Checkers Hypermarket voucher at the end of the second and third visits as appreciation for their attendance. The vouchers were valid for anything other than alcohol or cigarettes.

At each follow-up interview participants were asked if they had any questions about the study, and whether they still wanted to participate. Any queries were addressed before the interview started. The participants received a reminder card with the date and time of their next appointment, and with the interviewer's contact details. They were asked to contact the interviewer if they had queries or wanted to change or cancel the next appointment. Where correct contact details were available, a reminder SMS was sent to encourage participants to arrive for their follow-up appointments. When participants did not show up for their scheduled appointments, and where correct contact details were available either from the dietitian's records or those of the main PASS study, the dietitian phoned to reschedule appointments.

Representation of usual intake

Coverage of seasonal variation in food availability was ensured by recruiting participants over a one-year period with interviews evenly distributed across the seasons (Table 3.1).

Variability that may result from different eating habits on specific week days, specifically week versus weekend days, was addressed by conducting each 24-hour dietary recall of a particular participant on a different day of the week, including one weekend day.

Data capturing

Data from the 24-hour recalls were entered in FoodFinder ™ 3 by a registered dietitian experienced in capturing dietary data. Participant code, meal date, interview number, meal item code, total grams and energy and nutrient intake results were exported from FoodFinder ™ 3 to Excel spreadsheets where data was sorted and cleaned.

Cleaning of the 24-hour recall data

Cleaning of captured dietary data was done to eliminate methodological errors, and involved the following steps:

- The data was checked for any missing interview data by comparing a list of participant codes from the captured data file with that from the list compiled during the dietary data collection process. Three full 24-hour recalls had to have been captured for a participant's data to be included in the analyses.
- The resulting data set was checked for outliers in terms of quantities consumed. These values (either very low or very high) were compared to the original raw data from the recorded 24-hour recall and corrected if incorrectly captured.
- A list of each food item recorded across the 456 interviews was checked for consistency of food codes and portion sizes allocated for the same item.
- An independent dietitian rechecked all the raw data against the Excel spreadsheet and made suggestions for changes, including errors of omitted items and meals, added items, or where incorrect quantities were recorded in the spreadsheet. The primary researcher (interviewer) confirmed the final decisions in this regard with the senior nutrition researchers in the group.

The updated and corrected data sheet was once more run though data cleaning analyses. Any remaining outliers were inspected in terms of daily total energy and protein intakes and checked against the data captured in the 24-hour recalls. Based on these assessments all outliers were accepted as true values.

Estimation of the energy contribution of alcohol intake

Alcohol intake was one of the main outcome variables of the main PASS study. A detailed account of alcohol intake was therefore collected through a series of questions (Addendum A) by the fieldworkers of the main PASS study during the recruitment interview and at each follow-up interview of the main study. The consumption of alcohol was thus not prompted for during the interviews of the dietary sub-study and not recorded as part of the 24-hour recalls. Analysis of alcohol data was done separately to this sub-study. From this data, the average energy from alcohol intake was calculated in grams per day over a four-week period. For that time period, the average number of monthly standard drinks of alcohol was calculated by multiplying the frequency of alcohol consumption (days per month) with the average quantity of alcohol consumed (standard drinks per drinking day). This was divided by 28 days to give the average daily standard drinks of alcohol. The average daily alcohol energy was calculated by multiplying the average daily standard drinks by 13.6 (grams of alcohol per standard drink), and then multiplied by 29 (kilojoules per gram of alcohol). This was then added to the total energy intakes as calculated from the 24-hour recalls to calculate total energy intake including alcohol.

3.4 Interpretation of dietary intake data

3.4.1 Under- and over-reporters (validity measure)

Various formulas have been compiled to estimate REE and these can be used to check for under- or over-reporting with other measures (Ireton-Jones, 2012; Lee & Nieman, 2010). The Goldberg cut-off was created by calculating the minimum energy that a person would require for normal daily functioning using DLW and calorimetry. Although the Goldberg formula assumes a state of energy balance – energy intake equals energy expenditure, resulting in stable weight – the formula has been used in several studies with pregnant women (McGowan & McAuliffe, 2012; Winkvist, Persson & Hartini, 2001).

The average energy intake computed from the three 24-hour recalls was used to determine the levels of under-, accurate, and over-reporting by the study sample using the following steps:

1) The IOM equation for EER for pregnant women (shown below) was used to calculate the energy requirements of each participant as follows:

Adolescents: 135.3 – (30.8 x age) + PAL x (10 x weight + 934 x height) + 25 + 350
Adults: 354 – (6.91 x age) + PAL x (9.36 x weight + 726 x height) + 350
PAL= Physical activity level for a sedentary lifestyle was used (1.16)

2) The average energy intake (EI) of each participant derived from her three 24-hour recalls was divided by her calculated EER to derive an EI to EER ratio.
3) Goldberg et al. (1991) suggested a cut-off value below 0.8 for this ratio to identify under-reporting, and above 1.2 to identify over-reporting. However, the cut-off value of 0.9 that has been used in studies involving pregnant women was applied in this research. This cut-off value accommodates pregnancy-induced changes (Derbyshire et al., 2009; McGowan & McAuliffe, 2012). For the purposes of this study a value ≤0.9 indicates under-reporting, >0.9 but <1.2 indicates accurate reporting, and ≥1.2 indicates over-reporting.

3.4.2 Adequacy of dietary intake

To investigate the adequacy of dietary intake, the dietary intake data from the three 24-hour recalls were compared to the DRIs, SAFBDG and the NAR and MAR were calculated.

Interpretation of dietary adequacy using the dietary reference intakes (DRIs)

The dietary standards developed by the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes (DRI Committee) of the Food and Nutrition Board, Institute of Medicine of the National Academies in America have been adopted by South Africa to replace the RDAs as an improved indicator for good health, disease prevention, and the possible adverse effects of overconsumption (Trumbo et al., 2002). The DRIs (Table 3.3) incorporate a set of nutrient-based reference values that can be used to provide quantitative estimates of adequacy of nutrient intakes (Murphy et al., 2006).

TABLE 3.3 *The Dietary Reference Intakes* (Adapted from Murphy & Poos, 2002)

	ESTIMATED AVERAGE REQUIREMENTS	**RECOMMENDED DAILY ALLOWANCE**	**ADEQUATE INTAKE**	**TOLERABLE UPPER INTAKE LEVEL**
Definition	Intake estimated to meet the nutrient requirements of half of the healthy individuals in a specific age and gender group. Used as the basis in determining the RDA	Average intake level sufficient to meet the requirements of almost all (97-98%) healthy individuals in a specific age and gender group	An assumed adequate intake level based on observed or experimentally determined estimates on a group of healthy people. Used when insufficient data is available to estimate an EAR	The highest average intake level unlikely to pose risks of adverse health effects in almost all (97-98%) individuals in a specific age and gender group
Use in individuals	Determines possibility of inadequate intake in individuals	Low risk of inadequacy with intake at or above this level	Low risk of inadequacy with intake at or above this level	Risk of adverse effects if usual intake is above the UL
Use in groups	Estimates the prevalence of inadequate intake in groups	Not suitable for assessment of intake in groups	Low prevalence of inadequate intake at or above this level	Estimates the percentage of a population at risk of adverse effects

The EAR is the most appropriate reference value to use to assess the adequacy of dietary intake of a group (Murphy et al., 2006, Murphy & Poos, 2002). The distribution of intakes above or below the EAR are determined and interpreted for these purposes (Murphy et al., 2006). If an EAR has not been set for a specific nutrient, then the AI is used instead. With an intake above the AI, it can be assumed that the prevalence of inadequate intake will be low. However, an intake below the AI may not necessarily indicate inadequacy, but just that an increase in intake may be advantageous (Barr et al., 2002). In this study, where possible, the EARs were used for interpretation of results. Where available, reference values for the second trimester were used, as majority of the women were in the second trimester for at least two of the three days that dietary intake was assessed.

Assessment of the prevalence of adequate/inadequate intake of nutrients using the data derived from the three repeated 24-hour recalls per participant was done by applying the

EAR cut-point method (Murphy et al., 2006; IOM, 2000). This analysis requires the use of usual intake, rather than observed intake, to limit over- or underestimation of the prevalence of nutrient inadequacy (Gibson, 2005). While observed intake is the actual data collected using a dietary intake assessment method, usual intake can only be collected in this way if the collection period spans several days, weeks, months, or years, depending on what nutrient is being recorded (Gibson, 2005; Basiotis et al., 1987). However, it is possible to get a more accurate measure of usual intake based on three repeated 24-hour recalls by applying statistical modelling to observed intake data to correct for inter and intra-individual variability (Gibson, 2005; Murphy & Poos, 2002; IOM, 2003). The detail of the statistical modelling is explained as part of Section 3.5 (*Statistical procedures and analysis*)

Interpretation of dietary adequacy using the South African Food Based Dietary Guidelines

Analysis according to the FBDG was done in Excel spreadsheets using the following steps:

- All food items consumed were categorised into one of thirteen food groups (Milk and milk products; Nuts and seeds; Fats and oils; Fruit juice; Legumes and legume products; Meat and meat products; Fish and seafood; Eggs; Cereals and cereal products; Sugar, syrups and sweets; Fruit; Vegetables; Roots) (Table 3.4)
- The average serving sizes for each food item was entered based on serving sizes recommended by the FBDG.
- For each item, the actual intake was divided by the average serving size to calculate the number of portions of each food item. In this way, discrepancies between dry cereals and cooked cereals were compensated for (e.g. 100g of cornflakes equals just over three portions, while 100g of cooked oats equals one portion).
- Where complex food items were concerned (e.g. cottage pie or baked goods), the contribution to number of portions was split among two or three broad food categories in a ratio compatible with the macronutrient distribution of each food item. For example: Cottage pie has a suggested serving size of 120g. A 240g serving of cottage pie would equal 2 portions. Based on the macronutrient distribution of cottage pie, these portions contributed 3/5 to the "Roots" group, and 2/5 to the "Meat and meat products" group, equating to 1.2 portions of roots, and 0.8 portions of meat.

- The number of portions was summed for each of the 13 broad food groups. The food groups were then collated into six food groups that represent six of the FBDG, and the total number of portions for these groups was calculated by adding the number of portions for the broad food groups that were collated (Table 3.4).

TABLE 3.4 *Categories as per the South African food based dietary guidelines*

FOOD BASED DIETARY GUIDELINE	FBDG CATEGORY	TOTAL PORTIONS	TOTAL PORTIONS PER CAPITA
Make starchy foods the basis of most meals	Starch	1607.7	10.6
Eat plenty of vegetables and fruit every day	Vegetables and Fruit	453.6	3.0
Eat dry beans, split peas, lentils and soya regularly	Dry beans, split peas, lentils, soy	52.0	0.3
Chicken, fish, meat, milk or eggs can be eaten daily	Meat, poultry, fish, eggs	577.8	3.8
	Milk	182.7	1.2
Eat fats sparingly	Fats	1479.2	9.7
Use food and drinks containing sugar sparingly and not between meals	Sugar	1169.8	7.7
Use salt and foods high in salt sparingly	Salt	-	-

- The number of eaters was calculated by counting each individual that consumed a food item at least once within the three recorded days.
- The number of eaters was divided by the total sample size (n=152) and multiplied by 100 to give the percentage of eaters.
- The total quantity was calculated as the sum of every serving size (grams) for every time that that food item was consumed.
- The total mean portion size (grams) per eater per day was calculated by dividing the total quantity consumed by the number of eaters, divided by three days.
- The portion per capita was calculated by dividing total quantity consumed of a particular food item by the total sample size (n=152), divided by three days.
- The % of daily TE was calculated by dividing the energy provided by each food item with the total energy provided by all foods consumed combined.

- The total portions per capita were calculated by dividing the total number of portions consumed by the total number of participants in the study sample (n=152) (Table 3.5).

TABLE 3.5 *Broad food groups and per capita portions*

	Average serving sizes	**Total grams (3 days)**	**Total portions/ day**	**No. of eaters**	**% of eaters**	**No. Portion/ eater/ day**	**Portion size/ eater/ day**	**Portion size/ capita**	**No. Portions/ capita**
Cereals and cereal products	30g dry, 100g cooked 35g bread	181 625.5	1605.5	152.0	100.0	10.6	398.3	398.3	10.6
Roots	90g	66 904.7	247.8	147.0	96.7	1.7	151.7	146.7	1.6
Fruit	80g	77 192.5	287.8	127.0	83.6	2.3	202.6	169.3	1.9
Vegetables	80g	39 337.0	165.8	144.0	94.7	1.2	91.1	86.3	1.1
Meat and meat products	40g	64 055.5	480.3	152.0	100.0	3.2	140.5	140.5	3.2
Fish and seafood	60g	6 302.5	32.3	58.0	38.2	0.6	36.2	13.8	0.2
Eggs	50g	9 770.5	65.1	75.0	49.3	0.9	43.4	21.4	0.4
Legumes and legume products	100g	15 566.0	52.0	70.0	46.1	0.7	74.1	34.1	0.3
Milk and milk products	250mL milk 125mL yoghurt	94 361.5	182.7	147.0	96.7	1.2	214.0	206.9	1.2
Fats and oils	5g	8 971.0	1475.8	146.0	96.1	10.1	20.5	19.7	9.7
Nuts and seeds	15g	154.0	3.4	7.0	4.6	0.5	7.3	0.3	0.0
Sugar, syrups and sweets	10g	134 581.0	1115.1	152.0	100.0	7.3	295.1	295.1	7.3
Fruit juice	125mL	9 932.5	54.7	34.0	22.4	1.6	97.4	21.8	0.4

Per capita calculation: Portion per capita was calculated by dividing the total quantity of the food item consumed by the total study sample size respectively.

Interpretation of dietary adequacy using the nutrient adequacy ratio (NAR) and mean adequacy ratio (MAR)

The NAR and MAR are two ratios that can also be used to gauge the adequacy of dietary intake. The NAR was calculated for each participant by dividing observed intake of a nutrient by the recommended intake according to the EAR for the same nutrient (Steyn et al., 2005).

This was done for all nutrients assessed in the study. A value of 100% indicates that intake is the same as recommendations (Steyn et al., 2005).

The MAR was calculated for each participant by adding each NAR (to a maximum of 100%), and dividing it by the number of nutrients analysed. The MAR gives a measure of the overall adequacy of the diet. As with the NAR, a value of 100% indicates that intake is the same as recommendations, and thus adequate (Steyn et al., 2005).

3.4.3 Dietary diversity and variety

Dietary Diversity Score

The dietary diversity score (DDS) gives an indication of the number of food groups consumed per day by a participant (Hatloy et al., 1998; Steyn et al., 2005), regardless of the amount consumed from any specific food group. It is evident that DDSs calculated in previous studies differed in terms of the number of food groups as well as type of food groups included in these scores. We have calculated two different DDSs to assess both household and individual DDSs (Kennedy et al., 2012). Firstly, the number of different food groups consumed in each of the 24-hour recall periods was calculated for each participant by assigning each food item consumed to one of the following food groups to assess household DDS: 1) milk and milk products 2) nuts, seeds, legumes and legume products 3) fats and oils 4) meat, meat products, fish, and seafood 5) eggs 6) cereals and cereal products 7) vegetables and 8) fruit. Sugar, syrups, sweets, fruit juices as well as tea and coffee were not included in the DDS calculation because these items do not add diversity to dietary intake in pregnant women, and the majority of other studies also do not include this (De Cock et al., 2013). Secondly, the same procedure was followed to calculate individual DDS using different food group categories, namely: 1) milk and milk products 2) nuts, seeds, legumes and legume products 3) fats and oils 4) meat, meat products, fish, and seafood 5) eggs 6) cereals and cereal products 7) vitamin A-rich fruit and vegetables and 8) other fruit (excluding fruit juice) and 9) other vegetables. This was done to enable comparison with a variety of other studies done using the DDS where vitamin A-rich fruits and vegetables were calculated separately to other fruits and vegetables. The number of different food group categories included in each participant's daily intake was then calculated for each 24-hour recall period, and an average over the three days was then calculated. No established cut-off points are available (Kennedy et al., 2012), but the majority of studies assessed used a score below four as being indicative

of poor dietary diversity, and poor food security (Drimie et al., 2013; Labadarios et al., 2011; Oldewage-Theron & Kruger, 2011; Steyn et al., 2006; Hatloy et al., 2000).

Variety of intake

Variety of dietary intake was assessed by identifying the most commonly consumed foods, and ranking of foods based on contribution to total energy and macronutrient intakes. The top 20 most commonly consumed foods were based on foods consumed by the highest percentage of the study sample (not considering the quantity consumed). This was done for all food items combined, as well as for fruits and vegetables specifically. Similarly, the top ten foods contributing to energy, total protein, carbohydrates and fat, as well as fibre and added sugar intake was based on which foods contributed the highest total kilojoules in the case of energy and grams in the case of the nutrients. For these purposes, all data was sorted by food type, summing the specific nutrient value (e.g. protein) of each food, and then ranking the food items by this total nutrient value. For each of these nutrients the number and percentage of eaters was calculated for each food item. From this, the average portion per eater was calculated by dividing the total quantity of the food item consumed, by the number of people that consumed that specific food item. This was divided by three to give the average portion per eater per day. Portion per capita was calculated by dividing the total quantity of the food item consumed by the total study sample size respectively.

The same process was followed for adults and adolescents separately, and differences were displayed between the two groups by means of a rank column.

3.5 Statistical procedures and analysis

The data on socio-demographic, anthropometric and nutrient intake variables were entered in excel spreadsheets and cleaned prior to analyses. Statistica Version 12 (StatSoft Inc. 2014) and Microsoft Office Excel 2007 was used to analyse the data. Kolmogorov-Smirnoff and Shapiro-Wilk tests were used to check for normality of the data. Where necessary, natural log and square root transformations were done to improve normality of the macro- and micronutrient content derived from the 24-hour recalls. Categorical variables were described using frequencies, while means, standard deviations, medians and inter-quartile ranges were used for numerical variables. Comparisons between adolescent and adult participants were done using Chi-square and Fisher's exact tests.

A process developed by the National Research Council (NRC) and IOM to decrease the prevalence of inadequate and excessive intakes, namely data shrinking, was applied as follows to all nutrient variables (IOM, 2003; Naude, 2012):

1. A one-way ANOVA test was applied to the normalised data to generate the observed variance and within-person variance.
2. The observed and within-person variance were divided by the number of days of intake data (three), giving the observed variance and within-person variance for each of the distributions.
3. Within-person variance was subtracted from observed variance, and divided by three (number of days of intake), to give an estimated between-person variance. Between-person variance represents the 'true' variance of the distributions of usual intakes.
4. Standard deviations were calculated for between-person variance and observed variance by using their square roots.
5. To calculate each participant's adjusted intake from mean intake:

   Adjusted intake = [(participant's mean – group mean) x (SD between/ SD observed)] + group mean

Macro- and micronutrient intakes were compared with recommendations by using the EAR cut-point method, and was carried out by a statistitian at the MRC. Frequencies of intakes below the EAR reflect the risk of inadequate intake (IOM, 2000). The frequencies of usual intakes below the EAR were compared between adults and adolescents using Chi-square and Fisher's exact tests to determine any significant differences.

Correlation matrices (Pearson's for normally distributed and Spearman's for non-normally distributed data) were constructed for macro- and micronutrients and socio-demographic variables, nutrients and anthropometric variables, as well as for socio-demographic and anthropometric variables.

Comparisons between adolescent and adult DDS, NAR and MAR were performed using Chi-square and Fisher's exact tests.

For all statistical analyses, a value of $p < 0.05$ will be considered statistically significant.

3.6 Ethical considerations

The study was approved by the Human Research Ethics Committee of the University of Cape Town, and the Stellenbosch University Research Ethics Committee (HREC REF: 401/2012). The consent form reflects providing consent for the dietary validation study and the collection of three 24-hour recalls.

Informed consent was obtained from each participant after a full and adequate oral and written explanation of the study was provided by the investigator (Addendum B). Participation was voluntary, and subjects were able to withdraw from the study at any stage without stating a reason. Each participant was assigned a code by the main PASS study fieldworkers on recruitment and this code was used to ensure anonymity when entering data for analysis. This study provided no risks or direct benefits to the subjects.

CHAPTER FOUR

SOCIO-DEMOGRAPHIC AND ANTHROPOMETRIC PROFILE

4.1 Introduction

A healthy pregnancy outcome encompasses any situation in which a healthy child is delivered safely, and neither mother nor child suffers any life-threatening complications (Kaiser & Campbell, 2014; Gravett & Rubens, 2012). Socio-demographic factors and the anthropometric status of the mother before and during pregnancy have been shown to influence pregnancy outcomes (Kaiser & Campbell, 2014). Pre-pregnancy under- or over-weight has been shown to result in various adverse pregnancy outcomes, including but not limited to preterm birth (Hoellen et al., 2014), low birth-weight (LBW) (Jeric et al., 2012; Calvo & Lopez, 2012), small-for-gestational age (SGA) (Han et al., 2010), autism (Moss & Chugani, 2014) and cardiac defects (Calvo & Lopez, 2012). Some studies have shown pregnancy outcome to be adversely affected by poorer socio-economic status, lower educational levels (Luo et al., 2006; Savitz et al., 2004; Kramer et al., 2000; Meis et al, 1995), and lower income (Luo et al., 2006; Tuntiseranee et al., 1999). Both adolescents and older women (≥35 years of age) may be at increased risk for poorer pregnancy outcomes (Laopaiboon et al., 2014; Calvo & Lopez, 2012; Mills & Lavendar, 2010; Baker et al., 2009).

Insights in the anthropometric status and socio-demographic characteristics of pregnant women can advise the development and implementation of intervention strategies to address risks for poor pregnancy outcomes (Northstone et al., 2008; De Irala-Estevez et al., 2000; Barker et al., 1990). No studies on the profile of and associations between anthropometric status and socio-demographic factors of pregnant females of different age groups in South Africa have been published, despite the fact that poor socio-economic factors affect the majority of the population (Shisana et al., 2013). Because communities and their circumstances vary, it is ideal to assess such associations in target communities to ensure relevance and appropriateness of interventions (Gibson, 2005).

The aim of this study was 1) to assess, describe and compare the socio-demographic profile and anthropometric status of adolescent and adult pregnant females attending the Bishop Lavis MOU, and 2) to investigate associations between these variables in the total group and in each of the two age groups.

4.2 Results

Socio-demographic characteristics

The mean age of the participants in this study was 25.4 years (Table 4.1), ranging from 16.1 to 41.6 years. One in five women were classified as having a teen pregnancy (<19 years of age), while almost 10% were of advanced maternal age (≥35 years of age). All participants were of mixed ancestry. The majority of the sample was either married or in a relationship, and had fewer than 10 years of formal education. A total of only 19% of the study sample completed secondary education or had a tertiary qualification. The lowest level of schooling was grade 6. The majority of participants had an average monthly household income less than R5 000 which was used to support four or more individuals in the household. No significant differences were found between adolescent and adult participants for any of the socio-demographic variables (not included in table).

TABLE 4.1 *Socio-demographic profile of the total study sample (n = 152)*

	n	Mean (SD)	%
Maternal age, years	152	25.4 (6.2)	
Maternal age categories			
< 19 years	30		19.7
19 – 25 years	47		30.9
26 – 35 years	63		41.4
35 years and older	12		7.9
Married/ partnered (% yes) *	142		93.4
Years of formal education categories	152	9.8 (1.59)	
Formal education categories			
Grade 7 or lower	14		9.2
Grade 8 – 10	89		58.6
Grade 11	20		13.2
Grade 12	25		16.4
Tertiary education	4		2.6
Monthly household income categories	151		
Don't know	32		21.2
0 – 500	7		4.6
501 – 2 000	32		21.2
2 001 – 5 000	48		31.8
≥ 5 001	32		21.2
No. of individuals supported	150	5.03 (2.56)	
No. of individuals supported categories			
≤ 3	72		48.0
4 -5	27		18.0
≥ 6	51		34.0

Abbreviations: No. = number SD = standard deviation

* Remainder unmarried/ not partnered

Gestational age

Twenty-six (17.1%) of the participants were below 93 days gestational age, and were thus classified as being in the first trimester of pregnancy, while the majority of participants (82.9%) were classified as being in the second trimester of pregnancy. The mean (SD) gestational age of the study sample was 114.6 (18.2) days. Gestational age was the same between the two age groups.

Weight status

Only 53.3% of participants knew their pre-pregnancy weight. These participants' mean pre-pregnancy BMI was classified in the normal range (Table 4.2). The pre-pregnancy BMI of one in three participants was classified as overweight or obese. The pre-pregnancy BMI of the majority of the adolescents and half of the adults was classified as normal. The adults had a significantly higher mean pre-pregnancy weight and BMI, as well as recruitment weight and BMI compared to the adolescents (Table 4.2).

Weight change from pre-pregnancy weight to weight at recruitment ranged from -5.7 kg to 26.5 kg in the adolescents, and -14.8 kg to 24.2 kg in the adults. In the total group, underweight women gained the most weight followed by normal weight women. There were no significant differences between adolescent and adult participants for mean weight change per BMI category. Only 15.2% of the participants gained weight within the recommended guidelines for their gestational age at recruitment and pre-pregnancy weight status category, while the majority of women gained too much weight (Table 4.2).

TABLE 4.2 *Anthropometric profile of the total study sample (n = 152), and comparisons between adolescent and adult participants*

	TOTAL GROUP		**ADOLESCENTS**		**ADULTS**		
	n	**Value**	**n**	**Value**	**n**	**Value**	**p**
Height (mm), Mean (SD)	149 †	1585.0 (58.8)	30	1580.9 (73.4)	119	1586.1 (54.8)	0.6684 ɤ
Pre-pregnancy weight (kg)*, Mean (SD)	81	59.5 (15.0)	14	51.7 (7.6)	67	61.1 (15.6)	0.0311 ɤ
Pre-pregnancy BMI (kg/m^2)*, Mean (SD)	79	23.7 (5.8)	14	20.8 (3.2)	65	24.3 (6.1)	0.0372 ɤ
Pre-pregnancy BMI categories ǂ, column %							
Thinness / Underweight	10	12.7	1	7.1	9	13.8	0.1700 ɕ
Normal weight	45	57.0	12	85.7	33	50.8	
Overweight	10	12.7	1	7.1	9	13.8	
Obese (class I)	10	12.7	0	0.0	10	15.4	
Obese (class II+)	4	5.1	-	-	4	6.2	
Weight at recruitment (kg), Mean (SD)	149	61.8 (15.1)	30	55.9 (12.0)	119	63.3 (15.5)	0.0161 ɤ
BMI at recruitment (kg/m^2), Mean (SD) ¶	149	24.8 (5.9)	30	22.4 (4.6)	119	25.2 (6.0)	0.0184 ɕ
Weight gain (kg), Mean (SD) §	79	3.8 (7.3)	14	2.8 (8.4)	65	4.0 (7.1)	0.5766 ɤ
Weight gain (kg) Mean (SD) **by pre-pregnancy BMI category**							
Underweight	10	8.4 (4.9)	1	26.5	9	6.4 (4.9)	0.0069 ɤ
Normal weight	45	3.8 (6.3)	12	1.1 (3.7)	33	4.8 (6.4)	0.1651 ɤ
Overweight	10	1.9 (4.2)	1	0.2	9	2.1 (4.4)	0.0802 ɤ
Obese	14	2.0 (10.5)	0	0.0	14	2.0 (10.5)	-
Adequacy of weight gain (kg) by recommendations ††, n (%)							
Insufficient gain	31	39.2%	8	57.1%	23	35.4%	0.2897 ɕ
Adequate gain	12	15.2%	1	7.1%	11	16.9%	
Excessive gain	36	45.6%	5	35.7%	31	47.7%	

Abbreviations: BMI = Body Mass Index SD = Standard deviation

† Height was not taken for three participants

¶ Allocation to WHO BMI categories not appropriate during pregnancy

*Based on self-reported pre-pregnancy weight

ǂ BMI classifications: Adolescent: Severe thinness (below -3SD) (combined with thinness) Thinness (below -2SD), Normal weight, Overweight (above +1SD), Obese (above +2SD) (WHO 2006b; Cole et al., 2000 – classification used by Cole et al. (2000) is the same as that set out by the WHO, except "severe thinness" and "thinness" will be classified as "underweight"). Adult BMI classifications: Underweight (<18.5kg/m^2), Normal weight (18.5 – 24.9 kg/m^2), Overweight (25 – 29.9 kg/m^2), Obese class I (30 – 34.9 kg/m^2), Obese class II (≥35 kg/m^2) (WHO, 2006b)

§ Weight gain = weight at recruitment – self-reported pre-pregnancy weight

†† Interpreted using recommendations for adequate weight gain in pregnant women using gestational age at recruitment (IOM, 2009)

ɤ Independent samples T-test ɕ Pearson's Chi-square test

The mean MUAC of the total group was classified in the "no risk for malnutrition" category, while almost one in six participants were classified as having either a strong indication or high risk for malnutrition. Adolescents had a significantly lower mean MUAC, and were significantly more likely to be classified in the "risk for malnutrition" category compared to adults (Table 4.3).

The means of the TSF, AMA and AFA are presented in Table 4.3. Almost 30% of participants were classified as "risk of poor pregnancy outcomes" according to AFA categories. Adolescents had a significantly lower cAMA and AFA than adults (Table 4.3).

TABLE 4.3 *Arm anthropometry of total study sample (n=152) and comparisons between adolescent and adult participants*

	TOTAL GROUP		**ADOLESCENTS**		**ADULTS**		
	n	Value	n	Value	n	Value	P
TSF (mm), Mean (SD)	146	24.9 (9.5)	29	22.7 (6.9)	117	25.4 (9.9)	0.1574 ɤ
MUAC (cm), Mean (SD)	152	26.8 (6.5)	30	25.2 (3.1)	118	28.1 (5.0)	0.0036 ɤ
MUAC (cm) categories							
<22 High risk of malnutrition, LBW, adverse outcomes	8	5.4%	2	6.7%	6	5.1%	0.0120 ☪
<23 Strong indication of malnutrition	13	9.5%	3	10.0%	11	9.3%	
<25 Warning indicator for malnutrition	29	19.6%	12	40.0%	17	14.4%	
≥25 No risk	97	65.5%	13	43.3%	84	71.2%	
cAMA (cm^2), Mean (SD)	146	24.8 (9.1)	29	19.8 (4.4)	117	26.0 (9.5)	0.0009 ɤ
AFA (cm^2), Mean (SD)	146	30.8 (16.3)	29	25.7 (11.0)	117	32.2 (17.1)	0.0342 ɤ
AFA (cm^2) categories*							
<20cm^2 – Risk of poor pregnancy outcome	42	28.8%	10	34.5%	32	16.2%	0.4475 ☪
≥20cm^2	104	71.2%	19	65.5%	85	43.2%	

Abbreviations: AFA = Arm Fat Area cAMA = Corrected Arm Muscle Area MUAC = Mid-Upper Arm Circumference TSF = Triceps Skinfold

* AFA categories as suggested by Friis et al., 2004

ɤ Independent samples T-test ☪ Pearson's Chi-squared test

Correlations

A correlation matrix between select socio-demographic and anthropometric variables is presented in Table 4.4. Variables for which no significant correlations were found for the total group and within the adolescent and adult groups were not included in the tables below.

A significant negative correlation was found between number of individuals supported by the household income and number of years of formal education. An older maternal age was significantly correlated with higher pre-pregnancy weight, recruitment weight, BMI, and MUAC. There were no other significant correlations between other socio-demographic variables and anthropometry.

Significant positive correlations were also evident between pre-pregnancy weight and pre-pregnancy BMI, recruitment weight and BMI, TSF, MUAC, cAMA, and AFA; and also between pre-pregnancy BMI and recruitment weight and BMI, TSF, MUAC, cAMA and AFA. A higher pre-pregnancy BMI was correlated with a greater AFA. A greater height at recruitment was significantly correlated with higher pre-pregnancy and recruitment weights. Gaining more weight from pre-pregnancy to recruitment was significantly associated with a lower pre-pregnancy weight and BMI and a higher recruitment weight, BMI, MUAC and cAMA.

TABLE 4.4 *Correlation between socio-demographic and anthropometric data*

	Number supported by income	Pre-pregnancy weight (kg)	Pre-Pregnancy BMI (kg/m^2)	Weight at Recruitment (kg)	Weight change (kg)	BMI at recruitment (kg/m^2)	TSF (mm)	MUAC (mm)	cAMA (cm^2)	AFA (cm^2)
Years of formal education	**-0.1777**	0.0337	-0.0240	0.0632	0.1184	0.0417	0.1104	0.0349	-0.0696	0.0621
	p=0.0296	p=0.7651	P=0.8337	p=0.4436	p=0.302	p=0.6135	p=0.1831	p=0.6738	p=0.4036	p=0.594
Maternal Age (years)		**0.2633**	0.2208	**0.3549**	0.1229	**0.3181**	0.2149	**0.3727**	**0.4100**	**0.2900**
		p=0.0176	P=0.505	**p=0.0001**	p=0.284	**p=0.0001**	p=0.0890	**p=0.0001**	**p=0.0000**	**p=0.011**
Height at Recruitment (mm)		**0.3321**	0.0099	**0.2095**	0.1363	-0.1152	0.1184	-0.0493	-0.0292	0.0890
		p=0.0028	p=0.9309	**p=0.010**	p=0.234*	p=0.1620	p=0.302*	p=0.5517	p=0.7265	p=0.444
Pre-pregnancy weight (kg)		0	**0.9284**	**0.8599**	-0.2008	**0.7912**	**0.7350**	**0.7672**	**0.5686**	**0.8117**
			p=0.0000	**p=0.000**	p=0.078	**p=0.0000**	**p=0.0000**	**p=0.0000**	**p=0.000**	**p=0.000**
Pre-Pregnancy BMI			0	**0.7725**	**-0.2620**	**0.8356**	**0.7246**	**0.7783**	**0.5896**	**0.7979**
				p=0.0000	**p=0.021**	**p=0.0000**	**p=0.0000**	**p=0.0000**	**p=0.000**	**p=0.000**
Weight at Recruitment (kg)				0	**0.2856**	**0.9342**	**0.8215**	**0.9131**	**0.7462**	**0.8922**
					p=0.011	**p=0.0000**	**p=0.0000**	**p=0.0000**	**p=0.000**	**p=0.000**
Weight change					0	**0.3035**	0.2021	**0.2843**	**0.2515**	0.1658
						p=0.0065	p=0.076*	**p=0.0117**	**p=0.0263**	p=0.152
BMI at recruitment						0	**0.8445**	**0.9486**	**0.7741**	**0.8966**
							p=0.0000	**p=0.0000**	**p=0.000**	**p=0.000**
TSF (mm)							0	**0.8822**	**0.4740**	**0.9718**
								p=0.0000	**p=0.000**	**p=0.000**
MUAC (mm)								0	**0.8143**	**0.9612**
									p=0.0000	**p=0.000**
cAMA (cm^2)									0	**0.6708**
										p=0.000

Abbreviations: cAMA = Corrected Arm Muscle Area MUAC = Mid-Upper Arm Circumference TSF = Triceps Skinfold

Spearman's test used for all correlation analyses except where marked with * where Pearson's correlation tests were used

Variables for which no significant correlations were found for the total group and within the adolescent and adult groups were not included in the table

4.3 Discussion

The study sample was drawn from a population of pregnant adolescent and adult participants attending a community health centre in a low socio-economic area where income and education levels can be expected to be low. Based on the 2011 South African Census report, the household income of the majority of women in our sample (78.8% had income <R5 000) can be classified below the average monthly household incomes of South Africa as a whole (R8 600) or those living in the Western Cape (R11 955). Furthermore, 5.8% of the women

had very low household incomes of less than R500 per month. These results point to the fact that the majority of participants have a low socio-economic status, which may impact negatively on food security and access to a variety of food choices (Olsen & Strawderman, 2008; Sobal & Stunkard, 1989). The possible lack of household food security is supported by the fact that the already limited incomes were used to support an average of three to four people in the majority of cases. This is in line with South African and Western Cape statistics of 3.6 people per household (Census 2011). However, no significant associations were found between income, the number of people supported by income, and the anthropometric variables of study participants indicating that household income may not be impacting on the weight status of pregnant adolescent and adult participants in the area as could have been expected (Duflo, 2003). It needs to be noted that the more individuals who were supported by the household income, the lower the level of formal education of study participants was. The average number of years of formal education of 9.8 in our sample is in line with the 9.75 reported for the same age group of adolescent and adult participants in the 2003 SADHS (Department of Health, 2007) with none of our participants reporting no education. The lowest level of education reported in our sample was six years of schooling, whereas the SADHS and SANHANES reported that 12.2% and 7.1% of women respectively had none. The general level of formal education is relatively low, with only 19% of the study sample having completed secondary education, compared to the 32.3% reported by SANHANES (Shisana et al., 2013).

Pre-pregnancy weight and BMI are determinants of maternal health and nutritional status during pregnancy with a healthier pre-pregnancy weight status being associated with a greater chance of a healthy pregnancy and pregnancy outcome (Moss & Chugani, 2014; Tanaka et al., 2014; Calvo & Lopez, 2012; IOM, 2009). Obtaining reasonable estimates of pre-pregnancy weight in our sample proved to be challenging. Only 52% of the participants were able to provide information on pre-pregnancy weight (14 adolescents and 65 adults). Evidence shows that self-reported weight, height and BMI are generally strongly correlated with actual weight, height and BMI ($r>0.9$). However, subjects tend to underestimate their weight, resulting in underestimation of BMI and subsequent misclassification according to BMI categories (Spencer et al., 2002; Rossouw et al., 2000; Yoong et al., 2013; Gunnare et al., 2013; Niedhammer et al., 2002). Factors that have been found to influence accuracy of reporting (increased likelihood of under-reporting) include being female and heavier (Spencer

et al., 2002; Kolvalchik, 2008; Gunnare et al., 2013; Rossouw et al., 2000), time elapsed (Kolvalchik, 2008), age, end-digit preference, being an adolescent, lower level of education and lower grades of occupation (Spencer et al., 2002; Niedhammer et al., 2000). Yoong et al. (2013), Rossouw et al. (2001) and Niedhammer et al. (2000) agree that as a result of potential under-reporting of weight at an individual level, results of the classification of individuals according to BMI should be interpreted with caution. In our study we used self-reported weight to classify participants according to BMI categories and to calculate weight gain from conception to recruitment on an individual basis. All the results involving this variable should thus be interpreted with caution, especially also because the sample included females of lower levels of education and thus possibly lower levels of occupation.

The pre-pregnancy weight of almost half (43%) of the participants who gave a pre-pregnancy weight did not fall within the ideal weight range. One in eight (12.7%) participants were classified as underweight while 30.4% were classified as overweight. Although no South African data regarding pre-pregnancy weight status is available, large cohort studies done in Europe and the United States reported a lower prevalence of underweight of 2.9% in the National Norwegian Mother and Child Cohort Study (Haugen et al., 2014) and 5.5% in the Florida Birth Defects Register and the Florida singleton birth cohort (Block et al., 2013; Thompson et al., 2013). Data from developing countries is scarce, but some analyses have shown that women in developing countries with low pre-pregnancy weight have an increased risk of preterm labour (Han et al., 2011; Neggers, 2015) and IUGR (Neggers, 2015), but not of delivering LBW infants (Neggers, 2015). Our study's overweight prevalence was higher than the 17.8% reported for the Norwegian sample (Haugen et al., 2014) but lower than the 42.5% reported for the USA sample (Block et al., 2013; Thompson et al., 2013). These results may reflect on the double burden of overweight and underweight in developing countries such as South Africa (Toriola et al., 2012)

Adolescent pregnancy (younger than 19) or pregnancy at advanced maternal age (older than 35 years) may significantly influence health outcomes of the mother and foetus and/or infant, both during and after pregnancy. It is thus concerning that 27.6% of participants in our sample were in these risk age groups, although the proportion is slightly lower than the approximate 42% reported in the 2003 SADHS (SADHS, 2007). This proportion is much

lower in the study by Jaffer et al. (2008) who had only 13.8% of their sample falling in these at-rsik categories. Women older than 35 years have an increased risk for hypertension, miscarriage, ectopic pregnancy (Mills & Lavendar, 2010; Yogev et al., 2010; Huang et al., 2008), stillbirth, pre-term delivery, caesarean delivery (Laopaiboon et al., 2014; Flenady et al., 2011; Aliyu et al., 2008), macrosomia (Kenny et al., 2013), and LBW (Aliyu et al., 2008; Joseph et al., 2005). In our study older maternal age was significantly associated with higher pre-pregnancy weight, recruitment weight, BMI, MUAC, cAMA and AFA all of which reflect increased risk for obesity (except cAMA), which may result in further increased risks of the above mentioned adverse pregnancy outcomes.

It is well known that adolescence is a period of rapid development and tissue and bone growth, which increases nutritional requirements (Williamson, 2006). Addition of the increased requirements of pregnancy places pregnant adolescents and their foetuses, at high risk of malnutrition and adverse pregnancy outcomes. Our results show that adolescent participants had a poorer nutritional status than the adult participants as their mean pre-pregnancy weight and BMI, as well as recruitment weight, BMI, MUAC, cAMA and AFA were significantly lower than that of adults.

Interpretation of BMI in pregnant women in terms of weight status categories is inappropriate as classification cut-offs are not available for pregnancy. Mid upper arm circumference has been shown to be strongly correlated with body weight which makes it a suitable replacement or proxy for BMI in the assessment of weight status in individuals (Ogbonna et al., 2007; Thame et al., 2007; Kelly et al., 1996; Pelletier et al., 1995; James et al., 1994). This relationship remains evident during pregnancy (Calvo & Lopez, 2012; Lopez et al., 2011), as was found in our study, namely that increasing BMI was associated with increasing MUAC. Interpretation of nutritional status based on MUAC in pregnant females is possible as criteria are available to classify these individuals as "high risk", "strong indicator" or "warning" of malnutrition, LBW and adverse outcomes (Kruger, 2005). MUAC during early pregnancy has been reported to be a better predictor of prematurity (Liljestrand & Bergström, 1991) and LBW (Lopez et al., 2011; Ojha & Malla, 2007; Kruger, 2005) than maternal weight gain or BMI. It is thus concerning that adolescents in our study were significantly more likely to have a MUAC classified in the "warning" category for malnutrition. The pregnant adolescents may

thus be more at risk of having a preterm delivery, or giving birth to LBW and SGA babies (Calvo & Lopez, 2012; Baker et al., 2009; Chen et al., 2007).

Arm and skin-fold measurements other than MUAC can also be analysed in combination with weight to give an indication of body composition (subcutaneous fat stores and muscle mass) (Widen & Gallagher, 2014; Lee & Nieman, 2010) and maternal nutritional status (Widen & Gallagher, 2014; Lopez et al., 2011). The higher TSF, cAMA and AFA were significantly associated with higher pre-pregnancy and recruitment weight, MUAC, BMI and with weight change (except TSF and AFA). Although no specific interpretation criteria for TSF and cAMA are available for pregnant women, the mentioned associations generally support the insights gained in the nutritional status of participants based on MUAC. According to the guidelines by Friis et al. (2004), 30% of the study participants are at greater risk for poor pregnancy outcomes. Widen and Gallagher (2014) and Calvo and Lopez (2012) agree that more research is needed to identify meaningful changes in these measurements throughout pregnancy.

Adequate weight gain during pregnancy is an important determinant of pregnancy outcome infant health and health status later in life (Ehrenthal et al., 2014). Pre-pregnancy weight status and number of foetuses being carried determines how much weight a woman should gain during pregnancy to decrease the risk of adverse outcomes (Ehrenthal et al., 2014; IOM, 2009). Monitoring BMI and weight during pregnancy can give an indication of foetal growth and health (Calvo & Lopez, 2012; Lopez et al., 2011). Only 15.2% of the study participants who provided pre-pregnancy weight estimates (11 adults and one adolescent) gained weight within their specific recommended guidelines based on their pre-pregnancy BMI status, while 45.6% gained too much weight. Participants that were classified as underweight before conception gained almost twice as much weight than those who were normal weight at that point. Participants that were overweight or obese before conception gained approximately 50% less weight than those who were normal weight. These findings are reflected in the negative association found between weight gain and pre-pregnancy BMI which has been shown in several other studies (Heude et al., 2012; Nohr et al., 2009; Dietz et al., 2006; Edwards et al., 1996). While this trend is in line with recommendations for weight gain, it is

concerning that more than half of the adolescents gained insufficient weight during the first trimester of their pregnancy. It should also be noted that in contrast to these findings positive associations between weight gain and recruitment weight, BMI, MUAC and cAMA were found. Furthermore, the large standard deviations in weight gain – most probably as a result of weight losses of up to 14.7kg and gains of up to 26.5kg, which were calculated based on self-reported pre-pregnancy weight – speak to the possibility that the self-reported pre-pregnancy weights may not be accurate and that the results concerning weight gain need to be interpreted with caution.

Bearing in mind the limitation of self-reporting in general and those relating to self-reported weight specifically, it can be concluded that the study sample was characterized by a number of indicators that reflects lower socio-economic status, but that these indicators do not seem to be associated with the weight status of the adolescent and adult participants. Furthermore, adolescents seemed to be less well-nourished than adults, who may, in turn, be more likely to be overweight or obese. Nutrition interventions for pregnant adolescents should thus focus on ensuring sufficient nourishment for optimal growth and weight gain, while prevention of excessive weight gain during pregnancy and post-partum weight retention should be the focus for pregnant adults.

CHAPTER FIVE

DIETARY ADEQUACY

5.1 Introduction

Adequate dietary intake during pregnancy plays an important role in healthy pregnancy outcomes for both mother and foetus (NHMRC, 2013). Energy requirements increase from the first to the second and the second to the third trimesters, and protein and carbohydrate requirements increase from the first trimester of pregnancy. However, micronutrient requirements increase to a greater extent, necessitating a high nutrient-dense diet (IOM, 2002; 2005).

Adequacy of dietary intake of individuals, groups and populations can be assessed and expressed in terms of energy and nutrient recommendations as prescribed in the Dietary Reference Intakes (DRIs), quality of food choices and compliance with recommended number of servings consumed per food category when compared to food based dietary guidelines (FBDG) and nutrient adequacy ratios (NARs). Assessment of the adequacy of dietary intake determines the risk of deficiency of macro- and micronutrients under investigation, as well as the risk for development of non-communicable diseases.

Only seven studies could be traced that investigated the dietary intake of pregnant women in various regions in South Africa (Bopape et al., 2008; Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Tshitaudzi, 2003; Klinger, 2004; Mamabolo et al., 2004). Of these, only four analysed the diet for energy and macro- and micro-nutrients (Kesa & Oldewage-Theron, 2005; Mosteret et al., 2005; Tshitaudzi, 2004; Klinger, 2004), while other studies focused on a few specific nutrients only (Bopape et al., 2008; Mamabolo et al., 2004) or only assessed frequency of intake (Jaffer et al., 2008). When assessing the adequacy of dietary intake of a group, the EAR is the most appropriate DRI value to use (Murphy et al., 2006; Murphy & Poos, 2002). Only one study (Bopape et al., 2008) used the EARs as the reference point, while some used % RDA to reflect adequacy (Kesa & Oldewage-Theron, 2005; Klinger, 2004) and others used <67% RDA as an indicator of inadequacy (Mostert et al., 2005; Tshitaudzi, 2003). The different reference ranges used in dietary adequacy makes it challenging when comparing results, as does the varying methodologies, sample sizes and age of participants. Only one of these studies investigated the energy and nutrient intakes of pregnant women in Cape Town (Klinger, 2004). With the South African FBDG having been

recently updated in 2012, studies comparing intake to these guidelines may be useful in assessing effectiveness of the guidelines. Furthermore, these improved science-based guidelines can also provide a means to assess the quality of general population level dietary intake. None of the above mentioned studies made direct comparisons to the FBDG.

Several studies have investigated NARs in South African samples (Napier & Oldewage-Theron, 2015; Steyn et al., 2014; Oldewage-Theron & Kruger, 2010; Steyn et al., 2005). However, these were conducted mostly on children and adolescents and no studies have assessed the NAR in pregnant women in South Africa. Further studies on the adequacy of dietary intake of pregnant women in Cape Town are clearly needed to provide insights to advise the development of suitable interventions in order to optimise the nutritional status of these individuals, as well as pregnancy outcomes.

This research sets out to investigate the dietary adequacy of pregnant women attending the Bishop Lavis MOU in Cape Town in terms of the DRIs, the South African FBDG, NAR and MAR and associations thereof with anthropometric and select socio-demographic variables.

5.2 Results

Energy intake

The mean observed energy intake of the study sample was 10 168.4kJ with the majority of participants not meeting the EER (Table 5.1). However, the mean predicted energy requirement calculated using the IOM equations for pregnant adolescent and adult participants, was 9 043.7 kJ per day (IOM, 2005). Based on this predicted EER value, only 34.2% of the study sample had energy intakes lower than the requirements. There were no significant differences between adolescent and adult participants for any of the energy variables or in the mean values and proportions below the cut-points (not included in table).

TABLE 5.1 *Energy and macronutrient intakes of the study sample (n=152)*[#]

MACRONUTRIENTS	Mean (SD)	Median (IQR)	DRI	% TE	% < Cut point	% TE range [a]
Total energy (kJ)	10 168.4 (3147.9)	9 511.6 (7 998.0 – 12 099.8)	11 520.6 [b] 9 043.7*	-	79.6% 34.2%*	-
Adolescents	10 321.8 (3 096.5)	9 321.6 (8 570 – 11 059)	9 945.6 9 696.1*	-	56.7% 53.3%*	-
Adults	10 130.7 (3 171.9)	9 636.1 (7 785.8 – 12 224.1)	11 520.6 8 879.2*	-	77.9% 33.6%*	-
Calculated total energy (kJ)	9 043.7				34.2%	
Energy without alcohol (kJ)	10 136.0 (3145.4)	9 511.6 (7 998.0 – 12 098.4)	-	-	-	-
Total energy (kJ/kg current weight)	168.9 (48.9)	161.4 (133.8 – 198.1)	-	-	-	-
Protein (g)	78.8 (28.1)	75.3 (63.2 - 90.5)	71.0 [c]	10.2%	42.8%	10-15%
Protein (g/kg)	1.3 (0.6)	1.2 (1.0 – 1.6)	0.88 [e]	-	-	-
Total fat (g)	89.9 (34.9)	85.2 (65.1 - 111.1)	-	46.0%	-	20-35%
MUFA (g)	30.1 (12.8)	28.1 (21.0 – 37.4)	-	15.7%	-	Remainder of energy from fat
PUFA (g)	25.5 (11.2)	23.2 (17.5 – 32.2)	-	14.8%	-	6–10%
Saturated fat (g)	3.53 (5.59)	0.68 (0.03 – 3.71)	-	12.3%	-	6–10%
Cholesterol (g)	264.45 (133.47)	247.52 (174.7 – 359.3)	-	-	-	-
Carbohydrates (g)	303.5 (95.4)	292.5 (234.3 - 354.2)	135 [e]	42.8%	0.0%	55-75%
Fibre (g)	20.7 (8.8)	18.5 (14.6 - 24.9)	28 [d]	-	98.0%	-
Added Sugar (g)	89.0 (37.58)	83.1 (58.3 – 113.0)	-	20.5%	-	<10%
Alcohol (g)	1.12 (2.3)	0.0 (0.0 – 1.02)	0.0	0.34%	-	-

Abbreviations: MUFA = Mono-unsaturated fatty acids; PUFA = Poly-unsaturated fatty acids; SD = Standard deviation; IQR = Inter-quartile range; TE = Total energy

No significant differences between adolescent and adult participants

* Energy requirements calculated for each participant using the IOM EER calculations using age, height, weight, sedentary PAL and adolescent/ adult coefficients

[a] Institute of Medicine, 2005

[b] EER as outlined by Food and Nutrition board of the IOM, 2002, 2005

[c] RDA, [d] AI and [e] EAR as outlined by Food and Nutrition board of the IOM, 2005

Validity of energy intake estimate

In the total sample 18.1% were classified as under-reporters, 55% were classified as accurate reporters, and 26.9% as over-reporters (Table 5.2). There was a non-significant trend for under-reporting to be more likely in those with a BMI in the obese range, especially those with a BMI >35kg/ m^2, with 45.5% of participants in this category having under-reported. This proportion was triple in proportion to under-reporting found for the normal and overweight groups. Those in the normal and overweight categories were three times more likely to over-report than those in the BMI >35kg/ m^2 category. The majority of participants in other BMI categories were however classified as accurate reporters (Table 5.2).

TABLE 5.2 *Accuracy of reporting of energy intake according to the Goldberg cut-off method by BMI categories; row% and column %*

		Goldberg Categories			
Row %		**Under-reporters n (%)**	**Accurate reporters n (%)**	**Over-reporters n (%)**	**n (%)**
BMI categories (kg/m^2)	**< 25**	14 (16.1)	48 (55.2)	25 (28.7)	**87 (58.4)**
	25 - 29.9	5 (13.5)	21 (56.8)	11 (29.7)	**37 (24.8)**
	30 - 34.9	3 (21.4)	8 (57.1)	3 (21.4)	**14 (9.4)**
	≥ 35	5 (45.5)	5 (45.5)	1 (9.1)	**11 (7.4)**

Abbreviations: BMI = Body Mass Index

Pearson Chi Square p-value = 0.0929

Macronutrient intakes

Although the average protein intake exceeded recommendations, nearly half of the study sample did not meet the EAR (Table 5.1). The EAR for carbohydrates was met by all participants. The fibre intake of the majority of the study sample was below the AI, with only three (2%) participants meeting recommendations. Although average alcohol intake

contributed only a small amount to total energy intake (Table 5.1) 49 participants (32.2%) reported drinking alcohol around conception and during the first trimester of their pregnancies. Twelve (24.5%) of these were adolescents (not included in table).

Macronutrient distribution

Carbohydrates contributed a mean of 42.8% to total energy, which falls below the recommendation of 55% – 75% of total energy suggested by the IOM (2002) (Table 5.1). Added sugar contributed 20.5% to total energy intake, making it just under 50% of the total carbohydrate contribution. Protein contribution to total energy intake fell within the lower end of the recommended range, while total fat (46%) and saturated fat (12.3%) contribution exceeded the upper end of the recommended ranges. No participants had a fat intake below the recommended level. There were no significant differences in any of the macronutrient variables, between adult and adolescent participants (data not included in table).

Vitamins

All participants had inadequate vitamin D and folate intakes, while close to two thirds of the participants had inadequate intakes of pantothenic acid, biotin, and vitamin A (Table 5.3). Thiamin, vitamin E and vitamin C intakes were inadequate for approximately one in every three participants. One in ten participants had inadequate intakes of vitamin B12 and riboflavin.

Adolescent participants had a significantly ($p=0.0308$) higher Vitamin B12 intake [mean(SD) of 3.9(2.1)] than adult participants [mean(SD) 3.6(2.2)]. There were no other significant differences in vitamin intakes between adolescent and adult participants.

TABLE 5.3 *Mean and adequacy of vitamin intake by the study sample (n=152)*

VITAMINS	Mean (SD)	Median (IQR)	EAR/ AI [d]	% < Cut point
FAT SOLUBLE				
Vitamin A (mcg)	720.3 (536.2)	561.9 (393.2 - 832.0)	550	62.5%
Vitamin D (ug)	3.8 (2.4)	3.3 (1.9 - 5.4)	10	100.0%
Vitamin E (mg)	15.4 (7.3)	13.5 (10.3 - 18.5)	12	23.7%
WATER SOLUBLE				
Thiamin (mg)	1.4 (0.5)	1.4 (1.1 - 1.7)	1.2	29.6%
Riboflavin (mg)	2.6 (2.0)	2.0 (1.4 - 3.3)	1.2	7.9%
Niacin (mg)	25.6 (8.8)	25.1 (19.6 – 30.0)	14	0.0%
Vitamin B6 (mg)	3.9 (1.6)	3.5 (2.7 - 4.8)	1.6	0.0%
Vitamin B12 (mcg)	3.7 (2.1)	3.2 (2.4 - 4.6)	2.2	9.9%
Pantothenate (mg)	6.5 (3.6)	6.0 (4.2 - 7.9)	6 [d]	77.6% [d]
Biotin (mcg)	34.7 (20.8)	29.4 (21.9 - 40.2)	30 [d]	61.8% [d]
Folate (ug)	284.5 (134.9)	248.6 (191.3 - 346.2)	520	100.0%
Vitamin C (mg)	99.4 (82.5)	85.4 (53.5 - 123.4)	70	33.6%

Abbreviations: SD = Standard deviation; IQR = Inter-quartile range; EAR = Estimated average requirements AI = Adequate intake

[d] AI as outlined by Food and Nutrition board of the IOM, 2005

Minerals

With the exception of calcium, iron and potassium the mean intake of all minerals was above the respective EAR/AI cut-points (Table 5.4). Almost all participants (>95% below cut-point) had inadequate intakes of calcium, iron and potassium. Inadequate intakes of manganese and magnesium were also common (>67% and <80% below cut-point). Inadequate zinc intake was evident in one fifth of the study sample, while intakes of phosphorous, sodium and copper were adequate in majority of the sample. While only 5.9% had an intake below the cut-point for sodium, 81.1% of participants had an intake above the AI of 1500mg. No significant differences were found in mineral intakes between adolescent and adult participants.

TABLE 5.4 *Mean and adequacy of mineral intake by the study sample (n=152)*

MINERALS	Mean (SD)	Median (IQR)	EAR/ AI [d]	% < Cut point
Calcium (mg)	539.0 (295.6)	467.0 (325.0 - 715.4)	800	98.0%
Iron (mg)	14.1 (4.6)	13.5 (11.0 - 16.7)	22	99.3%
Magnesium (mg)	275.5 (91.5)	261.7 (215.3 - 318.7)	290	77.0%
Phosphorous (mg)	1096.8 (386.8)	1035.4 (848.6 - 1255.5)	580	1.3%
Potassium (g)	2674.7 (1012.0)	2519.7 (1962.0 - 3130.7)	4700 [d]	99.3% [d]
Sodium (mg)	2315.0 (936.1)	2148.9 (1674.8 - 2861.4)	1500 [d]	5.9% [d]
Zinc (mg)	12.0 (4.3)	11.2 (8.7 - 14.4)	9.5	21.7%
Copper (ug)	1.3 (0.5)	1.2 (1.0 - 1.6)	0.8	0.0%
Manganese (mg)	2117.0 (1004.4)	1872.8 (1356.1 - 2499.3)	2000 [d]	67.1% [d]

Abbreviations: SD = Standard deviation; IQR = Inter-quartile range; DRI = Dietary reference intakes AI = Adequate intake

[d] AI as outlined by Food and Nutrition board of the IOM (2005)

Adequacy of intake according to Food Based Dietary Guidelines

The results in Table 5.5 show that the average number of starch servings consumed was 1.5 times greater than that recommended by the South African FBDG. Intakes of fruits and vegetables, “milk” servings, as well as dry beans, split peas, lentils, and soy were below the recommended number of servings. The numbers of “meat, poultry, fish, and eggs” consumed were just above the guidelines. Sugar and fat servings were consumed seven to nine times daily, where the recommendations suggest using these “sparingly”. No statistically significant differences were found between adolescent and adult participants.

TABLE 5.5 *Average per capita daily number of servings per food group * consumed by the study sample compared to recommended servings according to the South African Food Based Dietary Guidelines***

Food groups	Standard Serving size **	FBDG recommended number of servings **	Average number of servings
Starch (Bread, cereal, rice, pasta)	1 slice of bread (30g); 30g dry/ 100g wet cereal; ½ cup cooked rice/ pasta/ porridge; 1 small potato	6 – 8 per day	10.6 per day
Vegetables and fruit	1 medium fruit; ½ cup cooked vegetables; 1 cup raw vegetables	5 per day	3.0 per day
Meat, poultry, fish, eggs	75-100g cooked chicken/ fish/ meat without bone; 2 eggs	2 – 3 per day	3.8 per day
Dry beans, split peas, lentils, soy	1 cup/ 200g cooked	At least 3 per week	0.3 per week
Milk	1 cup milk/ yoghurt; 40-50g cheese	2 per day	1.2 per day
Fats (including nuts and oils)	±1 teaspoon oil	'Sparingly'	9.7 per day
Sugar	±1 tablespoon sugar	'Sparingly'	7.7 per day
Salt	-	'Sparingly'	-

Abbreviations: FBDG = Food based dietary guidelines

* Derived from total grams consumed in a food group divided by the indicated serving size for each food item

** Food-Based Dietary Guidelines for South Africa. S Afr J Clin Nutr. 26(3) (Supplement):S1-S164

Analysis of Nutrient Adequacy Ratio (NAR) and Mean Adequacy Ratio (MAR)

Analysis of the NAR showed adequate intakes of protein, carbohydrates, vitamin E and C, and all B vitamins with the exception of folate (Table 5.6). The NAR for fibre, vitamin D, calcium, potassium and iron also indicate poor intakes of these nutrients. The NAR for vitamin B12 was significantly higher for adolescents [151.8 (38.3)] than adults [145.9 (41.8)] (Pearson's Chi^2 test p=0.0255). The mean(SD) MAR for the total group was 84.3(5.2)% with no significant difference between adolescent [mean(SD) 84.6(4.3)%] and adult [mean(SD) 84.3(5.5)%] participants (Independent sample t-test p=0.7775).

TABLE 5.6 *Nutrient adequacy ratios (NARs) for the dietary intake of the study sample (n=152)*

	NAR Mean (SD)		NAR Mean (SD)
Energy	86.1 (16.4)	Biotin	97.3 (24.9)
Protein	119.5 (25.2)	Folate	50.8 (11.7)
Carbohydrates	213.6 (39.8)	Vitamin C	116.7 (38.9)
Fibre	66.2 (13.5)	Calcium	56.8 (18.3)
Vitamin A	97.5 (25.5)	Iron	62.0 (12)
Vitamin D	30.4 (7.9)	Magnesium	89.6 (17)
Vitamin E	116.9 (23)	Phosphorous	178.3 (39.1)
Thiamin	113.5 (21.1)	Potassium	52.5 (10.4)
Riboflavin	157.9 (49.7)	Sodium	144.0 (28.2)
Niacin	168.0 (27.3)	Zinc	121.4 (24.5)
Vitamin B6	229.8 (47.2)	Copper	152.0 (29.5)
Vitamin B12	147.0 (41.1)[a]	Manganese	94.3 (25)
Pantothenate	88.3 (17.8)		

Abbreviations: SD = Standard deviation

NAR = observed intake/ recommended (DRI) intake; values <100 represent intake below the requirements

a Significant difference between adolescent and adult participants (Pearson's Chi^2 test p=0.0255).

Exploratory association analysis between dietary intakes, socio-demographic and anthropometric variables

A number of significant correlations between dietary intake, socio-demographic and select anthropometric variables were found (Table 5.7). However, these associations need to be interpreted with caution as the r-values were generally low (<0.2).

In the total sample, the greater the number of individuals supported by monthly income the lower the energy and protein intake. A higher number of years of formal education was associated with higher intakes of zinc and total protein. There were no significant correlations between dietary intake variables and maternal age, gestational age, recruitment BMI and MUAC in the total study sample (Table 5.7 to 5.9).

In the adolescent group, the greater the number of people supported by an income the higher the intake of biotin. A greater MUAC was associated with a higher energy intake (Table 5.7 to 5.9).

In the adult group, the greater the number of people supported by income, the higher the intake of energy, total protein, carbohydrates, calcium, zinc, magnesium, phosphorous, potassium, copper, vitamin B12 and alcohol. A greater number of years of formal education was associated with higher total protein, saturated fat, calcium, phosphorous, potassium, zinc, and vitamin A intakes. A higher intake of alcohol and saturated fat were associated with a greater MUAC (Table 5.7 to 5.9). Dietary variables for which no significant correlations were found for the total group and within the adolescent and adult groups were not included in the following tables.

TABLE 5.7 *Correlation matrix for association between* ***energy and macronutrient*** *intake and anthropometric and socio-demographic variables for total study sample and by age groups*

	Years of formal education	No. supported by income	Gestational age	Recruitment BMI	MUAC
Energy	NS	**-0.1672 p=0.0402**	NS	NS	NS
Adolescents	NS	NS	NS	NS	**-0.3714 =0.0433**
Adults	NS	**-0.184 p=0.0433**	NS	NS	NS
Energy without alcohol	NS	**-0.1677 p=0.0396**	NS	NS	NS
Adolescents	NS	NS	NS	NS	**-0.3714 =0.0433**
Adults	NS	**-0.1852 p=0.0419**	NS	NS	NS
Total protein	**0.1874 p=0.0208**	**-0.2089 p=0.0101**	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	**0.2024 p=0.0254**	**-0.1972 p=0.0302**	NS	NS	NS
Saturated fat	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	**0.2391 p=0.008**	NS	NS	NS	**0.1872 p=0.0423**
MUFA	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	NS	NS	NS	NS	**0.1825 p=0.0479**
Carbohydrates	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	NS	**-0.1812 p=0.0467**	NS	NS	NS
Fibre	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	**-0.4273 =0.0185**
Adults	NS	NS	NS	NS	NS

Abbreviations: MUAC = Mid-upper arm circumference BMI = Body mass index NS = Not significant

Spearman's correlation coefficient used for all variables except those marked with an * where Pearson's correlation coefficient was used

Dietary variables for which no significant correlations were found for the total group and within the adolescent and adult groups were not included in the table

TABLE 5.8 *Correlation matrix for association between* ***mineral*** *intake and anthropometric and socio-demographic variables for total study sample and by age groups*

	Years of formal education	No. supported by income	Gestational age	Recruitment BMI	MUAC
Calcium	NS	**-0.2005 p=0.0136**	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	**0.2553 p=0.0045**	**-0.2168 p=0.0169**	NS	NS	NS
Iron	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	NS	NS	**-0.0780 p=0.537 ***	NS	NS
Magnesium	NS	**-0.1788 p=0.0281**	NS	NS	NS
Adolescents	NS	NS	NS	NS	**-0.3582 p=0.0519**
Adults	NS	**-0.192 p=0.0349**	NS	NS	NS
Phosphorous	NS	**-0.2356 p=0.0036**	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	**0.2288 p=0.0113**	**-0.2502 p=0.0056**	NS	NS	NS
Potassium	NS	**-0.191 p=0.0188**	NS	NS	NS
Adolescents	NS	NS	**-0.5481 p=0.042 ***	NS	**-0.3981 p=0.0294**
Adults	**0.1967 p=0.0299**	**-0.237 p=0.0089**	NS	NS	NS
Zinc	**0.194 p=0.0166**	**-0.1971 p=0.0153**	NS	NS	NS
Adolescents	NS	NS	NS	NS	**-0.3763 p=0.0404**
Adults	**0.1933 p=0.0329**	**-0.1968 p=0.0305**	NS	NS	NS
Copper	NS	**-0.1788 p=0.028**	NS	NS	NS
Adolescents	NS	NS	**-0.5534 p=0.040 ***	NS	**-0.4477 p=0.0131**
Adults	NS	**-0.227 p=0.0123**	NS	NS	NS
Manganese	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	**-0.4134 p=0.0231**
Adults	NS	NS	NS	NS	NS

Abbreviations: MUAC = Mid-upper arm circumference BMI = Body mass index NS = Not significant

Spearman's correlation coefficient used for all variables except those marked with an * where Pearson's correlation coefficient was used

Dietary variables for which no significant correlations were found for the total group and within the adolescent and adult groups were not included in the table

TABLE 5.9 *Correlation matrix for association between* ***vitamin*** *intake and anthropometric and socio-demographic variables for total study sample and by age groups*

	Years of formal education	No. supported by income	Gestational age	Recruitment BMI	MUAC
Vitamin A	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	**0.2036 p=0.0245**	NS	NS	NS	NS
Niacin	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	**-0.3611 p=0.05**	NS
Adults	NS	NS	NS	NS	NS
Vitamin B6	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	NS	NS	NS	NS	NS
Vitamin B12	NS	**-0.2073 p=0.0106**	NS	NS	NS
Adolescents	NS	NS	NS	NS	NS
Adults	NS	**-0.2303 p=0.011**	NS	NS	NS
Pantothenic acid	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	**-0.4002 p=0.0284**	NS
Adults	NS	NS	NS	NS	NS
Biotin	NS	NS	NS	NS	NS
Adolescents	NS	**0.5974 p=0.024 ***	NS	NS	NS
Adults	NS	NS	NS	NS	NS
Vitamin D	NS	NS	NS	NS	NS
Adolescents	NS	NS	**0.6826 p=0.007 ***	NS	NS
Adults	NS	NS	NS	NS	NS
Vitamin E	NS	NS	NS	NS	NS
Adolescents	NS	NS	NS	**-0.3673 p=0.0459**	**-0.3863 p=0.035**
Adults	NS	NS	NS	NS	NS
Alcohol	NS	NS	NS	NS	NS
Adolescents	NS	NS	**0.6505 p=0.012 ***	NS	**0.3571 p=0.0527**
Adults	NS	**0.1854 p=0.0417**	NS	NS	NS

Abbreviations: MUAC = Mid-upper arm circumference BMI = Body mass index NS = Not significant

Spearman's correlation coefficient used for all variables except those marked with an * where Pearson's correlation coefficient was used

Dietary variables for which no significant correlations were found for the total group and within the adolescent and adult groups were not included in the table

5.3 Discussion

Energy requirements during pregnancy are influenced by several factors. These include age, pre-pregnancy BMI, rate of weight gain during pregnancy, genetics, physical activity and other lifestyle factors. The EER is an energy intake recommendation that is predicted to maintain energy balance in healthy, normal weight individuals, but does not necessarily reflect the exact energy requirements of each woman. The equation takes into consideration the age, height, weight and physical activity of each participant and a factor is added for the increased requirements of pregnancy (IOM, 2009). Although the mean energy intake (10 168kJ) of our sample was below the EER, it was higher than the 8 425.7kJ reported for pregnant women (16 to 35 years old) from Gauteng (Kesa & Oldewage-Theron, 2005) and the 9123kJ reported for pregnant adolescents (14 to 19 years old) from Limpopo (Tshitaudzi, 2003). The proportions of participants in our study who did not meet energy requirements using the EER of 11 520kJ (79.6%) and the calculated mean predicted energy requirement of 9 043.7kJ (34%) as cut points, were higher than the 26.1% reported for pregnant women (aged 13 to 40 years) from Limpopo (Mostert et al., 2005). It needs to be borne in mind that projected energy requirements of subjects included in our study and that of women in the above mentioned studies may not be comparable, as our study sample was mostly in their late first or early second trimester, while participants in the studies by Kesa and Oldewage-Theron (2005) and Mostert et al. (2005) were in their third trimester. Furthermore, participants in the study by Tshitaudzi (2003) were all adolescents. It needs to be noted that the predicted energy requirement calculated for our participants was based on a sedentary PA coefficient, thus assuming that all participants had low levels of physical activity, as we did not assess actual physical activity levels. Ritchie and King (2008) and others point out that physical activity levels influences pregnancy energy requirements, and as such, the mean predicted energy requirements of our sample may have been more in line with the EER if a PA coefficient based on actual physical activity had been used.

Inspection of accuracy of energy reporting using the Goldberg cut-off method (categories) showed that 55% of the study population could be classified as accurate reporters, with over-reporting being more common than under-reporting (26.9% and 18.1% respectively). These results are in line with published findings on accuracy of reporting in similar groups. Under-reporting was identified in 25.2% and 22% of non-pregnant women in West Africa (Becquey

& Martin-Prevel, 2010) and Washington DC (Tooze et al., 2012) respectively. Winkvist et al. (2002) reported that 30% and 16% of pregnant women from Indonesia under-reported their energy intake during their first and second trimesters respectively. However, the prevalence of under-reporters in our study is much lower than the 83% found in adults from a study in Khayelitsha, Cape Town (Orcholski et al., 2015). Our results show that participants in the BMI $\geq$35kg/m^2 category tended to be three times more likely to under-report their energy intake (45.5 %) than those in the normal and overweight categories (16.1% and 13.5 % respectively) and two times more likely than those in the BMI $\geq$30 and < 35kg/m^2 category (21.4%). These results are in line with findings that the obese are likely to under-report their dietary intake (Orcholski et al., 2015; Winkvist et al., 2002). The fact that a total of 45% of our sample was classified as either under- or over-reporters indicates that the results of ranking of individual participants according to DRI cut-offs needs to be interpreted with caution.

Availability of sufficient food to meet energy requirements is generally acknowledged to be linked to food security (income) (Hanson & Connor, 2014; Bruening et al., 2012). Our results support this contention as lower energy intake was associated with having a lower income and having to sustain greater numbers in the household. Poor food security may also impact negatively on the quality and variety of foods consumed and thus the adequacy of especially good quality protein and micronutrient intakes (alluded to in more detail later in this discussion) (Hanson & Connor, 2014; Brooks et al., 2010).

Within the context of the energy balance equation the expectation would be for energy intake to be associated with weight status outcomes. However, as has been found in many other studies (Caudwell et al., 2013; Blundell et al., 2012), BMI (at recruitment) was not significantly associated with energy intake in our study. This may be a reflection of the truth, but on the other hand may be the result of inaccurate reporting by 45% of the sample, as well as moderating effects of varying levels of physical activity, and thus energy expenditure. However, the positive association between energy intake and MUAC in adolescent participants supports the notion that sufficient energy intake results in improved nutritional outcomes in this age group. With a greater proportion of adolescents having an intake below their calculated EER, they may have an increased risk of malnutrition and poor pregnancy

outcomes, especially of LBW (Lopez et al., 2011; Kruger, 2005). This is expected in this subgroup as the increased requirements of pregnancy on top of the increased needs of adolescence are difficult to meet, especially in a resource-deprived environment.

Protein requirements increase substantially during pregnancy (Otten et al., 2006; Erick, 2008; Williamson, 2006). The mean protein intake of our study sample (both adolescent and adult participants) seems to be sufficient as it exceeded the DRI recommendation and contributed 10.1% to total energy intake, which is at the lower end of the recommended range. The NAR for protein of 119% also reflects sufficient protein intake. Further support for this comes from the assessment of adequacy of intake based on the FBDG, namely that the average intake of the meat, poultry, fish, and egg group was above the number of servings recommended by the FBDG (Schonfeldt et al., 2013). The mean protein intake (78.8 ±28.1g) of the current study sample was in line with that of 73.18 ± 23g (Kesa & Oldewage-Theron 2005) in a similar group of pregnant women and higher than the 60.9g reported by Tshitaudzi (2003) in pregnant adolescents. Despite the apparent overall sufficiency of intake, it is concerning that 42.8% of our study sample had inadequate protein intake according to the EAR cut-point method, which is much higher than the 24% in pregnant women from Limpopo reported by Mostert et al. (2005) based on the <67% of the RDA cut-point. Inadequate protein intakes hold serious risks for especially the foetus, and can negatively impact neurological and cognitive development (Antonow-Schlorke et al., 2011; Tolsa et al., 2004), as well as increase the risk of insulin resistance and hypertension in the child in later life (Sinclair & Watkins, 2013).

The range of protein intake found in our sample (minimum 8.3g and maximum 380.4g per day) may be explained by a number of factors. Firstly, the possibility of under and over-reporting needs to be considered as incorrect reporting of energy intake may inadvertently result in inaccuracies in estimations of the intake of other nutrients. Income is a further factor to consider as protein sources, specifically good quality sources of protein, are often expensive (Brooks et al., 2010). The poor socio-demographics (level of education and number of individuals in the household) of a large part of the study sample may be the prime explanation for the inadequate protein intake in almost half of the study sample. This is supported by the correlations found between a higher protein intake and a higher educational

level and lower number of people supported by household income. In line with these results, Bopape et al. (2008) and Jaffer et al. (2008) also reported low average intakes of meat and meat products in pregnant women from low socio-economic areas. The fact that participants with poorer protein intake during pregnancy had lower recruitment weights and gained less weight during pregnancy may also speak to poorer outcomes as a result of inadequate protein intake. Short-to-medium-term protein inadequacies may contribute to poor tissue and muscle formation in both mother and foetus (Ota et al., 2015; Blumfield et al., 2012; Erick, 2008; Hoppe et al., 2004). This may contribute to sub-optimal brain development, insulin resistance and hypertension in later life (Sinclair & Watkins, 2013)

The study sample is characterized by high mean percentage total fat (46.0% TE) and saturated fat (12.3%) contributions to total energy intake, with both exceeding current recommendations of 20-35% and <7% respectively. This can be explained by the high number of servings of fats that were consumed on a daily basis when compared to the FBDG that recommends using fat sparingly. The mean total fat intake in the current study (89.9g) is greater than that found by both Kesa and Oldewage-Theron (2005) (62.3g) and Tshitaudzi (2003) (48.4g). High fat intakes may be linked to the greater palatability of fatty foods such as fried foods, fatty meat cuts, full cream dairy products, as well as satiation effects when compared to low-fat options such as lean meats and low fat spreads (Weltens et al., 2015; Grabenhorst & Rolls, 2014). According to Brooks et al. (2010) high fat foods are also generally cheaper and more readily available than lower fat options in lower socio-economic status communities. Although no association was found between total fat intake and education or number of people in the household, a higher level of education was associated with higher saturated fat intake in the adult pregnant women, which could reflect higher intake of fats from dairy and meat (animal protein products), confirming the possibility that they may have the means to purchase more animal protein. Saturated fat and cholesterol intakes were positively correlated with MUAC in the adult pregnant women, indicating lower risk for malnutrition in those consuming animal fats (per implication possibly higher animal protein as well) (Kruger, 2005). However, these results should be interpreted with caution as no associations between fat intake and BMI at recruitment or prior to pregnancy were found. The intake of PUFAs exceeded the recommended ranges for total energy. However, this incorporates both omega-3 and omega-6 PUFAs, and these were not analysed separately in this study. We can expect that the majority of this intake was in the form of omega-6 PUFA

due to the high intake of foods prepared with sunflower oil and the low intake of omega-3-rich fish. As a result, it is possible that this particular sample of pregnant adolescent and adult participants have an increased risk of problems such as impaired cognitive and behavioural development (Innis, 2009) and increased risk of obesity in later life (Muhlhausler et al., 2010; Ailhaud et al., 2006) due to inadequate omega-3 intake. The MUFA intake of 15% is within the recommendations. No other studies conducted in South Africa on pregnant women reported on the intake of MUFAs and PUFAs.

Carbohydrate requirements increase during pregnancy, but an excessive intake can cause high blood glucose levels, which may increase the risk of gestational diabetes and excessive foetal size (Scholl et al., 2001). The risk of both the mother and child developing diabetes and heart disease later in life may also be increased (Bellamy et al., 2009). No participants in the current study had an inadequate carbohydrate intake. In fact, the mean intake was more than double the daily requirement. This is reiterated by a NAR for carbohydrates of over 200% and the frequency of intake of foods from the starch group that exceeded the FBDG recommendation (Vorster, 2013). Similarly, high carbohydrate intakes were also reported by Kesa and Oldewage-Theron (2005), Mostert et al. (2005), Klinger (2004), Tshitaudzi (2003), and Bopape et al. (2008). Jaffer et al. (2008) also listed starch-rich foods as being part of the most frequently consumed foods. Despite meeting the DRI that is set at a particular level to prevent ketosis (Erick, 2012), carbohydrates contributed less to total energy (42.8%) than the recommended 55% to 75%. As the protein contribution to total energy was at the lower end of the recommended range, it follows that the high fat intake accounts for the lower carbohydrate contribution. With many, often more refined, carbohydrates viewed as being more affordable, a higher intake in those with a lower income could be expected. However, the negative association found between carbohydrate intake and the number of people supported by household income in adult participants does not support this notion.

A major concern is the very high added sugar intake in our study sample. The mean intake of the total group was 89g per day which constitutes 20.5% of total energy intake. The FBDG suggest that foods and drinks containing sugar be consumed sparingly, and not between meals, and that this constitutes a maximum of 10% of total daily energy intake (Temple & Steyn, 2013). A recent WHO report strongly recommends an intake of free sugars of less than

10% total energy, while also making a conditional recommendation (as a result of the nature of the evidence) of further reducing this to 5% of total energy intake. New UK recommendations have adopted the lower intake of free sugars of 5% of total energy. With our study sample consuming just more than double the upper limit recommended by the WHO and FBDG, their risk of types 2 diabetes (WHO, 2015b; Aune et al., 2013; Greenwood et al., 2013), obesity (WHO, 2015b; Te Morenga et al., 2013), dental caries (WHO, 2015b; SACN, 2015) and possibly cardiovascular disease (WHO, 2014b, 2003; Johnson et al., 2009) is increased. None of the other studies done on South African pregnant adolescent and adult participants reported on quantities of sugar consumed, Kesa and Oldewage-Theron (2005) found sugar to be in the top ten most commonly consumed foods, and Jaffer et al. (2008) reported sugar to be consumed an average of 8.2 (cases = mothers of <2500g birth weight babies) and 9.6 (controls = mothers of ≥2500g birth weight babies) times per week. Lower sugar intakes of 38g/day and 51g/day were reported for black men and women respectively in Cape Town who participated in the Cardiovascular Risk in Black South Africans (CRIBSA), making up 11-15% of total energy intakes (Steyn & Temple, 2012).

The majority (96%) of the study population did not meet the daily fibre recommendations, verified by an NAR of 66%. This is indicative of a diet low in fruits, vegetables, and legumes, as is reflected in comparisons to the FBDG. Only 46.1% of participants reported eating any legumes on at least one day of the three days assessed with the average number of servings being 0.3 per day. These results are in line with the low intake of legumes reported by Tshitaudzi (2003), Mostert et al. (2005), and Jaffer et al. (2008) (1.8 servings per week). These studies also did not find regular intake of any fibre-rich foods in pregnant adolescents or adults. Low fibre intakes may increase the risks of constipation, haemorrhoids, and poor blood glucose control (Rizotto et al., 2010; Hibberts & Schizas, 2010).

Frequency of fruit and vegetable intake fell below the 5-per-day recommendations of the FBDG (Naude, 2013) with 5.3% of participants having no vegetables and 18.4% no whole fruit at all during the three days assessed. As with meats and meat products, vegetables were eaten only on Sundays by many of the participants. The low fruit (1.9 servings per day) and vegetable (1.1 servings per day) intake is especially concerning because of the increased requirements for micronutrients to support the growing and developing foetus, and ensure the

health of the mother. Jaffer et al. (2008) found similar low intakes of fruit and vegetables with the frequency of fruit intake in these adolescent and adult participants being 11.7 and 12.4 times per week for cases (birth weights ≤2500g) and controls (birth weights >2500g) respectively and vegetable intake 3.4 and 3.8 times per week respectively. Low fruit and vegetable intake could possibly be explained by lack of affordable options in the direct vicinity of the households (Temple & Steyn, 2011b)

The fact that protein, fibre containing starches, legumes and fruit and vegetable intake did not meet requirements, is reflected in inadequate intakes of some of the micronutrients. This is a concern as the EAR for several vitamins and minerals increase during pregnancy to meet the needs of the mother and foetus. The nutrient with the most substantial increase in requirements during pregnancy is folate. As reported by Mostert et al (2005) and Bopape et al. (2008), none of the participants in our study sample met the EAR for folate, which is also reflected in the NAR of 50.8%. This suggests a poor intake of foods such as liver, mushrooms, spinach, broccoli, lean beef and whole-wheat bread and may increase the risk of neural tube defects (Cox & Phelan, 2008), low birth weight, preterm birth and growth faltering (Erick, 2012; Scholtz et al., 2010). The low number of servings from the vegetable and protein groups consumed on a daily basis as well as the low fibre intake supports the possibility that these foods were not consumed regularly enough to meet the increased requirements of pregnancy.

Both the mean and median vitamin A intake was above the EAR for pregnancy, but a large portion of the study sample did not meet the requirements (62.5% below cut-off), reflecting the wide range of intakes (58.4 – 1599.3mcg). Other South African studies reported similar results. Mostert et al. (2005) found that the mean intake of their sample of pregnant adolescent and adult participants was 74.6% of the RDA, while 76% of their participants consumed less than 67% of the RDA. Tshitaudzi (2003) reported that the mean intake of their sample of pregnant adolescents was 94% of the RDA, with 52.5% of participants consuming less than the RDA. The wide range of intakes recorded for the three 24-hour recalls could be explained by the fact that an extended period of recall is required to accurately determine usual vitamin A intake. Basiotis et al. (1987) suggested that up to 44 days of recall would be required for more accurate measures of group intakes and ranking of individual intake.

Vitamin C intakes showed a similar pattern with mean intakes being above the EAR, yet a third of the study sample fell below the cut-point. Mostert et al. (2005) and Tshitaudzi (2003) reported that 73.9% and 25% of women in their samples had intakes of less than 67% RDA respectively, and Tshitaudzi reported an intake of 118% of the RDA. Basiotis et al. (1987) suggest at least 19 days of recall for valid assessment of mean vitamin C intake in groups of females. However, it could be argued that the inadequate vitamin A and C intakes in participants in our study are confirmed by the low frequency of fruit and vegetable consumption. Emphasising fruit and vegetable intake should thus be a major theme in nutrition education of pregnant women.

The fact that all participants had an inadequate intake of vitamin D should be interpreted with care as the South African Food Composition Tables (Wolmarans et al., 2010) have a high percentage of missing values for food items containing vitamin D. As a zero value is allocated if a value is missing, underestimation of actual intake invariably results. Although Tshitaudzi (2003) also found that 82% of their group of adolescents had an inadequate intake, this may reflect underestimation as their dietary data was also analysed using the South African Food Composition Tables. However, although there are food sources of vitamin D, the amounts are usually inadequate to maintain vitamin D sufficiency (Naude, 2012b), unless the food has been fortified with vitamin D, or supplements are consumed (Pettifor, 2005). Low exposure to sunlight may also cause vitamin D deficiency, as sunlight is required for the formation of pre-vitamin D in the skin (Naude et al., 2012; Naude, 2012b; Malik et al., 2009).

Calcium requirements do not increase during pregnancy for adults, but do increase for pregnant adolescents. Almost all participants showed inadequate intakes, with the mean intake being just 67% of the EAR for adults, and the NAR being 56.8%. Klinger (2004) and Mostert et al. (2005) reported similar results with 85.6% below the RDA and 89.1% <67% RDA respectively. This suggests a general poor intake of dairy products and green leafy vegetables which is supported by the findings of Jaffer et al. (2008) where 50% of the sample did not consume any milk or milk products, and Mostert et al. (2005) where milk and vegetable intakes were reportedly low. In contrast, Kesa and Oldewage-Theron (2005) indicated that fresh milk was the most frequently consumed product in their study sample. The high level of inadequacy in our sample is reiterated by our data which shows that an

average of 1.2 servings of milk products were consumed per day, which falls below the FBDG recommendations of 2 servings per day (Vorster et al. 2013). Canada's Food Guide, (2011) and the USDA guidelines (2011) recommend two and three daily servings of dairy products respectively for pregnant women. Furthermore, the intake of vegetables that are sources of calcium may have been low as overall fruit and vegetable intake did not meet the FBDG recommendation. Promotion of consumption of good sources of calcium during pregnancy should thus remain a focus in education initiatives to prevent the risks associated with low calcium intake, including bone loss (Hacker et al., 2012), osteoporosis (Gallager, 2012) and preeclampsia (Hofmeyer et al., 2014; Imdad & Bhutta, 2012) in the mother, as well as poor bone outcomes in the offspring in later life (Ganpule et al., 2006; Chang et al., 2003).

The general intake of B vitamins, with the exception of thiamine, pantothenate, biotin and folate, is adequate for the majority of the study sample. The NAR supports the finding of inadequate folate intake (50.8%), but not of thiamine (113.5%), pantothenate (88.3%) or biotin (97.3%). Our findings of inadequate thiamine, pantothenate and biotin intakes are not reflected in any other studies done on pregnant women in South Africa and suggests a low intake of fortified and whole grains, peanuts, soy protein, and eggs. While several other studies on pregnant women in South Africa reported inadequate intakes of vitamin B12 (Tshitaudzi, 2003; Klinger, 2004; Modjadji et al., 2007; Bopape et al., 2008) and vitamin B6 (Tshitaudzi, 2003; Mostert et al., 2005), only 9.9% and of the current study sample fell below the cut-point for vitamin B12 and none for vitamin B6. This suggests adequate intakes of meat, liver, milk, fish and cheese in the current study sample.

As with calcium, mean magnesium intake was below the EAR and also below the EAR cut-point for 77.0% of the study sample. None of the other studies reporting on the dietary intake of pregnant South African adolescents/ adults found similar inadequate magnesium intakes (Tshitaudzi, 2003; Klinger, 2003). Since the intake of good sources of magnesium, namely nuts, legumes and dark green leafy vegetables may have been inadequate based on comparison of the frequency of intake with the FBDG for intake of these foods, the study sample may be at risk of a magnesium deficiency. Increased consumption of these foods should thus be emphasised. Low magnesium intakes may result in preterm labour (Rylander,

2014; Hantoushzadeh et al., 2007; Durlach, 2004), hypertension, IUGR and LBW (Rylander, 2014; Jain et al., 2010; Cooke & Mimouni, 1997).

Sodium intake was above the AI for 81.1% of the study sample, with a mean intake of 2315.0 mg per day. The NAR of 144% reflects these values, as do the findings of high intakes of crisps, bread and processed meats. The other South African studies on pregnant women that were assessed did not comment on sodium intakes. This suggests an increased risk of the current study sample of developing PIH and pre-eclampsia, as the upper limit for pregnany is 2.3g/day (Steffen, 2014; Rakova et al., 2014).

Iron requirements are increased in pregnancy to ensure adequate expansion of maternal plasma volume for sustaining foetal growth and nutrient transport (Scholl, 2011). Low iron intakes thus clearly put the mother and foetus at risk of poor pregnancy outcomes. It is therefore very concerning that the mean intake of iron of our study sample was well below the EAR for pregnant women and 99% of participants had an intake below the cut-point. The NAR of 62.0% for iron supports the findings of low intake. Low iron intakes are usually the result of poor consumption of meat, eggs, legumes, whole-grain foods and fortified cereals (Gallagher, 2012). While comparisons to the FBDG show that the daily frequency of intake of items from the meat, poultry, fish and eggs group seems sufficient, the intake of legumes is very low, and based on the results of fibre intake starches that were consumed, were most probably refined options. Iron and folate supplementation is included in the Department of Health protocol for ante-natal care (DoH, 2007b) and may decrease the risk of iron deficiency. However, this does not detract from the importance of ensuring that the dietary intake for pregnant women during pregnancy is optimal.

Bearing in mind the limitations of self-reported dietary intake, especially the problem of under- and over-reporting, it can be concluded that while the pregnant adolescent and adult participants in our study sample have adequate intakes of carbohydrates, almost 50% was in the form of added sugar, and fibre intake was very low. Total fat intake was above recommended levels, with unhealthy fats in the form of saturated fats also being above recommendations. Mean protein intake was sufficient but varied greatly with 43% of the

study sample falling below the cut-point. Micronutrients that may be a concern include folate, vitamin A, vitamin D, calcium, and iron. The comparison of the daily frequency of intake of foods from the different food groups with FBDG recommendations mostly supports macro and micronutrient findings, thus reiterating the fact that the study population may be at nutrition risk. It is clear that antenatal care for women in this area should include a strong nutrition care component. The findings of this study provide some insights into the nutrition risks that need to become the focus of such interventions.

CHAPTER SIX

DIETARY VARIETY

6.1 Introduction

Several vitamin and mineral requirements increase during pregnancy to a much larger degree than the overall energy requirements, especially for the first trimester. For this reason, a nutrient-dense rather than energy-dense diet should be followed (Cox & Phelan, 2008). It is known that no one food or food group can provide all the nutrients required, making variety in dietary intake key to a healthy lifestyle (Steyn & Ochse, 2013), and, by extension, a healthy pregnancy. Consuming a varied diet is the most efficient way of attaining and maintaining the nutritional requirements of pregnancy to reduce the risks of adverse pregnancy outcomes (Christian & Stewart, 2010). Generally, pregnant women tend to be more open to following healthier diets as a means of providing the best that they can for their child (Wilkinson & Tolcher, 2010),

Several studies have been done on dietary diversity in South Africa. Those living in informal or rural areas have been found to have lower dietary diversity (Drimie et al., 2013; Oldewage-Theron & Kruger, 2011), although not all studies reported this association (Faber et al., 2014). Results from national level surveys in South Africa, however, support the notion of poor dietary variety in lower SES populations (Labadarios et al., 2007), and more so in some provinces (Labadarios et al., 2011). These studies were carried out on several population groups, from children, to students and adults. However, no studies could be traced that reported on the dietary variety in pregnant women in South Africa.

This research set out to investigate dietary variety in pregnant women attending a MOU in Bishop Lavis, Cape Town, in terms of the 20 most frequently consumed foods, fruits and vegetables and the top ten foods contributing to total energy and select macro- and micronutrient intakes as well as a diversity score, and associations between these variables and anthropometric and select socio-demographic variables.

6.2 Results

In total, 406 different food items were coded for the 456 24-hour recalls that were included in the analysis conducted for this study. After grouping similar foods where different codes

were allocated to differentiate between different cooking methods or different varieties of the same food item was coded for, 136 items remained (Addendum C).

The top 20 most commonly consumed foods are listed in Table 6.1. Sugar (sugar added to tea, coffee or cereals) was the most commonly consumed food item, with only three participants not reporting any sugar intake for the three days that the 24-hour recalls represented. Other foods containing sugar or refined carbohydrates also featured on the top ten list, including white bread, white rice, carbonated drinks and crisps. Seven items (chicken, milk, beef, cheese, eggs, sausage and polony) on the top 20 list have high protein content. Apples and tomatoes were the only fruit and vegetables on the top 20 list for the total sample and for adults, while for adolescents there were no fruits and only tomatoes as vegetable on this list. The top five most commonly consumed foods by adolescents included sugar, white bread, crisps, carbonated drinks and chicken, while that for adults included sugar, chicken, potatoes, margarine and milk.

The per capita intake for the top six items in terms of servings is 4 per day for sugar (1 serving = 5g), 1.5 for chicken (1 serving = 40g), ± 1.5 for potato (1 serving = 90g), 2 for margarine (1 serving = 5g), ±0.5 for milk (1 serving = 250mL) and 3 for white bread (1 serving = 35g). The per capita intake of white bread is three times higher than that of brown bread. The per capita gram intake of tomato equates to approximately a quarter vegetable serving per day (1 serving vegetable = 80g) and apple to half a fruit serving (1 fruit serving = 80g). Intake of carbonated beverages equates to 1 glass (200ml) per day while the intake of crisps equates to 1 small packet of 30g per day.

TABLE 6.1 *Top 20* ***most commonly consumed foods*** *for the total study sample (n=152) and for adolescent and adult participants*

	Food item	No. of eaters	% eaters	Mean portion size (g)	Mean total portions/ eater/ day (g)	Portion/ capita (g)	Adolescent rank (% eaters)	Adult rank (% eaters)
1	Sugar	149	98.0	33.1	40.1	39.3	1 (100.0)	1 (97.5)
2	Chicken	142	93.4	85.7	68.4	63.9	4 (86.7)	2 (95.1)
2	Potato	142	93.4	179.4	133.5	124.7	4 (86.7)	2 (95.1)
4	Margarine	138	90.8	14.1	11.1	10.1	4 (86.7)	4 (91.8)
5	Milk	137	90.1	153.2	144.6	130.3	8 (83.3)	4 (91.8)
5	White bread	137	90.1	122.7	100.3	90.4	2 (96.7)	7 (88.5)
7	White rice	135	88.8	126.6	90.6	80.5	8 (83.3)	6 (90.2)
8	Carbonated drink	115	75.7	407.1	289.1	218.7	4 (86.7)	9 (73.0)
8	Crisps	115	75.7	58.8	36.8	27.8	3 (90.0)	10 (72.1)
10	Tea	112	73.7	389.0	250.1	184.3	12 (63.3)	8 (76.2)
11	Coffee	104	68.4	356.9	243.7	166.7	10 (70.0)	11 (68.0)
12	Beef	94	61.8	96.9	47.7	29.5	10 (70.0)	12 (59.8)
13	Tomato	79	52.0	95.0	46.5	24.2	15 (56.7)	14 (50.8)
14	Cheese	77	50.7	45.8	22.2	11.3	16 (46.7)	13 (51.6)
15	Eggs	75	49.3	92.2	43.4	21.4	13 (60.0)	16 (46.7)
16	Pasta	70	46.1	205.5	104.7	48.2	13 (60.0)	18 (42.6)
17	Brown bread	69	45.4	130.6	70.0	31.8	24 (36.7)	15 (47.5)
18	Apples	65	42.8	199.3	95.1	40.6	24 (36.7)	17 (44.3)
19	Sausage	64	42.1	76.9	32.4	13.7	18 (43.3)	19 (41.8)
20	Polony	61	40.1	58.7	25.0	10.0	16 (46.7)	21 (38.5)

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Sugar" includes all sugar added to tea, coffee, cereals and porridges

"Potato" includes all boiled, mashed, oven roasted and sautéed potato

"Crisps" refer to store-bought packet chips e.g. Simba, NikNaks, Flings etc.

In the total sample and for adults, apples were the most commonly consumed fruit, followed by a variety of fruit juices. Fruit juice was the most commonly consumed fruit source for adolescents (Table 6.2). Oranges and bananas were consumed by almost one third of the study sample. Fruit consumption in general was not common in this study sample, with 21 (13.8%) participants not reporting any consumption of fruit (or fruit juice) for the three 24-hour recalls. Fruit juice was reported as the only 'fruit' consumed by four (2.6%) participants.

TABLE 6.2 *Top 20* ***fruit*** *consumed by the total study sample (n=152) and for adolescent and adult participants*

	Food item	No. of eaters	% eaters	Adolescent rank (% eaters)	Adult rank (% eaters)
1	Apples	65	42.8	2 (36.7)	1 (44.3)
2	Fruit juice	44	28.9	1 (40.0)	4 (26.2)
3	Oranges	44	28.9	3 (33.3)	3 (27.9)
4	Banana	42	27.6	4 (23.3)	2 (28.7)
5	Peaches	34	22.4	5 (20.0)	5 (23.0)
6	Pear	19	12.5	7 (10.0)	6 (13.1)
7	Naartjie	15	9.9	6 (16.7)	8 (8.2)
8	Dried fruit	13	8.6	8 (6.7)	7 (9.0)
9	Plum	11	7.2	9 (3.3)	8 (8.2)
10	Grapes	10	6.6	9 (3.3)	9 (7.4)
11	Coconut	3	2.0	10 (0.0)	10 (2.5)
12	Lemon	3	2.0	10 (0.0)	10 (2.5)
13	Pineapples	3	2.0	8 (6.7)	12 (0.8)
14	Strawberry	3	2.0	9 (3.3)	11 (1.6)
15	Nectarine	2	1.3	10 (0.0)	11 (1.6)
16	Watermelon	2	1.3	10 (0.0)	11 (1.6)
17	Apricot	1	0.7	10 (0.0)	12 (0.8)
18	Guava	1	0.7	10 (0.0)	12 (0.8)
19	Mango	1	0.7	10 (0.0)	12 (0.8)
20	Melon	1	0.7	10 (0.0)	12 (0.8)
21	Pawpaw	1	0.7	10 (0.0)	12 (0.8)

Tomatoes were the most commonly consumed vegetable, being consumed by just over half of the study sample (Table 6.3). Mixed vegetables and onions were consumed by just over a quarter of the study sample. The consumption of a variety of other vegetables was reported but in less than 20% of the study sample. Eight participants (5.3%) did not report consuming any vegetables for the three days assessed.

TABLE 6.3 *Top 20* ***vegetables*** *consumed by the total study sample (n=152) and for adolescent and adult participants*

	Food item	No. of eaters	% of eaters	Adolescent rank (% eaters)	Adult rank (% eaters)
1	Tomato	79	52.0	1 (56.7)	1 (50.8)
2	Mixed vegetables	46	30.3	2 (26.7)	2 (31.1)
3	Onion	38	25.0	2 (26.7)	3 (24.6)
4	Pumpkin	30	19.7	7 (10.0)	4 (22.1)
5	Carrots	26	17.1	4 (16.7)	5 (17.2)
6	Lettuce	24	15.8	4 (16.7)	6 (15.6)
7	Cabbage	22	14.5	7 (3.3)	5 (17.2)
8	Gem squash	21	13.8	6 (6.7)	6 (15.6)
9	Peas	21	13.8	6 (6.7)	6 (15.6)
10	Mealies	18	11.8	3 (20.0)	8 (9.8)
11	Peppers	16	10.5	5 (10.0)	7 (10.7)
12	Cauliflower	15	9.9	6 (6.7)	7 (10.7)
13	Broccoli	9	5.9	7 (3.3)	9 (6.6)
14	Butternut	8	5.3	8 (0.0)	9 (6.6)
15	Cucumber	8	5.3	7 (3.3)	10 (5.7)
16	Beetroot	7	4.6	7 (3.3)	11 (4.9)
17	Green beans	7	4.6	6 (6.7)	12 (4.1)
18	Mushrooms	5	3.3	8 (0.0)	12 (4.1)
19	Baby marrow	2	1.3	8 (0.0)	13 (1.6)
20	Parsnip	1	0.7	8 (0.0)	14 (0.8)
21	Spinach	1	0.7	8 (0.0)	14 (0.8)

Top ten foods contributing to energy and macronutrient intake

White bread contributed almost 10% to total energy intake for adults and adolescents, with sugar providing the second most energy for adolescents and third most for adults. Each participant consumed an average of 4.2 servings of white bread per day. The four energy dense food items (sugar, crisps, chips, and sugar sweetened beverages) on the top 10 list contributed to 19.8% of total energy intake for the total group and 23.2% for adolescents and 18.7% for adults. Chicken, brown bread and margarine contributed more to the total energy of adults than adolescents, while adolescents consumed more energy from crisps, carbonated drinks, pasta and chips. Maize meal was consumed by only a fifth of the study sample, and made up only 0.8% of total energy intake (not reported in table).

TABLE 6.4 *Top 10 food items* ***contributing to total energy intake*** *for the total study sample (n=152) and for adolescent and adult participants*

		No. of eaters	% eaters	Total quantity (g)	Total mean portion/ eater/ day (g)	Portion/ capita (g)	% of daily TE	Adolescent rank (% TE)	Adult rank (% TE)
1	White bread	137	90.1	41 238.0	100.3	90.4	9.2	1 (9.7)	1 (9.1)
2	Sugar	149	98.0	17 934.0	40.1	39.3	6.6	2 (7.4)	3 (6.4)
3	Potato	142	93.4	56 874.7	133.5	124.7	6.5	4 (6.3)	2 (6.5)
4	Crisps	115	75.7	12 691.5	36.8	27.8	6.4	3 (7.2)	4 (6.1)
5	Chicken	142	93.4	29 154.0	68.4	63.9	5.2	9 (4.0)	5 (5.5)
6	White rice	135	88.8	36 705.0	90.6	80.5	4.3	7 (4.2)	6 (4.3)
7	Carbonated drink	115	75.7	99 742.5	289.1	218.7	3.8	5 (4.6)	7 (3.5)
8	Milk	137	90.1	59 433.5	144.6	130.3	3.4	10 (3.6)	9 (3.4)
9	Brown bread	69	45.4	14 500.0	70.0	31.8	3.2	13 (2.0)	8 (3.5)
10	Margarine	138	90.8	4 608.5	11.1	10.1	3.0	14 (1.9)	10 (3.3)
11	Pasta	70	46.1	21 990.0	104.7	48.2	3.0	6 (4.6)	13 (2.6)
12	Chips	50	32.9	10 775.0	71.8	23.6	3.0	8 (4.0)	12 (2.7)

Abbreviations: TE = Total energy

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Sugar" includes all sugar added to tea, coffee, cereals and porridges

"Potato" includes all boiled, mashed, oven roasted and sautéed potato

"Chips" include all deep fried potatoes, while "Crisps" refer to store-bought packet chips e.g. Simba, NikNaks, Flings etc.

Animal derived proteins in the top ten protein sources were the most important contributors to protein intake (46% combined), with chicken making the greatest and beef the second greatest contributions (Table 6.5). Chicken was mostly consumed with the skin (67.2%), with 5.4% reporting eating a KFC-style fried chicken (not reported in table). Plant protein sources in the top ten contributed a total of 23.0% of total protein intake with bread, especially white bread, being the main contributors. Fish and seafood contributed only 3.4% of total protein intake (Table 6.5). Only 34.9% of fish and seafood choices came from omega-3 rich medium and high fat fish, and 49.4% of all fish consumed was fried (not reported in table). Prominent differences in ranking between adolescent and adult participants include pasta and potato that are ranked higher in adolescents and brown bread and mutton that are ranked higher in adults. Peanut butter was consumed by only 23% of the study sample, and made up only 1.1% of total protein intake (not reported in table).

TABLE 6.5 *Top 10 food items* ***contributing to total protein intake*** *for the total study sample (n=152) and for adolescent and adult participants*

		No. of eaters	% eaters	Total Protein (g)	Mean total/ eater/ day (g)	Portion/ capita (g)	% of Total Protein	Adolescent rank (% TP)	Adult rank (% TP)
1	Chicken	142	93.4	7 479.8	17.6	16.4	20.8	1 (17.2)	1 (21.7)
2	Beef	94	61.8	3 737.3	13.3	8.2	10.4	3 (10.1)	2 (10.5)
3	White bread	137	90.1	3 628.9	8.8	8.0	10.1	2 (11.3)	3 (9.8)
4	Milk	137	90.1	1 936.3	4.7	4.2	5.4	4 (6.1)	4 (5.2)
5	Brown bread	69	45.4	1 305.0	6.3	2.9	3.6	12 (2.4)	5 (3.9)
6	Fish and seafood	58	38.2	1 231.7	7.1	2.7	3.4	6 (3.9)	7 (3.3)
7	Pasta	70	46.1	1 231.5	5.9	2.7	3.4	5 (5.5)	10 (2.9)
8	Cheese	77	50.7	1 210.4	5.2	2.7	3.4	9 (3.4)	6 (3.4)
9	Eggs	75	49.3	1 098.3	4.9	2.4	3.1	8 (3.5)	9 (3.0)
10	Potato	142	93.4	1 066.8	2.5	2.3	3.0	7 (3.5)	12 (2.8)
11	Mutton	37	24.3	1 056.6	9.5	2.3	2.9	13 (2.1)	8 (3.2)
12	White rice	135	88.8	1 035.6	2.6	2.3	2.9	10 (3.0)	11 (2.8)

Abbreviations: TP = Total protein

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Potato" includes all boiled, mashed, oven roasted and sautéed potato

Crisps made the highest contribution to total fat intake (10.8%) with margarine being the second highest contributor to total fat intake (Table 6.6). The majority of participants reported using brick margarine i.e. Marvelo (not reported in table). Potatoes were ranked as the third highest contributor to total fat intake and this can be explained by the cooking methods used (roasted, sautéed, or butter/ margarine added to mashed potatoes). Fat intake from energy dense snacks (crisps, chips and chocolate) was 16.7%. Not a single 'healthy' fat made it onto the top ten list, with avocados ranking 15th on the total group list, making up only 2.1% of total fat, and nuts (excluding peanuts) contributing only 0.1% to total fat intake. Prominent differences in ranking between adolescent and adult participants include chips, polony and chocolate that are ranked higher for adolescents, and cheese and eggs that ranked higher for adults.

TABLE 6.6 *Top 10 food items* ***contributing to total fat intake*** *for the total study sample (n=152) and for adolescent and adult participants*

		No. of eaters	% eaters	Total Fat	Mean total/ eater/ day (g)	Portion/ capita	% of Total Fat	Adolescent rank (% TF)	Adult rank (% TF)
1	Crisps	115	75.7	4 444.7	12.9	9.7	10.8	1 (12.7)	1 (10.4)
2	Margarine	138	90.8	3 742.8	9.0	8.2	9.1	3 (6.1)	2 (9.9)
3	Potato	142	93.4	3 330.4	7.8	7.3	8.1	2 (7.8)	3 (8.2)
4	Chicken	142	93.4	2 950.8	6.9	6.5	7.2	4 (5.5)	4 (7.6)
5	Milk	137	90.1	1 990.1	4.8	4.4	4.9	6 (5.3)	6 (4.7)
6	Sausage	64	42.1	1 958.3	10.2	4.3	4.8	5 (4.1)	5 (4.9)
7	Beef	94	61.8	1 892.2	6.7	4.1	4.6	7 (4.8)	7 (4.6)
8	Chips	50	32.9	1 594.7	10.6	3.5	3.9	5 (5.4)	9 (3.5)
9	Cheese	77	50.7	1 594.7	6.9	3.5	3.9	12 (3.9)	8 (3.9)
10	Polony	61	40.1	1 295.6	7.1	2.8	3.2	9 (4.2)	11 (2.9)
11	Eggs	75	49.3	1 255.5	5.6	2.8	3.1	13 (3.6)	10 (2.9)
16	Chocolate	44	28.9	807.9	6.1	2.2	2.0	8 (4.5)	23 (1.4)

Abbreviations: TF = Total fat

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Potato" includes all boiled, mashed, oven roasted and sautéed potato

"Chips" include all deep fried potatoes, while "Crisps" refer to store-bought packet chips e.g. Simba, NikNaks, Flings etc.

The top ten foods contributing to total carbohydrate intakes are mostly refined, including white bread, sugar, carbonated drinks and white rice that together make up 41.2% of the total carbohydrate intake. Energy dense items (sugar, carbonated beverages, crisps and chips) contribute a combined 27.7% to total carbohydrate intake. In the total sample, whole-grain or high fibre foods were ranked 11th (high fibre cereal), 13th (apples), 22nd (oats), 25th (beans), and 32nd (lentils) (not reported in table). Combined these contribute 12.2% to total carbohydrate intake. Prominent differences in ranking between adolescent and adult participants include pasta and cereal that is ranked higher for adolescent participants, and brown bread and milk that are ranked higher for adult participants.

TABLE 6.7 *Top 10 food items* ***contributing to total carbohydrate intake*** *for the total study sample (n=152) and for adolescent and adult participants*

		No. of eaters	% eaters	Total CHO	Mean total/ eater/ day (g)	Portion/ capita	% of Total CHO	Adolescent rank (% TC)	Adult rank (% TC)
1	White bread	137	90.1	18 928.2	46.1	41.5	13.7	2 (13.9)	1 (13.6)
2	Sugar	149	98.0	17 912.2	40.1	39.3	12.9	1 (14.0)	2 (12.7)
3	Carbonated drink	115	75.7	10 273.5	29.8	22.5	7.4	3 (8.8)	4 (7.1)
4	White rice	135	88.8	9 949.8	24.6	21.8	7.2	4 (6.7)	3 (7.3)
5	Potato	142	93.4	8 515.4	20.0	18.7	6.2	5 (5.9)	5 (6.2)
6	Crisps	115	75.7	6 567.1	19.0	14.4	4.7	7 (5.2)	7 (4.6)
7	Brown bread	69	45.4	6 235.0	30.1	13.7	4.5	9 (2.7)	6 (5.0)
8	Pasta	70	46.1	5 079.6	24.2	11.1	3.7	6 (5.3)	8 (3.2)
9	Chips	50	32.9	3 782.0	25.2	8.3	2.7	8 (3.5)	9 (2.5)
10	Milk	137	90.1	3 010.0	7.3	6.6	2.2	13 (2.1)	10 (2.2)
16	Cereal	37	24.3	2 101.3	18.9	6.1	1.5	10 (2.7)	18 (1.2)

Abbreviations: CHO = Carbohydrates TC = Total carbohydrates

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Sugar" includes all sugar added to tea, coffee, cereals and porridges

"Potato" includes all boiled, mashed, oven roasted and sautéed potato

"Chips" include all deep fried potatoes, while "Crisps" refer to store-bought packet chips e.g. Simba, NikNaks, Flings etc.

Table sugar (added to tea, coffee, porridges and cereals) contributes almost half to total added sugar intake and carbonated beverages a quarter (Table 6.8). A variety of other confectionary items and beverages make up the rest of the sugar intake. Prominent differences in ranking between adolescent and adult participants include koeksisters that are ranked higher in adolescents and dairy-fruit juice mixes, jam and sugar added to pumpkin that are ranked higher in adults.

TABLE 6.8 *Top 10 food items* ***contributing to added sugar intake*** *for the total study sample (n=152) and for adolescent and adult participants*

		No. of eaters	% eaters	Total Sugar	Mean total/ eater/ day (g)	Portion/ capita	% of Total Sugar	Adolescent rank (% TS)	Adult rank (% TS)
1	Sugar	149	98.0	17 912.2	40.1	39.3	44.2	1 (45.3)	1 (43.8)
2	Carbonated drink	115	75.7	10 273.5	29.8	22.5	25.3	2 (28.6)	2 (24.4)
3	Sweets	58	38.2	2 180.6	12.5	4.8	5.4	4 (4.6)	3 (5.6)
4	Chocolate	44	28.9	1 515.7	11.5	3.3	3.7	3 (6.5)	5 (2.9)
5	Cake	35	23.0	1 451.8	13.8	3.2	3.6	5 (3.4)	4 (3.6)
6	Dairy-fruit juice mix	27	17.8	741.9	9.2	1.6	1.8	9 (0.8)	6 (2.1)
7	Cookies	37	24.3	696.9	6.3	1.5	1.7	7 (1.9)	8 (1.7)
8	Jam	25	16.4	695.5	9.3	1.5	1.7	14 (0.5)	7 (2.1)
9	Koeksister	14	9.2	644.8	15.4	1.4	1.6	6 (2.2)	11 (1.4)
10	Ice cream	21	13.8	594.7	9.4	1.3	1.5	8 (0.8)	9 (1.6)
11	Cold drink	21	13.8	505.2	8.0	1.1	1.2	10 (0.8)	12 (1.4)
12	Pumpkin	30	19.7	491.0	5.5	1.1	1.2	20 (0.2)	10 (1.5)

Abbreviations: TS = Total sugar

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Sugar" includes all sugar added to tea, coffee, cereals and porridges

White bread was the most prominent contributor to fiber intake, with potatoes and brown bread also making a reasonable contribution (Table 6.8). A variety of other foods contribute to the total fibre intake of the participants. Only two fruits were ranked in the top ten contributing foods, and no vegetables were consumed in amounts sufficient to contribute to fibre intake. Differences that stand out between adolescent and adult participants are chips and pasta that are ranked higher for adolescents, and brown bread, high fibre cereal and lentils that are ranked higher for adults.

TABLE 6.9 *Top 10 food items* ***contributing to total fibre*** *intake for the total study sample (n=152) and for adolescent and adult participants*

		No. of eaters	% eaters	Total Fibre	Mean total/ eater/ day (g)	Portion/ capita	% of Total Fibre	Adolescent rank (% TFib)	Adult rank (% TFib)
1	White bread	137	90.1	1 319.6	3.2	2.9	14.0	1 (15.7)	1 (13.6)
2	Potato	142	93.4	823.2	1.9	1.8	8.7	2 (9.1)	3 (8.6)
3	Brown bread	69	45.4	797.5	3.9	1.7	8.4	4 (5.6)	2 (9.1)
4	High fibre cereal	51	33.6	544.3	3.6	1.2	5.8	7 (5.2)	4 (5.9)
5	Beans	46	30.3	479.7	3.5	1.1	5.1	6 (5.2)	5 (5.0)
6	Apples	65	42.8	430.5	2.2	0.9	4.6	5 (5.6)	6 (4.3)
7	Chips	50	32.9	377.1	2.5	0.8	4.0	3 (5.7)	9 (3.6)
8	Oranges	44	28.9	375.4	2.8	0.8	4.0	9 (4.7)	7 (3.8)
9	Lentils	32	21.1	347.6	3.6	0.8	3.7	11 (3.5)	8 (3.7)
10	Pasta	70	46.1	307.0	1.5	0.7	3.2	8 (5.0)	11 (2.8)
11	Crisps	115	75.7	287.8	0.8	0.6	3.0	10 (3.6)	10 (2.9)

Abbreviations: TFib = Total fibre

Portion per capita = total grams consumed divided by the total sample n=152, and divided by three days

"Potato" includes all boiled, mashed, oven roasted and sautéed potato

"Chips" include all deep fried potatoes, while "Crisps" refer to store-bought packet chips e.g. Simba, NikNaks, Flings etc.

Fifty-one (33.6%) participants, 13 (43.3%) adolescents and 38 (31.1%) adults, reported consuming alcohol 30 days prior to conception, with a mean intake of 0.2 drinks per day (6 days per month), ranging from 0.002 to 0.9 drinks per day (0.06 – 27 days per month). There was no significant difference between adolescent and adult participants for alcohol intake.

Dietary diversity scores (DDS)

For the first DDS calculation, the mean ±SD DDS of the study sample was 5.0 ±1.2, and was similar between adolescents (5.1 ± 0.8) and adults (5.0 ±0.9) (Independent sample t-test p=0.9465). In the total group 12 participants (7.9%) had a DDS less than 4, and 140 participants (92.1%) had a DDS ≥4. There were no differences between adolescents [DDS<4: n=1 (3.3%); DDS≥4: n=29 (96.7%)] and adults [DDS<4: n=11 (9.0%); DDS≥4: n=111 (91.0%)] (Chi-square test p=0.2576).

The DDS was also calculated using vitamin A rich fruits and vegetables as a category separate to other fruit and other vegetables, and showed that other fruits and vegetables were consumed much more frequently than vitamin A-rich fruits and vegetables (Table 6.10). With these categories the mean DDS was 5.1 ±1.2, and remained the same between adolescent and adult participants (5.2 ±0.8, and 5.1 ±0.9 respectively) (Independent sample t-test p=0.8011). In the total group 13 participants (8.6%) had a DDS less than 4. There were no differences between adolescents [DDS<4: n=1 (3.3%); DDS≥4: n=29 (96.7%)] and adults [DDS<4: n=11 (9.0%); DDS≥4: n=111 (91.0%)] (Chi-square test p=0.3042). The food groups that contributed mostly to the latter DDS included milk and milk products, fats and oils, meat and meat products and cereals and cereal products (Table 6.10).

TABLE 6.10 *Number of eaters per day of dietary diversity score food groups for the total study sample (n=152)*

FOOD GROUPS PER DDS	No. of eaters Day 1	%	No. of eaters Day 2	%	No. of eaters Day 3	%	No. of eaters Average	%
Milk and milk products	113	74.3%	129	84.9%	116	76.3%	119.3	78.5%
Nuts and seeds	36	23.7%	32	21.1%	32	21.1%	33.3	21.9%
Fats and oils	122	80.3%	118	77.6%	113	74.3%	117.7	77.4%
Meat and meat products	140	92.1%	143	94.1%	147	96.7%	143.3	94.3%
Eggs	34	22.4%	35	23.0%	28	18.4%	32.3	21.3%
Cereals and cereal products	152	100.0%	152	100.0%	152	100.0%	152.0	100.0%
Vitamin A rich fruit and vegetables	33	21.7%	38	25.0%	42	27.6%	37.7	24.8%
Other fruit	64	42.1%	89	58.6%	75	49.3%	76.0	50.0%
Other vegetables	77	50.7%	78	51.3%	88	57.9%	81.0	53.3%

Abbreviations: DDS = Dietary diversity score No. = number

6.3 Discussion

One of the most notable characteristics of the dietary intake of this sample of pregnant women is the high intake of refined carbohydrates such as added sugar, refined starches, crisps, and carbonated drinks. Sugar was the number one most commonly consumed food item, the second highest contributor to total energy and carbohydrate intake, and the top contributor to added sugar in this study sample. By far the highest contributor is sugar added

to tea, coffee and/or cereals, with a mean total per eater per day of 40.1g, equating to approximately eight teaspoons of sugar. This is separate to sugar added to foods during preparation, most notably pumpkin, which, for just under a fifth of the population, added an extra teaspoon of sugar per day. The other South African studies that were conducted on pregnant women reported frequent and high sugar intakes (Kesa & Oldewage-Theron, 2005; Jaffer et al., 2008), although not as high or frequent as the current study. Kesa & Oldewage-Theron (2005) reported sugar to be the tenth most commonly consumed food in their study sample, while Jaffer et al. (2008) found sugar to be consumed between eight and ten times per week, by an average of 87.5% of their study sample. However, in the general population, even higher sugar intakes of 51 g/day (15% of TE) and 38 g/day (11% of TE) have been reported for young and older urban black adults respectively from Cape Town that participated in the Cardiovascular Risk Study in Black South Africans (CRIBSA) (Steyn & Temple, 2012). Furthermore, a five-year follow-up cohort of 2 010 urban and rural black adults from the North West province in South Africa that participated in the PURE study found that the intake of added sugar had approximately doubled from 2005 to 2010. The women in their follow-up sample consumed an average of 72.1g of sugar per day which contributed to 10.0% of their total energy intake (Vorster et al., 2014).

Other highly refined carbohydrate foods in the top 20 most commonly consumed foods included white bread, white rice, carbonated drinks and crisps. Carbonated drinks were the eighth most commonly consumed food item, the seventh highest contributor to total energy intake, and the third highest contributor to total carbohydrate intake. Mostert et al. (2005) also reported a high intake of such sugar sweetened beverages. The high intake of these energy dense foods may cause weight gain and irregularities in blood glucose levels, which may contribute to excessive foetal size (Scholl et al., 2001). Furthermore, since energy dense foods like carbonated drinks usually have a low micronutrient content, the risk for inadequate micronutrient intake increases. It is thus concerning that adolescents had an even higher intake of crisps, carbonated drinks, chocolate, koeksisters and ice-cream than adults.

As with the current study, starches also made up the bulk of food intake in three other South African studies done on pregnant women (Mostert et al., 2005; Jaffer et al., 2008; Tshitaudzi, 2003). The high intake of starches may contribute to achieving adequate micronutrient intake

due to the fact that foods such as maize meal and white and brown bread flours are fortified with vitamin A, thiamin, riboflavin, niacin, pyridoxine, folic acid, iron and zinc (UNICEF, 2007). It must be noted that while several studies conducted on pregnant women in South Africa (Bopape et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Tshitaudzi, 2003) reported maize meal to be one of the most commonly consumed foods, only a fifth of the current study sample consumed maize meal, which contributed 0.7% to total energy intake, and was the 18th highest contributor to carbohydrates.

It is also concerning that most of the starches consumed in this study sample were refined. White bread was the top contributor to total energy intake with an average consumption of four to five slices per woman per day. In fact, the intake of whole-grain or high fibre items was low, with only three such items (potatoes, cereals and apples) appearing on the top 20 list of foods contributing to total carbohydrate intake. Just under a quarter of the total fibre intake came from foods not high in fibre, suggesting a very large intake of those foods, with white bread being the highest contributor. Foods that are high in fibre (high-fibre cereal, beans, lentils and oats) were consumed by fewer participants, and in much smaller amounts than more refined foods. The higher fibre foods were consumed even less frequently by adolescents. The significant price difference between whole-wheat and white bread (white bread being ±17% cheaper) (Temple et al., 2011) would make it difficult for a low socio-economic population to afford the healthier option. While the beneficial effect of whole-grain, high fibre foods has been well researched (Mann, 2007; Key & Spencer, 2007; WHO, 2003), the recent general trend in South Africa has been to a diet of more refined carbohydrates, and more added sugar (Vorster et al., 2014; Steyn, 2006). A poor intake of fibre-rich foods was also evident from studies that included pregnant women in South Africa with Jaffer et al. (2008) reporting an average of just over one fruit and vegetable serving per day, and legumes being consumed twice per week. Furthermore, none of the top ten commonly consumed food items in the study by Kesa and Oldewage-Theron (2005) were high in fibre, while Mostert et al. (2005) reported “small amounts” of vegetables consumed.

The increase in micronutrient requirements during pregnancy is greater than the increase in energy requirements, necessitating a higher nutrient-dense diet (Erick, 2012, Williamson, 2006), and hence the need for plenty of fruits and vegetables. The general intake of fruits and

vegetables in this study sample was low. Tomatoes and apples were the only fruits and vegetables on the top 20 most commonly consumed food list, appearing only at the 13th and 18th positions respectively. Two participants reported not consuming any fruits or vegetables for the three days assessed. The mean intake of fruit and vegetables was only 126.4g per day which equates to an average of three portions per day. This is below the recommended 400g or five portions of 80g per day (Naude, 2013). Fruits and vegetables contributed nominally to total energy and carbohydrate intakes. Apples ranked the highest at 23rd in total energy and 13th in total carbohydrates, while pumpkin ranked 47th in total energy and 36th in total carbohydrates. Tomatoes ranked 56th in total energy and 41st in carbohydrates. Fruit and vegetable intake was similarly low amongst all assessed studies on pregnant South African women who reported on fruit and vegetable intake (Bopape et al., 2008; Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005). Of these studies, Jaffer et al. (2008) reported an average weekly intake of 7.8 portions of fruit and vegetables, while Bopape et al. (2008) and Kesa & Oldewage-Theron (2005) mention a poor intake, and no fruits or vegetables making the top ten most commonly consumed foods. Schneider et al. (2007) reported fruit and vegetable intake in South Africa with daily estimated intakes for women of 226g/day.

Apples, oranges, bananas and peaches were consumed by at least a fifth of the study sample, while the remaining sixteen different fruits reported were consumed by only nineteen or fewer women. Eating a variety of fruits as part of a healthful diet may reduce the risk of some chronic diseases (Naude, 2013b). It is recommended to consume one serving daily of a yellow-orange fruit (e.g. mango, paw-paw) (Naude, 2013), which, in the current study, ranked 19th and 21st in the total study sample. Fruits contain several essential nutrients including vitamin A, vitamin C, potassium, phytochemicals and fibre (Schneider et al, 2007). Apples, ranked as the most commonly consumed fruit, are good sources of fibre, while bananas, ranked fourth, are a good source of potassium. Oranges, ranked third overall are a reasonable amount of vitamin C. However, increased fruit intake and a greater variety of fruit are required to obtain the full health benefits.

It should be noted that fruit juice is included in the total gram amount calculated for fruit and vegetable intake and contributed 9 932.5g (5.4%) to this amount. Fruit juice was ranked as the fourth most commonly consumed ‘fruit’ amongst adults, whereas this was first amongst

the adolescents. Very few of the women reported purchasing and consuming freshly squeezed fruit juice which would offer more nutrients and less sugar than the more common commercial fruit juice concentrates and artificial juices. Fruit juice was also ranked high on the commonly consumed foods lists in the studies by Mostert et al. (2005) and Kesa & Oldewage-Theron (2005).

The general vegetable consumption was more varied, with each of the top five most commonly consumed vegetables being consumed by just over a quarter of the study sample. As with fruits, a variety of vegetables is recommended. It is suggested that consuming a single daily serving each of cruciferous vegetables (e.g. broccoli and cabbage), dark-green leafy vegetables (e.g. spinach) and yellow-orange vegetables (e.g. carrots, butternut) could have protective benefits against several non-communicable diseases (Naude, 2013; 2013b). The study sample did not reach intakes that will meet these recommendations. From the most commonly consumed vegetables, cabbage (ranked 7th) and broccoli (13th) were consumed by only 20.4% of the participants, spinach (22nd) by 0.7%, and pumpkin (4th), carrots (5th), and butternut (14th) was consumed by 42.1%. While the consumption of yellow-orange vegetables is still inadequate, they are consumed significantly more regularly than cruciferous vegetables. The intake of spinach was only reported by one participant on one of the three recall days.

The most commonly consumed protein source in this study sample was chicken. Chicken was also the second most commonly consumed food in the total sample, while other animal protein sources included cheese (8th), beef (12th), sausage (14th), eggs (15th) and polony (16th). Chicken also contributed largely to total energy intake, at least partly because the majority of chicken was consumed with the skin on, was roasted or fried with fat, or was deep fried. Although cheese is a relatively expensive item, it ranked higher on the most commonly consumed foods list than most other cheaper plant-based protein sources which were ranked very low. While the overall intake of saturated fats should be limited, a recent meta analysis found that hard cheeses may lower LDL-cholesterol (de Goede et al., 2015), and that the specific whey proteins in cheese may counter the effect of the high fat and added sodium in cheese (Tavara & Malcata, 2012). A meta analysis done on red and processed meat indicate a dose-response, with higher intakes increasing the risk of total, cardiovascular and cancer

mortality (Wang et al., 2015). Eggs are a source of complete protein, and have been shown to contribute towards satiety and weight management (Murphy & Allen, 2003). Despite the high cholesterol content of eggs, epidemiological studies have unfailingly shown a non-significant relationship between the intake of eggs and cardiovascular disease risk (Schonfeldt et al., 2013). The current intake of eggs in the study sample is good as the consumption of eggs are thus recommended, but preparation methods need to be considered. Looking at other South African studies, several found a low intake of protein-rich foods. Kesa and Oldewage-Theron (2005) did not have any high protein foods in their top ten most commonly consumed foods, while Mostert et al. (2005) reported only small amounts of chicken intake, and Tshitaudzi (2003) found a lower protein intake in pregnant adolescents than non-pregnant adolescents. Tshitaudzi (2003) also found a significant difference in protein intake from plant and animal sources, with a greater contribution from plant sources (38.1g vs. 22.6g).

The low intakes of fish, with only 38.1% of the participants reporting any fish intake in the three 24-hour recalls, is concerning. While several participants did consume pilchards, sardines, and even salmon (34.9%) which contain high amounts of readily bio-available omega-3 PUFAs, half of the fish consumed was battered and fried, and most fish consumed were lowfat fish such as hake and tuna that contain low amounts of the omega-3 PUFAs. The national food consumption survey (NFCS) in South Africa found intakes of PUFA to be low, especially in those who also had a low total fat intake, (Smuts & Wolmarans, 2013) and found fish intake to be much lower than the recommended two to three portions of 80-90g twice per week (Schonfeldt et al., 2013). Although the study was done on children between one and nine years of age, Smuts and Wolmarans (2013) suggest that although it does not necessarily reflect composition of the intake in adults, it may still provide some indication of the household diet. During pregnancy the intake of omega-3 PUFAs such as EPA and DHA become vital for brain and nervous system development (Koletzko et al. 2014; Gould et al., 2013; Campoy et al., 2012; Kris-Etherton & Innis, 2007).

Plant protein sources are generally cheaper and can contribute significantly to total protein intake if consumed in combinations that incorporate all essential amino acids and make up complete protein intake (FAO/ WHO 2002). White and brown bread were ranked as the third and fifth largest contributors to protein. Since bread does not contain a significant amount of protein, this indicates a very high intake of bread relative to high quality high protein sources. Other plant protein-rich sources did not rank very high on the most commonly consumed

foods or the total energy lists, and were consumed in smaller amounts and by fewer people than less plant protein-rich sources. Beans, being a cheap source of protein, was consumed by close to a third of the participants, ranked 21st on the contribution to total protein list (1.0% of total protein), and ranked as the 27th most commonly consumed food. Peanut butter, another plant protein source, ranked 20th in total protein contribution, and 40th on the most commonly consumed list. Peanut butter and nuts were not mentioned by any other studies on pregnant South African women, while intake of legumes was reported to be low by Jaffer et al. (2008). In the general South African diet of both adults and children, legume and nut intake was similarly low (Labadarios et al., 2011), being consumed by only 19.7% of the sample from the NFCS (Steyn & Ochse, 2013).

It is evident that none of the top ten foods contributing to fat intake are good sources of omega-3 PUFAs or MUFAs. Instead, crisps, margarine, chips and other fried potatoes, sausage, beef, cheese, polony, and pies make up just over half of the total energy intake from fat, and make up seven of the top ten contributors to total fat. This indicates a very high intake of saturated fatty acids. Although these foods are also high in essential omega-6 PUFAs, excessive intakes of omega-6 PUFAs have been shown to have pro-inflammatory effects (Keeren et al., 2015). These mostly processed foods are generally less expensive than sources like avocados, nuts, olives and fatty fish that would offer greater amounts of MUFAs and omega-3 PUFAs (Brooks et al., 2010). While chicken meat does contribute some unsaturated fats to the diet, several participants reported eating fried chicken (5.4%) and chicken with the skin (67.2%), which contributes more to trans fatty acids and saturated fatty acids.

When comparing snack foods between adolescent and adult participants, poor food choices become evident specifically in adolescents. Chocolate is ranked as the 8th highest contributor to fat intake in adolescents, but only 23rd in adults. The same is true with crisps, which was classified as the third most commonly consumed food for adolescents, but only tenth in adults Cake, koeksisters and ice cream were also more commonly consumed by adolescents than adults. However, sweets, cookies, jam, and sugar added to pumpkin dishes were more commonly consumed by adults than adolescents.

As a general indicator of dietary diversity, the DDS indicated an acceptably diverse intake of food groups within this study sample with fewer than 9% of the sample having an inadequate DDS. Similar findings were reported by Labadarios et al. (2011) that found a 15.7% inadequate DDS (<4) in adolescent and adult participants in the Western Cape province. However, Drimie et al. (2013) reported 37% of their study sample of adults from Johannesburg to have a DDS <4. While foods in the current sample are consumed from a variety of food groups, fruit and vegetable intake is very low, and further variety and diversity within other food groups may be lacking.

Bearing in mind the limitations of self-reported dietary intake, especially the problem of under- and over-reporting, it can be concluded that the dietary intake of pregnant women in this sample is characterised by a high intake of refined carbohydrates, especially added sugar, white bread and carbonated drinks and a low intake of whole grains and foods high in fibre. Intakes of fruits and vegetables are inadequate, specifically the intake of yellow fruit, cruciferous and green leafy vegetables. The food sources that contributed mostly to fat intake are typically high in saturated fatty acids (e.g. high fat and processed animal products) and omega-6 PUFAs (e.g. margarine, sunflower oil for cooking). While these fatty acids were also provided by the regular consumption of energy-dense snack foods, especially crisps and chocolate, limited amounts of food sources high in MUFA and omega-3 fatty acids were consumed. Protein was consumed largely from animal sources, with low intakes of legumes. Fish intake was insufficient and characterized by the consumption of low fat varieties such as hake while the intake of fish high in omega-3 PUFAs was specifically low. The majority of meat and fish was fried or deep-fried. In general, food choices made by adolescents were higher in energy, refined carbohydrates, sugar and saturated fats, and lower in vegetables and high-fibre options.

While the dietary diversity scores indicate adequate variety of the dietary intake from different food groups of this study sample, poor food choices within the food groups are apparent (e.g. frying food, high intake of refined foods, sugar added to vegetables) and may limit the amount of micronutrients consumed which may adversely influence pregnancy and pregnancy outcome. It is clear that antenatal care for women in this area should include dietary education with a strong focus on the intake of healthy and affordable food and beverage choices and healthy preparation methods.

CHAPTER SEVEN

OVERVIEW, CONCLUSIONS AND RECOMMENDATIONS

7.1 Overview

It is undisputed that adequate nutritional intake during pregnancy is important for optimal health outcomes for mother and infant (NHMRC, 2013; Wu et al., 2004) since several vitamin and mineral requirements increase substantially during pregnancy (Erick, 2012; Trumbo et al., 2002). This makes a nutrient-dense rather than energy-dense diet essential (Cox & Phelan, 2008), and since no one food or food group can provide all the nutrients required, variety in dietary intake is key to a healthy lifestyle and pregnancy outcome (Steyn & Ochse, 2013).

Despite the importance of adequate nutrition in pregnancy, very few studies have investigated this in South Africa. The studies that have been done (Bopape et al., 2008; Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Tshitaudzi, 2003; Klinger, 2004; Mamabolo et al., 2004) all found general high carbohydrate intakes and poor intakes of fibre, fruits and vegetables, iron, folate, vitamin C and zinc. The majority of these studies included both adolescent and adult participants (Bopape et al., 2008; Jaffer et al., 2008; Kesa & Oldewage-Theron, 2005; Mostert et al., 2005; Mamabolo et al., 2004), except for Tshitaudzi (2003) who assessed only adolescents and Klinger (2004) who assessed only adults. Four studies were done in the Limpopo province (Bopape et al., 2008; Mostert et al., 2005; Tshitaudzi, 2003; Mamabolo et al., 2004), one in Gauteng (Kesa & Oldewage-Theron, 2005), and two in the Western Cape (Jaffer et al., 2008; Klinger, 2004).

The aims of this study was to investigate the adequacy, diversity and variety of dietary intake and associated factors (anthropometry, socio-demographic status) in participants attending the Bishop Lavis MOU in Cape Town, and recruited into the Safe Passage Study (PASS). We assessed the socio-economic and anthropometric profiles of the study sample and investigated associations in the total group and between adolescent and adult participants. This was done to aid in implementing successful intervention strategies by highlighting possible causes of poor pregnancy outcomes. The dietary intake of participants was assessed using the average of three 24-hour recalls. This method is inexpensive, has relatively low respondent burden (Lee & Nieman, 2010), is better suited to study samples with lower literacy levels, and is less likely to influence actual dietary intake (Cade et al., 2002). The limitations of the 24-hour recall method need to be taken into account, as this method relies on memory and conceptualisation of portion sizes (Cade et al., 2002) and is prone to under-reporting of unhealthy foods (Lee & Nieman, 2010). Furthermore, the possibile influence of

non-responder bias should be borne in mind when interpreting the results of this study as no data of those that did not consent to participate were collected.

The data from the three 24-hour recalls was interpreted using DRIs to provide insights into adequacy of intakes of energy, macro- and micro-nutrients. Further insights into the adequacy of intake were gained from computing the NARs and MAR as well as comparing data with the SAFBDG by converting the 24-hour intake data to frequency of intake from specified food groups. Dietary variety was investigated by identifying the 20 most frequently consumed food items and also the most frequently consumed fruits and vegetables. The top ten foods contributing to toal energy, protein, carbohydrates and fats were identified to provide insights into the main sources of the macronutrients. Furthermore, two versions of the DDS were calculated to further assess variety of intake.

The results of the adequacy, quality, variety and diversity assessments of the three repeated 24-hour recalls need to be interpreted bearing in mind the limitations of this dietary assessment method. A single interviewer was used to conduct all interviews, coding of the recalls was done by one experienced dietitian, and various data cleaning steps were taken to increase the validity thereof. Inspection of the validity of the data using the Goldberg cut-offs showed that 55.0% were accurate reporters, while 18.1% under-reported and 26.9% over-reported. Furthermore, those with a BMI >35kg/m^2 tended to be more likely to under-report. These results regarding the potential validity of the 24-hour recall data indicate that cautious interpretations and conclusions should be made. This, however, does not detract from the contribution that the results can make to insights in current dietary intake as there is very limited research on the dietary intake of pregnant women in South Africa.

Findings were interpreted within the context of the work by Bopape et al. (2008), Jaffer et al. (2008), Kesa & Oldewage-Theron (2005), Mostert et al. (2005), Tshitaudzi (2003) and Klinger (2004). It needs to be noted that comparisons between studies were challenging due to a lack of standardisation in research aims, methods, and reference indicators. Only two of these studies were done in the Western Cape.

7.2 Integrated results and conclusions

Weight status

Weight status is one of the determinants of a healthy pregnancy outcome (Ehrenthal et al., 2014). In line with the growing obesity epidemic in South Africa, one in three of the study participants were classified as overweight or obese prior to pregnancy. Simultaneously, however, one in eight women were classified as underweight suggesting the double burden of over- and under-weight in this sample of pregnant women which is in line with results from South African populations (Toriola et al., 2012). Adolescents seemed to be less well-nourished than adults, who may, in turn, be more likely to be overweight or obese. The total amount and the rate of weight gain during pregnancy also influences pregnancy outcome (Ehrenthal et al., 2014; Calvo & Lopez, 2012). The majority of the study sample (84.4%) gained either insufficient or too much weight according to recommended guidelines, further increasing their risk of adverse pregnancy outcomes, as well as exacerbating their personal poor health and weight status (Davis et al., 2014; Han et al., 2010; Mamun et al., 2014; Crozier et al., 2010; IOM, 2009). While the risk of several adverse pregnancy outcomes is increased, the impact of these in-utero conditions may likely perpetuate the negative effects in the future of these offspring (Langley-Evans, 2014; Barker, 2002).

There are limitations to the anthropometric data that need to be considered. The pre-pregnancy weight, pre-pregnancy BMI and weight change variables were self-reported and need to be interpreted with caution. Self-reported weight relies on the individual knowing and remembering their weight of several months prior to the interview and reporting it accurately. More than half of the study participants were unable to provide an estimation of their weight prior to conception, reducing the study sample size for these variables.

Major concerns about dietary intake

The mean DDS of the study sample suggests an acceptably diverse intake of food groups. Although fewer of the study sample scored a low DDS when compared to other studies done in South Africa (Drimie et al., 2013; Labadarios et al. 2011), the food choices were generally not varied and healthy enough, as is reflected by the results of the most commonly consumed foods. These included sugar which was the most commonly consumed food item, followed by margarine in fourth, white bread in sixth, carbonated drinks in eighth and crisps in tenth place. Furthermore, the intake of several food groups did not meet the SAFBDG recommendations. Thus, although a variety of foods may have been consumed, healthy grain

and fat choices as well as portion sizes and frequency of consumption of fruits, vegetables and legumes may have been generally insufficient.

The excessive intake of sugar and other refined carbohydrate foods such as white bread and white rice is highly concerning. The daily mean per capita intake of added sugar of the total sample was 89.0g, and the daily per capita intake of table sugar was 39.3g. Thus approximately 10% of total energy intake comes from sugar added to tea, coffee and cereals by the person themselves, with carbonated drinks adding most of the rest. Added sugar contributed 20.5% to total energy intake, making up close to half of the total carbohydrate contribution. With the high proportion of carbohydrates coming from refined sources, the intake of whole grain foods is inadequate. These factors greatly increase the mother's risk of developing diabetes, either during pregnancy or afterwards (Malik et al., 2010). The adverse effects of sugar on glucose tolerance and the subsequent effect on weight status have been emphasised in several studies (Salas-Salvado et al., 2011; Malik et al., 2010; Mann, 2007). Poor blood glucose control has also been shown to have adverse effects on foetal development and the offspring's future health (Tanaka et al., 2014; Calvo & Lopez, 2012; Landon et al., 2009).

The total fat intake of the study sample contributed 46.0% to total energy intake, which is one and a half times the upper limit of the recommendation. A large portion of this was in the form of SFA (12.3% TE). The FBDGs recommends that fats should be consumed sparingly, but intake in the study sample equalled 9.7 servings per day. High intakes of fat are further supported by the fact that margarine was ranked as the fourth most commonly consumed food item with per capita portion sizes of 10.1g. Several of the protein choices made by the study sample were high in fat, particularly in saturated fat, with cheese, beef, sausages and eggs all ranking in the top 20 most commonly consumed foods. Preparation methods of potatoes and chicken also added substantially to the high fat intake. The high fat and saturated fat content of the diets of the study sample may increase their risk for obesity and cardiovascular disease (Smuts & Wolmarans, 2013).

The inadequate intake of folate, iron, zinc, calcium and magnesium in the total study sample is concerning because of the roles that each of these nutrients play in optimal pregnancy outcomes. The requirements of all these nutrients increase during pregnancy for adults and

adolescents, except that of calcium which increases only for pregnant adolescents (IOM, 2010; Cox & Phelan, 2008). The most likely explanation for these inadequate intakes is the poor variety, specifically in terms of low intakes of legumes, nuts, seeds, fish, milk, fruit and vegetables, combined with poor food choices, especially in terms of the consumption of refined carbohydrates rather than whole-grains as is reported in this study sample. The lack of dark-green leafy vegetable intake (being consumed by only one participant on one day) may be a good intervention point as this is a good source of several of the inadequately consumed micronutrients. Inadequacies of these nutrients increase the risk of prematurity (folate, iron, zinc, magnesium), LBW (folate, iron, magnesium), poor brain and cognitive development in the infant (iron, zinc), and pre-eclampsia and hypertension in the mother (folate, zinc, calcium, magnesium).

Contrary to dietary recommendations during pregnancy, the common food choices of the study sample were high in energy and low in nutrient density. Food and snack choices were particularly poor among adolescents. For example, chocolate was ranked as the 8th highest contributor to total fat in adolescents while ranking as only the 23rd highest fat contributor for adults. What is of particular concern is that adolescents, whose food choices were often poorer and whose fruit and vegetable intake was lower, require more nutrient dense foods to meet their further elevated nutrient requirements (Wendt et al., 2012; Brown, 2008). This puts adolescents at greater risk of adverse pregnancy outcomes.

Other concerns

The mean total energy intake (10 168kJ) of the study sample was below the EER, with 79.6% of the participants falling below the EER cut-point. When interpreting these results, it needs to be considered that only 55% seemed to have reported their energy intakes accurately. In addition, the majority of the sample who provided pre-pregnancy weights were either normal weight (57.0%), overweight (12.7%) or obese (17.8%). Poor energy intake, if present, is concerning especially during pregnancy where weight loss can be detrimental to the health of the foetus (IOM, 2009). Although there were no statistically significant differences in energy intake between adolescent and adult participants, poor energy intake is more concerning in pregnant adolescents because of their further increased energy and nutrient requirements and their generally micronutrient-poor diets (Northstone et al., 2013). However, the lower energy intake in adolescents may have been as a result of under-reporting which is more prevalent in adolescents than adults (Forrestal, 2011).

The mean protein intake and grams per kilogram per day was above the recommended values. Despite this, 42.8% of the study sample did not consume enough protein, which is understandable when considering the high cost of meat and meat products (Brooks et al., 2010) versus the general lower economic status of the study sample. What obscures this result is that the intake of relatively cheaper protein sources was very low with peanut butter and beans ranking as the 20th and 21st highest contributors to total protein. This may suggest a need for dietary education on good quality nutrition choices on a limited budget. Furthermore, the type of protein sources could generally be classified as unhealthy including high fat cuts or processed meats.

Results in the South African context

The findings from other studies done on pregnant South African women are similar to those from this study with excessive intakes of refined carbohydrates, sugar and fat, and low intakes of mean energy, fibre and fruits and vegetables. Folate, iron, zinc, calcium and magnesium intakes were insufficient to meet pregnancy requirements.

Good aspects of the diet

Results show that there was a low risk of deficiency for several nutrients including riboflavin, niacin, vitamin B6, vitamin B12, phosphorous, sodium and copper. This was supported by analysis of the NAR which showed adequate intakes of all B vitamins (except folate), phosphorous, sodium, zinc, copper and manganese. These findings may be explained by the adequate (and excessive) intake of foods from the "starch" and "meat, poultry, fish, and eggs" groups of the SAFBDG, as these foods are generally high in B vitamins, phosphorous, zinc and sodium (Gallagher, 2012). The mandatory fortification of maize meal and white and brown bread flour with folic acid, iron, vitamin A, thiamine, riboflavin, niacin, pyridoxine and zinc, may also be contributing to the adequate intakes of these nutrients. Concurrently, mean intake of protein was also within the DRI, and the NAR showed adequate intake of protein and carbohydrates.

Associated SES factors

With the study sample being classified as generally low in socio-economic status, associations between the dietary variables and income and education are insightful. Low SES

and lower income suggest less availability and less accessibility to adequate resources, including prenatal care and varied, nutritious foods. Whole grain cereals are generally more expensive than their refined counterparts (Temple et al., 2011), fruits and vegetables are commonly more expensive (Brooks et al., 2010; Temple et al., 2009), and foods high in fat are generally more affordable than good quality protein sources (Brooks et al., 2010). While fatty foods are not necessarily inexpensive, their palatability and the satiety that they provide may clarify why they are popular food choices (Weltens et al., 2015; Grabenhorst & Rolls, 2014). The poor socio-economic status of this study sample may therefore be increasing the risk of diabetes and cardiovascular disease, as fibre and micronutrients from fruits and vegetables have important beneficial effects on blood glucose levels and heart health respectively (Naude, 2013; Rizotto et al., 2010; Roman et al., 2014). A diet high in saturated and trans fats (and low in healthier unsaturated fatty acids) may have detrimental effects on both diabetes and cardiovascular health (Mennitti et al., 2015; Smuts & Wolmarans, 2013; Muhlhausler et al., 2010).

It was found that the greater the number of people relying on household income, the lower the intake of energy and protein. Low income was associated with years of education, an association that reflects the situation in South Africa where poor education decreases the chance of being able to achieve a higher income, and a low income challenges the ability to achieve a better education. Furthermore, lower levels of education may suggest less likelihood of knowing about or understanding the principles of a healthy diet and lifestyle. Overall, the effects of the social and economic factors of this study sample cannot be overlooked, and needs to be considered in targeted interventions.

Current interventions in South Africa for pregnant women

It is a concern that despite many national interventions being in place to address the nutrition related health of pregnant women and young children, we found that these women (adolescent and adult participants), may still be at nutrition risk. Within the South African context, the integrated nutritional supplementation/ therapeutic programme (NSP/ NTP) was initiated in 1995. It focused specifically on maternal and child health, and offered food and/ or micronutrient supplementation to women that met certain criteria (Grundlingh et al., 2013). An assessment done by Grundlingh et al. (2013) concluded that the program had been unsuccessful, with only a quarter of pregnant women interviewed being aware of the

program, and five out of six women that qualified for supplementation never being registered for it. The participants of the current study did not receive any supplements from these programmes, but did receive the routine iron and folic acid supplements.

The study by Mamabolo et al. (2004) assessed the Government initiated supplementation program at eight antenatal clinics in Limpopo and found the prevalence of iron and folate deficiencies to still be common in the area despite supplements being issued at the antenatal clinics. Side effects of iron supplements, concurrent high intake of tannins and phytates, and inadequate motivation were mentioned as possible barriers to greater efficacy.

The South African national food fortification programme was also initiated to provide nutrients found to be consumed below recommendations. This programme entails compulsory fortification of maize and white and brown bread flours with folic acid, iron, vitamin A, thiamine, riboflavin, niacin, pyridoxine and zinc, and was implemented in October 2003 to improve the nutritional composition of the average dietary intake of South Africans (UNICEF, 2007). Modjadji et al. (2007) reported that this programme had some positive effects on folate status in non-pregnant women in a small rural area in the Limpopo province, but not on iron or vitamin B12 status.

7.3 Recommendations

Well-targeted nutrition education is necessary and may assist in overcoming the dietary inadequacies of the targeted sample in question. In this study sample, nutrition education should be done with the aim of maintaining a healthy weight status throughout pregnancy in order to avoid the complications that arise due to both inadequate and excessive weight gains. Nutrition interventions for pregnant adolescents should focus on ensuring sufficient nourishment for optimal growth and weight gain, while prevention of excessive weight gain during pregnancy and post-partum weight retention should be the focus for pregnant adults.

The content of the nutrition education could focus on the following:

- Adverse effects of high intakes of processed foods, refined carbohydrates and saturated and trans fatty acids
- Examples of alternative, healthier, and cheaper protein sources and how to incorporate these into daily intake
- The use of lower-fat cooking methods such as boiling or steaming potatoes and vegetables, or dry-roasting chicken or fish
- Encouraging individuals to explore different ways of preparing foods
- Encouraging individuals to experiment with a larger variety of foods
- Affordable alternatives for the most commonly consumed foods. For example, whole wheat bread in place of white bread, brown rice (with added lentils) instead of white rice or maize meal, and small quantities of artificially sweetened beverages, or preferably clean water in place of sugar sweetened beverages
- Education on the adverse effects of excessive sugar intake and of sources of added sugar could be incorporated into school programs or offered as short courses at day clinics. These could provide practical tips on how to cut down on or wean off of added sugar consumption
- Including further ideas and tips on healthy, cost-effective snacks, especially in adolescents, such as fruits and vegetables. Where possible, schools, clinics and other public spaces could be encouraged to set up and run vegetable gardens to assist in improving the availability of fresh vegetables and fruits.

The socio-economic situation of the study sample must be taken into consideration when deciding on, developing and implementing intervention strategies. Current intervention programs can improve on their effectiveness by offering supplements that elicit fewer adverse side effects, by educating pregnant women on the benefits of necessary supplements, and by promoting and increasing awareness of available programs and the benefits that could be reaped from these. Informing all health care providers of these programs as well as ways in which they could assist their patients to register for them could increase the efficacy of such programs and make them available to a larger proportion of the population.

Concerning future research, several recommendations can be made:

- Future studies should use the standardised food group classifications for determing the DDS as suggested by the FAO (Kennedy et al., 2012) to allow for easy comparisons between studies and population groups.
- Further research could be done regarding the lack of association between weight and income to confirm the accuracy of this finding or to identify possible confounding factors, and to find factors that do influence pre-pregnancy weight status.
- A similar study could be carried out on a larger study sample, and participants could be followed up over the course of their pregnancy to assess nutritional intake and the dietary (and anthropometric) changes that take place as pregnancy progresses. From these findings, an intervention-based study can be set up to determine the feasibility and effectiveness of various intervention strategies to improve dietary intake as well as pregnancy outcomes.

In conclusion, this study shows that dietary intake of the study sample is characterised by excessive intakes of refined carbohydrates, sugar and fat along with inadequate intakes of mean energy, fibre, legumes, fish and fruits and vegetables. A lack of intake of these foods is likely a major cause of the inadequate intakes of folate, iron, zinc, calcium and magnesium which were insufficient to meet pregnancy requirements. As a result, the study sample is at greater risk of unfavourable pregnancy outcomes for both the infant and mother. It seems likely that nutrition education, especially with regard to food choices, may have beneficial effects on this study sample. Although programs are currently in place to assist pregnant women nutritionally they may not be operating as effectively as possible and may not be reaching all targeted individuals. These programs offer an ideal setting for nutrition education and they should be used as avenues for reaching those that have been identified as having or being at risk of deficiencies or inadequate intakes. Although effective supplementation and fortification programs may improve the nutritional status of pregnant women it is clear that interventions focussing on changing dietary intake is necessary. This may improve the general health of pregnant women and pregnancy outcomes which may in turn assist in breaking the perpetuated cycle of poor health, poor education and poor income.

REFERENCES

Abrams S. 2007. In utero physiology: Role in nutrient delivery and fetal development for calcium, phosphorus, and vitamin D. *Am J Clin Nutr*. 85(suppl):604S-7S.

Achkar M, Dodds L, Giguere Y, Forest JC, Armson BA, Woolcott C, Agellon S, SPencer A, Weiler HA. 2014. Vitamin D status in early pregnancy and risk of preeclampsia. *Am J Obstet Gynecol*. 212(1):1-7.

Adamson AJ, Baranowski T. 2014. Developing technological solutions for dietary assessment in children and young people. *J Hum Nutr Diet*. 27(s1):1-4.

Adibi SA, Baraona E, Lieber CS. 1992. Effects of ethanol on amino acids and protein metabolism. In *medical and nutritional complications of alcoholism: Mechanism and management*, pp. 127-55 [Lieber CS, editor]. New York: Plenum Press.

Aghajafari F, Nagulesapillai T, Ronksley PE, Tough SC, O'Beirne M, Rabi DM. 2013. Association between maternal serum 25-hydroxyvitamin D level and pregnancy and neonatal outcomes: Systematic review and meta-analysis of observational studies. *Brit Med J*. 346:1-14.

Aikins, MP. 1998. Alternative therapies for nausea and vomiting of pregnancy. *Obstet Gynecol*. 91:149–55.

Ailhaud G, Massiera F, Weill P, Legrand P, Alessandri JM, Guesnet P. 2006. Temporal changes in dietary fats: Role of n-6 polyunsaturated fatty acids in excessive adipose tissue development and relationship to obesity. *Prog Lipid Res*. 45:203–36.

Alaily AB, Carrol KB. 1978. Pulmonary ventilation in pregnancy. *Brit J Obst Gyn.* 85: 518–24.

Aliyu MH, Salihu HM, Wilson RE, Alio AP, Kirby RS. 2008. The risk of intrapartum stillbirth among smokers of advanced maternal age. *Arch Gyn Obst*. 278: 39–45.

Al-Jameil N, Tabassum H, Al-Mayouf H, Aljohar HI, Alenzi ND, Hijazy SM, Khan FA. 2014. Analysis of serum trace elements-copper, manganese and zinc in preeclamptic pregnant women by inductively coupled plasma optical emission spectrometry: a prospective case controlled study in Riyadh, Saudi Arabia. *Int J Clin Exp Pathol*. 7(5):1900-10.

Allen LH. 2005. Multiple micronutrients in pregnancy and lactation: an overview. *Am J Clin Nutr*. 81(suppl):1206S–12S.

American Congress of Obstetricians and Gynecologists (ACOG): ACOG Committee opinion number 315, Sept 2005. Obesity in pregnancy, *Obstet Gynecol*. 106(3):671.

Amugsi DA, Mittelmark MB, Lartey A. 2014. Dietary diversity is a predictor of acute malnutrition in rural but not in urban settings: evidence from Ghana. *Brit J Med & Med Research*. 4(25):4310-24.

Ananth CV, Basso O. 2010. Impact of pregnancy-induced hypertension on stillbirth and neonatal mortality. *Epidemiology*. 21: 118–23.

Anderson AS. 2001. Pregnancy as a time for dietary change? Symposium on 'Nutritional adaptation to pregnancy and lactation'. *P Nutr Soc*. 60:497-504.

Anderson MA. 2008. Medical nutrition therapy for low-birth-weight infants. In *Krause's food & nutrition therapy*, 12th ed., pp. 1117-1140 [Hebberd K, Alexopoulos Y, Bays H, editors]. Elsevier.

Anderson MA. 2012. Medical nutrition therapy for low-birth-weight infants. In *Krause's food & nutrition therapy*, 13th ed., pp. 972-95 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Antonow-Schlorke I, Schwab M, Cox LA. 2011. Vulnerability of the fetal primate brain to moderate reduction in maternal global nutrient availability. Proc Natl Acad Sci U S A. 108:3011–6.

Appleton KM, Roger PJ, Ness AR. 2010. Updated systematic review and meta-analysis of the effects of n-3 long-chain polyunsaturated fatty acids on depressed mood. *Am J Clin Nutr.* 91:757–70.

Araújo CL, Hallal PC, Nader GA, Neutzling MB, Defátima VM., Menezes AM et al. 2009. Effect of birth size and proportionality on BMI and skinfold thickness in early adolescence: Prospective birth cohort study. *Eur J Clin Nutr*. 63; 627–33.

Artal R, Lockwood CJ, Brown HL. 2010. Weight gain recommendations in pregnancy and the obesity epidemic, *Obstet Gynecol*. 115(1):152-5.

Aune D, Norat T, Romundstad P & Vatten L. 2013. Whole grain and refined grain consumption and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of cohort studies. *Eur J Epidemiol.* 28:845-58.

Azaïs-Braesco V, Pascal G. Vitamin A in pregnancy: requirements and safety limits. 2000. *Am J Clin Nutr.* 71(suppl):1325S–33S.

Baker PN, Wheeler SJ, Sanders TA, Thomas JE, Hutchinson CJ, Clarke K et al. 2009. A prospective study of micronutrient status in adolescent pregnancy. *Am J Clin Nutr*. 89:1114–24.

Bale TL, Baram TZ, Brown AS, Goldstein JM, Insel TR, McCarthy MM et al. 2010. Early life programming and neurodevelopmental disorders. *Biol psychiatry*. 68:314–9.

Ball EW, Giles C. 1964. Folic acid and vitamin B12 levels in pregnancy and their relation to megaloblastic anaemia. *J Clin Pathol.* 17:165–74.

Baranowski T, Islam N, Douglass D, Dadabhoy H, Beltran A, Baranowski J, Thompson D, Cullen KW, Subar AF. 2014. Food intake recording software system, version4 (FIRS St4): a self-completed 24-h dietary recall for children. *J Hum Nutr & Diet*. 27(s1):66-71.

Barker DJ, Eriksson JG, Forsen T, Osmond C. 2002. Fetal origins of adult disease: strength of effects and biological basis. *Int J Epidemiol*. 31:1235–9.

Barker DJ, Thornburg KL. 2013. Placental programming of chronic diseases, cancer and lifespan: a review. *Placenta*. 34: 841–845.

Barker DJ. 1990. The fetal infant origins of adult disease. *Brit Med J*. 301(11):1111.

Barker DJ. 2006. Adult consequences of fetal growth restriction. *Clin Obstet Gynecol*. 49:270–83.

Barker ME, McClean SI, ThompsonKA, Reid NG. 1990. Dietary behaviours and sociocultural demographics in Northern Ireland. *Brit J Nutr*. 64: 319-29.

Barr S, Murphy SP & Poos M. 2002. Commentary: Interpreting and using the Dietary

Basiotis PP, Welsh SO, Cronin FJ, Kelsay JL, Mertz W. 1987. Number of days of food intake records required to estimate individual and group nutrient intakes with defined confidence. *J Nutr*, 117, 1638-41.

Becquey E, Martin-Prevel Y. 2010. Micronutrient adequacy of women's diet in urban Burkina Faso is low. *J Nutr*. 140(11):20795-855.

Bekkers MBM, Elstgeest LEM, Scholtens S, Haveman-Nies A, de Jongste JC, Kerkhof M et al. 2012. Maternal use of folic acid supplements during pregnancy, and childhood respiratory health and atopy. *Eur Respir J*. 39:1468–74.

Bekkhus M, Skjøthaug T, Nordhagen R, Borge AIH. 2010. Intrauterine exposure to caffeine and inattention/overactivity in children. *Acta Paediatr*. 99(6):925–8.

Bellamy L, Casas JP, Hingorani AD, Williams D. 2009. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. *Lancet*. 373: 1773–9.

Benn CS, Aaby P, Arts RJ, Jensen KJ, Netea MG, Fisker AB. 2015. An enigma: why vitamin A supplementation does not always reduce mortality even though vitamin A deficiency is associated with increased mortality. *Int J Epidemiol.* dyv117.

Bertrand J, Floyd RL, Weber MK, O'Connor M, Riley EP, Johnson KA, et al. 2004. Fetal alcohol syndrome: Guidelines for referral and diagnosis. Atlanta, GA: *Centers for Disease Control and Prevention*.

Bhate VK, Joshi SM, Ladkat RS, Deshmukh US, Lubree HG, Katre PA et al. 2012. Vitamin B12 and folate during pregnancy and offspring motor, mental and social development at 2 years of age. *J Dev Orig Health Dis*. 3(2):123–30.

Biltoft-Jensen A, Matthiessen J, Rasmussen LB, Fagt S, Groth MV, Hels O. 2009. Validation of the Danish 7-day pre-coded food diary among adults: energy intake v. energy expenditure and recording length. *Brit J Nutr*. 102:1838–46.

Bingham SA, Gill C, Welch A, Day K, Cassidy A, Khaw KT et al. 1994. Comparison of dietary assessment methods in nutritional epidemiology: weighed records, 24 h recalls, food-frequency questionnaires and estimated-diet records. *Brit J Nutr*. 72: 619-43.

Biro G, Hulshof KF, Ovesen L, Amorim Cruz JA. 2002. Selection of methodology to assess food intake. *Eur J Clin Nutr*. 56(Suppl 2): S25–32.

Black AE, Prentice AM, Goldberg GR, Jebb SA, Bingham SA, Livingstone MBE et al. 1993. Measurements of total energy expenditure provide insights into the validity of dietary measurements of energy intake. *J Am Diet Assoc*. 93:572-9.

Black AE. 1999. Small eaters or under-reporters? In *Progress in obesity research*, pp. 223–8 [G Ailhaud and B Guy-Grand, editors]. Paris: John Libbey.

Black MM, Quigg AM, Hurley KM, Pepper MR. 2011. Iron deficiency and iron-deficiency anemia in the first two years of life: strategies to prevent loss of developmental potential. *Nutr Rev*. 69(Suppl. 1):S64–S70.

Black MM. 2003. Micronutrient deficiencies and cognitive functioning. *J nutr*. 133(11): 3927-31.

Block SR, Watkins SM, Salemi JL, Rutkowski R, Tanner JP, Correia JA, Kirby RS. 2013. Maternal pre-pregnancy Body Mass Index and risk of selected birth defects: Evidence of a dose-response relationship. *Paediatric & Perinatal Epidemiol*. 27(6):521-31.

Blumfield M, Hure A, MacDonald-Wicks L, Smith R, Simpson S, Raubenheimer D, Collins C. 2012. The association between the macronutrient content of maternal diet and adequacy of micronutrients during pregnancy in the women and their children's health (WATCH) study. *Nutrients*. 4(12):1958-76.

Blundell JE, Caudwell P, Gibbons C, Hopkins M, Näslund E, King NA, Finlayson G. 2012. Body composition and appetite: fat-free mass (but not fat mass or BMI) is positively associated with self-determined meal size and daily energy intake in humans. *Brit J Nutr. 107*(3):445-9.

Bodnar LM, Catov JM, Simhan HN, Holick MF, Powers RW, Roberts JM. 2007. Maternal vitamin D deficiency increases the risk of preeclampsia. *J Clin Endocrinol Metab*. 92:3517–22.

Bomela NJ. 2015. A cross-sectional analysis of the geographic distribution and causes of maternal mortality in South Africa: 2002 - 2006. *BMC*. 15:273-84.

Bopape MM, Mbhenyane XG, Alberts M. 2008. The prevalence of anaemia and selected micronutrient status in pregnant teenagers of Polokwane Municipality in the Limpopo Province. *S Afr J Clin Nutr.* 21(4):332-6.

Brabin BJ, Premji Z, Verhoeff F. 2001. Iron-deficiency anemia: Reexamining the nature and magnitude of the public health problem. *J Nutr*. 131:636S-48S.

Bradley CS, Kennedy CM, Turcea AM, Rao SSC, Nygaard IE. 2007. Constipation in Pregnancy: Prevalence, Symptoms, and Risk Factors. *Obstet Gynecol*.110:1351–7.

Brooks RC, Simpson SJ, Raubenheimer D. 2010. The price of protein: combining evolutionary and economic analysis to understand excessive energy consumption. *Obes Rev.* 11(12):887-94.

Broussard CN, Richter JE. 1998. Nausea and vomiting of pregnancy. *Gastroenterol Clin N*. 27:123–51.

Brown JE. 2008. Nutrition during pregnancy. In *Nutrition through the lifecycle*, pp. 83-154 [Adams P, Rose N, Feldman E, editors]. Thomson Wadsworth.

Bruening M, MacLehose R, Loth K, Story M, Neumark-Sztainer D. 2012. Feeding a family in a recession: food insecurity among Minnesota parents. *Am J Pub Health. 102*(3):520-6.

Brunetti-Pierri N, Hunter JV, Boerkoel CF. 2007. Gray matter heterotopias and brachytelephalangic chondrodysplasia punctata: A complication of hyperemesis gravidarum induced vitamin K deficiency?. *Am J Med Genetics* Part A, 143(2), 200-4.

Bullen JJ, Rogers HJ, Spalding PB, Ward CG. 2005. Iron and infection: The heart of the matter. FEMS Immunol Med Microbiol. 43:325–30.

Bureau S, Mouhoubi S, Touloumet L, Garcia C, Moreau F, Bédouet V, Renard CM. 2015. Are folates, carotenoids and vitamin C affected by cooking? Four domestic procedures are compared on a large diversity of frozen vegetables. *LWT-Food Sci &Technol*. 64(2):735-41.

Burke G, Robinson K, Refsum H, Stuart B, Drumm J, Graham I. 1992. Intrauterine growth retardation, perinatal death, and maternal homocysteine levels. *N Engl J Med.* 326:69–70.

Burrows TL, Martin RJ, Collins CE. 2010. A systematic review of the validity of dietary assessment methods in children when compared with the method of doubly labelled water. *J Am Diet Assoc*. 110:1501-10.

Butte NF, King JC. 2005. Energy requirements during pregnancy and lactation. *Public Health Nutr*. 8:1010-27.

Butte NF. 2000. Carbohydrate and lipid metabolism in pregnancy: normal compared with gestational diabetes mellitus. *Am J Clin Nutr*. 71: 1256S-61S.

Byrd-Bredbenner C, Schwartz J. 2004. The effect of practical portion size measurement aids on the accuracy of portion size estimates made by young adults. *J Hum Nutr Diet*. 17:351-7.

Cade J, Thompson R, Burley V, Warm D. 2002. Development, validation and utilisation of food frequency questionnaires – a review. *Public Health Nutr*. 5(4):567-87.

Cade JE, Burley VJ, Warm DL, Thompson RL, Margetts BM. 2004. Food-frequency questionnaires: a review of their design, validation and utilisation. *Nutr Res Rev*.17:5-22.

Callaway LK, O'Callaghan M, McIntyre DH. 2009. Obesity and the hypertensive disorders of pregnancy. *Hypertens Preg*. 28: 473-93.

Calvo EB, López LB. 2012. Reference charts for anthropometric changes during pregnancy. In *Handbook of Anthropometry: Physical measures of human form in health and disease*, pp. 999-1019 [Preedy VR, editor]. Springer Science+Business Media

Campoy C, Escolano-Margarit MV, Anjos T, Szajewska H, Uauy R. 2012. Omega 3 fatty acids on child growth, visual acuity and neurodevelopment. *Brit J Nutr*. 107:S85-S106.

Canada's Food Guide. 2011. *Eating well with Canada's Food Guide* http://www.hc-sc.gc.ca/fn-an/food-guide-aliment/track-suivi/table_female-femme_preg-ence_age19-50-eng.php Accessed [13 July 2015]

Carlin A, Alfirevic Z. 2008 Physiological changes of pregnancy and monitoring. *Best Pract Res Cl Ob*. 22(5):801–23.

Carlo WA, Goudar SS, Jehan I, Chomba E, Tshefu A, Garces A et al. 2010. Newborn-care training and perinatal mortality in developing countries. *N Engl J Med*. 362:614-23.

Carlsen MH, Lillegaard ITL, Karlsen A, Blomhoff R, Drevon CA, Andersen LF. 2010. Evaluation of energy and dietary intake estimates from a food frequency questionnaire using independent energy expenditure measurement and weighed food records. *Nutr J*. 9(1):37-46.

Carmichael SL, Arbrams B. 1997. A critical review of the relationship between gestational weight gain and preterm delivery. *Obstet gynecol*. 89:865-73.

Carmichael SL, Shaw GM, Schaffer DM, Laurent C, Selvin S. 2003. Dieting behaviour and risk of neural tube defects. *Am J Epidemiol*. 158:1127-31.

Carrillo JA, Benitez J. 1996. CYP1A2 activity, gender and smoking, as variables influencing the toxicity of caffeine. *Brit J Clin Pharmacol*. 41:605–8.

Casanueva E, Viteri FE. 2003. Iron and oxidative stress in pregnancy. *J Nutr.* 133:1700S–8S.

Catalano PM, Thomas A, Huston-Presley L, Amini SB. 2003. Increased fetal adiposity: a very sensitive marker of abnormal in utero development. *Am J Obstet Gynecol*. 189:1698-704.

Caudwell P, Finlayson G, Gibbons C, Hopkins M, King N, Näslund E, Blundell JE. 2013. Resting metabolic rate is associated with hunger, self-determined meal size, and daily energy intake and may represent a marker for appetite. *Am J Clin Nutr. 97*(1): 7-14.

Centers for Disease Control and Prevention (CDC). 1993. Fetal alcohol syndrome - United States, 1979-1992. *MMWR Morb Mortal Wkly Rep*. 42(17):339-41.

Centers for Disease Control and Prevention (CDC). 2004. National Health and Nutrition Examination Survey Anthropometry Procedures Manual [Internet]. Available from http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_an.pdf. Accessed 23 November 2015.

Centers for Disease Control and Prevention (CDC). 2014. Folic acid: Recommendations. http://www.cdc.gov/ncbddd/folicacid/recommendations.html. Accessed 7 January 2015.

Cetin I, Alvino G. 2009. Intrauterine growth restriction: Implications for placental metabolism and transport: a review. *Placenta*. 30(Supplement A):23 S77–S82.

Chambers E, McGuire B, Godwin S, McDowell M, Vecchio F. 2000. Quantifying portion sizes for selected snack foods and beverages in 24-hour dietary recalls. *Nutr Res*. 20:315-26.

Chang SC, O'Brien KO, Nathanson MS. 2003. Fetal femur length is influenced by maternal dairy intake in pregnant African American adolescents. *Am J Clin Nutr*. 77:1248–54.

Chen XK, Wen SW, Fleming N, Demissie K, Rhoads GG, Walker M. 2007. Teenage pregnancy and adverse birth outcomes: a large population based retrospective cohort study. *Int J Epidemiol*. 36:368–73.

Cheng D, Kettinger L, Uduhiri K, Hurt L. 2011. Alcohol consumption during pregnancy: Prevalence and provider assessment. *Obstet Gynecol*. 117:212–7.

Christian P, Murray-Kolb LE, Tielsch JM, Katz J, LeCLerq SC, Khatry SK. 2014. Associations between preterm birth, small-for-gestational age, and neonatal morbidity and cognitive function among school-age children in Nepal. *BMC Pediatr*. 14:58.

Christian P, Stewart CP. 2010. Maternal micronutrient deficiency, fetal development, and the risk of chronic disease. *J Nutr*. 140: 437–45.

Churchill JA, Berendes HW, Nemore J. 1969. Neuropsychological deficits in children of diabetic mothers. *Am J Obstet Gynecol*. 105:257–68.

Cianfarani S, Agostoni C, Bedogni G, Berni Canani R, Brambilla P, Nobili V et al. 2012. Effect of intrauterine growth retardation on liver and long-term metabolic risk. *Int J Obesity*. 1–8. DOI:10.1038/ijo.2012.54.

Clausson B, Granath F, Ekbom A, Lundgren S, Nordmark A, Signorello LB, Cnattingius S. 2002. Effect of caffeine exposure during pregnancy on birth weight and gestational age. *Am J Epidemiol*. 155, 429–36.

Cocchi G, Gualdi S, Bower C, Halliday J, Jonsson B, Myrelid, et al. 2010. International trends of Down Syndrome 1993–2004: Births in relation to maternal age and terminations of pregnancies. *Birth Defects Res* (Part A). 88:474–9.

Cogswell ME, Parvanta I, Ickes L, Yip R, Brittenham GM. 2003. Iron supplementation during pregnancy, anemia, and birth weight: A randomized controlled trial. *Am J Clin Nutr*. 78:773–81.

Cohen JH, Kim H. 2009. Sociodemographic and health characteristics associated with attempting weight loss during pregnancy. *Prev Chronic Dis.* 6(1).http://www.cdc.gov/pcd/issues/2009/jan/07_0192.htm. Accessed [7 June 2013].

Colak A, Yildiz O, Toprak B, Turkon H, Halıcıoglu O, Coker I. 2014. Correlation between calcium and phosphorus in cord blood and birth size in term infants. *Minerva pediatrica*.

Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. *Brit Med J*, *320*(7244): 1240.

Committee on Toxicity of Chemicals in Foods, Consumer Products and the Environment (COT). 2001. Statement on the reproductive effects of caffeine. London: Food Standards Agency.

Conen D, Tedrow UB, Cook NR, Buring JE, Albert CM. 2010. Birth weight is a significant risk factor for incident atrial fibrillation. *Circulation*.122:764-70.

Cooke LA, Mimouni FB. 1997. Whole blood ionized magnesium in the healthy neonate. *J Am Coll Nutr*. 16:181-3.

Cooper BA, Lowenstein L. 1964. Relative folate deficiency of erythrocytes in pernicious anemia and its correction with cyanocobalamin. *Blood J*. 24:502-21.

Cox JT, Phelan ST. 2008. Nutrition during pregnancy. *Obstet Gynecol Clin N Am*. 35:369–83.

Cox P, Marton T. 2009. Pathological assessment of intrauterine growth restriction. *Best Pract Res Cl Ob*. 23:751–64.

Cox S. 2008. Staying healthy on a vegetarian diet during pregnancy. *J Midwifery Wom Heal*. 53(1):91-2.

Craig WJ, Mengels AR. 2009. Position of the American Dietetic Association: Vegetarian Diets. *J Am Diet Assoc*. 109:1266-82.

Craig WJ. 2010. Nutrition concerns and health effects of vegetarian diets. *Nutr Clin Pract*. 25:613-20.

Crozier SR, Inskip HM, Godfrey KM, Cooper C, Harvey NC, Cole ZA et al. 2010. Weight gain in pregnancy and childhood body composition: findings from the Southampton Women's Survey. *Am J Clin Nutr*. 91:1745–51.

Crume TL, Scherzinger A, Stamm E, McDuffie R, Bischoff KJ, Hamman RF, Dabelea D. 2014. The long-term impact of intrauterine growth restriction in a diverse US cohort of children: The EPOCH study. *Obesity*. 22(2):608-15.

Crystal SR, Bowen DJ, Bernstein IL. 1999. Morning sickness and salt intake, food cravings, and food aversions. *Physiol Behav*. 67(2):181–7.

Cullen G, O'Donoghue D. 2007. Constipation and pregnancy. *Best Pract Res Clin Ga*. 21(5); 807–18.

Curtis EM, Moon RJ, Dennison EM, Harvey NC. 2014. Prenatal calcium and vitamin D intake, and bone mass in later life. *Current Osteoporosis Reports*. 12(2): 194-204.

Cypel YS, Guenther PM, Petot GJ. 1997. Validity of portion-size measurement aids: a review. *J Am Diet Assoc*. 97: 289–92.

Dall'Alba VD, Fornari F, Krahe C, Callegari-Jacques SM, de Barros SGS. 2010. Heartburn and regurgitation in pregnancy: The effect of fat ingestion. *Dig Dis Sci*. 55:1610–4.

Davis R, Hofferth SL, Shenassa ED. 2014. Gestational weight gain and risk of infant death in the United States. *Am J Pub Health*. 104(51):S90-5.

Dawson JE, Raymond AM, Winn LM. 2006. Folic acid and pantothenic acid protection against valproic acid-induced neural tube defects in CD-1 mice. Toxicol & applied pharmacol. 211(2), 124-132.

de Castro JM. 1999. Measuring real-world eating behavior. *Prog Obes Res*. 8: 215–21.

De Cock N, D'Haese MD, Vink N, van Rooyen CJ, Staelens L, Schonfeldr HC, D'Haese L. 2013. Food security in rural areas of Limpopo province, South Africa. *Food Security*. 5(2):269-82.

de Goede J, Geleijnse JM, Ding EL, Soedamah-Muthu SS. 2015. Effect of cheese consumption on blood lipids: a systematic review and meta-analysis of randomized controlled trials. *Nutr rev*. 73(5): 259-75.

De Irala-Estevez J, Groth M, Johansson L, Oltersdorf U, Prattala R, Martinez-Gonzalez MA. 2000. A systematic review of socio-economic differences in food habits in Europe: consumption of fruit and vegetables. *Eur J Clin Nutr*. 54: 706-14.

Delbaere I, Verstraelen H, Goetgeluk S, Martens G, De Backer G et al. 2007. Pregnancy outcome in primiparae of advanced maternal age. *Eur J Obstet Gynecol Reprod Biol*. 135:41–6.

Department of Health, Medical Research Council, OrcMacro. 2007. South Africa Demographic and Health Survey 2003. Pretoria: Department of Health.

Department of Health. 2007b. Guidelines for maternity care in South Africa: A manual for clinics, community health centres and district hospitals. 3rd edition. www.kznhealth.gov.za/family/Maternity_care_guidelines_2007.pdf Accessed [4 August 2015].

Derbyshire E, Davies GJ, Costarelli V, Dettmar PW. 2009. Habitual micronutrient intake during and after pregnancy in Caucasian Londoners. *Matern Child Nutr*, 5:1–9.

De-Regil LM, Fernández-Gaxiola AC, Dowswell T, Peña-Rosas JP. 2010. Effects and safety of periconceptional folate supplementation for preventing birth defects. *Cochrane Database of Systematic Reviews*. Issue 10. Art. No.: CD007950. DOI:

Deutsch AB, Lynch O, Alio AP, Salihul HM, Spellacy WN. 2010. Increased risk of placental abruption in underweight women. *Am J Perinatol*. 27(3): 235-40.

Dewey KG, Cohen RJ. 2007. Does birth spacing affect maternal or child nutritional status? A systematic literature review. *Matern Child Nutr*. 3:151–73.

Dewey KG, McCrory MA. 1994. Effects of dieting and physical activity on pregnancy and lactation. *Am J Clin Nutr*. 59:446S–52S.

DHIS: District Health Information System Database. National Department of Health. http://hisp.org/. 2012/13 financial year.

Ding M, Leach M, Bradley H. 2013. The effectiveness and safety of ginger for pregnancy-induced nausea and vomiting: a systematic review. *Women Birth*. 26:e26-30.

Dietz PM, Callaghan WM, Cogswell ME, Morrow B, Ferre C, Schieve LA. 2006. Combined effects of prepregnancy body mass index and weight gain during pregnancy on the risk of preterm delivery. *Epidemiol*. 17:170–7.

Dowswell T, Neilson JP. 2008. Interventions for heartburn in pregnancy (Review). The Cochrane Collaboration. Issue 4. Art. No.:CD007065. DOI: 10.1002/14651858.CD007065.pub2

Drake AJ, Walker BR. 2004. The intergenerational effects of fetal programming: non-genomic mechanisms for the inheritance of low birth weight and cardiovascular risk. *J Endocrinol*. 180:1–16.

Drake R, Reddy S, Davies J. 1998. Nutrient intake during pregnancy and pregnancy outcome of lacto-ovo-vegetarians, fish-eaters and non-vegetarians. *Veg Nutr*. 2:45-52.

Drimie S, Faber M, Vearey J, Nunez L. 2013. Dietary diversity of formal and informal residents in Johannesburg, South Africa. *Pub Health*. 13:911.

Drossman DA, Dumitrascu DL. 2006. Rome III: New standard for functional gastrointestinal disorders. *J Gastrointestin Liver Dis*. 15(3):237-41.

Duflo E. 2003. Grandmothers and granddaughters: old age pensions and intra-household allocation in South Africa. World Bank Econom Rev. 17(1):1–25.

Dukes KA, Burd L, Elliott AJ, Fifer WP, Folkerth RD, Hankins GDV, Hereld D, Hoffman HJ, Myers MM, Odendaal HJ, Signore C, Sullivan LM, Willinger M, Wright C, Kinney HC. 2014. The Safe Passage STudy: Designs, methods, recruitment, and follow-up approach. *Paed Perinat Epidemiol.* 1-11

Durlach J. 2004. New data on the importance of gestational Mg deficiency. *J Am Coll Nutr*. 23: 694S–700S.

Durnwald CP, Ehrenberg HM, Mercer BM. 2004. The impact of maternal obesity and weight gain on vaginal birth after cesarean section success. *Am J Obstet Gynecol.*191(3):954-7.

Edenberg HJ. 2007. The genetics of alcohol metabolism: Role of alcohol dehydrogenase and aldehyde dehydrogenase variants. *Alcohol Res Health*. 30(1):5–13.

Edwards LE, Hellerstedt WL, Alton IR, Story M, Himes JH. 1996. Pregnancy complications and birth outcomes in obese and normal-weight women: Effects of gestational weight change. *Obstet Gynecol*. 87:389–94.

Ehrenberg HM, Huston-Presley L, Catalano PM. 2003. The influence of obesity and gestational diabetes mellitus on accretion and the distribution of adipose tissue in pregnancy. *Am J Obstet Gynecol*. 189:944–8.

Ehrenthal BR, Minkovitz CS, Strobino DM. 2014. Maternal obesity, gestational weight gain, and childhood growth in the first year of life. In *Obesity during pregnancy in clinical practice*, pp. 229-32 [Nicholson W, Baptiste-Roberts K, editors]. Springer-Verlag London.

Eichenwald EC, Stark AR. 2008. Management and Outcomes of Very Low Birth Weight. *N Engl J Med*. 358:1700-11.

Einarson A, Maltepe C, Boskovic R, Koren G. 2007. Treatment of nausea and vomiting in pregnancy: An updated algorithm. *Can Fam Physician*. 53(12):2109-11.

Epping-Jordan JE, Galea G, Tukuitonga C, Beaglehole R. 2005. Preventing NCDs: taking stepwise action. Lancet. 366(9497):1667-71.

Erick M. 2008. Nutrition during pregnancy and lactation. In *Krause's food & nutrition therapy*, 12th ed., pp.160-98 [Hebberd K, Alexopoulos Y, Bays H, editors]. Elsevier.

Erick M. 2012. Nutrition in pregnancy and lactation. In *Krause's food and the nutrition care process*, 13th ed., pp.340-374 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Escamilla-Nuñez MC, Barraza-Villarreal A, Hernandez-Cadena L, Navarro-Olivos E, Sly PD, Romieu I. 2014. Omega-3 fatty acid supplementation during pregnancy and respiritory symptoms in children. *Chest*. 146(2):373-82.

Euser AG, Cipolla MJ. 2009. Magnesium sulfate for the treatment of eclampsia: A brief review. *Stroke*. 40:1169-75.

Faber M, Laubscher R, Berti C. 2014. Poor dietary diversity and low nutrient density of the complementary diet for 6-to 24-month-old children in urban and rural KwaZulu-Natal, South Africa. *Maternal & child nutrition*.

Faggiano F, Vineis P, Cravanzola D, Pisani P, Xompero G, Riboli E et al. 1992. Validation of a method for the estimation of food portion size. *Epidemiology*. 3:379-82.

Fall CHD, Fisher DJ, Osmond C, Margetts BM. 2009. Multiple micronutrient supplementation during pregnancy in low-income countries: A meta-analysis of effects on birth size and length of gestation. *Food Nutr Bull*. 30(4):5333-46.

FAO/WHO/UNU. 2002. Joint expert consultation on protein and amino acid requirements in human nutrition. Geneva, Switzerland.

FBDG-SA. 2013. Food-Based Dietary Guidelines for South Africa. S Afr J Clin Nutr. 26(3)(Supplement):S1-S164

Feskanich D, Willett WC. 1993. The use and validity of food frequency questionnaires in epidemiological research and clinical practice. *Medicine, Exercise, Nutrition, and Health*. 2:143-54.

Fishbane S. 1999. Review of issues relating to iron and infection. Am J Kidney Dis. 34[Suppl 2]:S47–S52.

Fleming TP, Kwong WY, Porter R, Ursell E, Fesenko I, Wilkins A et al. 2004. The embryo and its future. *Biol Reprod*. 71:1046–54.

Flenady V, Koopmans L, Middleton P, Froen JF, Smith GC, Gibbons K et al. 2011. Major risk factors for stillbirth in high-income countries: a systematic review and meta-analysis. *Lancet.* 377: 1331–40.

Food and Agriculture Organization (FAO)/World Health Organization (WHO). 2002. Human vitamin and mineral requirements. Report of a joint FAO/WHO expert consultation. Rome: FAO.

Food and Nutrition board of the IOM. 2006. Dietary reference intakes: The essential guide to nutrient requirements. Otten JJ, Hellwig JP, Meyers LD, Editors. *The National Academies Press*, Washington, D.C.

Food and Nutrition board of the IOM. 2010. Dietary reference intakes for calcium and vitamin D. [Ross AC, Taylor CL, Yaktine AL, Del Valle HB, editors]. *The National Academies Press*, Washington, D.C. 1103-16.

Food Guide Pyramid. 1992. A Guide to Daily Food Choices, US Dept of Agriculture. *Human Nutrition Information Service, Washington, DC, Home and Garden Bulletin*, (252).

Foote JA, Murphy SP, Wilkens LR, Basiotis PP, Carlson A. 2004. Dietary variety increases the probability of nutrient adequacy among adults. *J Nutr*. 134: 1779–85.

Forno E, Young OM, Kumar R, Simhan H, Celedon JC. 2014. Maternal obesity in pregnancy, gestational weight gain, and risk of childhood asthma. *Pediatrics*. 134(2):535-46.

Forrestal SG. 2011. Energy intake misreporting among children and adolescents: a literature review. *Maternal & child nutrition. 7*(2):112-7.

Foster E, Matthews JNS, Lloyd J, Marshall L, Mathers JC, Nelson M et al. 2008. Children's estimates of food portion size: The development and evaluation of three portion size assessment tools for use with children. *Br J Nutr*. 99;175-84.

Frederick IO, Williams MA, Sales AE, Martin DP, Killien M. 2008. Pre-pregnancy body mass index, gestational weight gain, and other maternal characteristics in relation to infant birth weight. *Matern Child Health J*. 12(5):557-67.

Friis H, Gomo E, Nyazema N, Ndhlovu P, Krarup H, Kaestel P, Michaelsen KF. 2004. Maternal body composition, HIV infection and other predictors of gestation length and birth size in Zimbabwe. Br J Nutr. 92:833–40.

Fugelstad A, Rao R, Georgieff MK. 2008. The role of nutrition in cognitive development. In: Nelson CA and Luciana M, ed. Handbook of Developmental Cognitive Neuroscience, 2nd ed. Cambridge, MA: MIT Press. 623–641.

Furneaux EC, Langley-Evans AJ, Evans SCL. 2001. Nausea and vomiting of pregnancy: endocrine basis and contribution to pregnancy outcome. *Obstet Gynecol Surv*. 56, 775-82.

Gagné A, Wei SQ, Fraser WD, Julien P. 2009. Absorption, transport, and bioavailability of vitamin e and its role in pregnant women. J Obstet Gynaecol Canada. 31(3):210-7.

Gallagher ML. 2008. The nutrients and their metabolism. In *Krause's food & nutrition therapy*, 12th ed., pp. 39-143 [Hebberd K, Alexopoulos Y, Bays H, editors]. Elsevier.

Gallagher ML. 2012. Intake: The nutrients and their metabolism. In *Krause's food and the nutrition care process*, 13th ed., pp.32-128 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Ganpule A, Yajnik CS, Fall CHD. 2006. Bone mass in Indian children – relationships to maternal nutritional status and diet during pregnancy: The Pune Maternal Nutrition Study. J Clin Endocrinol Metab. 91:2994–3001.

Garza-Gisholt AC, Rivas-Ruiz R, ClarkP. 2012. Maternal diet and vitamin D during pregnancy and association with bone health during childhood: Review of the literature. *Bol Med Hosp Infant Mex*. 69(2):83-90

Gaunekar NN, Crowther CA. 2004. Maintenance therapy with calcium channel blockers for preventing preterm birth after threatened preterm labour. Cochrane Database Syst Rev 3:CD004071.

Geissler PW, Shulman CE, Prince RJ, Mutemi W, Mnazi C, Lowe B. 1998. Geophagy, iron status and anaemia among pregnant women on the coast of Kenya. *Trans R Soc Trop Med Hyg*. 92(5):549–53.

Getahun D, Ananth CV, Oyelese Y, Chavez MR, Kirby RS, Smulian JC. 2007. Primary preeclampsia in the second pregnancy: effect of changes in prepregnancy body mass index between pregnancies. *Obstet Gynecol*. 110(6):1319-25.

Gibson RS. Principles of Nutritional Assessment, 2nd ed. New York, NY: Oxford University Press; 2005.

Gilbert-Barness, Debich-Spicer D. 2005. General principles. In *Handbook of pediatric autopsy pathology*, pp. 3-38 [Breaugh MJ, editor]. Springer.

Gill SK, Broussard C, Devine O, Green RF, Rasmussen SA, Reefhuis J. 2012. Association between maternal age and birth defects of unknown etiology - United States, 1997–2007.

Gill SK, Maltepe C, Koren G. 2009. The effect of heartburn and acid reflux on the severity of nausea and vomiting of pregnancy. *Can J Gastroenterol*. 23(4): 270–2.

Gluckman PD. 2003. Fetal origins of adult disease: Insulin resistance. *Medscape Obstet Gynecol Womens Heal*. 8:4-5

Godrey KM, Barker DJ. 2000. Fetal nutrition and adult disease. *Am J Clin Nutr*. 71: 1344S-52S

Goel A, Jain V, Gupta I, Varma N. 2003. Serial serum ferritin estimation in pregnant women at risk of preterm labor. *Acta Obstet Gynecol Scand*. 82:129-32.

Goldberg GR, Black AE, Jebb SA, Cole TJ, Murgatroyd PR, Coward WA. 1991. Critical evaluation of energy intake data using fundamental principles of energy physiology. I. Derivation of cut-off limits to identify underrecording. *Eur J Clin Nutr*. 45:569-81.

Golding J, Steer C, Emmett P, Davis JM, Hibbeln JR. 2009. High levels of depressive symptoms in pregnancy with low omega-3 fatty acid intake from fish. *Epidemiology*. 20: 598–603.

Gould JF, Smithers LG, Makrides M. 2013. The effect of maternal omega-3 (n− 3) LCPUFA supplementation during pregnancy on early childhood cognitive and visual development: a systematic review and meta-analysis of randomized controlled trials. *Am J Clin Nutr.* 97(3):531-44.

Graafmans WC, Richardus JH, Borsboom GJJM, Bakketeig L, Langhoff-Roos J, Bergsjo P et al. 2002. Birth weight and perinatal mortality: a comparison of "optimal" birth weight in seven western European countries. *Epidemiology*. 13:569-74.

Grabenhorst F, Rolls ET. 2014. The representation of oral fat texture in the human somatosensory cortex. *Human brain mapping. 35*(6):2521-30.

Gravett MG, Rubens CE. (2012). A framework for strategic investments in research to reduce the global burden of preterm birth. *Am J Obstet & Gynecol.* 207(5):368-73.

Gray R, Mukherjee RAS, Rutter M. 2009. Alcohol consumption during pregnancy and its effects on neurodevelopment: what is known and what remains uncertain. *Addiction*. 104, 1270-3.

Green R. 2008. Indicators for assessing folate and vitamin B12 status and for monitoring the efficacy of intervention strategies. *Food Nutr Bull*. 29(2):S52-63.

Greenwood DC, Alwan N, Boylan S, Cade JE, Charvill J, Chipps KC, et al. 2010. Caffeine intake during pregnancy, late miscarriage and stillbirth. *Eur J Epidemiol*. 25:275–80.

Greenwood DC, Thatcher NJ, Ye J, Garrard L, Keogh G, King LG, Cade J. 2014. Caffeine intake during pregnancy and adverse outcomes: a systematic review and dose-response meta-analysis. Eu*r J Epidemiol*. 29:725-34.

Greenwood DC, Threapleton DE, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, Burley VJ. 2013. Glycemic index, glycemic load, carbohydrates, and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. Diabetes Care 36, 4166-4171.

Grote NK, Bridge JA, Gavin AR, Melville JL, Iyengar S, Katon WJ. 2010. A meta-analysis of depression during pregnancy and the risk of preterm birth, low birth weight, and intrauterine growth restriction. *Arch Gen Psychiatry*. 67(10):1012-24.

Grundlingh H, Herselman M, Iversen PO. 2013. An assessment of the implementation of the National Therapeutic Programme for pregnant women within the City of Cape Town district. S Afr Med J. 103(8):549-51.

Guelinckx I, Devlieger R, Beckers K, Vansant G. 2008. Maternal obesity: pregnancy complications, gestational weight gain and nutrition. *Obes Rev.* 9(2):140-50.

Gunnare NA, Silliman K, Morris MN. 2013. Accuracy of self-reported weight and role of gender, body mass index, weight satisfaction, weighing behavior, and physical activity among rural college students. *Body Image.* 10(3): 406-10.

Gutierrez-Mazariegos J, Theodosiou M, Campo-Paysaa1 F, Schubert M. 2011. Vitamin A: A multifunctional tool for development. *Semin Cell Dev Biol*. 22:603–10.

Haberg SE, London SJ, Stigum H, Nafstad P, Nystad W. 2009. Folic acid supplements in pregnancy and early childhood respiratory health. *Arch Dis Child*. 94:180–4.

Habib F, Habib E, Alenazy M, Nooh R. 2009. Compliance to iron supplementation during pregnancy. J Obstet Gynaecol. 29(6):487-92.

Hacker AN, Fung EB, King JC. 2012. Role of calcium during pregnancy: maternal and fetal needs. Nutr Rev. 70(7):397-409.

Hadders-Algra M. 2011. Prenatal and early postnatal supplementation with long-chain polyunsaturated fatty acids: neurodevelopmental considerations. *Am J Clin Nutr*. 94(6):1874-9.

Hagen A, Entezami M, Gasiorek–Wiens A, Albig M, Becker R, Knoll U, et al. 2011. The impact of first trimester screening and early fetal anomaly scan on invasive testing rates in women with advanced maternal age. *Ultraschall Med.* 32(3):302–6.

Haggarty P. 2014. Meeting the fetal requirement for polyunsaturated fatty acids in pregnancy. *Curr Opin Clin Nutr & Metabol Care*. 17(2):151-5.

Haider BA, Bhutta ZA. 2012. Multiple-micronutrient supplementation for women during pregnancy. *Cochrane Database of Systematic Reviews* 2012, Issue 11. Art. No.: CD004905. DOI: 10.1002/14651858.CD004905.pub3.

Haider BA, Yakoob MY, Bhutta ZA. 2011. Effect of multiple micronutrient supplementation during pregnancy on maternal and birth outcomes. *BMC Pub Health*. 11(3):19-28.

Hales C, Barker D. 2001. The thrifty phenotype hypothesis. *Br Med Bull*. 60:5–20.

Hammond KA, Litchford MD. 2012. Clinical: Inflammation, physical, and functional assessments. In *Krause's food and the nutrition care process*, 13th ed., pp.163-77 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Hammond KA. 2012. Intake: Analysis of the diet. In *Krause's food and the nutrition care process*, 13th ed., pp.129-143 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Han Z, Mulla S, Beyene J, Liao G, McDonald S. 2011. Maternal underweight and the risk of preterm birth and low birth weight: a systematic review and meta-analyses. *Int J Epidemiol*. 40:65–101.

Hankin JH, Wilkins LR, Kolonel LN, Yoshizawa CN. 1991. Validation of quantitative diet history method in Hawaii. *Am J Epidemiol*. 133:616-28.

Hanson KL, Connor LM. 2014. Food insecurity and dietary quality in US adults and children: a systematic review. *Am J clin nutr. 100*(2): 684-92.

Hanson M, Godfrey KM, Lillycrop KA, Burdge GC, Gluckman PD. 2011. Developmental plasticity and developmental origins of non-communicable disease: theoretical considerations and epigenetic mechanisms. *Prog Biophys Mol Biol*. 106: 272–80.

Hantoushzadeh S, Jafarabadi M, Khazardoust S. 2007. Serum magnesium levels, musclecramps, and preterm labor. *Int J Gynaecol Obstet*. 98: 153–154.

Harnack L, Steffen L, Arnett D, Shujun G, Luepker RV. 2004. Accuracy of estimation of large food portions. *J Am Diet Assoc.* 104:804-6.

Hart PH, Lucas RM, Walsh JP, Zosky GR, Whitehouse AJ, Zhu K, Allen KL, Kusel MM, Anderson D, Mountain JA. 2015. Vitamin D in fetal development: Findings from a birth cohort study. *Pediatrics*. 135(1):167-73.

Hassan GI, Onu AB. 2006. Total serum vitamin C concentration in pregnant women: implications for a healthy pregnancy. *Rev Bras Saúde Matern Infant.* 6(3):293-6.

Hasse JM, Matarese LE. 2012. Medical Nutrition Therapy for hepatobiliary and pancreatic disorders. In *Krause's food and the nutrition care process*, 13th ed., pp.645-74 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Hatloy A, Hallund J, Diarra MM, Oshaug A. 2000. Food variety, socioeconomic status and nutritional status in urban and rural areas in Koutiala (Mali). Pub Health Nutr. 3(1): 57-65.

Hatloy A, Torheim LE, Oshaug A. 1998. Food variety – a good indicator of nutritional adequacy of the diet? A case study from an urban area in Mali, West Africa. *Eur J Clin Nutr*. 52: 891–8.

Hattingh Z, Walsh CM, Bester CJ, Oguntibeju OO. 2008. An analysis of dietary micronutrient intakes of two age groups of black South African women. *W Indian Med J*. 57(5), 431-6.

Haugen M, Brantsaeter AL, Winkvist A, Lissner L, Alexander J, Oftedal B, Magnus P, Meltzer HM. 2014. Associations of pre-pregnancy body mass index and gestational weight gain with pregnancy outcome and postpartum weight retention: a prospective observational cohort study. *BMC Pregnancy and Childbirth.* 14(201):1-11.

Hedegaard M, Henriksen TB, Sabroe S, Secher NJ. 1993. Psychological distress in pregnancy and preterm delivery. *Brit Med J*. 307:234–9.

Heimrath J, Paprocka M, Czekanski A, Ledwozyw A, Kantor A, Dus D. 2014. Pregnancy-Induced Hypertension is accompanied by decreased number of circulating endothelial cells and circulating endothelial progenitor cells. *Arch Immunol Ther Exp*. 62:353-6.

Heird WC, Lapillonne A. 2005. The role of essential fatty acids in development. *Annu Rev Nutr*. *25:* 549-71.

Helgstrand S, Andersen AN. 2005. Maternal underweight and the risk of spontaneous abortion. *Acta Obstet Gynecol Scand*. 84(12):1197-201.

Hercberg S, Preziosi P, Galan P. 2001. Iron deficiency in Europe. *Pub Health Nutr*. 4(2B): 537-45.

Hernandez T, Wilder L, Kuehn D, Rubotzky K, Moser-Veillon P, Godwin S et al. 2006. Portion size estimation and expectation of accuracy. *J Food Comp Anal*. 19(suppl): S14-S21.

Heude B, Thiébaugeorges O, Goua V, Forhan A, Kaminski M, Foliguet B, Schweitzer M, Magnin G, Charles M & EDEN Mother–Child Cohort Study Group. 2012. Pre-pregnancy body mass index and weight gain during pregnancy: relations with gestational diabetes and hypertension, and birth outcomes. *Maternal & child health J*. 16(2):355-63.

Hibbeln JR, Ferguson TA, Blasbalg TL. 2006. Omega-3 fatty acid deficiencies in neurodevelopment, aggression and autonomic dysregulation: opportunities for intervention. *Int Rev Psychiatry*. 18:107–18.

Hibberts F, Schizas A. 2010. Assessment and treatment of patients with haemorrhoids. *Nursing Standard. 24*(18): 51.

Hillbruner C, Egan R. 2008. Seasonality, household food security, and nutritional status in Dinajpur, Bangladesh. *Food Nutr Bull*. 29: 221-231.

Hoellen F, Hornemann A, Haertel C, Reh A, Rody A, Schneider S, Tuschy B, Bohlmann MK. 2014. Does maternal underweight prior to conception influence pregnancy risks and outcome? *In Vivo*. 28(6): 1165-70.

Hofmeyer G, Lawrie TA, Atallah ÁN, Duley L, Torloni MR. 2014. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. Cochrane Database of Systematic Reviews. Issue 6. Art. No.: CD001059. DOI: 10.1002/14651858.CD001059.pub4.

Hofmeyer GJ, Lawrie TA, Atallah ÁN, Duley L. 2010. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. Cochrane Database Syst Rev. 8(8).

Hoppe C, Molgaard C, Thomsen BL, Juul A, Michaelsen KF. 2004. Protein intake at 9 mo of age is associated with body size but not body fat in 10-y-old Danish children. *Am J Clin Nutr.* 79(3):494-501.

Horrobin DF, Bennett CN. 1999. Depression and bipolar disorder: relationships to impaired fatty acid and phospholipid metabolism and to diabetes, cardiovascular disease, immunological abnormalities, cancer, ageing and osteoporosis: possible candidate genes. *Prostaglandins Leukort Ess*. 60(4):217–34.

Hoyert DL Mathews TJ, Menacker F, Strobino DM, Guyer B. 2006. Annual summary of vital statistics: 2004. *Pediatrics*. 117(1):68.

Hronek M, Kudlácková Z. 2005. Deficient intake of nutrients and the resulting health complications in vegetarians in the course of pregnancy and lactation. *Ceska Gynekol*. 70(2):161-4.

Hronek M, Zadak Z, Hrnciarikova D, Hyspler R, Ticha A. 2009. New equation for the prediction of resting energy expenditure during pregnancy. *Nutrition.* 25:947-53.

Hsu W, Wu C, Hsieh CTC, Lo H, Lin J, Kao M. 2013. Low body weight gain, low white blood cell count and high serum ferritin as markers of poor nutrition and increased risk for preterm delivery. *Asia Pac J Clin Nutr*. 22 (1):90-9.

Huang L, Sauve R, Birkett N, Fergusson D, van Walraven C. 2008. Maternal age and risk of stillbirth: a systematic review. Can Med Assoc J. 178(2):165-72.

Huizinga MM, Carlisle AJ, Cavanaugh KL, Davis DL, Gregory RP, Schlundt DG et al. 2009. Literacy, numeracy, and portion-size estimation skills. *Public Health Nutr*. 36(4):324-8.

Hulshoff HE, Hoek HW, Susser E, Brown AS, Dingemans A, Schnack HG. 2000. Prenatal exposure to famine and brain morphology in schizophrenia. *Am J Psychiatry*. 157(7):1170–2.

Hutson JR, Stade B, Lehotay DC, Collier CP, Kapur BM. 2012. Folic acid transport to the human fetus is decreased in pregnancies with chronic alcohol exposure. *PLoS ONE*. 7(5): e38057. doi:10.1371/journal.pone.0038057

Huybregts L, Roberfroid D, Lachat C, Van Camp J, Kolsteren P. 2007. Validity of photographs for food portion estimation in a rural West African setting. *Public Health Nutr.* 11(6):581-7.

Hypponen E. Vitamin D for the prevention of preeclampsia? A hypothesis. 2005. *Nutr Rev*. 63:225

Imdad A, Bhutta ZA. 2011. Effect of balanced protein energy supplementation during pregnancy on birth outcomes. *BMC Pub Health*. 11(Suppl 3):S17.

Imdad A, Bhutta ZA. 2012. Effect of calcium supplementation during pregnancy on maternal, fetal and birth outcomes. *Paediatr Perinat Epidemiol*. 26(1):138-52.

Imdad A, Bhutta ZA. 2012b. Maternal nutrition and birth outcomes: effect of balanced protein-energy supplementation. *Paediatr Perinat Epidemiol*. 26:178-90.

Indumati V, Kodliwadmath MV, Sheela MK. 2011. The role of serum electrolytes in pregnancy induced hypertension. J Clin Diagnostic Res, 5, 66-9.

Iniguez G, Ong K, Bazaes R, Avila A, Salazar T, Dunger D, et al. 2006. Longitudinal changes in insulin-like growth factor-I, insulin sensitivity, and secretion from birth to small-for-gestational-age children. *J Clin Endocrinol Metab*. 91:4645–9.

Innis SM. 2007. Dietary (n-3) fatty acids and brain development. *J Nutr.* 137: 855–9.

Innis SM. 2009. Omega-3 Fatty acids and neural development to 2 years of age: do we know enough for dietary recommendations? *J Paediatr Gasteroenterol Nutr*. 48, Suppl 1: S16–S24.

Institute of Medicine of the National Academies. 2002. Dietary Reference Intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, D.C. *National Academy Press.*

Institute of Medicine of the National Academies. 2005. Dietary Reference Intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, D.C. *National Academy Press.*

Institute of Medicine. 2000. Dietary Reference Intakes: Applications in Dietary Assessment, Washington, D.C. *National Academies Press*.

Institute of Medicine. 2003. Dietary Reference Intakes: Applications in Dietary Planning. Washington, D.C. *National Academies Press*.

Institute of Medicine. 2006. Part II: Energy, macronutrients, water and physical activity; Protein and amino acids. pp. [Otten JJ, Hellwig JP, Meyers 144-55. LD, editors]. Washington, DC. *National Academy press*.

Institute of Medicine. 2009. Weight gain during pregnancy: Reexamining the guidelines. Washington, DC. *National Academy press*.

Ireton-Jones CS. 2012. Intake: Energy. In *Krause's food and the nutrition care process*, 13th ed., pp.19-31 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Izquierdo-Alvarez S, Castanon SG, Ruata ML, Aragues EF et al. 2007. Updating of normal levels of copper, zinc and selenium in serum of pregnant women. *J Trace Elem Med Biol.* 21(1): 49-52.

Jacques PF, Sulsky IS, Sadowski JA, Phillips JCC, Rush D, Willett WC. 1993. Comparison of micronutrient intake measured by a dietary questionnaire and biochemical indicators of micronutrient status. *Am J Clin Nutr*. 57:182-9.

Jaffer S, Swart EC, Jackson D. 2008. An assessment of the dietary intake of pregnant women in the West Coast/ Winelands region, Western Cape Province: Relation to low birth weight. Thesis.

Jain S, Sharma P, Kulshreshtha S, Mohan G, Singh S. 2010. The role of calcium, magnesium and zinc in pre-eclampsia. *Biol Trace Elem Res*. 133(2):162-70.

James WP, Mascie-Taylor GC, Norgan NG, Bistrian BR, Shetty PS, Ferro-Luzzi A. 1994. The value of arm circumference measurements in assessing chronic energy deficiency in Third World adults. *Eur J Clin Nutr*. 48:883–94.

Jansson T, Cetin I, Powell TL. 2006. Placental transport and metabolism in fetal overgrowth - a workshop report. *Placenta*. 27(suppl A):S109-S13.

Jeric M, Roje D, Medic N, Strinic T, Mestrovic Z, Vulic M. 2012. Maternal pre-pregnancy underweight and fetal growth in relation to institute of medicine recommendations for gestational weight gain. *Early Hum Dev.* 89(5):277-81.

Johnson K, Posner SF, Biermann J, Cordero JF, Atrash HK, Parker CS, et al. 2006. Recommendations to improve preconception health and health care — United States. A report of the CDC/ATSDR Preconception Care Work Group and the Select Panel on Preconception Care. *MMWR Recomm Rep*. 55(RR-6):1–23.

Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH. 2009. Dietary sugars intake and cardiovascular health: A scientific statement from the American Heart Association. Circulation. 120(11):1011–20.

Jones A, Lozoff B. 2014. Overweight and obesity in early pregnancy are associated with porer maternal iron status and inhibited placental iron transfer. In *142nd APHA Annual Meeting and Exposition* (November 15-November 19, 2014). APHA.

Jones CW. 2001. Gestational diabetes and its impact on the neonate. *Neonatal Netw*. 20:17–23.

Joseph KS, Allen AC, Dodds L, Turner LA, Scott H, et al. 2005. The perinatal effects of delayed childbearing. *Obstet Gynecol*. 105: 1410–1418.

Kaiser LL, Allen LH. 2002. Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome. *J Am Diet Assoc*. 102(10):1479-90.

Kaiser LL, Allen LH. 2008. Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome. *J Am Diet Assoc*.108:553-61.

Kaiser LL, Campbel CG. 2014. Practice paper of the Academy of Nutrition and Dietetics abstract: Nutrition and Lifestyle for a healthy pregnancy outcome. *J Academy Nutr & Dietet.* 114 (9):1447.

Kajantie E, Osmond C, Barker DJ, Forsen T, Phillips DI, Eriksson JG. 2005. Size at birth as a predictor of mortality in adulthood: a follow-up of 350 000 person-years. *Int J Epidemiol*. 34(3): 655–63.

Kass L, Weekes J, Carpenter L. 2012. Effect of magnesium supplementation on blood pressure – a meta-analysis. *Eur J Clin Nutr* 66:411–8.

Kawai K, Spiegelman D, Shankar AH, Fawzi WW. 2011. Maternal multiple micronutrient supplementation and pregnancy outcomes in developing countries: meta-analysis and meta-regression. *Bull World Health Organ*. 89(6).

Keen CL, Uriu-Adams JY, Skalny A, Grabeklis A, Grabeklis S, Green K et al. 2010. The plausibility of maternal nutritional status being a contributing factor to the risk for fetal alcohol spectrum disorders: The potential influence of zinc status as an example. *Biofactors*. 36(2): 125–35.

Kelly A, Kevany J, de Onis M. Shah P. 1996. A WHO collaborative study of maternal anthropometry and pregnancy outcomes. *Int J Gynecol Obstet*. 53(3):219–33.

Kennedy DA, Stern SJ, Moretti M, Matok I, Moumita S, Nickel C et al. 2011. Folate intake and the risk of colorectal cancer: A systematic review and meta-analysis. 35:2-10.

Kennedy G, Ballard T, Dop MC. 2012. Guidelines for measuring household and individual dietary diversity. *Food and Agriculture Organization of the United Nations.*

Kenny LC, Lavender T, McNamee R, O'Neill SM, Mills T, et al. 2013. Advanced maternal age and adverse pregnancy outcome: Evidence from a large contemporary cohort. PLoS ONE. 8(2): e56583. doi:10.1371/journal.pone.0056583

Kesa H, Oldewage-Theron W. 2005. Anthropometric indications and nutritional intake of women in the Vaal Triangle, South Africa. *Pub Health.* 119(4):294-300.

Key TJ, Spencer EA. 2007. Carbohydrates and cancer: an overview of the epidemiological evidence. *Eur J Clin Nutr.* 61 Suppl 1:S112-S121.

Khan Y, Tisman G. 2010. Pica in iron deficiency: a case series. *J Med Case Rep*. 4(1):86.

Khashan AS, Kenny LC. 2009. The effects of maternal body mass index on pregnancy outcome. *Europ J Epidemiol*. 24(11):697–705.

Khashu M, Narayanan M, Bhargava S, Osiovich H. 2009. Perinatal outcomes associated with preterm birth at 33 to 36 weeks'gestation: a population-based cohort study. *Pediatrics*. 123(1):109–13.

King JC. 2000. Determinants of maternal zinc status during pregnancy. *Am J Clin Nutr*. 71(suppl):1334S–43S.

Kirkpatrick SI, Subar AF, Douglass D, Zimmerman TP, Thompson FE, Kahle LL, George SM, Dodd KW, Potischman N. 2014. Performance of the automated self-administered 24-hour recall relative to a measure of true intakes and to an interviewer-administered 24-h recall. *Am J Clin Nutr*.

Klemmensen A , Tabor A, Østerdal M, Knudsen V, Halldorsson T, Mikkelsen T, Olsen S. 2009. Intake of vitamin C and E in pregnancy and risk of pre-eclampsia: prospective study among 57 346 women. Brit J Obstet Gynaec. 116:964–74.

Klinger I. 2004. The nutritional status of pregnant women in relation to alcohol consumption during pregnancy, and pregnancy outcome. Thesis

Kodituwakku P, Kodituwakku EL. 2013. Fetal Alcohol Syndrome. In *Neuroscience in the 21st century*, 1st ed., pp2411-30 [Pfaff DW, editor]. Springer New York.

Kodituwakku PW. 2009. Neurocognitive profile in children with fetal alcohol spectrum disorders. *Dev Disabil Res Rev*. 15:218-24.

Koletzko B, Bauer CP, Bung P, Cremer M, Flothkötter M, Hellmers C et al. 2012. [Nutrition in pregnancy - Practice recommendations of the Network "Healthy Start - Young Family Network"]. [Article in German]. *Dtsch Med Wochenschr*. 137(25-26):1366-72.

Koletzko B, Boey CCM, Campoy C, Carlson SE, Chang N, Guillermo-Tuazon MA, Joshi S, Prell C, Quak SH, Sjarif DR, Su Y, Supapannachart S, Yamashiro Y, Osendarp SJM. 2014. Current information and Asian perspectives on Long-Chain Polyunsaturated Fatty Acids in pregnancy, lactation, and infancy: Systematic review and practice recommendations from an early nutrition academy workshop. *Ann Nutr Metabol*. 65:49-80.

Koletzko B, Cetin I, Brenna JT. 2007. Consensus Statement: Dietary fat intakes for pregnant and lactating women. *Brit J Nutr*. 98:873-7.

Kotha VK, De Souza A. 2013. Wernicke's Encephalopathy following hyperemesis gravidarum: a report of three cases. The neuroradiology journal, 26(1), 35-40.

Kramer MS, Morin I, Yang H, Platt RW, Usher R, McNamara H et al. 2002. Why are babies getting bigger? Temporal trends in fetal growth and its determinants. *J Pediatr*.141:538-42.

Kramer MS, Seguin L, Lydon J, Goulet L. 2000. Socio-economic disparities in pregnancy outcome: why do the poor fare so poorly?. Paediatr & perinat epidemiol. 14(3):194-210.

Kris-Etherton PM, Innis S. 2007. Position of the American Dietetic Association and Dietitians of Canada: Dietary fatty acids. *J Am Diet Assoc.* 107(9):1599–611.

Kristal AR, Feng Z, Coates RJ, Oberman A, George V. 1997. Associations of race/ethnicity, education, and dietary intervention with the validity and reliability of a food frequency questionnaire: The women's health trial feasibility study in minority populations. *Am J Epidemiol*. 146: 856-69.

Kruger HS. 2005. Maternal anthropometry and pregnancy outcomes: a proposal for the monitoring of pregnancy weight gain in outpatient clinics in South Africa. *Curationis*. 28(4):40–9.

Kumar A, Devi SG, Batra S, Singh C, Shukla DK. 2009. Calcium supplementation for the prevention of pre-eclampsia. *Int J Gynecol Obstet*. 104(1):32-6.

Kurzel RB. 1991. Serum magnesium levels in pregnancy and preterm labor. *Am J Perinatol.* 8:119-27.

Labadarios D, Mchiza ZJR, Steyn NP, Gericke G, Maunder EMW, Davids YD, Parker W. 2007. Food security in SOuth Africa: a review of national surveys. Bull World Health Organ. 89(12). Genebra.

Labadarios D, Steyn NP, Nel J. 2011. How diverse is the diet of adult South AFricans? *Nutr J*. 10:33.

Lakin V, Haggarty P, Abramovich DR. 1998. Dietary intake and tissue concentrations of fatty acids in omnivore, vegetarian, and diabetic pregnancy. *Prost Leuk Ess Fatty Acids*. 58:209-220.

Lambe S, Mahajan B, Muddeshwar M. 2014. Comparitive study of serum calcium, magnesium and zinc levels in preeclampsia and normal pregnancy. *Int J Recent trends & Tech*. 9(3):422-6.

Lampl M, Jeanty P. 2004. Exposure to maternal diabetes is associated with altered fetal growth patterns: A hypothesis regarding metabolic allocation to growth under hyperglycemic-hypoxemic conditions. *Am J Hum Biol*. 6:237-63.

Landon MB, Spong CY, Thorn E, Carpenter MW, Ramin SM, Casey B et al. 2009. A multicenter, randomized trial of treatment for mild gestational diabetes. *N Engl J Med*. 361:1339-48.

Langley-Evans SC. 2014. Nutrition in early life and the programming of adult disease: a review. *J Hum Nutr Diet*. 28(Suppl. 1):1-14.

Laopaiboon M, Lumbiganon P, Intarut N, Mori R, Ganchimeg T, Vogel JP, Souza JP, Gulmezoglu AM. 2014. Advanced maternal age and pregnancy outcomes: a multicountry assessment. *BJOG – Int J Obstet Gy*. 121(1):49-56.

Larsson SC, Akesson A, Bergkvist L, Wolk A. 2010. Multivitamin use and breast cancer incidence in a prospective cohort of Swedish women. Am J Clin Nutr. 91(5):1268-72.

Laura HC, Menezes AB, Noal RB, Hallal PC, Araújo CL. 2010. Maternal anthropometric characteristics in pregnancy and blood pressure among adolescents: 1993 live birth cohort, Pelotas, southern Brazil. *BMC Pub Health*. 10(1):434-41.

Lawlor DA, Smith GD, O'Callaghan M, Alati R, Mamun AA, Williams GM et al. 2007. Epidemiologic evidence for the fetal overnutrition hypothesis: findings from the Mater-University study of pregnancy and its outcomes. *Am J Epidemiol*;

Lederman SA. 1985. Physiological changes of pregnancy and their relation to nutrient needs. *Curr Concepts Nutr*. 14:13-43

Lee RD, Nieman DC. 2010. Anthropometry. In *Nutritional Assessment*, 5th ed. [Lee RD, Nieman DC, editors] McGraw Hill.

Leeds M, Riyazi N, de Vries JI, Jakobs C, van Geijn HP, Dekker GA. 1998. Effects of folic acid and vitamin B6 supplementation on women with hyperhomocysteinemia and a history of preeclampsia or fetal growth restriction. *Am J Obstet Gynecol*. 179:135–9.

Lewis B, SIamusantu W, Dyer B, Talegawkar S, Klemm R, Schulze K, Palmer A, West K. 2014. Child dietary intake data by tablet-based 24-hour recall in rural Zambia. *FASEB J*. 28(1).

Ley SH, Hanley AJ, Sermer M, Zinman B, O'Connor DL. 2013. Lower dietary vitamin E intake during the second trimester is associated with insulin resistance and hyperglycemia later in pregnancy. *Euro J Clin Nutr*. 67(11):1154-6.

Li K, Kaaks R, Linseisen J, Rohrmann S. 2012. Associations of dietary calcium intake and calcium supplementation with myocardial infarction and stroke risk and overall cardiovascular mortality in the Heidelberg cohort of the European Prospective Investigation into Cancer and Nutrition study (EPIC-Heidelberg). *Heart*. 98: 920-5.

Licitra L, Pregazzi R, Troiano L, Guaschino S. 1998. Anthropometric skin-fold and impedance measurement assessment of the maternal nutritional status at the end of pregnancy. Comments deduced from a study of 54 cases. *Minerva Ginecologica*. 50:379–82.

Lieber CS. 2005. Metabolism of alcohol. *Clinics in liver disease. 9*(1): 1-35.

Liljestrand J, Bergström S. 1991. Antenatal nutritional assessment: the value of upper arm circumference. *Gynecol Obstet Inves*. 32:81–3.

Lindheimer M, Barron WM. 1998. Renal function and volume homeostasis. In *Principles and Practice of Medical Therapy in Pregnancy*, 3rd ed. [Gleicher N, Buttino L, Elkayam U, editors] Stanford, CT: Appleton and Lange, pp. 1043–52.

Litchford MD. 2012. Clinical: Biochemical assessment. In *Krause's food and the nutrition care process*, 13th ed., pp.191-208 [Alexopoulos Y, Frazier DM, editors]. Elsevier.

Liu LX, Arany Z. 2014. Maternal cardiac metabolism in pregnancy. *Cardiovasc Res.* 101: 545–53.

Loane M, Dolk H, Morris JK, EUROCAT Working Group. 2009. Maternal age-specific risk of non-chromosomal anomalies. *Brit J Obstet Gynaecol.* 116(8):1111–9.

Lof M, Olausson H, Bostrom K, Janerot-Sjo¨berg B, Sohlstrom A, Forsum E. 2005. Changes in basal metabolic rate during pregnancy in relation to changes in body weight and composition, cardiac output, insulin-like growth factor I, and thyroid hormones and in relation to fetal growth. *Am J Clin Nutr.* 81:678–85.

Loomans EM, Hofland L, van der Stelt O, van der Wal MF, Koot HM, Van den Bergh BRH et al.2012. Caffeine intake during pregnancy and risk of problem behavior in 5- to 6-year-old children. *Pediatrics*. 130(2):e305-13.

López LB, Calvo EB, Poy MS, del Valle Balmaceda Y, Cámera K. 2011. Changes in skinfolds and mid-upper arm circumference during pregnancy in Argentine women. Matern Child Nutr. 7(3):253–62.

Louie JCY, Markovic TP, Perera N, Foote D, Petocz P, Ross GP et al. 2011. A randomized controlled trial investigating the effects of a low–glycemic index diet on pregnancy outcomes in gestational diabetes mellitus. *Diabetes Care.* 34(11):2341-6

Lozoff B, Beard J, Connor J, Barbara F, Georgieff M, Schallert T. 2006. Long-lasting neural and behavioral effects of iron deficiency in infancy. *Nutr Rev*. 64(Suppl):S34–S43.

Lumish RA, Young SL, Lee S, Cooper E, Pressman E, Guillet R, O'Brien KO. 2014. Gestational iron deficiency is associated with Pica behaviors in adolescents. *J Nutr*. 144(10):1533-9.

Luo ZC, Wilkins R, Kramer MS. 2006. Effect of neighbourhood income and maternal education on birth outcomes: a population-based study. Canadian Med Assoc J. 174(10): 1415-20.

Lutsey PL, Dawe D, Villate E, Valencia S, Lopez O. 2008. Iron supplementation compliance among pregnant women in Bicol, Philippines. Pub health nutr. 11(1), 76.

Ma Y, Olendzki BC, Pagoto SL, Hurley TG, Magner RP, Ockene IS et al. 2009. Number of 24-hour diet recalls needed to estimate energy intake. *Ann Epidemiol*. 19:553–9.

MacDorman MF, Kirmeyer S. 2009. Fetal and perinatal mortality, United States, 2005. Centers for Disease Control and Prevention, National Center for Health Statistics, Division of Vital Statistics. 57(8):1-19.

Maddalozzo GF, Turner RT, Edwards CHT, Howe KS, Widrick JJ, Rosen CJ et al. 2009. Alcohol alters whole body composition, inhibits bone formation, and increases bone marrow adiposity in rats. *Osteoporos Int*. 20:1529–38.

Magee LA, Abalos E, von Dadelszen P, Sibai B, Easterling T, Walkinshaw S. 2011. How to manage hypertension in pregnancy effectively. *Br J Clin Pharmacol*. 72:3; 394–401.

Mahadevana U, Kaneb S. 2006. American gastroenterological association institute medical position statement on the use of gastrointestinal medications in pregnancy. *Gastroenterology*. 131(1): 278–82.

Mahon P, Harvey N, Crozier S, Inskip H, Robinson S, Arden N et al. Study Group SWS. 2010. Low maternal vitamin D status and fetal bone development: cohort study. *J Bone Miner Res*. 25:14–9.

Malik P, Gasser RW, Kemmler G, Moncayo R, Finkenstedt G, Kurz M, Fleischhacker WW. 2009. Low bone mineral density and impaired bone metabolism in young alcoholic patients without liver cirrhosis: A cross-sectional study. *Alcohol Clin Exp Res*. 33: 375–81.

Malik VS, Popkin BM, Bray GA, Despres JP, Willett WC, Hu FB. 2010. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care. 33(11):2477–83.

Mamabolo RL, Alberts M, Levitt NS, Delemarre-van de Waal HA, Steyn NP. 2007. Prevalence of gestational diabetes mellitus and the effect of weight on measures of insulin secretion and insulin resistance in third-trimester pregnant rural women residing in the Central Region of Limpopo Province, South Africa. *Diabet Med*. 24:233–9.

Mamabolo RL, Alberts M, Steyn NP, Delemarre-van de Waal HA, Nthangeni NG, Levitt NS. 2004. Evaluation of the effectiveness of iron and folate supplementation during pregnancy in a rural area of Limpopo province. *S Afr J Clin Nutr.*17(1):15-22.

Mamun AA, Callaway LK, O'Callaghan MJ, Williams GM, Najman JM, Alati R et al. 2010. Associations of maternal pre-pregnancy obesity and excess pregnancy weight gains with adverse pregnancy outcomes and length of hospital stay. *BMC Pregnancy Childbirth*. 62(11):1-9.

Mamun AA, Mannan M, Doi SAR. 2014. Gestational weight gain in relation to offspring obesity over the life course: a systematic review and bias-adjusted meta-analysis. *Obes Rev*. 15(4):338-47.

Mangels AR. 2008. Vegetarian diets in pregnancy. In *Handbook of Nutrition and pregnancy*. [Lammi-Keefe CJ, Couch SC, Philipson EH, editors] Humana Press, pp. 215-32.

Mann J, Truswell AS. 2002. Essentials of Human Nutrition. Oxford University Press: Oxford.

Mann JI. 2007. Dietary carbohydrate: relationship to cardiovascular disease and disorders of carbohydrate metabolism. *Eur J Clin Nutr*. 61 Suppl 1:S100-S111.

Manning MA, Hoyme HE. 2007. Fetal alcohol spectrum disorders: a practical clinical approach to diagnosis. *Neurosci Biobehav Rev*. 31(2):230–8.

March of Dimes, PMNCH, Save the Children, WHO. 2012. Born too soon: The global action report on preterm birth. [Howson CP, Kinney MV, Lawn JE, editors] World Health Organisation. Geneva.

Margann EF Doherty DA, Chauhan SP, Barrilleaux SP, Verity LA, Martin JN et al. 2003. Effect of maternal hydration on amniotic fluid volume. *Obstet Gynecol* 101:1261-5.

Margerison Zilko CE, Rehkopf D, Abrams B. 2010. Association of maternal gestational weight gain with short- and long-term maternal and child health outcomes. *Am J Obstet Gynecol.* 202:574.e1-8.

Martorell R, Stein AD, Schroeder DG. 2001. Early nutrition and later adiposity. *J Nutr*. 131:874S–80S.

Matthews A, Dowswell T, Haas DM, Doyle M, O'Mathúna DP. 2010. Interventions for nausea and vomiting in early pregnancy. *Cochrane Database of Systematic Reviews*. 9. Art. No.: CD007575. DOI: 10.1002/14651858.CD007575.pub2.

Matthews A, Haas DM, O'Mathúna DP, Dowswell T, Doyle M. 2014. Interventions for nausea and vomiting in early pregnancy. Cochrane Database of Systematic Reviews. 3. Art. No.: CD007575. DOI: 10.1002/14651858.CD007575.pub3.

May PA, Blankenship J, Marais AS, Gossage JP, Kalberg WO, Joubert B, *et al.* 2013. Maternal alcohol consumption producing fetal alcohol spectrum disorders (FASD): quantity, frequency, and timing of drinking. *Drug Alcohol Depend.* 133:502–12.

May PA, Blankenship J, Marais AS, Gossage JP, Kalberg WO, Barnard R, *et al.* 2013b. Approaching the prevalence of the full spectrum of fetal alcohol spectrum disorders in a South African population-based study. *Alcohol Clin Exp Res.* 37:818–30.

May PA, Brooke L, Gossage JP, Croxford J, Adnams C, Jones KL et al. 2000. Epidemiology of fetal alcohol syndrome in a South African community in the Western Cape Province. *Am J Public Health*. 90:1905–12.

May PA, Gossage JP, Marais AS, Adnams CM, Hoyme HE, Jones KL et al. 2007. The epidemiology of fetal alcohol syndrome and partial FAS in a South African community. *Drug Alcohol Depend.* 88:259-71.

May PA, Gossage JP, White-Country M, Goodheart K, Decoteau S, Trujillo PM et al. 2004. Alcohol consumption and other maternal risk factors for fetal alcohol syndrome among three distinct samples of women before, during, and after pregnancy: the risk is relative. *Am J Med Genet.* 127C: 10-20.

Mayer-Davis EJ, Vitolins MZ, Carmichael SL, Hemphill S, Tsaroucha G, Rushing J et al. 1999. Validity and reproducibility of a food frequency interview in a multi-cultural epidemiologic study. *Ann Epidemiol*. 9:314-24.

McArdle HJ, Gambling L, Kennedy C. 2014. Iron deficiency during pregnancy: the consequences for placental function and fetal outcome. *Proceedings of the Nutrition Society. 73*(01): 9-15.

McDonald SD, Han Z, Mulla S, Beyene J. 2010. Overweight and obesity in mothers and risk of preterm birth and low birth weight infants: Systematic review and meta-analyses. *Brit Med J.* 341:c3428.

McGowan CA, McAuliffe FM. 2012. Maternal nutrient intakes and levels of energy underreporting during early pregnancy. *Eur J Clin Nutr.* 66, 906–13.

McGuire S. 2011. US Department of Agriculture and US Department of Health and Human Services, Dietary Guidelines for Americans, 2010. Washington, DC: US Government Printing Office, January 2011. *Adv nutr: Int rev J*. 2(3): 293-4.

McMillen IC, Robinson JS. 2005. Developmental origins of the metabolic syndrome: prediction, plasticity, and programming. *Physiol Rev*. 85:571–633.

Mehta SH, Kruger M, Sokol RJ. 2011. Being too large for gestational age precedes childhood obesity in African Americans. *Am J Obstet Gynecol.*204:265.e1-5.

Meis PJ, Michielutte R, Peters TJ. 1995. Factors associated with preterm birth in Cardiff, Wales. I. Univariable and multivariable analysis. *Am J Obstet Gynecol.*173:590–596.

Mennitti LV, Oliveira JL, Morais CA, Estadella D, Oyama LM, do Nascimento CMO, Pisani LP. 2015. Type of fatty acids in maternal diets during pregnancy and/or lactation and metabolic consequences of the offspring. *The Journal of nutritional biochemistry. 26*(2): 99-111.

Merewood A, Mehta SD, Chen TC, Bauchner H, Holick MF. 2009. Association between vitamin D deficiency and primary cesarean section. *J Clin Endocrinol Metab*. 94:940–5.

Metzger BE, Buchanan TA, Coustan DR, de Leiva A, Dunger DB, Hadden DR et al. 2007. Summary and recommendations of the fifth international workshop-conference on gestational diabetes mellitus. *Diabetes care*. 30(2):s251-60.

Miao D, Young SL, Golden CD. 2014. A meta-analysis of pica and micronutrient status. *Am J Human Biol*. 27(1):84-93.

Millen AE, Bodnar LM. 2008. Vitamin D assessment in population-based studies: a review of the issues. *Am J Clin Nutr*. 87:1102S–5S.

Mills TA, Lavender T. 2010. Advanced maternal age. *Obstet Gynaecol Reprod Med*. 2(4):107-11.

Miyake Y, Tanaka K, Okubo H, Sasaki S, Arakawa M. 2014. Intake of dairy products and calcium and prevalence of depressive symptoms during pregnancy in Japan: a cross-sectional study. *Brit J obstet Gynae*. 1-8.

Mock DM, Stadler DD, Stratton SL, Mock NI. 1997. Biotin status assessed longitudinally in pregnant women. *J Nutr.*127(5):710Y716.

Mock DM. 2013. Biotin. In: Zempleni JS, Suttie JW, Gregory JF, Stover PJ, editors. Handbook of vitamins. 5th ed. Boca Raton (FL): CRC Press: p. 397–420.

Mock DM. 2014. Adequate intake of biotin in pregnancy: why bother?. J Nutr. 144(12):1885-6.

Mock DM. 2009. Marginal biotin deficiency is common in normal human pregnancy and is highly teratogenic in the mouse. *J Nutr*. 139:154–7.

Modjadji SEP, Alberts M, Mamabolo RL. 2007. Folate and iron status of South African non-pregnant rural women of childbearing age, before and after fortification of foods. *S Afr J Clin Nutr*. 20(3):89-93.

Moertl MG, Ulrich D, Pickel KI, Klaritsch P, Schaffer M, Flotzinger D et al. 2009. Changes in haemodynamic and autonomous nervous system parameters measured non-invasively throughout normal pregnancy. *Eur J Obstet Gyn R B*. 144S:S179-83.

Molloy AM, Kirke PN, Brody LC, Scott JM, Mills JL. 2008. Effects of folate and vitamin B12 deficiencies during pregnancy on fetal, infant, and child development. *Food Nutr Bull*. 29 (11)(suppl. 1):101-11.

Moodley J. 2011. Maternal deaths associated with hypertension in South Africa: Lessons to learn from the Saving Mothers report, 2005–2007. *Cardiovasc J Afr*. 22:31–5.

Moore KL, Persaud TVN. 1998. The Developing Human: Clinically Oriented Embryology, 6th ed, pp. 548 [Saunders WB, editor]. Philadelphia.

Moran VH. 2007. A systematic review of dietary assessments of pregnant adolescents in industrialised countries. *Brit J Nutr*. 97:411–25.

Moreira P, Padez C, Moura˜o-Carvalhal I, Rosado V. 2007. Maternal weight gain during pregnancy and overweight in Portuguese children. *Int J Obesity*. 31:608–14.

Morgane JP, MoklerDJ, Galler JR. 2002. Effects of prenatal protein malnutrition on the hippocampal formation. Neurosci & Biobehav Rev. 26(4):471-83.

Morgane PJ, Austin-LaFrance RJ, Bronzino J, Tonkiss J, Diaz-Cintra S, Cintra L, Kemper T, Galler JR. 1993. Prenatal malnutrition and development of the brain. Neurosci Biobehav Rev. 17:91–128

Mori R, Ota E, Middleton P, Tobe-Gai R, Mahomed K, Bhutta ZA. 2012. Zinc supplementation for improving pregnancy and infant outcome. *Cochrane Database of Systematic Reviews*. Issue 7. Art. No.: CD000230. DOI: 10.1002/14651858.CD000230.pub4

Morkbak AL, Hvas AM, Milman N, Nexo E. 2007. Holotranscobalamin remains unchanged during pregnancy. Longitudinal changes of cobalamins and their binding proteins during pregnancy and postpartum. *Haematologica*. 92(12):1711–2.

Mose T, Kjaerstad MB, Mathiesen L, Nielsen JB, Edelfors S, Knudsen LE. 2008. Placental passage of benzoic acid, caffeine, and glyphosate in an ex vivo human perfusion system. *J Toxicol Environ Health A*. 71(15):984–91.

Moss BG, Chugani DC. 2014. Increased risk of very low birth weight, rapid postnatal growth, and autism in underweight and obese mothers. *Am J Health Promot*. 28(3):181-8.

Mostert D, Steyn NP, Temple NJ, Olwagen R. 2005. Dietary intake of pregnant women and their infants in a poor black South African community. *Curationis*. 28(4):12-9.

Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. 2011. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 364(25):2392-404.

Muhlhausler BS, Gibson RA, Makrides M. 2010. Effect of long-chain polyunsaturated fatty acid supplementation during pregnancy or lactation on infant and child body composition: a systematic review. *Am J Clin Nutr*. 92:857–63.

Murphy SP, Allen LH. 2003. Nutritional importance of animal source foods. *J Nutr*. 133(11 Suppl 2):3932S-3935S.

Murphy SP, Guenther PM & Kretsch MJ. 2006. Commentary: Using the Dietary Reference

Murphy SP, Poos MI. 2002. Dietary Reference Intakes: Summary of applications in dietary assessment. *Pub Health Nutr*. 5(6A):843-9.

Napier C, Oldewage-Theron W. 2015. Dietary intake and nutritional status of adolescent girls and young women in Durban, South Africa. *J Family Ecol & Consumer Sci*. 43.

Narmaki E, Koohdani F, Qorbani M, Shiraseb F, Ataie-Jafari A, Sotoudeh G. 2015. Dietary diversity as a proxy measure of blood antioxidant status in women. *Nutrition*.

Naude C. 2013. "Eat plenty of vegetables and fruit every day": a food-based dietary guideline for South Africa. *S Afr J Clin Nutr*. 26(3) (Supplement):S46-S56.

Naude C. 2013b. Would an increase in vegetable and fruit intake help to reduce the burden of nutrition-related disease in South Africa? An umbrella review of the evidence. S Afr J Clin Nutr. 26(3):104-14.

Naude CE, Carey PD, Laubscher R, Fein G, Senekal M. 2012. Vitamin D and calcium in South African adolescents with alcohol use disorders. Nutrients. 4:1076-94.

Naude CE. 2012. Heavy alcohol use in adolescents: Potential influences on nutritional status. Thesis.

Naude CE. 2012b. Vitamin D: The sunshine nutrient. The Nutrition Association of South Africa. http://www.nutritionsociety.co.za/index.php/11-useful-information/44-vitamin-d-the-sunshine-nutrient Accessed [15 July 2015]

Neggers YH. 2015. The relationship between preterm birth and underweight in Asian women. *Reproductive Toxicol*. 56:170-4.

Nelson M, Atkinson M, Darbyshire S. 1994. Food photography I: The perception of food portion size from photographs. *Brit J Nutr*. 72:649-63.

Ngai I, Govindappagari S, Neto N, Marji M, Landsberger E, Garry D. 2014. Outcome of pregnancy when gestational diabetes is diagnosed before or after 24 weeks of gestation. *Obstet & Gynecol*. 123.

NHMRC. 2013. National Health and Medical Research Council: Australian Dietary Guidelines. Canberra.

NICE (National Institute for Health and Clinical Excellence) 2010. NICE Public Health Guidance, No 27. Dietary interventions and physical activity interventions for weight management before, during and after pregnancy. NICE: London.

Niebyl JR. 2010. Nausea and vomiting in pregnancy. *N Engl J Med*. 363(16):1544-50.

Niedhammer I, Bugel I, Bonenfant S, Goldberg M, Leclerc A. 2000. Validity of self-reported weight and height in the French GAZEL cohort. *Int J obes relat metab disord.*24(9):1111-8.

Nohr EA, Vaeth M, Baker JL, Sorensen TI, Olsen J, Rasmussen KM. 2009. Pregnancy outcomes related to gestational weight gain in women defined by their body mass index, parity, height, and smoking status. *Am J Clin Nutr*. 90:1288–94.

Northstone K, Emmett P, Rogers I. 2008. Dietary patterns in pregnancy and associations with socio-demographic and lifestyle factors. *Eur J Clin Nutr*. 62:471-9.

Northstone K, Smith ADAC, Cribb VL, Emmett PM. 2013. Dietary pattern in UK adolescents obtained from a dual-source FFQ and their associations with socio-economic position, nutrient intake and modes of eating. Pub Health Nutr. 17(7):1476-85.

Nulman I, Rovet J, Barrera M, Knittel-Keren D, Feldman BM, Koren G. 2009. Long-term neurodevelopment of children exposed to maternal nausea and vomiting of pregnancy and diclectin. *J pediatr*. 155(1):45-50

O'Connor DL. 1994. Folate status during pregnancy and lactation. *Adv Exp Med Biol*. 352:157-72.

Ogbonna C, Woelk GB, Ning Y, Mudzamiri S, Mahomed K, Williams MA. 2008. Maternal mid-arm circumference and other anthropometric measures of adiposity in relation to infant birth size among Zimbabwean women. *Acta Obstet Gyn Scan*. 86:26–32.

Ohara N, Tsujino T, Maruo T. 2004. The role of thyroid hormone in trophoblast function, early pregnancy maintenance, and fetal neurodevelopment. *J Obstet Gynaecol Can*. 26(11):982-90.

Ohkawa N, Shoji H, Kitamura T, Suganuma H, Yoshikawa N, Suzuki M et al. 2010. IGF-I, leptin and active ghrelin levels in very low birth weight infants during the first 8 weeks of life. *Acta Pædiatrica*. 99:37–41.

Ojha N, Malla DS. 2007. Low birth weight at term: Relationship with maternal anthropometry. *J Nepal Med Assoc*. 46(2)166:S2-6.

Ojo OA, Louwagie G, Morojele N, Rendall-Mkosi K, London L, Olorunju S et al. 2010. Factors associated with female high-risk drinking in a rural and urban South African site. *S Afr Med J*. 100:180-2.

Oldewage-Theron W, Kruger R. 2011. Dietary diversity and adequacy of women caregivers in a peri-urban informal settlement in South Africa. Nutrition. 27(4):420-7.

Olson CM, Strawderman MS, BA Dennison. 2009. Maternal weight gain during pregnancy and child weight at age 3 years. *Matern Child Health J*. 13:839–46.

Orcholski L, Luke A, Plange-Rhule J, Bovet P, Forrester TE, Lambert EV, Dugas LR, Kettmann E, Durazo-Arvizu RA, Cooper RS, Schoeller DA. 2015. Under-reporting of dietary energy intake in five populations of the Africa diaspora. *Brit J Nutr*. 113:464-72.

Osrin D, Vaidya A, Shrestha Y, Baniya RB, Manandhar DS, Adhikari RK, Anthony MDL. 2005. Effects of antenatal multiple micronutrient supplementation on birthweight and gestational duration in Nepal: double-blind, randomised controlled trial. The lancet, 365(9463), 955-962.

Ota E, Hori H, Mori R, Tobe-Gai R, Farrar D. 2015. Antenatal dietary education and supplementation to increase energy and protein intake. *Cochrane Database of Systematic Reviews*. 6. Art. No.: CD000032. DOI: 10.1002/14651858.CD000032.pub3.

Ovaskainen ML, Paturi M, Reinivuo H, Hannila ML, Sinkko H, Lehtisalo J et al. 2008. Accuracy in the estimation of food servings against the portions in food photographs. *Eur J Clin Nutr*. 62:674–81.

Palermo M, Pellegrini N, Fogliano V. 2014. The effect of cooking on the phytochemical content of vegetables. *J Sci Food & Agriculture*. 94(6):1057-70.

Pannacciulli N, Cantatore FP, Minenna A, Bellacicco M, Giorgino R, De Pergola G. 2001. C-reactive protein is independently associated with total body fat, central fat, and insulin resistance in adult women. *Int J Obes Relat Metab Disord*. 25:1416–20.

Parker JA, Conway DL. 2007. Diabetic ketoacidosis in pregnancy. *Obstet Gynecol Clin North Am*. 34:533–43.

Pelletier D, Arimond M, Johnson FC, Liang E, Low J, Mvula P et al. 1995. Maternal anthropometry predictors of intrauterine growth retardation and prematurity in the Malawi maternal and child nutrition study. *B World Health Organ*. 73 (Suppl.), 80–1.

Pena-Rosas JP, Viteri FE. 2009. Effects and safety of preventive oral iron or iron+folic acid supplementation for women during pregnancy. *Cochrane Database of Systematic Reviews*

Pencharz P, Jahoo F, Kurpad A, Michaelsen KF, Slater C, Tome D, Weisell R. 2014. Current issues in determining dietary protein and amino-acid requirements. *Eur J Clin Nutr*. 68:285–6.

Penney DS, Miller KG. 2008. Nutritional counseling for vegetarians during pregnancy and lactation. *J Midwifery Wom Heal*. 53(1):37–44.

Perry CA, Caudill MA. 2012. Biotin: critical for fetal growth and development yet often overlooked. Nutr Today. 47(2):79-85.

Perry CA, West AA, Gayle A, Lucas LK, Yan J, Jiang X, Caudill MA. 2014. Pregnancy and lactation alter biomarkers of biotin metabolism in women consuming a controlled diet. *J nutr*. 144(12): 1977-84.

Pettifor JM. 2005. Rickets and vitamin D deficiency in children and adolescents. *Endocrinol Metab Clin North Am*. 34(3):537-53, vii.

Phillips AK, Roy SC, Lundberg R, Guilbert TW, Auger AP, Blohowaik SE, Coe CL, Kling PJ. 2014. Neonatal iron status is impaired by maternal obesity and excessive weight gain during pregnancy. *J Perinatol*. 34:513-8.

Portincasa P, Di Ciaula A, Wang HH, Palasciano G, van Erpecum KJ, Moschetta A, Wang DQ. 2008. Coordinate regulation of gallbladder motor function in the gut-liver axis. *Hepatology*. 47:2112-26.

Powers HJ, Corfe BM, Naano E. 2011. Riboflavin in development and cell fate. Subcell Biochem. 56:229:45.

Prather CM. 2004. Pregnancy-related constipation. *Curr Gastroenterology Reports*. 6(5):402-4.

Procter SB, Campbell CG. 2014. Position of the Academy of Nutrition and Dietetics: Nutrition and lifestyle for healthy pregnancy outcome. *J Acad Nutr Diet*. 114:1099-103.

Pulver LS, Guest-Warnick G, Stoddard GJ, Byington CL, Young PC. 2009. Weight for gestational age affects the mortality of late preterm infants. *Pediatrics*. 123: e1072–7.

Quartarone G. 2013. Gastroesophageal reflux in pregnancy: a systematic review on the benefit of raft forming agents. *Minerva Ginecologica.* 65(5):541-9.

Rakova N, Muller DN, Staff AC, Luft FC, Dechend R. 2014. Novel ideas about salt, blood pressure, and pregnancy. *J reproductive immunol*. 101:135-9.

Rasmussen M, Holstein BE, Melkevik O, Damsgaard MT. 2013. Validity of self-reported height and weight among adolescents: the importance of reporting capability. *BMC Med Research Methodol.* 13(85):1-11.

Refsum H. 2001. Folate, vitamin B12 and homocysteine in relation to birth defects and pregnancy outcome. *Brit J Nutr*. 85(Suppl 2): S109–13.

Resnicow K, Odom E, Wang T, Dudley WN, Mitchell D, Vaughn R et al. 2000. Validation of three food frequency questionnaires and 24-hour recalls with serum caretenoid levels in a sample of African-American adults. *Am J Epidemiol*. 152, 1072-80.

Ribot B, Aranda N, Viteri F, Herna´ndez-Martı´nez C, Canals J, Arija V. 2012. Depleted iron stores without anaemia early in pregnancy carries increased risk of lower birthweight even when supplemented daily with moderate iron. *Human Reproduction*. 27(5):1260–6.

Ricci E, Parazzini F, Chiaffarino F, Cipriani S, Polverino G. 2010. Pre-pregnancy body mass index, maternal weight gain during pregnancy and risk of small-for-gestational age birth: Results from a case–control study in Italy. *J Matern-Fetal Neonat Med*. 23(6):501-5.

Rich-Edwards JW, Kleinman K, Michels KB, Stampfer MJ, Manson JE, Rexrode KM et al. 2005. Longitudinal study of birth weight and adult body mass index in predicting risk of coronary heart disease and stroke in women. *Brit Med J*. 330(7500):1115-8.

Riley EP, McGee CL. 2005. Fetal alcohol spectrum disorders: an overview with emphasis on changes in brain and behavior. *Exp Biol Med*. 230(6):357–65.

Ritchie LD, King JC. 2008. Nutrient recommendations and dietary guidelines for pregnant women. In *Handbook of nutrition and pregnancy*. [Lammi-Keefe CJ, Couch SC, Philipson EH editors]. Humana Press. pp. 3-25.

Rizotto JM, Giusti J, Higgins L. 2009. Nutrition in pregnancy. In *Diabetes in women: Pathophysiology and therapy*. [Tsatsoulis et al. editors]. Humana Press. pp. 255-72.

Robinson SM, Crozier SR, Borland SE, Hammond J, Barker DJ, Inskip HM. 2004. Impact of educational attainment on the quality of young women's diets. *Eur J Clin Nutr*. 58(8):1174-80.

Robson PJ, Livingstone MBE. 2000. An evaluation of food photographs as a tool for quantifying food and nutrient intakes. *Publ Health Nutr*. 3(2):183-92.

Rockett HRH, Wolf AM, Colditz GA. 1995. Development and reproducibility of a food frequency questionnaire to assess diets of older children and adolescents. *J Am Diet Assoc*. 95:336-40.

Roglic G, Colagiuri S. 2014. Gestational Diabetes Mellitus: Squaring the Circle. *Diabetes care*. 37(6): 143-4.

Roman P, Qin D, Marta S. 2014. Pregnancy outcome and breastfeeding pattern among vegans, vegetarians and non-vegetarians. *J Diet Res Nutr.* 1(1):1-4.

Ronnenberg AG, Wood RJ,Wang X, Xing H, Chen C, Chen D et al. 2004. Preconception hemoglobin and ferritin concentrations are associated with pregnancy outcome in a prospective cohort of Chinese women. *J Nutr*. 134:2586–91.

Roseboom T, de Rooij S, Painter R. 2006. The Dutch famine and its long-term consequences for adult health. *Early Hum Dev*. 82:485–91.

Rosenberg A. 2008. The IUGR Newborn. *Semin Perinatol*. 32:219-24.

Rossouw K, Senekal M, Stander I. 2001. The accuracy of self-reported weight by overweight and obese women in an outpatient setting. *Pub health nutr. 4*(1): 19-26.

Roth C, Magnus P, Schjolberg S, Stoltenberg C, Sure'n P, McKeague IW et al. 2011. Folic acid supplements in pregnancy and severe language delay in children. *J Am Med Assoc*. 306(14):1566-73.

Rozovski SJ, Winick M. 1979. Nutrition and cellular growth. In *Human Nutrition: pre- and postnatal development*. pp. 61-102 [Winick M, editor]. New York: Plenum Press.

Ruel MT. 2003. Operationalizing dietary diversity: a review of measurement issues and research priorities. *J Nutr*. 133:3911-26.

Rumbold A, Crowther CA. 2014. Vitamin E supplementation in pregnancy (review). *Cochrane Database of Systematic Reviews* (2), 38.

Rumpler WV, Kramer M, Rhodes DG, Moshfegh AJ, Paul DR. 2008. Identifying sources of reporting error using measured food intake. *Eur J Clin Nutr.* 62:544–52.

Rylander R. 2014. Magnesium in pregnancy blood pressure and pre-eclampsia – A review. *Pregnancy Hypertension: An International Journal of Women's Cardiovascular Health*. 4(2):146-9.

Saab RO, Bassil BJ, El Khoury MI, Rassam PW, Snaifer EE, Jabbour RA. 2014. Wernicke Encephalopathy following bariatric surgery and hyperemesis gravidarum. From the Editor, 7(10).

SADHS. 2007. Department of Health, Medical Research Council, OrcMacro. South Africa Demographic and Health Survey 2003. Pretoria: Department of Health.

Sahu S, Abraham R, Vedavalli R, Daniel M. 2009. Study of lipid profile, lipid peroxidation and vitamin E in pregnancy induced hypertension. *Indian J Physiol Pharmacol*. 53(4):365–9.

Salas-Salvado J, Martinez-Gonzalez MA, Bullo M, Ros E. 2011. The role of diet in the prevention of type 2 diabetes. Nutr Metab Cardiovasc Dis. Suppl 2:B32-48.

Sanders T. 1994. Good nutrition for the vegetarian mother. *Mod Midwife*. 4(4):23-6.

Santos IS, Victora CG, Huttly S, Carvalhal JB. 1998. Caffeine intake and low birth weight: a population-based case–control study. *Am J Epidemiol*. 147:620–7.

Saving Mothers fourth report 2005–2007. 2009. National Committee on Confidential Enquiries into Maternal Deaths. Pretoria: Department of Health.

Saving Mothers. 2014. Saving Mothers 2011–2013 Sixth report on the confidential enquiries into maternal deaths in South Africa. Pretoria: Department of Health (South Africa).

Savitz DA, Kaufman JS, Dole N, Siega-Riz AM, Thorp JM, Kaczor DT. 2004. Poverty, education, race, and pregnancy outcome. Ethnicity & disease. 14(3): 322-9.

Schneider M, Norman R, Steyn NP, Bradshaw D. 2007. Estimating the burden of disease attributable to low fruit and vegetable intake in South Africa in 2000. *S Afr Med J*. 97(8):717-23.

Scholl TO, Sowers M, Chen X, Lenders C. 2001. Maternal glucose concentration influences fetal growth, gestation, and pregnancy complications. *Am J Epidemiol*. 154(6):514–20.

Scholl TO. 1998. High third-trimester ferritin concentration: Associations with very preterm delivery, infection, and maternal nutritional status. *Am J Obstet Gynecol*. 92:161-6.

Scholl TO. 2005. Iron status during pregnancy: Setting the stage for mother and infant. *Am J Clin Nutr*. 81(5):1218S-22S.

Scholl TO. 2011. Maternal iron status: relation to fetal growth, length of gestation, and iron endowment of the neonate. *Nutr Rev*. 69(Suppl.1):S23–9.

Scholl TO, Sowers M, Chen X, Lenders C. 2001. Maternal glucose concentration influences fetal growth, gestation, and pregnancy complications. *Am J Epidemiol*. 154(6): 514-20.

Scholl TO, Reilly T. 2000. Anemia, iron and pregnancy outcome. *J nutr*. 130(2): 443-7.

Scholtz W, Jones A, Phillips DIW, Gale CR, Robinson SM, Godfrey KM. 2010. Lower maternal folate status in early pregnancy is associated with childhood hyperactivity and peer problems in offspring. *J Child Psychol Psychiatry*. 51(5):594–602.

Schonfeldt HC, Pretorius B, Hall N. 2013. "Fish, chicken, lean meat and eggs can be eaten daily": a food-based dietary guideline for South Africa. *S Afr J Clin Nutr*. 26(3)(Supplement):S66-S76.

Schuchardt JP, Huss M, Stauss-Grabo M, Hahn A. 2010. Significance of long-chain polyunsaturated fatty acids (PUFAs) for the development and behaviour of children. *Eur J Pediatr.* 169:149–64.

Scientific Advisory Committee on Nutrition (SACN). 2015. Carbohydrates and health report. TSO. Accessed 6 August 2015 [https://www.gov.uk/government/publications/sacn-carbohydrates-and-health-report]

Sealy-Potts C, Potts AC. 2014. An assessment of dietary diversity and nutritional status of preschool children. *Austin J Nutri Food Sci*. 2(7):1040-4

Sebire NJ, Jolly M, Harris JP, Wadsworth J, Joffe M, Beard RW et al. 2001. Maternal obesity and pregnancy outcome: a study of 287,213 pregnancies in London. *Int J Obes Relat Metab Disord*. 25(8):1175-82.

Sengpiel V, Bacelis J, Myhre R, Myking S, Pay ASD, Haugen M, Brantsaeter AL, Meltzer HM, Nilsen RM, Magnus P, Vollset SE, Nilsson S, Jacobsson B. 2014. Folic acid supplementation, dietary folate intake during pregnancy and risk for spontaneous preterm delivery: a prospective observational cohort study. *BMC Pregnancy and Childbirth*. 14:375.

Serfontein M, Venter G, Kruger A, MacIntyre U, Pisa PT. 2010. Alcohol intake and micronutrient density in a population in transition: the transition and health during urbanisation in South Africa (THUSA) study. *S Afr J Clin Nutr.* 23(3)(Suppl. 1):22-8.

Sevak L, Mangtani P, McCormack V, Bhakta D, Kassam-Khamis T, dos Santos Silva I. 2004. Validation of a food frequency questionnaire to assess macro- and micro-nutrient intake among South Asians in the United Kingdom. *Eur J Nutr*. 43,160-8.

Shankar K, Ronis MJJ, Badger TM. 2007. Effects of pregnancy and nutritional status on alcohol metabolism. *Alcohol Res Health*. 30(1);55-9.

Shaw GM, Finnell RH, Blom HJ, Carmichael SL, Vollset SE, Yang W, Ueland PM. 2009. Choline and risk of neural tube defects in a folate-fortified population. Epidemiol. 20(5): 714-19.

Shearer MJ. 2009. Vitamin K deficiency bleeding (VKDB) in early infancy. Blood Rev.23:49–59.

Shibata M, Suzuki A, Sekiya T, Sekiguchi S, Asano S, Udagawa Y et al. 2011. High prevalence of hypovitaminosis D in pregnant Japanese women with threatened premature delivery. *J Bone Miner Metab*. 29(5):615-20.

Shisana O, Labadarios D, Rehle T, Simbayi L, Zuma K, Dhansay A et al. 2013. *South African National Health and Nutrition Examination Survey (SANHANES-1)*. Cape Town: HSRC Press

Shrim A, Boskovic R, Maltepe C, Navioz Y, Garcia Bournissen F, Koren G. 2006. Pregnancy outcome following use of large doses of vitamin B6 in the first trimester. *J Obstet Gynaecol.* 26(8):749-51.

Sibai BM, Viteri OA. 2014. Diabetic Ketoacidosis in pregnancy. *Obstet & Gynecol*. 123(1):167-78.

Sie KKY, Medline A, van Weel J, Sohn KJ, Choi SW, Croxford R et al. 2011. Effect of maternal and postweaning folic acid supplementation on colorectal cancer risk in the offspring. Gut. 60:1687-94.

Siega-Riz AM, Hartzema AG, Turnbull C, Thorp J, McDonald T, Cogswell ME. 2006. The effects of prophylactic iron given in prenatal supplements on iron status and birth outcomes: A randomized controlled trial. *Am J Obstet Gynecol*. 194:512–9.

Simpson E, Mull JD, Longley E, East J. 2000. Pica during pregnancy in low income women born in Mexico. *West J Med*. 173(1):20–4.

Sinclair KD, Watkins AJ. 2013. Parental diet, pregnancy outcomes and offspring health: metabolic determinants in developing oocytes and embryos. *Reproduction, Fertility and Development.* 26(1): 99-114.

Small L, Sidora-Arcoleo K, Vaughan L, Creed-Capsel J, Chung KY, Stevens C. 2009. Validity and reliability of photographic diet diaries for assessing dietary intake among young children. *Infant Child Adolesc Nutr*. 1(1):27-36.

Smith SM, Garic A, Flentke GR, Berres ME. 2014. Neural crest development in fetal alcohol syndrome. *Birth Defects Res*. 102(3):210-20.

Smithers G, Finch S, Doyle W, Lowe C, Bates CJ, Prentice A et al. 1998. The National Diet and Nutrition Survey: people aged 65 years and over. *Nutrition & Food Science*. 98(3):133-4.

Smuts CM, Wolmarans P. 2013. The importance of the quality or type of fat in the diet: a food-based dietary guideline for South Africa. *S Afr J Clin Nutr.* 26(3):(Supplement):S87-S99.

Sobal J, Stunkard AJ. 1989. Socioeconomic status and obesity: A review of the literature. *Psychol Bull.* 105(2):260–75.

Spencer EA, Appleby PN, Davey GK, Key TJ. 2002. Validity of self-reported height and weight in 4808 EPIC–Oxford participants. *Pub health nutr. 5*(4): 561-5.

Stan CM, Boulvain M, Pfister R, Hirsbrunner-Almagbaly P. 2002. Hydration for treatment of preterm labour. *Cochrane Database of Systematic Reviews*, Issue 2. Art. No.: CD003096. DOI: 10.1002/14651858.CD003096.

Stanga Z, Brunner A, Leuenberger M, Grimble RF, Shenkin A, Allison SP, Lobo DN. 2008. Nutrition in clinical practice - the refeeding syndrome: illustrative cases and guidelines for prevention and treatment. Eur J Clin Nutr. 62:687.

Stats, S.A. 2012. Census 2011. *Mobile information. http://mobi. statssa. gov. za/census/Inter net. html*.

Steer P, Alam MA, Wadsworth J, Welch A. 1995. Relation between maternal haemoglobin concentration and birth weight in different ethnic groups. *Brit Med J*. 310:489-91.

Steer PJ. 2000. Maternal hemoglobin concentration and birth weight. *Am J Clin Nutr*. 71: S1285-7.

Steffen LM. 2014. Lower levels of sodium intake and reduced cardiovascular risk: the challenge to achieve lower sodium recommendations. *Circulation*. 129(9): 956-7.

Stephens TV, Payne M, O Ball R, Pencjarz PB, Elango R. 2015. Protein requirements of healthy pregnant women during early and late gestation are higher than current recommendations. *J Nutr*. 145:73–8.

Stephensen CB. 2001. Vitamin A, infection, and immune function. Annu Rev Nutr. 21:167–92.

Steyn NP & Senekal M. 2004. Dietary Assessment and Education Kit (DAEK): Chronic

Steyn NP, Labadarios D, Nel JH. 2011. Factors which influence the consumption of street foods and fast foods in South Africa - A national survey. *Nutr J.* 10:104.

Steyn NP, Nel JH, Nantel G, Kennedy G, Labadarios D. 2005. Food variety and dietary diversity scores in children: are they good indicators of dietary adequacy? *Pub Health Nutr. 9(5):644-50.*

Steyn NP, Senekal M, Norris SA, Whati L, MacKeown JM, Nel JH. 2006. How well do adolescents determine portion sizes of food and beverages? *Asia Pac J Clin Nutr*. 15:35-42.

Steyn NP, Nel J, Labadarios D, Maunder EMW, Kruger HS. 2014. Which dietary diversity indicator is best to assess micronutrient adequacy in children 1 to 9 y?. *Nutrition*. 30(1):55-60.

Steyn NP, Temple NJ. 2012. Evidence to support a food-based dietary guideline on sugar consumption in South Africa. BMC *Pub Health*. 12:502.

Steyn NP. 2006. Nutrition and chronic diseases of lifestyle in South Africa. In: *Chronic diseases of lifestyle in South Africa: 1995-2005.* [Steyn K, Fourie J, Temple N, eds]. Cape Town: Medical Research Council; p. 33-47.

Steyn NP, Ochse R. 2013. "Enjoy a variety of foods": a food-based dietary guideline for South Africa. *S Afr J Clin Nutr*. 26(3): 13-7.

Stocker CJ, Arch JR, Cawthorne MA. 2005. Fetal origins of insulin resistance and obesity. *Proc Nutr Soc*. 64:143–151.

Stuebe AM, Oken E, Gillman MW. 2009. Associations of diet and physical activity during pregnancy with risk for excessive gestational weight gain. *Am J Obstet Gyne*col. 201(1): 58.e1–58.e8. doi:10.1016/j.ajog.2009.02.025.

Su KP, Huang SY, Chiu TH, Huang KC, Huang CL, Chang HC et al. 2008. Omega-3 fatty acids for major depressive disorder during pregnancy: results from a randomized, double-blind, placebo-controlled trial. *J Clin Psychiatry*. 69:644–51.

Subar AF, Crafts J, Zimmerman TP, Wilson M, Mittl B, Islam NG et al. 2010. Assessment of the accuracy of portion size reports using computer-based food photographs aids in the development of an automated self administered 24-hour recall. *J Am Diet Assoc*. 110:55-64.

Subar AF, Kipnis V, Troiano RP, Midthune D, Schoeller DA, Bingham S et al. 2003. Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: The OPEN Study. *Am J Epidemiol*. 158:1-13.

Surgeon General. 2005. U.S. Surgeon General releases advisory on alcohol use in pregnancy: urges women who are pregnant or who may become pregnant to abstain from alcohol. Available at: www.surgeongeneral.gov/pressreleases/sg02222005.html (accessed 31 August 2012).

Swindale A, Bilinsky P. 2005. Household dietary diversity score (HDDS) for measurement of household food access: indicator guide. *Food and Nutrition Technical Assistance Project.* Washington DC.

Syddall HE, Sayer AA, Simmonds SJ, Osmond C, Cox V, Dennison EM et al. 2005. Birth weight, infant weight gain, and cause-specific mortality: the Hertfordshire Cohort study. *Am J Epidemiol*. 161:1074–80.

Symonds ME, Stephenson T, Gardner DS, Budge H. 2007. Long-term effects of nutritional programming of the embryo and fetus: mechanisms and critical windows. *Reprod Fertil Dev*. 19(1):53–63.

Tamura T, Goldenberg RL, Johnston KE, Chapman VR. 2004. Relationship between prepregnancy BMI and plasma zinc concentrations in early pregnancy. *Brit J Nutr*. 91:773–7.

Tamura T, Picciano MF. 2006. Folate and human reproduction. *Am J Clin Nutr*. 83:993–1016.

Tan PC, Jacob R, Quek KF, Omar SZ. 2007. Pregnancy outcome in hyperemesis gravidarum and the effect of laboratory clinical indicators of hyperemesis severity. *J Obstet Gynaecol Res.*3:457-64.

Tanaka T, Ashihara K, Nakamura M, Kanda T, Fujita D, Yamashita Y, Terai Y, Kamegai H, Ohmichi M. 2014. Associations between the pre-pregnancy body mass index and gestational weight gain with pregnancy outcomes in Japanese women. *J Obstet & Gynaecol Res*. 40(5):1296-303.

Tavares TG, Malcata FX. 2012. The Portuguese Paradox: Why do some inhabitants of Portugal appear to live so long when their diet is based on whey cheese?. *Food Chemistry.* 131(3):727-9.

Te Morenga L, Mallard S, Mann J. 2013. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *Brit Med J*. 346: e7492.

Temple NJ, Steyn NP, Fourie J, De Villiers A. 2011. Price and availability of healthy food: a study in rural South Africa. *Nutrition.* 27(1): 55-8.

Temple NJ, Steyn NP. 2011b. The cost of a healthy diet: A South African perspective. *Nutr. 27*(5):505-8.

Temple NJ, Steyn NP. 2013. Sugar and health: a food-based dietary guideline for South Africa. S Afr J Clin Nutr. 26(3): (Supplement):S100-S104.

Temple NK, Steyn NP, Fourie J, De Villiers A. 2011. Price and availability of healthy food: A study in rural South Africa. Nutr. 27:55-8.

Thame M, Trotman H, Osmond C, Fletcher H, Antoine M. 2007. Body composition in pregnancies of adolescents and mature women and the relationship to birth anthropometry. *Eur J Clin Nutr*. 61:47–53.

Thaver D, Saeed MA, Bhutta ZA. 2006. Pyridoxine (vitamin B6) supplementation in pregnancy. *Cochrane Database of Systematic Reviews*. Issue 2. Art. No.: CD000179. DOI: 10.1002/14651858.CD000179.pub2.

Thomas MK, Demay MB. 2000. Vitamin D deficiency and disorders of vitamin D metabolism. *Endocrinol Metab Clin N Am*. 29:611–27.

Thompson FE, Byers T. 1994. Dietary assessment resource manual. *J Nutr.* 124(Suppl.): 2245S-311S.

Thompson LA, Zhang S, Black E, Das R, Ryngaert M, Sullivan S, Roth J. 2013. The associationof maternal pre-pregnancy Body Mass Index with breastfeeding initiation. *Matern Child Health J*. 17:1842–1851

Tolsa CB, Zimine S, Warfield SK. 2004. Early alteration of structural and functional brain development in premature infants born with intrauterine growth restriction. *Pediatr Res*. 56:132–8.

Tooze JA, Krebs-Smith SM, Troiano RP, Subar AF. 2012. The accuracy of the Goldberg method for classifying misreporters of energy intake on a food frequency questionnaire and 24-h recalls: comparison with doubly labeled water. *Eur J Clin Nutr*. 66:569-76.

Torheim LE, Barikmo I, Hatley A, Diakite M, Solvoll K, Diarra MM et al. 2001. Validation of a quantitative food-frequency questionnaire for use in Western Mali. *Pub Health Nutr*. 4: 1267-77.

Toriola AL, Moselakgomo VK, Shaw BS, Goon DT. 2012. Overweight, obesity and underweight in rural black South African children. *S Afr J Clin Nutr. 25*(2): 57-61.

Trabulsi J. Schoeller DA. 2001. Evaluation of dietary assessment instruments against doubly labeled water, a biomarker of habitual energy intake. *Am J Physiol Endocrinol Metab*. 281:891-9.

Trumbo P, Schlicker S, Yates AA, Poos M. 2002. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. *J Am Diet Assoc.* 102(11):1621–30.

Tshitaudzi GT. 2003. Nutritional status of pregnant women (under 20 years of age) with special emphasis on iron and folic acid status. Thesis.

Tuntiseranee P, Olsen J, Chongsuvivatwong V, Limbutara S. 1999. Socioeconomic and work related determinants of pregnancy outcome in southern Thailand. J epidemiol & community health. 53(10): 624-9.

Turner RT. 2000. Skeletal response to alcohol. *Alcohol Clin Exp Res.* 24(11):1693–701.

Uauy R, Dangour AD. 2006. Nutrition in brain development and aging: role of essential fatty acids. *Nutr Rev*. 64: S24–S33.

Underwood BA. 1994. Maternal vitamin A status and its importance in infancy and early childhood. *Am J Clin Nutr.* 59:517S–24S.

UNICEF and Department of Health of South Africa. 2007. A reflection of the South African maize meal and wheat flourfortification programme (2004 - 2007).

UNICEF. 2008. South Africa launches its first ever Vitamin "A" supplementation campaign http://www.UNICEF.org/media/media_45583.html Accessed [13 July 2015]

UNICEF. 2009. State of the world's children: Maternal and newborn health. [Moccia P, Anthony D, editors] Virginia.

UNICEF. 2014. Trends in maternal mortality: 1990 to 2013. Estimates by WHO, UNICEF, UNFPA, The World Bank and the United Nations Population Division.

Urban M, Chersich MF, Fourie LA, Chetty C, Olivier L, Viljoen D. 2008. Fetal alcohol syndrome among grade 1 schoolchildren in Northern Cape Province: prevalence and risk factors. *S Afr Med J*. 98(11):877–82.

US Teratology Society. 1987. Vitamin A during pregnancy. *Teratology*. 35:267–8.

USDA. 2011. *USDA Super Tracker: My foods. My fitness. My health.* http://www.mypyramid.gov/mypyramidmoms/index.html Accessed [13 July 2015].

van Eijsden M, Smits LJM, van der Wal MF, Bonsel GJ. 2008. Association between short inter pregnancy intervals and term birth weight: the role of folate depletion. *Am J Clin Nutr*. 88:147–53.

Vazquez J. 2010. Constipation, haemorrhoids, and heartburn in pregnancy. Clin Evid. 8:1411.

Veena SR, Krishnaveni GV, Srinivasan K, Wills AK, Muthayya S, Kurpad AV et al. 2010. Higher maternal plasma folate but not vitamin B-12 concentrations during pregnancy are associated with better cognitive function scores in 9- to 10- year-old children in South India. *J Nutr*. 140:1014–22.

Venkatachalam PS, Belavady B, Gopalan C. 1962. Studies on vitamin A nutritional status of mothers and infants in poor communities of India. *J Pediatr.* 61:262–8.

Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, & Maternal and Child Undernutrition Study Group. 2008. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet*. 371(9609):340-57.

Vikse BE, Irgens LM, Leivestad T, Hallan S, Iversen BM. 2008. Low birth weight increases risk for end-stage renal disease. *J Am Soc Nephrol*.19:151–7.

Viljoen DL, Gossage JP, Brooke L, Adnams CM, Jones KL, Robinson LK et al. 2005. Fetal alcohol syndrome epidemiology in a South African community: a second study of a very high prevalence area. *J Stud Alcohol.* 66(5):593–604.

Viljoen E, Visser J, Koen N, Musekiwa A. A systematic review and meta-analysis of the effect and safety of ginger in the treatment of pregnancy-associated nausea and vomiting. *Nutr J*. 13(20):1-14.

Villar J, Belizan JM. 2000. Same nutrient, different hypotheses: disparities in trials of calcium supplementation during pregnancy. *Am J Clin Nutr*. 71(Suppl 5):S1375–9.

Villar J, Say L, Shennan A, Lindheimer M, Duley L, Conde-Agudelo A et al. 2004. Methodological and technical issues related to the diagnosis, screening, prevention and treatment of pre-eclampsia and eclampsia. *Int J Gynecol Obstet*. 85(Suppl 1):S28–S41.

Vollset SE, Refsum H, Irgens LM, Emblem BM, Tverdal A, Gjessing HK et al. 2000. Plasma total homocysteine, pregnancy complications, and adverse pregnancy outcomes: the Hordaland Homocysteine Study. *Am J Clin Nutr*. 71: 962–8.

Vorster HH, Badham JB, Venter CS. 2013. An introduction to the revised food-based dietary guidelines for South Africa. *S Afr J Clin Nutr*.26(3):S1-S164

Vorster HH, Kruger A, Wentzel-Viljoen E, Kruger SH, Margetts BM. 2014. Added sugar intake in South Africa: findings from the Adult Prospective Urban and Rural Epidemiology cohort study. Am J Clin Nutr. 99:1479–86.

Walker ARP, Walker BF, Jones J, Verardi M, Walker C. 1985. Nausea and vomiting and dietary cravings and aversions during pregnancy in South African women. *Brit J Obstet Gyn*. 92(5): 484-9.

Wang L, Manson JE, Sesso HD. 2012. Calcium intake and risk of cardiovascular disease. Am J Cardiovasc Drugs. 12(2):105-16.

Wang X, Lin X, Ouyang YY, Liu J, Zhao G, Pan A, Hu FB. 2015. Red and processed meat consumption and mortality: dose-response meta-analysis of prospective cohort studies. Pub Health Nutr. Available on CJO2015. doi:10.1017/S1368980015002062.

Wardlaw GM, Kessel MW. 2002. In: *Perspectives in Nutrition*. [Mc Graw Hill, editor]. 5th ed. Boston Burr Ridge.

Warren K, Floyd L, Calhoun F, Stone D, Bertrand J, Streissguth A et al. 2004. Consensus statement on FASD. Washington, DC: National Organization on Fetal Alcohol Syndrome.

Warren KR, Hewitt BG. 2009. Fetal alcohol spectrum disorders: when science, medicine, public policy, and laws collide. *Dev Disabil Res Rev*. 15:170-5.

Watanabe K, Naruse K, Tanaka K, Metoki H, Suzuki Y. 2013. Outline of definition and classification of "Pregnancy induced Hypertension (PIH)". *Hypertens Res Pregnancy*. 1:3-4.

Waters TP, Huston-Presley L, Catalano PM. 2012. Neonatal body composition according to the revised Instititue of Medicine recommendations for maternal weight gain. *J Clin Endocrinol Metab*. 97 (10):3648-54.

Weltens N, Zhao D, Oudenhove L. 2014. Where is the comfort in comfort foods? Mechanisms linking fat signaling, reward, and emotion. *Neurogastroenterology & Motility. 26*(3): 303-15.

Wen W, Shu XO, Jacobs DR, Brown JE. 2001. The associations of maternal caffeine consumption and nausea with spontaneous abortion. *Epidemiology.*12:38–42.

Wendt A, Gibbs CM, Peters S, Hogueb CJ. 2012. Impact of increasing inter-pregnancy interval on maternal and infant health. *Paediatr Perinat Ep*. 26(Suppl. 1):239–58.

Weng X, Odouli R, Li D-K. 2008. Maternal caffeine consumption during pregnancy and the risk of miscarriage: a prospective cohort study. *Am J Obstet Gynecol*. 198(3):279-87.

Whincup PH, Kaye SJ, Owen CG, Huxley R, Cook DG. 2008. Birth weight and risk of type 2 diabetes: a systematic review. *J Am Med Assoc*. 300(24):2886-97.

Whitaker RC. 2004. Predicting preschooler obesity at birth: the role of maternal obesity in early pregnancy. *Pediatrics*. 114:e29–36.

White ND. 2014. Lifestyle and complementary medicine for common gastrointestinal disorders in pregnancy. *Am J Lifestyle Med*. 8)2):97-9.

Widen E, Siega-Riz AM. 2010. Prenatal nutrition: a practical guide for assessment and counselling. *J Midwifery Womens Helath*. 55(6):540-9.

Widen EM, Gallagher D. 2014. Body composition changes in pregnancy: measurement, predictors and outcomes. *Eur J Clin Nutr*. 68:643-52.

Wien TN, Pike E, Wisloff T, Staff A, Smeland S, Klemp M. 2012. Cancer risk with folic acid supplements: a systematic review and meta-analysis. Brit Med J Open. 2(1):1-15.

Wilcox AJ. 2001. On the importance- and the unimportance- of birthweight. *Int J Epidemiol.* 30:1233-41.

Wilkinson SA, Tolcher D. 2010. Nutrition and maternal health: What women want and can we provide it? *Nutr & Diet*. 67(1): 18-25.

Willett W. 1998. Food-frequency methods. In: *Nutritional Epidemiology, Monographs in Epidemiology and Biostatistics*. [Willett W, editor]. 2nd ed. New York: Oxford University Press, Vol. 30

Williamson CS. 2006. Nutrition in pregnancy. *Nutr Bull*. 31:28–59

ADDENDUM A – PASS STUDY RECRUITMENT INTERVIEW

THE SAFE PASSAGE STUDY

RECRUITMENT INTERVIEW	Participant ID

Date of Interview: [q1_Date] ___ ___ / ___ ___ / ___ ___ ___ ___ (month) (day) (year)	**Interviewer Name:** [q1_Interviewer] _____ _____ ___ (Last) (First) (MI)
Language of Interview: [q1_Language] ___ ___ (use codes below)	**Location of Interview:** [q1_Location] ___ ___ (use codes below)
Time Interview Began: [q1_TimeBegan] ___ ___ : ___ ___ (military time in your local time zone)	**Time Interview Ended:** [q1_TimeEnded] ___ ___ : ___ ___ (military time in your local time zone)

Background Information

1. What is your marital status now (in the last month): *(circle one)*

[q1_1]

Married	1
Partnered *(boyfriend or girlfriend)*, living together	2
Partnered *(boyfriend or girlfriend)*, not living together	3
Separated	4
Divorced	5
Single	6
Widowed	7
Other [q1_1_s1] *(please specify)*	8

→ a. How long have you been in this relationship:
[q1_1a1] years [q1_1a2] months [q1_1a3] weeks
(specify number) *(specify number)* *(specify number)*
(if married be sure to include the length of the entire relationship)

2. How many years of formal education have you completed: *(circle one)*

[q1_2]

	(Northern Plains)		*(South Africa)*
Grade School	Kindergarten	0	Primary School
	Grade 1	1	
	Grade 2	2	
	Grade 3	3	
	Grade 4	4	
	Grade 5	5	
	Grade 6	6	
	Grade 7	7	
	Grade 8	8	
High School	Grade 9	9	High School
	Grade 10	10	
	Grade 11	11	
	Grade 12 or GED	12	
College	Grade 13	13	College or University
	Grade 14	14	N/A
	Grade 15	15	
	Grade 16	16	
Graduate or Professional School	Grade 17+	17	

3. When did you find out you were pregnant: ___ ___ / ___ ___ / ___ ___ ___ ___ *(month) (day) (year)*
[q1_3]

(check all that apply)
Unsure of month ❑ [q1_3um]
Unsure of day ❑ [q1_3ud]

Language of Interview:
01=English
02=Afrikaans
03=Afrikaans and English

Location of Interview:
01=Belhar Antenatal Clinic
02=Bishop Lavis Midwife Obstetric Unit
03=Tygerberg Hospital
04=Pine Ridge Indian Health Services
05=Oglala Lakota College, Department of Nursing
06=Native American Women's Health Center
07=Rapid City Regional Hospital
08=Sanford Health
09=Altru Clinic
10=The Johnson House
11=Mercy Hospital
12=Spirit Lake Native American Maternal Health Child Program

Maternal Measurements

4. What did you weigh before you became pregnant: [q1_4_1] ____ lbs *OR* [q1_4_2] ____ . ____ kgs
(lbs) (kgs)

Instructions for the Clinical Coordinator

The following measurements must be taken at the time of the interview and must be in accordance with the Maternal Measurements Protocol. For each anthropometric measurement (items 5 – 9), two measurements must be taken and recorded. If the measurement differs by more than the acceptable range indicated, reposition the participant and take and record a third measurement.

5. Weight (record to the nearest 1 lb or 0.5 kg):

a. [q1_5a1] ____ lbs *OR* [q1_5a2] ____ . ____ kgs
(lbs) (kgs)

b. [q1_5b1] ____ lbs *OR* [q1_5b2] ____ . ____ kgs
(lbs) (kgs)

c. If not in acceptable range of **2 lbs or 1 kg**, record third measurement:
[q1_5c1] ____ lbs *OR* [q1_5c2] ____ . ____ kgs
(lbs) (kgs)

6. Height (record to the nearest $^1/_8$ in. or 1 mm):

a. [q1_6a1] ft [q1_6a2] [q1_6a3] $/_8$ in *OR* [q1_6a4] ____ mm
(ft) (in) (fraction) (mm)

b. [q1_6b1] ft [q1_6b2] [q1_6b3] $/_8$ in *OR* [q1_6b4] ____ mm
(ft) (in) (fraction) (mm)

c. If not in acceptable range of $^1/_4$ **in. or 2 mm**, record third measurement:
[q1_6c1] ft [q1_6c2] [q1_6c3] $/_8$ in *OR* [q1_6c4] ____ mm
(ft) (in) (fraction) (mm)

7. Head Circumference (record to the nearest 1 mm):

a. [q1_7a] ____ mm
(mm)

b. [q1_7b] ____ mm
(mm)

c. If not in acceptable range of **2 mm**, record third measurement: [q1_7c] ____ mm
(mm)

8. Arm Circumference (record to the nearest 1 mm):

a. [q1_8a] ____ mm
(mm)

b. [q1_8b] ____ mm
(mm)

c. If not in acceptable range of **2 mm**, record third measurement: [q1_8c] ____ mm
(mm)

9. Left Triceps Skinfold Thickness (record to the nearest 1 mm):

a. [q1_9a] ____ mm
(mm)

b. [q1_9b] ____ mm
(mm)

c. If not in acceptable range of **2 mm**, record third measurement: [q1_9c] ____ mm
(mm)

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

Interviewer's Script

Now, I am going to ask you some questions about smoking. Because these questions are personal, any information you share with me will be kept confidential. You will be identified by a number only, not by name. Your name will not be placed on this form. Here is a calendar for you to refer to. *(SHOW CALENDAR)*

Smoking and Tobacco Use History – *Section A (Cigarettes)*

[q1_10_1

10A. If you ever smoked, when was your last cigarette: MM] / DD] / YYYY] *(check all that apply)*

(month) (day) (year)

Unsure of month ❑[q1_10_1um]
Unsure of day ❑[q1_10_1ud]

OR

[q1_10_2]

NEVER SMOKED CIGARETTES ❑ ***If checked, SKIP TO 10B (Section B – top of page 4)***

Instructions for the Clinical Coordinator

Determine the following date range: ___ ___ / ___ ___ / ___ ___ ___ ___ *to* ___ ___ / ___ ___ / ___ ___ ___ ___

(1 year before the LMP date, mm/dd/yyyy) *(LMP date from Eligibility Form, mm/dd/yyyy)*

Based on the participant's response to Question 10A, check here if the date of the last cigarette was more than one year before the LMP date: ❑ [q1_10cc] ***If checked, SKIP TO 10B (Section B – top of page 4)***

11A. In the year before you became pregnant, how often did you smoke a cigarette: *(SHOW CARD A)*

[q1_11] *(circle one)*

None	0 →	*SKIP TO Interviewer's Script A*
Monthly or less	1	
2 to 4 days a month (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

12A. How many cigarettes did you smoke on a typical day when you were smoking in the year before you became pregnant:

Number of cigarettes: [q1_12] - [q1_12max]

(specify number OR range)

Interviewer's Script A

You said your last cigarette was on ___ ___ / ___ ___ / ___ ___ ___ ___. Let's look at the **30 days before** that date.

(month) (day) (year)

13A. How often did you smoke a cigarette in the 30 days before your last cigarette: *(SHOW CARD A)*

[q1_13] *(circle one)*

None during the 30 day period	0 →	*SKIP TO 10B (Section B – top of page 4)*
1 time	1	
2 to 4 days (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

14A. In the 30 days before your last cigarette, how many cigarettes did you have on a typical day when you smoked:

Number of cigarettes: [q1_14] - [q1_14max]

(specify number OR range)

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

Smoking and Tobacco Use History – *Section B (Hookah and Pipe)*

10B. If you ever smoked tobacco from a hookah or pipe, when was your last smoke:

[q1_10b_1 MM] (month) / DD] (day) / YYYY] (year)

(check all that apply)

Unsure of month ❑ [q1_10b_1um]

Unsure of day ❑ [q1_10b_1ud]

OR

[q1_10b_2]

NEVER SMOKED TOBACCO FROM A HOOKAH OR PIPE: ❑ ⟶ *If checked, SKIP TO 15*

Instructions for the Clinical Coordinator

Determine the following date range: ___ ___ / ___ ___ / ___ ___ ___ ___ (1 year before the LMP date, mm/dd/yyyy) *to* ___ ___ / ___ ___ / ___ ___ ___ ___ (LMP date from Eligibility Form, mm/dd/yyyy)

Based on the participant's response to Question 10B, check here if the date of the last smoke from a hookah or pipe was more than one year before the LMP date: ❑ ⟶ **[q1_10bcc], *SKIP TO 15***

11B. In the year before you became pregnant, how often did you smoke tobacco from a hookah or pipe: *(SHOW CARD A)*

[q1_11b] *(circle one)*

None	0 ⟶	*SKIP TO Interviewer's Script B*
Monthly or less	1	
2 to 4 days a month (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

12B. In the year before you became pregnant,

i. **how long was the duration of a smoking session on a typical day:** Number of hours: [q1_12b1] _ [q1_12b1max] (specify number OR range)

ii. **how many people shared the hookah or pipe, including yourself, on a typical day:** Number of people: [q1_12b2] [q1_12b2max] (specify number OR range)

Interviewer's Script B

You said your last smoke from a hookah or pipe was on ___ ___ (month) / ___ ___ (day) / ___ ___ ___ ___ (year).

Let's look at the **30 days before** that date.

13B. How often did you smoke tobacco from a hookah or pipe in the 30 days before your last smoke: *(SHOW CARD A)*

[q1_13b] *(circle one)*

None during the 30 day period	0 ⟶	*SKIP TO 15*
1 time	1	
2 to 4 days (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

14B. In the 30 days before your last smoke from a hookah or pipe:

i. **how long was the duration of a smoking session on a typical day:** Number of hours: [q1_14b1] _ [q1_14b1max] (specify number OR range)

ii. **how many people shared the hookah or pipe, including yourself, on a typical day:** Number of people: [q1_14b2] [q1_14b2max] (specify number OR range)

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

Environmental Smoking Exposure

15. How many people currently smoke tobacco inside the home where you live (excluding yourself):

Number of people: [q1_15] _ [q1_15max]
(specify number OR range)

16. Does your workplace allow smoking inside the building (not including a smoking room):

[q1_16] *(circle one)*

No	0
Yes	1

Interviewer's Script

Now, I am going to ask you about tobacco use. Because these questions are personal, any information you share with me will be kept confidential. Please refer to the calendar.

17. If you ever chewed, when was your last chew: [q1_17_1] ___ ___ / _____ / ___ ___ ___ ___
(month) (day) (year)

(check all that apply)
Unsure of month ❑ [q1_17_1um]
Unsure of day ❑ [q1_17_1ud]

OR

NEVER CHEWED ❑ [q1_17_2] ***If checked, SKIP TO Interviewer's Script D***

Instructions for the Clinical Coordinator

Determine the following date range: ___ ___ / ___ ___ / ___ ___ ___ ___ *to* ___ ___ / ___ ___ / ___ ___ ___ ___
(1 year before the LMP date, mm/dd/yyyy) *(LMP date from Eligibility Form, mm/dd/yyyy)*

Based on the participant's response to Question 17, check here if the date of the last chew was more than one year before the LMP date: ❑ [q1_17cc] ***If checked, SKIP TO Interviewer's Script D***

18. In the year before you became pregnant, how often did you chew tobacco: *(SHOW CARD A)*

[q1_18] *(circle one)*

None	0	→ *SKIP TO Interviewer's Script C*
Monthly or less	1	
2 to 4 days a month (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

19. How many dips or packets did you chew during a typical week when you were chewing in the year before you became pregnant:

Number of dips: [q1_19_1] _ [q1_19_1max]
(specify number OR range)

OR

Number of packets: [q1_19_2] _ [q1_19_2max]
(specify number OR range)

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

Interviewer's Script C

You said your last chew was on _____ / _____ / ___ _____ ___ . Let's look at the **30 days before** that date.
(month) (day) (year)

20. How often did you chew tobacco in the 30 days before your last chew: *(SHOW CARD A)*

[q1_20] *(circle one)*

None during the 30 day period	0	→ *SKIP TO Interviewer's Script D*
1 time	1	
2 to 4 days (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

21. In the 30 days before the date of your last chew, how many dips or packets did you chew in a typical week when you were chewing:

Number of dips: [q1_21_1] - [q1_21_1max]
(specify number OR range)

OR

Number of packets: [q1_21_2] - [q1_21_2max]
(specify number OR range)

Interviewer's Script D

Now, I am going to ask you about alcohol use. Because these questions are personal, any information you share with me will be kept confidential. Please refer to the calendar.

Alcohol Use

22. If you ever drank alcohol, when was your last drink: [q1_22_1] ___ ___ / ___ ___ / ___ ___ ___ ___
(month) (day) (year)

(check all that apply)
Unsure of month ❑ [q1_22_1um]
Unsure of day ❑ [q1_22_1ud]

OR

NEVER DRANK ❑ [q1_22_2] ***If checked, questionnaire is complete – enter Time Interview Ended on page 1.***

Instructions for the Clinical Coordinator

Determine the following date range: ___ ___ / ___ ___ / ___ ___ ___ ___ *to* ___ ___ / ___ ___ / ___ ___ ___ ___
(1 year before the LMP date, mm/dd/yyyy) *(LMP date from Eligibility Form, mm/dd/yyyy)*

Based on the participant's response to Question 22, check here if the date of the last drink was more than one year before the LMP date: ❑ [q1_22cc] ***If checked, questionnaire is complete – enter Time Interview Ended on page 1.***

23. In the year before you became pregnant, how often did you have a drink containing alcohol: *(SHOW CARD A)*

[q1_23] *(circle one)*

None	0	→ *SKIP TO Instructions for the Clinical Coordinator TLFB*
Monthly or less	1	*(only started drinking when they became pregnant)*
2 to 4 days a month (approx. once a week)	2	
2 to 3 days a week	3	
4 to 6 days a week	4	
7 days a week	5	

Coding Key:
-7 = Don't Know
-8 = Refused to Answer
-9 = Does Not Apply

Instructions for the Clinical Coordinator

Please refer to the Recruitment Interview TLFB Interviewer's Guide for interview's script in order to complete the next sections. The following fields are required: ***Type of Alcohol***, ***Size of Container***, *and* ***# Sharing****. If the* ***# of Containers*** *field requires additional computations, leave the field blank and complete the next two columns:* ***# of People purchasing or bringing*** *and* ***# of Containers purchased or brought by each****. DM-STAT will determine the* ***# of Containers****.*

24. What did you drink on a typical day when you were drinking in the year before you became pregnant:

Type of Alcohol *Alcohol Code* [q1_24a]	*Specify Type of Alcohol* [q1_24a_s1]	Size of Container *specify volume* [q1_24b]	*oz OR ml* [q1_24c]	# of Containers *specify number OR range* [q1_24d1] [q1_24d1max]	# Sharing *(including participant)* [q1_24e] [q1_24emax]	Ice or Frozen NO YES [q1_24g]
___	_____	____.__	___	___.__-___.__	___-___	0 1
___	_____	____.__	___	___.__-___.__	___-___	0 1
___	_____	____.__	___	___.__-___.__	___-___	0 1
___	_____	____.__	___	___.__-___.__	___-___	0 1

Total duration of drinking *(hours)*: ___ [q1_24f1]- ___ [q1_24f1max] *(specify number OR range)* ***OR*** Start Time: __ __ [q1_24f2] *(military time)* End Time: __ __ [q1_24f3] *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** ⟶ ☐ [q1_24h]

25. Think about the MOST you had to drink on any one day in the year before you became pregnant.

What did you drink on that day:

OR **Check if response is same as Question 24:** ☐ [q1_24_Same]

Type of Alcohol *Alcohol Code* [q1_25a]	*Specify Type of Alcohol* [q1_25a_s1]	Size of Container *specify volume* [q1_25b]	*oz OR ml* [q1_25c]	# of Containers *specify number OR range* [q1_25d1] [q1_25d1max]	# Sharing *(including participant)* [q1_25e] [q1_25emax]	Ice or Frozen NO YES [q1_25g]
___	_____	____.__	___	___.__-___.__	___-___	0 1
___	_____	____.__	___	___.__-___.__	___-___	0 1
___	_____	____.__	___	___.__-___.__	___-___	0 1
___	_____	____.__	___	___.__-___.__	___-___	0 1

Total duration of drinking *(hours)*: ___ [q1_25f1]- ___ [q1_25f1max] *(specify number OR range)* ***OR*** Start Time: __ __ [q1_25f2] *(military time)* End Time: __ __ [q1_25f3] *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** ⟶ ☐ [q1_25h]

Instructions for the Clinical Coordinator TLFB

Please refer to the Recruitment Interview TLFB Interviewer's Guide for interview's script and detailed instructions along with the TLFB Calendar in order to complete the next sections. Once completed, make sure to indicate ***Time Interview Ended*** *on page 1 of the Recruitment Interview.*

POST – RECRUITMENT INTERVIEW COMPLETION PROCEDURES

When the Participant's PASS visit has been completed in its entirety, transcribe the detailed alcohol information from the completed TLFB calendar page(s) onto the Recruitment Interview – Items 26 and 27. Place a Participant ID on the pages of the TLFB Calendar and keep with the Participant's records.

Item 26 Completion Instructions:

When transcribing information from the TLFB Calendar to Item 26, record the LMP recall period **Start Day**, **Start Month** and **Start Year** in the first row. Continue to list every date in the first column for the entire 30 day LMP recall period. Indicate which days were drinking days by circling **NO = 0** or **YES = 1** and transcribe all **Special Occasion Codes** from the TLFB Calendar. Complete the Last Drinking Day recall period following the same steps. If the LMP and LDD days overlap, only transcribe information for non-overlapping days for the LDD recall period. For each **Unique Drinking Day**, assign a two digit number starting with 01. For all drinking days which the participant drank the same exact amount and for the same duration of time, record the same **Unique Drinking Day** number in the last column.

Item 27 Completion Instructions:

Complete detailed alcohol information for each **Unique Drinking Day** number by transcribing all information from the TLFB calendar to Item 27. Use as many Additional Sheets as necessary to record all detailed alcohol information.

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

26. TLFB Summary Sheet

Data Entry Document

Last Menstrual Period

[q1_26a_1] Start Month __ __ [q1_26a_2] Start Day __ __ [q1_26a_3] Start Year __ __

	Date	Drinking Day (circle one for each item) NO	YES	Occasion Code (specify code)	Unique Drinking Day # (specify number)
1.	[q1_26a1a]	[q1_26a1b]		[q1_26a1c]	[q1_26a1d]
2.	[q1_26a2a]	[q1_26a2b]		[q1_26a2c]	[q1_26a2d]
3.	[q1_26a3a]	[q1_26a3b]		[q1_26a3c]	[q1_26a3d]
4.	[q1_26a4a]	[q1_26a4b]		[q1_26a4c]	[q1_26a4d]
5.	[q1_26a5a]	[q1_26a5b]		[q1_26a5c]	[q1_26a5d]
6.	[q1_26a6a]	[q1_26a6b]		[q1_26a6c]	[q1_26a6d]
7.	[q1_26a7a]	[q1_26a7b]		[q1_26a7c]	[q1_26a7d]
8.	[q1_26a8a]	[q1_26a8b]		[q1_26a8c]	[q1_26a8d]
9.	[q1_26a9a]	[q1_26a9b]		[q1_26a9c]	[q1_26a9d]
10.	[q1_26a10a]	[q1_26a10b]		[q1_26a10c]	[q1_26a10d]
11.	[q1_26a11a]	[q1_26a11b]		[q1_26a11c]	[q1_26a11d]
12.	[q1_26a12a]	[q1_26a12b]		[q1_26a12c]	[q1_26a12d]
13.	[q1_26a13a]	[q1_26a13b]		[q1_26a13c]	[q1_26a13d]
14.	[q1_26a14a]	[q1_26a14b]		[q1_26a14c]	[q1_26a14d]
15.	[q1_26a15a]	[q1_26a15b]		[q1_26a15c]	[q1_26a15d]
	[q1_26a_LMP_a]	[q1_26a_LMP_b]		[q1_26a_LMP_c]	[q1_26a_LMP_d]
16.	[q1_26a16a]	[q1_26a16b]		[q1_26a16c]	[q1_26a16d]
17.	[q1_26a17a]	[q1_26a17b]		[q1_26a17c]	[q1_26a17d]
18.	[q1_26a18a]	[q1_26a18b]		[q1_26a18c]	[q1_26a18d]
19.	[q1_26a19a]	[q1_26a19b]		[q1_26a19c]	[q1_26a19d]
20.	[q1_26a20a]	[q1_26a20b]		[q1_26a20c]	[q1_26a20d]
21.	[q1_26a21a]	[q1_26a21b]		[q1_26a21c]	[q1_26a21d]
22.	[q1_26a22a]	[q1_26a22b]		[q1_26a22c]	[q1_26a22d]
23.	[q1_26a23a]	[q1_26a23b]		[q1_26a23c]	[q1_26a23d]
24.	[q1_26a24a]	[q1_26a24b]		[q1_26a24c]	[q1_26a24d]
25.	[q1_26a25a]	[q1_26a25b]		[q1_26a25c]	[q1_26a25d]
26.	[q1_26a26a]	[q1_26a26b]		[q1_26a26c]	[q1_26a26d]
27.	[q1_26a27a]	[q1_26a27b]		[q1_26a27c]	[q1_26a27d]
28.	[q1_26a28a]	[q1_26a28b]		[q1_26a28c]	[q1_26a28d]
29.	[q1_26a29a]	[q1_26a29b]		[q1_26a29c]	[q1_26a29d]
30.	[q1_26a30a]	[q1_26a30b]		[q1_26a30c]	[q1_26a30d]

Last Drinking Day

[q1_26b_1] Start Month __ __ [q1_26b_2] Start Day __ __ [q1_26b_3] Start Year __ __

	Date	Drinking Day (circle one for each item) NO	YES	Occasion Code (specify code)	Unique Drinking Day # (specify number)
1.	[q1_26b1a]	[q1_26b1b]		[q1_26b1c]	[q1_26b1d]
2.	[q1_26b2a]	[q1_26b2b]		[q1_26b2c]	[q1_26b2d]
3.	[q1_26b3a]	[q1_26b3b]		[q1_26b3c]	[q1_26b3d]
4.	[q1_26b4a]	[q1_26b4b]		[q1_26b4c]	[q1_26b4d]
5.	[q1_26b5a]	[q1_26b5b]		[q1_26b5c]	[q1_26b5d]
6.	[q1_26b6a]	[q1_26b6b]		[q1_26b6c]	[q1_26b6d]
7.	[q1_26b7a]	[q1_26b7b]		[q1_26b7c]	[q1_26b7d]
8.	[q1_26b8a]	[q1_26b8b]		[q1_26b8c]	[q1_26b8d]
9.	[q1_26b9a]	[q1_26b9b]		[q1_26b9c]	[q1_26b9d]
10.	[q1_26b10a]	[q1_26b10b]		[q1_26b10c]	[q1_26b10d]
11.	[q1_26b11a]	[q1_26b11b]		[q1_26b11c]	[q1_26b11d]
12.	[q1_26b12a]	[q1_26b12b]		[q1_26b12c]	[q1_26b12d]
13.	[q1_26b13a]	[q1_26b13b]		[q1_26b13c]	[q1_26b13d]
14.	[q1_26b14a]	[q1_26b14b]		[q1_26b14c]	[q1_26b14d]
15.	[q1_26b15a]	[q1_26b15b]		[q1_26b15c]	[q1_26b15d]
16.	[q1_26b16a]	[q1_26b16b]		[q1_26b16c]	[q1_26b16d]
17.	[q1_26b17a]	[q1_26b17b]		[q1_26b17c]	[q1_26b17d]
18.	[q1_26b18a]	[q1_26b18b]		[q1_26b18c]	[q1_26b18d]
19.	[q1_26b19a]	[q1_26b19b]		[q1_26b19c]	[q1_26b19d]
20.	[q1_26b20a]	[q1_26b20b]		[q1_26b20c]	[q1_26b20d]
21.	[q1_26b21a]	[q1_26b21b]		[q1_26b21c]	[q1_26b21d]
22.	[q1_26b22a]	[q1_26b22b]		[q1_26b22c]	[q1_26b22d]
23.	[q1_26b23a]	[q1_26b23b]		[q1_26b23c]	[q1_26b23d]
24.	[q1_26b24a]	[q1_26b24b]		[q1_26b24c]	[q1_26b24d]
25.	[q1_26b25a]	[q1_26b25b]		[q1_26b25c]	[q1_26b25d]
26.	[q1_26b26a]	[q1_26b26b]		[q1_26b26c]	[q1_26b26d]
27.	[q1_26b27a]	[q1_26b27b]		[q1_26b27c]	[q1_26b27d]
28.	[q1_26b28a]	[q1_26b28b]		[q1_26b28c]	[q1_26b28d]
29.	[q1_26b29a]	[q1_26b29b]		[q1_26b29c]	[q1_26b29d]
30.	[q1_26b30a]	[q1_26b30b]		[q1_26b30c]	[q1_26b30d]
	[q1_26b_LDD_a]	[q1_26b_LDD_b]		[q1_26b_LDD_c]	[q1_26b_LDD_d]

Occasion Codes:
PD = Pay Day
SO = Special Occasion

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

27. TLFB Summary Sheet – Unique Drinking Days

Data Entry Document

Unique Drinking Day #: 01 [q1_27_1]

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol* [q1_27a] [q1_27a_s1]	Size of Container *specify volume* *oz OR ml* [q1_27b] [q1_27c]	# of Containers *specify number OR range* [q1_27d1] [q1_27d1max]	# Sharing *(including participant)* [q1_27e] [q1_27emax]	Ice or Frozen NO YES [q1_27g]
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1

Total duration of drinking *(hours)*: [q1_27f1] ___ . ___ - [q1_27f1max] ___ . ___ *(specify number OR range)* ***OR*** Start Time: [q1_27f2] ___ : ___ *(military time)* End Time: [q1_27f3] ___ : ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** → ☐ [q1_27h]

Unique Drinking Day #: 02

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	Size of Container *specify volume* *oz OR ml*	# of Containers *specify number OR range*	# Sharing *(including participant)*	Ice or Frozen NO YES
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1

Total duration of drinking *(hours)*: ___ . ___ - ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ : ___ *(military time)* End Time: ___ : ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** → ☐

Unique Drinking Day #: 03

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	Size of Container *specify volume* *oz OR ml*	# of Containers *specify number OR range*	# Sharing *(including participant)*	Ice or Frozen NO YES
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1

Total duration of drinking *(hours)*: ___ . ___ - ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ : ___ *(military time)* End Time: ___ : ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** → ☐

Unique Drinking Day #: 04

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	Size of Container *specify volume* *oz OR ml*	# of Containers *specify number OR range*	# Sharing *(including participant)*	Ice or Frozen NO YES
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0 1

Total duration of drinking *(hours)*: ___ . ___ - ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ : ___ *(military time)* End Time: ___ : ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** → ☐

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

27. TLFB Summary Sheet – Unique Drinking Days *(Additional Sheet)* *Data Entry Document*

Unique Drinking Day #: ___ ___ **Type of Alcohol** *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** NO YES
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out. ⟶ ❑**

Unique Drinking Day #: ___ ___ **Type of Alcohol** *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** NO YES
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out. ⟶ ❑**

Unique Drinking Day #: ___ ___ **Type of Alcohol** *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** NO YES
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out. ⟶ ❑**

Unique Drinking Day #: ___ ___ **Type of Alcohol** *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** NO YES
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1
___ ___ ___ ______________	___ ___ ___ . ___ ___ ___	___ ___ . ___ - ___ ___ . ___	___ ___ - ___ ___	0 1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out. ⟶ ❑**

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

27. TLFB Summary Sheet – Unique Drinking Days *(Additional Sheet)*

Data Entry Document

Unique Drinking Day #: ___ ___

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** **NO**	 **YES**
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** ⟶ ☐

Unique Drinking Day #: ___ ___

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** **NO**	 **YES**
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** ⟶ ☐

Unique Drinking Day #: ___ ___

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** **NO**	 **YES**
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** ⟶ ☐

Unique Drinking Day #: ___ ___

Type of Alcohol *Alcohol Code* *Specify Type of Alcohol*	**Size of Container** *specify volume* *oz OR ml*	**# of Containers** *specify number OR range*	**# Sharing** *(including participant)*	**Ice or Frozen** **NO**	 **YES**
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1
___ ___ ___ ________________	____ . __ ___	___ . __ - ___ . __	___ - ___	0	1

Total duration of drinking *(hours)*: ___ ___ . ___ - ___ ___ . ___ *(specify number OR range)* ***OR*** Start Time: ___ ___ : ___ ___ *(military time)* End Time: ___ ___ : ___ ___ *(military time)*

OR **Check if unable to compute duration of drinking due to black out or passing out.** ⟶ ☐

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

Interviewer's Script E

Now, I am going to ask you about drug use. Because these questions are personal, any information you share with me will be kept confidential. Please refer to the calendar.

Drug Use

28. Have you ever used:

		NO	YES	
a.	**Marijuana (hashish, pot, grass, weed, dope or dagga)** [q1_28a]	0	1	→ ***If NO, skip to Question 28.b***

1. What is the date you last used: [q1_28a MM] __ / [DD] __ / [YYYY] __ __ (month) (day) (year) [q1_28a um] [ud] Unsure of: ❑ Month ❑ Day

2. How often did you use in the 30 days before that date: *(SHOW CARD A)*

	(circle one)
None during the 30 day period	0
1 time	1
2 to 4 days (approx. once a week)	2
2 to 3 days a week	3
4 to 6 days a week	4
7 days a week	5

[q1_28a2]

3. In the year before you became pregnant, how often did you use: *(SHOW CARD A)*

	(circle one)
None	0
Monthly or less	1
2 to 4 days a month (approx. once a week)	2
2 to 3 days a week	3
4 to 6 days a week	4
7 days a week	5

[q1_28a3]

		NO	YES	
b.	**Methamphetamines (crank, uppers, tik, chalk, meth, crystal meth, ice, glass, speed or quick)** [q1_28b]	0	1	→ ***If NO, skip to Question 28.c***

1. What is the date you last used: [q1_28b MM] __ / [DD] __ / [YYYY] __ __ (month) (day) (year) [q1_28b um] [ud] Unsure of: ❑ Month ❑ Day

2. How often did you use in the 30 days before that date: *(SHOW CARD A)*

	(circle one)
None during the 30 day period	0
1 time	1
2 to 4 days (approx. once a week)	2
2 to 3 days a week	3
4 to 6 days a week	4
7 days a week	5

[q1_28b2]

3. In the year before you became pregnant, how often did you use: *(SHOW CARD A)*

	(circle one)
None	0
Monthly or less	1
2 to 4 days a month (approx. once a week)	2
2 to 3 days a week	3
4 to 6 days a week	4
7 days a week	5

[q1_28b3]

Coding Key:
(7) = Don't Know
(8) = Refused to Answer
(9) = Does Not Apply

28. Have you ever used *(continued)*:

	NO	YES	If YES, date last used (month / day / year)
c. **Hairspray-mixers**	[q1_28c] 0	1	[q1_28c MM] / DD] / YYYY] Unsure of: ❑ Month [um] ❑ Day [ud]
d. **Other #1** [q1_28d_s1] _____ *(please specify)* ...	[q1_28d] 0	1	[q1_28d MM] / DD] / YYYY] Unsure of: ❑ Month [um] ❑ Day [ud]
e. **Other #2** [q1_28e_s1] _____ *(please specify)* ...	[q1_28e] 0	1	[q1_28e MM] / DD] / YYYY] Unsure of: ❑ Month [um] ❑ Day [ud]
f. **Other #3** [q1_28f_s1] _____ *(please specify)* ...	[q1_28f] 0	1	[q1_28f MM] / DD] / YYYY] Unsure of: ❑ Month [um] ❑ Day [ud]
g. **Other #4** [q1_28g_s1] _____ *(please specify)* ...	[q1_28g] 0	1	[q1_28g MM] / DD] / YYYY] Unsure of: ❑ Month [um] ❑ Day [ud]

Coding Key:
⑦ = Don't Know
⑧ = Refused to Answer
⑨ = Does Not Apply

ADDENDUM B – INFORMED CONSENT DOCUMENT

Stellenbosch University (SU)

Informed Consent document

Title: Assessment of the validity and reliability of the PASS South African Diet Screener

Principal Investigators: Janetta Harbron, MSc, RD
University of Cape Town
Hein Odendaal, FRCOG, MD
Department of Obstetrics and Gynaecology
Coen Groenewald, FCOG(CMSA), MD
Project manager
Celeste de Bruyn, RD
Masters student, University of Cape Town

Statement of Research

It is a basic ethical principle that a participant who is to participate in the research must give her informed consent to such participation. This consent must be based on the understanding of the nature and risks of the research. This document provides information important for this understanding. Research projects include only participants who choose to take part. Please take your time to make your decision. If at any time you have questions, please ask.

What is the purpose of this study? The purpose of this ancillary or sub-study is to assess the reliability and validity of the Diet Screener (questionnaire) designed for women enrolled in the Safe Passage Study in South Africa. Validation means to confirm that the data collected by the Diet Screener accurately represents the type and quantity of food you eat on a regular basis. Reliability refers to the 'repeatability' of the Screener, the degree to which the same conclusion is reached with multiple tests on the same person in the same conditions.

How many people will participate? This study will be done on a sub-set of the participants currently enrolled in the Safe Passage Study. Approximately 150 women enrolled and presenting for their first research visit (i.e. recruitment) between 14-20 weeks of pregnancy, will be invited to participate in this sub-study. Women will be enrolled from the Bishop Lavis Midwife Obstetric Unit. You have been invited to participate because you are a Safe Passage study participant.

How long will I be in this study? Your participation in this sub-study will last for the next four weeks. In this study, the assessments will be done with an interviewer at the clinic. A total of three 24-hour recalls, three Diet Screeners and two other forms will be filled in with an interviewer over three visits to the clinic. Each visit will last 30-45 minutes.

What will happen during this study? At the time of your 14-20 weeks research visit (i.e. recruitment), you will be asked to complete the Diet Screener with the help of an interviewer. The Screener will ask questions about your eating habits around the time of conception. This will take about 20 minutes. The interviewer will then complete another food questionnaire which will ask you a standard set of questions on food and drinks that you have had in the past 24 hours. This will take approximately 20 minutes. Then we will organize with you when you will need to come to the clinic again for the follow-up visits to complete the next questionnaires. During the next 4 weeks, you will need to come to the clinic on 2 separate occasions to be interviewed again by a Dietitian. The Dietitian will complete the same two food questionnaires during these sessions. To help you with estimating the food portion sizes during these interviews, there will be pictures available from the interviewer that you can look at during the interview.

What are the risks of the study? There are no known risks to participation in this study. At any time during the interview, you may refuse to answer any of the questions and you may take a break. You may also stop your participation at any time. Your decision whether or not to participate in this sub-study will have no effect on your participation in the Safe Passage Study.

What are the benefits of this study? There are no direct benefits to you from participating in this sub-study. However, you will be helping us learn more about eating habits of pregnant women in the Tygerberg area.

Are there alternatives to participating in this study? The alternative is not to participate in this study.

Will it cost me anything to be in this study? There is no cost for you to be in this study.

Will I be paid for participating? Yes. You will receive R100 for each completed follow-up interview. The money will be given to you at the end of the interviews. If you complete all three assessments, this would total vouchers worth R200.

Will information about me be kept confidential? The records of this study will be kept private. In any report about this study that might be published, you will not be identified. Several people may look at this data including: the individuals from the National Institutes of Health that are funding the research, the Committee for Human Research of Stellenbosch University, the federally appointed Advisory and Safety Monitoring Board, the data coordinating center, and researchers that are part of the project.

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. Confidentiality will be maintained by assigning you a subject identification number. Copies of all questionnaires will be labeled with this number and not your name. All information with your name and other identifying information (e.g. phone number) will be kept secret.

Will I or my doctor be given research results? It is very important that you understand that the results from the interviews will not be given to you or your doctor, even if you ask that this be done. The results from the interviews will not be sent to your hospital. These assessments will be done for specific research purposes and are not designed to or intended to find medical or other problems.

Who do I call if I have questions? If you have any questions now or in the future, you may contact the Study Office (021-938-4748), Prof. Hein Odendaal (021-938-9601) or Janetta Harbron (021-406-6310). If you have questions or concerns about your rights as a research participant, you can contact the Committee for Human Research of the Stellenbosch University (021-938-9207).

Your signature indicates that this research study has been explained to you, your questions have been answered, and that you agree to participate in this ancillary study. You will receive a copy of this form.

_____
Name of the participant

_____ _____
Signature of Participant Date

I have explained the risks, benefits and the procedures involved with participation in this study.

_____ _____
Signature of study representative Date

ADDENDUM C – FOOD ITEMS CONSUMED BY STUDY SAMPLE

Apples	Custard	Melon	Pumpkin
Apricot	Dairy-fruit juice mix	Milk	Rooibos tea
Avocado	Doughnut	Milk tart	Roti
Baby marrow	Dried fruit	Mixed vegetables	Rusk, home-made, buttermilk, white (HM)
Bacon	Eggs	Muffin	Salt, table
Banana	Fish	Mushrooms	Salt, table, iodised
Beans	Fish paste	Mutton	Samoosa
Beef	Fruit juice	Naartjie	Samp and beans, 1:1
Beetroot	Garlic	Nectarine, raw	Sauce, savoury
Broccoli	Gem squash	Nesquik powder	Sauce, sweet
Brown bread	Grapes	Nuts	Sausage
Brown rice	Gravy	Oats	Soup
Butter	Green beans	Offal, cooked (tripe / brawn / brain / tongue)	Spinach
Butternut	Guava	Oil	Split peas
Cabbage	Ham	Onion	Spread (sandwich), pork / beef
Cake	High fiber cereal	Oranges	Strawberry
Carbonated drink	Hot cross bun	Parsnip, boiled	Sugar
Carrots	Ice cream	Pasta	Sweet potato
Cauliflower	Icing	Pawpaw	Sweets
Cereal	Jam	Peaches	Tart
Cheese	Jam tart	Peanut butter	Tea
Chicken	Jelly	Peanuts, raw	Tomato
Chips	Koeksister	Pear	Tomato paste
Chocolate	Lemon	Peas	Toppers, cooked
Chutney, fruit	Lemon juice, fresh	Peppers	Turkey
Coconut	Lentils	Pie	Vetkoek
Coffee	Lettuce	Pineapples	Water
Cold drink	Liver	Pizza	Watermelon
Cookies	Maize meal	Plum	Wheat flour, cake flour
Crackers	Mango	Polony	White bread
Cream	Margarine	Popcorn	White rice
Creamer (coffee and tea)/Non-dairy powder	Marmite, yeast extract	Pork	White sauce
Crisps	Mayonnaise	Potato	Wholewheat bread
Cucumber	Mealie	Pudding, trifle	Yoghurt