

# Maternal dietary patterns and gestational diabetes mellitus

Ellen Alma Tryggvadóttir

Supervisors: Professor Ingibjörg Gunnarsdóttir and Associate professor Bryndís Eva Birgisdóttir

A thesis submitted to the degree of Master of Science in Human Nutrition Faculty of Food Science and Nutrition School of Health Sciences University of Iceland 2014



# Fæðuval íslenskra kvenna á meðgöngu og tengsl við meðgöngusykursýki

Ellen Alma Tryggvadóttir

Leiðbeinendur: Ingibjörg Gunnarsdóttir prófessor og Bryndís Eva Birgisdóttir dósent

Meistaraprófsverkefni í næringarfræði unnið á Rannsóknastofu í næringarfræði við Háskóla Íslands og Landspítala Háskólasjúkrahús

Matvæla- og næringarfræðideild Heilbrigðisvísindasvið Háskóla Íslands Júní 2014

#### Maternal dietary pattern and gestational diabetes mellitus

Ellen Alma Tryggvadóttir

Supervisors: Professor Ingibjörg Gunnarsdóttir and Associate professor Bryndís Eva Birgisdóttir

Thesis for the degree of Master of Science in Human Nutrition carried out at the Unit for Nutrition Research, University of Iceland and Landspitali - The National University Hospital of Iceland

Faculty of Food Science and Nutrition School of Health Sciences University of Iceland June 2014 Ritgerð þessi er til meistaragráðu í næringarfræði og er óheimilt að afrita ritgerðina á nokkurn hátt nema með leyfi rétthafa. © Ellen Alma Tryggvadóttir 2014 Prentun: Háskólaprent Reykjavík, Ísland 2014

### Ágrip

Inngangur: Góð næring á meðgöngu er afar mikilvæg fyrir heilbrigði móður og barns. Rannsóknir hafa bent til þess að fæðuval sé heilsusamlegra hjá þunguðum konum í kjörþyngd fyrir meðgöngu miðað við hjá þunguðum konum sem eru of feitar eða í ofþyngd fyrir meðgöngu. Offita við upphaf meðgöngu er einn áhættuþátta meðgöngusykursýki. Meðgöngusykursýki getur haft verulega slæmar afleiðingar fyrir bæði móður og barn. Markmið: 1) Að kanna hvort tengsl séu á milli fæðumynsturs á meðgöngu og hættu á meðgöngusykursýki. 2) Samanburður á fæðuvali þungaðra kvenna í kjörþyngd fyrir þungun og þeirra sem eru of þungar/feitar fyrir þungun.

Aðferðir: Um er að ræða ferilrannsókn framkvæmda í samtarfi við aðra rannsókn á kvennadeild Landspítalans þar sem íslenskum konum á aldrinum 18 - 40 ára var boðin þátttaka við 20. vikna ómskoðun á Fósturgreiningardeild Landspítalans. Þátttakendur vigtuðu allan mat og drykk í fjóra daga fljótlega eftir þátttöku (19. - 24. viku meðgöngu) og gengust síðan undir sykurþolspróf í kringum 23. - 28. viku meðgöngu. Fæðuupplýsingar voru skráðar í ICEFOOD forritið með kóðum úr ÍSGEM gagnagrunninum. Matardagbækur fengust frá 98 konum í kjörþyngd, 46 konum í yfirþyngd og 39 of feitum konum (n=183) sem voru nýttar í samanburð á fæðuvali. Af þessum konum fóru 86, 44 og 38 í sykurþolspróf (n=168) og hjá þeim síðarnefndu voru skoðuð tengsl fæðumynsturs við meðgöngusykursýki

**Niðurstöður:** 1) Fæðumynstur sem samanstóð af fisk og sjávarréttum; eggjum; grænmeti; ávöxtum og berjum; jurtaolíum; hnetum og fræjum; pasta; morgunverðarkorni; kaffi og te ásamt neikvæðu samhengi við gosdrykki og franskar kartöflur tengdist minni hættu á meðgöngusykursýki (OR: 0.54 95% CI: 0.30, 0.98). Tengslin voru enn til staðar eftir leiðréttingu ýmissa þátta (OR: 0.36 95% CI: 0.14, 0.94).

2) Konur í kjörþyngd fyrir meðgöngu virðast velja meira af hollum fæðutegundum en konur sem eru of feitar fyrir meðgöngu. Neysla ávaxta, grænmetis, fisks, trefja og Omega-3 virðist ekki vera nægileg meðal þungaðra kvenna á Íslandi. Skortur á D-vítamíni, joði og járni gæti verið til staðar hjá fjölda þeirra.

**Ályktanir:** Heilsusamlegt fæðumynstur gæti reynst verndandi gegn meðgöngusykursýki, sérstaklega hjá konum sem eru þegar í aukinni áhættu vegna ofþyngdar/offitu fyrir meðgöngu. Stór hluti þungaðra kvenna á Íslandi fylgja ekki ráðleggingum í fæðuvali.

#### Abstract

**Background:** A healthy diet during pregnancy is important for mother and child. Studies have implied that pregnant women of normal weight before pregnancy have healthier diets than those overweight or obese before pregnancy. Obesity is one of the risk factors for gestational diabetes mellitus (GDM), which is associated with negative health effects on both mother and child.

**Objective:** 1) Investigate associations between maternal dietary pattern and GDM.

2) Compare maternal diets for women of normal weight before pregnancy and overweight/obese before pregnancy.

**Methods:** A prospective observational study performed in cooperation with a separate study at the Gynecology department at the National hospital where Icelandic women aged 18 - 40 years were recruited at routine 20 week ultrasound at the Pre-natal diagnosis department. All participants kept a four day weighed food record as soon as possible following recruitment (weeks 19 - 24). All underwent an oral glucose tolerance test in weeks 23 - 28 Food data was recorded into the ICEFOOD calculating program based on the Icelandic food database (ISGEM). Food records were obtained from 98 normal weight women, 46 overweight women and 39 obese women (n=183), used to compare diets. Not all of these women underwent the OGTT or 86, 44 and 38 respectively (n=168) and only they were included in the study for GDM associations.

**Results:** 1) A dietary pattern comprising of seafood, eggs, vegetables, fruit and berries, vegetable oils, nuts and seeds, pasta, breakfast cereals, coffee and tea with a negative correlation to intake of soft drinks and french fries was associated with lower risk of GDM (OR: 0.54 95% CI: 0.30, 0.98). The association was still present in the adjusted model (OR: 0.36 95% CI: 0.14, 0.94). 2) Women of normal weight before pregnancy appear to have somewhat healthier maternal diet choices then pregnant women who are obese before pregnancy. Dietary intake of fruits, vegetables, fish, fiber and Omega-3 is lacking among pregnant women in Iceland. A number of the women may be at risk of deficiency for vitamin D, Iodine and Iron.

**Conclusions:** Adhering to a prudent dietary pattern in pregnancy may prove beneficial in preventing GDM, especially among women already at higher risk due to overweight/obesity before pregnancy. Maternal diet in Iceland could be improved.

#### Aknowledgements

The work presented in this thesis was carried out at the Unit for Nutrition Research, Faculty of Food Science and Nutrition, University of Iceland and Landspitali-The National University Hospital of Iceland and funded by The University of Iceland's research fund.

I would like to thank Professor Ingibjörg Gunnarsdóttir for the opportunity to work on this project and to thank both her and Associate professor Bryndís Eva Birgisdóttir for their invaluable guidance and assistance along the way. I am fortunate to have had such amazing supervisors who have both taught me so much.

I would like to thank all the staff at the Institution of nutrition research and Faculty of food science and nutrition who are always so helpful and friendly. I would also like to thank those behind the GDM-study at Landspítalinn, Helga Medek and Reynir Tómas Geirsson, the staff at the Pre-natal diagnosis department and all the study participants.

A special thanks to my fellow students and friends working on their own projects at the Unit for Nutrition Research and yet always ready to help in any way, especially Ph.D. student Birna Þórisdóttir and M.Sc. students Áróra Rós Ingvadóttir, Jóna Björk Viðarsdóttir and Harpa Hrund Hinriksdóttir.

Finally I would like to thank my boyfriend Hörður S. Dan. for all his patience and help and my family and friends for all their invaluable belief and support. I am so grateful for having you all in my life.

## **Table of contents**

Á	grip		. 3
A	bstract		. 3
A	knowle	edgements	. 4
T	able of	contents	. 5
Ir	dex of	tables	. 7
Ir	dex of	figures	. 7
A	bbrevi	ations	. 8
1	Intr	oduction	. 9
2	Bac	kground	10
	2.1	Gestational diabetes mellitus	10
	2.2	Diagnosing gestational diabetes mellitus	10
	2.3	Proposed mechanism	11
	2.4	Negative effects of gestational diabetes mellitus	12
	2.5	Risk factors	13
	2.5.	1 Weight gain in pregnancy	13
	2.6	Dietary guidelines in pregnancy	14
	2.6.	1 Folate	14
	2.6.	2 Vitamin D in pregnancy	15
	2.7	Dietary patterns	16
	2.7.	Diet and gestational diabetes mellitus	17
	2.7.	2 Healthy dietary pattern carbohydrates	20
	2.8	Benefits of dietary treatment	21
3	Met	hods	22
	3.1	Participants	22
	3.2	Dietary assessment	23
	3.3	Statistical data	23
	3.4	Author's contribution	24

4 R	esults of secondary aim	26
4.1	Dietary intake of normal weight, overweight and obese pregnant women	26
5 D	viscussion and future perspectives	35
5.1	Summary of findings	35
5.2	Discussion	35
6 C	onclusions	38
7 R	eferences	39
8 A	ppendices	46
8.1	Appendix I – Manuscript draft	46
8.2	Appendix II – Recipes from the Directorate of Health	63
8.3	Appendix III - Food groups in ISGEM	72
8.4	Appendix IV – Calculated loss (%) of nutrients due to cooking	75

# **Index of tables**

Table 1 - An overview of studies assessing the relationship between total diet quality and
risk of GDM
Table 2 - Dietary intake (g/day) divided by body mass index (BMI) before pregnancy 28
Table 3 - Macronutrient intake divided by body mass index (BMI) before pregnancy 30
Table 4 - Contribution of energy providing nutrients to total energy intake (E%)
Table 5 - Micronutrient intake as well as intake of selected heavy metals, caffeine and
aspartame divided by body mass index (BMI) before pregnancy
Index of figures
Figure 1
Figure 2

#### **Abbreviations**

**AR** Average requirement

**BMI** Body mass index

**DASH** Dietary approach to stop hypertension

**DHA** Docosahexaenoic acid

**EPA** Eicosapentaenoic acid

**FPG** Fasting plasma glucose

**g** Gram

G<sub>0</sub> Fasting glucose

**GDM** Gestational diabetes mellitus

**GDM-study** Get diabetics moving-study

GI Glycemic index

**HEI** Healthy Eating Index

WHO World Health Organization

**HOMA-IR** Homeostasis Model Assessment of Insulin Resistance

 $I_0$  Fasting insulin

**IOM** Institute of medicine

**IPAQ** International Physical Activity Questionnaire

**MET** Metabolic Equivalent of Task

mg Milligram

**NTD** Neural tube defects

**OGTT** Oral glucose tolerance test

**RDI** Recommended daily intake

**TAC** Total antioxidant capacity

**VIF** Variance inflation factor

μ**g** Microgram

#### 1 Introduction

A diverse and healthy diet is extremely important during pregnancy for the mother and child. During this time the mother needs to fulfill both her own energy- and nutrient requirements in addition to those of her child. The availability of all necessary nutrients is crucial during this time to ensure a normal and healthy growth process [1, 2]. Maternal diet studies have implied that a large number of pregnant women do not follow dietary guidelines and that in several cases they are not able to reach the recommended intake for many nutrients. The results vary somewhat between different countries [3-11]. A few observational studies have been performed in Iceland assessing dietary intake for pregnant women [12-16], suggesting that fish intake is limited in part of the pregnant population, vegetable intake is low and many women do not reach recommended intake levels of vitamin D [12, 13, 16]. Several studies have emphasized the importance of the maternal diet by demonstrating its associations to the mother's risk for gestational diabetes mellitus (GDM) [17-21]. However most of these studies have focused on specific food groups or nutrients and only a limited number have studied dietary patterns. As foods may have a combined effect these are important additional analyses. It may prove useful to gather information on local dietary patterns as they may vary between different countries. Results of such studies may provide the tools to create successful intervention studies.

The primary aim of this thesis was to investigate maternal dietary patterns and the associations to gestational diabetes mellitus in a group of pregnant women in Iceland (Appendix I). The secondary aim was to compare maternal diets of women who are normal weight before pregnancy and those who are either overweight or obese before pregnancy.

#### 2 Background

#### 2.1 Gestational diabetes mellitus

Gestational diabetes mellitus has been defined as glucose intolerance at any degree that either commences or is diagnosed during pregnancy. The same definition has been used whether insulin treatment is needed in combination to dietary modifications or not, even if symptoms remain after pregnancy. Therefore it is both possible that the glucose intolerance has existed before the pregnancy or that it started during pregnancy [22]. Most people who have impaired glucose tolerance exhibit less glucose uptake stimulated by insulin. To compensate for this the β-cells start secreting more insulin and thus create a state of hyperinsulinemia [23]. βcells and the liver seem to regulate concentrations of both plasma glucose and insulin levels in a loop of negative feedback. GDM is like other forms of diabetes when insulin levels secreted are not sufficient to meet demands. It has been proposed that the metabolic dysfunctions associated with gestational diabetes begin during the first trimester [24]. However, during the first half of pregnancy it is quite normal for both postprandial and fasting plasma glucose to be lower than normally seen in non-pregnant women [25] as pregnancy is a time of complex metabolic and hormonal changes. These changes involve an increase in insulin resistance which is meant to increase maternal plasma glucose and subsequently its availability to the fetus [26]. When insulin mediated glucose uptake of cells is impaired, the pancreas needs to increase the amount of insulin secreted to prevent hyperglycemia [23].

#### 2.2 Diagnosing gestational diabetes mellitus

There is to date a lack of global uniformity regarding the diagnoses of GDM and screening methods may also differ [27]. One method of screening is a two hour 75 gram oral glucose tolerance test, where fasting blood glucose is first measured, then blood glucose is tested at one and two hours after glucose administration [27]. Screenings for women considered at risk, are usually performed between week 24 and 28 of gestation [25]. According to recent recommendations from the World Health Organization (WHO) there should be two different references used when diagnosing diabetes in pregnancy. One is aimed at identifying those with diabetes mellitus in pregnancy without previous diabetes diagnoses, yet display severely high glucose levels. The other classifies those who have GDM. According to the recommendations from WHO, diabetes mellitus in pregnancy should be diagnosed in cases

where fasting plasma glucose is measured  $\geq 7.0$  mmol/l or the two hour plasma glucose is  $\geq$  11.1 mmol/l after a 75 gram oral glucose tolerance test. Also if the person displays symptoms of diabetes and a random test of plasma glucose is  $\geq$  11.1 mmol. The reference for GDM is fasting plasma glucose between 5.1 and 6.9 mmol/l, the one hour plasma glucose measured  $\geq$  10.0 mmol/l or the two hour plasma glucose is between 8.5 and 11.0 mmol/l after a 75 gram oral glucose tolerance test. If one, two or all of these criteria are met the woman is diagnosed with GDM [25].

#### 2.3 Proposed mechanism

The reasons for the dysfunction of cells in the pancreas leading to the lack of insulin in GDM are not entirely clear. In most women diagnosed with GDM the diminished insulin secretion is because of either β-cell dysfunction caused by genetic reasons, autoimmune dysfunction or a chronic state of insulin resistance [28]. Obese women have higher rates of insulin resistance than normal weight women in the beginning of pregnancy which when added to the insulin resistance accompanying a normal pregnancy (usually an increase of 50-60%) has an effect on the metabolism, leading to an excess of cytokines and nutrients in the environment surrounding the fetus [24, 29]. GDM is thought to display a similar pathophysiology as displayed in the metabolic syndrome in relation to inflammation, hypertension and hyperlipidemia, the link being hyperinsulinemia and an increase in insulin resistance. So it becomes more likely for a woman to develop gestational diabetes when she has prepregnancy insulin resistance [24, 29]. Women who have GDM have been shown to have higher concentrations of triglycerides in blood [30] and to demonstrate less insulin secretion when compared to women with normal glucose tolerance. If insulin resistance persists after gestation it may lead to an inability to sustain the excess insulin secretion needed, which can lead to the progression of type 2 diabetes [28]. A woman diagnosed with GDM should undergo testing six weeks or more after pregnancy to determine whether she has normoglycemia, diabetes type 1 or 2, impaired fasting glucose or impaired glucose tolerance [25, 31]. Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) is a calculation method used to assess levels of insulin resistance by using results of fasting glucose and fasting insulin measurements [32]. In GDM there appear to be two forms of insulin resistance. One is a physiological type of insulin resistance beyond what is normally seen in the last trimester of pregnancy in a normal pregnancy, which is displayed by altered insulin signaling that may result in a decrease in glucose uptake stimulated by insulin in the skeletal muscles. Usually the insulin resistance dissipates after gestation which may suggest that it is brought on by factors associated with the pregnancy [24, 28, 31]. The other form of impaired insulin response is associated with pre-pregnancy metabolic dysfunction which in addition to the physiological type of insulin resistance that occurs during normal pregnancy may result in more severe cases of insulin resistance during pregnancy. It involves both increased hepatic production of glucose and a decrease in glucose uptake stimulated by insulin. In the skeletal muscles an insulin signal enables glucose uptake as a result of the insulin receptor tyrosine being phosphorylated. One of the proposed mechanisms leading to the obesity related increased insulin resistance has been a decrease in tyrosine phosphorylation in the skeletal muscles. Another mechanism may be the inhibition of insulin signaling due to an increase in serine phosphorylation of the insulin receptor. An increase in insulin resistance in pregnancy has been associated with cytokine disruptions of insulin signaling [24, 28].

#### 2.4 Negative effects of gestational diabetes mellitus

There are many negative factors that have been associated with GDM for both mother and child. Having GDM has been associated with a greater risk of miscarriage, gestational hypertension, pre-eclampsia, trauma at birth and having infants that are very large, which in turn can lead to a higher risk of caesareans, prematurity and shoulder dystocia [33-37]. Women who have been diagnosed with GDM are also at a greater risk of diabetes later in life [37-40]. GDM may also be associated with a delay in lactation initiation [41]. Infants born to GDM women are at a higher risk of suffering from various malformations and Erb's palsy due to shoulder dystocia [33]. For the child there is also a risk of in utero growth restriction, hypoglycemia [34] and an increased risk of diabetes 2 later in life [37, 42-46]. Any increase in maternal plasma glucose has been demonstrated to have a related increase in birth weight due to hyperinsulinemia [47] and adiposity present in the infant at birth [48]. According to the hypothesis by Pedersen the increase in transference of nutrients to the infant is due to elevated glucose levels because of the presence of diabetes in the mother. Increased glucose transferred to the fetus then results in a hyper secretion of insulin in utero and a subsequent increase of glucose utilization by the fetus [49]. An increase in placental cytokine production and expression in GDM may be related to the insulin resistance developed in pregnancies. It may be possible that fetal insulin can have a modifying effect on gene expression in the placenta, glycogen deposits and expansion of blood vessels [28]. The increase in fetal growth may also be associated with an increase in lipid availability due to changes in maternal metabolism [29]. Children born to obese women are more likely to become obese themselves and develop dysfunctions in metabolism during childhood. And thus if the child is female she

may be more likely to be obese herself during childbearing years continuing the cycle of obesity and diabetes risk [50].

#### 2.5 Risk factors

Women who are overweight or obese have a greater risk of being diagnosed with GDM than women of normal weight [14, 40, 51]. According to a recent Icelandic study the rate of GDM was 10% among obese women, 2.7% among women who were overweight and 1.3% among women of normal weight [52]. A higher risk of being diagnosed with GDM is also associated with maternal age, a family history of diabetes 2, previous diagnoses of GDM or glucose in the urine, previous delivery of a large child and having been diagnosed with Polycystic Ovarian Syndrome (PCOS) [28, 53, 54]. Hyperlipidemia may also increase the risk of GDM by influencing cytokine expression. The aforementioned risk factors are all unmodifiable for the women once they are pregnant but additional risk factors that pregnant women are able to modify in an effort to decrease their risk for GDM are gestational weight gain, maternal diet and exercise. Exercise before and during pregnancy is thought to possibly lower risk of GDM [55, 56] although quality evidence is considered limited [57]. However incorporating physical activity in pregnancy for at least 30 minutes a day is recommended for all pregnant women [56, 58].

#### 2.5.1 Weight gain in pregnancy

In Iceland the official recommendation for gestational weight gain is 12-18 kg for normal weight women (BMI <25) and 7–12 kg for women who are overweight or obese (BMI ≥25) before pregnancy [59]. This is similar to recommendations from the Institute of medicine (IOM) where the optimal weight gain in pregnancy is 12.5–18 kg for underweight women (BMI <18.5 kg/ m2), 11.5–16 kg for normal weight women (BMI 18.5–24.9 kg/m2), 7–11.5 kg for overweight women (BMI 25.0–29.9 kg/ m2) and 5–9 kg for obese women (BMI ≥30.0 kg/m2) [60]. A high rate of gestational weight gain, particularly in early pregnancy, has been linked to a greater risk of GDM [61, 62]. Excessive gestational weight gain has also been associated with several adverse maternal and neonatal outcomes [63, 64] and has also been linked to lifestyle factors, although social- and genetic factors are also involved [64]. When women gain excessive weight during pregnancy it increases both birth weight and postpartum weight, in addition to being a likely prediction of obesity in the future for both mother and child [65, 66]. By using different criteria's for recommended pregnancy weight gain based on a calculated pre-pregnancy BMI (kg/m²) it may be possible to achieve better pregnancy

outcomes [67] as it seems that women who are overweight are more likely to gain excess weight during pregnancy [14]. Being overweight or obese before pregnancy presents an independent risk of adverse pregnancy outcomes and so it is especially important for these women to avoid excessive weight gain in pregnancy [68, 69]. A combination of healthy diet choices and increased physical activity during pregnancy may prove useful in preventing excessive gestational weight gain [70] in addition to lowering weight retention after birth [71].

#### 2.6 Dietary guidelines in pregnancy

The Icelandic guidelines for diet during pregnancy recommend eating a variety of nutrient dense foods and to limit consumption of processed foods and added sugar. The goal is to eat at least 500 grams of fruit and vegetables every day, fish at least two times a week, choose whole grains and fiber rich cereals, choose low fat dairy products, use oil or liquid fats instead of solid fats, use salt conservatively, take cod liver oil or choose another source of vitamin D and drink water [59]. Dietary recommendations for pregnant women in several countries such as: Denmark, Sweden, Norway, Britain and the United states appear to be similar to the Icelandic guidelines [72-77]. However in Denmark the recommendation is six pieces of fruits and vegetables per day and fish and fish products several times a week [75] which is similar to Norway where further a maximum limit of fatty fish has been set to 400g per week. They also recommend eating no more than 500g of red meat per week [73]. All Icelandic women of childbearing age are recommended to take folic acid supplements.

#### **2.6.1** Folate

Folate is a water soluble vitamin-B complex that can be found in foods such as leafy green vegetables, fruit and beans. Folic acid is the oxidized and more active form of this vitamin and has greater bioavailability than folate. Folic acid is the form used as a supplement and food additive [78]. Folate is necessary for many aspects of overall health, normal growth and development. It acts as a cofactor in many biological reactions including roles regarding the genome and gene expression, amino acid metabolism and neurotransmitter synthesis [79]. Women of childbearing age in Iceland are advised to supplement with 400 µg daily. This is especially important one month prior to conception and during the first trimester [79-81]. It has become widely accepted that pre-conceptional supplementing of folic acid can reduce the prevalence of Neural tube defects (NTD) [79, 82-86]. Neural tube defects are thought to be caused by both genetic disposition and environmental factors combined. The result of

which is failure of the spine to close during embryonic stage which can be fatal or cause paraplegia with paralysis in the lower extremities [78, 79]. Some research has indicated that folic acid intake may also reduce the risk of cleft lip and palate in the offspring [87-89] although not all studies agree on this and it has been proposed that the risk reduction is rather due to multivitamin supplementation than folic acid alone [90]. In addition folate is thought to have beneficial effects on brain development and function in the offspring [91] and that it may possibly prevent some cardiovascular malformations [92]. Recent studies have also demonstrated a positive association between folate intake and improved birth weight [93, 94]. Some countries have chosen to fortify a number of foods with folic acid since supplementation recommendations have not always been effective [95, 96]. There has been some speculation regarding the safety of folic acid supplementation and fortification as folate may contribute to masking B<sub>12</sub> deficiency. Some fear that it may also have a negative effect on cancer and tumor growth [97] but this has been refuted by others and the hypothesis is thought to lack sufficient evidence [98]. Recently there has been debate as to whether it may be beneficial to change recommendations toward multivitamin supplementation instead of folate alone [99-101] but more evidence is needed before such suggestions can be formally made [102].

#### 2.6.2 Vitamin D in pregnancy

Insufficient vitamin D levels during pregnancy appear to be a worldwide problem, even in some countries with high levels of sun exposure where clothing habits may interfere [103-105]. Vitamin D is known to have an integral part in the maintenance of calcium homeostasis in the body. That is achieved by its involvement in calcium absorption, reabsorption and bone mineralization. Recent studies have indicated that the role of vitamin D may be more extensive than previously thought and that it may also include a part in immune function and other health related factors [106, 107]. The body is able to synthesize vitamin D from direct exposure to the sun but in northern parts of the world such as Iceland, that option is only viable during the summer and so an alternative source of vitamin D is specifically recommended during the remaining months of the year [81, 108]. Some foods such as fortified milk and cereals, oil, fatty fish and eggs contain a small amount of vitamin D but in Iceland the main source is Lýsi (cod liver oil). Currently the recommended daily dosage in Iceland of vitamin D is 15 µg for women of childbearing age [109]. Because of the involvement vitamin D has in calcium homeostasis it is important to maintain adequate levels during pregnancy to prevent rickets and improve bone health in the offspring [103, 106, 107,

110-112]. A possible link between vitamin D deficiency and abnormal glucose metabolism has also been demonstrated. In a number of studies pregnant women with gestational diabetes appeared to have lower levels of vitamin D [104, 105, 113-115]. In addition some of the recent literature has indicated that low vitamin D levels may be associated with an increased risk of preeclampsia [112, 115, 116] and bacterial vaginosis [112, 115]. In accordance to this some studies have indicated that by supplementing vitamin D during pregnancy it is possible to raise its levels and subsequently decrease the risk of infections and preterm birth [111], atopy and asthma in the child later on [117], preeclampsia [116, 118], bacterial vaginosis [118] and improve fetal growth and infant immune function [116]. But not all researchers agree with the hypotheses on the safety and health benefits of vitamin D supplementation [119, 120] and caution may be warranted regarding over supplementation, since some studies have demonstrated an adverse effect of high levels of vitamin D, such as a higher risk of food allergy in the offspring [121] and pregnancy-associated breast cancer [118]. There seems to be a lack of worldwide consensus regarding the optimal dosage and procedure regarding vitamin D supplementation during pregnancy but most studies highlight the urgent need for more randomized control trials on this subject [106, 113, 114, 120, 122-124].

#### 2.7 Dietary patterns

Recently there has been a greater focus on investigating the combined effect of various foods or food groups on health and health related factors as an addition to investigating isolated foods or nutrients. By ascertaining specific dietary patterns from information on dietary intake in large groups or countries it is then possible to investigate associations between the patterns and available health factors. There are mostly two methods used to find dietary patterns. One is a data-driven statistical methodology or a posteriori and the other is the index methodology or a priori [125, 126]. In the first method dietary patterns are found by using cluster analysis, factor analysis or principal component analysis in datasets to identify any dietary patterns among participants. The second one is when a predefined base such as dietary recommendations for example are used in conjunction with results of dietary data to create an index of adherence to the recommendations. Dietary patterns that have been demonstrated and consequently studied are for example the Mediterranean diet, Western diet and a Prudent diet. However the definitions for foods and food groups within those patterns may vary between different investigating parties [126].

#### 2.7.1 Diet and gestational diabetes mellitus

Some studies have suggested that pregnant women of normal weight have a healthier diet choice than pregnant women who are overweight or obese [127-130]. One study performed in Iceland demonstrated that overweight or obese women were more likely to excessively increase their overall energy consumption and consequently gain excessive weight during their pregnancy [14]. A healthy diet prior to pregnancy is thought to decrease the risk for GDM [131, 132]. Some studies credit certain factors of the diet to be directly associated with the risk of GDM such as higher consumption of soft drinks [133], increased consumption of energy [21], fat especially saturated fat [134] and decreased consumption of polyunsaturated fat and carbohydrates [17-21]. Long chain polyunsaturated fatty acids are thought to have an anti-inflammatory effect [24]. A few studies have been conducted on the association between vitamin D and GDM. Some demonstrate a link between low plasma 25(OH) D and an increased risk of GDM [135, 136] while others do not [137]. Some research suggests that a lack of vitamin D is associated with an imbalance in glucose metabolism [104, 138, 139]. However even though studies point to direct associations between isolated foods or nutrients and GDM it must be considered that the relevant intake may correlate with intake for a different food or nutrient which may have a combined effect. For instance a high consumption of vegetables, nuts and seeds may be strongly correlated to fish consumption which in combination provides good sources of folate, iodine, vitamin D and Omega-3. Following either a healthy-, Mediterranean or DASH diet has been related to a decrease in risk for GDM [140]. One study demonstrated a greater GDM risk for Latin women following an unhealthy Western type diet when compared to a prudent dietary pattern [141].

An overview of studies assessing the relationship between total diet quality and risk of GDM is given in Table 1. More studies are needed in order to define dietary patterns associated with lower GDM [142] and results from observational studies need to be confirmed in interventional studies [143, 144].

Table 1

Author, year, country/Study design	Population subject characteristics/ Dietary assessment method	Exposure/Intervention	Outcome	Results
Karamanos B [143], Greece, 2013 Observational study	1076 pregnant women from ten Mediterranean countries Validated questionnaire	Mediterranean diet index was computed and its adherence associations to GDM investigated	GDM assessed by 75-g OGTT at the 24th-32nd week of gestation	Adherence to a Med Diet pattern of eating is associated with lower incidence of GDM (OR=0.655 $P$ =0.004.) and better degree of glucose tolerance, even in women without GDM
Asemi Z [144] 2013 Iran. Randomized ctrl trial	32 pregnant women diagnosed with GDM at 24 to 28 week gestation	Participants were randomly assigned to consume either the control (n = 16) or DASH diet (n = 16) for 4 weeks	Fasting blood samples were taken at baseline and after 4 week of intervention to measure fasting plasma glucose (FPG), serum insulin, and hs-CRP, homeostasis model of assessment-insulin resistance (HOMA-IR), plasma total antioxidant capacity (TAC), and total glutathione levels (GSH)	Consumption of the DASH diet in pregnant women with GDM had beneficial effects on FPG (-7.62 vs 3.68 mg/dL $P$ =0.02), serum insulin levels (-2.62 versus 4.32 $\mu$ IU/mL, $P$ =0.03), HOMA-IR score (-0.8 versus 1.1; $P$ = 0.03), plasma TAC (45.2 versus -159.2 mmol/L; $P$ < 0.0001), and total GSH levels (108.1 versus -150.9 $\mu$ mol/L; $P$ < 0.0001)
Asemi Z [145] 2012, Iran. Randomized ctrl trial	34 women diagnosed with GDM at 24-28 weeks gestation	Subjects were randomly assigned to consume either the control diet (n 17) or the DASH eating pattern (n 17) for 4 weeks	Fasting blood samples were taken at baseline and after 4 weeks of intervention to measure fasting plasma glucose, glycated Hb (HbA1c) and lipidprofiles. Participants underwent a 3 h oral glucose tolerance tests and blood samples were collected at 60, 120 and 180 min to measure plasma glucose levels	Consumption of the DASH eating pattern for 4 weeks among pregnant women with GDM resulted in beneficial effects on glucose tolerance and lipid profiles compared with the control diet

Author, year, country/Study design	Population subject characteristics/ Dietary assessment method	Exposure/Intervention	Outcome	Results
Tobias DK [131], 2012, Boston- Harvard Observational study	15,254 Nurses' Health Study II participants. Pregnancies were free of pre-pregnancy chronic disease or previous GDM Validated food- frequency questionnaire	Pre-pregnancy dietary pattern adherence scores were computed based on participants' usual intake of the patterns' components such as fruit, vegetables, nuts, legumes, white/red meat ratio, fiber, dairy etc	872 incident cases of GDM were documented	Pre-pregnancy adherence to healthful dietary patterns is significantly associated with a lower risk of GDM. MED was associated with a 24% lower risk (RR: 0.76; 95% CI: 0.60, 0.95), DASH with a 34% lower risk (RR: 0.66; 95% CI: 0.53, 0.82), and HEI with a 46% lower risk (RR: 0.54; 95% CI: 0.43, 0.68)
Zhang C [146] 2006 Boston Observational study	13,110 women free of cardiovascular disease, cancer, type 2 diabetes and history of GDM in the Nurses' Health Study II. Validated semi-quantitative food frequency questionnaire	Two major dietary patterns (i.e. 'prudent' and 'Western') were identified through factor analysis	758 incident cases of GDM were documented	These findings suggest that pre- pregnancy dietary patterns may affect women's risk of developing GDM. A diet high in red and processed meat was associated with a significantly elevated risk (RR: 1.63 (95% CI 1.20-2.2 p (trend) 0.001)

Several other studies have demonstrated associations between dietary patterns and other pregnancy related complications [147-149].

#### 2.7.2 Healthy dietary pattern carbohydrates

Carbohydrates should be a part of a healthy dietary pattern. Fiber rich and whole grain carbohydrates are the recommended type for a healthy diet [81]. Foods that are rich in dietary fibers such as: vegetables, whole grain, cereals, legumes, pulses and fruits are believed to have multiple beneficial health effects and may possibly reduce the risk of developing several diseases such as cardiovascular disease, cancer, obesity and type 2 diabetes [150-155]. Grains are considered to be whole grains when they still contain its endosperm, bran and germ. When grains are refined the bran and part of the germ is usually removed [156]. Example of whole grain foods are whole wheat, oats, rye, barley, brown rice, and bulgur [155]. Many fiber rich foods are low-GI foods, but not all and GI of food depends on many other factors such as cell structure. A dietary pattern consisting of low-GI foods has been demonstrated to have a beneficial effect on pregnant women with GDM but more intervention studies on the matter are lacking [157]. Choosing low GI food items is recommended in treatment of diabetes and GDM [158-160].

Whole grain foods contain many health beneficial components such as: dietary fiber, vitamins, minerals, lignans, phenolic compounds, phytic acid, tannins and more [156]. The reasons for the possible protective effects of wholegrain against diabetes 2 still remain under debate. Dietary fiber content is one of the factors that is thought to influence the body's glycemic response to foods. Whole grain foods tend to lead to slower digestion and absorption of carbohydrates [156] possibly due to enzyme inhibitors [161] and slower absorption of nutrients [155]. It has been demonstrated that increasing wholegrain consumption can lead to lower fasting glucose and –insulin [155, 162] and potentially lower rates of diabetes 2 [163, 164] which is debated [165]. It is also possible that the antioxidants that are found in wholegrain- and other foods rich in dietary fiber could be one reason for the protecting effect by reducing the activity of free radicals and subsequently lowering the risk of diseases [156]. In Iceland the current recommendations are for consumption of at least 25 grams of fiber each day for all adults by means of fruit, vegetables, legumes and wholegrain products [81]. Recent literature has demonstrated that fiber intake during pregnancy is below recommendations in many countries [166]. There is a lack of reliable evidence regarding the role of whole grains and fiber in the prevention of diseases such as gestational diabetes, and there is need for more quality trials [167].

#### 2.8 Benefits of dietary treatment

Identifying and subsequently managing gestational diabetes has been linked to a lower infant mortality and morbidity rate [28]. Women with GDM should receive counseling from a dietitian to assist them in choosing a diet that keeps glucose levels normal in addition to providing all required nutrients for both mother and fetus, without exceeding recommended gestational weight gain [168]. This service is not readily available at this time in Iceland. One reason for this may be that the need has increased greatly over a short period of time, with the steadily growing rate of overweight and obesity making it difficult for the healthcare system to adjust in time. Another possibility is that current information regarding treatments is still limited and if the most effective evidence based treatment is to be found, relevant intervention trials are needed. Previous treatment interventions for GDM have demonstrated a possibility of improving the women's quality of life related to their health in addition to reducing perinatal morbidity [169] and risk of macrosomia, caesarians, shoulder dystocia and hypertension, excessive weight gain, pre-eclampsia, and the need for insulin therapy. Outcomes are also better in regards to macrosomia, neonatal hypoglycemia, and birth weight due to better glycemic control [25, 29, 170, 171]. And thus by diagnosing and properly treating GDM it may be possible to increase the odds of a normal pregnancy outcome [172]. A study comparing adverse outcomes between treated and untreated groups of women diagnosed with GDM, where caloric intake was restricted to 25/35 kcal/kg with recommendations for 3 meals and 4 snacks a day, demonstrated much lower rates of adverse outcomes for the treated group and in some cases similar results for the treated group as seen in a non-diabetic group [173]. If healthy diet choices are successfully implemented during a GDM pregnancy and continued by the women after the pregnancy it may possibly reduce her risk for diabetes mellitus type 2 later in life [140]. The one thing that is clear today is the importance of encouraging all women to choose a healthy and diverse diet. It is also important to study associations of modifiable risk factors to GDM in the aims of providing women with the right tools to possibly prevent its occurrence and subsequently increase their quality of life as well as their offspring's.

#### 3 Methods

The data for this study was gathered as part of a larger study, called Get diabetics moving (GDM-study), which was conducted at the National university hospital. The aim of the GDM-study is to study the impact of increased physical activity on blood sugar levels, weight, metabolism and oxygen transfer to the fetus for women with GDM. All study participants underwent a 2 hour, 75 gram oral glucose tolerance test (OGTT) at gestation weeks 23 - 28 and a fetal Echocardiography at gestation weeks 31 - 38. Information on both pre-pregnancy and weight gain during pregnancy was gathered. Dietary intake was assessed by a four day weighed food record either from Wednesday - Saturday or Saturday - Tuesday as soon as possible following recruitment (weeks 19 - 24). See also the method section in the attached manuscript.

#### 3.1 Participants

Participants were recruited over a period of 18 months from April 2012 - October 2013 at a routine 20 week ultrasound with the help of staff at the Pre-natal diagnosis department at the National university hospital. Researchers were randomly present during recruitment period at the pre-natal diagnosis department. The department staff introduced the study to eligible women during their ultrasound and if they were interested they were forwarded to meet with a researcher who introduced the study protocol in detail. Initially the criteria for participation were: Age between 18 - 40 years, first - third pregnancy, non smoker, no family history of diabetes or gestational diabetes and BMI between 18.5-24.9 kg/m<sup>2</sup> (normal weight) or 30-<40 kg/m<sup>2</sup> (obese). After six months of recruiting the criteria were altered to include women with a BMI of 25-29.9 kg/m<sup>2</sup> (overweight) and allow women with a family history of diabetes. The main reason for the changed protocol was that the recruitment process was delayed, mainly due to the fact that the participation rate in the obese group was lower than expected. The change in protocol was approved by the steering committee of the GDM-study. A total of 217 women were recruited, 56 women declined participation (participation rate 79%). Food records used to compare diets were obtained from 98 normal weight women, 46 overweight women and 39 obese women (n=183). Of those 86, 44 and 38 respectively (n=168) underwent the OGTT and where thereby eligible for studying the association between dietary intake and GDM. Recent guidelines from the World health organization were used to determine the presence of GDM: Fasting plasma glucose between 5.1 and 6.9 mmol/l, the one hour plasma glucose measured ≥ 10.0 mmol/l or the two hour plasma glucose between 8.5 and 11.0 mmol/l after a 75 gram oral glucose tolerance test. If one, two or all of these criteria were met the woman was diagnosed with GDM [25].

#### 3.2 Dietary assessment

During recruitment participants were provided with both vocal and written instructions on how to properly fill out a four day food weighed food record and were provided with a food scale for weighing. They were instructed to record intake of all food and drink, including all supplements for the duration of four days, either from Wednesday – Saturday or Saturday – Tuesday as soon as possible after recruitment (in weeks 19 - 24). The selection of weekdays was made in order to offer some flexibility while still gathering information regarding dietary intake for two week days and two weekend days, where Friday was considered a weekend day as it has been shown to have similar intake variance as Saturday and Sunday [174]. This decision was based on studies demonstrating that energy intake may vary over the course of the week and is possibly increased on weekends [175]. Participants were instructed to weigh each food type separately and record: time of meal, type of food with brand name and the amount of each separate food type in grams. If they were unable to weigh foods separately, they were instructed to either describe the ratio of different foods or write down the relevant recipe.

#### 3.3 Statistical data

The food data was recorded into the ICEFOOD calculating program version 2.0 that is based on nutritional composition values on 514 ingredients from the Icelandic food database ISGEM and 607 food recipes from the Directorate of Health (Appendix II and III) [176, 177]. The nutritional data from ISGEM anticipates loss of certain nutrients during different cooking methods (Appendix IV). Energy is calculated as: 9 kcal/g for fats, 7 kcal/g for alcohol, 4 kcal/g for protein and carbohydrates and 2 kcal/g for fiber [177]. The results for participants nutritional data is displayed in Microsoft Excel for each recorded day, where the average intake was calculated for each participant. Those results in addition to all information regarding participants gathered during recruiting, and results from the OGTT were transferred to the program IBM SPSS Statistic version 20, where all statistical analysis took place. To determine significance of dietary intake differences Mann Whitney U test was used for comparing two weight groups and Kruskal-Wallis non-parametric test the for all three weight groups. Criteria for recommended daily intake of micronutrients for pregnant women in

Iceland was obtained from the Icelandic Directorate of health [81] and women's average requirements from the new Nordic nutrition recommendations [126].

#### 3.4 Author's contribution

My work on this project was divided into four separate work stages.

#### 1. Recruiting participants

My work on this project began with my assistance in recruiting participants to the study: Get Diabetics Moving, from September 2012 until September 2013. I was situated at the pre-natal diagnosis department at random times to introduce and explain all aspects of the study to possible participants coming from their 20<sup>th</sup> week ultrasound. Participants were required to sign an Informed consent form, fill out informational forms and a physical activity questionnaire. During the same time I calculated the persons BMI and gestational age and recorded all informational data into a Microsoft excel sheet. I then scheduled the participant's appointment for the OGTT in addition to the next appointment with the Ph.D student Helga Medek. I explained to the participants how to properly fill out a weighed food diary and provided them with all their relevant documents and materials.

#### 2. Recording data from food diaries

All data from food diaries was recorded into excel sheets associated with the ICEFOOD calculating program which is based on values from the Icelandic food database ISGEM. Each day of the four day food diaries is recorded in a separate sheet and named according to the participant's number and day of diary. Specific codes are then assigned to each type of food for the calculating process. Once all diaries were recorded and properly coded, the calculating process is initiated. The results are displayed in an excel sheet where I calculated the average results for all four days. Food groups were created for the analysis, as presented in the method section in the attached manuscript.

#### 3. Statistical analysis and presentations

I transferred all results from the ICEFOOD calculating program an addition to all other gathered participant information to the program IBM SPSS Statistic version 20. There I reviewed the data in an effort to find and correct errors in the data. Subsequently I conducted all relevant statistical analysis for the data, with guidance from my instructors. I presented main results for the scientific article draft in a video recording in April 2014, as a part of Science in spring days at the National university hospital. I also created a poster explaining my master's project and some preliminary results which was displayed at a presentation at a Graduate study gathering for the Food Science- and nutrition department at the University in Iceland.

#### 4. Writing theses

I wrote a thesis including a draft for a scientific paper using the results of the analyzed data. I used mostly Pubmed and Science direct in the search of relevant articles for references although some were retrieved from citations in other articles. Reference searches consisted of search words such as: Pregnancy, Nutrition, Diet, Gestational Diabetes Mellitus, Dietary patterns and Principal Component analysis.

#### 4 Results of secondary aim

Results related to the primary aim of the thesis are presented in Appendix I, in the draft manuscript named: Association of a maternal dietary pattern derived from pregnant women in Iceland to GDM risks.

Results that belong to the secondary aim of comparing maternal diets of women who are normal weight before pregnancy and those who are either overweight or obese before pregnancy are presented below.

#### 4.1 Dietary intake of normal weight, overweight and obese pregnant women

When dietary intake of 27 common food groups was compared between all three weight groups (Table 1) there was a significant difference for intake of potato chips/popcorn (P < 0.01).

When comparing the dietary intake between the normal weight - and the overweight women it revealed that overweight women had a tendency towards higher intake of French fries (P=0.08), poultry (P=0.07), milk and dairy products (P=0.10) of borderline significance and lower intake of sugar, candy and honey (P=0.06) compared to normal weight women.

The obese women had a significantly higher intake of milk and dairy products (P=0.04), soft drinks (P=0.04), potato chips/popcorn (P<0.01) compared with the normal weight women. They also had a lower intake of vegetable oil (P=0.04), and tended to consume less fruit than the normal weight women, although the difference was not statistically significant (P=0.08).

No significant difference was observed in intake of total energy, energy providing nutrients or contribution of energy giving nutrients to total energy intake between the three groups (Table 2 and Table 3) when including both diet and supplements. However, intake of total omega-3 fatty acid, Eicosapentaenoic acid (EPA) and Docosahexaenoic acid (DHA) intake was of borderline significance higher in the diet of normal weight women compared with those who were obese prior to pregnancy (P= 0.09, P= 0.07 and P= 0.10, respectively).

Table 4 is an overview of the intake of vitamins, minerals and other substances from the diet. Intake of different vitamins and minerals is outside the scope of the present thesis and the results are only provided for descriptive purpose and no statistical analysis assessing possible differences between normal weight-, overweight- and obese women have been performed at this point. Possible associations between intake of specific nutrients or other substances and GDM have not yet been assessed.

The total percentage of all the pregnant women able to reach the average requirements of micronutrients, as suggested for non-pregnant women [126] and the recommended daily intakes for pregnant women [81] respectively, can be seen in Figure 1.

**Table 2 -** Dietary intake (g/day) divided by body mass index (BMI) before pregnancy

		N	Iormal	weight	n=98 (B	MI:18.5	-24.9)				Overw	eight n=	=46 (BN	ИI: 25-2	29.9)		C	bese n=	39 (BM	II: 30-40	0)		
	Percentiles Percentiles													es			Percentiles						
	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90		
Vegetables	96	55	39	56	86	125	168	116	72	33	60	105	162	219	115	87	45	58	83	152	209		
Nuts and seeds	6	15	0	0	0	5	23	5	8	0	0	0	7	15	3	6	0	0	0	2	15		
Fruits	166	115	22	81	157	234	317	151	113	28	61	134	217	285	133 <sup>x</sup>	100	15	73	119	175	240		
Fruit juices, no added sugar	144	148	0	0	110	249	348	106	111	0	0	91	165	262	112	153	0	0	59	124	413		
Potatoes <sup>1</sup>	22	27	0	0	15	33	60	22	24	0	0	13	38	60	27	31	0	0	19	47	76		
French fries	10	18	0	0	0	20	30	16 <sup>x</sup>	21	0	0	0	29	47	14	19	0	0	0	28	42		
Bread and crisp bread	103	44	51	62	103	131	164	103	47	58	69	95	120	161	102	34	64	84	103	124	138		
Pasta, couscous	20	34	0	0	0	29	62	22	34	0	0	0	34	82	18	28	0	0	0	32	62		
Wholegrain cereals	40	32	3	18	31	60	90	37	30	0	12	31	55	77	31	25	2	11	28	50	74		
Breakfast cereals	47	54	0	12	33	62	101	55	55	0	18	43	74	123	49	48	0	12	37	74	114		
Fish, fish products and shellfish	26	26	0	0	20	50	61	32	36	0	0	19	50	100	20	22	0	0	11	38	50		
Poultry	28	27	0	0	23	41	63	38 <sup>x</sup>	37	0	12	37	51	76	38	37	0	0	32	58	74		
Meat and meat products	53	36	7	24	50	79	104	52	31	15	27	49	75	104	61	43	19	28	50	79	143		
Eggs	15	18	0	0	9	24	40	14	18	0	2	9	19	39	11	12	0	0	4	20	32		
Milk and dairy products	291	196	84	171	258	377	520	335 <sup>x</sup>	181	133	205	297	448	636	336*	159	111	207	347	472	535		
Cheese	43	25	16	25	39	59	77	45	25	20	30	41	49	84	45	26	16	24	41	59	86		

Overweight n=46 (BMI: 25-29.9)

Obese n=39 (BMI: 30-40)

Data is displayed as mean and standard deviation (SD) and percentiles

Normal weight n=98 (BMI:18.5-24.9)

<sup>\*</sup> Significantly different from the normal the weight group ( $P \le 0.05$ )

<sup>&</sup>lt;sup>x</sup> Of borderline significant difference from the normal weight group (P < 0.1)

Mann Whitney U test was used to compare differences between groups

<sup>&</sup>lt;sup>1</sup>Not including French fries

<sup>&</sup>lt;sup>2</sup> Includes all animal fats (except fish), butter, margarine and hydrogenated fats

<sup>&</sup>lt;sup>3</sup> Bread salads are mayonnaise based salads

Table 3 - Macronutrient intake divided by body mass index (BMI) before pregnancy

		Normal weight n=98 (BMI:18.5-24.9) Overweight n=46 (BMI: 25-29.9)													Obese n=39 (BMI: 30-40)									
					Percentil	es						Perc	entiles				Percentiles							
	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90			
Energy, total (Kcal/day)	2157	405	1543	1880	2140	2408	2687	2092	457	1419	1741	2146	2356	2688	2195	533	1543	1814	2113	2553	3023			
Protein, total (g/day)	81	17	59	69	79	89	98	86*	17	61	76	88	95	106	84	20	58	67	84	97	117			
Fat, total (g/day)	84	20	61	70	83	97	111	80	21	54	65	78	95	109	84	28	50	64	78	100	116			
Saturated fat (g/day)	34	9	24	27	32	40	45	31	9	19	24	31	35	44	33	11	22	24	31	38	50			
Monounsaturated fat (g/day)	27	7	18	23	27	31	35	25	7	17	20	25	30	36	27	10	16	20	25	31	42			
Polyunsaturated fat (g/day)	14	4	9	11	14	17	20	13	5	8	10	12	16	21	14	6	7	8	13	18	21			
Omega-3 fatty acids (g/day)	0.6	0.8	0.1	0.1	0.3	1.0	1.6	0.5	0.7	0.1	0.1	0.2	0.5	1.5	0.4 <sup>x</sup>	0.6	0.0	0.1	0.2	0.5	1.6			
EPA (g/day)	0.2	0.3	0.0	0.0	0.1	0.3	0.6	0.2	0.2	0.0	0.0	0.1	0.2	0.5	0.2 <sup>x</sup>	0.2	0.0	0.0	0.1	0.2	0.6			
DHA (g/day)	0.3	0.4	0.0	0.1	0.1	0.5	0.9	0.3	0.4	0.1	0.1	0.1	0.3	0.8	0.2 <sup>x</sup>	0.3	0.0	0.0	0.1	0.3	0.8			
Carbohydrates, total (g/day)	258	60	176	214	257	299	333	246	62	171	200	241	296	338	265	67	165	217	261	295	391			
Added sugar (g/day)	66	30	30	41	62	87	113	62	30	28	37	56	82	115	70	35	33	44	64	87	122			
Fiber, total (g/day)	19	6	12	14	18	22	26	19	6	10	14	18	24	27	20	7	13	15	17	24	29			

Data is displayed as mean results and standard deviation (SD) and as percentiles \* Significantly different from the normal the weight group ( $P \le 0.05$ ) \* Of borderline significant difference from the normal weight group (P < 0.1) Mann Whitney U test was used to compare differences between groups

 $Table\ 4\ -\ Contribution\ of\ energy\ providing\ nutrients\ to\ total\ energy\ intake\ (E\%)$ 

	Norma	l weight	n=98 (	BMI:1	8.5-24	.9)	Ove	rweight	n=46 (	BMI: 2	25-29.9	)	Obese n=39 (BMI: 30-40)							
			Pe	ercentil	les					P	ercentil	es		Percentiles						
	Mean	10	25	50	75	90	Mean	10	25	50	75	90	Mean	10	25	50	75	90		
Protein	15	14	16	16	15	15	14	17	14	17	18	17	16	15	16	13	17	16		
Fat	36	37	36	36	36	37	35	34	35	35	35	36	35	33	34	34	36	34		
Saturated fat	14	14	14	14	15	15	14	12	13	14	13	14	14	15	13	13	14	15		
Monounsaturated fat	12	11	12	12	11	12	11	11	10	11	11	12	11	10	11	11	11	12		
Polyunsaturated fat	6	5	5	6	6	6	6	5	5	5	6	7	6	5	4	6	7	6		
Omega-3 fatty acids	0.3	0.0	0.1	0.1	0.4	0.5	0.2	0.1	0.1	0.1	0.2	0.5	0.2	0.0	0.0	0.1	0.2	0.5		
Carbohydrates	49	47	48	49	49	49	48	49	47	48	49	49	49	49	51	50	48	51		
Added sugar	13	8	9	12	14	17	12	8	9	11	14	17	13	10	10	12	14	16		

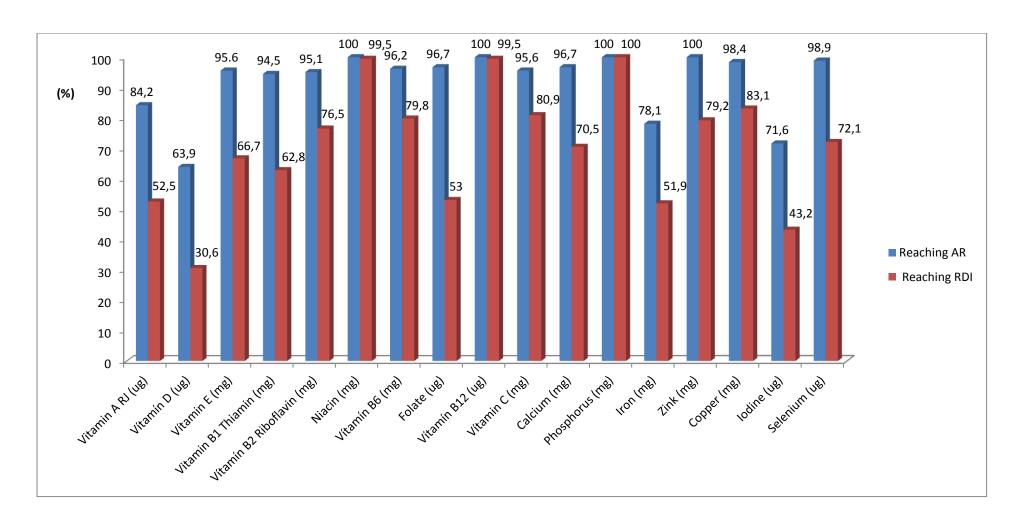
Table 5 - Micronutrient intake as well as intake of selected heavy metals, caffeine and aspartame divided by body mass index (BMI) before pregnancy

		Norn	nal weig	ht n=98	(BMI:1	8.5-24.9	")		Overweight n=46 (BMI: 25-29.9)								Obese n=39 (BMI: 30-40)						
				Perce	entiles							Percent	tiles				Percentiles						
	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90		
Vitamin A RJ (ug)	1152	735	443	613	992	1479	2203	977	592	412	549	754	1270	1807	1057	681	463	607	775	1375	2147		
Retinol (ug)	932	701	283	419	739	1255	1827	825	583	334	433	579	1108	1747	859	651	285	420	613	1217	1838		
Beta Carotene (ug)	2446	2360	440	907	1809	3198	4725	1721	1304	444	735	1272	2532	3382	2275	2091	429	787	1561	3799	5109		
Vitamin D (ug)	14	12	2	4	11	17	29	12	9	3	5	11	18	25	13	12	2	4	9	20	31		
Vitamin E (mg)	16	9	7	10	14	20	27	13	7	6	8	11	17	21	15	9	5	8	14	20	30		
B1,Thiamin (mg)	2.1	1.2	1.0	1.2	1.9	2.5	3.7	1.9	1.2	1.0	1.3	1.4	2.2	3.8	2.2	1.1	1.1	1.3	2.0	2.7	3.4		
B2,Riboflavin (mg)	2.6	1.4	1.3	1.5	2.4	3.1	4.5	2.4	1.3	1.4	1.6	2.1	2.9	4.2	2.7	1.3	1.4	1.8	2.6	3.3	4.0		
Niacin (mg)	26	14	12	16	23	33	42	24	14	13	17	20	28	41	28	15	15	16	26	37	47		
Vitamin B6 (mg)	3	2	1	2	3	3	5	3	2	1	2	2	3	5	3	2	1	2	3	4	5		
Folate (ug)	584	338	232	307	547	738	984	550	321	232	334	482	685	973	607	285	278	402	602	748	998		
Vitamin B12 (ug)	6	3	3	4	6	8	12	6	3	3	4	6	7	10	6	2	4	5	6	8	10		
Vitamin C (mg)	159	92	75	96	141	205	255	159	177	58	87	116	176	296	177	125	57	103	157	225	265		
Calcium (mg)	1099	390	695	837	1060	1264	1608	1130	345	674	925	1114	1346	1553	1100	332	762	880	1023	1314	1598		
Phosphorus (mg)	1503	344	1121	1264	1439	1679	1935	1561	342	1092	1309	1602	1783	2005	1564	358	1095	1336	1520	1781	2185		

		Norma	al weight	n=98 (E	3MI:18.5	-24.9)			Ove	erweight	n=46 (B)	MI: 25-2	9.9)	Obese n=39 (BMI: 30-40)							
				F	Percentile	es					I	Percentile	es	Percentiles							
	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90	Mean	SD	10	25	50	75	90
Magnesium (mg)	307	82	208	243	295	354	419	308	79	194	247	296	374	425	311	85	210	249	315	354	424
Potassium (mg)	2723	617	2008	2245	2618	3147	3618	2788	752	1789	2165	2768	3266	3934	2790	751	1962	2245	2661	3149	3796
Iron (mg)	22	24	8	10	16	22	38	17	19	7	9	14	19	28	18	8	10	12	17	22	31
Zink (mg)	17	10	7	9	12	23	27	15	9	7	10	13	18	31	19	10	8	12	16	25	33
Copper (mg)	2	1	1	1	1	3	4	2	1	1	1	1	2	3	2	1	1	1	2	3	4
Iodine (ug)	186	127	67	89	156	241	348	180	112	84	93	151	238	354	193	113	84	104	167	251	362
Selenium (ug)	84	38	46	59	75	100	130	82	34	46	59	75	98	134	86	38	48	57	70	112	138
Cadmium (ug)	10	3	6	8	9	11	13	9	3	6	7	9	11	13	10	3	7	8	9	12	13
Lead (ug)	25	23	9	11	16	34	55	18	12	7	9	15	22	33	16	9	7	10	15	21	29
Mercury (ug)	3	2	1	1	3	4	6	4	6	1	1	2	4	7	2	1	1	1	2	4	4
Caffeine (mg)	48	44	2	14	33	72	114	53	44	3	23	36	93	124	47	44	1	14	37	68	119
Aspartame (mg)	11	32	0	0	0	0	30	19	45	0	0	0	8	96	35	74	0	0	0	45	105

Data is displayed as mean and standard deviation (SD) and percentiles

Figure 1 - Percentage of pregnant women (n=183) reaching the estimated average requirement (AR) and recommended daily intake (RDI) of micronutrients, including both dietary intake and supplements.



# 5 Discussion and future perspectives

The results related to association of maternal dietary patterns of pregnant women in Iceland to GDM risk is discussed in Appendix I, in the manuscript draft named: Association of a maternal dietary pattern derived from pregnant women in Iceland to GDM risks.

## **5.1** Summary of findings

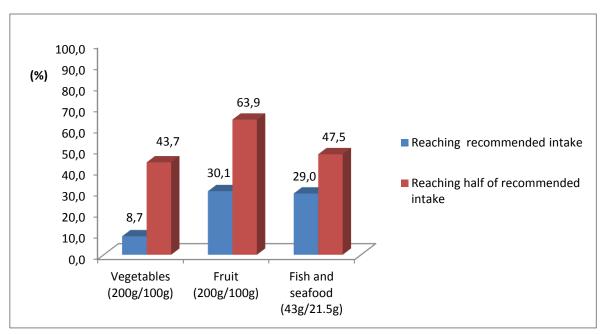
Only one clear dietary pattern was extracted from the cohort and as it closely resembled the Icelandic dietary recommendations it was referred to as a prudent dietary pattern. It appears that when pregnant women in Iceland adhere to this prudent dietary pattern, their risk of GDM is lower, even when adjusting for weight gain in pregnancy and physical activity. This was most evident in the group of overweight/obese women in this study as they are at a much higher risk for GDM than normal weight women. To confirm these results it would be very interesting to conduct a clinical trial based on the results. Viewing dietary intake of the different weight groups revealed a slightly stronger tendency for healthy food choices among the normal weight women, especially when compared to the obese women. Intake for several foods and nutrients that are strongly emphasized as important in the dietary recommendations for pregnant women is lacking for most of the women.

### 5.2 Discussion

When dietary intake was compared between the different weight groups it did not reveal a number of significant results, especially when comparing the overweight- and normal weight women but despite some of the results not being quite significant (P < 0.1) they were deemed as noteworthy due to the fact that both the overweight and obese group consisted of much fewer women and the study might lack statistical power to detect differences in food intake that still might be of relevance. When intake of the overweight – and obese women was added together (n=85) and compared to the normal weight women (n=98) it revealed significant differences in intake of Milk and dairy products (P=0.02), potato chips/popcorn (P=0.01) and poultry (P=0.04) and indicated significant differences (P<0.1) for intake of French fries, vegetable oil, soft drinks and fruit juice. Viewing the differences between the obese women and normal weight women seems to suggest several less healthy dietary choices in the obese group. Their intake of unhealthy foods such as soft drinks and potato chips/popcorn were higher whereas the intake for healthier foods like fruits and vegetable oils was lower. Both the overweight and obese women had a higher intake for milk and dairy products compared to the normal weight group.

Unfortunately an overall view of the women's consumption of foods of special focus in the dietary recommendations [81], is repeatedly demonstrated as inadequate.

**Figure 2**Percentage of pregnant women (n=183) reaching the recommended/half of the recommended intake of vegetables (200g/100g), fruit (200g/100g) and fish and seafood (43g/21.5g).



For example hardly any of the women reach the 200g recommended minimum of vegetable intake. Similarly more than half of the women have fruit intake less than 200g per day. So it is perhaps not so surprising that so few women are able to reach the recommended minimum fiber intake of 25g per day. By eating more fruit and vegetables the women could increase their intake of several vitamins, minerals and other protective phytochemicals in addition to a lower risk of excessive weight gain [178]. According to dietary recommendations, daily consumption of fish should be at least 43g per day which is the equivalent of eating 150g of fish twice a week. At least half of all the women report fish consumption well below recommendation. This is in accordance to previous studies performed in Iceland indicating that maternal consumption of fish and fish oil is not in line with recommendations for a large number of women [12, 13, 16] and that vegetable intake is fairly low [16]. Studies conducted in other countries report similar results. In Poland the maternal intake for fish, Omega-3, fruits and vegetables was not sufficient and the consumption of sweets was fairly high [4]. An English study reported very low maternal intakes for both fruits and vegetables [11] and two separate studies regarding diet in pregnancy in Spain indicated an intake lacking in Omega-3 fatty acids whereas the intake for fruit and vegetables exceeded the recommendations [3, 5].

The overall distribution of macronutrients appears to be fairly similar in all three weight groups. Intake of saturated fats and added sugar is higher than recommended in all three weight groups. According to the new Nordic Nutrition recommendations the intake of the essential Omega-3 acids should represent at least one percent of total energy and thereof 200 mg/day of Docosahexaenoic acid (DHA) for pregnant and lactating women [126]. This is evidently not the case for any of the three weight groups. Not even in the highest percentiles for any of the weight groups, does the total Omega-3 intake represent one percent of total energy. Omega-3 fatty acids are necessary in the early development of the brain and have an essential role in central nervous system functions. Low levels of omega-3 fatty acids have been linked to dysfunctions in brain development and growth impairment [179]. The recommended intake for DHA was not reached by over half of all the women. DHA serves an important role in optimizing maturation of both the brain and retina [180].

This not only indicates that the dietary intake for a large number of pregnant women may be lacking but also that these are the women's dietary habits from before pregnancy. Studies have demonstrated the importance of a healthy pre-pregnancy diet in relation to GDM risk. This stresses the need to implement healthful dietary choices as early as possible as healthful dietary choices are maintained throughout pregnancy [131, 132, 181].

The micronutrient intake for all the participants revealed relatively low intake of some nutrients. When a person is unable to reach the average requirements of a micronutrient it suggests that the person is likely to suffer from deficiency [126]. Since a high percentage of the women are unable to reach the average requirements for vitamin D (36%), iron (22%) and iodine (28%) it would suggest that a number of these women may have a deficiency. These numbers may even be higher since the reference for average requirements used here is aimed at non-pregnant women and should actually be higher for pregnant women. These results are troubling as all these nutrients are of extreme importance in pregnancy, which is clearly emphasized in the dietary recommendations for pregnant women in Iceland [59]. An earlier study in Iceland also reported low vitamin D intakes [16] and results of a Spanish study indicated that a large number of pregnant women did not reach half of the recommended intake for Vitamin D, folate and iron [5]. The signs of iron deficiency are usually well monitored during pregnancy but the same does not apply to Iodine deficiency which if severe may lead to dysfunctions in brain development [182]. Folate and vitamin D are specifically recommended for pregnant women in Iceland but there is obviously a need for more effective means of increasing compliance. Perhaps it is necessary to implement dietary counseling into health- and maternal care in order to achieve this. Some studies have demonstrated such counseling to improve diet choices in pregnancy [183, 184]. It would also prove useful to further analyze the data regarding the intake of vitamins, minerals and other substances from the diet, to see the amount derived from dietary intake and supplements, respectively.

# 6 Conclusions

Pregnant women who are normal weight before pregnancy appear to have somewhat healthier diet choices then pregnant women who are obese before pregnancy. Dietary intake of various foods such as: Fruits, vegetables, fish, fiber and Omega-3, which are specifically recommended in dietary guidelines is severely lacking among pregnant women in Iceland. Adherence to a healthy or prudent dietary pattern may prove beneficial in preventing gestational diabetes mellitus, especially for women already at higher risk due to overweight or obesity before pregnancy. A number of pregnant women may be at risk of deficiency for several vitamins that are vital during pregnancy such as: vitamin D, iodine and iron.

### 7 References

- 1. Williamson, C.S., *Nutrition in pregnancy*. Nutrition Bulletin, 2006. **31**(1): p. 28-59.
- 2. Rao, K.R., I.J. Padmavathi, and M. Raghunath, *Maternal micronutrient restriction programs* the body adiposity, adipocyte function and lipid metabolism in offspring: a review. Rev Endocr Metab Disord, 2012. **13**(2): p. 103-8.
- 3. Rodriguez-Bernal, C.L., et al., *Dietary intake in pregnant women in a Spanish Mediterranean area: as good as it is supposed to be?* Public Health Nutr, 2012: p. 1-11.
- 4. Suliga, E., [Nutritional behaviours of pregnant women]. Pediatr Endocrinol Diabetes Metab, 2011. **17**(2): p. 76-81.
- 5. Ortiz-Andrellucchi, A., et al., [Assessment of nutritional quality in healthy pregnant women of the Canary Islands, Spain]. Med Clin (Barc), 2009. **133**(16): p. 615-21.
- 6. Haugen, M., et al., *Dietary supplements contribute substantially to the total nutrient intake in pregnant Norwegian women.* Ann Nutr Metab, 2008. **52**(4): p. 272-80.
- 7. O'Neill, J.L., et al., *Are women in early pregnancy following the national pyramid recommendations?* Ir Med J, 2011. **104**(9): p. 270-2.
- 8. Fowles, E.R., Comparing pregnant women's nutritional knowledge to their actual dietary intake. MCN Am J Matern Child Nurs, 2002. **27**(3): p. 171-7.
- 9. Giddens, J.B., et al., *Pregnant adolescent and adult women have similarly low intakes of selected nutrients.* J Am Diet Assoc, 2000. **100**(11): p. 1334-40.
- 10. Pick, M.E., et al., Assessment of diet quality in pregnant women using the Healthy Eating Index. J Am Diet Assoc, 2005. **105**(2): p. 240-6.
- 11. Wen, L.M., et al., *Dietary behaviours during pregnancy: findings from first-time mothers in southwest Sydney, Australia.* Int J Behav Nutr Phys Act, 2010. **7**: p. 13.
- 12. Thorsdottir, I., et al., Association of fish and fish liver oil intake in pregnancy with infant size at birth among women of normal weight before pregnancy in a fishing community. Am J Epidemiol, 2004. **160**(5): p. 460-5.
- 13. Olafsdottir, A.S., et al., *Relationship between dietary intake of cod liver oil in early pregnancy and birthweight.* BJOG, 2005. **112**(4): p. 424-9.
- 14. Olafsdottir, A.S., et al., *Maternal diet in early and late pregnancy in relation to weight gain.* Int J Obes (Lond), 2006. **30**(3): p. 492-9.
- 15. Olafsdottir, A.S., et al., Relationship between high consumption of marine fatty acids in early pregnancy and hypertensive disorders in pregnancy. BJOG, 2006. **113**(3): p. 301-9.
- Gústavsdóttir, A.G., Joðhagur barnshafandi kvenna á Íslandi. Fylgni við fæðutengdar ráðleggingar. 2011.
- 17. Ying, H. and D.F. Wang, [Effects of dietary fat on onset of gestational diabetes mellitus]. Zhonghua Fu Chan Ke Za Zhi, 2006. **41**(11): p. 729-31.
- 18. Saldana, T.M., A.M. Siega-Riz, and L.S. Adair, *Effect of macronutrient intake on the development of glucose intolerance during pregnancy.* Am J Clin Nutr, 2004. **79**(3): p. 479-86.
- 19. Jing, X., et al., [Gestational diabetes mellitus and the lifestyle and dietary structure of pregnant women: a case-control study]. Wei Sheng Yan Jiu, 2010. **39**(2): p. 209-11, 227.
- 20. Ley, S.H., et al., Effect of macronutrient intake during the second trimester on glucose metabolism later in pregnancy. Am J Clin Nutr, 2011. **94**(5): p. 1232-40.
- 21. Park, S., et al., Gestational diabetes is associated with high energy and saturated fat intakes and with low plasma visfatin and adiponectin levels independent of prepregnancy BMI. Eur J Clin Nutr, 2013. **67**(2): p. 196-201.
- 22. Metzger, B.E., *Summary and recommendations of the Third International Workshop-Conference on Gestational Diabetes Mellitus*. Diabetes, 1991. **40 Suppl 2**: p. 197-201.
- 23. Reaven, G.M., *Banting lecture 1988. Role of insulin resistance in human disease.* Diabetes, 1988. **37**(12): p. 1595-607.
- 24. Catalano, P.M., *Trying to understand gestational diabetes.* Diabet Med, 2014. **31**(3): p. 273-81.
- 25. World Health Organization. *Diagnostic Criteria and Classification of Hyperglycaemia First Detected in Pregnancy*. 2013: Geneva.
- 26. Di Cianni, G., et al., *Intermediate metabolism in normal pregnancy and in gestational diabetes.* Diabetes Metab Res Rev, 2003. **19**(4): p. 259-70.

- 27. American Diabetes, A., *Standards of medical care in diabetes--2014.* Diabetes Care, 2014. **37 Suppl 1**: p. S14-80.
- 28. Metzger, B.E., et al., *Summary and recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus*. Diabetes Care, 2007. **30 Suppl 2**: p. S251-60.
- 29. Catalano, P.M. and S. Hauguel-De Mouzon, *Is it time to revisit the Pedersen hypothesis in the face of the obesity epidemic?* Am J Obstet Gynecol, 2011. **204**(6): p. 479-87.
- 30. Di Cianni, G., et al., *Maternal triglyceride levels and newborn weight in pregnant women with normal glucose tolerance.* Diabet Med, 2005. **22**(1): p. 21-5.
- 31. Expert Committee on the, D. and M. Classification of Diabetes, *Report of the expert committee* on the diagnosis and classification of diabetes mellitus. Diabetes Care, 2003. **26 Suppl 1**: p. S5-20.
- 32. Turner, R.C., et al., *Insulin deficiency and insulin resistance interaction in diabetes: estimation of their relative contribution by feedback analysis from basal plasma insulin and glucose concentrations.* Metabolism, 1979. **28**(11): p. 1086-96.
- 33. Fadl, H.E., et al., *Maternal and neonatal outcomes and time trends of gestational diabetes mellitus in Sweden from 1991 to 2003.* Diabet Med, 2010. **27**(4): p. 436-41.
- 34. Yogev, Y. and G.H. Visser, *Obesity, gestational diabetes and pregnancy outcome.* Semin Fetal Neonatal Med, 2009. **14**(2): p. 77-84.
- 35. Mitanchez, D., A. Burguet, and U. Simeoni, *Infants Born to Mothers with Gestational Diabetes Mellitus: Mild Neonatal Effects, a Long-term Threat to Global Health.* J Pediatr, 2014. **164**(3): p. 445-50.
- 36. Negrato, C.A., R. Mattar, and M.B. Gomes, *Adverse pregnancy outcomes in women with diabetes*. Diabetol Metab Syndr, 2012. **4**(1): p. 41.
- 37. Buchanan, T.A., A.H. Xiang, and K.A. Page, *Gestational diabetes mellitus: risks and management during and after pregnancy.* Nat Rev Endocrinol, 2012.
- 38. Cheung, N.W. and K. Byth, *Population health significance of gestational diabetes.* Diabetes Care, 2003. **26**(7): p. 2005-9.
- 39. 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.
- 40. Ben-Haroush, A., Y. Yogev, and M. Hod, *Epidemiology of gestational diabetes mellitus and its association with Type 2 diabetes.* Diabet Med, 2004. **21**(2): p. 103-13.
- 41. Matias, S.L., et al., *Maternal prepregnancy obesity and insulin treatment during pregnancy are independently associated with delayed lactogenesis in women with recent gestational diabetes mellitus.* Am J Clin Nutr, 2014. **99**(1): p. 115-21.
- 42. Boden, G., *Fuel metabolism in pregnancy and in gestational diabetes mellitus.* Obstet Gynecol Clin North Am, 1996. **23**(1): p. 1-10.
- 43. Dabelea, D., et al., *Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships.* Diabetes, 2000. **49**(12): p. 2208-11.
- 44. Dabelea, D., W.C. Knowler, and D.J. Pettitt, *Effect of diabetes in pregnancy on offspring:* follow-up research in the Pima Indians. J Matern Fetal Med, 2000. **9**(1): p. 83-8.
- 45. Hillier, T.A., et al., *Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia.* Diabetes Care, 2007. **30**(9): p. 2287-92.
- 46. Bottalico, J.N., *Recurrent gestational diabetes: risk factors, diagnosis, management, and implications.* Semin Perinatol, 2007. **31**(3): p. 176-84.
- 47. Group, H.S.C.R., et al., *Hyperglycemia and adverse pregnancy outcomes.* N Engl J Med, 2008. **358**(19): p. 1991-2002.
- 48. Group, H.S.C.R., *Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study:* associations with neonatal anthropometrics. Diabetes, 2009. **58**(2): p. 453-9.
- 49. Pedersen, J., Weight and length at birth of infants of diabetic mothers. Acta Endocrinol (Copenh), 1954. **16**(4): p. 330-42.
- 50. Catalano, P.M., *Obesity and pregnancy--the propagation of a viscous cycle?* J Clin Endocrinol Metab, 2003. **88**(8): p. 3505-6.
- 51. Cypryk, K., et al., [Overweight and obesity as common risk factors for gestational diabetes mellitus (GDM), perinatal macrosomy in offspring and type-2 diabetes in mothers]. Przegl Lek, 2005. **62**(1): p. 38-41.
- 52. Eliasdottir, O.J., H. Harethardottir, and T. Thornorkelsson, [The effect of maternal weight on pregnancy outcome]. Laeknabladid, 2010. **96**(11): p. 691-6.
- 53. Berkowitz, G.S., et al., *Race/ethnicity and other risk factors for gestational diabetes.* Am J Epidemiol, 1992. **135**(9): p. 965-73.

- 54. American Diabetes, A., *Standards of medical care in diabetes--2009.* Diabetes Care, 2009. **32 Suppl 1**: p. S13-61.
- 55. Oostdam, N., et al., *Interventions for preventing gestational diabetes mellitus: a systematic review and meta-analysis.* J Womens Health (Larchmt), 2011. **20**(10): p. 1551-63.
- 56. Colberg, S.R., K. Castorino, and L. Jovanovic, *Prescribing physical activity to prevent and manage gestational diabetes.* World J Diabetes, 2013. **4**(6): p. 256-262.
- 57. Han, S., P. Middleton, and C.A. Crowther, *Exercise for pregnant women for preventing gestational diabetes mellitus*. Cochrane Database Syst Rev, 2012. **7**: p. CD009021.
- 58. Landlæknir, Ráðleggingar um hreyfingu. 2008.
- 59. Landlæknisembættið, M.m., Lýðheilsustöð og Umhverfisstofnun and n. Matvælastofnun). Matur og meðganga, Fróðleikur fyrir konur á barnseignaraldri. 2006.
- 60. Medicine., I.o., in *Weight Gain During Pregnancy: Reexamining the Guidelines*, K.M. Rasmussen and A.L. Yaktine, Editors. 2009: Washington (DC).
- 61. Hedderson, M.M., E.P. Gunderson, and A. Ferrara, *Gestational weight gain and risk of gestational diabetes mellitus*. Obstet Gynecol, 2010. **115**(3): p. 597-604.
- 62. Liu, Z., et al., Gestational weight gain and risk of gestational diabetes mellitus among Chinese women. Chin Med J (Engl), 2014. **127**(7): p. 1255-60.
- 63. Muktabhant, B., et al., *Interventions for preventing excessive weight gain during pregnancy.* Cochrane Database Syst Rev, 2012. **4**: p. CD007145.
- 64. Gaillard, R., et al., *Risk factors and outcomes of maternal obesity and excessive weight gain during pregnancy.* Obesity (Silver Spring), 2013. **21**(5): p. 1046-55.
- 65. Phelan, S., et al., Reducing excessive gestational weight gain: lessons from the weight control literature and avenues for future research. Womens Health (Lond Engl), 2011. **7**(6): p. 641-61.
- 66. Thorsdottir, I. and B.E. Birgisdottir, *Different weight gain in women of normal weight before pregnancy: postpartum weight and birth weight.* Obstet Gynecol, 1998. **92**(3): p. 377-83.
- 67. Most, O. and O. Langer, *Gestational diabetes: maternal weight gain in relation to fetal growth, treatment modality, BMI and glycemic control.* J Matern Fetal Neonatal Med, 2012. **25**(11): p. 2458-63.
- 68. Forsum, E., et al., Weight loss before conception: A systematic literature review. Food Nutr Res, 2013. **57**.
- 69. Vinter, C.A., et al., [Obese pregnant women and complications in relation to pregnancy and birth]. Ugeskr Laeger, 2012. **174**(16): p. 1079-82.
- 70. Stuebe, A.M., E. Oken, and M.W. Gillman, *Associations of diet and physical activity during pregnancy with risk for excessive gestational weight gain.* Am J Obstet Gynecol, 2009. **201**(1): p. 58 e1-8.
- 71. Weissgerber, T.L., et al., *Exercise in the prevention and treatment of maternal-fetal disease: a review of the literature.* Appl Physiol Nutr Metab, 2006. **31**(6): p. 661-74.
- 72. LIVSMEDELSVERKET, N.f.a.S. *Food for you who are pregnant*. 2014 [cited 2014 03.04.14]; Available from: <a href="http://www.slv.se/en-gb/Group1/Food-and-Nutrition/Dietary-guidelines/Advice-about-food-for-you-who-are-pregnant/">http://www.slv.se/en-gb/Group1/Food-and-Nutrition/Dietary-guidelines/Advice-about-food-for-you-who-are-pregnant/</a>.
- 73. von Ruesten, A., et al., Adherence of pregnant women to Nordic dietary guidelines in relation to postpartum weight retention: results from the Norwegian Mother and Child Cohort Study. BMC Public Health, 2014. **14**: p. 75.
- 74. ernæring., N.r.f. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer 2011 [cited 2014 03.04.14]; Available from: <a href="http://helsedirektoratet.no/publikasjoner/kostrad-for-a-fremme-folkehelsen-og-forebygge-kroniske-sykdommer/Publikasjoner/kostrad-for-a-fremme-folkehelsen-2011.pdf">http://helsedirektoratet.no/publikasjoner/kostrad-for-a-fremme-folkehelsen-og-forebygge-kroniske-sykdommer/Publikasjoner/kostrad-for-a-fremme-folkehelsen-2011.pdf</a>.
- 75. The Danish National Board of health. *HEALTHY HABITS before, during and after PREGNANCY*. 2010 [cited 2014 03.04.14]; Available from: <a href="http://sundhedsstyrelsen.dk/publ/Publ2010/CFF/English/SundeVaner\_en.pdf">http://sundhedsstyrelsen.dk/publ/Publ2010/CFF/English/SundeVaner\_en.pdf</a>.
- 76. choices, N. *Have a healthy diet in pregnancy*. 2013 [cited 2014 03.04.14]; Available from: http://www.nhs.uk/conditions/pregnancy-and-baby/pages/healthy-pregnancy-diet.aspx.
- 77. Services, U.S.D.o.A.a.U.S.D.o.H.a.H. *Dietary Guidelines for Americans*, 2010. 2010 [cited 2014 03.04.14]; Available from: http://www.health.gov/dietaryquidelines/dga2010/DietaryGuidelines2010.pdf.
- 78. Pitkin, R.M., Folate and neural tube defects. Am J Clin Nutr, 2007. **85**(1): p. 285S-288S.
- 79. Djukic, A., Folate-responsive neurologic diseases. Pediatr Neurol, 2007. 37(6): p. 387-97.
- 80. Allen, V.M., et al., *Teratogenicity associated with pre-existing and gestational diabetes.* J Obstet Gynaecol Can, 2007. **29**(11): p. 927-44.

- 81. Landlæknir, *Ráðleggingar um mataræði og næringarefni fyrir fullorðna og börn frá tveggja ára aldri*, Landlæknir, Editor. 2006.
- 82. Jian, M., J. Wang, and H. Sun, [Meta-analysis of effect of intervention with folic acid on neural tube defects]. Wei Sheng Yan Jiu, 2009. **38**(6): p. 682-4.
- 83. Toriello, H.V., Policy, and G. Practice Guideline Committee of the American College of Medical, *Policy statement on folic acid and neural tube defects*. Genet Med, 2011. **13**(6): p. 593-6.
- 84. Imdad, A., M.Y. Yakoob, and Z.A. Bhutta, *The effect of folic acid, protein energy and multiple micronutrient supplements in pregnancy on stillbirths*. BMC Public Health, 2011. **11 Suppl 3**: p. S4.
- 85. De-Regil, L.M., et al., *Effects and safety of periconceptional folate supplementation for preventing birth defects.* Cochrane Database Syst Rev, 2010(10): p. CD007950.
- 86. Blencowe, H., et al., *Folic acid to reduce neonatal mortality from neural tube disorders*. Int J Epidemiol, 2010. **39 Suppl 1**: p. i110-21.
- 87. Molina-Solana, R., et al., Current concepts on the effect of environmental factors on cleft lip and palate. Int J Oral Maxillofac Surg, 2013. **42**(2): p. 177-84.
- 88. Badovinac, R.L., et al., Folic acid-containing supplement consumption during pregnancy and risk for oral clefts: a meta-analysis. Birth Defects Res A Clin Mol Teratol, 2007. **79**(1): p. 8-15.
- 89. Goh, Y.I., et al., *Prenatal multivitamin supplementation and rates of congenital anomalies: a meta-analysis.* J Obstet Gynaecol Can, 2006. **28**(8): p. 680-9.
- 90. Johnson, C.Y. and J. Little, *Folate intake, markers of folate status and oral clefts: is the evidence converging?* Int J Epidemiol, 2008. **37**(5): p. 1041-58.
- 91. Morse, N.L., Benefits of docosahexaenoic acid, folic acid, vitamin D and iodine on foetal and infant brain development and function following maternal supplementation during pregnancy and lactation. Nutrients, 2012. **4**(7): p. 799-840.
- 92. Czeizel, A.E., *Periconceptional folic acid-containing multivitamin supplementation for the prevention of neural tube defects and cardiovascular malformations.* Ann Nutr Metab, 2011. **59**(1): p. 38-40.
- 93. Fekete, K., et al., Effect of folate intake on health outcomes in pregnancy: a systematic review and meta-analysis on birth weight, placental weight and length of gestation. Nutr J, 2012. **11**: p. 75.
- 94. Lassi, Z.S., et al., *Folic acid supplementation during pregnancy for maternal health and pregnancy outcomes.* Cochrane Database Syst Rev, 2013. **3**: p. CD006896.
- 95. Yi, Y., et al., *Economic burden of neural tube defects and impact of prevention with folic acid: a literature review.* Eur J Pediatr, 2011. **170**(11): p. 1391-400.
- 96. Botto, L.D., et al., *International retrospective cohort study of neural tube defects in relation to folic acid recommendations: are the recommendations working?* BMJ, 2005. **330**(7491): p. 571.
- 97. Kloosterman, J., et al., [Folic acid fortification: prevention as well as promotion of cancer]. Ned Tijdschr Geneeskd, 2006. **150**(26): p. 1443-8.
- 98. Herrmann, W. and R. Obeid, *The mandatory fortification of staple foods with folic acid: a current controversy in Germany.* Dtsch Arztebl Int, 2011. **108**(15): p. 249-54.
- 99. Haider, B.A., M.Y. Yakoob, and Z.A. Bhutta, *Effect of multiple micronutrient supplementation during pregnancy on maternal and birth outcomes.* BMC Public Health, 2011. **11 Suppl 3**: p. S19.
- 100. Shah, P.S., et al., Effects of prenatal multimicronutrient supplementation on pregnancy outcomes: a meta-analysis. CMAJ, 2009. **180**(12): p. E99-108.
- 101. Bhutta, Z.A., et al., *Is it time to replace iron folate supplements in pregnancy with multiple micronutrients?* Paediatr Perinat Epidemiol, 2012. **26 Suppl 1**: p. 27-35.
- Haider, B.A. and Z.A. Bhutta, *Multiple-micronutrient supplementation for women during pregnancy*. Cochrane Database Syst Rev, 2012. **11**: p. CD004905.
- Dawodu, A. and R.C. Tsang, *Maternal vitamin D status: effect on milk vitamin D content and vitamin D status of breastfeeding infants*. Adv Nutr. 2012. **3**(3): p. 353-61.
- 104. Senti, J., D.K. Thiele, and C.M. Anderson, *Maternal vitamin D status as a critical determinant in gestational diabetes.* J Obstet Gynecol Neonatal Nurs, 2012. **41**(3): p. 328-38.
- 105. Lau, S.L., et al., Serum 25-hydroxyvitamin D and glycated haemoglobin levels in women with gestational diabetes mellitus. Med J Aust, 2011. **194**(7): p. 334-7.
- 106. Wagner, C.L., et al., *Vitamin D and its role during pregnancy in attaining optimal health of mother and fetus.* Nutrients, 2012. **4**(3): p. 208-30.

- 107. Thorne-Lyman, A. and W.W. Fawzi, *Vitamin D during pregnancy and maternal, neonatal and infant health outcomes: a systematic review and meta-analysis.* Paediatr Perinat Epidemiol, 2012. **26 Suppl 1**: p. 75-90.
- 108. Schroth, R.J., C.L. Lavelle, and M.E. Moffatt, *Review of vitamin D deficiency during pregnancy: who is affected?* Int J Circumpolar Health, 2005. **64**(2): p. 112-20.
- 109. Landlæknir. Ráðlagðir dagskammtar af ýmsum vítamínum 2013 [cited 2014 06.04]; Available from: <a href="http://www.landlaeknir.is/servlet/file/store93/item21457/Radlagdir\_dagskammtar\_tafla\_2013.p">http://www.landlaeknir.is/servlet/file/store93/item21457/Radlagdir\_dagskammtar\_tafla\_2013.p</a> df.
- 110. Winzenberg, T. and G. Jones, *Vitamin D and bone health in childhood and adolescence*. Calcif Tissue Int, 2013. **92**(2): p. 140-50.
- 111. Wagner, C.L., et al., *A randomized trial of vitamin D supplementation in 2 community health center networks in South Carolina*. Am J Obstet Gynecol, 2013. **208**(2): p. 137 e1-13.
- 112. Hollis, B.W. and C.L. Wagner, *Vitamin D and pregnancy: skeletal effects, nonskeletal effects, and birth outcomes.* Calcif Tissue Int, 2013. **92**(2): p. 128-39.
- 113. Alzaim, M. and R.J. Wood, *Vitamin D and gestational diabetes mellitus*. Nutr Rev, 2013. **71**(3): p. 158-67.
- 114. Poel, Y.H., et al., *Vitamin D and gestational diabetes: a systematic review and meta-analysis.* Eur J Intern Med, 2012. **23**(5): p. 465-9.
- 115. Aghajafari, F., et al., Association between maternal serum 25-hydroxyvitamin D level and pregnancy and neonatal outcomes: systematic review and meta-analysis of observational studies. BMJ, 2013. **346**: p. f1169.
- 116. Roth, D.E., *Vitamin D supplementation during pregnancy:* safety considerations in the design and interpretation of clinical trials. J Perinatol, 2011. **31**(7): p. 449-59.
- 117. Hollams, E.M., *Vitamin D and atopy and asthma phenotypes in children.* Curr Opin Allergy Clin Immunol, 2012. **12**(3): p. 228-34.
- 118. Christesen, H.T., et al., *The impact of vitamin D on pregnancy: a systematic review.* Acta Obstet Gynecol Scand, 2012. **91**(12): p. 1357-67.
- 119. Nassar, N., et al., *Systematic review of first-trimester vitamin D normative levels and outcomes of pregnancy.* Am J Obstet Gynecol, 2011. **205**(3): p. 208 e1-7.
- 120. Rosen, C.J., et al., *The nonskeletal effects of vitamin D: an Endocrine Society scientific statement.* Endocr Rev, 2012. **33**(3): p. 456-92.
- 121. Weisse, K., et al., *Maternal and newborn vitamin D status and its impact on food allergy development in the German LINA cohort study.* Allergy, 2013. **68**(2): p. 220-8.
- 122. Paxton, G.A., et al., *Vitamin D and health in pregnancy, infants, children and adolescents in Australia and New Zealand: a position statement.* Med J Aust, 2013. **198**(3): p. 142-3.
- 123. Urrutia, R.P. and J.M. Thorp, *Vitamin D in pregnancy: current concepts.* Curr Opin Obstet Gynecol, 2012. **24**(2): p. 57-64.
- 124. De-Regil, L.M., et al., *Vitamin D supplementation for women during pregnancy.* Cochrane Database Syst Rev, 2012. **2**: p. CD008873.
- 125. Kant, A.K., *Dietary patterns: biomarkers and chronic disease risk.* Appl Physiol Nutr Metab, 2010. **35**(2): p. 199-206.
- 126. Nordic Council of Ministers, Nordic Nutrition Recommendations 2012. 2014.
- 127. Fazio Ede, S., et al., [Dietary intake of pregnant women and maternal weight gain after nutritional counseling]. Rev Bras Ginecol Obstet, 2011. **33**(2): p. 87-92.
- 128. Laraia, B.A., L.M. Bodnar, and A.M. Siega-Riz, *Pregravid body mass index is negatively associated with diet quality during pregnancy.* Public Health Nutr, 2007. **10**(9): p. 920-6.
- 129. Tsigga, M., et al., *Healthy Eating Index during pregnancy according to pre-gravid and gravid weight status*. Public Health Nutr, 2011. **14**(2): p. 290-6.
- 130. Guelinckx, I., R. Devlieger, and G. Vansant, *Pregnancies complicated by obesity: clinical approach and nutritional management.* Verh K Acad Geneeskd Belg, 2010. **72**(5-6): p. 253-76.
- 131. Tobias, D.K., et al., *Prepregnancy adherence to dietary patterns and lower risk of gestational diabetes mellitus*. Am J Clin Nutr, 2012. **96**(2): p. 289-95.
- Thang, C., et al., *Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus.* Diabetes Care, 2006. **29**(10): p. 2223-30.
- 133. Chen, L., et al., *Prospective study of pre-gravid sugar-sweetened beverage consumption and the risk of gestational diabetes mellitus.* Diabetes Care, 2009. **32**(12): p. 2236-41.
- 134. Bo, S., et al., Dietary fat and gestational hyperglycaemia. Diabetologia, 2001. 44(8): p. 972-8.
- 135. Burris, H.H., et al., *Vitamin D deficiency in pregnancy and gestational diabetes mellitus.* Am J Obstet Gynecol, 2012. **207**(3): p. 182 e1-8.

- 136. Zhang, C., et al., *Maternal plasma 25-hydroxyvitamin D concentrations and the risk for gestational diabetes mellitus*. PLoS One, 2008. **3**(11): p. e3753.
- 137. Baker, A.M., et al., First-trimester maternal vitamin D status and risk for gestational diabetes (GDM) a nested case-control study. Diabetes Metab Res Rev, 2012. **28**(2): p. 164-8.
- 138. Perez-Ferre, N., et al., Association of low serum 25-Hydroxyvitamin D levels in pregnancy with glucose homeostasis and obstetric and newborn outcomes. Endocr Pract, 2012: p. 1-18.
- 139. McLeod, D.S., et al., Associations of serum vitamin D concentrations with obstetric glucose metabolism in a subset of the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study cohort. Diabet Med, 2012. **29**(8): p. e199-204.
- Tobias, D.K., et al., *Healthful Dietary Patterns and Type 2 Diabetes Mellitus Risk Among Women With a History of Gestational Diabetes Mellitus*. Arch Intern Med, 2012: p. 1-7.
- 141. Chasan-Taber, L., *Physical activity and dietary behaviors associated with weight gain and impaired glucose tolerance among pregnant Latinas*. Adv Nutr, 2012. **3**(1): p. 108-18.
- 142. Morisset, A.S., et al., *Prevention of gestational diabetes mellitus: a review of studies on weight management.* Diabetes Metab Res Rev, 2010. **26**(1): p. 17-25.
- 143. Karamanos, B., et al., *Relation of the Mediterranean diet with the incidence of gestational diabetes*. Eur J Clin Nutr, 2014. **68**(1): p. 8-13.
- 144. Asemi, Z., et al., A randomized controlled clinical trial investigating the effect of DASH diet on insulin resistance, inflammation, and oxidative stress in gestational diabetes. Nutrition, 2013. **29**(4): p. 619-24.
- 145. Asemi, Z., et al., Favourable effects of the Dietary Approaches to Stop Hypertension diet on glucose tolerance and lipid profiles in gestational diabetes: a randomised clinical trial. Br J Nutr, 2013. **109**(11): p. 2024-30.
- 2 Zhang, C., et al., A prospective study of dietary patterns, meat intake and the risk of gestational diabetes mellitus. Diabetologia, 2006. **49**(11): p. 2604-13.
- 147. Brantsaeter, A.L., et al., A dietary pattern characterized by high intake of vegetables, fruits, and vegetable oils is associated with reduced risk of preeclampsia in nulliparous pregnant Norwegian women. J Nutr, 2009. **139**(6): p. 1162-8.
- 148. Haugen, M., et al., *Mediterranean-type diet and risk of preterm birth among women in the Norwegian Mother and Child Cohort Study (MoBa): a prospective cohort study.* Acta Obstet Gynecol Scand, 2008. **87**(3): p. 319-24.
- 149. Englund-Ogge, L., et al., *Maternal dietary patterns and preterm delivery: results from large prospective cohort study.* BMJ, 2014. **348**: p. g1446.
- 150. Damon, S., et al., *Nutrition and diabetes mellitus: an overview of the current evidence.* Wien Med Wochenschr, 2011. **161**(11-12): p. 282-8.
- 151. Slavin, J.L., *Position of the American Dietetic Association: health implications of dietary fiber.* J Am Diet Assoc, 2008. **108**(10): p. 1716-31.
- 152. Overby, N.C., et al., Dietary fiber and the glycemic index: a background paper for the Nordic Nutrition Recommendations 2012. Food Nutr Res, 2013. **57**.
- 153. Sartorelli, D.S. and M.A. Cardoso, [Association between dietary carbohydrates and type 2 diabetes mellitus: epidemiological evidence]. Arq Bras Endocrinol Metabol, 2006. **50**(3): p. 415-26.
- 154. Giacosa, A. and M. Rondanelli, *The right fiber for the right disease: an update on the psyllium seed husk and the metabolic syndrome.* J Clin Gastroenterol, 2010. **44 Suppl 1**: p. S58-60.
- 155. Ye, E.Q., et al., *Greater whole-grain intake is associated with lower risk of type 2 diabetes, cardiovascular disease, and weight gain.* J Nutr, 2012. **142**(7): p. 1304-13.
- 156. Slavin, J., Why whole grains are protective: biological mechanisms. Proc Nutr Soc, 2003. **62**(1): p. 129-34.
- 157. Louie, J.C., J.C. Brand-Miller, and R.G. Moses, *Carbohydrates, glycemic index, and pregnancy outcomes in gestational diabetes*. Curr Diab Rep, 2013. **13**(1): p. 6-11.
- 158. Mann, J.I., et al., Evidence-based nutritional approaches to the treatment and prevention of diabetes mellitus. Nutr Metab Cardiovasc Dis, 2004. **14**(6): p. 373-94.
- 159. Moses, R.G., et al., Can a low-glycemic index diet reduce the need for insulin in gestational diabetes mellitus? A randomized trial. Diabetes Care, 2009. **32**(6): p. 996-1000.
- 160. Landspítali, T.N.U.h.i.l. Clinical guidelines for Gestational diabetes mellitus. . 2012.
- 161. Sievenpiper, J.L., et al., Effect of non-oil-seed pulses on glycaemic control: a systematic review and meta-analysis of randomised controlled experimental trials in people with and without diabetes. Diabetologia, 2009. **52**(8): p. 1479-95.

- 162. Nettleton, J.A., et al., Interactions of dietary whole-grain intake with fasting glucose- and insulin-related genetic loci in individuals of European descent: a meta-analysis of 14 cohort studies. Diabetes Care, 2010. **33**(12): p. 2684-91.
- 163. Murakami, K., H. Okubo, and S. Sasaki, *Effect of dietary factors on incidence of type 2 diabetes: a systematic review of cohort studies.* J Nutr Sci Vitaminol (Tokyo), 2005. **51**(4): p. 292-310.
- 164. Imam, M.U., et al., *Antidiabetic properties of germinated brown rice: a systematic review.* Evid Based Complement Alternat Med, 2012. **2012**: p. 816501.
- 165. Priebe, M.G., et al., *Whole grain foods for the prevention of type 2 diabetes mellitus.* Cochrane Database Syst Rev, 2008(1): p. CD006061.
- Blumfield, M.L., et al., Systematic review and meta-analysis of energy and macronutrient intakes during pregnancy in developed countries. Nutr Rev, 2012. **70**(6): p. 322-36.
- 167. Tieu, J., C.A. Crowther, and P. Middleton, *Dietary advice in pregnancy for preventing gestational diabetes mellitus*. Cochrane Database Syst Rev, 2008(2): p. CD006674.
- 168. Cheung, N.W., *The management of gestational diabetes.* Vasc Health Risk Manag, 2009. **5**(1): p. 153-64.
- 169. Crowther, C.A., et al., *Effect of treatment of gestational diabetes mellitus on pregnancy outcomes.* N Engl J Med, 2005. **352**(24): p. 2477-86.
- 170. Landon, M.B., et al., *A multicenter, randomized trial of treatment for mild gestational diabetes.* N Engl J Med, 2009. **361**(14): p. 1339-48.
- 171. Thomaz de Lima, H., et al., Systematic review; Nutritional therapy in gestational diabetes mellitus. Nutr Hosp, 2013. **28**(6): p. 1806-14.
- 172. Lapolla, A., et al., Gestational diabetes mellitus in Italy: a multicenter study. Eur J Obstet Gynecol Reprod Biol, 2009. **145**(2): p. 149-53.
- 173. Langer, O., et al., *Gestational diabetes: the consequences of not treating.* Am J Obstet Gynecol, 2005. **192**(4): p. 989-97.
- 174. Haines, P.S., et al., Weekend eating in the United States is linked with greater energy, fat, and alcohol intake. Obes Res, 2003. **11**(8): p. 945-9.
- 175. Jackson, K.A., et al., *Minimizing random error in dietary intakes assessed by 24-h recall, in overweight and obese adults.* Eur J Clin Nutr, 2008. **62**(4): p. 537-43.
- 176. Landlæknisembættið. [cited 2014 09.05]; Available from: http://www.landlaeknir.is/.
- 177. Matis. Available from: <a href="http://www.matis.is/thjonusta/naeringargildi-matvaela-isgem/">http://www.matis.is/thjonusta/naeringargildi-matvaela-isgem/</a>.
- 178. Streuling, I., et al., Weight gain and dietary intake during pregnancy in industrialized countries-a systematic review of observational studies. J Perinat Med, 2011. **39**(2): p. 123-9.
- 179. Rombaldi Bernardi, J., et al., *Fetal and neonatal levels of omega-3: effects on neurodevelopment, nutrition, and growth.* ScientificWorldJournal, 2012. **2012**: p. 202473.
- 180. Rogers, L.K., C.J. Valentine, and S.A. Keim, *DHA supplementation: current implications in pregnancy and childhood.* Pharmacol Res, 2013. **70**(1): p. 13-9.
- 181. Crozier, S.R., et al., *Women's dietary patterns change little from before to during pregnancy.* J Nutr, 2009. **139**(10): p. 1956-63.
- 182. Pearce, E.N., *Monitoring and effects of iodine deficiency in pregnancy: still an unsolved problem?* Eur J Clin Nutr, 2013. **67**(5): p. 481-4.
- 183. Kinnunen, T.I., et al., Effects of dietary counselling on food habits and dietary intake of Finnish pregnant women at increased risk for gestational diabetes a secondary analysis of a cluster-randomized controlled trial. Matern Child Nutr, 2012.
- 184. Thangaratinam, S., et al., Effects of interventions in pregnancy on maternal weight and obstetric outcomes: meta-analysis of randomised evidence. BMJ, 2012. **344**: p. e2088.

8 Appendices

Appendix I – Manuscript draft 8.1

Association of a maternal dietary pattern derived from pregnant women in Iceland to

GDM risks.

Author(s): Tryggvadottir EA<sup>1</sup>, Medek H<sup>2</sup>, Birgisdottir BE<sup>1</sup>, Geirsson RT<sup>2</sup>, Gunnarsdottir I<sup>1</sup>

Affiliation(s): Unit for Nutrition Research, National University Hospital and Faculty of Food

Science and Nutrition, University of Iceland<sup>1</sup>. Department of Obstetrics and Gynecology,

Women's Clinic, National University Hospital, Reykjavik<sup>2</sup>.

Corresponding author:

Ellen Alma Tryggvadóttir

Unit for Nutrition Research, National University Hospital and Faculty of Food Science and

Nutrition, University of Iceland. Eiriksgata 28, 101 Reykjavik. Iceland. Tel: +354-690-1110

Key words: Diet, Dietary pattern, Pregnancy, Gestational diabetes mellitus

46

#### **Abstract**

**Background:** Gestational diabetes mellitus (GDM) is associated with negative health effects for both the mother and child.

**Objective:** To investigate the association between maternal dietary pattern and GDM.

**Methods:** A prospective observational study including 168 pregnant Icelandic women aged 18-40 years. These were recruited at routine 20 week ultrasound at Landspitali/National-University Hospital in Iceland. All participants kept a four day weighed food record as soon as possible following recruitment (gestational weeks 19 - 24). Food data was recorded into the ICEFOOD calculating program based on the Icelandic food database (ISGEM). Principal component analysis was used to extract dietary patterns from 29 food groups and a healthy eating index was constructed. All women underwent an oral glucose tolerance test in weeks 23 - 28.

**Results:** One clear dietary pattern (eigenvalue 2.4) was extracted comprising of seafood; eggs; vegetables; fruits and berries; vegetable oils; nuts and seeds; pasta; breakfast cereals; coffee and tea with a negative correlation to intake of soft drinks and French fries. Variance explained was 8.2%. The prevalence of GDM was 2.3% among women of normal weight before pregnancy and 18.3% among overweight/obese women. The pattern was associated with lower risk of GDM (OR: 0.54 95% CI: 0.30, 0.98). When adjusting for age, parity, prepregnancy weight, energy intake, weekly weight gain and total Metabolic Equivalent of Task (MET) the association remained (OR: 0.36 95% CI: 0.14, 0.94).

**Conclusions:** Adhering to a prudent dietary pattern in pregnancy may prove beneficial in preventing GDM, especially among women already at higher risk due to overweight/obesity before pregnancy.

### Introduction

The number of women diagnosed with gestational diabetes mellitus (GDM) continues to grow worldwide. Many negative health aspects have been associated with GDM for both mother and child. Having GDM has been associated with a greater risk of diabetes later in life [1-4], miscarriage, hypertension, pre-eclampsia and delivering very large infants. This can in turn lead to a higher risk of prematurity, trauma at birth, caesareans, and shoulder dystocia [2, 5-8]. Infants born to women with GDM are at a higher risk of various malformations [5], growth restriction during gestation, hypoglycemia following the lack of glucose supply through the umbilical cord after delivery [6] as well as diabetes 2 later in life [2, 9-13]. It is well known that women who are overweight and especially obese before pregnancy have a greater risk of being diagnosed with GDM than women of normal weight [4, 14, 15]. Eating a healthy diet, exercise and adhering to the recommended gestational weight gain should be an emphasis for women at risk of GDM [16]. Some studies credit certain factors of the diet to be directly associated with the risk of GDM such as higher consumption of soft drinks [17], increased consumption of energy [18], fat especially saturated fat [19] and decreased consumption of polyunsaturated fat and carbohydrates [18, 20-23]. However, there is still a lack of convincing evidence demonstrating what type of diet might be most effective in preventing GDM [24]. Recently there has been a greater focus on investigating the combined effect of various foods on health and health related factors instead of isolated foods or nutrients, for example by using dietary patterns or healthy eating index [25, 26]. There are few existing studies that have investigated the relationship between dietary patterns and the The aim of this study was to investigate the association between maternal risk of GDM. dietary patterns, using both principal component analysis and a healthy eating index, and GDM.

#### **Methods**

The data for this study was gathered in co-operation with a separate study called Get diabetics moving (GDM-study), which was conducted at the National University Hospital in Iceland. The aim of the GDM-study was to study the impact of increased physical activity on blood sugar levels, body weight, metabolism and oxygen transfer to the fetus for women with GDM. All study participants underwent a 2 hour, 75 gram oral glucose tolerance test (OGTT) between 23 - 28 weeks of gestation. All participants were required to keep a 4 day weighed

food record either from Wednesday - Saturday or Saturday - Tuesday as soon as possible following recruitment in week 19 - 24.

## **Participants**

Participants were recruited over a period of 18 months from April 2012 - October 2013 at a routine 20 week ultrasound with the help of staff at the Pre-natal diagnosis department at the National University Hospital. Initially the criteria's for participation were: Icelandic women living in Reykjavik with age between 18 - 40 years, first - third pregnancy, non-smoker, no reported family history of diabetes or GDM and a BMI between 18.5-24.9 kg/m<sup>2</sup> (normal weight) or 30 - <40 kg/m<sup>2</sup> (obese). After six months of recruiting the criteria were altered to include women with a BMI of 25-29.9 kg/m<sup>2</sup> (overweight) and to include women with a family history of diabetes. This alteration was made as much fewer obese women were being recruited than normal weight women. A total of 217 women were recruited within the study period, 56 women declined participation (participation rate 79%). A total of 168 women (86 normal weight, 44 overweight and 38 obese) returned food records and additionally underwent the OGTT. All participants answered the International Physical Activity Questionnaire (IPAQ) from which The Metabolic Equivalent of Task (MET) was estimated. Pre-pregnancy weight was self-reported. Weight was recorded at recruitment in gestation week 19 - 24 and again at a fetal Echocardiography at 31 - 38 weeks. Each participant's weight gain is reported as weekly weight gain, due to the variance of recorded weight. To obtain weekly weight gain, the differences between the two recorded weights were calculated and divided by number of weeks passed in each case. Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) is a calculation method used to assess levels of insulin resistance by using results of fasting insulin (I<sub>0</sub>) and fasting glucose (G<sub>0</sub>) measurements calculated as (I<sub>0</sub> x G<sub>0</sub>)/22,5 [27]. Recent guidelines from the World Health Organization (WHO) were used to determine the rate of GDM in the group at 23 - 28 weeks of gestation: Fasting plasma glucose between 5.1 and 6.9 mmol/l, the one hour plasma glucose measured  $\geq$ 10.0 mmol/l or the two hour plasma glucose between 8.5 and 11.0 mmol/l after a 75 gram oral glucose tolerance test. If one, two or all of these criteria were met the woman was diagnosed with GDM [28].

### Statistical data and analysis

The food data was recorded into the ICEFOOD calculating program that is based on values from the Icelandic food and nutrient database ISGEM [29]. The average food intake was calculated for each participant divided into 18 main food groups which consist of subgroups as defined in ISGEM [29]. Average intake of energy and nutrients including supplements was

also calculated. The dietary pattern was extracted from 29 food groups. Thereof were 11 of the main food groups: Milk and dairy products; cheese; ice cream; meat and meat products; fish/fish products and shellfish; poultry; eggs; potato chips and popcorn; sauces; soups and bread salads; pre-prepared foods; sugar/honey and candy. The subgroups of six of the main food groups were utilized to construct the remaining food groups. Various types of fats were divided into three groups: solid fats; vegetable oils; fish oil. Drinks were divided into three groups: coffee/tea and cocoa powder; soft drinks and sports drinks; pure fruit juices. Vegetables were split into three groups: all vegetables; potatoes; French fries. Fruit subgroups were divided into two groups: nuts and seeds; fruit, berries and jams. Grains were divided into five groups: Grains; breakfast cereals; bread and crisp bread; cookies and cakes; pasta and couscous. The two subgroups of supplement foods were used: diet- and protein shakes; vitamin and mineral supplements.

The dietary pattern was extracted by using Principal component analysis with the orthogonal rotation Varimax with Kaiser Normalization. The suitability of our data was tested with the Bartlett's test of Sphericity and Kaiser-Meyer-Olkin measure of sampling adequacy. The Bartlett test of Sphericity was significant (P < 0.01). However, the Kaiser Mayer Olkin test was 0.5 which is borderline acceptable. To support our findings we determined the highest adherence to the prudent dietary pattern by dividing the associated extracted factor into tertiles where the highest intake for each factor (lowest for the two negative factors) scored highest. Those who ranked in the highest tertile for all factors combined were determined as having the highest total adherence. Furthermore, the same method of tertiles was used to determine best adherence to a healthy eating index using the food based dietary guidelines from the Icelandic Directorate of Health as criteria. The index included fish and seafood; vegetables; fruits; vegetable oils; nuts and seeds; unground/wholeground cereals (i.e. bran, germ, oats, rice and corn); vitamin D intake and soft drinks. The Kolmogorov–Smirnov test was used to test all data for normality. Data is presented as means and standard deviations and also as median and Interquartile range when appropriate.

Differences in maternal characteristics over the three groups of BMI before pregnancy were tested with the Kruskall-Wallis test. The relationship between the results of the pattern analysis and GDM diagnosis was determined using logistic regression. The Chi-square test was used to test significance when comparing the number of GDM diagnoses in groups with highest vs. lower adherence to the prudent dietary pattern or healthy eating index. The association of maternal characteristics to the prudent dietary pattern adhering scores, fasting

glucose and HOMA-IR were tested with Mann Whitney U test in the case of parity and Kruskall-Wallis test in other cases. The variance inflation factor (VIF) used to detect colinearity for factors used for adjustment reported no values > 4. All statistical analysis was performed in IBM SPSS Statistic version 20. Significance level was set at P = 0.05.

#### **Results**

Table 1 shows the maternal characteristics. Women who were overweight or obese were on average a year older than normal weight women (29 vs. 30 years) (P=0.01). Obese women had gained less weight at recruiting in weeks 19 - 24 of pregnancy than overweight women or normal weight women, 2.7, 5.5 and 4.6 kg, respectively (P=0.02). The difference in weekly weight gain between weeks 19 - 24 and 31 - 38 was also significantly different between the groups where the normal weight women gained more weight than overweight and obese women (P<0.01). The prevalence of GDM for women of normal weight before pregnancy was 2.3% and among overweight or obese women it was 18.3%. None of the women had glucose levels above the GDM criteria which would indicate diabetes mellitus in pregnancy.

In the pattern analysis one clear dietary pattern was extracted (eigenvalue 2.4) (Table 2) positive for seafood, eggs, vegetables, fruit and berries, vegetable oils, nuts and seeds, pasta, breakfast cereals, coffee and tea and negative for soft drinks and French fries. Variance explained was 8.2%. This pattern was labeled as a "Prudent pattern". The other extracted pattern had eigenvalue < 2 explained variance was 6.6% and was not as clearly defined. Furthermore it did not demonstrate associations to the outcome under investigation. Adherence to this prudent dietary pattern varied somewhat when divided by maternal characteristics (Table 3). Adherence increased significantly for women in the oldest tertile (P=0.03) as did fasting glucose levels (P=0.01). Fasting glucose and HOMA-IR levels were significantly higher with increasing pre-pregnancy BMI (P<0.01 and P<0.01).

The prudent dietary pattern was associated with a significantly decreased risk of GDM (Table 4). That difference was still present after adjusting the model for: age, parity, prepregnancy weight, energy intake, weekly weight gain and total MET. The final model demonstrated no significant results regarding total MET. However the analysis demonstrated an independent association between weekly weight gain and GDM-risk (OR: 0.02 95% CI: 0.00, 0.54).

We split the participants into a normal weight group (n=86/GDM:n=2) and overweight/obese group (n=82/GDM:n=15). As cases in the normal weight group were too few, no further analysis was made. When performing the same analysis for the overweight/obese group the

significant association to the decreased risk of GDM remained (Table 4). Analyzing the effects of each dietary factor separately demonstrated no significant associations with GDM except in the case of seafood consumption and additionally pasta in the overweight/obese group. The related odds ratio for those factors was much higher than observed in the logistic regression for the prudent dietary pattern.

When all participants were divided into tertiles depending on adherence to the prudent dietary pattern HOMA-IR values were lower in the tertile with the highest adherence although of borderline significance (P=0.054). When further split up into normal weight and overweight/obese groups the lower HOMA-IR values were significant only for the overweight/obese group (P < 0.01) (Table 5).

Comparing the number of women diagnosed with GDM in the group with the highest adherence to the extracted prudent pattern to the group with lower adherence, for all women and overweight/obese women respectively is shown in Table 6. The results for all the women demonstrated a diagnosis rate of 1.8% for the women with the highest adherence and 14.3% for the women with the lower adherence (P=0.01). The overweight/obese group demonstrated a GDM diagnosis rate of 3.7% for women with the highest adherence to the extracted pattern and 25.5% for women with lower adherence (P=0.02).

We repeated the same analysis for adherence to a healthy eating index based on dietary guidelines and compared the group with the highest adherence to the group with lower adherence for all women and the overweight/obese women respectively. The rate of GDM diagnosis was 3.6% for the women with the highest adherence and 13.4% for those with a lower adherence (P=0.05). The results for the overweight/obese women demonstrated a 3.8% rate of diagnosis for the women with the highest adherence and 25% for women with lower adherence (P=0.02).

### **Discussion**

In our present study we found that adhering to a prudent dietary pattern as well as scoring high on a healthy eating index is associated with a decreased risk for GDM. This was especially true for women who were either overweight or obese before pregnancy, but the rate of GDM diagnosis is several times higher in this group. Similar results have been found previously in an observational study including 1076 women in ten countries where adherence to a Mediterranean dietary pattern was associated with better glucose tolerance and decreased incidence of GDM [30]. Another study based on results from the Nurses' Health Study II indicated that a pre-pregnancy adherence to the Dietary Approach to Stop Hypertension (DASH), a Mediterranean- or Healthy Eating Index (HEI) diet was associated with a lower GDM risk. The strongest relationship was seen for the HEI diet which like the other two patterns includes higher intake of vegetables, fruits, nuts and legumes and additionally cereal fiber and polyunsaturated fats [31]. This suggests that adherence to these dietary patterns continued throughout pregnancy [32]. A prospective study by Chen et al examined the effects of a prudent diet that included vegetables, fruit, fish and poultry and a westernized diet, which includes high intake of red and processed meat, pizza, French fries, candy and refined grains. They discovered an association between the prudent dietary pattern and a decreased risk for GDM as well as an increased risk for GDM associated with the westernized pattern [33]. A randomized control trial also demonstrated the benefits of a healthy diet for women already diagnosed with GDM, where eating according to a DASH diet, which consists of plenty of fruits and vegetables, whole grains and low-fat dairy products, led to a lower number of women needing insulin treatment, fewer caesarians and better pregnancy outcomes [34]. Even though these studies all seem to highlight the combined benefits of healthy foods on GDM risks they are still relatively few and results need to be verified with more intervention studies.

Although our extracted prudent dietary pattern contained pasta which is not necessarily associated with a healthy dietary pattern, it may possibly be explained by the fact that it is a popular food and often consumed by most women in the study. Furthermore most types of pasta are a low glycemic index food (below 55) and pasta including vegetables can also easily be a part of a healthy diet [35, 36]. The fact that adherence to the prudent dietary pattern increased with age points to the possibility that the women become more health conscious with increasing age.

The results for the association between the different food groups included in the pattern indicate that the benefit of the dietary factors combined is stronger than demonstrated by the

isolated foods. In this study only two of the 86 women of normal weight before pregnancy were diagnosed with GDM, bedding for low power of any analysis including GDM. Therefore, even though the association demonstrated between the prudent dietary pattern and GDM appeared to be more apparent for the overweight/obese group it is possible that the results would be more similar in a larger group of normal weight women [31]. Weight gain in pregnancy also appears to be an obvious risk factor in association with GDM risk.

## Strengths and limitations

Information regarding food intake was acquired through weighed food diaries where intake of all food and drink, including all supplements was recorded for the duration of four days, either from Wednesday - Saturday or Saturday - Tuesday as soon as possible after recruitment at week 19 - 24. The food diaries were filled out and delivered before the diagnosis of GDM. The volume of information available is another strength. As age and overweight/obesity are both risk factors for GDM we adjusted for age and pre-pregnancy BMI in our model. We also adjusted for total MET and a calculated weight gain per week between week 19 - 24 and 31 -38 where weighing was performed on two occasions. There are a few limitations to this study as well, such as the change in criteria during recruiting. That was due to the fact that it proved difficult to recruit participants that were obese and had no family history of diabetes. However, when the model was adjusted for family history of diabetes it demonstrated no associations and so it appears to be irrelevant in this case. Even though physical activity was adjusted for in the model it may have an association to dietary habits, as the two factors often correlate and increased physical activity is often associated with healthier diet choices [37]. For instance when total MET was substituted with vigorous activity in the regression model, the association between dietary patterns and GDM was somewhat attenuated. Future studies should account for both physical activity and dietary intake when assessing the associations between lifestyle and risk of GDM.

Conclusions: These results indicate that adhering to a healthy or prudent dietary pattern may prove beneficial in preventing GDM, especially among women already at higher risk due to overweight or obesity before pregnancy. Promoting a healthy diet for prevention of GDM, with a special focus on women who are already at an increased risk for GDM due to overweight or obesity might turn out to be meaningful and merits testing with intervention studies. The results could contribute to changes in dietary advice in an effort to lower the rates of GDM.

**Table 1 -** Maternal characteristics

	Normal weight	Overweight	Obese
	n = 86	n = 44	n = 38
	Mean SD	Mean SD	Mean SD
Age (years)	<b>29.0</b> ± 4.8	<b>30.0</b> ± 4.3	<b>30.0</b> ± 4.6
Height (m)	<b>168</b> ± 5.6	<b>167</b> ± 5.6	<b>168</b> ± 6
Pre-pregnancy weight (kg)	<b>61.1</b> ± 6.5	<b>76.2</b> ± 5.3	<b>93.6</b> ± 9.8
Weight at recruiting week 19-23 (kg) <sup>x</sup>	<b>65.9</b> ± 6.8	<b>81.9</b> ± 7.0	<b>96.8</b> ± 10.3
BMI pre pregnancy (kg/m <sup>2</sup> ) <sup>x</sup>	<b>21.6</b> ± 1.6	<b>27.2</b> ± 1.2	<b>33.2</b> ± 2.7
Gestational age at recruiting (weeks+days)*	<b>20/2</b> ± 3.4	<b>21/0</b> ± 6.8	<b>20/4</b> ± 3.7
BMI at recruiting (kg/m <sup>2</sup> ) <sup>x</sup>	<b>23.3</b> ± 1.8	<b>29.3</b> ± 1.7	<b>33.8</b> ± 2.3
Weight gain at recruiting (kg) <sup>x</sup>	<b>4.6</b> ± 2.7	<b>5.5</b> ± 4.1	<b>2.7</b> ± 4.1
Weekly weight gain between weight recordings (10-17 weeks) (kg) <sup>x</sup>	<b>0.7</b> ± 0.2	<b>0.6</b> ± 0.2	<b>0.5</b> ± 0.3
Parity	<b>0.6</b> ± 0.8	<b>1.0</b> ± 0.9	<b>0.7</b> ± 0.7
Number of GDM diagnoses (%)	<b>2</b> (2.3)	<b>4</b> (9.1)	<b>11</b> (28.9)

Data is presented as mean  $\pm$  standard deviation (SD)

<sup>&</sup>lt;sup>x</sup> Information about weight at recruiting is missing for 13 normal weight subjects, one overweight subject and 7 obese subjects

<sup>\*</sup> Gestational age presented as weeks and days ± standard deviation of days

**Table 2 -** The extracted Prudent dietary pattern

Dietary pattern food	Factor loading coefficient*
Vegetables	0.58
Eggs	0.56
Vegetable oils <sup>a</sup>	0.47
Seafood <sup>b</sup>	0.47
Soft drinks <sup>c</sup>	-0.45
Breakfast cereals	0.40
Fruit and berries <sup>d</sup>	0.39
Nuts and seed	0.36
Pasta/couscous	0.34
French fries	-0.33
Tea, coffee, cocoa powder	0.33

<sup>\*</sup>The Factor loading coefficient describes the correlation (r) between intake of the food groups and the extracted factor

<sup>a</sup>Includes all vegetable oils, peanut and seed butters

<sup>b</sup> Includes all fish, shellfish and seafood products

<sup>c</sup> Includes soda- and sports drinks (sugar sweetened and sugar-free)

<sup>d</sup> Includes all fruit, berries and jams

 $\textbf{Table 3 -} \textbf{ Associations between characteristics of the participants and the prudent pattern score, fasting glucose and HOMA-IR**$ 

Characteristics	n (%)	Prudent dietary pattern	Fasting glucose (mmol/l)	SD	HOMA-IR*	SD
Maternal age (years)						_
18 - 25	45 (27)	-0.11	4.4	(0.4)	2.6	(2.5)
26 – 33	91 (54)	-0.11	4.5	(0.4)	3.4	(4.6)
34 - 40	32 (19)	0.46	4.6	(0.4)	2.1	(0.9)
P-value		0.03	0.01		0.70	
Parity						
Para 0	79 (47)	0.03	4.4	(0.4)	2.8	(2.7)
Para 1-3	89 (53)	-0.03	4.5	(0.4)	3.1	(4.3)
P-value		0.87	0.22		0.79	
Pre-pregnancy BMI (kg/m²)						
18.5 - 24.9	86 (51)	0.1	4.3	(0.4)	2.6	(4.2)
25.0 - 29.9	44 (26)	0.07	4.6	(0.4)	3.2	(3.4)
≥ 30	38 (23)	-0.3	4.7	(0.4)	3.6	(2.4)
P-value		0.68	< 0.01		< 0.01	
Energy intake (kcal)						
Lowest energy quartile	42 (25)	-0.01	4.5	(0.4)	2.9	(3.3)
Second energy quartile	42 (25)	0.01	4.6	(0.4)	3.4	(4.7)
third energy quartile	42 (25)	-0.11	4.5	(0.4)	2.8	(3.9)
Highest energy quartile	42 (25)	0.11	4.4	(0.4)	2.8	(2.7)
P-value		0.98	0.16		0.59	

Data is displayed as mean and standard deviation (SD)

<sup>\*</sup> The homeostatic model assessment of insulin resistance (HOMA-IR)

**Table 4** - Association between the Prudent dietary pattern and its components, total MET, weekly weight gain and gestational diabetes mellitus

	1	U <b>nadjuste</b> d	l		Adjusted <sup>1</sup>			
All participants (n=168)	OR		CI		OR	CI		
Extracted prudent dietary pattern	0.54	(0.30	,	0.58)*	0.44	(0.21	,	0.90)*
Seafood	0.98	(0.95	,	1.02)	0.84	(0.72	,	0.97)*
Eggs	0.99	(0.95	,	1.03)	0.98	(0.90	,	1.06)
Vegetables	1.00	(0.99	,	1.01)	1.00	(0.99	,	1.00)
Fruit and berries	1.00	(1.00	,	1.01)	1.09	(0.99	,	1.01)
Vegetable oils	0.95	(0.78	,	1.16)	0.80	(0.58	,	1.10)
Nuts and seeds	1.01	(0.95	,	1.07)	0.94	(0.76	,	1.17)
Pasta, couscous	0.98	(0.95	,	1.01)	0.89	(0.81	,	0.99)*
Breakfast cereal	1.00	(0.99	,	1.01)	1.02	(0.99	,	1.05)
Coffee, tea and Cocoa powder	1.00	(1.00	,	1.01)	1.00	(0.99	,	1.01)
Soft drinks	1.00	(1.00	,	1.00)	0.99	(0.98	,	1.00)
French fries	1.01	(0.99	,	1.04)	1.02	(0.99	,	1.11)
Overweight/obese before pregnar	ncy (n=82)							
Extracted prudent dietary pattern	0.38	(0.18	,	0.83)*	0.31	(0.13	,	0.75)*
Seafood	0.96	(0.93	,	1.00)*	0.96	(0.93	,	1.00)*
Eggs	0.98	(0.94		1.03)	0.98	(0.93	,	1.03)
Vegetables	1.00	(0.99)	,	1.00)	0.09	(0.99)	,	1.00)
Fruit and berries	1.00	(1.00)	,	1.01)	1.05	(0.92	,	1.20)
Vegetable oils	0.96	(0.70	,	1.32)	0.90	(0.67	,	1.22)
Nuts and seeds	0.99	(0.91	,	1.07)	1.05	(0.92	,	1.20)
Pasta, couscous	0.96	(0.92	,	1.00)	0.95	(0.91	,	1.00)*
Breakfast cereal	0.99	(0.98	,	1.01)	0.99	(0.98	,	1.01)
Coffee, tea and cocoa powder	0.96	(0.90	,	1.03)	0.95	(0.89	,	1.03)
Soft drinks	1.00	(1.00	,	1.00)	1.00	(1.00	,	1.00)
French fries	1.01	(0.99	,	1.04)	1.02	(0.99	,	1.05)

<sup>&</sup>lt;sup>1</sup>Adjusted for age, parity, pre-pregnancy weight, energy intake (kcal), weekly weight gain and total MET

<sup>\*</sup>Association is significant

**Table 5 -** Relationship between different prudent dietary pattern adherences scores (lowest to highest tertile) and HOMA-IR, 120 min outcomes for glucose and insulin at oral glucose tolerance test and the Metabolic Equivalent of Task (MET).

	All participans (n=168)		Normal weight (n=86)		Overweight/ obese (n=82)	
Prudent dietary pattern			HOMA-IR			
Lowest score tertile	2.34	(2.37)	1.46	(0.94)	3.24	(2.19)
Medium score tertile	2.23	(1.76)	1.73	(1.04)	3.18	(1.65)
Highest score tertile	1.88	(1.04)	1.53	(1.1)	2.18	(1.05)
P-value	0.054		0.73		< 0.01	
		Glucos	se 120 min (r	nmol/L)		
Lowest score tertile	5.8	(1.6)	5.2	(2.0)	5.9	(1.2)
Medium score tertile	5.6	(1.7)	5.3	(0.9)	5.9	(1.3)
Highest score tertile	5.3	(1.6)	5.1	(1.4)	5.9	(1.4)
P-value	0.18		0.92		0.51	
		Insul	in 120 min (	mU/L)		
Lowest score tertile	56.1	(44)	48.9	(52)	69.2	(46)
Medium score tertile	61.1	(66)	57.9	(33)	99.1	(98)
Highest score tertile	50.3	(44)	50.1	(39)	66.3	(37)
P-value	0.25		0.52		0.06	
			Total MET			
Lowest score tertile	2202	(4878)	2217	(3751)	1680	(5553)
Medium score tertile	1670	(2978)	1665	(2890)	1853	(3275)
Highest score tertile	2319	(2814)	2523	(2891)	2361	(2541)
P-value	0.17		0.14		0.77	

Data is displayed as medians (Interquartile range)

Significance in differences was found using The Kruskal–Wallis test

mU/L: milliunits per liter

**Table 6** - Adherence to food based dietary pattern and food based dietary guidelines and rate of gestational diabetes mellitus

gestational diabetes menitus				
	Pruden	t dietary p	oattern	
	GDM diagnosis	Non- GDM	Total	GDM
	n	N	n	%
All participants (n=168)				
Highest adherence	1	55	56	1.8
Low/medium adherence	16	96	112	14.3
				P = 0.01
Overweight/obese women (n=82)				
Highest adherence	1	26	27	3.7
Low/medium adherence	14	41	55	25.5
				P= 0.02
	Health	ny eating l	Index	
	GDM diagnosis	Non- GDM	Total	GDM
All participants (n=168)				
Highest adherence	2	54	56	3.6
Low/medium adherence	15	97	112	13.4
				P = 0.05
Overweight/obese women (n=82)				
Highest adherence	1	25	26	3.8
Low/medium adherence	14	42	56	25.0
				P = 0.02

Chi-squared test was used to define significance of differences

### References

- 1. Cheung, N.W. and K. Byth, *Population health significance of gestational diabetes*. Diabetes Care, 2003. **26**(7): p. 2005-9.
- 2. Buchanan, T.A., A.H. Xiang, and K.A. Page, *Gestational diabetes mellitus: risks and management during and after pregnancy.* Nat Rev Endocrinol, 2012.
- 3. 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.
- 4. Ben-Haroush, A., Y. Yogev, and M. Hod, *Epidemiology of gestational diabetes mellitus and its association with Type 2 diabetes.* Diabet Med, 2004. **21**(2): p. 103-13.
- 5. Fadl, H.E., et al., *Maternal and neonatal outcomes and time trends of gestational diabetes mellitus in Sweden from 1991 to 2003.* Diabet Med, 2010. **27**(4): p. 436-41.
- 6. Yogev, Y. and G.H. Visser, *Obesity, gestational diabetes and pregnancy outcome.* Semin Fetal Neonatal Med, 2009. **14**(2): p. 77-84.
- 7. Mitanchez, D., A. Burguet, and U. Simeoni, *Infants Born to Mothers with Gestational Diabetes Mellitus: Mild Neonatal Effects, a Long-term Threat to Global Health.* J Pediatr, 2014. **164**(3): p. 445-50.
- 8. Negrato, C.A., R. Mattar, and M.B. Gomes, *Adverse pregnancy outcomes in women with diabetes*. Diabetol Metab Syndr, 2012. **4**(1): p. 41.
- 9. Boden, G., *Fuel metabolism in pregnancy and in gestational diabetes mellitus.* Obstet Gynecol Clin North Am, 1996. **23**(1): p. 1-10.
- 10. Dabelea, D., et al., Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships. Diabetes, 2000. **49**(12): p. 2208-11.
- 11. Dabelea, D., W.C. Knowler, and D.J. Pettitt, *Effect of diabetes in pregnancy on offspring:* follow-up research in the Pima Indians. J Matern Fetal Med, 2000. **9**(1): p. 83-8.
- 12. Hillier, T.A., et al., *Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia.* Diabetes Care, 2007. **30**(9): p. 2287-92.
- 13. Bottalico, J.N., Recurrent gestational diabetes: risk factors, diagnosis, management, and implications. Semin Perinatol, 2007. **31**(3): p. 176-84.
- 14. Cypryk, K., et al., [Overweight and obesity as common risk factors for gestational diabetes mellitus (GDM), perinatal macrosomy in offspring and type-2 diabetes in mothers]. Przegl Lek, 2005. **62**(1): p. 38-41.
- 15. Olafsdottir, A.S., et al., *Maternal diet in early and late pregnancy in relation to weight gain.* Int J Obes (Lond), 2006. **30**(3): p. 492-9.
- 16. Catalano, P.M., *Trying to understand gestational diabetes*. Diabet Med, 2014. **31**(3): p. 273-81.
- 17. Chen, L., et al., *Prospective study of pre-gravid sugar-sweetened beverage consumption and the risk of gestational diabetes mellitus.* Diabetes Care, 2009. **32**(12): p. 2236-41.
- 18. Park, S., et al., Gestational diabetes is associated with high energy and saturated fat intakes and with low plasma visfatin and adiponectin levels independent of prepregnancy BMI. Eur J Clin Nutr, 2013. **67**(2): p. 196-201.
- 19. Bo, S., et al., Dietary fat and gestational hyperglycaemia. Diabetologia, 2001. **44**(8): p. 972-8.
- 20. Ying, H. and D.F. Wang, [Effects of dietary fat on onset of gestational diabetes mellitus]. Zhonghua Fu Chan Ke Za Zhi, 2006. **41**(11): p. 729-31.
- 21. Saldana, T.M., A.M. Siega-Riz, and L.S. Adair, *Effect of macronutrient intake on the development of glucose intolerance during pregnancy.* Am J Clin Nutr, 2004. **79**(3): p. 479-86.
- 22. Jing, X., et al., [Gestational diabetes mellitus and the lifestyle and dietary structure of pregnant women: a case-control study]. Wei Sheng Yan Jiu, 2010. **39**(2): p. 209-11, 227.
- 23. Ley, S.H., et al., Effect of macronutrient intake during the second trimester on glucose metabolism later in pregnancy. Am J Clin Nutr, 2011. **94**(5): p. 1232-40.
- 24. Morisset, A.S., et al., *Prevention of gestational diabetes mellitus: a review of studies on weight management.* Diabetes Metab Res Rev, 2010. **26**(1): p. 17-25.
- 25. Kant, A.K., *Dietary patterns: biomarkers and chronic disease risk.* Appl Physiol Nutr Metab, 2010. **35**(2): p. 199-206.
- 26. Nordic Council of Ministers, Nordic Nutrition Recommendations 2012. 2014.
- 27. Wallace, T.M., J.C. Levy, and D.R. Matthews, *Use and abuse of HOMA modeling.* Diabetes Care, 2004. **27**(6): p. 1487-95.

- 28. Organization, W.H., *Diagnostic Criteria and Classification of Hyperglycaemia First Detected in Pregnancy* Diagnostic Criteria and Classification of Hyperglycaemia First Detected in *Pregnancy*. 2013: Geneva.
- 29. Matis. Available from: <a href="http://www.matis.is/thjonusta/naeringargildi-matvaela-isgem/">http://www.matis.is/thjonusta/naeringargildi-matvaela-isgem/</a>.
- 30. Karamanos, B., et al., *Relation of the Mediterranean diet with the incidence of gestational diabetes*. Eur J Clin Nutr, 2014. **68**(1): p. 8-13.
- 31. Tobias, D.K., et al., *Prepregnancy adherence to dietary patterns and lower risk of gestational diabetes mellitus*. Am J Clin Nutr, 2012. **96**(2): p. 289-95.
- 32. Crozier, S.R., et al., *Women's dietary patterns change little from before to during pregnancy.* J Nutr, 2009. **139**(10): p. 1956-63.
- 33. Zhang, C., et al., *Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus*. Diabetes Care, 2006. **29**(10): p. 2223-30.
- 34. Asemi, Z., et al., Favourable effects of the Dietary Approaches to Stop Hypertension diet on glucose tolerance and lipid profiles in gestational diabetes: a randomised clinical trial. Br J Nutr, 2013. **109**(11): p. 2024-30.
- 35. Aston, L.M., et al., Determination of the glycaemic index of various staple carbohydrate-rich foods in the UK diet. Eur J Clin Nutr, 2008. **62**(2): p. 279-85.
- 36. Foster-Powell, K., S.H. Holt, and J.C. Brand-Miller, *International table of glycemic index and glycemic load values: 2002.* Am J Clin Nutr, 2002. **76**(1): p. 5-56.
- 37. Gillman, M.W., et al., *Relationships of physical activity with dietary behaviors among adults.* Prev Med, 2001. **32**(3): p. 295-301.

# 8.2 Appendix II – Recipes from the Directorate of Health

#### BRAUÐ, KEX OG KÖKUR

Baguette Beyglur

Brauð, byggbrauð

Brauð, Fitty, Orkubr.frá Myllunni, Bónus kjarna

Brauð, fjölkornabrauð Brauð, franskbrauð

Brauð, heilhveitibrauð, heimilisbrauð

Brauð, malt, danskt rúg, Fitty kjarna, Sólkj, Ráðskbr

Brauð, rúgbrauð, seytt

Brauð, speltbr (Myllan), Heilkornabr Mosfelba.

Brauðstangir (á pítsustöðum)

Bruður, fínar Bruður, grófar Croissant Croutons f. salat Flatkökur

Hamborgarabrauð Heilhveitihorn Hrökkbrauð, gróf Hrökkbrauð, fín Hvítlauksbrauð Ítölskbrauð, ciabatta

Kringlur, fínar Kringlur, heilhveiti Langloka, gróf Langloka, fín Nanbrauð

Pítsusnúðar Pítubrauð, fín

Ostaslaufur

Pítubrauð, gróf Pólarbrauð Pylsubrauð Rúnstykki, gróf Rúnstykki, fín

Skonsur Taco skeljar Tortilla

Brauðterta

Heitur brauðréttur Kjúklingaburritos frá Sóma Langloka m/kjúklingi, léttsósa

Langloka m/kalkúni og beikoni

Langloka m/skinku, eggjum, grænm. og sósu Langloka m/skinku, osti og sinnepssósu

**Nautaburritos** 

Pítubrauð, Pólarbrauð indverskur kjúklingur Pítubrauð, Pólarbrauð, m/skinku og salati Samloka m/eggjum og grænm. heilsubiti Samloka m/miklu kjöti t.d. American Style

Samloka m/roastbeef Samloka m/rækjusalati Samloka m/skinku og osti Samloka m/túnfisksalati Subway bræðingur, án sósu Subway Club, án sósu

Subway klassískur ítalskur, án sósu

Subway m/kalkún, án sósu Tortilla kjúklingur- Tikka masala

Tortilla, reykt skinka, egg, grænmeti pítusósa

Hafrakex, heilhveitikex

Hrískökur Kransakökur Kremkex Mjólkurkex, fínt Mjólkurkex, gróft

Saltkex Smákökur

Smákökur, lítil fita t.d. marenstoppar

Súkkulaðikex Tekex

Döðlubrauð, bananabrauð

Eplakaka, paj Gulrótarkaka

Hjónabandssæla, Figroll

Jólakaka

Kanelsnúður, ömmusnúður

Kleinuhringir Kleinur

Kleinur Gæðabakstur Kleinur Ömmubakstur Kókoskúlur, úr bakaríum

Marengsterta Muffins

Möndlukaka m/bleiku kremi

Ostakaka Pönnukökur Rjómaterta Rúlluterta, brún

Snúðar, gersnúðar (sænskir)

Snúður (bakarí) Súkkulaðikaka Tebolla Tíramísúkaka

Vínarbrauð, smjörkaka Vínarterta (randalín)

Vöfflur

Samloka m/hangikjöti og baunasalati

Samloka m/kalkún og beikoni

Samloka m/kjúklingi eða kalkúni Þeyttur rjómi Samloka m/kjúklingi og rauðu pestói Þeyttur jurtarjómi

ÁLEGG

Fetaostur, í olíu Roastbeef Fetaostur, í saltlegi, Fetaostur frá Mjólku Rúllupylsa

Fjörostur Skinka, Hunangsskinka

Sulta

Gráðaostur Spægipylsa Kotasæla Steik

Léttfeti

Mozzarella ostur 21% Áleggspasta (kavíar)
Mozzarella ostur, ferskur í kúlum Lax, silungur, reyktur

Mozzarella ostur, ferskur i külum Lax, silungur, reyktu Mygluostar (fita ca. 33%), Brie, Dala-hr.,Höfðingi Rækjur

Mygluostar (fita ca. 25%), Camembert, Hrókur Síld, maríneruð Mygluostar (fita ca. 36%), Gullostur, Stóri-Dímon Síld, maríneruð í sósu

Mygluostar (fita ca. 36%), Dalayrja, Kastali

Smurostur 18%, Gotti smurostur

Mysuostur, smyrjanlegur Ítalskt salat, majónes

Ostur 17%, Létt-Brie, rifinn salatostur

Ostur 26%, rifinn pasta, pizzu og gratin

Ostur 11%

Rækiusalat majónes

Ostur, 11% Rækjusalat, majónes
Parmesanostur Rækjusalat, sýrður rjómi

Rjómaostur, til matargerðar í stóru boxi (26%) Skinkusalat

Rjómaostur, smyrjanlegur í lítilli dós (19%)

Smurostur, létt 6% Túnfisksalat, majónes

Sojaostur Túnfisksalat, sýrður rjómi Steyptir, bræddir ábætisost (ca. 32%)

Egg Hummus
Hamborgarahryggur/lúxus skinka, magurt Hunang
Hamborgarahryggur/lúxus skinka, m/fiutrönd Pestó

Hangiálegg Sulta/marmelaði

Hangiálegg, fituskert Súkkulaðiálegg

Kindakæfa Kjúklingaskinka, kalkúnaskinka Agúrka

Lifrarkæfa Bananar
Lifrakæfa, fituminni Epli
Lifrarpate Paprika

Malakoff Pepperóní

**MORGUN KORN** 

All-bran Múslí án sykurs

Pran-flakes Westaflakes Westas Múslí sætt

Bran-flakes, Weetaflakes, Weetos, Múslí sætt
Cheerios Rice Krispies
Cocoa Puffs, Lucky Charms, Coca pops, Guldkorn Sesamfræ

Bran-flakes, Weetaflakes, Weetos Sólblómafræ Hafragrautur Special K

Hafragrjón Sol Gröd m/epli&kanil og m/bláberjum

Honey nut Cheerios Weetabix, Spelt flögur Hörfræ Havre Fras, Crunchy bran

Hveitikím Rug Fras

Tómatur

#### Kornflögur

#### SÝRÐAR MJÓLKURVÖRUR

AB-mjólk, lífræn AB-mjólk

AB-mjólk m/ávöxt.og músli, fitu og sykursk.

Létt-ABT-mj m/ávöxt.og músli, fitu og sykursk.

Létt-drykkjarjógúrt, Léttur Ab drykkur

ABT-mjólk hrein Létt-drykkjarjógúrt, án viðbætts sykurs

ABT-mjólk með ávöxtum og múslí Léttjógúrt, fitusk.(m/trefjum), Orkujógúrt f. Mjólku Benecol, m/ávöxtum, sykurskert Léttjógúrt, fitu og sykursk (melónu), LGG+ jógúrt

Bíómjólk Létt-súrmjólk

Bíómjólk m/vanillu, sykurskert Létt-súrmj m/eplum og peru, fitu-og sykursk.

Engjaþykkni LGG+

FrúTína jógúrt LGG+, epli og perur, án viðbætts sykurs

FrúTína jógúrtdrykkur Pasqual, feitt
Grísk jógúrt Pasqual, fitusnautt
Hrísmjólk m/sætri sósu Pasqual, létt

Jógúrt m/ávöxtum/bragðefni, lífræn jógúrt SMS skyr

Skyr, hreint
Jógúrt, hrein
Skyr m/ávöxtum, Kea skyr, skyr.is

KEA skyrdrykkur m/agave sírópi Skyr m/ávöxtum, m. sætuefni Krakkaskyr Skyr.is drykkur m/ávöxtum

Létt AB mjólk, hrein Skyr.is drykkur m/ávöxtum, m.sætuefni

Létt-AB-mjólk m/ávöxtum, fituskert Súrmjólk

Létt-AB-mj m/eplum og gulrót. fitu og sykursk. Súrmjólk m/ávöxtum eða bragðefnum

Létt-ABT-mj m/ávöxt. og músli, fituskert Þykkmjólk m/ávöxtum

#### **GRAUTAR**

Ávaxtagrautur, sykurskertur Hrísgrjónagrautur, (úr léttmjólk) Ávaxtagrautur, venjulegur Hrísgrjónagrautur, (úr nýmjólk)

Hafragrautur

## ÚTÁLÁT

Fjörmjólk Rjómi

Léttmjólk, Létt G mjólk

Nýmjólk, lífrænmjólk, G-mjólk Púðursykur Undanrenna Sykur

Kaffirjómi Síróp, agavesíróp

Matreiðslurjómi

### **ÍS OG BÚÐINGUR**

Dýfa á ísÍssósaÍs úr vélBragðarefurÍs, rjómaís, SkafísMjólkurhristingurÍs, Hversdagsís EmmessMjólkurbúðingurÍs, KjörísRjómabúðingur, frómas

Ítalskur ís Sun Lolly klaki

Jógúrtís

Íspinni, ísblóm, toppar Þeyttur rjómi Frostpinni, klaki Þeyttur jurtarjómi

Snickers, Mars, Kit Kat ís, Ben & Jerry's ís

#### **SÚPUR**

Baunasúpa (kjöt sér)

Brauðsúpa

Kjötsúpa (kjöt sér)

Pastasúpa

Grænmetissúpa, heimalöguð Sætsúpa, ávaxtasúpa

Grænmetissúpa, heimalöguð m/rjóma Grænmetissúpa m/kókosmjólk og curry paste

Grænmetissúpa, tær pakkasúpa Grænmetissúpa, þykkt, pakkasúpa

Kakósúpa

Tómats m/rjómaosti, t.d. fiskisúpa (fiskur sér) Tómatsúpa, grunnur t.d. fiskisúpa (fiskur sér)

#### FISKUR OG FISKRÉTTIR

Fiskibollur úr dós

Fiskibollur, búðingur, steikt úr steikingarfeiti

Gellur, kinnar, soðnar

Hrogn, soðin

Humar

Keila, langa, bökuð/grilluð

Lax, silungur, steikt

Lax, silungur, soðið/bakað/grillað

Lifur, soðin

Karfi, rauðspretta, steinbítur, steikt

Karfi, rauðsp, steinb, soðið/bakað/grillað

Plokkfiskur (kartöflur í rétti)

Plokkfiskur m/olíu (kartöflur í rétti)

Rækjur

Rækjur, djúpsteiktar

Saltfiskur, reyktur fiskur

Saltfiskur í tómat-ólífusósu, suðrænn saltfiskur

Skötuselur

Smálúða, grilluð/bökuð

Smálúða, steikt

Stórlúða, soðin/bökuð/grilluð Ýsa, djúpsteikt, skyndibitastað

Ýsa, ofnbökuð m/lauk og osti

### KJÖT OG KJÖTRÉTTIR

Chili con carne m/hakki og baunum

Hakk m/tómatsósu án steikingarfeiti

Hakk m/tómatsósu, m/steikingarfeiti

Hakk m/niðurs.tóm, grænm. án steikingarf.

Hakk m/niðurs.tóm, grænm m/steikingarf.

Hamborgari án brauðs án steikingarfeiti

Hamborgari án brauðs, m/steikingarfeiti Kjötbollur úr hakki, steiktar m/steikingarfeiti

Lasagna m/hakki

Tortilla m/hakk og grænmetis fyllingu

Bixímatur

Bjúga, soðin

Kjötfarsbollur, steikt m/steikingarfeiti

Kjötfars, soðið

Pylsur, soðnar

Kjúklingur m/skinni, grillaður/bakaður

Kjúklingur án skinns, grillaður/bakaður/steiktur

Kjúklingabitar, djúpsteiktir

Kjúklinganaggar

Ýsa, þorskur steikt m/raspi og matarolíu Ýsa, þorskur, steikt m/raspi og smjöri

Ýsa, þorskur steikt m/raspi og smjörlíki

Ýsa, þorskur steikt m/raspi óþ.steikingarfeiti

Ýsa, þorskur án rasps, steikt/grillað í steikingarf.

Ýsa, þorskur, soðið/bakað/grillað án feiti

Ýsa í malasíu karrí (Hagkaup)

Ýsa í rjómasósu

Ýsa í rjómasósu m/grænmeti (50:50)

Ýsa í tilb.sósu, úr fiskbúð (verslun)

Ýsa í tilb.sósu m/grænmeti, úr fiskbúð (verslun)

Ýsunaggar

Harðfiskur, bitafiskur

Hákarl Hvalur, súr Sardínur

Síld, maríneruð Síld, maríneruð í sósu

Túnfiskur í olíu Túnfiskur í vatni

Sushi, lax nigiri, lax maki

Sushi, california

Saltkjöt, feitt

Hangikjöt, Londonlamb

Nautakjöt, magurt, steikt/bakað Nautakjöt, millifeitt, steikt/bakað Nautakjöt, feitt, bakað/steikt

Svínakjöt, magurt, bakað/steikt Svínakjöt, millifeitt, bakað/steikt

Svínaköt, feitt (hnakki), bakað/grillað/steikt

Skinka, hamborgarahryggur

Skinka, hamborgarahryggur, magurt

Hreindýrakjöt Hrefnukjöt

Hrossakjöt, reykt

Hrossakjöt, saltað Hrossakjöt, soðið

Hrossakjöt, steikt

Raspsteikt kjöt, t.d. snitsel, naggar steikt

Kjúklingaréttur m/sósu, án rjóma, lítið grænmti

Kjúklingaréttur m/rjómasósu, lítið grænmeti Kjúklingarétt. m/sósu og grænm (50:50) án rjóma Kjúklingarétt. m/rjómasósu og grænmeti (50:50)

Kjúklingaréttur m/tilbúinni sósu (t.d. Tikka masala), lítið

grænmeti

Kjötpottur, gúllas m/sósu, án rjóma Kjötpottur, gúllas m/rjómasósu

Kjötpottur m/sósu og grænm (50:50) án rjóma

Kjötpottréttur m/rjómas. og grænmeti (50:50) Grænmetispottréttur m/litlukjöti (25%)

Kjúklingaréttur m/tilbúinni sósu (t.d. Tikka masala) og

grænmeti (50:50)

Beikon Blóðmör, soðinn Lifrarpylsa, soðin

Lambakjöt, magurt, bakað/grillað/steikt Lambakjöt, læri, bakað (lítil fita borðuð)

Lambakjöt, millifeitt, bakað/grillað/steikt

Lambakjöt, millifeitt, soðið Lambakjöt,feitt,bak/grill/steikt t.d.kótilettur m/fitu

Lambakjöt, feitt, soðið (t.d.súpukjöt)

Lifur, steikt Svartfugl, lundi Svið, soðin, sviðasulta

Saltkjöt, magurt

### PÍTSA, PASTA, EGGJA, GRÆNMETIS- OG BAUNARÉTTIR

Baunir, hvítar niðursoðnar í tómatsósu

Baunir, soðnar, nýrna-, kjúkl.-, linsu- og soyab.

Baunabuff

Bökur m/grænmeti Chili m/baunum, án kjöts

Egg, soðin Egg, steikt

Eggjakaka, omeletta Gratínerað grænmeti Grænmetislasagna Núðluréttur, instant

Núðluréttur Nings m/kjúkl., grænm. og egg Hrísgrjónaréttur Nings m/kjúkl, grænm og egg

Heilsuréttur Nings-brún hrísgr m/egg,kjúkl.og grænm. Pastar. heitur (feitur), pepperón, grænm, rjómasósa

Pastaréttur heitur m/skinku,grænm.sýrðum rjóma

Pastasalat, kalt m/grænmeti, án sósu Pastasalat, kalt m/kjúklingi, skinku, án sósu Pastasalat, kalt m/túnfiski og grænmeti, án sósu Pítsa, eldbökuð, m/pepperóní, ananas og sveppum Pítsa, eldbökuð m/kjúkl., hnetum, sólþ.tóm. & svep.

Pítsa m/grænmeti

Pítsa m/pepperóni og lauk Pítsa m/skinku og ananas Pítsa m/sósu og osti (margaríta) Pítsu, hvítlauksbrauð, pitsastaðir

Sojakjöt, tofu Tortellini

Tortilla, burrito, m/bauna og grænmetisfyllingu

Vorrúllur, Kínarúllur

#### **SKYNDIBITAR**

Fylltar pönnukökur (Crepes)

Hamborgari m/brauði, grænmeti og sósu

Hamborgari McDonalds

Kjúklingabitar

Kjúklingaborgari KFC og MacDonalds

KjúklingaTwister KFC

Kjúklingasalat, KFC Zinger salat

Kjúklingaburritos frá Sóma

Nautabúrítós frá Sóma

Núðluréttur Nings m/kjúkl., grænm. og eggjum

Hrísgrjónaréttur Nings m/kjúkl, grænm og egg

Heilsuréttur Nings-brún hrísgr m/egg,kjúkl.og grænm.

Píta m/buffi, grænmeti og sósu Píta m/grænmeti og sósu

Pylsa í brauði m/tómatsósu og/eða sinnepi

Pylsa í brauði, m/öllu Rækjur, djúpsteiktar

Tortilla, burrito, m/bauna og grænmetisfyllingu

Vorrúllur, djúpsteiktar

Ýsa, djúpsteikt

#### KARTÖFLUR OG MEÐLÆTI

Bygg, soðið Eggjapasta, núðlur Heilhveitipasta Hrísgrjón

Hýðishrísgrjón

Kartöflur, franskar, ofnsteiktar, steiktar

Kartöflur, soðnar, bakaðar Kartöflusalat m/majónesi Kartöflusalat m/sýrðum rjóma

Kús kús

Kartöflugratín m/rjóma Rótargrænmeti (sætar kartöflur, kartöflur, gulrætur)

Kartöflumús Spaghetti, pasta Kartöflur, brúnaðar Sætar kartöflur

Kartöflur, franskar, af skyndibitastað

SOĐIĐ, STEIKT GRÆNMETI

Asíur, súrsað grænmeti, relish Laukur, steiktur, í steikingarfeiti Blómkál soðið Laukur, steikur þurrkaður

Eggaldin, steikt Maískorn
Grænar baunir Ólívur
Grænmetisblanda, frosin, soðin Rauðkál
Laukur, papríka, gulrætur, steikt í olíu Rauðrófur
Laukur, papríka, gulrætur, steikt, í smjörlíki Rósakál

Laukur, papríka, gulrætur, steikt, í steikingarfeiti Sólþurrkaðir tómatar í olíu

Gulrófur, soðnar Spergilkál Gulrætur, soðnar Spínat

Hvítkál, soðiðSveppir, niðursoðnirHvítlaukur í olíuSveppir, steiktir, í matarolíuKúrbítur, steikturSveppir, steiktir, í smjörlíki

Laukur, steiktur, í matarolíu Sveppir, steiktir, í steikingarfeiti

Laukur, steiktur, í smjörlíki

HRÁTT GRÆNMETI

Agúrkur Laukur, rauðlaukur, púrrulaukur Blómkál Papríka, græn, gul og rauð

Gulrófur Ruccola, spínat ofl. dökkgrænt kál

Gulrætur Salat (iceb, kínak, tómatar, gúrka, papríka)
Hrásalat (hvítkál, gulrætur, rófur) Salat (ruccola, spínat, tómatar, gúrka, papríka)

Hrásalat í majonessósu Spergilkál Iceberg, kínakál, blaðsalat Tómatar

ÁVEXTIR

Ananas Vatnsmelóna Appelsínur Vínber

Ávextir, blandaðir, skornir

Avocado (lárpera) Niðursoðinn ananas

Bananar Niðursoðnir blandaðir ávextir

Bláber

Epli Döðlur Fíkjur

Ferskjur Furuhnetur

Greipaldin

Graskersfræ Hunangsmelónur Hnetur, salthnetur Jarðarber Kantelópa Hnetur, aðrar Kíví Hörfræ Krækiber Möndlur Mandarínur Rúsínur Nektarínur Sesamfræ Mangó Sólblómafræ

Mangó Sölblóma Perur Sveskjur

Plómur Þurrkaðir ávextir, aðrir

#### VIÐBIT, FEITI, STEIKINGARFEITI

Bertolli (áður Olivio) Smyrill Flora Proactive Smyrja

Klípa Sólblóma 65% Létt og laggott Hamsar

Létt og laggott með ólífuolíu Hörfræsolía
Létta Olíublanda
Kókosolía Ólívuolía

Smjör Smjörlíkisblanda Smjörvi Steikingarfeiti

## SÓSUR, SÝRÐUR RJÓMI, SALATSÓSUR

Bernessósa Majónes, extra light (5% fita)

Brún sósa (soðsósa, lítil fita)

Guacamole

Kókosmjólk

Jafningur (mjólk)

Kókosmjólk, létt

Malasíu karrísása fyrir fiek

Malasíu karrísósa fyrir fisk Olíuediksdressing Ostasósa (ca 15%) Pestósósa

Rjómasósa (nær bara rjómi) Piparsósa

Sósa úr tómötum Pítusósa keypt/búin til úr majónesi (70%fita)

Sósa úr smurosti (18%) og léttmjólk Pítusósa eggjalaus (28% fita)

Sojasósa Remúlaði (58%fita)
Súrsætsósa Salatsósa, salatbar í Hagkaup

Tikkamasalasósa ofl. tilbúnar sósur úr krukku Salsasósa

Tandori og jógúrtsósa m/grænmeti fyrir fisk Saisasosa
Saisasosa

Uppbökuð sósa Sinnepssósa (21% fita)
Súrmjólkursósa, jógúrtsósa

Graflaxsósa Syrður rjómi, 5%

Grænmetissósa (58%) Sýrður rjómi 10% Hamborgarasósa (40% fita) Sýrður rjómi 18% Hamborgarasósa, eggjalaus (21% fita) Sýrður rjómi 36%

Hvítlaukssósa (53% fita)

Thousand Island dressing
Kokteilsósa úr majónesi (70% fita)

Thousand Island dressing, létt

Kokteilsósa, eggjalaus (25% fita)

Tómatsósa (ketchup)

Létt Pítusósa (40% fita)

Majónes Salt

Majónes, létt

## DRYKKIR

Ávaxtadrykkir (smoothy) Gosdrykkir, kóladrykkir sykurlausir

Boozt, skyr.is og ávextir Fresca

Boozt, skyr.is án viðbætts sykurs og ávextir Gosdrykkir sykraðir, aðrir Fjörmjólk Gosdrykkir sykurlausir, aðrir

Fjörmjólk Gosdrykkir syk FrúTína jógúrtdrykkur Maltöl

Fru lina jogurtdrykkur Maltol Kakó úr nýmjólk Pilsner

Kakó, úr léttmjólk, kókómjólk, Kappi kókómjók
KEA skyrdrykkur, (Agave síróp)
Burn orkuskot

Maria Maria

Kókómjólk Extreme orkuskot, Redfin

Kókómjólk, sykurskert Íþróttadrykkir (Aqarius, Gatorade, Leppin, Isostar)

Létt-drykkjarjógúrt,Léttur Ab drykk. m.ávöxtum Orkudrykkir (Burn, Cult, Red Bull, Bomba)

Létt-drykkjarjógúrt (án viðbætts sykurs) Orkudrykkir (Magic, Orkan)

Léttmjólk, Létt-G mjólk Powerade

Nýmjólk, lífrænmjólk, G-mjólk

Skyr.is drykkur m/ávöxtum

Bjór

Skyr.is drykkur m/ávöxtum án viðbætts sykurs

Hrísmjólk, kalkbætt

Sojamjólk Swiss miss Undanrenna

Ávaxta-, Berg- og Eðaltoppur m.andox/trefjum Kolsýrt vatn, Toppur, Egils kristall m/bragðefn.

Kristall +

Kristall sport (m/sætuefnum)

Vatn

Appelsínusafi, greipsafi, hreinn Ávaxtasafi, vítamínbættur

Eplasafi, hreinn

Engiferdrykkur, My secret

Gulrótarsafi Heilsusafi Nektar

Svali/Frissi fríski, djús, sykraður

Svali, djús, sykurskertur

Gosdrykkir, kóladrykkir sykraðir

SÆLGÆTI, SNAKK

Brjóstsykur Bland í poka Bounty

Buff, staur Corny

Hlaup

Hraun

Hrískúlur Karamellur

Kitkat

Kúlur

Lakkrís Lakkrískonfekt

M&M's, Smartís

Mars, Snickers

Nizza

Ópal, Tópas, með sykri Ópal, Tópas, sykurlaust

Pipp

Prince Polo

Rolo

Síríuslengja

Special K bar

Súkkulaði, fyllt Súkkulaði, rjóma Bjór lite Brennd vín Hvítvín

Líkjör Rauðvín

Sherry, millisterk vín

Cappuccino m/léttmjólk Cappuccino m/nýmjólk

Kaffi

Kaffi Latte m/léttmj, Macchiato m/léttmj. Kaffi Latte m/nýmj, Macchiato m/nýmj.

Te

Kaffirjómi í kaffi, te Léttmjólk í kaffi, te Nýmjólk í kaffi, te Rjómi í kaffi, te

Canderel (aspartam)

Hunang

Nutrasweet í kaffi, te Síróp, agavesíróp Sykur í kaffi, te

Súkkulaði, suðu Súkkkulaði, 70%

Súkkulaði m. karmellu ofl., Súkkulaðihnetur, m&m hnetur

Súkkulaðirúsínur

Twix

Tyggjó, með sykri Tyggjó, sykurlaust

Hnetur, salthnetur Hnetur - allar teg. Hnetubar-bland í poka

Kartöfluflögur Nasl, skrúfur Poppkorn, heimap Poppkorn, tilbúið Poppkorn, örbylgju

Poppkorn örbylgju Popp Secret Poppkorn, örbylgju, létt

Poppkorn, örbylgju Richfood Light

Saltstangir Söl

Tortilla, chips

### **FÆÐUBÓTAREFNI**

B-vítamíntöflur C-vítamíntöflur D-vítamíntöflur E-vítamíntöflur

Fjölvítamín án A- og D-vítamíns Fjölvítamín með A- og D-vítamíni

Fólasíntöflur Frískamín Hákarlalýsishylki

Heilsutvenna, lýsishylki (án A og D vítamín)

Heilsutvenna, vítamíntafla

Járntöflur Krakkalýsi

Lýsi og liðamín, liðamínstafla

Lýsi og liðamín, lýsishylki (án A og D vítamín)

Lýsi+Dvít & kalk, kalktafla Lýsi + Dvít & kalk, lýsishylki

Omega-3, hylki Omega-3 forte, hylki Omega-3 fiskiolía

Sportþrenna, fjölvítamíntafla (karnitín sleppt)

Sportþrenna, Omega-3 hylki

Udo´s Oil 3-6-9 Ufsalýsi Þorskalýsi

Þorskalýsisperlur Önnur bætiefni

Próteinduft, Myoplex original, deluxe, Profitt protein

Próteinduft, 100% Whey protein

Próteindrykkur, Myoplex tilbúinn til neyslu Próteindrykkur, Herbalife, Nupo létt

Próteindrykkur, Hámark Próteindrykkur, Hleðsla

Próteinstykki t.d. Myoplex, Easy body, Hreysti, Kraftur,

OhYeah, Styrkur

Próteinstykki, Herbalife gull

Þyngingarblanda

# 8.3 Appendix III - Food groups in ISGEM

# 1. Mjólk, mjólkurvörur

- 1.1 Nýmjólk, léttmjólk, undanrenna, rjómi, kakómjólk, kakó, bragðbættir mjólkurdrykkir
- 1.2 Sýrðar mjólkurvörur, sýrður rjómi, jógúrt, jógúrtdrykkir og skyr
- 1.3 Mjólkurgrautar og –súpur, mjólkurbúðingar, rjómabúðingar, tiramisú
- 1.4 Nýmjólkurduft, undanrennuduft

### 2. Ostar

- 2.1 Allir ostar úr mjólk eða mjólkurvörum
- 2.2 Ostar úr soja eða öðru jurtapróteini

## 3. Ís

- 3.1 Mjólkurís, rjómaís, jógúrtís
- 3.2 Jurtaís
- 3.3 Vatnsís

## 4. Kornmatur, brauð og kökur

- 4.1 Ómalað og heilmalað korn, kím og klíð. Hrísgrjón, maís og hafragrjón
- 4.2 Mjöl
- 4.3 Morgunverðarkorn, mjölgrautar
- 4.4 Brauð, hrökkbrauð, tvíbökur, bruður, skonsur
- 4.5 Kex, sætt, ósætt, smákökur
- 4.6 Kökur, sætabrauð (annað en kex og smákökur), tertur, ostakökur
- 4.7 Pasta, kús-kús

# 5. Grænmeti og kartöflur

- 5.1 Nýir, frystir rótarávextir, nema kartöflur
- 5.2 Nýtt, fryst grænmeti: stönglar, blöð, aldin
- 5.3 Nýjar kartöflur
- 5.4 Nýjar, frystar baunir, ertur
- 5.5 Nýir sveppir
- 5.6 Ferskar kryddjurtir
- 5.7 Niðursoðið og niðurlagt grænmeti, tómatmauk
- 5.8 Þurrkað grænmeti, kartöfluduft
- 5.9 Franskar kartöflur

# 6. Ávextir, ber, hnetur og fræ

6.1 Nýir, frystir ávextir

- 6.2 Ný, fryst ber
- 6.3 Hnetur, fræ
- 6.4 Niðursoðnir ávextir, ber, ávaxtagrautar, ávaxtamauk
- 6.5 Þurrkaðir ávextir og ber
- 6.6 Sultur

# 7. Kjöt og kjötvörur.

- 7.1 Lambakjöt, kindakjöt, nýtt, fryst, saltað, reykt, hakkað
- 7.2 Nautakjöt
- 7.3 Svínakjöt
- 7.4 Hrossakjöt
- 7.5 Hreindýra- hvalkjöt
- 7.6 Fars, farsvörur, pylsur, bjúgu, áleggspylsur
- 7.7 Innmatur, slátur, svið, kæfa
- 7.8 Niðursoðin kjötvara

# 8. Fiskur, fiskafurðir og skeldýr

- 8.1 Ferskur og frystur fiskur. Fiskhakk, hrogn, lifur
- 8.2 Þurrkaður og hertur fiskur
- 8.3 Fiskfars og farsvörur, fiskipate
- 8.4 Saltfiskur, reyktur fiskur, siginn, kæstur og grafinn fiskur
- 8.5 Niðurlagður og niðursoðinn fiskur og skeldýr
- 8.6 Fersk og fryst skeldýr

# 9. Fuglakjöt

- 9.1 Alífuglar
- 9.2 Villtir fuglar

# 10. Egg og eggjavörur

- 10.1 Egg, ný, fryst, heil eða fljótandi
- 10.2 Þurrkaðar eggjavörur

# 11. Feitmeti: smjör, smjörlíki, olíur o.fl

- 11.1 Jurtaolíur, jurtafeiti
- 11.2 Fiskolíur, lýsi
- 11.3 Tólg, mör, kjötfita
- 11.4 Smjör, Smjörvi, Létt og laggott, Klípa
- 11.5 Smjörlíki, hert fita
- 11.6 Hnetusmjör, fræsmjör

## 12. Drykkir, nema mjólkurdrykkir

- 12.1 Te, kaffi, kakóduft
- 12.2 Gosdrykkir, svaladrykkir
- 12.3 Blandaðir ávaxta- og berjadrykkir, saft
- 12.4 Hreinir safar, ávaxtasafar, berjasafar, grænmetissafar
- 12.5 Íþrótta- og orkudrykkir
- 12.6 Bjór, pilsner, maltöl
- 12.7 Borðvín
- 12.8 Millisterk vín, brennd vín, líkjör
- 12.9 Vatn, sódavatn með og án bragðefna

# 13. Matarsalt, edik, ger, krydd og kraftur

- 13.1 Matarsalt, edik, krydd og kraftur
- 13.2 Ger og hjálparefni
- 13.3 Gervisætuefni

# 14. Snakk: poppkorn, flögur o.fl.

- 14.1 Poppkorn
- 14.2 Flögur, skrúfur, kornstangir, annað snakk

# 15. Sósur, súpur og áleggssalöt

- 15.1 Allar sósur og ídýfur: Salatsósur, majones og majonessósur, olíusósur, rjóma og ostasósur, sinnep, tómatsósur, sósur úr grænmeti, uppbakaðar, jafnaðar sósur, súrsætar sósur o.fl.
- 15.2 Súpur, súpuduft
- 15.3 Áleggssalöt, majonessalöt, salöt úr sýrðum rjóma

## 16. Tilbúnir réttir

- 16.1 Pitsur, samlokur, pítur, brauðréttir, hamborgarar, pylsa í brauði
- 16.2 Pastaréttir, lasagna
- 16.3 Kjötréttir
- 16.4 Fiskréttir
- 16.5 Grænmetisréttir
- 16.6 Eggjaréttir

# 17. Fæðubótarefni, næringardrykkir, sérfæði

- 17.1 Vítamín, steinefni, önnur fæðubótarefni
- 17.2 Megrunar- og próteindrykkir, næringardrykkir, próteinstykki

## 18. Sykur, hunang og sælgæti

- 18.1 Sykur, púðursykur, flórsykur
- 18.2 Hunang
- 18.3 Sælgæti

8.4 Appendix IV – Calculated loss (%) of nutrients due to cooking

									_	
	1-3	4	5-6	5.1	5.2	7	8	9	10	11
<u>A-vítamín</u>										
suða	0	10	10	5	5	20	20	45	5	15
steiking	10	10	10	10	10	20	20	25	20	50
bakstur	10	10	10	10	10	5	10	30	20	15
<u>E-vítamín</u>										
suða	20	0	0	0	0	20	0	45	0	25
steiking	20	0	0	0	0	20	0	25	0	80
bakstur	20	0	0	0	0	20	0	20	0	25
<u>B₁-vítamín</u>										
suða	0	15	25	25	40	60	20	60	10	0
steiking	0	20	25	10	10	20	20	30	15	0
bakstur	0	20	25	25	10	20	20	40	15	0
B <sub>2</sub> -vítamín										
suða	10	10	35	30	35	30	30	5	5	0
steiking	10	5	35	5	5	20	10	10	10	0
bakstur	10	5	35	5	5	20	10	10	20	0
bakstui	10	J	33	J	J	20	10	10	20	U
<u>Níasín</u>										
suða	0	30	35	30	35	50	30	40	0	0
steiking	0	5	35	5	5	20	10	20	0	0
bakstur	0	5	35	10	5	20	10	20	0	0
B <sub>6</sub> -vítamín										
suða	10	40	40	20	35	50	30	40	10	0
steiking	10	40	40	10	10	40	20	40	20	0
bakstur	10	10	40	10	10	40	20	35	20	0
<u>Fólat</u>										
<u></u> suða	20	30	40	50	50	30	30	40	10	0
steiking	20	30	40	30	30	20	20	30	20	0
bakstur	20	50	40	25	30	20	20	30	30	0
B <sub>12</sub> -vítamín										
suða	5	0	0	0	30	30	20	50	0	0
steiking	5	0	0	15	20	20	10	30	0	0
bakstur	5	0	0	20	30	20	10	30	0	0
<u>C-vítamín</u>										
suða	50	30	50	40	50	20	20	20	0	0
steiking	50	15	50	20	20	20	20	20	0	0
bakstur	50	30	50	15	30	20	20	20	0	0
<u>Steinefni</u>										
suða	5	5	5	5	5	5	5	5	5	5
steiking	0	0	0	0	0	0	0	0	0	0
bakstur	0	0	0	0	0	0	0	0	0	0
มสหรเนเ	U	U	U	U	U	U	U	U	U	U