

2013

# Dietary change after gestational diabetes mellitus for prevention of type two diabetes mellitus

Nancy Cinnadaio  
*University of Wollongong*

---

## Recommended Citation

Cinnadaio, Nancy, Dietary change after gestational diabetes mellitus for prevention of type two diabetes mellitus, Masters of Science - Research thesis, School of Health Sciences, University of Wollongong, 2013. <http://ro.uow.edu.au/theses/4435>

## **UNIVERSITY OF WOLLONGONG**

### **COPYRIGHT WARNING**

You may print or download ONE copy of this document for the purpose of your own research or study. The University does not authorise you to copy, communicate or otherwise make available electronically to any other person any copyright material contained on this site. You are reminded of the following:

Copyright owners are entitled to take legal action against persons who infringe their copyright. A reproduction of material that is protected by copyright may be a copyright infringement. A court may impose penalties and award damages in relation to offences and infringements relating to copyright material. Higher penalties may apply, and higher damages may be awarded, for offences and infringements involving the conversion of material into digital or electronic form.

**UNIVERSITY OF  
WOLLONGONG**



**School of Health Sciences**

**DIETARY CHANGE AFTER GESTATIONAL DIABETES MELLITUS FOR  
PREVENTION OF TYPE TWO DIABETES MELLITUS**

**Nancy Cinnadaio**

**"This thesis is presented as part of the requirements for the  
award of the Degree of Masters of Science Research  
of the  
University of Wollongong"**

**December 2013**

## ABSTRACT

Women with a history of gestational diabetes mellitus (GDM) are at an increased risk of developing type two diabetes mellitus (T2DM). Nutritional guidelines for the prevention of T2DM recommend a reduction of 7% of body weight if overweight, reduction in the intake of fat and an increase in the intake of dietary fibre. Current research indicates overweight and obesity and consumption of excessive dietary fat, are prevalent problems amongst women with prior GDM. Thus, there is a need to develop interventions targeted towards lifestyle change in these women. This thesis is a secondary analysis of dietary data from a pilot behavioural lifestyle intervention in women with prior GDM: the Healthy Living Program after Gestational Diabetes Mellitus (HeLP GDM) study. The following two central hypotheses were tested:

1. dietary change was achieved in the HeLP GDM study
2. the associated dietary intakes collected from two dietary assessment methods provided relatively comparable estimates of macronutrient intake.

*Methods:* In the HeLP GDM study 59 overweight or obese women with prior GDM were randomised to either a six month telephone diet and physical activity behavioural change intervention group or a no treatment control group. For this thesis changes in intake of energy, fat and fibre from baseline to six months were analysed using nutrient estimates obtained from three day estimated food records and seven day telephone diet histories. Additionally change in body weight from baseline to six months was assessed using self-reported weight. To assess relative validity baseline diet history and food record paired data were compared for group differences. Change in bias from baseline to six months was assessed using paired t tests with individual precision and presence of systematic error determined by Bland Altman scatterplots.

*Results:* At six months diet history analysis demonstrated the intervention group compared to the control group reduced energy by -1251 kJ (95%CI -2169, -332), total fat by -16.8 g (95% CI -28.5, -5.0) and saturated fat by -7.2 g (95% CI -12.2, -2.1). Analysis of food record data demonstrated only a relative reduction in saturated fat of -4.7 g (95% CI -9.0, -0.4) in the intervention group. The reduction in energy by -849 kJ (95% CI -1774, 76) and total fat by -

11.5 kJ (95% CI -23.2, 0.2) approached but failed to achieve statistical significance. Intervention participants were more likely than control participants to consume an intake of total fat less than  $\leq 30$  of total energy [9.2 greater odds (95% CI 2.1, 41.3)] and 15g per 4200kJ of fibre [8.5 greater odds (95% CI 2.0, 35.4)] identified by the diet history but not the food record. Greater reduction in energy intake by the intervention group was confirmed by a small but clinically significant reduction in weight of -2.4 kg (95% CI -4.5, -0.3).

Relative validity analysis identified mean bias between paired food record and diet history estimates for all variables were not significantly different at baseline. For all variables no significant change was found in bias at six months. However for each nutrients there was a large standard deviation of the mean bias which indicated low precision between dietary assessment estimates. No linear relationships were demonstrated between bias and mean intake indicating an absence of systematic error for all variables.

*Conclusion:* Dietary intervention targeted at overweight or obese women with prior GDM resulted in short term decreases in intake of energy, total fat and saturated fat, while an appropriate intake of dietary fibre was maintained. Furthermore the reduction in energy intake was accompanied by a decrease in body weight. Overall data obtained from both the diet history and food record compared well at the group level and both methods identified similar dietary change trends.

## **ACKNOWLEDGMENTS**

Firstly I would like to thank my supervisors Professor Linda Tapsell and Dr. Yasmine Probst for their constant patience, understanding and encouragement to keep writing.

Thank you to the members of the HeLP GDM pilot collaboration team: Associate Professor Wah Cheung, Dr. Hidde van der Ploeg, Associate Professor Ben Smith, Professor Adrian Bauman and Melissa Gwizd as without this study there would be no thesis.

To Roslyn Hogan, as together we approached almost one thousand women to participate in the HeLP GDM study.

To Dr Marijka Batterham for assistance in all statistical related matters.

A thank you to my wonderful parents Maria and Nicola for their endless support and constant hours of babysitting. Without their commitment writing this thesis would not have been possible. Thank you for believing in me.

Thank you to my partner Rik for his patience and support.

Last but not least I dedicate this thesis to my beautiful children Sasha and Siana, to show you if something is important enough see it through.

## Contents

ABSTRACT .....	2
ACKNOWLEDGMENTS .....	4
LIST OF FIGURES .....	7
LIST OF TABLES .....	8
GLOSSARY .....	9
CHAPTER ONE - LITERATURE REVIEW.....	11
Introduction .....	11
GDM and T2DM: what is the link .....	11
Evidence for the role of diet in prevention of T2DM .....	14
Total fat and saturated fat .....	15
Polyunsaturated fat .....	18
Monounsaturated fat .....	19
Dietary Fibre.....	21
Food and dietary patterns.....	24
Dietary change and prevention of T2DM.....	26
Summary of dietary change and prevention of T2DM.....	32
Dietary assessment methods.....	32
The validity of dietary assessment methods.....	33
Food records.....	35
Diet history interview .....	36
Summary of dietary assessment methods.....	39
Literature review summary.....	40
Chapter Two - Research Design and Hypothesis.....	41
Introduction .....	41
Aims and hypotheses.....	41
Thesis hypotheses.....	41
Thesis aims .....	42
The HeLP GDM study: a summary.....	42
Study setting.....	43
Selection criteria.....	43
Pilot phases .....	44
Pre - screen.....	44
Baseline assessment.....	45

Six month assessment.....	45
Randomisation.....	47
The intervention .....	47
Ethics.....	49
CHAPTER THREE - DIETARY CHANGE AFTER GESTATIONAL DIABETES MELLITUS IN THE HeLP GDM STUDY .....	50
Introduction .....	50
Method .....	50
Dietary assessment.....	50
Anthropometry.....	51
Data Entry.....	52
Statistical analysis.....	54
Results.....	55
Discussion .....	68
Limitations and strengths of the current dietary analysis .....	72
CHAPTER FOUR - RELATIVE VALIDITY OF DIETARY ASSESSMENT IN THE HeLP GDM STUDY .....	76
Introduction .....	76
Method .....	76
Statistical analysis.....	76
Results.....	77
Discussion .....	86
CHAPTER FIVE - CONCLUSIONS AND RECOMMENDATIONS.....	91
Conclusion.....	91
Recommendations for the future.....	92
REFERENCES.....	94
APPENDIX A: PROFORMA FOR PHYSICAL ACTIVITY COUNSELLING IN THE HeLP GDM STUDY.....	101
APPENDIX B: PROFORMA FOR DIETARY COUNSELLING IN THE HeLP GDM STUDY .....	116
APPENDIX C: SEVEN DAY TELEPHONE DIET HISTORY PROFORMA.....	128
APPENDIX D: THREE DAY FOOD RECORD PROFORMA .....	136



## LIST OF FIGURES

Figure 1: Plan for implementation of the HeLP GDM study .....	46
Figure 2: Eligibility, recruitment, retention and completion of the HeLP GDM study with shaded cells showing the dietary sub-study of this thesis .....	57
Figure 3 : Bland- Altman plot: agreement of estimated energy intake between a seven day telephone diet history and three day estimated food record at baseline .....	80
Figure 4: Bland- Altman plot: agreement in estimated total fat intake between a seven day telephone diet history and three day estimated food record at baseline .....	80
Figure 5: Bland- Altman plot: agreement in estimated saturated fat intake between a seven day telephone diet history and three day estimated food record at baseline .....	81
Figure 6: Bland- Altman plot: agreement in estimated fibre intake between a seven day telephone diet history and three day estimated food record at baseline .....	81
Figure 7: Bland- Altman plot: agreement in estimated %E intake from total fat between a seven day telephone diet history and three day estimated food record at baseline .....	82
Figure 8: Bland- Altman plot: agreement in estimated %E intake from saturated fat between a seven day telephone diet history and three day estimated food record at baseline .....	82
Figure 9: Bland- Altman plot: agreement in estimated energy intake between a seven day telephone diet history and three day estimated food record at six months .....	83
Figure 10: Bland- Altman plot: agreement in estimated total fat intake between a seven day telephone diet history and three day estimated food record at six months.....	83
Figure 11: Bland- Altman plot: agreement in estimated saturated fat intake between a seven day telephone diet history and three day estimated food record at six months.....	84
Figure 12: Bland- Altman plot: agreement in estimated fibre intake between a seven day telephone diet history and three day estimated food record at six months .....	84
Figure 13: Bland- Altman plot: agreement in estimated %E intake from total fat between a seven day telephone diet history and three day estimated food record at six months.....	85
Figure 14: Bland- Altman plot: agreement in estimated %E intake from saturated fat between a seven day telephone diet history and three day estimated food record at six months .....	85

## LIST OF TABLES

Table 1: Relative validity between self reported weight and measured weight at baseline.....	55
Table 2: Baseline demographic data of participants at enrolment and those remaining in the trial at six months.....	58
Table 3: Baseline anthropometry: all participants and remaining participants at six months .....	59
Table 4: Baseline assessment of the dietary stages of change: all participants and remaining participants at six months.....	59
Table 5: Assessment of macronutrient intake via the seven day diet history by experimental group at baseline and six months: intention to treat.....	60
Table 6 Assessment of macronutrient intake via the seven day diet history by experimental group at baseline and six months: completers .....	61
Table 7: Assessment of macronutrient intake via the three day food record by experimental group at baseline and six months: intention to treat.....	62
Table 8: Assessment of macronutrient intake via the three day food record by experimental group at baseline and six months: completers .....	63
Table 9 Assessment of weight and body mass index by experimental design at baseline and six months: intention to treat and completers .....	65
Table 10: Odds ratios for meeting dietary targets assessed by the seven day diet history and three day food record by experimental group at baseline and six months: intention to treat .....	66
Table 11: Odds ratios for meeting dietary targets assessed by the seven day diet history and three day food record by experimental group at baseline and six months: completers .....	67
Table 12: Relative validity of energy and macronutrient intake between the seven day telephone diet history and three day estimated food record at baseline and six months.....	79

## **GLOSSARY**

AHS	Australian Health Survey
AIHW	Australian Institute of Health and Welfare
BA	Bland Altman
BMI	Body Mass Index
DM	Diabetes Mellitus
DLW	Doubly Labelled Water
DPP	Diabetes Prevention Program
DPS	Diabetes Prevention Study
EHC	Euglycaemic-Hyperinsulinaemic Clamp
EE	Energy Expenditure
EI	Energy Intake
FFQ	Food Frequency Questionnaire
FR	Food Record
GDM	Gestational Diabetes Mellitus
HCF	High Cereal Fibre
HOMA-IR	Homeostasis Model Assessment of Insulin Resistance
HP	High Protein
IGT	Impaired Glucose Tolerance
IS	Insulin Sensitivity
ITT	Intention to Treat
IVGTT	Intravenous Glucose Tolerance Test
LOA	Limit of Agreement
MW	Measured Weight
MUFA	Monounsaturated Fatty Acid
NI	Nitrogen Intake
OGTT	Oral Glucose Tolerance Test

PA	Physical Activity
PSEA	Portion Size Estimation Aid
PUFA	Polyunsaturated Fatty Acids
RCT	Randomised Controlled Trial
RDM	Recent Diabetes Mellitus
RR	Relative Risk
SDB	Social Desirability Bias
SFA	Saturated Fatty Acid
SRW	Self Reported Weight
T1DM	Type 1 Diabetes Mellitus
T2DM	Type 2 Diabetes Mellitus
UDM	Undiagnosed Diabetes Mellitus
UN	Urinary Nitrogen
WHO	World Health Organisation
%E	Percent of Total Energy

## **CHAPTER ONE - LITERATURE REVIEW**

### **Introduction**

Women with previous gestational diabetes mellitus (GDM) are at high risk of developing type two diabetes mellitus (T2DM) [1]. Insulin sensitivity (IS) measures which are used as surrogate markers for T2DM are strongly influenced by dietary fat and fibre intake.[2-5] Large randomised controlled trials (RCT) across different ethnic populations at risk of T2DM have consistently shown increased physical activity (PA) and a lower total fat, higher fibre diet reduces T2DM incidence.[6-9] Women with a history of GDM often do not consume a diet in line with current guidelines to reduce their risk of developing T2DM and studies have reported a number of barriers to achieving a healthy diet.[10-13] However, difficulties exist in accessing and retaining women postpartum in clinical trials[14-16] and despite their increased risk of T2DM, there are few dietary behavioural intervention trials targeting women postpartum with a history of GDM.[17-20] These key issues provide the foundation for the research presented in this thesis.

What follows is an in depth narrative literature review developed to outline key issues pertinent to the thesis. To develop the review a search strategy was used with Pubmed and the Cochrane database of systematic reviews. The following search terms were entered: GDM/gestational diabetes, diabetes mellitus, postpartum period/postnatal, dietary/diet/nutrition, dietary fats, dietary fibre, weight/weight loss, lifestyle.

As there are a limited number of studies published in the area of postpartum interventions in women with previous GDM no literature search limits were imposed. Reference and bibliographical lists in the articles retrieved were scanned for additional relevant publications. Previous relevant PhD and Masters theses which were freely available are also reviewed.

### **GDM and T2DM: what is the link**

Diabetes Mellitus (DM) is a broad term used to describe a disorder characterised by high blood glucose levels resulting from defects in insulin secretion, insulin action, or both.[21] The chronic hyperglycaemia of DM leads to dysfunction and failure of the eyes, kidneys,

nerves, heart and blood vessels.[21] There are four distinct categories of DM with the following definitions:

**Type 1 Diabetes Mellitus (T1DM):** a form of DM resulting from a cellular-mediated autoimmune destruction of the beta ( $\beta$ ) cells of the pancreas.[21]

**T2DM:** Individuals with T2DM suffer from insulin resistance. Insulin resistance occurs when normal concentrations of insulin produce a less than normal biological response.[22]. Additionally, in T2DM a relative rather than absolute insulin deficiency exists. T2DM is the most common form, contributing to 85% of the prevalence of diabetes.[21]

**GDM:** any degree of glucose intolerance with onset or first recognition during pregnancy.[21]

**Other specific types:** DM which arises from a collection of causes including: genetic causes, excess levels of certain hormones (growth hormone, cortisol, glucagon, epinephrine) which antagonise insulin action, drug or chemical induced DM, infections, diseases affecting the pancreas and uncommon forms of immune mediated DM[21]

The following review will, however, focus only on T2DM and GDM.

In 2000 an estimated 171 million people worldwide were living with DM with future global projections estimating the number will approximately double by 2030[23]. A 2006 forecast model from a community based patient cohort the Freemantle Diabetes Study, determined if the age specific and sex specific prevalence rates in Australia continue to rise; an extremely conservative estimate of the projected national direct health care costs of T2DM at 2051, is predicted to be \$2.6 billion.[24]

Measured blood glucose from the 2011–13 Australian Health Survey (AHS) revealed in 2011–12, 5.1% of Australians aged 18 years and over had T2DM. This comprised 4.2% with known T2DM and 0.9% with T2DM newly diagnosed from their test results conducted for

the survey.[25] The prevalence of T2DM has more than doubled since 1989-90.[26] However, it would appear testing for T2DM has improved as a previous national survey reported one undiagnosed case of T2DM for each known case.[27] In contrast 2011-13 figures indicate approximately one undiagnosed case of T2DM for every four diagnosed cases.[25] It has been projected that unless trends in T2DM are reversed by 2025 there will be two million Australians living with T2DM.[22]

A diagnosis of GDM transiently reveals the woman's predisposition to T2DM. Pregnancy is itself a period of increased insulin resistance. Increased insulin secretion required to maintain normal blood glucose levels during an insulin resistant state, is decreased in women with GDM.[28] According to the Australian Institute of Health and Welfare (AIHW), in 2005-2006 GDM was diagnosed in 4.6% of confinements among women aged 15-49 years.[29] Depending on ethnicity and diagnostic criteria, the incidence of GDM in the literature is quoted to be between 2.2 and 8.8% of the pregnant population.[30]

The incidence of GDM according to ethnicity is evident in Australia with the incidence as high as 10.5% in women reporting a country within South Asia as their country of birth.[31] While the prevalence of T2DM has increased, so too has the prevalence of GDM. A study from Melbourne's Mercy Hospital found between two five year intervals from 1979 to 1983 and 1984 to 1988 the incidence of GDM more than doubled from 3.3 to 7.5%.[32]

GDM and T2DM share similar non modifiable and modifiable risk factors. Risk factors such as age, ethnicity and family history cannot be changed while carrying excess weight can be. Body mass index (BMI) a common although not always accurately applied measure of body fatness, is positively associated with development of T2DM.[25] An individual with a BMI of  $\geq 25.00$ - $29.99 \text{ kg/m}^2$  is considered overweight with those measuring  $\geq 30.00 \text{ kg/m}^2$  considered obese.[33] A 10 year follow up from 1986 to 1996 from two large epidemiological studies the Nurses Health Study and the Health Professionals Follow Up study, found overweight individuals were more than three times more likely to develop T2DM compared with individuals of normal weight.[25] Additionally, the follow up found

morbidly obese individuals ( $\text{BMI} \geq 35.00 \text{ kg/m}^2$ ) were approximately 20 times more likely to develop T2DM.

A diagnosis of GDM itself is a risk factor for T2DM. A meta-analysis of women with prior GDM found the conversion rate to T2DM ranged from 2.6 to 70% over a period of six weeks to 28 year postpartum.[1] The cumulative incidence in this study was greatest within five years, reaching a plateau at ten years. However, a more recent large Australian retrospective cohort study found the cumulative incidence to continue past ten years, with 25% of the women developing T2DM at 15 years postpartum.[34] Women with prior GDM most at risk of developing T2DM are those with the highest glucose levels during pregnancy. Increasing BMI and insulin use have been inconsistently documented as long term predictors of risk in this population.[1, 34]

It has been estimated that up to one third of the cases of T2DM among parous women in Australia are associated with previous GDM.[30] As routine screening for GDM occurs in Australian hospitals, one third of women with T2DM are able to be identified earlier via a GDM pregnancy. Thus an ideal opportunity is presented to reduce the risk of T2DM in women who have already had antenatal diabetes education and theoretically should be aware of their future elevated risk.

### **Evidence for the role of diet in prevention of T2DM**

There have been a very limited number of RCTs conducted for longer than 12 months which have examined the effect of specific dietary patterns on T2DM risk. Given the complexities of conducting large scale nutrition intervention studies, evidence of the association between specific nutrients and prevention of T2DM have been based mainly on epidemiologic research and short term RCTs. The short term trials have used IS as a T2DM surrogate endpoint. IS is a measure of insulin responsiveness as well as insulin resistance.[35] Impaired IS is an intermediate step for development of T2DM. Hence strategies to prevent a reduction in IS may have a large impact on reducing populations affected with T2DM.[36]



Many macro and micro nutrients and dietary patterns have been investigated for their role in prevention or promotion of T2DM. In the 2003 World Health Organisation (WHO) technical report on recommendations for the prevention of T2DM dietary intake of saturated fat acid (SFA) was considered a probable risk factor for developing T2DM, while the intake of dietary fibre was considered a probable protective factor.[37] The American Diabetes Association (ADA) position statement on the nutritional recommendations for T2DM primary prevention in individuals at high risk specify that programs be recommended which emphasise the following lifestyle changes: weight loss if required, regular PA, reduction in calories and dietary fat to approximately 30% of total calories and an intake of 15g of fibre per 1000 calories.[38] These ADA nutritional recommendations are derived from several large RCTs which effectively demonstrated intensive lifestyle change significantly reduces the risk of T2DM.[6-9] As dietary fat and fibre targets have been the cornerstone for dietary change in relation to reduction of T2DM only a discussion of the relevance of these macronutrients will be further addressed below.

#### **Total fat and saturated fat**

The WHO 2003 technical report on diet nutrition and the prevention of chronic disease guidelines has suggested there is a *possible* increased risk of higher total fat intake and development of T2DM. While it has been stated that there is a probable increased risk of higher SFA intake and development of T2DM,[37] consistency of the existence of this relationship appears however to be dependent on research design. Animal studies and prospective cohort studies using biomarkers rather than dietary intakes consistently providing evidence of detrimental effects of high SFA on IS and diabetes risk.

Positive associations between total fat and SFA and diagnosis of T2DM were found in a cross sectional case control study of n =602, across six Mediterranean countries in Europe.[39] In the study the diets of individuals recently diagnosed with diabetes (RDM) and those with undiagnosed diabetes (UDM) were compared with sex, age, BMI, and centre-matched non diabetic individuals. The RDM and UDM groups relative to their healthy controls were found to have significantly higher intakes of total fat (30.2% versus 27.8% and 34.7%vs 30.4%, respectively) and SFA from animal fat sources (12.2% versus 10.8% and 14.2% versus 10.6%, respectively).

Furthermore, a cross sectional study in healthy individuals which examined the relationship between SFA content in muscle and measures of insulin resistance via the euglycaemic-hyperinsulinaemic clamp (EHC)<sup>1</sup> technique demonstrated SFA content of the muscle membrane was positively correlated to fasting serum insulin[40]. In another study of 30 non diabetic patients both lean and obese, the amount of the SFA palmitic acid and skeletal muscle triglycerides, were found to be determinants of impaired IS.[41] Although a 2012 systematic review and meta-analysis of cohort studies measuring dietary macronutrient intake and T2DM risk did not find total fat and SFA to be associated with T2DM risk,[42] it has been suggested that dietary components may have varying effects in individuals with different predisposing genes and/or lifestyles and at different stages in the natural history of T2DM. [43]

It must be noted that any systematic underreporting of fat intake in heavier individuals in combination with the positive association between obesity and T2DM would be expected to bias the relative risk estimates for fat intake and T2DM downward and potentially negate any existing association.[43] This seems to be reinforced by the more consistent results seen with prospective biomarker studies in which SFA levels have been associated with T2DM surrogate endpoints or T2DM incidence. Long term prospective studies of sample sizes ranging from n= 895 to over n= 3500 in both genders across Finland, Sweden, America and Australia, have demonstrated significant associations between a high proportion of SFAs in serum cholesterol esters and phospholipids and T2DM incidence.[44-47].

Animal studies have consistently demonstrated impairment in glucose tolerance after diets high in SFA, while omega 3 polyunsaturated fatty acids (PUFA) have been shown to enhance IS or reverse abnormal glucose tolerance caused by high SFA diets.[48-50] There is evidence in humans that manipulation of dietary fat intake influences IS. The isoenergetic dietary RCT

---

<sup>1</sup> The gold standard for investigating and quantifying insulin resistance. The plasma insulin concentration is acutely raised and maintained at 100  $\mu$ U/mL by a continuous infusion of insulin. Meanwhile, the plasma glucose concentration is held constant at basal levels by a variable glucose infusion. When the steady-state is achieved, the glucose infusion rate equals glucose uptake by all the tissues in the body.

named KANWU recruited men and women who ranged from a healthy weight to the overweight.[5] Participants with IGT were also included. All participants were assigned to one of the following two diets, both containing 37 percent of total energy (%E) from total fat: a high SFA diet (17%E) or a high monounsaturated fatty acid (MUFA) diet (23%E). Food products were provided to participants to assist in achieving dietary targets and dietary compliance was tested by three day food records and by blood samples. IS was measured via the intravenous glucose tolerance test (IVGTT)<sup>2</sup>.

After three months participants attained an increased MUFA intake of 8% to achieve %E intake from MUFA of 21%. IS was significantly decreased by 10% on the high SFA diet compared with the high MUFA diet, which did not change. There was no low fat study group comparison however post hoc analysis revealed no beneficial effect of replacing SFA with MUFA when %E from total fat consumed was above the median intake of 37%E. Hence this indicates any benefit in changing quality of dietary fat in the diet is negated by a diet high in total fat.

Reduction in total fat has been included as a dietary target in large long term successful RCTs conducted to determine if T2DM can be delayed or prevented by pharmacological intervention and or by encouraging positive dietary and PA behaviour.[6-9] A post hoc analysis from one of the major RCTs showed reduction in total fat was a significant predictor of the weight loss achieved in the study.[51] With weight loss the dominant predictor of reduced T2DM incidence, reduction in total fat, therefore, had an indirect effect on reduction in T2DM incidence.

In another RCT further discussed in Dietary change in high risk individuals, reduction of total fat to  $\leq 30\%$ E was included as part of five study goals, with a strong inverse correlation found between achievement of a greater number of the study goals and incidence of T2DM. These findings indicate reduction in total fat is albeit an indirectly beneficial dietary strategy for the reduction of T2DM risk, reinforced by the previously mentioned ADA guidelines. There is a

---

<sup>2</sup>A clinical approach to study beta cell function. This technique consists of the injection of a glucose bolus and frequent sampling of plasma glucose and insulin or C-peptide concentrations.

substantial body of evidence despite the inconsistencies across different research designs to implicate SFA as a nutrient associated with the impairment of IS and development of T2DM. Although there is no specific target set by ADA for reduction in SFA, a dietary target of less than 10% of total energy from SFA as was trialled in the large RCTs discussed below, appears an appropriate target for future prevention trials.

### **Polyunsaturated fat**

With the evidence suggesting potential harm for a high intake of SFA on T2DM risk, questions have been raised regarding an appropriate dietary fat replacement for SFA. Consistent evidence has accumulated for the inverse association between PUFA intake and T2DM risk. However, consideration of the individual's PUFA intake in the context of overall wellbeing not just its relation to T2DM risk must be considered.

In patients undergoing surgery for coronary artery disease, the phospholipid fraction of muscle biopsy samples demonstrated long chain PUFA, arachidonic acid and the total percentage of PUFA, were inversely associated with fasting serum insulin concentration.[40] Conversely, IS was positively correlated with these same PUFA in their sample of healthy men determined by EHC.[40] Additionally several prospective studies have found an inverse association of dietary intake of PUFA and incidence of T2DM. In the Nurses Health Study total PUFA intake was inversely associated with diabetes risk when all known risk factors were controlled for (RR 0.75 95% CI 0.65, 0.88). Furthermore, a 5% increase in energy from vegetable fat was associated with a reduced risk of diabetes (RR 0.79 95% CI 0.74, 0.84).[52] In the Health professionals follow up study an inverse relationship between intake of the PUFA linoleic acid and diabetes incidence was demonstrated only in men under the age of 65 (RR 0.74, 95% CI 0.60, 0.92).[53]

The 2012 systematic review and meta-analysis of cohort studies aforementioned concluded vegetable fat was significantly associated with a lower risk of T2DM (RR 0.79 95% CI: 0.71, 0.86). However no other sub class of PUFA or total PUFA was found to be significantly associated with T2DM risk. Thus it appears PUFA intake from vegetable fat rather than from animal products may confer the benefit seen.

Concern is warranted, however, against advice to increase intake of foods high in omega 6 PUFA as excess intake has been implicated in other chronic diseases.[54] The current adoption of PUFA-rich fats and oils has led to relatively high intakes of omega six (n-6) PUFA and has resulted in a high ratio of n-6 to omega 3 (n-3) PUFA intake.[45] It has been currently estimated the n-6 to n-3 consumption in a typical western diet is in the range of 15:1 to 20:1.[55] In the Melbourne Collaborative Cohort Study the ratio was 9:1[45] when the optimal n-6 to n-3 intake for the human diet is suggested to be 1-2:1.[56]

In a post hoc analysis of KANWU a higher n-6 to n-3 intake was characterised by a significant deterioration of IS within the duration of the study, independent of group assignment (high SFA or high MUFA diet).[57] Moreover, individuals with a higher n-6 to n-3 intake at baseline demonstrated greater deterioration in IS at three months. Recommendations to limit SFA intake should take into account the possible importance of the n-6 to n-3 intake and subsequent adverse effects of excess n-6 consumption.[45] Dietary replacement of excess SFA by MUFA as is seen in the next section, shows more promise than increasing the overall PUFA intake.

### **Monounsaturated fat**

There is evidence to suggest that replacing intake of SFA with MUFA may have some benefit for the reduction of diabetes risk. At the very least the Nurses Health Study and Health Professionals follow up study found MUFA intake was not associated with T2DM incidence.[52, 53] In addition two prospective cohort studies in Southern Europe found a lower incidence of T2DM with increasing adherence to the Mediterranean diet in baseline healthy individuals[58] and myocardial infarction survivors.[59] The traditional Mediterranean diet is high in MUFA as it is a diet characterised by: high consumption of vegetables, legumes, grains, fruits, nuts and olive oil, moderate consumption of fish and wine and a low consumption of red and processed meat and whole fat dairy.[60] The MUFA content of the Mediterranean diets accounts for 16–29%E with olive oil the predominant source of MUFA.[61]

As presented in the section Total fat and saturated fat diets high in MUFA have been compared with diets high in SFA. In KANWU when MUFA intake replaced some of the SFA in the diet, IS was not impaired. Moreover, when analysis was conducted according to the median fat intake of 37 %E, in individuals below the median intake, difference in IS between the two diets was significant. This sub analysis demonstrated a 12.5% decrease in IS in the high SFA diet and an 8.8% increase in IS in the high MUFA diet.[5]

Increasing evidence of the benefit of a diet high in MUFA as a replacement for SFA intake comes from a large study of the Mediterranean dietary pattern. A sub study of the trial determined the Mediterranean diet's effect on T2DM incidence in men and women aged between 55-80 years, who were at risk of cardiovascular disease.[60] In the three arm RCT 418 individuals free of diabetes followed three non-calorie restricted diets: a low fat (<35%E from total fat "control") diet, a Mediterranean diet enriched with olive oil and a Mediterranean diet enriched with mixed nuts. It is important to note both olive oil and nuts deliver a high amount of MUFA.

The Mediterranean diet included advice to do the following: use abundant olive oil for cooking and dressing, increase consumption of fruit, vegetables, legumes and fish, reduce total meat consumption recommending white meat instead of red or processed meat, prepare homemade sauce with tomato, garlic, onion and spices, avoid butter, cream fast food, sweets pastries and sugar sweetened drinks and moderate consumption of red wine for those who drank alcohol. Those in the Mediterranean diet groups were given either one litre per week of olive oil or mixed nuts 30g per day. Exercise was not promoted.

Participants were followed for a median of four years. Weight loss was similar and non-significant across all groups as was exercise. After adjustment for sex, age, BMI, baseline energy intake, waist circumference, PA, smoking status, fasting glucose, use of lipid lowering drugs, Mediterranean diet score and weight change, the incidence of T2DM determined by

yearly blood samples from the oral glucose tolerance test (OGTT)<sup>3</sup>, was reduced by 52% when the two Mediterranean diet groups were combined, compared to the control group. However, only 21% of the control group managed to achieve their goal of fat intake <35% total energy and as this was a study of the benefit of a particular dietary pattern, there was no mention of each group's actual achieved MUFA intake. Overall the evidence is so far accumulating for the benefits of a relative higher MUFA intake on improvement in IS and T2DM incidence. Further trials in other at risk populations however are needed to make definitive conclusions.

### **Dietary Fibre**

Consistent evidence has accumulated for the benefit of an increased consumption of dietary fibre and reduced risk of T2DM. Several prospective trials have investigated the association between dietary fibre or wholegrain cereal intake and T2DM risk reduction. Foods are considered to be wholegrain if all components of the kernel (bran, germ and endosperm) are present in their natural proportions. Wholegrain foods, therefore, have comparatively greater levels of cereal fibre than foods based on refined grain in which the bran and germ have been removed.

In a Cochrane Review of 11 prospective studies, intake of cereal fibre was inversely associated with T2DM, with the RR range of 0.63-0.72.[3] A meta-analysis of six prospective cohort studies reviewing the association between wholegrain intake and incidence of T2DM found total wholegrain intake was inversely associated with T2DM risk, with a RR of 0.79 (95% CI 0.72, 0.87) for each two serving per day increment in whole grain intake.[62] According to this meta-analysis, results from the Nurses Health Study One and Two indicated bran but not germ intake was significantly associated with a lower risk of T2DM (RR 0.70, 95% CI 0.62, 0.79).[62]

---

<sup>3</sup> Under fasted conditions the individual consumes an oral dose of glucose most commonly 75grams and blood glucose is measured over a time period, most commonly over two hours.

Results from the Tehran Lipid and Glucose Study, a study population of 2457 adults aged 19-84 years, showed total dietary fibre intake (OR 0.53, 95% CI 0.39, 0.74) both soluble (OR 0.60, 95% CI 0.43, 0.84) and insoluble (OR 0.51, 95% CI 0.35, 0.72) were significantly and inversely associated with the risk of the metabolic syndrome after adjustment for main and dietary confounders.[2] Among the sources of dietary fibre studied, fruit fibre (OR 0.51, 95% CI 0.37, 0.72) and legume fibre (OR 0.73, 95% CI 0.53, 0.99) were also significantly inversely associated with the risk of the metabolic syndrome. Results from these prospective studies and the subsequent reviews indicate advice to increase fibre intake from a range of food sources such as fruit, legumes and grain products, may all contribute to the benefit of dietary fibre on T2DM risk reduction.

There is also considerable evidence from short term RCTs that manipulation of fibre intake influences IS. A double blind 18 week parallel RCT compared the effect on IS of four isoenergetic diets: high protein (HP) diet versus high cereal fibre (HCF) diet versus control diet versus mixed diet of moderate protein and moderate cereal fibre.[63] 111 overweight individuals with features of the metabolic syndrome were assigned to one of the four diets.

In addition to emphasis of plant and vegetable sources to achieve dietary targets and restriction in all groups of high fat animal products, all participants consumed a supplement specific to group assignment. The HCF group consumed 30g dietary fibre and the HP group consumed 58g protein, with the dietary target of 25-30%E from protein. A fibre intake target is not mentioned however cereal fibre was emphasised only in HCF diet and the mixed diet and participants were expected to consume at minimum 30g of fibre per day. Dietary compliance was good, with lower adherence to the HP diet found between week six and week 18 of the study. Overall IS in the HCF group improved significantly from baseline and significantly decreased in the HP group resulting in a significant 25% difference in IS between participants in the HCF and HP groups after week six. After week 18, differences in IS were no longer significant partly explained by lower adherence to the HP diet. In the HCF group at week 18 IS remained significantly increased by 16% compared with baseline values.



Considered a third type of fibre alongside insoluble and soluble fibre is resistant starch, starch which escapes digestion in the small intestine. In a single blind randomised placebo controlled trial, supplementation of 40g per day of resistant starch for 12 weeks in 20 middle aged adults with elevated fasting insulin levels improved IS by 19%, with 14% deterioration in the placebo group ( $p=0.023$ ).[64] Resistant starch escapes digestion in the small intestine and is considered a third type of fibre alongside insoluble and soluble fibre. In contrast with these results, no changes in glucose and insulin concentrations or markers of insulin resistance and sensitivity were found in a 12 week three arm parallel RCT involving manipulation of fibre intake in 226 middle aged healthy adults[65]. The participants in this trial were assigned to either: a control diet consisting of refined cereals and white bread or a diet where three servings of refined cereals foods were substituted with either: three servings of whole-wheat foods (serving size of 70–80 g whole-meal bread and 30–40 g whole-grain cereals) or a diet with one serving of whole wheat foods and two servings of oats, with the oat serving size not specified.

To meet dietary targets participants were given refined or wheat- or oat-based wholegrain foods. Each dietary group was designed to be practical and realistic for free-living individuals to achieve, with no alteration of their usual lifestyle. The fibre intake in this study at 12 weeks was considerably lower (16.8 and 18.5g) in the two active treatment groups when compared to the fibre intake of 30g or more achieved in intervention trials mentioned prior that achieved significant change in IS.[63, 64].

In the prospective Tehran Lipid and Glucose Study participants who consumed  $\geq 30$ g per day of total dietary fibre were found to have a small but significantly lower risk of the metabolic syndrome (-5%  $p=0.012$ ). Together with results from the short term RCTs and as will be seen in the next section, an intake of approximately 30g of fibre or 15g per 4200kJ as recommended by ADA, seems to be the amount of fibre required to confer benefit of improvements to IS and reduced T2DM risk.

### Food and dietary patterns

For individuals at risk of T2DM achieving the ADA dietary targets for prevention of T2DM may require change to the consumption of specific foods and/or general food patterns, with a number of studies linking both individual foods and dietary patterns to T2DM risk. Red meat is a dominant source of total and SFA in western diets. Although the nutritional composition of red meat can vary according to breed, feeding regimen, season and meat cut, prospective studies have linked higher consumption of red meat to an increased risk of T2DM.[66]

Additionally, the change in consumption of red meat intake over time and subsequent risk of T2DM in three large prospective cohorts has been investigated. When FFQ data from the Health Professionals Follow Up Study and the Nurses Health Study one and two were pooled, compared with the reference group of no change in red meat intake, increasing red meat intake of more than 0.50 servings per day was associated with a 48% elevated risk in the subsequent four year period.[67] The association was only modestly attenuated after further adjustment for initial BMI and concurrent weight gain.

In a 2007 review higher consumption of dairy foods was associated with the prevention of T2DM. A 2011 meta analysis of seven cohort studies which investigated the influence of type, fat content and daily consumption of dairy and risk of T2DM, found consumption of low-fat dairy foods was associated with an 18% reduced risk of T2DM development.[68] However, intake of high fat dairy foods and whole milk were not associated with such risk. The meta analysis speculated the protective effect of dairy may largely due to nutrients other than fat in milk and its products. Thus greater consumption of low fat dairy may confer protection while avoiding potential harm from excessive SFA consumption.

Research using the cluster analysis method from food frequency questionnaire data has found certain dietary patterns are associated with either an increase or reduction in T2DM. Data from 10 308 individuals in the Whitehall II study of 20 civil service departments in London demonstrated the “healthy pattern” compared with the unhealthy pattern reduced the risk of T2DM (HR 0.74, 95% CI 0.58, 0.94), after adjustment for age, sex, ethnicity, dietary energy misreporting, social position, smoking status, and leisure-time physical activity. The “healthy

pattern” was characterised by: higher than average consumption of wholemeal bread, fruit and vegetables, and polyunsaturated margarine and average to low consumption of red meat, sweet foods, and wine and beer, while the unhealthy pattern was characterised by: higher than average consumption of meat and sausages, white bread, fries, and full cream milk, average consumption of wine and beer and very low consumption of fruit and vegetables.

The association between dietary patterns and development of T2DM specifically in individuals at risk of diabetes including women with prior GDM, have contributed to the body of evidence that indicates those individuals consuming a comparatively healthier diet, have a lower risk of T2DM. In 20 835 overweight and obese participants in the Dutch part of the EPIC-NL study, a diet high in soft drinks, fries and snacks and low in fruit and vegetables, was associated with a higher risk of T2DM (HR 1.70 95 % CI 1.31, 2.20)

In 4413 women with history of GDM from the Nurses Healthy Study Two, the following three healthy dietary patterns: the alternate Mediterranean diet (aMED), the Dietary Approaches to Stop Hypertension (DASH), and the alternate Healthy Eating Index (aHEI) were inversely associated with the risk of T2DM. The three dietary patterns promoted to some degree higher consumption of fruit and vegetables, legumes, nuts, soy, fish and seafood, wholegrains and cereal fibre and lower consumption of red and processed meat, sugary foods and salt. The aMED pattern was associated with 40% lower risk of T2DM (HR 0.60, 95% CI 0.44, 0.82); the DASH pattern, a 46% lower risk (HR 0.54 95% CI 0.39, 0.73) and the aHEI pattern, a 57% lower risk (HR 0.43 95% CI 0.31, 0.59). Adjustment for BMI partially attenuated the effect estimates for the three dietary patterns, however estimates were still significant.

The Mediterranean dietary pattern is characterised by: high consumption of vegetables, legumes, grains, fruits, nuts and olive oil, moderate consumption of fish and wine and a low consumption of red and processed meat and whole fat dairy. As mentioned previously, the Mediterranean diet when followed over four years, resulted in a significant reduction in T2DM incidence in older age adults.

In summary the consistent association between a healthy dietary pattern and reduced risk of T2DM and the emerging evidence of the long term benefits of consuming a Mediterranean diet, affirm the suitability of encouraging individuals to consume a diet which is: predominant in vegetables, fruit, wholegrains, legumes, nuts, MUFA oils and low fat dairy, moderate in its intake of red meat and limited in processed meat and energy dense snacks such as dessert items.

## **Dietary change and prevention of T2DM**

### ***Dietary change in high risk individuals***

Moving on from evidence of individual nutrients and dietary patterns, large RCTs have been conducted to determine if T2DM can be delayed or prevented by pharmacological intervention and or by encouraging a range of positive dietary and PA behaviours.[6-9] These trials were conducted across different ethnic populations involving individuals with IGT. In two well-designed studies the American Diabetes Prevention Program (DPP) and the Finnish Diabetes Prevention Study (DPS), lifestyle intervention resulted in a significant 58% reduction in the incidence of T2DM compared to the control group.[6, 9]

The reduction in T2DM incidence was of a similar magnitude amongst men and women, different ethnic groups and amongst younger and older participants. A small but significant reduction of T2DM incidence by 26 – 31% with use of metformin, an oral glucose lowering medication compared to a placebo group has been reported.[6, 8] However, combining lifestyle intervention and metformin use has not been found to be significantly more effective than lifestyle alone in reducing T2DM risk.[8] Amongst the studies the specific components of the lifestyle intervention varied, with an overall focus on decreasing fat and energy intake, increasing dietary fibre and encouraging daily PA with emphasis on walking.

In the DPP a loss of approximately five kilograms was estimated to account 55% of the reduction in T2DM incidence. A significant reduction in diabetes incidence of 46% was found amongst participants who completed 150 minutes minimum of moderate PA per week without achieving the study's weight loss goal. The weight loss goal was to reduce and maintain weight reduction of at least 7% initial body weight, if overweight. Change in dietary

intake such as total energy or total fat were not associated with the reduction in T2DM risk. However together with an increase in PA, lower baseline total fat intake and a reduction in intake of total fat after one year were found to be significant predictors of weight loss both in the shorter and longer term.

Results from the smaller Finnish DPS substantiate the results of the DPP.[9] Participants in the DPS were encouraged to achieve the following goals: consumption of  $\leq 30\%$ E from total fat and less than  $\leq 10\%$ E from SFA, consumption of 15g fibre per 1000kcal intake, weight loss of 5% of initial body weight if overweight and completion of 30 mins of moderate PA per day. As in the DPP, participants in the intervention arm of the DPS lost significantly more weight than those in the control group at one year (-4.2kg versus -0.8kg). Among the intervention group, those meeting the PA goal without achieving their weight loss goal had a lower odds ratio of T2DM compared to those who did not meet the weight loss goal and maintained a sedentary lifestyle (OR 0.3 CI 0.1, 0.7). Overall a strong inverse correlation was noted between achievement of a greater number of study goals and incidence of T2DM, indicating dietary change as part of lifestyle change is important for such risk reduction.

In a sub analysis of the DPP the difference in efficacy of the intervention between women who did, or did not have a history of GDM were examined. In women with prior GDM, treatment with metformin when compared to placebo resulted in a similar risk reduction achieved by lifestyle intervention (50% versus 53% reduction in risk respectively).[69] However treatment with metformin in women without such a history appears to be less effective, with only a 14% risk reduction of T2DM found.

The report estimated five to six women with IGT and a history of GDM would need to be treated over 3 years with either metformin or lifestyle intervention to prevent one case of diabetes. In women without a history of GDM, these estimated numbers needed to prevent a single case of diabetes over 3 years were 24 for metformin therapy and 9 for lifestyle intervention. These estimates reaffirm the need to encourage women with prior GDM to adopt lifestyle change which has proven effective in reducing risk of T2DM. Although metformin was also found to be effective in women with prior GDM, consumption of lifelong

metformin would be costly and the long term potential side effects of such a practice are not established in this population.[69]

### *Dietary change in women with prior GDM*

#### *Dietary patterns reported in women with GDM*

While it is possible to delay or prevent onset of T2DM via a healthy lifestyle, the current lifestyle and dietary behaviour of women with prior GDM is not encouraging. Amongst a cohort of 4718 American women with and without prior GDM, the former were significantly heavier (BMI 27.4 kg/m<sup>2</sup> versus 25.4 kg/m<sup>2</sup>) with a prevalence of overweight and obesity of 28.4% and 25.6%, respectively.[10] Only approximately one quarter of the sample of women with previous GDM met the American fruit and vegetable consumption guidelines of five or more servings per day and 36% ate less than three serves of fruit and vegetables daily.

Despite the smaller sample sizes amongst Australian studies the trends are just as alarming. In a rural sample in Victoria of 53 women with prior GDM, 66% were above healthy weight range with 24% classed as overweight and 42% as obese.[12] In a random sample of 226 women with prior GDM in Western Sydney, the mean BMI was 32.0kg/m<sup>2</sup> and 30% were obese.[13] Additionally the dietary intake of fruit and vegetables was low with only 5% of the women meeting the recommended five or more vegetable servings per day, with 38% consuming one or less servings per day. Furthermore, over half this sample of women consumed less than the recommended two serves of fruit per day.

Conversely, fat intake in Australian women with prior GDM is higher than the recommended less than 30 %E intake from total fat and less than 10 %E intake from SFA. Total fat and SFA intakes in these women are reported in the range of 34% and 13-15.5% respectively.[11, 70] In women with a recurring history of GDM, fat intake was reported to be as high as 41.4%.[11]

The food frequency questionnaire (FFQ) administered to the above Western Sydney cohort revealed women were engaging in dietary behaviours which favour high intakes of total fat

and SFA. 50% of the sample consumed full cream milk and 25% consumed fried foods two or more times per week.[13] According to a secondary analysis of the DPP, the subgroup of women with previous GDM were unable to sustain weight loss achieved over a three year period when compared to women without a previous GDM. Women with previous GDM achieved weight loss of  $5.1 \pm 0.4$  kg at six months with a steady weight regain to a mean weight loss of only  $1.6 \pm 0.8$  kg at year three, compared with a loss of  $6.4 \pm 0.2$  kg and a mean weight loss of  $4.0 \pm 0.4$  kg at three years in the women without previous GDM.[69]

In the small rural Victorian sample of women with previous GDM, the majority agreed eating a healthy diet, maintaining weight and doing PA were important for preventing diabetes and prevention was important.[12] However only 15 out of the 53 women in the study were meeting PA guidelines and managing their weight, either classified within a healthy BMI or currently losing weight. Psychosocial variables that influence the dietary behaviour of women with a prior GDM has been explored with “Busy lifestyle” the most common barrier reported.[13] Other barriers which featured predominantly were “too great a change from current diet”, “cost” and “dislike of healthy foods by others in the household”. [13] It is of interest to note the survey found a third of the sample reported a lack of knowledge of what foods to consume to reduce their diabetes risk, given the women would have had at minimum dietary education on managing GDM during the pregnancy. As a whole these results indicate a need to improve the efficacy of the limited risk reduction strategies currently offered to women post a GDM pregnancy.

#### Study Design issues for intervention research in women with GDM

When conducting clinical interventions in women post pregnancy and or with young children, it is crucial to address the social context of the woman’s life stage. High attrition rates ranging from 21 to 43% are cited for behavioural lifestyle interventions targeted at postpartum women including those with prior GDM.[71] To minimise attrition and maximise participation, the feasibility of obtaining assessment measures and intervention design must be carefully considered when developing research protocol for such studies.

A prior qualitative case study of a sample of women with prior GDM found physical and emotional disturbances such as lack of sleep, fatigue, depression, stress, return to work and the overall sense of feeling rushed were prevalent themes.[70] These issues demonstrate that the physical capacity of mothers is limited. Not only can this influence the potential efficacy of a lifestyle intervention but it may also limit the capacity of these women to partake in the intervention at all or in a manner that is useful to the research team. In choosing methods of data collection therefore while the quality of outcome data is important, the ability to obtain sufficient outcome data must be considered.

The difficulty in obtaining biochemical data relating to T2DM intervention outcomes has previously been highlighted in a behavioural intervention where the oral glucose tolerance test (OGTT) which diagnoses IGT and T2DM was collected in only 13 of 20 the women who provided data.[14] An OGTT requires the participant be available for two hours, fasted and with minimal movement made during this time. It can only be speculated that this may poses problems for mothers with limited childcare available, as childcare arrangements may be required. Thus although obtaining measures of insulin sensitivity or diagnosis of T2DM is ideal, research methods in the context of women with prior GDM who have young families must be realistic, given at present there is limited literature on the efficacy and feasibility of dietary change in these women.

In behavioural change interventions assessing dietary change, choice of dietary assessment method must also be carefully considered. In the context of women with young children the delivery of dietary assessment and degree of subject burden associated with a given method may impact on quality and or quantity of results. These factors were the driving force for decisions made regarding choice of dietary assessment in the current thesis.

The optimal timing and content of a post birth lifestyle interventions both in women with or without complications in their pregnancies also remains undefined.[72] Recruitment of women with or without previous GDM six weeks after birth has consistently resulted in high drop out rates, slow recruitment or low participation[15, 72, 73]. Difficulties in recruiting for a healthy lifestyle intervention at a minimum of six weeks postpartum is further highlighted



in a study where 49 of a possible 3298 participants were randomised, giving a 1.5% recruitment rate. Such a low recruitment rate occurred even though the study required no face to face counselling and only two visits to the hospital for data collection.[74] The challenge thus remains in designing an appropriate trial which maximises recruitment and retention in order to demonstrate dietary change is possible.

#### Dietary change in women with prior GDM is possible

Previous large scale interventions in individuals at high risk of diabetes have shown diabetes is preventable.[6-9] There are comparatively less large scale trials evaluating the efficacy of diabetes risk reduction interventions on T2DM specifically in women with prior GDM.[17, 19] Since the design and implementation of the intervention from which this thesis is derived and during the write up of this thesis, several studies have been published which have examined lifestyle change in women with prior GDM.[18, 20] Within this literature dietary outcomes are mostly secondary to changes in T2DM incidence or weight.

The studies have been conducted in Australian, American and Chinese women with study sample sizes ranging from 38 in a small Australian pilot of rural women,[18] to a currently on-going large scale Chinese RCT of 1180 women.[19] Most studies have delivered lifestyle change involving dietary intervention in the postpartum period and beyond predominantly via telephone, with one study beginning intervention immediately after the diagnosis of GDM and continuing seven months thereafter.[20] The intervention period in these studies have ranged from six months to a median of 51 months of follow up.

The majority have demonstrated significant reductions in body weight, total fat in absolute amounts and as %E and greater increases in intake of fibre post intervention.[18-20, 75] An Australian study in which women were given healthy diet and PA advice and counselling by a dietitian every three months for a median of 51 months, however, demonstrated no significant difference in incidence of T2DM between the intervention and control group.[17] The large Chinese study has T2DM incidence as its primary outcome however final study results are yet to be released at the time of writing this review.

### **Summary of dietary change and prevention of T2DM**

Different levels of evidence demonstrate a relationship between consumption of a diet high in SFA and an increased risk of T2DM and conversely a greater consumption of fibre and a decreased risk of T2DM. One well designed RCT has indicated replacement of SFA intake with sources of MUFA may improve IS, provided overall fat intake does not exceed 37 %E. Individuals counselled to follow a Mediterranean dietary pattern had a lower T2DM incidence when followed over four years compared with a control low fat diet. RCTs which targeted a fibre intake above 30 grams per day found IS improved in individuals with features of the metabolic syndrome.[67, 68].

It is well established that T2DM can be prevented in high risk populations by reducing weight and increasing PA. Combined with the other dietary and PA goals aforementioned, decreasing total and SFA and increasing fibre intakes reduced the incidence of T2DM in individuals with IGT. Subsequently reduction in body weight and intake of total fat and an increase in fibre intake have been recommended by the ADA for inclusion in subsequent T2DM prevention programs. Consumption of “healthy” food patterns has consistently been associated with a lower risk of T2DM, most likely as such patterns enable the recommended dietary fat and fibre targets for T2DM risk reduction to be achieved. Levels of overweight and obesity are prevalent in women with prior GDM which further increases their risk of T2DM. Additionally the poor quality of their diet as published in the literature indicates the need for intervention to achieve a dietary pattern consistent with diabetes risk reduction and to achieve the ADA dietary recommendations. The barriers which both limit participation in research and prevent women with prior GDM from eating a healthy diet should be addressed to maximise research participation and intervention efficacy.

### **Dietary assessment methods**

The FFQ, 24 hour recall, food record and the diet history interview are the most common methods available to quantify and assess dietary intake. Dietary assessment is vulnerable to considerable error.[76, 77] Although no gold standard amongst these dietary methods exists the repeated 24 hour recall and the weighed food record are considered the closest available.

The 24 hour recall requires the participant to remember all food and beverages consumed in the previous 24 hours, with the interviewer using food models and household measures to

help quantify the actual amount of food and beverages consumed.[78] The food record involves either: estimating or weighing and measuring all foods and beverages consumed using scales and household measures over a specified period of time, often three, four or seven days. [78] The diet history is an intensive interview which generates information about usual eating habits and often includes a food frequency checklist.[78] The FFQ asks questions regarding the intake of specific food items and if it is a quantitative FFQ portion size is included in order to generate usual intakes.[78]

The dietary assessment method chosen in a research setting is dependent on the aims of the study, the resources available and the population under study. The food record and the diet history were the means by which outcome data was collected for the HeLP GDM study and subsequently the thesis.. Collecting a diet history was considered necessary, as a diet history allows for a thorough investigation into the individuals dietary consumption. Consequently the focus for the remainder of literature review concerns only these two dietary methods.

### **The validity of dietary assessment methods**

Evaluating the validity of reported energy intake (EI) provides a valuable check on the general quality of the dietary data in any study.[79]. If EI is underreported it is probable that the intakes of other nutrients correlated to EI are also underestimated.[79] Misreporting, and specifically underreporting of EI is persistent in all dietary assessments. Underreporting of energy in a particular dietary assessment is commonly cited as a measure of criterion validity in validation studies, both for justification of its use of in research design and for comparison between assessment methods.[79-83] Previous work in the area of dietary methodology found the degree to which participants underreport varies depending on the context and characteristics of the participants of the study. [84]

An individual is considered to be in energy balance when energy intake is equal to energy expenditure (EE). Underreporting is defined as a discrepancy between reported EI and measured energy expenditure (EE) without any assumed or observed change in body mass.[82] To determine underreporting a method of measuring EE is required, alternatively underreporting can be calculated by evaluating reported intake against presumed

requirements. Either way studies have reported different cut offs to distinguish underreporting. Doubly labelled water (DLW) is the gold standard for measuring EE under free-living conditions. The DLW method is costly and due to its expense, many validation studies determine underreporting by using the Goldberg cutoff technique. This method evaluates EI against estimated energy requirements EI:BMR, with the ratio subsequently compared with a specific physical activity level (PAL) cut off value. The cutoff value represents the lowest value of EI/BMR that could reasonably reflect the energy expenditure if the person led a sedentary lifestyle.[85] Aside assessing the validity of reported energy intakes, studies have validated nutrient intakes using urinary biomarkers. One example is validation of protein intake by using nitrogen excretion levels in 24 hour urine samples.

According to two reviews on the validity of dietary assessments, BMI is most consistently positively associated with underreporting.[79, 82] However underreporting is not limited to overweight or obese individuals. In a nationwide quantitative FFQ administered in Norway, 52% of under reporters had a BMI of  $<25.0\text{kg/m}^2$ [86]. Across several studies, women have also been found more likely to underreport[82, 87] with several aspects of dieting (reported trying to lose weight, dieting history, weight changes in the last five years, self-perception of feeling to heavy, dieting during the study period) also linked to underreporting.[79, 82] Underreporting of dietary intake via self-report methods in samples of overweight women with prior GDM is thus likely. Underreporting published in the literature by the main dietary assessment methods pertinent to the thesis, is discussed below. However individual underreporting of dietary intake was not investigated in the current thesis.

In addition to determining the true validity of intakes obtained from dietary assessment, dietary assessment methods are compared amongst each other to determine how well values obtained agree, otherwise known as relative validity. Distinction must be made between validity at the group level (the mean value) and validity at the individual level (correct ranking). A method may provide a valid mean but rank poorly due to poor precision or variable bias across subjects.[79] When measuring dietary change in a RCT, the assessment of dietary data validity within the study context can strengthen the dietary study's primary findings. Given both diet history and food record datasets were analysed in regards to the

primary aim of this thesis which surrounds dietary change, an analysis of the relative validity between the datasets became a secondary aim of this thesis.

### Food records

The food record is a written record of actual food and drink consumption over a specified period of time. The food record is usually weighed or estimated. Although the food record is not free of error, of the above four methods of measuring dietary intake, the weighed food record is often used as the reference method in validating new tools to assess nutrient intake.[88] The food record however, places a much greater burden on the participant who may change actual intake to make recording easier. High subject burden in general may effect compliance and accuracy in data documentation [89] and relies to some extent on the participant's literacy. [90]

In a 2003 review of the markers of validity of reported energy intake, with inclusion of over 22 studies, the ratio of estimated EI:EE as determined by DLW for both the estimated and weighed food record was 0.84. [79] The corresponding 'acceptable reporters' range was defined as a ratio between 0.76–1.24. Thus the two variations of the food record method underestimate energy intake equally within accepted limits. Additionally moderate correlations between dietary intake assessed by the estimated food record and urinary biomarkers have been reported for urinary nitrogen ( $r=0.66$ ), sodium ( $r=0.48$ ) and potassium (0.58).[91]

Comparison between the estimated and weighed food record have been studied in different populations with different time points for data collection which may contribute to differences in study findings. Exact protocols for completing records have varied with portion estimation in the estimated food record assisted by using photographs or drawings. Relative validity studies have generally demonstrated the estimated food record compared with the weighed food record does not obtain significantly different dietary intake values for energy, fat and fibre.[92, 93] .

However in a study of 60 individuals in Costa Rica a seven day weighed food record, weighed independently by researchers when compared with a seven day estimated food record. showed the estimated food record significantly underestimated mean intakes of energy, dietary fibre and all other macronutrients with exception of absolute amount of total fat.[89] However the nutrient intakes reported by the estimated record occurred within 10% of the weighed food record values. Greater differences in estimates of mean energy and nutrient intake were detected among participants from rural areas compared to urban areas which contributed to the overall differences. Thus again emphasising the importance of study context when interpreting results.

### **Diet history interview**

Compared with other forms of dietary assessment the diet history is a less standardised technique and can be conducted in several styles, depending on the interviewer's technique. Although the format may vary, the questioning is predominantly of an open ended nature. The diet history is most commonly a face to face interview in which beginning with the first meal eaten, the individual reports their intake, portion size and frequency of consumption; over a usual day, week or other specified time frame.

To facilitate estimation of portion size, a range of portion size estimation aids (PSEA) exist such as food models, photographs of foods or standard household measures. Diet histories in clinical practice are an essential tool for identifying aspects of the diet which require change. The diet history is however, labour intensive, time consuming and dependent on memory recall of the interviewee.[94]

Much fewer studies have validated the use of the diet history against DLW, expected energy requirement or biomarkers. The 2003 review aforementioned found the ratio of estimated EI:EE as determined by DLW for diet histories which spanned either seven days or usual intake of the previous month was also 0.84. The 'acceptable reporters' range as previously mentioned was defined as a ratio between 0.76–1.24.[79] The review indicates the diet history is a valid dietary assessment with both the diet history and food record method underestimating true energy intake equally within the accepted limits.

In an English study of 48 middle aged women the diet history was compared to a 16 day food record and validated against urinary nitrogen (UN) DLW and study specific Goldberg cutoffs.[95] The UN: nitrogen intake (NI) showed slight underreporting of protein intake with no differences in magnitude between the diet history and food record. In a subsample of 16 individuals who completed DLW testing the  $EI:BMR_{\text{measured}}$  ratio was 1.46 for weighed records, and 1.60 for diet history compared with a measured physical activity level of 1.65 and a Goldberg cut-off value of 1.52. In the context of the Goldberg cutoff the food record mean EI was biased to under-reporting whereas the mean EI from the diet history was considered valid indicating relatively better performance of the diet history.

As the diet history represents a wide range of approaches which focus on an account of usual intake, comparisons across relative validity studies of the diet history can be difficult. Unlike the abundance of literature available comparing the relative validity of the other three dietary assessment methods, there is sparse literature on relative validity of the diet history. Difficulty in obtaining such literature may also arise due to the use of different wording to term the diet history interview. The following section compares the performance of the diet history with the food record in different populations and cultures with a number of studies assessing relative validity in the clinical intervention context.

In healthy women and men and additionally in the overweight at high risk of T2DM or those already diagnosed with T2DM, the diet history in majority of studies underestimated energy and fat intake compared with the food record[80, 83, 96] A number of reports have however reported an overestimation of energy, fat and fibre.[70, 97]. In majority of relative validity analysis differences between mean estimates of macronutrients obtained were not significant, otherwise differences in macronutrient intake in absolute values or when translated to the context of actual foods were negligible.[83] Significant differences between methods in energy intakes however are not negligible and have varied from -695kJ to -935kJ.[80, 96] Large standard deviations of the mean bias were present in most studies.[70, 80, 96] A large standard deviation of the mean bias demonstrates a wide extent of intra-individual variation indicating low individual precision of agreement between methods.

A study which assessed changes in bias between the diet history and food record over time in two clinical trials of overweight individuals and those with T2DM found the magnitude of bias for energy and fat was stable overtime.[96] Differences in context between the two trials including study participants and intervention outcomes were found to influence the direction and magnitude of the bias for MUFA, which confirms the importance of study context in any assessment of relative validity. In both clinical trials change in MUFA was the primary outcome. While there were no differences between the clinical trials in overall bias in the diet history measurement of energy and %E from protein, carbohydrate, alcohol, total fat PUFA and SFA, in the clinical trial of healthy individuals mean bias for %E from MUFA was small and underestimated by the diet history. In the clinical trial of individuals with T2DM the mean bias was larger and overestimated by the diet history.

In the first clinical trial underestimation of MUFA intake was suggested to have resulted from changes in actual food intake at the time of recording, to resemble the study goals of increasing MUFA.[96] It was speculated that in the second clinical trial since prescription related reporting in people with T2DM has previously been demonstrated, they may have simply reported intakes that were in line with the intervention goals. However change in MUFA was not reflected in the food record as the food record reflected actual food intake.[96]

The downside to conducting a diet history is the time needed to complete the assessment and subsequently the cost. Traditionally most interview methods are conducted face to face however to save time, money and increase recruitment other modes of administration have been investigated, of which most common is the telephone.[98] Literature is available on the validity of the computer assisted diet history[99] however at the time of writing the thesis candidate was unaware of any published literature which has explicitly documented the relative validity of the diet history conducted via telephone compared with the traditional face to face method or any other dietary assessment.

A 1992 review of the telephone survey as a method of dietary assessment found intakes obtained via telephone for the FFQ and 24 hour recall correlated well however the review



called for more literature in this area.[98] Two more recent studies conducted in 2000 have compared the dietary data from the 24 hour recall method via telephone with that collected face to face in adult women. The studies confirm the relative validity of using the telephone as a vehicle for dietary assessment.[100, 101]

In one study energy intakes were compared to energy expenditure measured by DLW. Underreporting was present with EI significantly lower than expenditure for both telephone and face to face interviews with no significant difference between the two in the magnitude of underreporting.[100] Additionally there was no significant difference at group level in energy intakes between the telephone and face to face interview. In the second study no significant differences in intake of energy, total fat and SFA or fibre were found between the telephone interview or the face to face interview.[101]

One potential source of error in dietary intake studies is portion size estimation.[102] The PSEAs which can be used are limited when dietary assessment is conducted via telephone compared with in person assessment. 2D PSEA for example photographs of portion size are predominantly used, as they can be mailed to respondents before interviewing. A study has compared the accuracy with which respondents reported food intake in the 24 hour recall when using 2D versus 3D PSEAs, during an in person versus telephone interview in 120 people aged 18-65 years.[102] No significant differences were found in the accuracy with which respondents reported intake when using the same set of 2D aids during in-person or telephone interviews indicating portion estimation was not affected by change in administration of the assessment.

### **Summary of dietary assessment methods**

It appears the diet history underreports energy intake to same degree as the weighed or estimated food record when underreporting is determined by objective biomarkers. A number of studies show small differences between the diet history and food record group mean estimates. The precision of individual estimates from the diet history and food record to agree however is low. Overall the results from relative validity analyses indicate compared with food record estimates, diet history values may underreport energy and dietary fat. However

the context of the trial including outcomes of interest in the intervention, nutrients studied and sample population recruited, may affect direction and magnitude of the bias. Validation of the use of the telephone to administer the diet history appears to be unknown although research indicates dietary intakes obtained via telephone for other dietary assessment methods are comparable to those obtained in a face to face interview.

### **Literature review summary**

Women with prior GDM are at a high risk of developing T2DM. ADA nutritional guidelines for the prevention of T2DM recommend a reduction in body weight, reduction in dietary fat and an increase in dietary fibre for individuals at high risk of diabetes. Survey data of postpartum women with previous GDM show their current dietary practices and macronutrient intakes favour progression towards T2DM. Small and large scale studies in women with prior GDM have modelled their dietary targets on targets defined in prior large scale T2DM prevention studies in individuals at high risk of T2DM. Intervention studies in women with prior GDM have demonstrated positive dietary change for T2DM risk reduction is possible, however in the majority, study samples are small and intervention duration has been short. The current thesis are secondary analyses of dietary data collected from a feasibility pilot assessing lifestyle change in postpartum women with prior GDM within the western Sydney Local Health District.

## **Chapter Two - Research Design and Hypothesis**

### **Introduction**

This thesis contains secondary analyses of data from the randomised controlled pilot the Healthy Living Program after Gestational Diabetes Mellitus (HeLP GDM) study, a lifestyle behavioural intervention conducted in a sample of women with previous GDM. Under the guidance of the collaborators mentioned below the role of the thesis candidate was the design and implementation of the HeLP GDM study. More specifically the thesis candidate recruited the participants, solely administered the intervention component of the pilot and completed all anthropometry and dietary data entry.

Currently a primary outcome paper is in the process of manuscript revision for publication. Although change in study participant's physical activity was a primary outcome for the HeLP GDM study, the current thesis pertains only to analyses of the dietary and anthropometric data. The following members of the collaboration team HvP and BS, as mentioned on page 42 were responsible for the analysis and reporting of the PA outcomes. PA outcomes from the HeLP GDM study were reported in the aforementioned primary outcome paper. Subsequently no PA outcome data or discussion surrounding increasing levels of PA and prevention of T2DM is presented in this thesis.

The following section details the aims and hypotheses of the thesis. The results and associated discussion contained in chapter three address the primary aim and that of chapter four address the secondary aim. The remainder of chapter two provides a summary of the HeLP GDM study.

### **Aims and hypotheses**

#### **Thesis hypotheses**

The two central hypotheses tested in this thesis were:

1. dietary change was achieved in the HeLP GDM study

2. the associated dietary intakes collected from two dietary assessment methods provided relatively comparable estimates of macronutrient intake.

From these hypotheses arose two separate research questions:

1. Did the women in the HeLP GDM study achieve dietary targets associated with T2DM risk reduction?
2. How well did the two measures of dietary intake obtained in the study compare?

## **Thesis aims**

### **Primary aim**

To determine if the intervention group in the HeLP GDM study compared to the control group significantly decreased their intake of total fat, SFA and energy and increased their total intake of fibre. It was hypothesised that the intervention group in the HeLP GDM study would:

- decrease total fat intake significantly more than the control group
- decrease SFA significantly more than the control group
- decrease energy intake significantly more than the control group
- increase dietary fibre significantly more than the control group

### **Secondary aim**

To determine the relative validity between a seven day telephone diet history and a three day estimated food record, the two dietary assessment methods used to obtain dietary data in the HeLP GDM study. It was hypothesised the seven day telephone diet history and three day estimated food record would be relatively comparable in estimating energy and macronutrient intake.

## **The HeLP GDM study: a summary**

The HeLP GDM study was a single blind parallel randomised controlled trial conducted to evaluate the effectiveness of a targeted intervention to dietary and PA change, in women with

recent previous GDM. The intervention was designed and implemented within a research setting at Westmead hospital NSW. The design and implementation was due to a collaboration between the following individual's and research units: the endocrinologist (WC) and clinical trials co-ordinator/dietitian also the thesis candidate at Westmead Hospital, nutrition researchers at University of Wollongong (LT, JoS, YB) and exercise physiology and medical and nutrition researchers within the Cluster for Physical Activity and Health (CPAH) at Sydney University (HvP, AB, MG) and Monash University (BS). The pilot approach was chosen to determine if the intervention would be feasible to implement on a much greater scale within the NSW hospital system.

### **Study setting**

Women who were registered as attending the Diabetes in Pregnancy Service at Westmead Hospital were considered potential participants. The pilot population included all women who were seen for the medical management of GDM and had given birth within six to 48 months of the recruitment phase (1 September 2009 to 31 May 2010).

### **Selection criteria**

#### ***Inclusion criteria***

- i) Women who have had GDM and gave birth in the previous 6-48 months
- ii) Overweight or obese using culturally specific BMI cutoff for the non-Indian Asian populations: BMI  $\geq 25.00$  kg/m<sup>2</sup> for all excluding the non-Indian Asian subgroup (BMI  $\geq 23.00$  kg/m<sup>2</sup>) [103]
- iii) Report an intention to adopt changes in diet or PA according to the Transtheoretical model (TTM) (ie, individual is in contemplation, preparation, action or maintenance).

#### ***Exclusion criteria***

- i) Diagnosed T1DM or T2DM
- ii) Pregnant or planning pregnancy within six months
- iii) Unable to or unfit to exercise due to chronic illness
- iv) Prescribed medication which may affect glucose or weight control

- v) Precontemplators for both diet and PA as defined by the TTM
- vi) Poor written and spoken English proficiency
- vii) Failure to complete all the baseline assessments

### **Pilot phases**

The pilot consisted of five stages: pre-screen, baseline assessment, randomisation to the intervention or control group, intervention period and six month follow up. For a summary of the phases refer to Figure 1.

### **Pre - screen**

Women who were registered as attending the Diabetes in Pregnancy Service at Westmead within the last 6-48 months were contacted via a letter to participate. Women registered as having experienced a GDM pregnancy 48 months from the start of the recruitment phase were contacted first and those who experienced a GDM pregnancy six months from recruitment phase were contacted last. Women were called within two weeks of receiving their invitation to participate.

If the woman expressed interest in the study in order to determine eligibility, she was asked to self report her weight and height for calculation of BMI. The woman was also asked to answer the two brief questionnaires based on the TTM to assess her stage of behaviour change for diet[104] and PA.[105] The TTM conceptualises behaviour change as a progression through five distinct stages of change. An individual's readiness to change at a given moment can be identified within one of the following five stages: precontemplation (not intending to make changes), contemplation (considering change), preparation (making small changes), action (actively engaging in new behaviour) and maintenance (sustaining the change over time).[106]

The woman needed to score at minimum a contemplator in either the diet or physical activity stage of behaviour change questionnaires to be eligible for enrolment. Questions were asked to ensure the woman did not meet any of the exclusion criteria. If the woman was eligible, an

appointment was made to sign the consent form and provide baseline anthropometric measures. To maximise recruitment numbers home visits were offered for all aspects of the pilot, to women who reported interest in the study but had difficulties attending the hospital.

#### **Baseline assessment**

Eligible participants completed a baseline evaluation. Weight, height, BMI and waist circumference was recorded. Instructions were given regarding completion of a three day estimated food record and standard measuring cups and spoons were provided. The seven day diet history, a previously validated health and lifestyle survey regarding PA habits and beliefs and attitudes towards PA and diet[107] and self-reported weight were collected via telephone by MG. Diet history, food record and survey data obtained formed the foundation for intervention counselling and provided outcome data.

#### **Six month assessment**

Women were not asked to attend the hospital for six month follow up to encourage recruitment into the study and reduce participant burden. Thus waist measurement was not recorded and weight at six month final assessment was self reported. Women were sent their three day food record form. The seven day diet history and health and lifestyle survey was conducted via telephone as per baseline assessment. During data collection MG was blinded to the participant's group assignment.

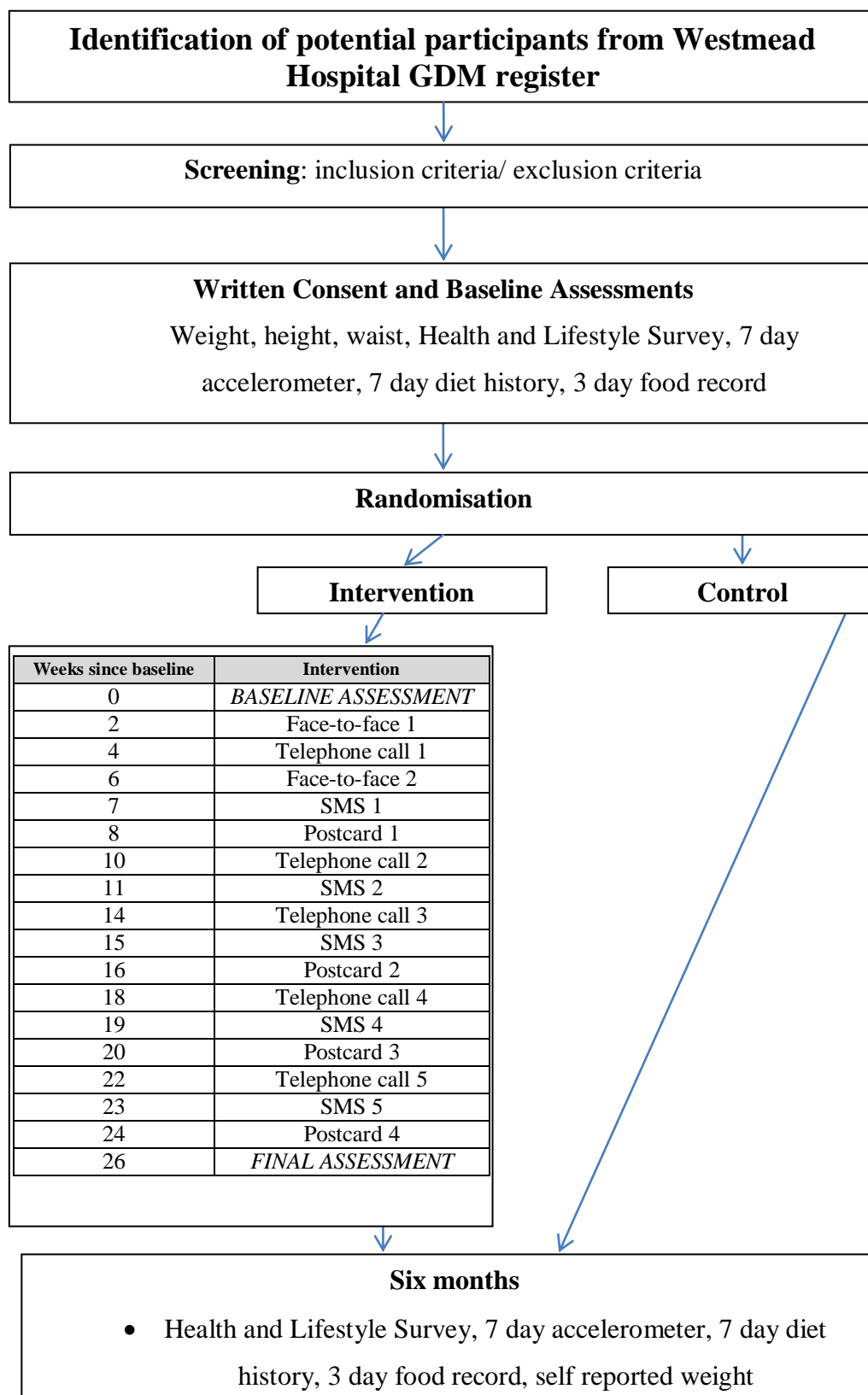


Figure 1: Plan for implementation of the HeLP GDM study



### **Randomisation**

The women were assigned to either the six month intervention or a no treatment control group. Independently prepared randomisation envelopes were opened for each subject after baseline assessment. Randomisation occurred in blocks of eight with equal numbers of intervention or control in each group.

### **The intervention**

The intervention component comprised of two face to face counselling sessions, five telephone follow ups, five individualised mobile text messages and four generic mailed postcards (see Figure 1). The timing and mode of the HeLP GDM study intervention were guided by earlier formative studies conducted amongst women after GDM within this Area Health Service. These earlier studies included a behaviour change program for 25 women post a GDM pregnancy[14] and a pilot RCT with 43 women post a GDM pregnancy.[108] The RCT timeline of 2 face-to-face counselling sessions one month apart, and a telephone session in between, was adapted for use in the HeLP GDM study.

Periodic prompts are defined as messages, reminders or brief feedback communicated to participants at various intervals.[109] A 2009 review regarding the use and efficacy of periodic prompts and reminders in dietary and PA health promotion and health behaviour interventions reported positive findings in 11 of the 19 studies reviewed.[109] Tailoring of periodic prompts through regular contact with a counsellor was also found to be effective in six studies. The review concluded the effectiveness of periodic prompts is enhanced if prompts are frequent, with most studies providing weekly prompts and personal contact with a counsellor is included.[109] Hence the decision to include a number of SMS texts tailored to the individual participant and provision of generic postcards relevant to the study population.

the intervention followed the patient counselling model and a proforma based on this model was used by the thesis candidate to guide the face to face and telephone calls.[110] The proforma focused on behavioural goals, self efficacy and barriers to change that in prior

research has been identified as important among women with prior GDM in the Western Sydney Area health service.[107] For details of the proforma see appendix A and B.

The counselling sessions involved the use of goal setting and problem solving to attain dietary goals agreed upon by the participant and the thesis candidate. Goals were set depending on current dietary patterns while acknowledging the participants stage of change. Strategies to change dietary intake were negotiated with the primary aim of reducing total fat and SFA intake and increasing fibre if required, to reduce calorie intake, as most participants were personally motivated to lose weight.

Problem solving included discussion on potential barriers to achieving goals and possible action taken to overcome them. Discussion also involved the role of family and friends in providing support and potential rewards for goals achieved. Dependent on the individual case, structured meal plans were provided and self-monitoring was encouraged in the form of recording number of times goal were achieved or recording food intake, for feedback by the thesis candidate.

Each participant in the intervention received a journal titled “*my journey to a healthier me*”. The journal was provided as an aid to assist with the counselling process and its use was not mandatory. The journal provided a place to record discussion with the thesis candidate regarding goals and to monitor goal achievement and dietary intake. The journal also contained practical food information, ideas to assist in achieving dietary fat reduction and increased fibre intake and a list of websites to refer to. The journal was an unpublished work of the thesis candidate which was reviewed and approved by the members of the collaboration team.

Four postcards containing pictures accompanied by generic statements about healthy lifestyle and T2DM prevention were mailed to the participant’s home. Two of the postcards contained messages relating to healthy eating. The remaining two were in regards to partaking in regular PA to meet the PA aims of the HeLP GDM intervention group. These postcards were

mailed to the participant via Australia post inside an envelope, thereby maintaining the confidentiality of the participants and postcards. A mobile text message was sent to the woman one week after the second face to face counselling session and all telephone calls thereafter. The text was individualised to reflect the previous telephone conversation between participant and the thesis candidate and contained a reminder or positive reinforcement of goals set.

### **Ethics**

All aspects of the HeLP GDM study were approved by the Westmead Hospital Human Research Ethics Committee and written consent to participate was obtained from all participants. All information and data obtained during the HeLP GDM study is stored in the diabetes and endocrinology research department at Westmead Hospital for a minimum of 7 years, after which it can be destroyed in a confidential manner.

## CHAPTER THREE - DIETARY CHANGE AFTER GESTATIONAL DIABETES MELLITUS IN THE HeLP GDM STUDY

### Introduction

The results and associated discussion in this chapter pertain to the nutrition data from the HeLP GDM study as described in Chapter 2. In this study change in intake of total fat, SFA, fibre and energy from baseline to six months were the outcomes of interest. To assess dietary change dietary intake was measured using the seven day diet history and the three day estimated food record. To confirm changes in energy intake, change in self-reported weight was also determined.

### Method

#### Dietary assessment

##### *Seven day telephone diet history administration*

At baseline the seven day diet history was conducted prior to randomisation. The seven day diet history obtained followed a similar format to a previously validated open ended interview [80] assessing the individual's usual intake over the last month. The interview proceeded according to meals consumed and was divided into breakfast, lunch, dinner, morning tea, afternoon tea and supper. Each meal was kept as a separate entity so the participant could focus on their food and drink consumption at that time of the day. Within each meal, food intake was required to add up to seven days of intake including number of days where meals may have been missed.

Meal preparation practices were obtained such as fat trimming on meat and or chicken, type of oil and fats used and type of bread and milk consumed. The interview concluded with a food frequency checklist to cross check no main food or drink items such as yoghurt or juice had been missed. Portion sizes were estimated by the participants with the aid of the standard measuring cups and spoons provided to them at baseline. Palm of the hand, fist size or take away containers were additionally used to elicit portion sizes for items such as meat, fish, potato, certain fruits and take away meals. All information was recorded on an interview proforma provided in appendix C .

### *Three day estimated food record*

To complete the three day food record, in addition to standard measuring cups and spoons, participants were given a generic form which requested the following information: meal type, time food was consumed, food type with as much detail as possible, amounts consumed, cooking method and location where food was cooked. Participants were asked to provide a record for two weekdays and one weekend day. Emphasis was placed on recording all meals, snacks, nibbling, and beverages including water and any alcohol. Including brand names was encouraged. The food record was returned via a reply paid envelope or in some instances faxed or scanned and emailed back. For a copy of the food record form see appendix D.

## **Anthropometry**

### *Body weight*

Body weight was measured using digital scales (Soehnle Personenwaage jumbo, Art-Nr 62513) with participants barefoot and wearing minimal clothing. Weight was recorded to the nearest 0.1 kg. Weight at baseline was self-reported during the telephone health survey within approximately three weeks after measured weight was obtained. At six months self-reported weight obtained via the same health survey, was the only available measure of body weight.

### *Height*

Height was measured using a free standing stadiometer to the nearest 0.5 cm. Participants were barefoot and stood facing away from the stadiometer with heels at the rod of the stadiometer. The participant was asked to look straight ahead and the measurement was taken in line with the top of the head using the stadiometer's attached indicator.

### *BMI*

BMI is a commonly applied clinical measure to estimate body fat in individuals [111] and is calculated using the equation  $BMI = \text{body weight (kg)} / \text{height}^2 \text{ (m)}$ . Overweight is defined as a BMI of  $\geq 25.00$ - $29.99 \text{ kg/m}^2$  and obesity defined as  $BMI \geq 30.00 \text{ kg/m}^2$  [33]

### *Waist circumference*

Waist circumference in cm was taken only at baseline to describe the study sample. The measurement was taken on bare skin. The waist was defined as the midpoint between the lower border of the ribcage and the iliac crest. If excess body fat made the two points difficult to find, the measurement was taken at the umbilicus. One measurement was obtained.

### *Data entry*

#### *Dietary assessment*

To minimise researcher bias during data entry, food record and diet history hard copy data for each participant were recoded with a new study number. The recode was completed by an independent party. The thesis candidate completed all baseline and six month data entry. Once data entry was completed, recoded data were matched against the original study number to ensure no error occurred during the recode.

Food record and diet history information was entered into the nutrient analysis program, FoodWorks Professional (version 6, 2009: Xyris Software, Pty Ltd, Queensland, Australia). The Food Standards Australia New Zealand (Canberra) AUSNUT 2007 food, nutrient and supplement database within the Foodworks software was utilised for all analyses. Foods were entered individually according to the meal in which they were consumed. If a food was not available in the database, the approximate macronutrient profile of the food was searched on Calorie King using the Australian version of the website found at the following link <http://www.calorieking.com.au/>. [112] By using the recipe option, a profile of the food was created which reflected the available information of the food's energy, total fat, SFA and fibre content. The profile was then used for all subsequent entries where the particular food was mentioned. In the literature this method has not been validated as a method of assumption work. This assumption method was chosen based on personal discussion with MG as it was part of the protocol for data entry of information collected for the Sydney Diabetes Prevention Program. [113]

If a food's nutrient profile was not available then the most appropriate substitution in the database was chosen arbitrarily depending on the available information provided. For cultural

meals in which no database substitution was deemed appropriate and no recipe was given, the following steps were taken in order to make a suitable assumption: At least three Internet recipes if at all available of the meal in question were sourced to determine a common pattern of ingredients. A recipe was then chosen which arbitrarily was considered to include the common ingredients in measurements that were easily transferable to the FoodWorks software. The recipe assumption was then used for all further entries where the meal was named but no recipe was provided.

If portion size was missing depending on the available information, portion size was searched on Calorie King or if the product or its manufacturer had its own website. If no helpful information was given the unspecified serve option in the FoodWorks database was chosen. If the portion provided by the participant did not match serve sizes available in the FoodWorks the following steps were taken: in FoodWorks a similar food which offered the portion size listed by the participant was searched. The equivalent weight in grams of the portion size was then determined and applied to the original food in question. This method was applied as the gram option was always available in FoodWorks.

All substitutions, assumptions regarding portion size and created food profiles were recorded for the purpose of maintaining consistency of data entry. After initial data entry was completed all entries were rechecked for errors and to confirm data entry consistency by the thesis candidate. Data were then transferred to an SPSS statistical software database.

### *Anthropometry*

Weight, height and waist circumference data were recorded on the participant's information sheet and later transferred to a spread sheet. The spread sheet once checked for data entry error was exported alongside the dietary data into the same SPSS database for statistical analysis.

### Statistical analysis

Statistical analyses were performed using SPSS (version 19.0; SPSS Chicago IL, USA). An alpha error of  $p < 0.05$  was used to determine statistical significance for all analyses. Significant differences in baseline demographic information was assessed using independent t tests for continuous variables and the fishers exact test due to small sample size for categorical variables. Additionally the baseline characteristics of those who enrolled in the study and those who remained in the study are provided for visual comparison. However no formal testing for differences between participants who dropped out and those who remained in the study was conducted due to the small numbers in each group who dropped out. For detail on the number and reason for drop out of the study refer to Figure 2.

Results pertaining to the primary aim of the thesis are presented both as intention to treat and completers analyses. With the intention to treat analysis, if the six month measurement was not available, the participant's baseline value was assumed to be unchanged and carried forward. The completers analysis included only the data of participants who provided six month data. Thus sample size varies between completers analysis of diet history and food record data as less food records were completed at six months.

The **primary outcomes** of this study were changes in intake of total fat, SFA, fibre and energy. Baseline mean macronutrient intakes between groups were compared using the independent sample t-test. Mean group differences at six months for total fat, SFA and fibre and energy were tested for significance using a linear regression model, which adjusted for the baseline macronutrient value.

To determine the face validity of the dietary data, change in body weight was assessed at six months. In order to determine whether SRW could substitute for MW at six months, the relative validity of SRW compared with MW at baseline was assessed using paired samples t-test, the Pearson's correlation coefficient and the Bland Altman scatterplot. One participant from the intervention group was unable to provide her SRW at baseline and six months thus in the intervention group  $n = 28$  at baseline and  $n=23$  at six months. There was no significant difference in mean bias between SRW and MW and the two measurements were highly



correlated (Table 1). There was also no linear relationship between bias and mean weight (Table 1). Therefore SRW was used for all weight and BMI analyses.

The Fishers exact test was performed to test for significant group differences at baseline in the proportion of participants meeting dietary targets for %E from total fat and SFA and intake of fibre based on kilojoule intake. The defined targets in this analysis reflect ADA recommendations and targets previously achieved in large T2DM prevention trials.[7, 9] The dietary targets were: intake 15g of fibre per consumption of 4200kJ, consumption of  $\leq 30\%$  of total energy from total fat intake and  $\leq 10\%$  of energy intake from SFA. Odds Ratios (OR) with 95% CI were calculated using binary logistic regression to detect significant group differences in the proportion of participants meeting each dietary target at six months. For each variable, the baseline proportion of participants meeting the dietary recommendation was entered as a covariate.

**Table 1: Relative validity between self reported weight and measured weight at baseline**

	<b>SRW</b>	<b>MW</b>	<b>Paired t</b>	<b>R value</b>	<b>Bias<sub>mean</sub></b>	<b>*R<sup>2</sup> (p)</b>
	<b>Mean</b>	<b>Mean</b>	<b>test P</b>	<b>(p)</b>	<b>(SD)</b>	
	<b>(SD)</b>	<b>(SD)</b>				
Weight (kg)	78.5	78.9	0.18	0.99 (.00)	-0.42 (2.3)	0.00 (.86)

## Results

Data from the 59 women who completed the baseline assessments and were randomised to one of the two groups in the HeLP GDM study were used. Information on recruitment, retention and completion is given in Figure 2. The mean age of control and intervention participants was 35.1 and 35.7 years, respectively. Participants in both groups had a mean of two children. Participants were predominantly well educated, with 63% in the control group and 55% in the intervention group reporting a university education. Additionally, 47% and 52% of participants in the control and intervention group respectively were in paid employment. The sample was predominantly multi-cultural, with 67% and 72% in the intervention and control group respectively naming a country other than Australia as their

country of birth. Apart from a significant difference in the number of hours in paid employment between the groups for the women in paid employment, no other significant differences in baseline characteristics were found. These baseline results are shown in Table 2

The mean baseline BMI ( $31.6 \text{ kg/m}^2$ ) of the women in the intervention group was within the obese range while in the control group the mean BMI ( $29.4 \text{ kg/m}^2$ ) was still within the overweight range (Table 3). The difference was not however, significant. The stage of change questionnaires identified the majority of participants in both groups were currently in the preparation stage of change for diet [43% and 41% respectively (Table 4)]

Intention to treat and completers analyses showed the mean energy and macronutrient intake between the groups at baseline were not significantly different for both diet history and food record data (Table 5, Table 6, Table 7, Table 8) Decreases in continuous measures of energy and all macronutrient intakes occurred in both the intervention and control group.

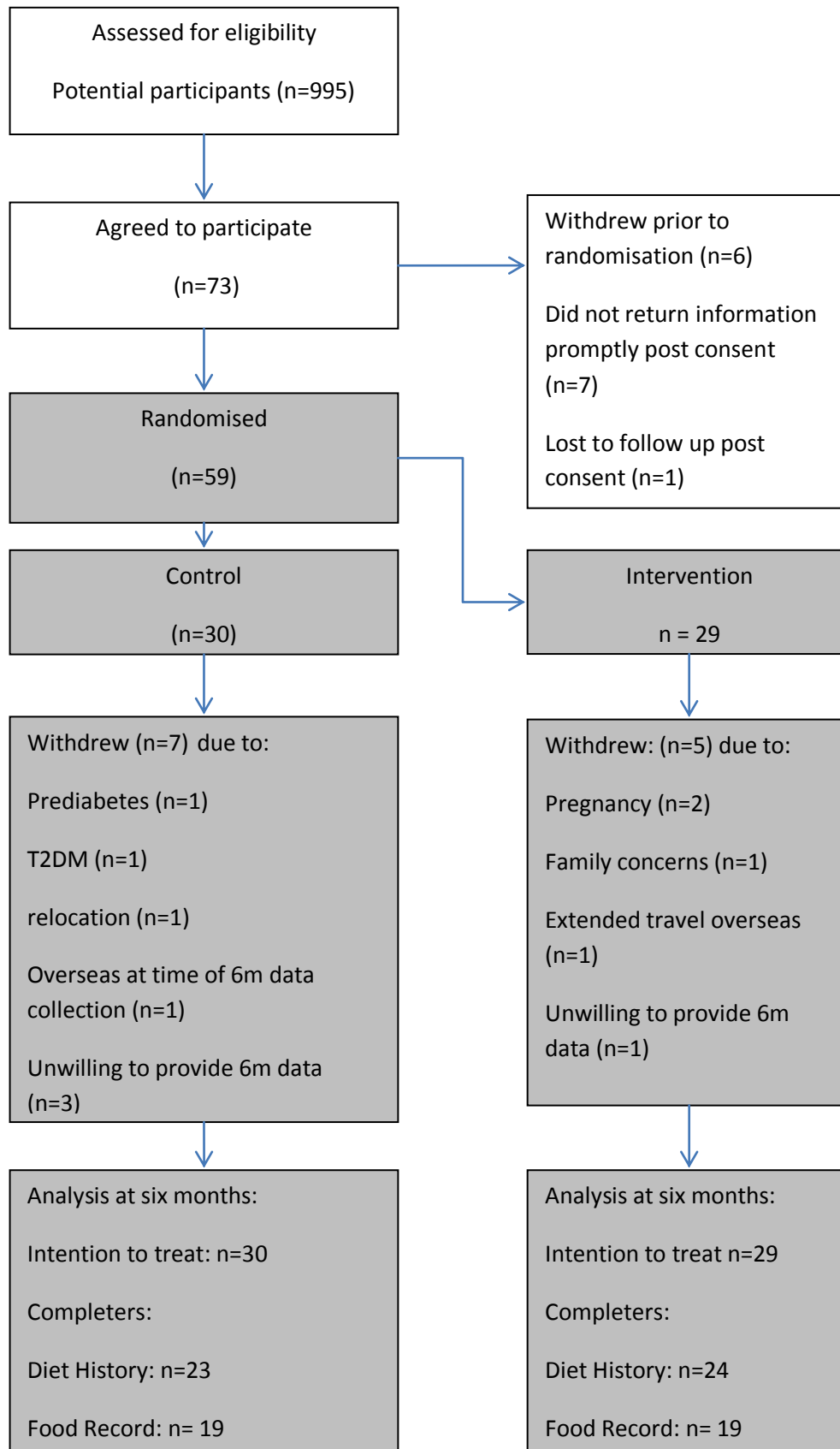


Figure 2: Eligibility, recruitment, retention and completion of the HeLP GDM study with shaded cells showing the dietary sub-study of this thesis

Table 2: Baseline demographic data of participants at enrolment and those remaining in the trial at six months

	Enrolled participants			Remaining participants	
	Control n = 30	Intervention n = 29	P	Control n= 23	Intervention n=24
Age*	35.1 (4.2)	35.7 (4.7)	0.64	35.2 (4.6)	36.2 (4.8)
Children*	2.3 (1.2)	1.9 (0.9)	0.23	2.3 (1.3)	1.9 (0.7)
GDM pregnancies*	1.3 (0.6)	1.3 (0.5)	0.68	1.3 (0.6)	1.3 (0.5)
<b>Education <sup>1</sup></b>					
Some high school	2 (6.7)	5 (17.2)		2 (8.7)	4 (16.7)
Completed all high school years	6 (20)	7 (24.1)		2 (8.7)	7 (29.2)
Trade/technical certificate/ diploma	3 (10)	1 (3.4)		2 (8.7)	1(4.2)
University	19 (63.3)	16 (55.2)	0.47	17 (73.9)	12 (50)
<b>Paid work</b>					
No	16 (53.3)	14 (48.3)		10 (43.5)	11 (45.8)
Yes	14 (46.7)	15 (51.7)	0.80	13 (56.5)	13 (54.2)
paid work (hours)	37.9 (9.0)	29.1 (11.4)	0.03	38.8 (8.6)	29.5 (11.0)
<b>COB</b>					
Australian	10 (33.3)	8 (27.6)		8 (34.8)	7 (29.2)
Non Australian	20 (67.3)	21 (72.4)	0.78	15 (65.2)	17 (70.8)
Non Australian born:					
Asia (including middle east)	15 (75)	15 (71.4)		11 (73.3)	12 (70.6)
Other^	5 (25)	6 (28.6)	1.00	4 (26.7)	5 (29.4)

\*mean (SD)

<sup>1</sup> highest level attained

COB: Country of Birth

^Values are shown as n (%), \*Europe, Africa, South America, America, Canada, New Zealand & Pacific Islands

**Table 3: Baseline anthropometry: all participants and remaining participants at six months**

	<i>Enrolled participants</i>			<i>Remaining participants</i>	
	Control	Intervention	P	Control	Intervention
	n = 30	n = 29		n= 23	n= 24
BMI (kg/m <sup>2</sup> )	29.4 (5.2)	31.6 (5.3)*	0.11	29.3 (5.0)	31.8 (5.6)^
Weight (kg)	75.0 (15.2)	82.2 (16.8)*	0.09	74.6 (13.9)	83.7 (17.9)^
Waist (cm)	94.8 (11.3)	99.1 (11.2)	0.14	93.7 (11.2)	100.3 (11.5)

Values are mean (SD), \*n = 28, ^n=23

**Table 4: Baseline assessment of the dietary stages of change: all participants and remaining participants at six months**

	<i>Enrolled participants</i>			<i>Remaining participants</i>	
	Control	Intervention	P	Control	Intervention
	n = 30	n = 29		n= 23	n= 24
Contemplation	11 (36.7)	7 (24.1)		8 (34.8)	6 (25)
Preparation	13 (43.3)	12 (41.4)		10 (43.5)	9 (37.5)
Action	5 (16.7)	7 (24.1)		4 (17.4)	6 (25)
Maintenance	1 (3.3)	3 (10.3)	0.54	1 (4.3)	3 (12.5)

Values are n (%)

Table 5: Assessment of macronutrient intake via the seven day diet history by experimental group at baseline and six months: intention to treat

	<i>Baseline, mean (SD)</i>			<i>6 month follow up, mean (SD)</i>		<i>Linear regression analysis</i>		
	Control (n = 30)	Intervention (n= 29)	P*	Control (n = 30)	Intervention (n= 29)	Adjusted change <sup>1</sup> (95% CI)	mean	P
Energy (kJ)	8631 (2840)	7778 (2625)	0.24	7840 (2782)	6088 (1814)	-1251 (-2169, -332)		0.01
Protein (g)	92.6 (26.7)	85.1 (26.6)	0.29	88.6 (27.8)	77.3 (25.2)	-5.6 (-14.8, 3.5)		0.22
Total fat (g)	74.8 (41.9)	62.8 (27.5)	0.20	66.8 (37.7)	43.2 (18.4)	-16.8 (-28.5, -5.0)		0.01
SFA (g)	26.9 (17.7)	22.3 (12.7)	0.26	23.6 (17.3)	13.7 (7.2)	-7.2 (-12.2, -2.1)		0.01
CHO (g)	242.1 (100.4)	223.5 (93.8)	0.47	217.6 (101.4)	174.9 (57.8)	-31.3 (-61.7, -0.8)		0.04
Alcohol (g)	0.9 (2.2)	1.5 (6.5)	0.65	0.8 (1.6)	1.0 (3.7)	-0.3 (0.1, 0.4)		0.00
Fibre (g)	25.5 (10.4)	24.8 (8.4)	0.78	23.6 (8.2)	25.2 (7.9)	2.1 (-1.1, 5.2)		0.20
<b>%E from:</b>								
Fat	31.1 (8.8)	29.4 (6.9)	0.42	30.8 (8.2)	25.5 (6.7)	-4.4 (-7.8, -1.0)		0.01
Protein	19.2(4.9)	19.3 (5.8)	0.91	20.2 (5.9)	22.0 (5.4)	1.6 (-0.6, 3.9)		0.15
CHO	46.1 (10.5)	47.2 (8.9)	0.67	45.3 (10.1)	47.6 (7.1)	1.6 (-1.4, 4.6)		0.30
Other <sup>^</sup>	3.6 (1.6)	4.0 (2.9)	0.49	3.6 (1.1)	4.8 (1.9)	1.1 (0.3, 1.8)		0.01
SFA	10.9 (4.2)	10.3 (3.5)	0.55	10.5 (4.3)	8.0 (3.1)	-2.1 (-3.7, -0.5)		0.01

\*independent t-test, <sup>1</sup>mean change in the intervention compared to control group, adjusted for baseline levels of outcome, CI: Confidence Interval

SFA: Saturated fatty acid, CHO: Carbohydrate, <sup>^</sup> Percentage of total energy from alcohol, fibre and other combined

Table 6 Assessment of macronutrient intake via the seven day diet history by experimental group at baseline and six months: completers

	<i>Baseline, mean (SD)</i>			<i>6 month follow up, mean (SD)</i>		<i>Linear regression analysis</i>	
	Control (n = 23)	Intervention (n= 24)	P*	Control (n = 23)	Intervention (n= 24)	Adjusted change <sup>1</sup> (95% CI)	mean p
Energy (kJ)	8124 (2434)	8090 (2702)	0.96	7092 (2035)	6048 (1869)	-1030 (-1996, -63)	0.04
Protein (g)	92.0 (23.6)	87.6 (27.5)	0.56	86.8 (24.9)	78.1 (26.4)	-5.6 (-16.8, 5.5)	0.32
Total fat (g)	70.2 (36.5)	68.2 (26.6)	0.83	59.7 (27.7)	44.5 (18.9)	-14.5 (-26.9, -2.2)	0.02
SFA (g)	23.8 (13.8)	24.4 (12.8)	0.87	19.6 (11.7)	14.0 (7.7)	-5.8 (-10.9, -0.6)	0.03
CHO (g)	223.4 (74.6)	227.2 (99.4)	0.89	191.5 (65.8)	168.5 (55.3)	-24.5 (-54.6, 5.6)	0.11
Alcohol (g)	0.9 (2.0)	1.7 (7.1)	0.57	0.7 (1.1)	1.2 (4.0)	0.2 (-1.4, 1.9)	0.78
Fibre (g)	24.6 (10.2)	25.6 (8.7)	0.73	22.1 (6.7)	26.1 (8.1)	3.6 (-0.1, 7.3)	0.06
<b>%E from:</b>							
Fat	31.0 (8.2)	31.1 (6.0)	0.96	30.6 (7.2)	26.4 (6.7)	-4.2 (-8.2,-0.2)	0.04
Protein	20.0 (4.6)	19.1 (5.8)	0.55	21.4 (5.6)	22.3 (5.4)	1.4 (-1.3, 4.1)	0.29
CHO	45.3 (8.9)	45.7 (8.7)	0.87	44.2 (8.3)	46.2 (6.6)	1.8 (-1.7, 5.3)	0.32
Other <sup>^</sup>	3.7 (1.8)	4.1 (3.2)	0.61	3.8 (1.2)	5.1 (1.9)	1.2 (0.3, 2.1)	0.01
SFA	10.5 (3.6)	11.0 (3.4)	0.58	9.9 (3.6)	8.2 (3.3)	-1.9 (-3.7, 0.0)	0.05

\*independent t-test, <sup>1</sup>mean change in the intervention compared to control group, adjusted for baseline levels of outcome, CI: Confidence Interval

SFA: Saturated fatty acid, CHO: Carbohydrate

<sup>^</sup> Percentage of total energy from alcohol, fibre and other combined

Table 7: Assessment of macronutrient intake via the three day food record by experimental group at baseline and six months: intention to treat

	<i>Baseline, mean (SD)</i>			<i>6 month follow up, mean (SD)</i>		<i>Linear regression analysis</i>		
	Control (n = 30)	Intervention (n= 29)	P*	Control (n = 30)	Intervention (n= 29)	Adjusted change <sup>1</sup> (95% CI)	mean	p
Energy (kJ)	8032 (2079)	8427 (2663)	0.53	8005 (2187)	7462 (2865)	-849 (-1774, 76)		0.07
Protein (g)	84.7 (24.0)	91.8 (26.4)	0.29	86.7 (26.2)	90.0 (41.1)	-2.4 (-17.1, 12.3)		0.74
Total fat (g)	70.9 (26.5)	73.8 (28.4)	0.69	86.7 (26.2)	60.7 (25.0)	-11.5 (-23.2, 0.2)		0.05
SFA (g)	26.8 (12.3)	25.2 (11.7)	0.60	25.8 (12.2)	20.1 (9.5)	-4.7 (-9.0, -0.4)		0.03
CHO (g)	223.0 (68.9)	231.2 (96.7)	0.71	221.8 (66.8)	205.1 (95.2)	-22.8 (-50.9, 5.3)		0.11
Alcohol (g)	1.8 (7.4)	1.6 (5.5)	0.89	0.7 (1.9)	0.8 (3.3)	0.1 (-1.1, 1.3)		0.87
Fibre (g)	22.8 (8.8)	26.3 (11.2)	0.19	23.9 (9.0)	27.5 (12.8)	0.8 (-3.3, 4.9)		0.70
<b>%E from:</b>								
Fat	32.3 (8.1)	32.2 (6.2)	0.96	31.6 (7.2)	30.0 (6.0)	-1.5 (-4.7, 1.6)		0.32
Protein	18.2 (3.8)	19.3 (5.4)	0.39	19.0 (5.4)	20.8 (5.7)	1.1 (-1.4, 3.6)		0.37
CHO	45.9 (8.9)	44.7 (8.4)	0.59	46.0 (8.6)	44.9 (6.1)	-0.3 (-2.8, 2.1)		0.79
Other <sup>^</sup>	3.5 (2.3)	3.8 (2.4)	0.64	3.4 (1.2)	4.3 (2.2)	0.8 (0.0, 1.6)		0.04
SFA	12.2 (4.5)	11.0 (3.6)	0.27	11.6 (3.6)	10.0 (3.2)	-1.0 (-2.5, 0.5)		0.19

\*independent t-test, <sup>1</sup>mean change in the intervention compared to control group, adjusted for baseline levels of outcome, CI: Confidence Interval

SFA: Saturated fatty acid, CHO: Carbohydrate

<sup>^</sup> Percentage of total energy from alcohol, fibre and other combined



Table 8: Assessment of macronutrient intake via the three day food record by experimental group at baseline and six months: completers

	<i>Baseline, mean (SD)</i>			<i>6 month follow up, mean (SD)</i>		<i>Linear regression analysis</i>	
	Control (n = 19)	Intervention (n= 19)	P*	Control (n = 19)	Intervention (n=19)	Adjusted change <sup>1</sup> (95% CI)	mean p
Energy (kJ)	8300 (2093)	8240 (2400)	0.94	8257 (2268)	6766 (2475)	-1456 (-2791, -122)	0.03
Protein (g)	90.6 (23.1)	89.3 (26.2)	0.87	93.7 (25.9)	86.6 (47.1)	-6.3 (-29.2, 16.6)	0.58
Total fat (g)	75.6 (24.4)	73.3 (30.1)	0.80	74.8 (30.7)	53.2 (21.2)	-20.8 (-37.1, -4.5)	0.01
SFA (g)	28.4 (11.8)	25.1 (12.3)	0.40	26.8 (11.8)	17.3 (7.5)	-8.2 (-14.0, -2.3)	0.01
CHO (g)	222.4 (70.5)	221.9 (80.1)	0.98	220.4 (67.0)	182.0 (67.8)	-38.2 (-76.6, 0.3)	0.05
Alcohol (g)	2.4 (9.2)	2.4 (6.7)	1.00	0.7 (2.0)	1.2 (4.0)	0.5 (-1.3, 2.4)	0.58
Fibre (g)	22.1 (8.6)	27.3 (11.2)	0.12	23.8 (9.1)	29.1 (13.5)	1.8 (-4.7, 8.3)	0.58
<b>%E from:</b>							
Fat	33.8 (7.7)	32.2 (6.5)	0.49	32.7 (6.3)	28.9 (5.8)	-3.7 (-7.8, 0.4)	0.07
Protein	18.9 (3.6)	19.0 (5.4)	0.92	20.2 (5.9)	21.4 (5.9)	1.2 (-2.5, 4.9)	0.52
CHO	43.7 (7.4)	44.4 (9.3)	0.81	43.9 (7.0)	44.7 (5.9)	0.5 (-3.0, 3.9)	0.78
Other <sup>^</sup>	3.5 (2.8)	4.3 (2.8)	0.39	3.3 (1.3)	5.1 (2.4)	1.5 (0.4, 2.6)	0.01
SFA	12.7 (4.7)	10.9 (3.1)	0.19	11.7 (3.3)	9.4 (2.0)	-2.0 (-3.9, -0.2)	0.03

\*independent t-test, <sup>1</sup> mean change in the intervention compared to control group, adjusted for baseline levels of outcome, CI: Confidence Interval

SFA: Saturated fatty acid, CHO: Carbohydrate

<sup>^</sup> Percentage of total energy from alcohol, fibre and other combined

Intention to treat analysis of diet history data showed in the intervention compared with the control there was a greater reduction in intake of: energy (-1251kJ,  $p=0.01$ ), total fat (-16.8g,  $p=0.01$ ), SFA (-7.2g,  $p=0.01$ ) and intake of %E from total fat (-4.4%,  $p=0.01$ ) and SFA (-2.1%,  $p=0.01$ ) at six months (Table 5). A greater reduction in carbohydrate and alcohol intake at six months also occurred in the intervention group (-31.3g,  $p=0.04$  and -0.3g,  $p=0.00$ , respectively). Decreases in carbohydrate and alcohol however were not primary outcomes of interest. Completers analysis of diet history data demonstrated a similar trend to the former intention to treat analysis (Table 6). In the intervention group significantly greater intake of fibre and a reduction in %E from SFA approached significance (3.6g,  $p=0.06$  and -1.9g,  $p=0.05$  respectively) at six months with no significant reduction in CHO or alcohol.

Intention to treat analysis of food record data showed there was only a greater reduction in the intervention group in SFA intake (-4.7g,  $p=0.03$ ) at six months (Table 7). Greater reduction of total energy (-849kJ,  $p=0.07$ ) and total fat (-11.5g,  $p=0.05$ ) intake in the intervention group at six months however approached significance. Completers analysis of food record data showed similar results to those of the diet history intention to treat analysis. There were greater reductions in the intervention group compared with the control group at six months in energy (-1456kJ,  $p=0.03$ ), total fat (-20.8g,  $p=0.01$ ) and SFA (-8.2g,  $p=0.01$ ) and in the %E from SFA (-2.0%,  $p=0.03$ ) (Table 8). Greater reduction in %E from total fat in the intervention group at six months approached significance (-3.7g,  $p=0.07$ ) (table 8).

Reduction in energy intake was confirmed by changes in body weight. Both the intention to treat and completers analyses showed participants in the intervention lost a small but significant amount of weight at six months [(-2.4kg  $p=0.03$  and -2.8kg  $p=0.04$ , respectively)] (Table 9). The proportion of participants who achieved the study goal of an energy intake derived from total fat of  $\leq 30\%$  at six months was greater in the intervention group in both analyses of diet history data (Table 10, Table 11). Intention to treat and completers analysis of diet history data and the food record completers analysis demonstrated participants in the intervention group were more likely than the controls to achieve the dietary fibre target at six months (Table 10, Table 11).

Table 9 Assessment of weight and body mass index by experimental design at baseline and six months: intention to treat and completers

	<i>Baseline, mean (SD)</i>			<i>6 month follow up, mean (SD)</i>		<i>Linear regression analysis</i>	
	Control	Intervention	P <sup>^</sup>	Control	Intervention	Adjusted change <sup>1</sup> (95% CI)	mean p
Weight (kg)							
Intention to treat	75.0 (15.2)	82.2 (16.8)*	0.09	75.0 (14.6)	79.2 (15.4)*	-2.4 (-4.5, -0.3)	0.03
completers	74.6 (13.9)	83.7 (17.9)*	0.06	74.7 (13.0)	80.1 (16.6)**	-2.8 (-5.4, -0.2)	0.04
BMI (kg/m <sup>2</sup> )							
Intention to treat	29.4 (5.2)	31.6 (5.3)*	0.11	29.4 (5.1)	30.4 (4.8)*	-1.0 (-1.8, -0.2)	0.02
completers	29.3 (5.0)	31.8 (5.6)*	0.11	29.3 (4.8)	30.4 (5.1)**	-1.1 (-2.1, -0.2)	0.02

Table 10: Odds ratios for meeting dietary targets assessed by the seven day diet history and three day food record by experimental group at baseline and six months: intention to treat

<i>Dietary target</i>	<i>Baseline, n(%)</i>			<i>6 month follow up, n(%)</i>		<i>Logistic Regression</i>	
	Control n=30	Intervention n=29	P*	Control n=30	Intervention n=29	Odds Ratio (95% CI)	P
%E from total fat $\leq 30$							
diet history *	14 (46.7)	14 (48.3)	1.00	13 (43.3)	23 (79.3)	9.2 (2.1, 41.3)	0.00
food record^	11 (36.7)	13 (44.8)	0.60	12 (40)	16 (55.2)	1.8 (0.6, 5.4)	0.32
%E from SFA fat $\leq 10$							
diet history*	14 (46.7)	14 (48.3)	1.00	18 (60.0)	23 (79.3)	2.8 (0.8, 9.7)	0.10
food record^	13 (43.3)	16 (55.2)	0.44	14 (46.7)	19 (65.5)	2.0 (0.6, 6.5)	0.24
Dietary fibre 15g per 4200kJ							
diet history*	10 (33.3)	11(37.9)	0.79	9 (30.0)	20 (69.0)	8.5 (2.0, 35.4)	0.00
food record^	7 (23.3)	10 (34.5)	0.40	8 (26.7)	15 (51.7)	3.7 (0.8, 16.0)	0.09

\*Fishers Exact test

Table 11: Odds ratios for meeting dietary targets assessed by the seven day diet history and three day food record by experimental group at baseline and six months: completers

<i>Dietary target</i>	<i>Baseline, n(%)</i>			<i>6 month follow up, n(%)</i>		<i>Logistic Regression</i>	
	Control	Intervention	P <sup>#</sup>	Control	Intervention	Odds Ratio (95% CI)	P
%E from total fat ≤30							
diet history *	11 (47.8)	9 (37.5)	0.56	10 (43.5)	18 (75.0)	6.9 (1.5, 31.3)	0.01
food record <sup>^</sup>	7 (36.8)	9 (47.4)	0.74	8 (42.1)	12 (63.2)	2.3 (0.6, 8.6)	0.21
%E from SFA fat ≤10							
diet history*	11 (47.8)	9 (37.5)	0.56	15 (65.2)	18 (75.0)	1.8 (0.5, 6.6)	0.38
food record <sup>^</sup>	8 (42.1)	9 (47.4)	1.00	9 (47.4)	12 (63.2)	1.9 (0.5, 7.0)	0.35
Dietary fibre 15g per 4200kJ							
diet history*	8 (34.8)	8 (33.3)	1.0	7 (30.4)	17 (70.8)	8.2 (1.9, 35.8)	0.01
food record <sup>^</sup>	3 (15.8)	9 (47.4)	0.08	4 (21.1)	14 (73.7)	8.2 (1.6, 41.9)	0.01

#Fishers Exact Test

\*: control n=23, intervention 24,

<sup>^</sup>: control n =19, intervention n= 19

## Discussion

The HeLP GDM study demonstrated positive dietary change associated with reduction of T2DM risk was achieved in a sample of women with prior GDM. Reduction in energy, total fat and SFA were evident regardless of dietary assessment method, though with some differences apparent. The differences seen, however, may have been caused by the statistical treatment of data, rather than a discrepancy between dietary assessments of actual dietary change.

The method of choice for the intention to treat analysis was baseline value carried forward. The intention to treat method assumes over the intervention period there was no change in the outcome of interest. Such an assumption has the potential to underestimate the intervention effect and it appears this may be the case with the current food record analysis.

In the food record completers analysis significantly greater reduction in energy and total fat in the intervention group occurred. However, with one third of the number of food records assuming no change at six months, these results instead approached significance or were no longer significant in the corresponding intention to treat analysis. Additionally the power calculations for the HeLP GDM study were based on an earlier pilot examining a physical activity intervention and not based on expected dietary change. Thus it seems the food record analysis became underpowered. A larger sample size of participants randomised may have thus clarified the discrepancy.

Consumption of  $\leq 30$  of %E from total fat as previously mentioned, has been recommended by the ADA.[38] Furthermore, consumption of  $\leq 10$ %E from SFA has been previously tested as an appropriate dietary fat intake target for the prevention of T2DM in at risk individuals.[9] In this thesis only the diet history analyses showed intervention participants were more likely than controls to achieve an intake of  $\leq 30$  of %E from total fat. Such data indicates change in total dietary fat intake to recommended levels is achievable however lack of significant results from the food record data indicates further substantiation is needed. Despite the greater absolute reduction in SFA achieved in the intervention group,

dichotomising the data to determine success in achieving dietary targets decreased the power to show a significant difference in meeting the SFA target.

There was only limited impact of the intervention on increased dietary fibre intake with non-significant differences in absolute intake of dietary fibre intake between the intervention and control. However, there were a greater proportion of participants in the intervention at six months who consumed a fibre intake of 15g per 4200kJ compared with the control group. These results indicate a greater proportion of individuals in the intervention than those in the control, managed to maintain an adequate level of fibre intake in the face of the energy intake reduction that occurred in both groups.

The HeLP GDM study was not specifically a weight loss intervention but an intervention aimed at dietary change of fat, fibre and energy intake. However that said, the greater reduction in energy intake reported by the intervention group was confirmed by the small but significant consistent reduction in body weight and BMI. Although the mean reduction of 2.4kg falls short of the clinically defined beneficial 5-10% loss of body weight,[114-116] DPP data demonstrated a relative risk reduction of T2DM of 16% per kilogram of weight lost, after adjustment for exercise and diet composition.[51] Furthermore it is encouraging to note that the weight lost as a result of the dietary change intervention is comparable to the amount of weight lost reported in a Cochrane review of long-term non-pharmacological weight loss interventions for adults with pre-diabetes (-2.8kg 95% CI -1.0 to -4.7).[117]

Previous large scale interventions in individuals at high risk of diabetes have shown diabetes is preventable. [6-9] Due to repeated calls in the literature for development of interventions to reduce T2DM risk in women with prior GDM it appears that a number of studies of varying size including the HeLP GDM study were concurrently conducted.[18-20]. However the pool of available literature is still small. Given the average baseline BMI in all studies at minimum has been within the overweight range, a comparison of the dietary outcome across studies is possible albeit limited, given dietary change is often secondary to change in T2DM incidence or weight.

The dietary change particularly for total fat reported in this thesis is comparable to two other studies.[18, 20] One of the studies was also an Australian pilot RCT although conducted in a rural setting with a smaller ( $n = 38$ ) sample size.[18] The intervention of this rural study was comprised of six months of telephone counselling administered by two diabetes educators trained in motivational interviewing. Counselling centred around the Australian guidelines to health eating and national recommendations for PA. The intervention involved five weekly phone calls and subsequently one call a month for the remaining 6 months. A similar reduction in total fat was reported (  $-19$  g/d; 95%CI:  $-37$  to  $-1$ ) with a slightly greater reduction in BMI ( $-1.5\text{kg/m}^2$ , 95%CI:  $-2.8$ ,  $-0.1$ ) and weight ( $-4.0\text{kg}$ , 95%CI:  $-7.6$ ,  $-0.5$ ) occurring in the rural study.

The second study a 2011 American lifestyle intervention involving 197 women with a GDM pregnancy, demonstrated reduction in intake of total fat and changes in weight are sustained postpartum, when intervention is commenced shortly after GDM diagnosis.[20] The intervention run predominantly by dietitians via telephone was modelled on the DPP curriculum previously mentioned in section. The primary aim was for the women to achieve their pregravid weight, or to lose 5% of body weight if overweight, with the secondary aim to consume 25%E or less from fat per day and to increase PA. When compared with the results of this thesis, a slightly lower but significant decrease in %E from total fat ( $-3.55$ ) at seven months postpartum was also achieved.[20]The difference in the greater absolute proportion of intervention participants achieving their weight goal however failed to reach significance ( $16.1\%$ ,  $p = 0.07$ ) with absolute reductions in weight not reported.

The study's weight data was also stratified according to whether women exceeded gestational weight gain (GWG) guidelines. The proportion of women who did not exceed GWG who reached their weight goal at 12 month postpartum in the intervention was significantly greater than those who did not exceed the GWG in the control group ( $22.5\%$   $p = 0.04$ ). A strong positive association exists between increased GWG and postpartum weight retention.[119] Consequently these results suggest that targeting dietary behaviour and weight management during the GDM pregnancy rather than at postpartum, may increase the effectiveness of a postpartum T2DM risk reduction intervention, by preventing excessive GWG.[20]



Not all studies of postpartum women with prior GDM have shown improvement in dietary behaviour or subsequent reduction in T2DM incidence after a dietary intervention.[17] In an Australian RCT of 200 women with prior GDM who were also diagnosed with IGT, the women were contacted by a dietitian for a dietary review three monthly for a median of 51 months. The trial reported no difference between intervention and control in T2DM prevalence and BMI increased in both groups. The absolute values for macronutrient intake were not reported.

Diet scores for fat and fibre in the published paper are mentioned with no information on the validity or background on how the scores were determined. Nevertheless there were comparable non-significant improvement in fat and fibre scores in both intervention and control at final assessment. Very limited information is provided regarding the content of the intervention and by the author's own admission the intervention may not have been sufficiently intensive to have resulted in a reduction of T2DM prevalence.

A more definitive study of lifestyle intervention involving dietary change is currently in progress in China. The two year RCT titled Tianjin Gestational Diabetes Mellitus Prevention Program (TGDMPP) which recruited between 2005 to 2009 has enrolled 1180 postpartum women with prior GDM.[19] The study will assess whether an individually designed diet and exercise program can prevent or delay the onset of T2DM in these women, with the primary outcome of the trial development of T2DM. The dietary fat targets of TGDMPP are identical to those within this thesis. However, in TGDMPP adequate dietary fibre intake is defined as a range between 20-30g.

The intervention component of the TGDMPP trial promotes an overall healthy eating pattern similar to that of the HeLP GDM study. The intervention involves multiple face to face individual counselling with additional telephone calls with a dietitian. Dietary intake is monitored over the first year five times via 3 day food records with corresponding feedback provided. The TGDMPP interim one year data report demonstrated in the subgroup of overweight women there was a comparable amount of weight lost in their intervention group, when compared with the weight loss reported in this thesis.

Baseline %E from total fat in the TGDMP was comparable to the intakes recorded in this thesis. However, at baseline fibre intake per 4200kJ in TGDMP was approximately six grams in both the intervention and control group, which is as low as half the baseline dietary intake of participants in the HeLP GDM study. In this thesis SFA intake was reported to be between 11-13% compared with in TGDMP where it was approximately 8%.

Traditional Chinese food patterns focus on vegetables, fruits, small quantities of meat, and either rice or wheat products with few if any dairy products. This traditional pattern provides less SFA, than the typical Western dietary pattern.[120] The comparison of baseline intakes of fat and fibre between this study and the current thesis suggest required changes to the woman's diet to reduce T2DM may be linked to culture and emphasis on areas of required change may differ accordingly.

In summary dietary and weight changes in women with prior GDM have been reported in a number of research settings, with varying levels of intervention intensity at differing time points after diagnosis of GDM and across a number of ethnicities.[18-20] The changes in dietary intake and weight reported in this thesis has compared favourably with other similar lifestyle trials[18] and overall the accumulating literature surrounding behavioural intervention for women with prior GDM indicate postpartum dietary and weight change is achievable in women at risk of developing T2DM.

### **Limitations and strengths of the current dietary analysis**

The dietary data of this thesis was obtained during a pilot conducted as a single blind RCT, with the RCT considered the most rigorous way of determining whether a cause-effect relation exists between treatment and outcome.[122] The participants in the HeLP GDM study were from culturally diverse backgrounds previously associated with an increased risk of GDM [31, 32] and T2DM [123] thus allowing the findings to be generalised across more than the Australian Caucasian population.

Several limitations must be noted when evaluating dietary data from the HeLP GDM study, of which first is the small sample size of only n=59. Furthermore the substantial number of missing data at six months further illustrates the challenges of working with this population of women with young families. The HeLP GDM study was a pilot for a potentially larger trial, with ascertaining the effect size of the intervention one of the pilot's main aims.

As previously mentioned imputation of the baseline value at six months for approximately one third of the participant's food record may have substantially underestimated the dietary change achieved as results from the food record completers analysis suggests. It is probable that the majority of these women had prior dietary counselling during their GDM pregnancy not only to manage their diabetes at the time but for future T2DM prevention. It is possible that women who maintained positive dietary change from their counselling into the postpartum period may not have shown significant improvement in the study, thereby reducing the overall intervention effect.

Exclusive breastfeeding is recommended by the WHO until an infant is 6 months of age after which it is recommended breastfeeding continue alongside nutritionally adequate foods until two years of age.[124] Women who breastfeed require an increase in their energy requirements and breastfeeding has been shown to accelerate recovery of prepregnancy weight.[124] However the effects of breastfeeding on dietary intake and weight in this study were minimal as participants were recruited six months to four years post their GDM pregnancy, with only two participants having had children under one and none were still breastfeeding at the time of baseline data collection.

It is unknown what effect inclusion of Calorie King nutrient data when the appropriate substitutions were lacking in the FoodWorks software had on the energy and macronutrient intakes obtained. However the food substitution information obtained from Calorie King was applied consistently and should not have affected the changes over time that was seen.

Apart from baseline measured weight the remaining information collected was self-reported. Self-reported data can be affected by social desirability bias (SDB), the tendency to respond in such a way as to avoid criticism and respond in a manner consistent with expected norms or to seek social approval.[125] Previous research has shown even a brief intervention can promote socially desirable responses by participants assigned to the intervention group.[125] In the study women were blindly assigned to either an intervention or control group prior to a telephone interview regarding dietary intake and health.

The intervention group prior to the interview were provided with an information sheet containing messages regarding healthy intake of fruit and vegetables. While the intervention and control group reported similar data in regard to most characteristics analysed, total fruit, total vegetable and total fruit and vegetable intake were significantly higher in the intervention group.[125] In the HeLP GDM intervention to reduce the possibility of SDB on the self-reported data, MG was blinded to group assignment while she conducted all dietary assessments.

No objective biomarkers were collected to validate energy or protein intake in the HeLP GDM intervention. As SDB can overestimate an intervention effect, any future trial in this population should include a sub study in which biomarker information is compared with self-reported dietary data. In the current thesis analysis no assessment as to the degree of underreporting at any time point in the pilot was completed. Thus it is unknown if the intervention which reinforced the message to consume lower fat, higher fibre wholegrain foods resulted in any greater amount of under or over reporting in the intervention group compared to the control.

Prior formative research has been conducted in a purposely selected sample of Cantonese/Mandarin, Arabic and English speaking women with a history of GDM from the same area health service. The research indicated this population of women wanted individually tailored, culturally appropriate guidance regarding their diet, cooking methods and menu planning, preferably in their native language. Budget constraints did not allow for translated materials to be provided in the HeLP GDM study. Thus it is unknown whether the

intervention delivered, was sufficiently culturally sensitive within this area health service of NSW. It is unknown if a more culturally tailored intervention ie provision of translated materials or dietitians of the same cultural background, may have resulted in a greater intervention effect.

Despite the limitations it can be summarised that a lifestyle intervention in women with prior GDM targeting dietary change of fat, fibre and energy intake resulted in statistically significant and clinically favourable change in such intakes. [126]

## CHAPTER FOUR - RELATIVE VALIDITY OF DIETARY ASSESSMENT IN THE HeLP GDM STUDY

### Introduction

Given that the assessment of dietary data validity within the study context can strengthen a dietary study's primary findings the secondary aim of the thesis, was to conduct a relative validity analysis on dietary data collected in the HeLP GDM study. This chapter of the thesis is an analysis of baseline and six month dietary data from the HeLP GDM study which details the agreement in estimates of energy and macronutrient intake at the group and individual level, between the seven day telephone diet history and three day estimated food record.

### Method

The section Dietary assessment and Data entry in chapter three discussed the dietary assessment methods and the dietary data entry respectively. The proceeding section outlines the statistical methods chosen to assess relative validity.

### Statistical analysis

It is difficult to obtain the true value for macronutrient intake via self-reported methods. The assessment of agreement between the seven day telephone diet history and three day estimated food record is between two 'non-perfect' measures of dietary intake each with their own different source of measurement error as specified in the Dietary assessment methods section of the literature review. A decision regarding agreement between two assessment methods is a relatively subjective one.

In the current analysis relative validity was assessed using the paired t-tests, Pearson product moment correlation coefficients and Bland Altman scatterplots. In addition the paired t test was used to determine any significant difference in mean bias ( $\text{mean}_{\text{bias}}$ )<sup>4</sup> at six months. The Pearson product moment correlation coefficient gives information about the strength of a

---

<sup>4</sup> Bias is computed as the value determined by one method minus the value determined by the other method. The mean bias is thus the average difference between the two methods of measurement.

relationship and the paired t-test confirms whether group means are significantly different. To determine the level of individual agreement across a given range, Bland Altman scatterplots were constructed. [127]

The limits of agreement<sup>5</sup> (LOA) in a Bland Altman scatterplot demonstrate the extent of precision around the mean<sub>bias</sub>. A greater degree of separation between the LOA indicates a larger variability in the difference between methods and a lower precision of agreement between measurements. Individuals reporting an energy intake outside the plausible energy intake range of  $\geq 2101\text{kJ}$  to  $\leq 14699\text{kJ}$  were excluded from the analyses. [128]

## Results

Dietary estimates from both the diet history and food record were obtained for n= 59 participants at baseline. At baseline two participants were excluded from relative validity analyses as their reported baseline energy intakes were above the upper limit of the plausible energy intake range: one participant reported an intake of 15405kJ via the diet history and the second participant a food record intake of 16012kJ. The total sample was thus n = 57 at baseline. Due to missing food record data, sample size was n = 38 for tests of agreement at six months and analysis of change in mean<sub>bias</sub> between baseline and six months. These analyses involved n=19 from each treatment group with no reported intake data excluded.

At baseline mean<sub>bias</sub> showed the diet history overestimated energy and dietary fibre and underestimated total fat and SFA with clinically small mean biases for each variable. At six months, however, the diet history underestimated all variables. There was no significant difference between mean estimate of energy, total fat, SFA, dietary fibre and %E from total fat and SFA from the diet history and food record at baseline. There was also no significant difference in bias between baseline and six months.

Moderate correlation coefficients between estimates were found for all variables at baseline and six months (Table 12). Non-significant  $r^2$  values for all variables except fibre at six

---

<sup>5</sup> The LOA define the limits within which 95% of the differences between the two methods are expected to fall.

months demonstrated there was no linear relationship between bias and mean intake. For dietary fibre intake at six months, upon removal of an outlier where the bias was -36g, the linear relationship was no longer demonstrated ( $r^2$  0.049  $p=0.19$ ).

At baseline and six months the standard deviation of the mean bias was large for all variables indicating a considerable extent of intra individual variation in measurements made with the diet history and food record. At baseline for all variables more than 5% of the cases were outside the 95% confidence intervals of the mean bias. However at six months this was no longer the case.

Limits of agreement calculated at baseline and six months were clinically large. At baseline the LOA for total energy intake illustrated that any estimate energy from the diet history differed by between -4072 and 4163kJ compared to the food record (Figure 3). Additionally the LOA for total fat, SFA, fibre, %E from total fat and %E from SFA were respectively: -65g and 58g (Figure 4), -25g and 22g (Figure 5), -14g and 16g (Figure 6), -18.5% and 14.5% (Figure 7), -9% and 7% (Figure 8). At six months the LOA for energy total fat, SFA, fibre, %E from total fat and %E from SFA from the diet history differed respectively by between -5096 and 3562 kJ (Figure 9), -61.9g and 40.9g (Figure 10), -29.1g and 14.1g (Figure 11), -20.3g and 16.1g (Figure 12), -15.4% and 10.6% (Figure 13), -7.5% and 4.7% (Figure 14)



**Table 12: Relative validity of energy and macronutrient intake between the seven day telephone diet history and three day estimated food record at baseline and six months**

	Baseline n = 57							Six months n=38		Difference in bias over time n = 38			
	DH	Mean	FR	Mean	Paired	R value	Bias <sub>mean</sub> (SD)	*R <sup>2</sup> (p)	R value	*R <sup>2</sup> (p)	Baseline	Six months	Paired
		(SD)		(SD)	t-test P	(p)			(p)		Bias <sub>mean</sub> (SD)	Bias <sub>mean</sub> (SD)	t-test P
Energy (kJ)		8095		8049	0.87	0.63 (.00)	46 (2101)	0.06 (.06)	0.55	0.03	-174 (2018)	-767 (2209)	0.16
		(2621)		(2148)					(.00)	(.31)			
Fat (g)		67.2 (31.9)		70.8 (26.5)	0.39	0.43 (.00)	-3.6 (31.4)	0.04 (.13)	0.54	0.01	-4.3 (33.1)	-10.5 (26.2)	0.34
									(.00)	(.57)			
SFA (g)		23.9 (13.9)		25.5 (11.6)	0.32	0.59 (.00)	-1.6 (11.8)	0.05 (.11)	0.61	0.00	-2.3 (11.9)	-4.5 (9.5)	0.32
									(.00)	(.80)			
Fibre (g)		24.9 (9.3)		23.9 (9.4)	0.36	0.67 (.00)	0.9 (7.6)	0.00 (.92)	0.61	0.23	0.4 (8.3)	-2.1 (9.3)	0.17
									(.00)	(.00)			
%E from:													
Total fat (g)		30.2 (7.6)		32.2 (7.2)	0.08	0.35 (.01)	-2.0 (8.4)	0.00 (.68)	0.45	0.00	-1.5 (8.2)	-2.4 (6.6)	0.66
									(.01)	(1.0)			
SFA (g)		10.6 (3.7)		11.6 (4.0)	0.07	0.46 (.00)	-1.0 (4.0)	0.01 (.52)	0.50	0.01	-0.9 (4.2)	-1.4 (3.1)	0.59
									(.00)	(.57)			

\*linear regression

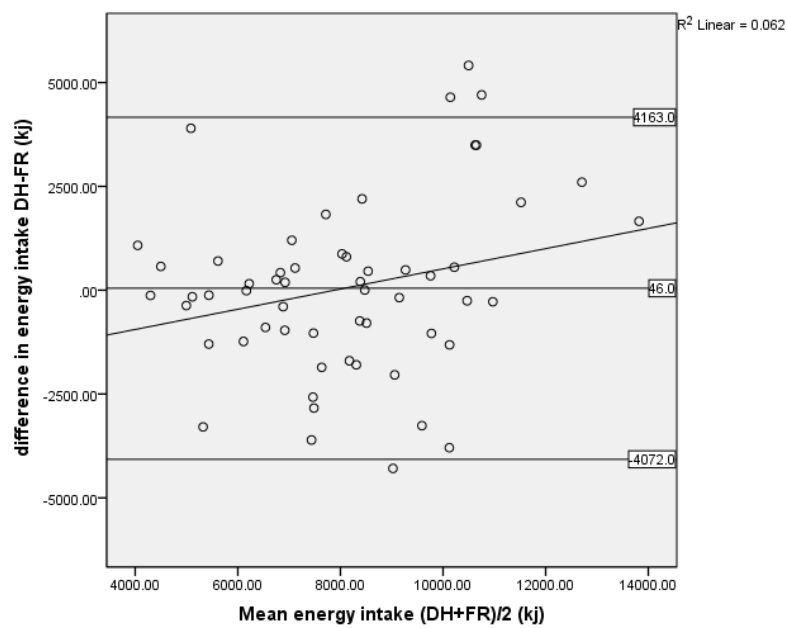


Figure 3 : Bland- Altman plot: agreement of estimated energy intake between a seven day telephone diet history and three day estimated food record at baseline

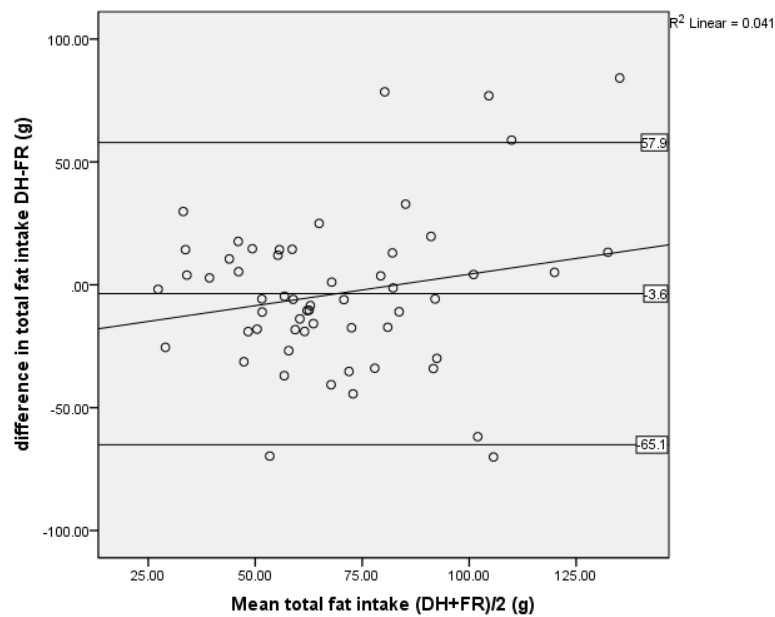


Figure 4: Bland- Altman plot: agreement in estimated total fat intake between a seven day telephone diet history and three day estimated food record at baseline

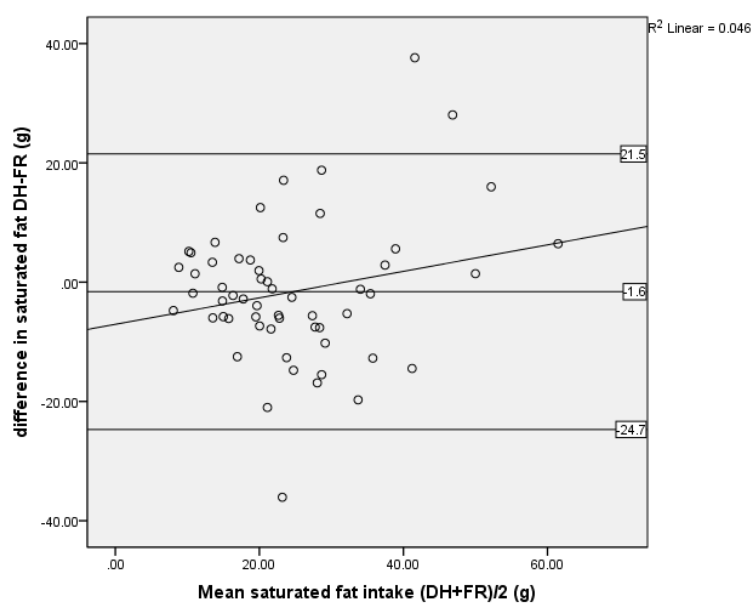


Figure 5: Bland- Altman plot: agreement in estimated saturated fat intake between a seven day telephone diet history and three day estimated food record at baseline

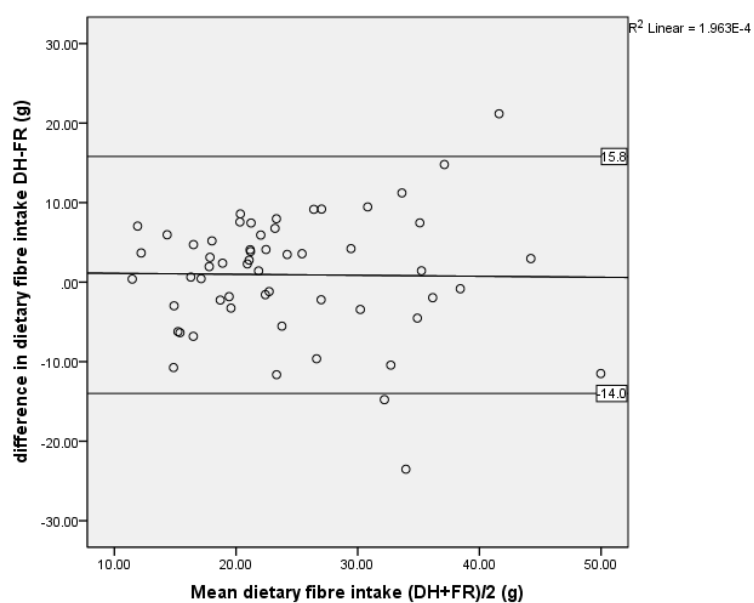


Figure 6: Bland- Altman plot: agreement in estimated fibre intake between a seven day telephone diet history and three day estimated food record at baseline

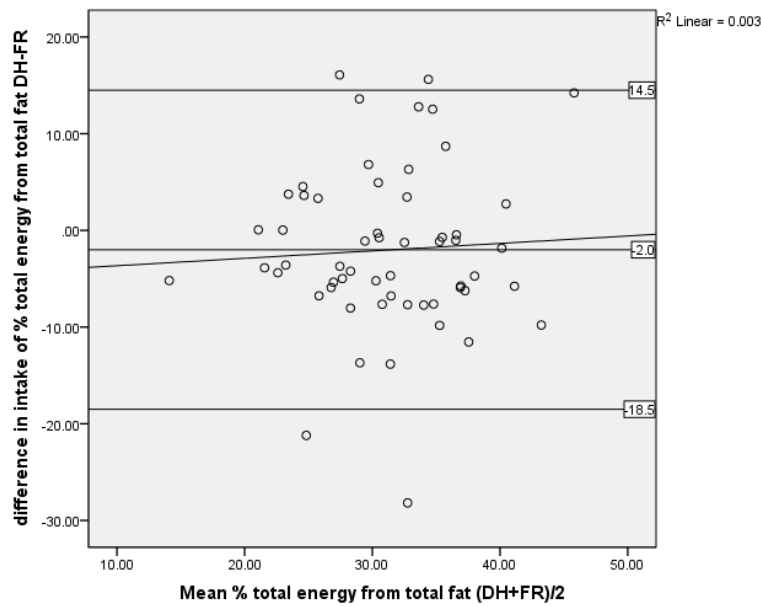


Figure 7: Bland-Altman plot: agreement in estimated %E intake from total fat between a seven day telephone diet history and three day estimated food record at baseline

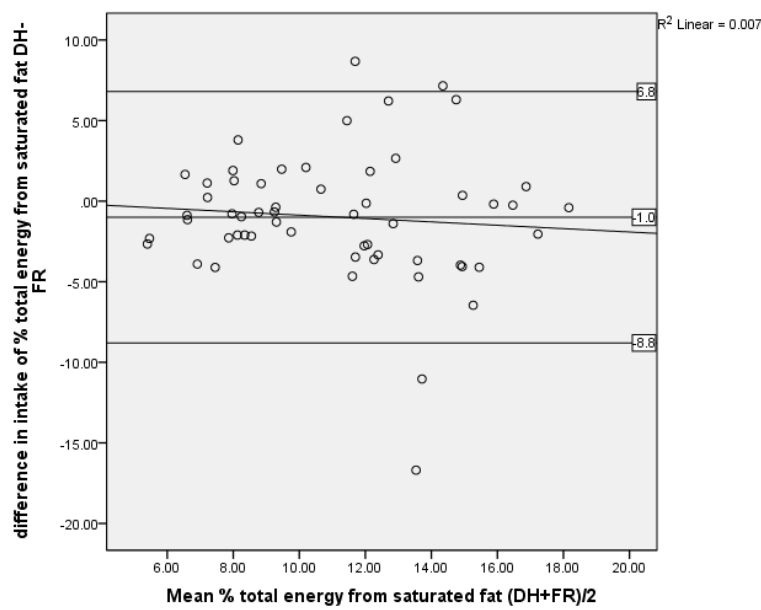


Figure 8: Bland-Altman plot: agreement in estimated %E intake from saturated fat between a seven day telephone diet history and three day estimated food record at baseline

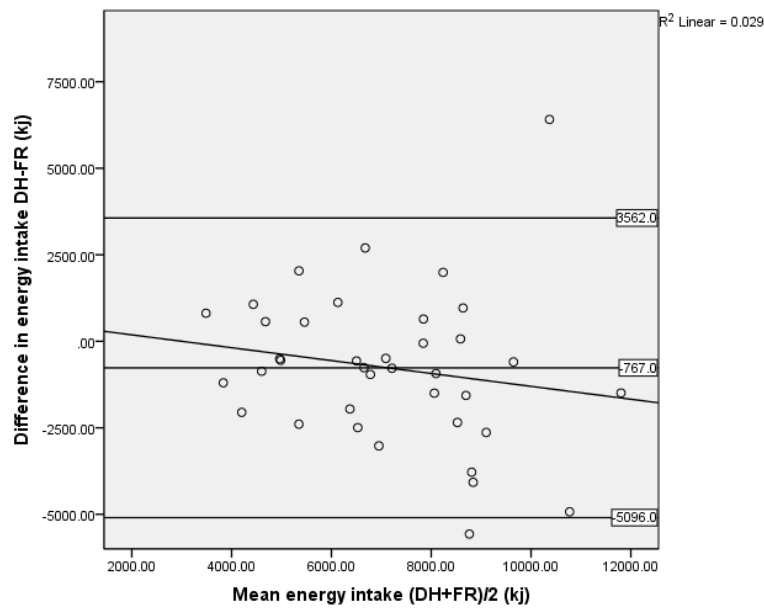


Figure 9: Bland- Altman plot: agreement in estimated energy intake between a seven day telephone diet history and three day estimated food record at six months

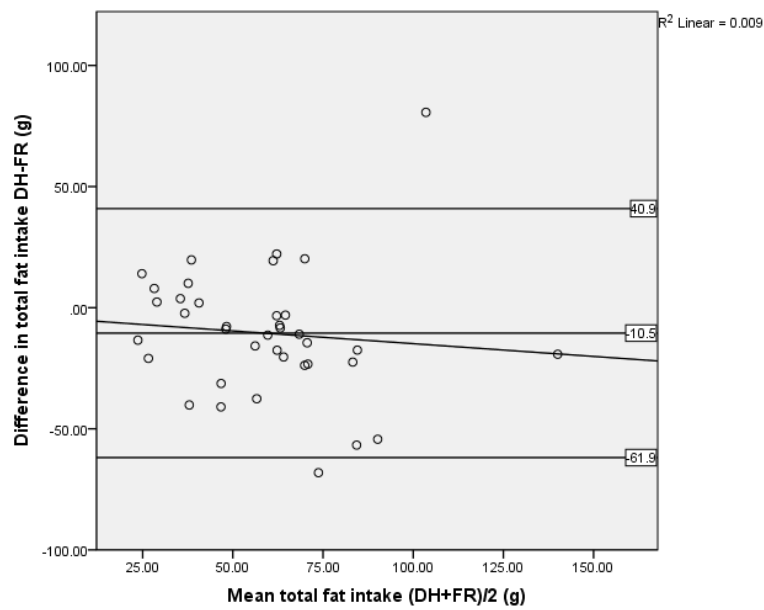


Figure 10: Bland- Altman plot: agreement in estimated total fat intake between a seven day telephone diet history and three day estimated food record at six months

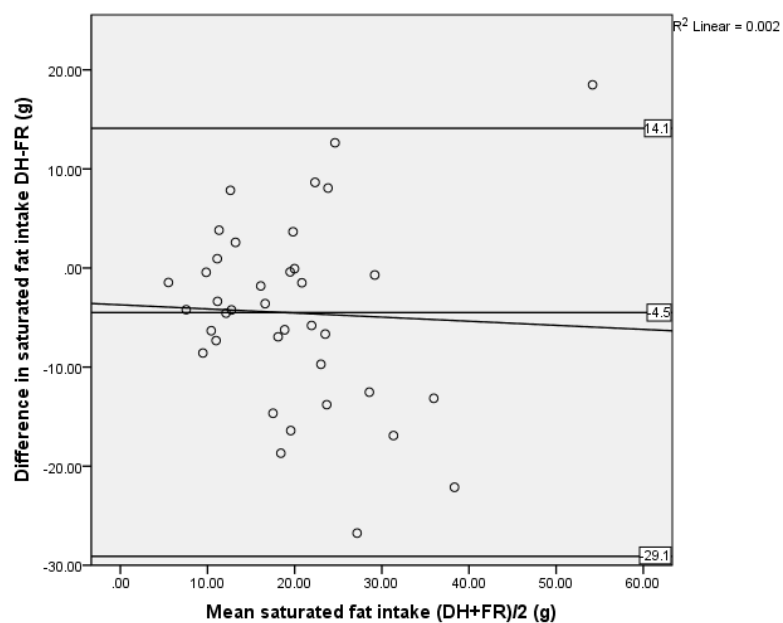


Figure 11: Bland- Altman plot: agreement in estimated saturated fat intake between a seven day telephone diet history and three day estimated food record at six months

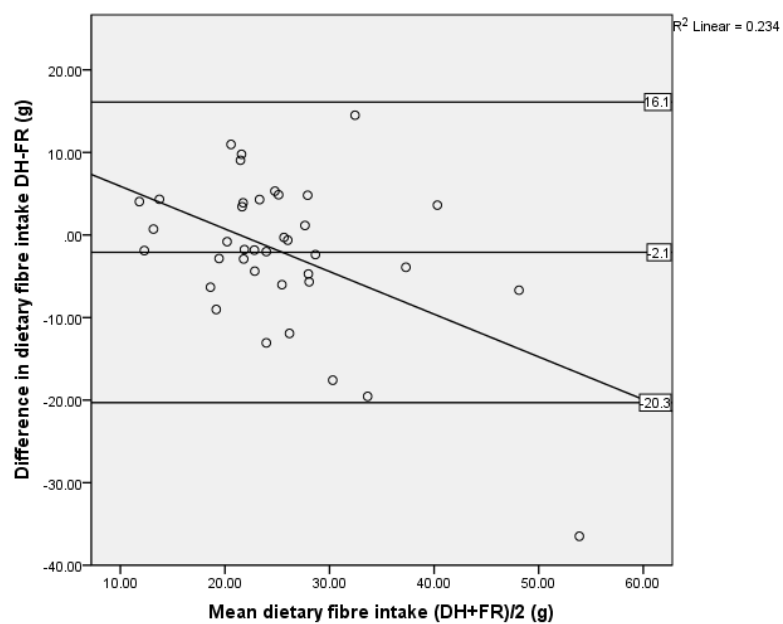


Figure 12: Bland- Altman plot: agreement in estimated fibre intake between a seven day telephone diet history and three day estimated food record at six months

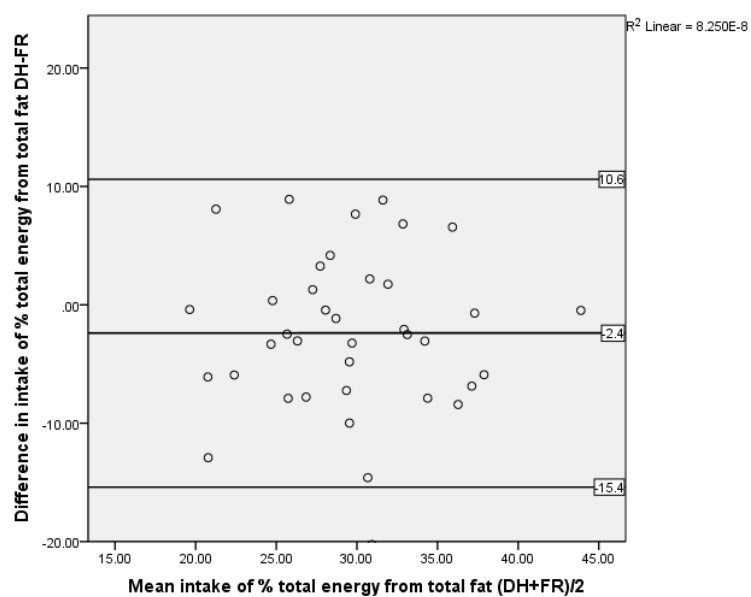


Figure 13: Bland- Altman plot: agreement in estimated %E intake from total fat between a seven day telephone diet history and three day estimated food record at six months

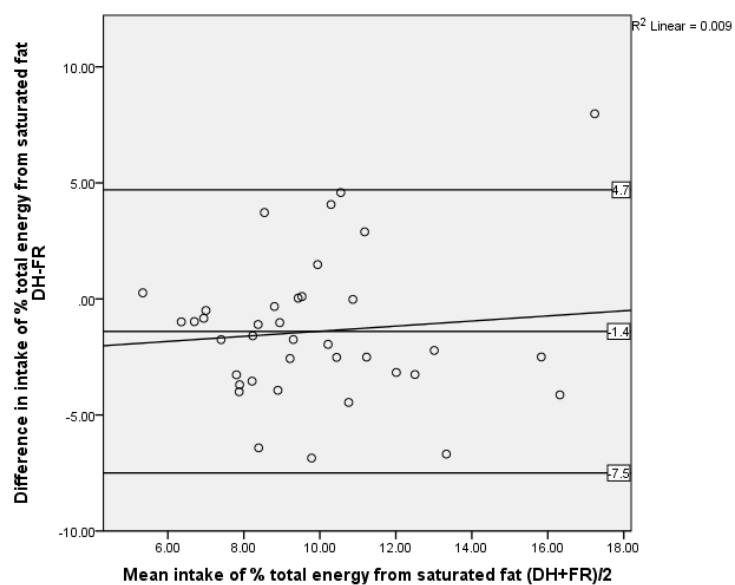


Figure 14: Bland- Altman plot: agreement in estimated %E intake from saturated fat between a seven day telephone diet history and three day estimated food record at six months

## Discussion

The relative validity analysis between the diet history and food record demonstrated that in a sample of women with prior GDM there was good agreement between the methods at the group level. Comparability across relative validity studies in dietary assessment can be difficult due to different assessment methods compared, different populations and variables under study, statistical manipulation of data prior to analysis and statistical assessment.

In previous literature of both healthy individuals and those with T2DM, the diet history has been found to both under or overestimate dietary intake, with underestimation cited by the majority.[70, 80, 96] Sparse information is known about the relative validity of estimates of fibre obtained by the diet history compared with the food record possibly due to the greater focus in the earlier years on dietary fat. Only one validation study reported a non-significant mean overestimation of fibre by the diet history which concurs with the baseline results of this thesis. [97]

A prior analysis in women with recent GDM found the diet history to overestimate energy and total fat but not SFA compared to the food record.[70] Overestimation of energy reported in the sample was however much larger than the current overestimation (563kJ versus 46kJ, respectively) and non-significantly different from their food record estimate. Between the two studies energy and SFA as %E followed the same trend however results for total fat differed, with total fat underestimated at both time points in the current study.

Most studies report large SD of mean bias for all dietary variables however this large intra-individual variation is expected. The diet history and the food record incorporate different characteristics, with the diet history a reflection of usual intake and a three day food record in reality a measure of actual intake. In the current study by six months half the sample had been treated dietetically. This may have contributed to a more stable pattern of dietary intake, a better awareness of portion size and or a general increased awareness of intake, all of which may have contributed to the reduction in outliers and narrower LOA seen at six months.



When compared with the other relative validity studies the moderate correlation coefficients obtained in this thesis analysis is within the range published in the literature for energy  $r = 0.47 - 0.7$ , [80, 81, 95, 96] However there is less consistency in correlation coefficient for %E from total and SFA. The moderate correlation coefficients found in this study were however lower than most studies for %E from total fat ( $r = 0.35$  vs  $r = 0.57$ , [81]  $r = 0.62$ , [96]  $r = 0.64$  [80]) and %E from SFA ( $r = 0.46$  vs  $r = 0.61$ , [70]  $r = 0.64$ , [96]  $r = 0.75$ , [80]) Within the current study there was minimal variation in correlation coefficients between baseline and six months.

At the end of the HeLP GDM intervention significant reductions in total and SFA were demonstrated by diet history and food record data. With this study and others suggesting an underestimation of energy, fat and SFA by the diet history [80, 96] caution could be encouraged in considering the magnitude of the intervention effect. The food record completers analysis however found there were greater reductions in these variables than those reported by the diet history.

There are a number of limitations to this relative validity analysis between the telephone diet history and estimated food record. Data for the analysis was not stratified according to treatment group therefore it is unknown if there are differences between methods according to treatment group. Actual accuracy of both methods is unknown as neither dietary measure was measured against criterion validity for energy intake. Furthermore, exclusion from analyses for underreporting or over reporting was not study specific and the plausible energy intake range included wide limits. Such wide limits which may have resulted in true under or over reporters to have remained in the analysis thereby influencing the variation in precision.

The decision to administer the traditional open ended diet history interview via the telephone and to request an estimated rather than weighed food record was chosen to maximise recruitment and minimise missing data. It was believed a change in administration of the methods would reduce participant burden in a population who are often difficult to recruit and retain to dietary interventions.[72] Previous literature in the 24 hour recall method indicated no difference in underreporting between estimates obtained in a face to face

interview compared with those obtained via the telephone.[100] However it is unknown the exact impact different mode of diet history administration had on the precision of diet history's to agree with food record estimates.

Misrepresentation of portion size of food consumed which contributes to overall misreporting, is problematic in any dietary assessment. It is conceivable that portion size estimation was limited even further by use of the telephone compared to face to face. In prior conversational analysis of the diet history use of the term "probably" reflected how individuals estimate portion size.[129] In a face to face diet history difficulty in portion estimation identified by this word could be lessened by using visual portion size aids. Use of such strategy is, however, limited when the diet history is conducted over the telephone.

In addition the study found sensitive topics such as consumption of chocolate, take away, butter or margarine and red meat or pork were accompanied with hesitation, pauses, explanation and laughter.[129] In this paper written by Tapsell et al they advise "*the diet history interviewer recognise these "marked" responses require special attention, with hesitation and or explanations be supported and considered positively*".[129] A number of these responses such as hesitation or pause may be potentially missed or misinterpreted when an interview is conducted over the telephone, resulting in decreased opportunities to increase reporting accuracy. It is of interest to note the large SD of mean bias identified in the study is comparable to those reported in other relative validity studies of the diet history. That said, further study of the diet history administered by both methods would clarify the extent of this potential error.

In order to assist in portion size estimation, participants in the HeLP GDM pilot were told to have available the standard measuring cups and spoons they were given to complete the food record with them during the telephone diet history. Despite the limitations associated with using standard measuring cups and spoons as a main portion size estimation aid, such practice would have enabled consistency in portion determination across both dietary assessment methods. Furthermore, portion size prompts such as a female fist size for a medium potato provided to participants on the food record booklet were used during the diet

history. Although such prompt lacks standardisation as fist size varies across women, use of these prompts should not have affected the relative validity results as the prompts were used in both methods.

The relative validity analysis of the diet history and food record must be interpreted in the context of dietary data obtained from women from diverse cultural backgrounds all with prior GDM. Although no sub analysis was conducted according to ethnicity, a highly multicultural sample may have influenced the agreement between the telephone diet history and the estimated food record in several ways. Firstly although individuals with insufficient English language skills were excluded, such judgement was arbitrarily decided during recruitment. Participants were asked if they were able to complete a food record. They may have believed their English skills to be adequate but many completed food records lacked sufficient detail.

Brief food records may have led to a greater amount of assumption work during data entry compared with greater detailed description elicited during the diet history. Although food assumptions made were applied identically whether required in a diet history or food record, if assumptions occurred more often in the food record its effect on the direction of bias between the two methods is unknown. Any future work in relative validity of dietary assessment in highly multicultural samples should consider the amount of assumptions required across methods, whether due to omission of detail by the participant or due limited food choices inherent in the food composition database at the point of data entry.

A highly multicultural sample may have also influenced results due to potential miscommunication. It has previously been noted when researcher and respondent do not share similar culture, culture-specific miscommunication can easily go undetected by professionals embedded in their own ethnocultural and disciplinary ways of knowing, thinking and communicating.[130] It is possible this issue may present itself during a diet history interview.

A prior study identified how the acceptability of the use of a telephone interview may differ across culture. The study examined the relevance of a Canadian fruit and vegetable FFQ in cultural subgroups living in Canada and found the Asian and Portuguese migrants surveyed believed a face to face survey was more effective than a telephone interview. The older Cantonese- and Mandarin-speaking participants in this study indicated that the telephone is not a common mode of communication and they would be unlikely to respond to a telephone survey. [130] Thus telephone use in dietary assessment should consider the cultural context and appropriateness prior to its implementation which was not investigated prior to the HeLP GDM study.

In summary the telephone diet history and estimated food record resulted in comparable group estimates of intake for energy, total fat and SFA and fibre at baseline with no significant change in bias at six months. Furthermore the dietary change demonstrated in Chapter three can be supported by the current relative validity analysis.

## CHAPTER FIVE - CONCLUSIONS AND RECOMMENDATIONS

### Conclusion

Women with a history of GDM are a population group with an increased risk of developing T2DM. While it is known that changes in diet and lifestyle can delay or prevent the onset of T2DM in at risk individuals, research in behaviour change associated with T2DM risk reduction specifically in women with prior GDM, is lacking. Additionally current clinical practice provides very little in the way of strategies to reduce risk of T2DM in these women. Thus the HeLP GDM study a six month behavioural intervention targeting dietary and PA change was piloted in a multicultural diverse sample of women with prior GDM.

The central hypothesis of this thesis was that dietary change was achieved in the HeLP GDM intervention. Results as detailed in chapter three prove this hypothesis to be correct, with significant reductions in intake of fat and energy identified and simultaneous maintenance of adequate dietary fibre. Analysis of diet history data demonstrated the women in the intervention were more likely than the controls to achieve the ADA recommendations to reduce intake of total fat to  $\leq 30\%$  and to achieve an intake of fibre of 15 grams per 4200 kJ. Furthermore the concurrent self reported weight lost within the intervention group strengthens the reduction in energy intake seen.

Diet histories in clinical practice are an essential tool for identifying aspects of the diet which require change hence its inclusion in the HeLP GDM study. Given the intervention context of the pilot, food records were also obtained. Previous literature has demonstrated no difference in energy underreporting between the diet history and food record. In this thesis comparable estimates of energy and macronutrient intakes obtained by the diet history and food record was hypothesised.

It is important to note that previous work in the area of dietary methodology has advocated the context of the trial including the purpose of the trial and the individual characteristics of the participants enrolled, can affect the findings of a validity study.[84] In the context of dietary information collected during the HeLP GDM study, the dietary assessments were

modified from their traditional form to suit the needs of the target group. The relative validity analysis in chapter four demonstrated the two dietary assessment methods provided comparable group estimates in the intake of fat, fibre and energy.

Extent of intra-individual variation among study participants was high indicating low precision for the two assessments to agree. However, the wide variation identified was not notably different to previous literature reporting the relative validity of a face to face diet history with estimated or weighed food records. The finding of low precision between the two methods is of less importance to the intervention context given the methods demonstrated they provided comparable mean estimates both at baseline and at six months.

Similar trends in dietary change at six months were evident across both methods of dietary assessment however some differences were apparent. Diet history data across both statistical analyses demonstrated consistent reductions in fat and energy. However, this was not the case with the food records. The differences seen, however, may have been caused by the statistical treatment of data, rather than a discrepancy between dietary assessments of actual dietary change.

With the primary analysis, missing values at six months led to an assumption of no change in the participant. The food records completers analysis which included only complete six month data, identified the same trends the diet history analyses. However, when the primary analysis of food records assumed no change at six months for one third of the sample, change in several variables failed to reach significance, with only a significant reduction in SFA. Overall the results of this thesis show promise for the inclusion of dietary change outcomes in subsequent trials that aim to achieve reduction in incidence of T2DM in women with prior GDM.

### **Recommendations for the future**

Dietary change in the HeLP GDM study context was limited to women at minimum thinking of changing dietary of PA behaviour. Future research into the efficacy of dietary intervention

in women with prior GDM should include women in the precontemplation stage of behaviour change, given the increased risk of developing T2DM remains whether or not they are thinking of changing. Recruitment to dietary intervention studies remains a problem in this target group and future research may simply need to use collaboration across multiple sites to achieve a satisfactory sample size and adequate power to address the research question.

Prior research has indicated approaching women during the GDM pregnancy rather than at postpartum may result in sustained dietary changes. Recruitment during a GDM pregnancy rather than at postpartum allows for a relationship to develop between the woman, health care provider and researcher, while the woman is still involved in the healthcare system. An established relationship between these parties may lead to greater numbers of women recruited and minimise study drop outs.

The HeLP GDM study included the use of five mobile text messages. Although no formal data was collected on the women's feedback regarding study design, future studies in this area may benefit from increasing the focus on mobile text messaging as a means of communication. Short Message Service otherwise known as SMS, is widespread, convenient, rapid and a less invasive form of communication for individuals who may not feel comfortable beginning dialogue regarding behaviour change. SMS used as periodic prompts can be tailored to the individual's stage of change and personal circumstances. This way all women not just those motivated to change can be targeted.

With the increasing use of technology smartphones now have available applications that allow the user to enter dietary intake at the point of consumption. Given that women with young children are generally time poor and are common users of smartphone technology, future studies should consider validating the use of such applications to assist with dietary data collection. Additionally future relative validity analysis conducted in highly multicultural samples should investigate the impact and influence of chosen food assumptions at the point of data entry and additionally how the frequency may vary across the dietary assessment methods in question.

## REFERENCES

1. Kim, C., K.M. Newton, and R.H. Knopp, *Gestational diabetes and the incidence of type 2 diabetes: a systematic review*. Diabetes Care, 2002. **25**(10): p. 1862-8.
2. Hosseinpour-Niazi, S., et al., *Inverse association between fruit, legume, and cereal fiber and the risk of metabolic syndrome: Tehran Lipid and Glucose Study*. Diabetes Res Clin Pract, 2011. **94**(2): p. 276-83.
3. Priebe, M.G., et al., *Whole grain foods for the prevention of type 2 diabetes mellitus*. Cochrane Database of Systematic Reviews, 2008(1): p. CD006061.
4. Riserus, U., W.C. Willett, and F.B. Hu, *Dietary fats and prevention of type 2 diabetes*. Progress in Lipid Research, 2009. **48**(1): p. 44-51.
5. Vessby, B., et al., *Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study*. Diabetologia, 2001. **44**(3): p. 312-9.
6. Knowler, W.C., et al., *Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin*. New England Journal of Medicine, 2002. **346**(6): p. 393-403.
7. Pan, X.R., et al., *Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study*. Diabetes Care, 1997. **20**(4): p. 537-44.
8. Ramachandran, A., et al., *The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1)*. Diabetologia, 2006. **49**(2): p. 289-97.
9. Tuomilehto, J., et al., *Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance*. New England Journal of Medicine, 2001. **344**(18): p. 1343-50.
10. Kieffer, E.C., B. Sinco, and C. Kim, *Health behaviors among women of reproductive age with and without a history of gestational diabetes mellitus*. Diabetes Care, 2006. **29**(8): p. 1788-93.
11. Moses, R.G., J.L. Shand, and L.C. Tapsell, *The recurrence of gestational diabetes: could dietary differences in fat intake be an explanation?* Diabetes Care, 1997. **20**(11): p. 1647-50.
12. Swan, W., G. Kilmartin, and S.T. Liaw, *Assessment of readiness to prevent type 2 diabetes in a population of rural women with a history of gestational diabetes*. Rural & Remote Health, 2007. **7**(4): p. 802.
13. Zehle, K., et al., *Psychosocial factors related to diet among women with recent gestational diabetes: opportunities for intervention*. Diabetes Educator, 2008. **34**(5): p. 807-14.
14. Cheung, N.W., et al., *A group-based healthy lifestyle program for women with previous gestational diabetes*. Diabetes Research & Clinical Practice, 2007. **77**(2): p. 333-4.
15. Ostbye, T., et al., *Active Mothers Postpartum: a randomized controlled weight-loss intervention trial*. Am J Prev Med, 2009. **37**(3): p. 173-80.
16. O'Toole, M.L., M.A. Sawicki, and R. Artal, *Structured diet and physical activity prevent postpartum weight retention*. J Womens Health (Larchmt), 2003. **12**(10): p. 991-8.
17. Wein, P., et al., *A trial of simple versus intensified dietary modification for prevention of progression to diabetes mellitus in women with impaired glucose tolerance*. Australian & New Zealand Journal of Obstetrics & Gynaecology, 1999. **39**(2): p. 162-6.
18. Reinhardt, J.A., et al., *Implementing lifestyle change through phone-based motivational interviewing in rural-based women with previous gestational diabetes mellitus*. Health Promotion Journal of Australia, 2012. **23**(1): p. 5-9.
19. Hu, G., et al., *Tianjin Gestational Diabetes Mellitus Prevention Program: study design, methods, and 1-year interim report on the feasibility of lifestyle intervention program*. Diabetes Research & Clinical Practice, 2012. **98**(3): p. 508-17.



20. Ferrara, A., et al., *A pregnancy and postpartum lifestyle intervention in women with gestational diabetes mellitus reduces diabetes risk factors: a feasibility randomized control trial*. Diabetes Care, 2011. **34**(7): p. 1519-25.
21. American Diabetes Association, *Diagnosis and classification of diabetes mellitus*. [Erratum appears in Diabetes Care. 2010 Apr;33(4):e57]. Diabetes Care, 2010. **33** Suppl 1: p. S62-9.
22. Kahn, C.R., *Insulin resistance, insulin insensitivity, and insulin unresponsiveness: a necessary distinction*. Metabolism: Clinical & Experimental, 1978. **27**(12 Suppl 2): p. 1893-902.
23. Wild, S., et al., *Global prevalence of diabetes: estimates for the year 2000 and projections for 2030*. Diabetes Care, 2004. **27**(5): p. 1047-53.
24. Davis, W.A., et al., *The obesity-driven rising costs of type 2 diabetes in Australia: projections from the Fremantle Diabetes Study*. Internal Medicine Journal, 2006. **36**(3): p. 155-61.
25. Australian Bureau of Statistics, *Australian Health Survey:biomedical results for chronic diseases 2011-12* 2011-2013, Australian Bureau of Statistics.
26. Australian Institute of Health and Welfare, *Diabetes prevalence in Australia: an assessment of national data sources*, in *Diabetes series*. 2009, AIHW: Canberra.
27. Dunstan, D.W., et al., *The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study*. Diabetes Care, 2002. **25**(5): p. 829-34.
28. Kuhl, C., *Etiology and pathogenesis of gestational diabetes*. Diabetes Care, 1998. **21** Suppl 2: p. B19-26.
29. Templeton, M. and I. Pieris-Caldwell, *Gestational diabetes mellitus in Australia 2005-06*. 2008, Australian Institute of Health and Welfare: Canberra.
30. Cheung, N.W. and K. Byth, *Population health significance of gestational diabetes*. Diabetes Care, 2003. **26**(7): p. 2005-9.
31. Anna, V., et al., *Sociodemographic correlates of the increasing trend in prevalence of gestational diabetes mellitus in a large population of women between 1995 and 2005*. Diabetes Care, 2008. **31**(12): p. 2288-93.
32. Beischer, N.A., et al., *Incidence and severity of gestational diabetes mellitus according to country of birth in women living in Australia*. Diabetes, 1991. **40** Suppl 2: p. 35-8.
33. World Health Organisation, *Obesity: preventing and managing the global epidemic*, in *WHO Technical Report Series*. 2000. p. 9.
34. Lee, A.J., et al., *Gestational diabetes mellitus: clinical predictors and long-term risk of developing type 2 diabetes: a retrospective cohort study using survival analysis*. Diabetes Care, 2007. **30**(4): p. 878-83.
35. Akinkuolie, A.O., et al., *Omega-3 polyunsaturated fatty acid and insulin sensitivity: a meta-analysis of randomized controlled trials*. Clinical Nutrition, 2011. **30**(6): p. 702-7.
36. Galgani, J.E., et al., *Effect of the dietary fat quality on insulin sensitivity*. British Journal of Nutrition. **100**(3): p. 471-9.
37. World Health Organisation, *Diet, nutrition and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation*, in *WHO Technical Report Series*. 2003, WHO: Geneva, Switzerland.
38. American Diabetes Association, *Nutrition Recommendations and Interventions for Diabetes: A position statement of the American Diabetes Association*. Diabetes Care, 2008. **31**(Supplement 1): p. S61-S78.
39. Thanopoulou, A.C., et al., *Dietary fat intake as risk factor for the development of diabetes: multinational, multicenter study of the Mediterranean Group for the Study of Diabetes (MGSD)*. Diabetes Care, 2003. **26**(2): p. 302-7.
40. Borkman, M., et al., *The relation between insulin sensitivity and the fatty-acid composition of skeletal-muscle phospholipids*. New England Journal of Medicine, 1993. **328**(4): p. 238-44.
41. Manco, M., et al., *Insulin resistance directly correlates with increased saturated fatty acids in skeletal muscle triglycerides*. Metabolism: Clinical & Experimental, 2000. **49**(2): p. 220-4.

42. Alhazmi, A., et al., *Macronutrient Intakes and Development of Type 2 Diabetes: A Systematic Review and Meta-Analysis of Cohort Studies*. Journal of the American College of Nutrition, 2012. **31**(4): p. 243-258.
43. Marshall, J.A. and D.H. Bessesen, *Dietary Fat and the Development of Type 2 Diabetes*. Diabetes Care, 2002. **25**(3): p. 620-622.
44. Vessby, B., et al., *The risk to develop NIDDM is related to the fatty acid composition of the serum cholesterol esters*. Diabetes, 1994. **43**(11): p. 1353-7.
45. Hodge, A.M., et al., *Plasma phospholipid and dietary fatty acids as predictors of type 2 diabetes: interpreting the role of linoleic acid*. American Journal of Clinical Nutrition, 2007. **86**(1): p. 189-97.
46. Laaksonen, D.E., et al., *Serum fatty acid composition predicts development of impaired fasting glycaemia and diabetes in middle-aged men*. Diabetic Medicine, 2002. **19**(6): p. 456-64.
47. Wang, L., et al., *Plasma fatty acid composition and incidence of diabetes in middle-aged adults: the Atherosclerosis Risk in Communities (ARIC) Study*. American Journal of Clinical Nutrition, 2003. **78**(1): p. 91-8.
48. Lamping, K.G., et al., *Modification of high saturated fat diet with n-3 polyunsaturated fat improves glucose intolerance and vascular dysfunction*. Diabetes, Obesity & Metabolism, 2013. **15**(2): p. 144-52.
49. Tishinsky, J.M., et al., *Fish oil prevents high-saturated fat diet-induced impairments in adiponectin and insulin response in rodent soleus muscle*. American Journal of Physiology - Regulatory Integrative & Comparative Physiology, 2012. **302**(5): p. R598-605.
50. Wein, S., et al., *Medium-chain fatty acids ameliorate insulin resistance caused by high-fat diets in rats*. Diabetes/Metabolism Research Reviews, 2009. **25**(2): p. 185-94.
51. Hamman, R.F., et al., *Effect of weight loss with lifestyle intervention on risk of diabetes*. Diabetes Care, 2006. **29**(9): p. 2102-7.
52. Salmeron, J., et al., *Dietary fat intake and risk of type 2 diabetes in women*. Am J Clin Nutr, 2001. **73**(6): p. 1019-26.
53. van Dam, R.M., et al., *Dietary fat and meat intake in relation to risk of type 2 diabetes in men*. Diabetes Care, 2002. **25**(3): p. 417-24.
54. de Lorgeril, M. and P. Salen, *New insights into the health effects of dietary saturated and omega-6 and omega-3 polyunsaturated fatty acids*. BMC Med, 2012. **10**: p. 50.
55. Wertz, P.W., *Essential fatty acids and dietary stress*. Toxicol Ind Health, 2009. **25**(4-5): p. 279-83.
56. Simopoulos, A.P., *The importance of the ratio of omega-6/omega-3 essential fatty acids*. Biomedicine & Pharmacotherapy, 2002. **56**(8): p. 365-79.
57. Giacco, R., et al., *Fish oil, insulin sensitivity, insulin secretion and glucose tolerance in healthy people: is there any effect of fish oil supplementation in relation to the type of background diet and habitual dietary intake of n-6 and n-3 fatty acids?* Nutrition Metabolism & Cardiovascular Diseases, 2007. **17**(8): p. 572-80.
58. Martinez-Gonzalez, M.A., et al., *Adherence to Mediterranean diet and risk of developing diabetes: prospective cohort study*. Bmj, 2008. **336**(7657): p. 1348-51.
59. Mozaffarian, D., et al., *Incidence of new-onset diabetes and impaired fasting glucose in patients with recent myocardial infarction and the effect of clinical and lifestyle risk factors*. Lancet, 2007. **370**(9588): p. 667-75.
60. Salas-Salvado, J., et al., *Reduction in the incidence of type 2 diabetes with the Mediterranean diet: results of the PREDIMED-Reus nutrition intervention randomized trial*. Diabetes Care, 2011. **34**(1): p. 14-9.
61. Gillingham, L.G., S. Harris-Janz, and P.J.H. Jones, *Dietary monounsaturated fatty acids are protective against metabolic syndrome and cardiovascular disease risk factors*. Lipids, 2011. **46**(3): p. 209-28.

62. de Munter, J.S.L., et al., *Whole Grain, Bran, and Germ Intake and Risk of Type 2 Diabetes: A Prospective Cohort Study and Systematic Review*. PLoS Med, 2007. **4**(8): p. e261.
63. Weickert, M.O., et al., *Effects of supplemented isoenergetic diets differing in cereal fiber and protein content on insulin sensitivity in overweight humans*. Am J Clin Nutr, 2011. **94**(2): p. 459-71.
64. Johnston, K.L., et al., *Resistant starch improves insulin sensitivity in metabolic syndrome*. Diabetic Medicine, 2010. **27**(4): p. 391-7.
65. Tighe, P., et al., *Effect of increased consumption of whole-grain foods on blood pressure and other cardiovascular risk markers in healthy middle-aged persons: a randomized controlled trial*. Am J Clin Nutr, 2010. **92**(4): p. 733-40.
66. Pan, A., et al., *Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis*. American Journal of Clinical Nutrition, 2011. **94**(4): p. 1088-96.
67. Pan, A., et al., *Changes in red meat consumption and subsequent risk of type 2 diabetes mellitus: three cohorts of US men and women*. JAMA Internal Medicine, 2013. **173**(14): p. 1328-35.
68. Tong, X., et al., *Dairy consumption and risk of type 2 diabetes mellitus: a meta-analysis of cohort studies*. Eur J Clin Nutr, 2011. **65**(9): p. 1027-31.
69. Ratner, R.E., et al., *Prevention of diabetes in women with a history of gestational diabetes: effects of metformin and lifestyle interventions*. J Clin Endocrinol Metab, 2008. **93**(12): p. 4774-9.
70. Barnard, J., *Evaluation of clinical indicators and social context for developing lifestyle modification programs after gestational diabetes mellitus: a case study for a cohort of women in the Illawarra region, Australia*. 2005, University of Wollongong: Wollongong.
71. Craigie, A.M., et al., *Supporting postpartum weight loss in women living in deprived communities: design implications for a randomised control trial*. Eur J Clin Nutr, 2011. **65**(8): p. 952-8.
72. McIntyre, H.D., et al., *Pilot study of an individualised early postpartum intervention to increase physical activity in women with previous gestational diabetes*. Int J Endocrinol, 2012. **2012**: p. 892019.
73. Stendell-Hollis, N.R., et al., *Recruitment of lactating women into a randomized dietary intervention: successful strategies and factors promoting enrollment and retention*. Contemp Clin Trials, 2011. **32**(4): p. 505-11.
74. Kim, C., et al., *A web-based pedometer programme in women with a recent history of gestational diabetes*. Diabet Med, 2012. **29**(2): p. 278-83.
75. Cheung, N.W., et al., *A group-based healthy lifestyle program for women with previous gestational diabetes*. Diabetes Research & Clinical Practice. **77**(2): p. 333-4.
76. Mertz, W., *Food intake measurements: is there a "gold standard"?* J Am Diet Assoc, 1992. **92**(12): p. 1463-5.
77. Kubena, K.S., *Accuracy in dietary assessment: on the road to good science*. J Am Diet Assoc, 2000. **100**(7): p. 775-6.
78. Lee-Han, H., V. McGuire, and N.F. Boyd, *A review of the methods used by studies of dietary measurement*. Journal of Clinical Epidemiology, 1989. **42**(3): p. 269-79.
79. Livingstone, M.B.E. and A.E. Black, *Markers of the validity of reported energy intake*. Journal of Nutrition, 2003. **133** Suppl 3: p. 895S-920S.
80. Tapsell, L.C., K. Pettengell, and S.L. Denmeade, *Assessment of a narrative approach to the diet history*. Public Health Nutr, 1999. **2**(1): p. 61-7.
81. Martin, G.S., et al., *Relative validity of a diet history interview in an intervention trial manipulating dietary fat in the management of Type II diabetes mellitus* ☆. Prev Med, 2003. **36**(4): p. 420-428.

82. Poslusna, K., et al., *Misreporting of energy and micronutrient intake estimated by food records and 24 hour recalls, control and adjustment methods in practice*. British Journal of Nutrition, 2009. **101 Suppl 2**: p. S73-85.
83. Hoidrup, S., et al., *Assessment of habitual energy and macronutrient intake in adults: comparison of a seven day food record with a dietary history interview*. Eur J Clin Nutr, 2002. **56**(2): p. 105-13.
84. Martin, G.S., *The interviewer administered, open-ended diet history method for assessing usual dietary intakes in clinical research: relative and criterion validation studies*. 2004, University of Wollongong: Wollongong.
85. Coulston, A.M., et al., *Nutrition in the Prevention and Treatment of Disease*. 2012, San Diego: Academic Press.
86. Johansson, L., et al., *Under- and overreporting of energy intake related to weight status and lifestyle in a nationwide sample*. American Journal of Clinical Nutrition, 1998. **68**(2): p. 266-74.
87. Trabulsi, J. and D.A. Schoeller, *Evaluation of dietary assessment instruments against doubly labeled water, a biomarker of habitual energy intake*. American Journal of Physiology - Endocrinology & Metabolism, 2001. **281**(5): p. E891-9.
88. Swierk, M., et al., *Validation of an Australian electronic food frequency questionnaire to measure polyunsaturated fatty acid intake*. Nutrition, 2011. **27**(6): p. 641-6.
89. Chinnock, A., *Validation of an estimated food record*. Public Health Nutr, 2006. **9**(7): p. 934-41.
90. Larkin, F.A., H.L. Metzner, and K.E. Guire, *Comparison of three consecutive-day and three random-day records of dietary intake*. J Am Diet Assoc, 1991. **91**(12): p. 1538-42.
91. McKeown, N.M., et al., *Use of biological markers to validate self-reported dietary intake in a random sample of the European Prospective Investigation into Cancer United Kingdom Norfolk cohort*. American Journal of Clinical Nutrition, 2001. **74**(2): p. 188-96.
92. Edington, J., et al., *Assessment of nutritional intake using dietary records with estimated weights*. Journal of Human Nutrition and Dietetics, 1989. **2**(6): p. 407-414.
93. Bingham, S.A., et al., *Comparison of dietary assessment methods in nutritional epidemiology: weighed records v. 24 h recalls, food-frequency questionnaires and estimated-diet records*. British Journal of Nutrition, 1994. **72**(4): p. 619-43.
94. Hartwell, D.L., *Comparison of a self-administered quantitative food amount frequency questionnaire with 4-day estimated food records*. International Journal of Food Sciences and Nutrition, 2001. **52**(2): p. 151-159.
95. Black, A.E., A.A. Welch, and S.A. Bingham, *Validation of dietary intakes measured by diet history against 24 h urinary nitrogen excretion and energy expenditure measured by the doubly-labelled water method in middle-aged women*. British Journal of Nutrition, 2000. **83**(4): p. 341-54.
96. Martin, G.S., et al., *Relative bias in diet history measurements: a quality control technique for dietary intervention trials*. Public Health Nutr, 2002. **5**(4): p. 537-45.
97. Chinnock, A., *Validation of a diet history questionnaire for use with Costa Rican adults*. Public Health Nutr, 2008. **11**(1): p. 65-75.
98. Fox, T.A., J. Heimendinger, and G. Block, *Telephone surveys as a method for obtaining dietary information: a review*. J Am Diet Assoc, 1992. **92**(6): p. 729-32.
99. Probst, Y.C., *An evaluation of automated dietary assessment: a case study into the development, implementation and evaluation of computer assisted survey technology as an adjunct to professional dietary consultation*. 2006, University of Wollongong: Wollongong.
100. Tran, K.M., et al., *In-person vs Telephone-administered Multiple-pass 24-hour Recalls in Women*. J Am Diet Assoc, 2000. **100**(7): p. 777-783.

101. Yanek, L.R., et al., *Comparison of the Effectiveness of a Telephone 24-hour Dietary Recall Method vs an In-person Method among Urban African-American Women*. J Am Diet Assoc, 2000. **100**(10): p. 1172-1177.
102. Godwin, S.L., E.t. Chambers, and L. Cleveland, *Accuracy of reporting dietary intake using various portion-size aids in-person and via telephone*. J Am Diet Assoc. **104**(4): p. 585-94.
103. World Health Organisation Expert Consultation, *Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies*. Lancet, 2004. **363**(9403): p. 157-63.
104. Robinson, A.H., et al., *Validating stage of change measures for physical activity and dietary behaviors for overweight women*. International Journal of Obesity, 2008. **32**(7): p. 1137-44.
105. Marcus, B.H., et al., *Self-efficacy and the stages of exercise behavior change*. Research Quarterly for Exercise & Sport, 1992. **63**(1): p. 60-6.
106. Greene, G.W., et al., *Dietary Applications of the Stages of Change Model*. J Am Diet Assoc, 1999. **99**(6): p. 673-678.
107. van der Ploeg, H.P., Smith B, Cheung, N.W, Razee, H, Blignault, I, Bauman, A, *Understanding and Measuring Health Behaviours and Related Psychosocial Factors among Women with a History of Gestational Diabetes Mellitus*. 2009, NSW Department of Health: Sydney.
108. Cheung, N.W., et al., *A pilot structured behavioural intervention trial to increase physical activity among women with recent gestational diabetes*. Diabetes Res Clin Pract, 2011. **92**(1): p. e27-9.
109. Fry, J.P. and R.A. Neff, *Periodic prompts and reminders in health promotion and health behavior interventions: systematic review*. J Med Internet Res, 2009. **11**(2): p. e16.
110. Rosal, M.C., et al., *Facilitating dietary change: the patient-centered counseling model*. J Am Diet Assoc, 2001. **101**(3): p. 332-41.
111. Suchanek, P., et al., *Which index best correlates with body fat mass: BAI, BMI, waist or WHR?* Neuroendocrinology Letters, 2012. **33 Suppl 2**: p. 78-82.
112. CalorieKing. 2011 1 April 2011 to December 2011; Available from: <http://www.calorieking.com.au/>.
113. Colagiuri, S., et al., *The Sydney Diabetes Prevention Program: A community-based translational study*. BMC Public Health, 2010. **10**(1): p. 328.
114. Douketis, J.D., et al., *Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice*. International Journal of Obesity, 2005. **29**(10): p. 1153-67.
115. Klein, S., et al., *Weight management through lifestyle modification for the prevention and management of type 2 diabetes: rationale and strategies: a statement of the American Diabetes Association, the North American Association for the Study of Obesity, and the American Society for Clinical Nutrition*. Diabetes Care, 2004. **27**(8): p. 2067-73.
116. Penn, L., et al., *Importance of Weight Loss Maintenance and Risk Prediction in the Prevention of Type 2 Diabetes: Analysis of European Diabetes Prevention Study RCT*. PLoS ONE, 2013. **8**(2): p. e57143.
117. Norris, S.L., et al., *Long-term non-pharmacological weight loss interventions for adults with prediabetes*. Cochrane Database of Systematic Reviews, 2005(2): p. CD005270.
118. Hodge, A., et al., *The Anti Cancer Council of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in young to middle-aged women in a study of iron supplementation*. [Erratum appears in Aust N Z J Public Health. 2003 Aug;27(4):468]. Australian & New Zealand Journal of Public Health, 2000. **24**(6): p. 576-83.
119. Lan-Pidhainy, X., E.A. Nohr, and K.M. Rasmussen, *Comparison of gestational weight gain-related pregnancy outcomes in American primiparous and multiparous women*. American Journal of Clinical Nutrition, 2013. **97**(5): p. 1100-6.
120. Lv, N. and J.L. Brown, *Chinese American Family Food Systems: Impact of Western Influences*. Journal of Nutrition Education and Behavior, 2010. **42**(2): p. 106-114.

121. Zhai, F.M.D., et al., *Lifespan nutrition and changing socio-economic conditions in China*. Asia Pacific Journal of Clinical Nutrition, 2007. **16**: p. 374-82.
122. Sibbald, B. and M. Roland, *Understanding controlled trials: Why are randomised controlled trials important?* Bmj, 1998. **316**(7126): p. 201.
123. Abouzeid, M., et al., *Type 2 diabetes prevalence varies by socio-economic status within and between migrant groups: analysis and implications for Australia*. BMC Public Health, 2013. **13**: p. 252.
124. Organisation, W.H., *Infant and Young Child Feeding: Model Chapter for textbooks for medical students and allied health professionals*. 2009, WHO Press: Switzerland.
125. Miller, T.M., et al., *Effects of social approval bias on self-reported fruit and vegetable consumption: a randomized controlled trial*. Nutr J, 2008. **7**: p. 18.
126. Razee, H., et al., *Beliefs, barriers, social support, and environmental influences related to diabetes risk behaviours among women with a history of gestational diabetes*. Health Promotion Journal of Australia, 2010. **21**(2): p. 130-7.
127. Bland, J.M.D.G., *Comparing methods of measurement*. Lancet, 1995. **346**(8982): p. 1085.
128. Willet, W., *Nutritional Epidemiology: Second Edition*. Vol. 30. 1998: Oxford University Press.
129. Tapsell, L.C., V. Brenningner, and J. Barnard, *Applying conversation analysis to foster accurate reporting in the diet history interview*. J Am Diet Assoc, 2000. **100**(7): p. 818-24.
130. Paisley, J., M. Greenberg, and J. Haines, *Cultural relevance of a fruit and vegetable food frequency questionnaire*. Can J Diet Pract Res, 2005. **66**(4): p. 231-6.

## APPENDIX A: PROFORMA FOR PHYSICAL ACTIVITY COUNSELLING IN THE HeLP GDM STUDY

NOTE: [instructions to counselor in italics]

*General suggestions for counseling: be sympathetic and understanding, don't judge the participant, try to look for solutions in a conversational way (don't superimpose them), aim for realistic plans and goals.*

### **First face-to-face meeting (2wks after baseline)**

The program will consist of 2 sessions with me face to face and I will be calling you 5 times of the next 6 months.

What we will discuss today is your reasons for wanting to join the program, discuss the areas you need to work on to achieving a more healthier lifestyle and if you are willing set some goals towards achieving either a more active lifestyle and or eating a more healthier diet.

We have developed this journal for you to use during our counseling. The information and worksheets provided are intended to help you through the program to achieve your goals. I will be explaining the sections of the journal as we go along.

*Go through reasons for entering program in the journal*

I would like to talk to you now about the role that physical activity can play in improving your health and reducing your future risk of type 2 diabetes and the strategies you could use to increase your physical activity participation. In this session I would like to:

explore the benefits that undertaking more physical activity could have for you

consider your recent levels of physical activity

if you are willing, set some goals for participating in more physical activity

*Briefly discuss the 'physical activity recommendation' section in the journal.*

When we met earlier, you completed a question (PA stage of change) on your physical activity, which suggested that currently you are (select one):

-not regularly physically active, but you are thinking about becoming more physically active in the next 6 months.

-physically active, but not regularly.

-regularly physically active, but you only started being so in the last 6 months.

-regularly physically active, and you have been so for longer than 6 months.

NOTE: regularly physically active = being moderately physically active for at least 30 minutes per day on at least five days per week. (explain this to the women if needed)

Is that about right?

Now, let's explore this a little bit further in your journal.



Complete the 'my habits & goals physical activity' section of the journal together. Have a quick discussion on the women's main barriers and possible solutions (show her the 'barrier solution' section in the journal, just so she is aware this is available, don't discuss this in detail). Finish with setting some short term physical activity goals until your next appointment with her (~2 weeks by telephone).

I think if you can start to put your plan into action you will feel a difference and if you keep it up it will bring you health benefits as well. What you set out to do is achievable, and with the discussions we have had today I am optimistic that you will be able to overcome any barriers you may face in trying to become more active.

As part of this lifestyle program we are offering some short telephone follow-up contacts over the next 6 months – five in total with the next one in about two weeks time to see how you are going in making a start with your physical activity. There will also be one more face to face meeting in about a month.

Would it be alright for me to call you then and catch up with how you are going, and talk about any questions or challenges you are facing?

Arrange time for follow-up contact.

Do you have any questions?

Thanks for your time and good luck.

### Telephone check up 1 (4wks after baseline)

[instructions to counselor in italics]

I am calling to follow-up from our discussion at the hospital about physical activity.

How are you going in relation to your physical activity plan?

*If necessary: Further prompts could be:*

What was it like trying to increase your physical activity these past 2 weeks?

Were you able to incorporate what we discussed last time?

If not meeting goals, acknowledge the challenges in starting to be more active, particularly for busy mums

What sort of barriers are you facing?

What have you tried to overcome that difficulty (or those difficulties)?

*Brainstorm with the participant on possible strategies for overcoming barriers, avoiding being too didactic (if necessary see barriers section in the journal)*

Would it be helpful to set some new goals now? or would you still like to keep working with the ones you have got?

If necessary set some new goals, aiming possibly for shorter bouts of activity, or less frequency in sessions

*Offer encouragement to keep going, and reinforce belief that she can make progress towards establishing a good physical activity routine. Remind her that face to face meeting will be in 2 weeks time to discuss how she is going and to offer some support.*

Arrange time for follow-up contact, thanks her for her time and make wish her good luck.

If meeting goals, congratulate the participant and tell them what a great job they have done

How did it feel to be physically active and to achieve your goal?

Do you find what you are doing is practical?

Do you feel you can keep going with this?

Can you anticipate that there will be any interruptions to your routine? If so do you have any ideas about how to deal with these?

*Discuss strategies for accommodating interruptions i.e adjusting frequency and/or type of activity, or accepting these and starting again as soon as possible*

Are you ready to perhaps set some new goals

If so, work on setting of new goals, in terms of duration, frequency and/or type of activity

Offer encouragement to keep going, and reinforce belief that she can continue to make progress towards establishing a good physical activity routine. Remind that face to face meeting will be in 2 weeks time to discuss how she is going and to offer some support.

Arrange time for follow-up contact, thanks her for her time and wish her good luck.

## **Second face-to-face meeting (6wks after baseline)**

[instructions to counselor in italics]

I am calling to follow-up from our discussion at the hospital about physical activity.

How are you going in relation to your physical activity plan?

*If necessary: Further prompts could be:*

What was it like trying to increase your physical activity these past 2 weeks?

Were you able to incorporate what we discussed last time?

If not meeting goals, acknowledge the challenges in starting to be more active, particularly for busy mums

What sort of barriers are you facing?

What have you tried to overcome that difficulty (or those difficulties)?

*Brainstorm with the participant on possible strategies for overcoming barriers, avoiding being too didactic (if necessary see barriers section in the journal)*

If meeting goals, congratulate the participant and tell them what a great job they have done

How did it feel to be physically active and to achieve your goal?

Do you find what you are doing is practical?

Do you feel you can keep going with this?

Can you anticipate that there will be any interruptions to your routine? If so do you have any ideas about how to deal with these?

*Discuss strategies for accommodating interruptions i.e adjusting frequency and/or type of activity, or accepting these and starting again as soon as possible*

In the remainder of this session we will focus in more detail on the following:

talk about social support and physical activity

discuss techniques that you could use to establish a habit of regular physical activity

### **Social support**

A factor that can play a big part in whether women, particularly those who are mothers, are able to find time for physical activity is whether they receive support and assistance from family and friends.

This support can come in the form of helping with child care, housework, giving you verbal encouragement, arranging to participate in physical activity with you, and various other actions.

Doing physical activity with your child(ren), family or friends is not just good for you but can be fun and beneficial for them as well. What kind of physical activities would be fun to do with your child(ren), family or friends?

Overall, do you feel supported by family/friends to participate in physical activity? If yes....What types of support do you find most helpful?

Do you think it would be helpful if you received more assistance from family members or friends, and if so in what ways?

Do you think you could start a discussion with .... about arranging to get the type of support that you need (reiterate the support that participant thinks would be helpful)?

*If woman finds that asking for help to be active is hard or leads to conflict, consider the following suggestion:* A good way to request support from others is to communicate using “I” statements, particularly when talking to your partner or others who might feel that they are being accused of not helping out enough. So instead of saying “I wish you would look after the kids more so I can get some exercise”, you could say “I would like to start doing a bit more exercise, but I am having trouble finding time to do that. Could we talk about finding a time when you could ... look after kids, make lunches etc ... so I could start to do more exercise”. “I” statements involve starting out with how you feel and what you need and then asking the other person to help you address that need. Rather than telling people what they have to do for you, it involves inviting them to talk about how you could address the need you have. Would this be something you could try?

### **Self monitoring**

We are going to give you this pedometer and would like to ask you to keep it and use it as a way of keeping track of the progress you are making. You can use this to firstly find out how many steps you carry out on a usual day, and then to monitor if you are making increases in your daily steps if you put your physical activity plan into action. You can actually adjust your goals to set them in terms of steps rather than minutes (e.g increase steps by 1000 or 2000 per day). The health recommendations advise people to take at least 10,000 steps per day, but again you do not necessarily have to reach this on your first day, it is good to set goals and keep increasing these goals.

Talk her through the pedometer instructions in the journal and explain the use of the pedometer step log.

This pedometer step log can be used to record your steps and keep a record of your progress. You can use this just for the first week or two, or possibly continue to use it, or perhaps just reuse it on certain weeks to see how you are going.

Another good way to monitor your own physical activities is to write them up in a physical activity log. Show her and explain the activity log in the journal. Like the pedometer step log, you could try it for a week or two and then continue to use it if it suits you well. If you prefer just to use the pedometer step log or just the physical activity log, that is fine too.

### **Other physical activity strategies**

Besides self monitoring there are a number of other helpful strategies to become and stay more physically active. Take the woman through the 'other strategies to become and stay active' section of the journal.

*Finish with setting some new short term physical activity goals until your next appointment with her.*



Arrange time for follow-up contact, thanks her for her time and wish her good luck.

**Telephone check up 2-5 (10, 14, 18, 22wks after baseline)**

[instructions to counselor in italics]

I am calling to talk about how you are going with your physical activity and diet.

How are you going in recent weeks with your physical activity plan?

If necessary: Were you able to incorporate what we discussed last time (give examples)?

*Explore type, frequency and duration of physical activity usually undertaken.*

*Consider how this compares with the original plans that were made.*

Have you been using the pedometer, step log, and or activity log? If yes, in what ways and what was helpful and what not? If no, why not?

*If not meeting goals, acknowledge the challenges in starting to be more active, particularly for busy mums*

What has made it difficult to stay on track?

*Address internal attribution for lapse if this arises (e.g. I don't have the willpower) by pointing out that lapses are common, that she has made progress to date, and there are strategies to reinstate her physical activity habit. The worst result is to use a lapse as a reason for not pursuing physical activity goals.*

What have you tried to overcome that difficulty (or those difficulties)?

What else do you think would be helpful to you in keeping to your plan?

*Brainstorm with the participant on possible strategies for overcoming barriers, avoiding being too didactic (if necessary see barriers section in the journal)*

Are you getting support from family and friends with your physical activity? In what ways?

*If not getting support, explore types of support that would be helpful and ways that could be arranged*

Would it be helpful to set some new goals now, or would you still like to keep working with the ones you have got?

*If necessary set some new goals, aiming possibly for shorter bouts of activity, or less frequency in sessions*

*Offer encouragement to keep going, and reinforce belief that she can make progress towards establishing a good physical activity routine. Remind her when the next meeting will be to discuss how she is going and to offer some support.*

*Arrange time for follow-up contact, thanks her for her time and make wish her good luck.*

*If meeting goals, congratulate the participant and tell them what a great job they have done*

Do you find that you are getting into a routine yet with your physical activity?

Is it making any difference to how you feel?

Are you finding that there are any strategies you are using which are helpful in keeping up your activity (e.g. type of activity, timing, support from husband/others, use pedometer, etc)

How confident are you that you can keep going with this?

Are you getting any support from family and friends with your physical activity?

Can you anticipate that there will be any difficulties in keeping up your routine? If so, how confident are you that you can overcome these?

If confident discuss plans about how difficulties will be overcome, if not confident discuss strategies for dealing with barriers.

Are you ready to perhaps set some new goals?

If so, work on setting of new goals, in terms of duration, frequency and/or type of activity

*Offer encouragement to keep going, and reinforce belief that she can continue to make progress towards establishing a good physical activity routine. Remind her when the next meeting will be to discuss how she is going and to offer some support.*

Arrange time for follow-up contact, thanks her for her time and wish her good luck.

## APPENDIX B: PROFORMA FOR DIETARY COUNSELLING IN THE HeLP GDM STUDY

NOTE: *[instructions to counselor in italics]*

### First face-to-face meeting (2 wks after baseline)

*General suggestions for counseling: be sympathetic and understanding, don't judge the participant, try to look for solutions in a conversational way (don't superimpose them), aim for realistic plans and goals.*

Thank you for completing the food record and spending the time to talk to our research dietitian about your eating patterns.

What we will discuss today is your reasons for wanting to join the program, discuss the areas you need to work on to achieving a more healthier lifestyle and if you are willing set some goals towards achieving either a more active lifestyle and or eating a more healthier diet

We have developed this journal for you to use during our counseling. The information and worksheets provided are intended to help you through the program to achieve your goals. I will be explaining the sections of the journal as we go along.

Go through reasons for entering program in the journal

I would like to talk to you now about the importance that healthy eating can play in improving your health and reducing your future risk of type 2 diabetes and the strategies you could use to establish a healthy eating pattern. In this session I would like to:

In regards to diet:

Use the brainstorm map to discuss what problem areas have been identified and if she feels they are a problem by using the following questions below

How do you feel about your current intake of (issues identified)

Are you interested in trying to...(mention areas identified) ?

What do you see as your barriers to eating a healthier diet?

*Questions to ask while exploring barriers:*

What is a typical day when you experience this barriers, is the barrier pretty constant, have you tried anything to get over this, was it helpful, what did you learn?

*If self efficacy is a problem: setting small goals and be positive that they have joined the program*

*Use goals worksheet in journal to set goals*

I think if you can start to put your plan into action you will feel a difference and if you keep it up it will bring you health benefits as well. What you set out to do is achievable, and with the discussions we have had today I am optimistic that you will be able to overcome any barriers you may face in trying to improve your eating habits

As I mentioned previously, as part of this lifestyle program we are offering some short telephone follow-up contacts over the next 6 months – five in total with the next one in about two weeks time to see how you are going in making a start with your goals. There will also be one more face to face meeting in about a month.

Would it be alright for me to call you then and catch up with how you are going, and talk about any questions or challenges you are facing?

*Arrange time for follow-up contact.*

Do you have any questions?

Thanks for your time and good luck.



## Telephone check up 1

*[instructions to counsellor in italics]*

I am calling to follow-up from our discussion at the hospital about eating patterns and or physical activity

How are you going in relation to your food goals?

*If necessary: Further prompts could be:*

*What was it like trying to*

Were you able to incorporate what we discussed last time?

*If not meeting goals, acknowledge the challenges in changing behaviours, particularly for busy mums*

What sort of barriers are you facing?

What have you tried to overcome that difficulty (or those difficulties)?

Brainstorm with the participant on possible strategies for overcoming barriers, avoiding being too didactic

Would it be helpful to set some new goals now, or would you still like to keep working with the ones you have?

If necessary set some new goals

*Offer encouragement to keep going, and reinforce belief that she can make progress towards establishing a good healthy eating routine. Remind that face to face meeting will be in 2 weeks time to discuss how she is going and to offer some support.*

*Arrange time for follow-up contact, thanks her for her time and wish her good luck.*

If meeting goals, congratulate the participant and tell them what a great job they have done

How did it feel to make these food changes and to achieve your goal?

Do you find what you are doing is practical?

Do you feel you can keep going with this?

Can you anticipate that there will be any interruptions to your routine? If so do you have any ideas about how to deal with these?

Discuss strategies for accommodating interruptions or accepting these and starting again as soon as possible

Are you ready to move on to other goals we set at the first face to face counseling session?

*If so, work on next set of goals discussed at the previous face to face counselling session*

*Offer encouragement to keep going, and reinforce belief that she can continue to make*

*progress towards establishing a healthy eating pattern. Remind that face to face meeting will be in 2 weeks time to discuss how she is going and to offer some support.*

*Arrange time for follow-up contact, thanks her for her time and wish her good luck.*

## **Second face-to-face meeting (6wks after baseline)**

How are you going in relation to your food goals?

If necessary: Further prompts could be:

What was it like trying to?

Were you able to incorporate what we discussed last time?

*If not meeting goals, acknowledge the challenges in changing behaviours, particularly for busy mums*

What sort of barriers are you facing?

What have you tried to overcome that difficulty (or difficulties)?

*If social support is an issue: questions to ask (do you think it would be helpful if you received more assistance from family members or friends and if so in what ways*

*talk about using I statements to tell them how you feel/what you need and then inviting them to talk about how they can help address your needs rather than blame. Could this be something they are willing to try?*

*Brainstorm with the participant on possible strategies for overcoming barriers, avoiding being too didactic*

Would it be helpful to set some new goals now, or would you still like to keep working with the ones you have have?

*If necessary set some new goals*

*Offer encouragement to keep going, and reinforce belief that she can make progress towards establishing a good healthy eating routine. Remind that telephone contact meeting will be in 4 weeks time to discuss how she is going and to offer some support.*

*If there are remaining areas to be discussed for diet start discussing additional goals even if patient is not planning to implement them immediately*

*Arrange time for follow-up contact, thanks her for her time and wish her good luck.*

*If meeting goals, congratulate the participant and tell them what a great job they have done*

How did it feel to make these food changes and to achieve your goal?

Do you find what you are doing is practical?\_

Do you feel you can keep going with this

Can you anticipate that there will be any interruptions to your routine? If so do you have any ideas about how to deal with these

*Discuss strategies for accommodating interruptions or accepting these and starting again as soon as possible*

Are you ready to move on to other areas/goals we set at the first face to face counselling session

*If so, work on next remaining areas to be discussed as established at the previous face to face counseling session*

*Offer encouragement to keep going, and reinforce belief that she can continue to make progress towards establishing a healthy eating pattern. Remind that telephone contact will be in 4 weeks time to discuss how she is going and to offer some support. Arrange time for follow-up contact, thanks her for her time and wish her good luck.*

**Telephone check up 2-5 (10, 14, 18, 22wks after baseline)**

*[instructions to counselor in italics]*

I am calling to talk about how you are going with your physical activity and diet.

How are you going in recent weeks with your dietary intake?

*If necessary: Were you able to incorporate what we discussed last time (give examples)?*

*Consider how their current eating patterns this compares with the original plans that were made.*

*If not meeting goals, acknowledge the challenges*

What has made it difficult to stay on track?

*Address internal attribution for lapse if this arises (e.g. I don't have the willpower) by pointing out that lapses are common, that she has made progress to date, and there are strategies to restart towards goals. The worst result is to use a lapse as a reason for not pursuing better eating patterns.*

What have you tried to overcome that difficulty (or those difficulties)?

What else do you think would be helpful to you in keeping to your plan?

*Brainstorm with the participant on possible strategies for overcoming barriers, avoiding being too didactic (if necessary see barriers section in the journal)*

Are you getting support from family and friends? In what ways?

*If not getting support, explore types of support that would be helpful and ways that could be arranged*

Would it be helpful to set some new goals now, or would you still like to keep working with the ones you have got?

*If necessary set some new goals*

*Offer encouragement to keep going, and reinforce belief that she can make progress towards establishing better dietary patterns. Remind her when the next meeting will be to discuss how she is going and to offer some support.*

*Arrange time for follow-up contact, thanks her for her time and wish her good luck.*

*If meeting goals, congratulate the participant and tell them what a great job they have done*

Do you find that you are getting into a routine yet with your dietary intake

Is it making any difference to how you feel?

Are you finding that there are any strategies you are using which are helpful in keeping up your changes

How confident are you that you can keep going with this?

Are you getting any support from family and friends?



Can you anticipate that there will be any difficulties in keeping up your routine? If so, how confident are you that you can overcome these?

*If confident discuss plans about how difficulties will be overcome, if not confident discuss strategies for dealing with barriers.*

Are you ready to perhaps set some new goals?

*If so, work on setting of new goals*

*Offer encouragement to keep going, and reinforce belief that she can continue to make progress towards establishing a good dietary intake. Remind her when the next meeting will be to discuss how she is going and to offer some support.*

*Arrange time for follow-up contact, thanks her for her time and wish her good luck.*

## APPENDIX C: SEVEN DAY TELEPHONE DIET HISTORY PROFORMA

Participant ID:

Diet Hx #:

Checked:



### Diet History The HeLP GDM Study

Are you currently breastfeeding?

Yes

No

Core Food Choices: **Please indicate the *type* of foods you select in these categories**

Food group	Type
<b>Milk (full fat, skim)</b>	
<b>Cheese (full fat, reduced fat)</b>	
<b>Yoghurt (full fat, reduced fat, diet)</b>	
<b>Bread (white, grain)</b>	
<b>Sugar(in drinks)</b>	
<b>Dressings:</b> <b>Sandwich</b> <b>Salad</b>	

### **Part 1: Food Preparation Practices**

#### **Butter/Margarine**

What type do you usually use?

Butter

Dairy blend

Margarine - polyunsaturated, regular

Margarine - polyunsaturated, reduced fat

Margarine - monounsaturated, regular

Margarine – monounsaturated, reduced fat

Other \_\_\_\_\_

**Oil/Fat in cooking**

What type of oil/fat do you use in cooking?

Butter

Olive oil

Dairy blend

Canola oil

Margarine - polyunsaturated, regular

Soybean oil

Margarine - polyunsaturated, reduced fat

Gold'n Canola

Margarine - monounsaturated, regular Other \_\_\_\_\_

**Fat on Meats/Chicken**

How much fat is trimmed from meat before cooking/eating?

None

25%

50%

All

How much of the skin on chicken do you remove before cooking/eating?

None

25%

50%

All

Other, please specify: \_\_\_\_\_

## Part 2: Breakfast

How often do you eat this meal?

[Home](#)

Away

[illegible]

### Part 3: Lunch

How often do you eat this meal?

[Home](#)

Away

[illegible]

## Part 4: Dinner

How often do you eat this meal?

[Home](#)

Away

[illegible]

### Part 5: Snacks / mid-meals (i.e. foods consumed outside main meals)

How often do you eat this meal?

[Home](#)

Away

[illegible]



## Part 6: Food Frequency Checklist

Food Category	Amount	Frequency
Bread/crumpet		
Biscuits		
Crispbreads/crackers		
Cakes/scones/muffins/pastries		
Pancakes		
Beans/legumes		
Fruit		
Fruit juice		
Soft drinks/cordials		
Chocolate/lollies		
Chips		
Alcohol		
Milk		
Yoghurt		
Ice cream		
Cheese		
Dip/cream cheese/cheese spread		
Soy milk		
Soy cheese		
Soy yoghurt		
Eggs/omega eggs		
Salmon/tuna (fresh/canned)		
Sardines/Mackerel		
White fish varieties		
Oysters		
Walnuts		
Pecans		
Other nuts		
Seeds		

## APPENDIX D: THREE DAY FOOD RECORD PROFORMA

**SYDNEY WEST**  
AREA HEALTH SERVICE

**NSW**  **HEALTH**

University of Wollongong



Participant ID:

Date:

Food record #:

Checked:

# 3 Day Food Record: Healthy Living Program after Gestational Diabetes (HeLP GDM)

Please fill in this food record on 2 weekdays and 1 weekend day.  
Select days that will most closely resemble your eating habits.

Please record all the food/drink you have on those days including the amount (cup or spoon measures). The record should include all meals, snacks, nibbling, and beverages including water and any alcohol. Record what you eat and drink as soon as you can to reduce the chance of forgetting.

Please provide as much detail as you can eg whether fish was grilled, fried, baked, type of bread eg wholegrain or rye. For increased accuracy, brand names where possible should be recorded.

If the food is eaten raw then list the raw measurement OR if the food is eaten cooked then list the cooked measurement (see food

record example for the procedure to follow if it is not possible to provide cooked measurements.)

Use the attached pages to help fill the food record as accurately as possible. For each new day start a new page.

Please don't be afraid or embarrassed to write down everything you eat and drink. All the information you provide is very important to us and will be kept confidential.

<b>Beverages</b>	<p>Did you add sugar or milk?</p> <p>Was it regular or decaffeinated?</p>	<b>Sandwiches</b>	<p>Type of bread used?</p> <p>Type of fillings: estimate number of slices of meat, cheese</p> <p>If salad sandwich what type of salad eg beetroot, lettuce etc</p>
<b>Breads</b>	<p>Did you spread on butter or margarine? Which brand and type? If it is a roll is it small, medium or large?</p>	<b>Meats</b>	<p>What preparation method did you use?</p> <p>What type of meat? (eg beef, chicken)</p> <p>Did you remove any visible fat?</p> <p>Is the weight listed for cooked or raw meat?</p> <p>If the meal was for 4 how much did you eat: <math>\frac{1}{4}</math> , <math>\frac{1}{2}</math>, <math>\frac{1}{3}</math> etc</p>
<b>Spreads</b>	<p>Measure usual spread by the teaspoon/tablespoon as it goes on.</p> <p>Jam, honey etc?</p>	<b>Soups</b>	<p>Was it prepared with milk, water, or cream?</p> <p>Was it low-sodium or regular?</p>
<b>Cereal</b>	<p>Did you add milk? Was it full cream, low fat, skim, added calcium?</p> <p>Did you add sugar or fruit?</p> <p>Type, brand amount?</p>	<b>Sugars / Sweets</b>	<p>Was it regular or reduced-calorie? Brands?</p> <p>Don't forget hard candy as well as chocolate.</p>
<b>Dairy</b>	<p>Is your yogurt fruited or plain?</p> <p>What % is your milk?</p>	<b>Vegetables</b>	<p>Was it raw or cooked? Was it fresh, frozen, or canned?</p>

			<p>Was it low-sodium or regular?</p> <p>Did you add any butter/margarine/oil or sauce?</p>
<i>Fats/Oils</i>	<p>Is the butter regular or salt-free?</p> <p>Do you use margarine?</p> <p>Which type of oils?</p> <p>Is it regular or reduced fat?</p>	<i>Take- aways</i>	Estimate quantities in cups
<i>Fruit</i>	<p>Did you add sugar to it?</p> <p>Was it fresh, canned, or frozen?</p> <p>Fruit juice? Brand? Is It 100% fruit juice or fruit drink</p> <p>If fruit is small eg strawberries or apricot list amount eg 3 strawberries</p>		

**Day:**\_\_\_\_\_ **Date:**\_\_\_\_\_

<b>Time</b>	<b>Meal type</b>	<b>Food</b>	<b>Amount</b>	<b>How was it cooked (fried, grilled, boiled)</b>	<b>Where was it made (home, café, restaurant)</b>
6am	Breakfast	Uncle Toby's traditional rolled oats	1.5 cups cooked	Boiled	Home
		Shape skim milk	1 cup		
		Coffee	1.5 cups		
		Shape skim milk	2 tablespoons		
		Raw sugar	2 teaspoons		
10am	Morning Tea	Arnott's Jatz biscuits	6 round biscuits		
		Coon cheddar cheese 30% reduced fat	2 slices		
		Water	600ml		
1pm	Lunch	1 steak and sandwich:			home
		Beef sirloin steak, fat removed	100 gram cooked	Grilled	
		Coon cheddar cheese: 30% reduced fat	1 slice		
		Cos lettuce	1 cup		
		Canned and drained beetroot	2 slices		
		Tomato	Half a small tomato		
		Flora pro-active margarine (regular fat)	2 teaspoons		
4pm	Afternoon Tea	Banana	1 medium		
		Skim capuccino	1 small		cafe

Day: \_\_\_\_\_ Date: \_\_\_\_\_

Time	Meal type	Food	Amount	How was it cooked (fried, grilled, boiled)	Where was it made (home, café, restaurant)
6pm	Dinner	Indian chicken curry:	Recipe makes 4 serves and I ate 1 serve		home
		Ghee	2 teaspoons		
		Chicken thigh fillets – skin & half fat removed	900 gram raw (according to the packet label)		
		Brown onion	1 medium raw		
		Curry paste	Half a cup		
		Campbell's reduced salt chicken stock	1 cup		
		Canned diced tomatoes	400 gram can		
		Sweet potato (orange and peeled)	500 gram raw (according to the label)	Boiled	
		Green beans	200 gram raw (according to the package)	Boiled	
		Basmati rice	1.5 cups cooked	boiled	
9pm	Supper	Chocolate cake	Cake makes 8 serves and I ate 2 serves		Home
		Brown sugar	1 cup		
		Dairy farmers full fat milk	3 quarters of a cup		
		Weston star regular fat butter	125g (using packet as a guide)		
		Cocoa powder	Half a cup		
		Self raising flour	1 cup		
		Bicarbonate of soda	Quarter teaspoon		
		Plain flour	2 tablespoons		
		Egg large	3		

Day:\_\_\_\_\_ Date:\_\_\_\_\_

Time	Meal type	Food	Amount	How was it cooked (fried, grilled, boiled)	Where was it made (home, café, restaurant)

#### PORTION GUIDE

Medium potato = woman's fist    40g hard cheese    = matchbox    180g fish fillet = whole hand

Medium fruit    = Fist                      100g cooked meat = palm of hand