1

General introduction
General introduction

During the last decades obesity has spread as a pandemic over Europe (1). More fertile women are getting (more) obese, (more) children are getting (more) obese and maternal weight gain in pregnancy increases over time. This has created a vicious circle, since obese mothers are more likely to have obese children (2-5).

Pregnancies, and especially excessive gestational weight gain, are believed to be important factors for body weight retention and the subsequent development of maternal overweight and obesity, or worsening existing overweight (6,7).

Prepregnancy obesity and gaining too much weight during pregnancy are also related to an increased risk of complications for mother and child during pregnancy, delivery and neonatal life (8-13). Complications for the mother are for example an increased risk for miscarriage, hypertension, preeclampsia, gestational diabetes, a complicated delivery and a higher risk for wound infections and venous thromboembolism. Risks for the offspring are stillbirths and neonatal deaths, macrosomia, complications during delivery and childhood obesity (8-13).

Guidelines on gestational body weight gain exist (14). In these Institute of Medicine guidelines, the importance of a normal body weight before pregnancy and weight gain within certain limits during pregnancy, are emphasized. A normal body weight before and controlled body weight gain during pregnancy are related to ‘keeping in shape’, a better pregnancy outcome and less obesity in the offspring (15,16). For this reason monitoring maternal weight gain during pregnancy has become medical practice by some.

But despite these guidelines, most women do not gain weight as recommended and 20% have weight retention of 5 kg or more between 6 and 12 months postpartum (17-19).

Body weight and weight gain are thought to be the result of a positive energy balance. When energy intake (i.e. food consumption) outweighs energy expenditure (i.e. by physical activity), this positive energy balance will result in weight gain.

In pregnant women, the energy balance is often upset. Pregnant women eat differently and are often less active compared to the prepregnancy period (20,21). This may result in excessive weight gain, and accumulation of body fat (22,23). Many efforts for managing weight (gain) during and after pregnancy have therefore focused on either food intake, physical activity, or a combination of both. However, the best strategy for managing body weight (gain) is currently not clear (24,25).
Obviously, weight gain in pregnancy is a physiological process. Most women gain 10-15 kg, consisting of the weight of the infant (circa 3 kg), adipose tissue (2-5 kg), placenta, membranes and amniotic fluid (circa 2-3 kg), an increase in tissue (uterus and breast (circa 2 kg)), and total body water (circa 3-5 kg) (26-28).

As a clinician it is important to differentiate between an increase in adipose tissue, pregnancy products or the collection of fluid. Furthermore, when maternal adipose tissue increases, it is important to know if the increase is mainly in the visceral or in the peripheral compartment. Excess visceral fat is considered a risk factor for coronary diseases and diabetes (29-31).

Whether the increase in adipose tissue in pregnancy is mainly peripheral or visceral is still under debate (27,32).

In this thesis, maternal weight change during pregnancy and the year after is the central theme.

We have addressed the following questions: What is the general recommendation regarding nutrition (including energy intake) during pregnancy in the Netherlands? What are the changes in body weight during and after pregnancy? How to measure the increase in adipose tissue during pregnancy? What is the relationship between eating style on weight gain in pregnancy, what is the relationship of physical activity on weight change, and how can this relationship be influenced? What is the effect of physical activity on hormonal markers? Finally the question will be addressed how important gestational weight gain is in the perspective of the prevention of obesity.

Outline of this thesis

In chapter 2 evidence is brought together in a narrative review on recommendations for maternal nutrition, including energy intake, during pregnancy in the Netherlands.

The effects of counselling on food intake and physical activity in pregnancy were evaluated in a Randomized Controlled Trial, the New Life(style) study, and results on body weight gain and body weight retention are presented in the chapter 3.

Because food intake is related to certain eating behaviours and these eating behaviours might be characteristic for the individual, the effects of eating style, attitude, social norm and self-efficacy on gestational weight gain were studied (chapter 4).

For a clinician it is important to know by means of a simple, practical test, whether or not body weight gain mostly consists (apart from the pregnancy products) of adipose tissue, because adipose tissue is a risk factor for several diseases. Such a simple practical test is the measurement of BMI or skinfolds. However, leptin is considered as a proxy measure for adipose tissue and might therefore also be used as such a simple test. In chapter 5 concurrent
validity between leptin, BMI and skinfolds was assessed, as a proxy of maternal adipose tissue.

In the IOM guidelines, the importance is emphasized of a normal body weight before pregnancy and weight gain within certain limits during pregnancy. In chapter 6 data from the New life(style) study on body weight change preceding, during and after pregnancy are presented. We studied the effects of smoking, sedentary behaviour and breastfeeding on maternal body weight change, weight retention and pregnancy outcome.

Physical activity is strongly related with body weight. Not much is known about the effect of physical activity in pregnancy on insulin-like growth factor-I (IGF-I) and other hormonal markers, related to the energy homeostasis. In chapter 7 the effect of different levels of physical activity on factors influencing maternal body weight such as maternal glucose, insulin, IGF-I and IGF binding protein 3 were studied.
References