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## Factors affecting weight development after pregnancy — The SPAWN (Stockholm Pregnancy And Women's Nutrition) study

by

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Doctoral thesis:

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#### **ABSTRACT**

Obesity is a growing problem all over the world. For some women pregnancy is a trigger event for developing overweight or obesity and 73% of female patients at the Obesity Unit indicated that they had retained more than 10 kg after each of their pregnancies. For this subgroup weight development after pregnancy was of crucial importance for their future health.

The SPAWN (Stockholm Pregnancy And Women's Nutrition) study is a long-term follow-up study of women who delivered children in 1984-85 in Stockholm (n =2342). 1423 participants completed questionnaires, which covered eating behaviour and exercise, demographic information including social situation and status and details of the pregnancy before, during and up to one year after pregnancy. Fifteen years later these women were invited to take part in the follow-up study (SPAWN). Anthropometric measurements and the same questionnaire data were collected on the 563 women who participated. Detailed drop-out analyses indicate that this subsample was representative. We have also monitored a group of women with gestational diabetes (GDM) and studied sweet food intake in women.

Our main findings are:

Both high weight gainers and high weight retainers had higher BMI values at 15 years of follow-up, although only 56% of the high weight gainers during pregnancy ended up as the high weight retainers one year after pregnancy. Weight retention at the end of the postpartum year, however, predicts future overweight.

Contrary to popular belief and official recommendations, high BMI before pregnancy is not a predictor for weight retention. Women with overweight before pregnancy do not have a higher risk of postpartum weight retention than normal weight women.

Women who are diagnosed with GDM have a considerably higher risk of developing type 2 diabetes mellitus later in life. More active strategies for future weight control and lifestyle advice after delivery might therefore be indicated for women with GDM.

Our SPAWN data furthermore suggests that women with depressive traits have higher intakes of sweet foods, suggesting a link between mood regulation and consumption of in particular chocolate.

Salivary counts of mutans streptococci and lactobacilli are simple, cheap and chair-side tests known to mirror the consumption of fermented carbohydrates and may thus be used as an objective indicator of the sweet food intake in dietary surveys.

#### **THESIS**

The present thesis is based on the following papers, which will be referred to by their Roman numerals:

- Linne Y, Barkeling B, Rössner S.
   Long-term weight development after pregnancy.
   Obesity Review. 2002 May;3(2):75-83.
- II. Linné Y, Dye L, Barkeling B and Rössner S.
   Weight development over time in parous women The SPAWN study-15 years follow-up.
   International Journal of Obesity and Related Metabolic Disorder, submitted.
- III. Linné Y, Dye L, Barkeling B and Rössner S
   Effects of pregnancy on long-term weight development a 15 years follow-up of the SPAWN women.
   Obesity Research, submitted.
- IV. Linné Y, Rössner S.

Interrelationships between weight development and weight retention in subsequent pregnancies - The SPAWN study.

Acta Obstetrica and Gynaecologica Scandinavia, in press.

V. Linné Y, Barkeling B, Rössner S.

Natural course of gestational diabetes mellitus – long-term follow-up of women in the SPAWN study.

British Journal of Obstetrics and Gynaecology, in press.

VI. Barkeling B, Linne Y, Lindroos AK, Birkhed D, Rooth P, Rössner S. Intake of sweet foods and counts of cariogenic microorganisms in relation to body mass index and psychometric variables in women.

International Journal of Obesity and Related Metabolic Disorder. 2002 Sep;26(9):1239-44.

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#### **BACKGROUND**

#### Introduction

#### Obesity as a disease

Obesity is a growing problem all over the world. The present most commonly used measurement of obesity in adults is the Body Mass Index (BMI) which is calculated as body weight (kg) divided by the square of the height (m²). The WHO has defined overweight as BMI over 25.0 kg/m² and obesity as BMI over 30.0 kg/m² and has stated that obesity is spread as an epidemic disease over the world¹. Obesity is associated with co-morbidities such as type 2 diabetes mellitus, hypertension and cardiovascular disease¹.

Type 2 diabetes was previously a disease in elderly people but increasing numbers of young people and even teenagers are being diagnosed with type 2 diabetes<sup>2, 3</sup>.

Weight development during adult life is characterised by a gradual increase<sup>4</sup>. Body weight at one age is strongly correlated with body weight decades later, which suggests genetic influences<sup>5</sup>.

However, the genotypical body weight development depends on environmental influences such as lifestyle<sup>6, 7</sup>, and various life events illness<sup>1</sup> (eg hypothyreosis), long-term medication<sup>8</sup> (anti-epileptics, glucocorticosteroids) and for women, pregnancy<sup>9</sup> can alter the genetically programmed weight gain trajectory.

#### Obesity in women

Obesity is increasing rapidly all among women over the world<sup>10, 11</sup>, and more women in fertile ages become overweight and obese. In Sweden the

percentage of overweight women has increased from 8.8% in 1980/1981 to 11.9% in 1996/97<sup>4</sup>. The largest proportional change was for women aged 16-44 years, among whom the prevalence of overweight has doubled. Overweight women have similar comorbidity as men, but also have some different problems, as listed in table 1<sup>12-14</sup>.

Women who are overweight and obese before pregnancy have a higher risk of complications during pregnancy and delivery.

Infants born by an obese mother have a nearly two-fold increased risk of death within the first year<sup>13</sup>. A Swedish study by Cnattingius et al reported that women who are overweight before conception are at greater risk of having a stillborn child than smokers<sup>15</sup>.

#### **Previous research**

#### Obesity as a result of pregnancy

The phenomenon of weight gain after pregnancy was already recognized in the thirties, when Greene at al described that women gained weight after pregnancy and sometimes increased their weight up to 125% of the weight they had when they married<sup>16</sup>. Some of the first studies were based on demographic surveys showing that women with children have a higher weight than women without children, and that body weight increases with each child<sup>17-20</sup>.

Table 1. Diseases and conditions associated with overweight and obesity.

#### General

Choice of contraceptive pills

Hyperandrogonadism

Amenorrhoea

Dysovualation

Infertility

Hyperinsulinemia

Polycystic ovary syndrome

Depression

Endometrial cancer

Breast cancer

#### **During pregnancy**

Gestational diabetes

Hypertention

Preeclampcia

Dyslipidemia

Impaired endothelia function

Back pain

Preterm delivery

More induced deliveries

Prolonged delivery

Vulval or perineal tears

Caesarean section and its complications

Thromboemublism

#### Effects on baby

Spina bifida

Microsomia

Head trauma

Shoulder dystocia

Brachial plexus lesion

Fracture of the clavicle

Increase perinatal mortality

Haemorrhage after delivery

#### Menopause

Choice of hormone replace therapy

Williamson et al<sup>19</sup> found increasing weight after each child with a steeper increase after five or more children, after correcting for eight confounders. Overweight women world wide have reported that their weight problems started after pregnancy<sup>3-5</sup> and in a study at the Obesity Unit at Huddinge University Hospital, 73% of the female patients reported that their weight problems started with a pregnancy after which they gained more than 10 kg.

## Normal weight gain during pregnancy

Weight gain during pregnancy, due to the foetus, placenta, breast growth and expanded blood-volume, has been calculated to be around 9.1 kg<sup>21</sup>. The rest is an energy reserve for the mother to nourish her child. The mother increases her appetite and lays down a buffer of over 30 000 kcal against the possibility of nutrition deprivation in late pregnancy, when foetal demands increase rapidly. This mechanism may have evolved and been of great protective value at a time when food supplies were uncertain and there was a need for pregnant women to perform

hard physical work, as is still the case in many developing countries today. The actual weight gain has varied over time. In 1944 Chesley<sup>22</sup> reviewed the literature from the 1800's until 1943 and found that the average weight gain during pregnancy was 10.9 kg. In the 1980's several studies took place which found a mean weight gain of 13-15 kg<sup>10</sup>, 23-27

### Recommendations for weight gain during pregnancy

The recommendations for weight gain during pregnancy have changed over time. In the 1950's the recommended weight gain during pregnancy was 7-8 kg, to prevent makrosomia<sup>28</sup> and complicated deliveries. But when caesarean section became more common and alleviated the problem, the recommendations became more liberal. In the USA, the Institute of medicine (IOM) has made recommendations according to pre-pregnancy weight and these recommendations are wide: from 6.8 kg for overweight women to an upper limit of 18 kg for underweight women (Table 2).

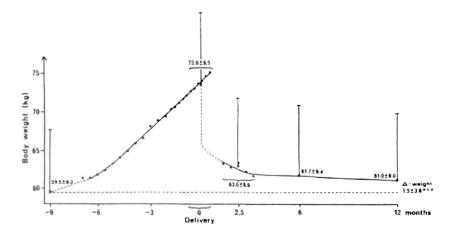
**Table 2**. Weight recommendations from the Institute of medicine (IMO).

Pre-pregnancy BMI	Recommended weight gain
Low pre-pregnancy weight (BMI under 19.8 kg/m²)	12.5-18.0 kg
Normal pre-pregnancy weight (BMI 19.8-26.0 kg/m <sup>2</sup> )	11.5-16.0 kg
High pre-pregnancy weight (BMI 26.0-29.0 kg/m <sup>2</sup> )	7.0-11.5 kg
Very High pre-pregnancy weight (BMI over 29.0 kg/m²)	6.8* kg *Recommended minimum weight gain

#### Weight loss after delivery

Most women lose weight in the first period postpartum. After the initial rapid weight loss period, the weight reduction slows down<sup>10</sup>. In a study by Öhlin et al<sup>10</sup> the pre-pregnant mean weight was  $59.5 \text{ kg} \pm 8.1 \text{ kg}$  (SD) and the mean weight increase during pregnancy was 14.1 kg  $\pm$  4.1 kg. After delivery, weight fell immediately in most women with mean body weights being  $63.0 \pm 3.6$  kg,  $61.7 \pm 3.9$  kg and  $61.0 \pm 3.6$  kg at 2.5, 6 and 12 months respectively (Figure 1). Prentice and Prentice have suggested three hypothetical mechanisms, which could explain why weight loss after delivery does not take place: reduced basal metabolism, impaired thermogenesis or reduced physical activity <sup>29, 30</sup>. However, these concepts have not been reconfirmed by other studies. Spaaij studied in detail and prospectively a group of women throughout pregnancy and lactation and could not find any statistically

significant changes in any of these factors <sup>30</sup>. Furthermore, in a Swedish study women were monitored after removal of a contraceptive device before a planned pregnancy, which allowed proper measurements of anthropometric data before, during and after pregnancy<sup>31</sup>. The changes in resting metabolic rate found were minor and very variable, and these authors underscore the fact that possibly these change are so small that they cannot be properly identified with the methods to measure energy balance, which are technically available and ethically acceptable under these circumstances<sup>31</sup>. A number of studies have found that weight retention is not only a failure to lose weight. Some women actually gain weight after pregnancy 10, 32-35.



**Figure 1.** Body weight development of 1423 women in the Stockholm pregnancy and weight development study from pre-pregnancy until one year of follow-up. Numbers in figure show mean at 2.5, 6 and 12 months postpartum<sup>10</sup>.

#### Weight retention after pregnancy

Most studies show a mean weight gain after pregnancy with a range of 0.4-3.8 kg. It is difficult to compare the studies since they have different follow duration, from 4 weeks to 10 years. Within the studies the follow-up time can also vary with a wide range of months to years. This also implies that the median observation time differ wildly (Table 3 and 4). The classification of weight and overweight also differ, some based on the WHO definition, some based on pre-pregnant BMI classification of the IOM or other. Some of the studies have corrected for one or more factors such as age, the time passing by <sup>10</sup> or socio-economic status <sup>32, 36</sup>. Harris et al have listed 31 different variables which interact with each other and that are hard to rule out (Table 5). Here follows a presentation on the most important studies based on relevance to these thesis:

Williamson et al<sup>19</sup> analysed data from 2547 women in their First National Health and Nutrition Examination Survey. After correcting for 10 confounders, they compared parous women, who however did not give birth during the study period, with nulliparous women, and found that the mean excess weight gain was 1.6 kg for nulliparous and 1.7 kg for the parous women. Women having one, two and three live births the weight gain was 1.7 kg, 1.7 kg, and 2.2 kg, respectively. Among women who were nulliparous at baseline, those that had their live births during the study period gained similar amounts of weight to that of women who began childbearing before the beginning of the study. The risk of gaining more than 13 kg was increased by 40-60%, and the risk of becoming overweight was increased by 60-110% in women having live births during the study. The authors conclude that the

average weight gain associated with childbearing after the age of 25 is quite modest. However, for some women who give birth after the age of 25 the risk of major weight gain and of becoming overweight clearly is increased with childbearing 19 Schauberger et al<sup>37</sup> studied a group of 795 women who were monitored with frequent weight measurements and questionnaires about their activities for six months postpartum. The mean net weight gain from the first prenatal visit to six months postpartum was  $1.4 \pm 4.8$ kg. Weight gain during prenatal care was the variable, most highly correlated to weight loss. Return to work outside the home, parity, and smoking also correlated significantly to weight loss. Breast-feeding, exercise, season of the year, age, and marital status were not related. Route of delivery was associated with weight loss at two and six weeks, but not at six months. Thus, counselling women about weight gain during pregnancy and weight loss requires an understanding of these variables with a long-term perspective of at least six months. Ronney et al<sup>38</sup> made a follow-up study of the Schouberger study. Weight was recorded through medical records with a mean of 8.5 years. Of the original cohort, 540 women had a documented weight beyond 5 years. The average weight gain from pre-pregnancy to follow-up was 6.3 kg. There was no difference in weight gain by prepregnancy BMI. Women who gained less than the recommended weight by the IOM, during their pregnancy were 4.1 kg heavier at follow-up, those gaining the recommended amount were 6.5 kg heavier, and those gaining more than recommended were 8.4 kg heavier. Women who lost all pregnancy weight by 6 months postpartum were 2.4 kg heavier at follow-up than women with retained weight, who weighed 8.3 kg more at follow-up.

**Table 3.** Cross-sectional studies, which have examined the relationship between parity and maternal body weight, modified after Harris et al<sup>39</sup>.

Country Date of collection		Sample size (n)	Numbers of confounders controlled for	Reference	
England	1945-55	5 081	2	Lowe and Gibson <sup>20</sup>	
Scotland	_	5830	2	Thomson and Billewicz <sup>40</sup>	
Sweden	1962-63	378	3	Cederlöf and Kaij <sup>41</sup>	
Netherlands	1980	2092	7	Baecke <sup>42</sup>	
USA	1968-69	755	4	Lee-Feldstein 43	
Sweden	1968-69	1373	8	Noppa and Bengtsson 44	
Finland	1966-72	17688	7	Heliövaara and Aromaa <sup>45</sup>	
Gambia	1978-79	139	1	Prentice et al <sup>46</sup>	
Wales	1965-79	35556	6	Newcombe <sup>17</sup>	
USA	1977-78	1133	8	Forster et al. 47	
England and	1972	7013	5	Rona and Morris <sup>48</sup>	
Scotland					
USA	1981	514	8	Caan et al <sup>49</sup>	
Bangladesh	1975-78	24446	1	Chowdhury <sup>50</sup>	
USA	1971-80	7414	3	Kumanyika 51	
USA	1984-87	1186	3	Kritz-Silverstein <sup>52</sup>	
USA	1987	87	3	Rodin <sup>53</sup>	
USA	1985	41184	6	Brown <sup>18</sup>	
USA	1976	113606	1	Manson <sup>54</sup>	
Sweden	1984-85	1423	2	Öhlin <sup>55</sup>	
USA	1971-84	2547	10	Williamsson <sup>19</sup>	
USA	1991	21	2	Hunt <sup>56</sup>	
USA	1980-90	985	10	Gunderson <sup>57</sup>	
UK	-	76	0	Sultani <sup>58</sup>	

**Table 4.** Longitudinal studies whose primary aim was to investigate the effect of pregnancy<sup>39</sup>.

***************************************					<del>-</del>
Country	Date of	Sample	Time of follow-up	Weight retention	Reference
	collection	size (n)			
England	1949	50	6 weeks	1.0	Beazley and Swinhoe <sup>59</sup>
USA	-	182	6 weeks	2.7 kg	Olsen and Mundt <sup>60</sup>
Canada	1979	371	6 week	5.3 kg	Muscanti <sup>61</sup>
USA	1985-88	274	6 months	4.8 kg	Scholl <sup>62</sup>
England	1957	1110	12-24 months	2.3 kg	McKeown and Record <sup>63</sup>
Scotland	-	5830	18-108 months	05-1.0	Thomson and Billewicz <sup>40</sup>
USA	1989-90	795	6 months	1.4 kg	Schauberger <sup>37</sup>
Netherlands	1980-86	49	9 months	1.6 kg	Rookus <sup>64</sup>
USA	-	345	7-12 months	5.0 kg	Boardley <sup>65</sup>
USA	1988	1592	10-18 months	1.3 kg	Kappel <sup>66</sup>
Sweden	1984-85	1423	1 year	1.5 kg	Öhlin <sup>55</sup>
USA	-	37	9 months	3.8 kg	Parham <sup>32, 67</sup>
USA	1985-91	202	12-60 months	5.3 kg	Smith <sup>67</sup>
USA	-	75	18 months	2.3 kg	Walker <sup>68</sup>
USA	1971-84	2547	24-156 months	1.7	Williamsson <sup>19</sup>
USA	1980-90	985	Median 2 years	2.0-4.6 kg *	Gunderson <sup>57</sup>
USA	1990-2000	540	Mean 8.5 years	6.3 kg	Rooney <sup>38</sup>
USA	1959-65	7116	<6 years	1.5kg	Green <sup>36</sup>

• Dividied by groups based on pre-pregnancy BMI.

Women in the high tertile had retained significantly more weight up through the sixth postpartum month. Parham concludes that a significant portion of maternal weight gain is likely to be retained, putting some women at risk of obesity <sup>32</sup>.

Smith et al<sup>67</sup> examined a total of 2788 women (53% black) aged 18 through 30 years. The women were assessed at baseline and reassessed three and six years later. Women who remained nulliparous (n = 925) during the five-year follow-up were compared with women who had a single pregnancy of at least 28 weeks' duration during that period and who were at least 12 months postpartum at follow-up. They adjusted for four confounders (age, education, smoking and physical activity), and baseline level of adiposity.

Primiparas within both race groups gained 2 to 3 kg more weight during the five-years period than did nulliparas in both adjusted and unadjusted analyses. Primiparas also had a greater increase in waist-to-hip ratio that was independent of weight gain. Multiparas did not differ from nulliparas in adiposity change in either race group. At each level of parity, black women demonstrated greater adverse changes in adiposity than did white women.

Rookus et al<sup>64</sup> studied the effect of pregnancy on the BMI and change in the BMI from pre-pregnancy through nine months postpartum by comparing 49 pregnant women with 400 non-pregnant women. In addition, the body weight of the pregnant women was measured at six and 12 months postpartum. Nine months postpartum the total group of pregnant women had gained as much body mass as was to be expected from ageing.

The same was true for the subgroup of women who did not breast-feed their child or who breast-fed for a period shorter than two months.

Unexpectedly, women who breast-fed

their child for more than two months gained +0.6 kg/m<sup>2</sup> more weight than the non-pregnant women. Compared to the latter group, women who used bromocriptine to stop lactation lost more weight.

Prentice et al<sup>46</sup> studied the energy intake of 143 pregnant and lactating Gambian women measured prospectively each week for a year. Birth weight, breast milk output and anthropometric measurements were also obtained. The average energy intake ( $\pm$ SE) during the dry and wet seasons respectively was: pregnancy,  $1483 \pm 22$  and  $1417 \pm$ 41 kcal/day; lactation (1st trimester),  $1773 \pm 31$  and  $1474 \pm 42$  kcal/day; lactation (subsequent trimesters), 1662  $\pm$  16 and 1413  $\pm$  37 kcal/day. Pregnant women gained 1.4 kg body weight per month in the dry season, but only 0.4 kg in the wet season. Lactating women gained weight in the dry season and lost weight in the wet season.

Maternal nutritional status did not deteriorate with increasing parity. Mean birth weights ( $\pm$  SE) were: 2.94  $\pm$  0.07 and 2.78  $\pm$  0.11 kg in dry and wet seasons, respectively. Breast milk output and quality and early infant growth were close to values from well-nourished communities during the dry season, but deteriorated markedly during the wet season.

Wet season energy intake was clearly inadequate, but these results indicate that, despite being low by international standards, the dry season intake was compatible with a good lactational performance. Except for a somewhat low birth weight, which is partly explained by small maternal stature, progress during pregnancy was also remarkably normal during the dry season.

Harris et al<sup>34, 39, 69-73</sup> studied the independent associations between parity and maternal BMI, and between parity and maternal weight gain.

**Table 5.** Confounders identified by Harrison <sup>74</sup>.

- 1. maternal age
- 2. marital status
- 3. maternal height
- 4. heterozygosity (monozygotic twins)
- 5. education
- 6. occupation, social class and employment status
- 7. urbanisation
- 8. religion and church attendance
- 9. ethnicity
- 10. size and housing
- 11. husband's age
- 12. income
- 13. capacity for work
- 14. smoking status
- 15. fertility
- 16. husband's BMI
- 17. obesity during adolescence and adult life
- 18. age at menopause and/or menopausal problems
- 19. birthweight of previous child
- 20. body weight during first pregnancy
- 21. household size
- 22. age at menarche
- 23 contraceptive and/or HRT use
- 24. alcohol use
- 25. physical activity
- 26. medical history and health status
- 27. family history of breast and/or endometrial cancer
- 28. menstrual cycle rhythmicity
- 29. dieting and weight cycling
- 30. lactation
- 31. interbirth interval

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They examined the change in maternal body weight from the beginning of one pregnancy to the beginning of the next. The study population was a group of 523 multiparous women who had been weighed regularly during pregnancy, and none of whom had become pregnant less than 12 months after the birth of their previous child. Parity was found to be independently associated with maternal BMI. Gestational weight gain and inter-pregnancy weight gain were also associated. Women of different parities were found to be at differential risk of long-term weight gain for two reasons. First, primiparous women are at risk of long-term weight gain because they gain the most weight during pregnancy, and high gestational weight gain is in itself a risk factor for long-term weight gain. Second, women of higher parity (4+) are at risk of longterm weight retention because they gain more weight in association with each pregnancy, irrespective of the amount of weight they gain during their pregnancies. For women of parity 3 or less, the association between maternal body weight and parity appears to be the result of cumulative weight gain during successive pregnancies. For women of greater parity, the association between maternal body weight and parity is partly the result of cumulative excess gestational weight gained during successive pregnancies, and partly the result of gaining more weight from the beginning of one pregnancy to the next at later pregnancies. Harris et al<sup>39</sup> have also studied the psychosocial and behavioural changes in 74 mothers. They found there was some evidence that lifestyle changes which accompany pregnancy and motherhood, increase some women's vulnerability as regards psychopathology. Mothers who did less physical activity after pregnancy than before have a grater risk for weight gain after delivery. Harris et al<sup>75</sup> have also

studied women from South Africa, where obesity among young women is common. They found no relationship between pre-pregnant weight and weight retention up to 2.5 years after delivery.

Öhlin et 10, 55, 76, 77 studied 1423 women up to one year after delivery. The women gained on average 0.5 kg but with a wide rage and 1.5% of the women retaining at least 10 kg. Large variances meant that when statistically significant relationships were found these were rather weak. The most important risk factor for sustained weight gain/retention one year after delivery was weight increase during pregnancy.

Usually viewed as a key factory in postnatal weight control, lactation frequency was significantly associated with post-partum weight change but the relationship was also very weak r=0.09 (p<0.01). Age, irregular eating habits and physical inactivity after delivery were also found to explain some of the variance in weight retention after delivery, but only to a small extent. As expected, women who gave up smoking when they learned that they were pregnant retained more weight than never/non-smokers. Furthermore, women who had experienced a change in weight of  $\pm$  6 kg due to previous pregnancies were more likely to retain weight  $(2.5 \pm 4.9 \text{ kg (p} < 0.05))$ . Prepregnant body weight, parity, contraceptive practices, social class, occupation, marital status, nationality and dietary advice during pregnancy were not found to predict body weight one year after delivery<sup>77</sup>. Gunderson et al<sup>57</sup> examined differences in the pattern of weight changes during and after pregnancy among four prepregnancy BMI groups in 985 women.

Maternal body weights were available before conception and delivery, and at six weeks postpartum for the first (index) pregnancy, and weight before

for the second was registered. Height and two pre-pregnancy weights were self-reported. Weights at delivery and six weeks postpartum were measured. Net delivery weight was defined as delivery weight minus infant birth weight. Three non-overlapping sequential weight changes were constructed: (1) net gestational gain (net delivery weight minus pre-pregnancy weight at the index pregnancy); (2) early net postpartum weight change (6weeks postpartum weight minus net delivery weight); and (3) late postpartum weight change (prepregnancy weight at the second pregnancy minus 6-weeks postpartum weight). Means adjusted for eight covariates (parity, race/ethnicity, education, mode of delivery, smoking, hypertension of pregnancy, age, height) and time intervals were not altered appreciably. Early net postpartum weight losses were similar for all pregravid BMI groups. Late (median of two years) postpartum weight losses were four kg higher in the low and average BMI groups, compared with the highest BMI group. About half of the net gestational gain was lost by six weeks postpartum, and the percentage that was lost decreased over time. Gunderson concluded that early postpartum weight loss does not vary by maternal pre-pregnancy BMI group, but late postpartum weight change does. Serial weight measurements are needed in epidemiological studies to differentiate retention of gestational gain from weight gain during the late postpartum period.

Even if the mean weight gain is not pronounced, the range is wide, from a weight loss of 13.6 kg to a weight gain of 29.5 kg<sup>39, 68</sup>. Parhan et al have shown in a study of 37 women that 11 % of the women were more than six kg heavier nine months after delivery. Kappel et al<sup>66</sup> found in data from 2845 women

that 25% of white women and 40% of the black women had gained more than nine pounds at 8-10 weeks after delivery. Öhlin et al found in 1432 women that before pregnancy 13 % were overweight and that these numbers had increased to 21% at one year postpartum 10. On the other hand, Williams et al <sup>19</sup> have compared nonpregnant and pregnant women regarding weight gain and shown that nonpregnant women gained more weight than those who gave birth, although these women were followed for only a short time period. Furthermore, in a Swedish study Hallberg et al<sup>78</sup> compared women with no, one, two and three children in three different age groups and found no weight differences.

# Factors during and in the near postpartum year which effects weight retention

#### Weight before pregnancy

A number of studies have looked into the relationship between pre-pregnant weight or BMI and weight gain during pregnancy <sup>25, 79-83</sup>. Some found no consistent relationship between prepregnant weight and weight gain during pregnancy<sup>23, 80, 81</sup>. Abrams et al<sup>25</sup> evaluated 4674 women with "Good" pregnancy outcomes. Pre-pregnancy BMI groups were constructed as underweight (BMI<19.2), normal weight (BMI 19.2-25.6), overweight (BMI 25.6-28.9) and very obese (BMI>28.9). Compared to the normal weight group, the underweight group gained on average 0.65 kg less, while the very obese group gained two kg less. But the variation was higher in the very obese group.

Kleiman et al<sup>82</sup> analysed a subsample of the 1980 NNS (National Survey) data.

The sample was divided into groups according to body mass: low (BMI < 19.8), moderate (BMI 19.8-26.0) and high (BMI >26.0). For some analyses, a fourth group labelled "very high" with a BMI > 29 was included.

Results of multiple regression analysis showed that there was a very small difference in mean weight gain between low and moderate BMI groups, but women in the high BMI group gained 2.7 kg less than the two lower groups. Öhlin et al<sup>10</sup> have shown that the degree of weight retention up to one year varies more in overweight and obese women (Figure 2).

#### Weight gain during pregnancy

High weight gain during pregnancy is the most important risk factor for high weight retention after pregnancy <sup>55, 70, 84, 85</sup>. These increases in weight have added very little to the birthweight of the child<sup>45-47</sup>. There is also a relationship between the length of the gestation and the weight gained during pregnancy<sup>27</sup>. Here is a methodological problem.

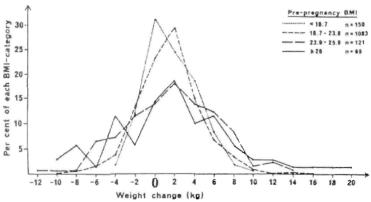
If weight retention is studied too soon after delivery the women do not have had enough time to lose the weight they naturally lose.

Measurements of body weight shortly after delivery 61, 66, 86 may indicate falsely high weight retention However, if the studies are conducted for longer time periods, it is likely to assume that the impact of the pregnancy and birth is lost among numerous other factors affecting the lifestyle of the mother <sup>19, 67</sup>. Thus, the ideal point in time to determine when the pregnancy has had its overall effect on weight development is controversial. A study by Greene et al including 7116 women examined weight gain between pregnancies<sup>36</sup>. All women had more than one child. In this study, women who gained the most during pregnancy had a higher risk of weight gain in the interpregnant interval.

#### Smoking and smoking cessation

Smoking increases the basal metabolism with about 10%<sup>87</sup> and those who quit smoking usually gain weight<sup>88</sup>.

Smoking is not only detrimental for the mother's health but also for the child<sup>15</sup>. Smoking cessation, which fortunately often occurs when women learn of their pregnancy, will also make women retain more weight<sup>10</sup>.



**Figure 2.** Distribution of weight change one year postpartum compared with pre-pregnancy body weight in groups with different pre-pregnancy BMI.

In a study by Öhlin et al $^{55}$ , women who gave up smoking when they learnt about their pregnancy retained more weight than others,  $3.4 \pm 4.3$  kg (p<0.01). Also Kleinman et al $^{82}$  found that mothers who quit smoking gained 0.5 kg more and that women who continued to smoke during pregnancy had a higher likelihood of gaining less than 6.8 kg. Also other studies have shown that mothers who smoked during their pregnancy gained less weight per week $^{89}$  compared to non-smoking mothers, and that women who continued to smoke during their pregnancy tended to have lower long-term weight gain $^{10,36,37}$ .

#### Socio-economic factors

Socio-economic factors are strong predictors for overweight and obesity in general 90. On the other hand, other studies that have measured socio-economical status by occupation or social class have found no significant associations 10, 36, 37. Most studies show that women with lower income or less education are at increased risk of retaining weight postpartum 68, 70, 91. Greater weight retention has been observed among unmarried women when compared to married women 33, 70, 91.

#### Lactation

In clinical practice mothers have generally been informed that full-time breastfeeding will be helpful to revert to pre-pregnancy weight. This seems logical, since a full lactation requires about 500 kcal/day, which for many women in this age group represents about 20% of their daily energy requirements <sup>92</sup>. From an evolutionary point of view it seems reasonable to assume that the adipose tissue deposition during pregnancy would serve as a nutritional reserve to ensure an adequate energy supply for the newborn. However, in reality breastfeeding has been found to be of surprisingly little help in postpregnancy weight control. Although some studies have shown that lactation increases weight loss after delivery 93, 94, other studies show little or no such influence 17, 64, 89, 95-97. To some extent this may reflect methodological shortcomings. Lactation has not been well defined and the total amount of energy lost through lactation has not been possible to quantify with any precision. In an attempt to overcome this inherent problem, a simple scoring system was developed in the Stockholm Pregnancy and Weight Development Study, which was meant to reflect both duration and intensity of breastfeeding. The women scored both the number of months they were lactating and whether their breastfeeding was partial or complete. This system made it possible to sum up a so-called lactation score, which would roughly correspond to the total energy of the milk. With this method the expected relationship observed was found, but was very weak  $r=0.05 (p<0.01)^{55}$ The fact that lactation in well-nourished societies seems to play little role for future weight control after delivery can be explained by several factors. It is possible that the changes in eating behaviour, with food being abundant, and a new lifestyle, encourage women to take in the extra energy needed by their eating habits rather than using their stored energy reserve.

#### Diet and dieting

Even though no women who become pregnant nowadays are recommended to eat for two, weight gain of any kind is a result of an increase of energy intake in relation to energy expenditure. Changes in dietary intake are common among pregnant women and in the postpartum period<sup>70,77</sup>. Harris et al found that more than 70% of the women felt that they ate differently than before pregnancy <sup>71</sup>. Öhlin et al found that women who displayed a less well structured eating pattern at one year after delivery retained more weight<sup>77</sup>. Women who increased their body weight also reported that their energy intake had risen during pregnancy and that they ate larger

portions and snacks more frequently. These women also skipped breakfast and lunch more frequently.

Both Öhlin et al and Schauberger et al have shown that many women undertake dieting in some form during the first year after delivery<sup>37,77</sup>. However, it is not clear whether such active dieting makes women return to their pre-pregnancy weight.

#### Physical activity

Little information is available regarding the potential importance of physical activity in affecting weight development during and after delivery. A number of methodological issues cloud the association 98. Studies are generally uncontrolled. The amount, activity and intensity are generally not well reported. Recall bias may be a problem and many women might give embellished pictures of reality, when they report on the change in lifestyle that has followed after pregnancy and childbirth. Early return to work has been associated with less weight retention. Schauberger et al found that women who went back to work within two weeks after delivery retained less weight at 6 weeks of follow-up than those who stayed at home<sup>37</sup>. However neither Öhlin et al<sup>77</sup> nor Harris et al<sup>39</sup> found any association between post-pregnancy physical activity and long-term weight development. Harris at al found that women who reported less physical activity after pregnancy gained 3.1 kg after pregnancy<sup>39</sup>. Samselle et al<sup>99</sup> found that women engaged in physical activity gained less at 6 weeks postpartum.

#### Heredity and ethnicity

Obesity is found to have strong genetic influence<sup>5</sup> and ethnicity plays an important role for the development of obesity and comorbidity. Few studies have looked upon the genetic influence on weight retention after pregnancy. Studies suggest that if a woman's mother is obese, she is more likely to retain weight after pregnancy <sup>100</sup>,

<sup>101</sup>. Harris et al assessed the relative importance of heritable characteristics and lifestyle in the development of body weight following pregnancy. Seventy-four mothers were followed up to 2.5 years after delivery. They were asked to score the degree of obesity in their parents, using a serie of nine silhouette drawings. Women who scored that their mothers were overweight and obese were more likely to retain more weight after pregnancy <sup>102</sup>. However there is no genetic study on factors affecting weight retention after obesity.

The few studies published on ethnicity and weight retention are exclusive in black and whites in the

United States <sup>66-68, 91, 103, 104</sup>. These studies consistently show pregnancy related weight gains to be greater in Afro-American than in white women even after correcting for confounders.

#### Age and parity

Weight goes up with age<sup>4</sup>. However there is no strong evidence that older mothers gain or retain more weight after pregnancy. Some studies show no effect at all <sup>36, 71</sup> and some show a negative influence <sup>10, 33, 35</sup>. Wolfe et al 104 show that there was a different weight retention pattern in white and black women. Older white women gained more than younger white women and the opposite happened in black women <sup>104</sup>. Janney et al<sup>33</sup> showed that age rather than parity influenced the rate of postpartum weight retention in the first and second pregnancy<sup>33</sup>. The same result was showed by Friis et al<sup>105</sup>. Older women were more unlikely to return to their prepregnant weight than younger women. The older women also showed a slower rate of weight loss than the younger one. The interaction between age and parity is complex. After accounting for difference in maternal age, an effect of parity on bodyweight is only consistently observed at high

parities 19, 40, 70.

Harris et al<sup>70</sup> showed that first time mothers are at risk for long-term weight retention because they gained the most during pregnancy<sup>70</sup>. However women with more than four children seem to also to have a higher weight retention because they gained more weight in association with each pregnancy irrespectively of the amount of weight they gained during pregnancy<sup>70</sup>.

Lederman et al showed that an increase in weight gain observed with increasing parity is the result of women of higher parity, having their pregnancies at older ages <sup>106</sup>. However advanced analyses have shown the effects of parity on body weight to be independent of maternal age <sup>19,70</sup>. Howevere, mothers of high parity might gain more weight in association with pregnancy as a result of different effects of motherhood in different parities.

#### **Psychology**

Very little has been published about psychosocial factors and weight retention after pregnancy. To have children must be one of the most stressful situations for women and men. Harris et al<sup>39, 84</sup> and Walker et al found that high levels of stress or low levels of social support to be associated with high weight retention after pregnancy <sup>68</sup> 107. Walker at al also showed that women think that the extra weight they have put on, lower their self-esteem<sup>68</sup>. Harris et al have examined factors regarding body image and change in body image of women during pregnancy. Women who were dissatisfied with their body image were more likely to have a higher weight retention after delivery<sup>39</sup>. Harris concluded that these associations suggest that either increased body weight or dissatisfaction predisposes mothers to gain more weight or that alternatively mothers who gain more weight are more dissatisfied.

#### Gestational diabetes

Obesity and gestational diabetes are closed linked together<sup>2, 3</sup>. It is clear that women with gestational diabetes (GDM) have a higher risk of developing type 2 diabetes melltius (DM). Although it is generally stated that 1-3% of women develop GDM during pregnancy 108, 109, the further development into a clinically manifest type 2 DM seems to follow different routes. The risk depends on numerous factors: Some of these are technical, such as the diagnostic criteria, which have been the cause of much international debate 110. Some factors clearly cannot be modified, such as ethnicity, pre-pregnancy, weight, age, parity, family history of diabetes, degree of hyperglycaemia in pregnancy and immediately post-partum<sup>111</sup>. Other risk factors can be modified after a pregnancy, such as future weight development and further pregnancies<sup>10</sup>. In earlier studies GDM women have been

In earlier studies GDM women have been reported to be heavier than matched pregnant women with a normal glucose tolerance <sup>112</sup>. Weight gain during pregnancy has on the other hand been found to be about 1.5 to 2 kg less in GDM women than in controls <sup>112-114</sup>. A similar pattern was found, in that GDM women are heavier at conception but gain less during pregnancy. Since some of these studies were retrospective and weight at conception, as is generally the case, was self-reported, there remains some uncertainty, but the pattern seems to be consistent.

Since this group of women is at high risk for type 2 DM, it is pertinent to also monitor long-term weight development in parallel to the natural history of type 2 DM.

#### Sweet food intake in women

Whether the intake of sweet foods is correlated to BMI in women is controversial. The majority of crosssectional food surveys has shown an inverse relationship between sugar intake or sugar-fat intake and BMI in women 100, . However, in a study by Macdiarmid et al<sup>119</sup>, the relationship was reversed when clear under-reporters were excluded. indicating that under-reporting plays a crucial role for the results. Under-reporting is a concern in all dietary surveys and especially for foods which are considered to be unhealthy, like sweet foods <sup>120, 121</sup>. Furthermore, under-reporting is a major problem in dietary studies of obese subjects 9. Apparently, there is a great need for objective tests for the intake of sweet foods <sup>122</sup>. The salivary counts of the cariogenic microorganisms mutans streptococci and lactobacilli are known to be positively related to the sugar consumption and may therefore be used in attempts to more objectively describe the sugar intake <sup>123-125</sup>. In a recent study by Barkeling et al <sup>126</sup>, the reported premenstrual intake sweet foods and the number of mutans streptococci were significantly higher in obese women compared to normal weight subjects.

Sweet food intake and psychometric variables

It has also been suggested that there is a link between mood and food, and that the use of sweet carbohydrate-rich foods and chocolate has been regarded as candidates for mood regulation (for a review see <sup>124</sup>, <sup>127, 128</sup>). In clinical and experimental studies it has been suggested that these foods sometimes are used as self-administered "medicine" in subjects with different affective disorders such as depression, seasonal affective disorders, pre-menstrual syndrome and also in the obese (see <sup>127, 128</sup>). However, the underlying mechanisms

mediating this mood regulating effect, lack scientific support <sup>127, 128</sup>. There are no cross-sectional data from the general population available where the actual intake of sweet foods is linked to the psychiatric status of the population of women.

#### **AIM OF THE STUDY**

The overall aim of the SPAWN (Stockholm Pregnancy And Women's Nutrition study) was to examine factors during and in the near postpartum year that effect weight development after pregnancy (Papers I-V).

#### Furthermore:

- To evaluate those women whose weight trajectories changed after their pregnancies and in particular in those women who were normal weight before pregnancy but became overweight at 15 years of follow-up (paper II).
- To compare weight and weight development at three presumably critical time periods; before, during and one year after pregnancy in order to evaluate which time period is most predictive for further weight development (paper III).
- To examine and evaluate risk factors for weight development in the first pregnancy and their impact on the second pregnancy (paper IV).
- To assess to what extent GDM women develop clinical type 2 diabetes later in life, and to what extent that development could be predicted, and if the impaired carbohydrate metabolism is related to other aspects of the metabolic syndrome (paper V).
- To measure the intake of sweet foods in a large cross-sectional sample of women, using subjective as well as more objective measurements, and describe how this intake is related to BMI and to psychometric variables reflecting mood (paper VI).

#### **METHODS AND SUBJECTS**

#### Study design

A longitudinal study of women's weight development during pregnancy, at one year postpartum and at 15 years of follow-up (Figure 3).

#### Selection procedures 1984-85

Women were selected at the check-up 2.5 months after delivery when they routinely visited the maternally clinic. All mothers who during one year, came to the maternity clinic for the last routine control, i.e. 6-15 weeks after the delivery, were invited by their midwives to take part in the study. It included two additional visits to the maternity clinic at 6 and 12 months after delivery. Women who were going to move within a short time and those with obvious language and communication problems were not invited. The sample represents a mixed metropolitan population from both the inner city area and suburb districts with a distribution in social groups that can be assumed to correspond to the population in the Stockholm area. Ninety-six per cent of the entire sample was Swedish citizens and the rest came from miscellaneous. mainly nordic countries.

#### **Subjects**

A total of 2342 women accepted to participate in the study. Forty-seven women were excluded for different reasons: 20 had twin births, 12 used insulin during pregnancy, 12 had gastrointestinal problems with severe energy losses (heavy vomiting or diarrhoea) and in three cases prepregnant body weight data was not available. Information about the women

until 2.5 months post-partum was thus available in 2295 cases.

The drop-out frequency during the first post-partum year was 38 per cent (872 cases). Fifteen per cent dropped-out from the 6 months visit and 23 per cent from the 12 months visit post-partum. For the majority (674 cases) the reasons for dropping-out were unknown, 89 women were pregnant again, 60 had moved or were on holiday, 41 reported lack of interest or unsuitable time, and eight reported miscellaneous reasons.

### Data collection in the first year after delivery

Data was collected concerning age, parity, body weight (before and during pregnancy and 2.5, 6 and 12 months after delivery), weight retention after previous pregnancy, experience of slimming, lactation pattern, contraceptive practices, profession and smoking habits.

The data was collected from pregnancy and delivery routine records from the maternity clinics and from questionnaires at 6 and 12 months after delivery. The staff at the maternity clinic administrated the routine records and the questionnaires. They also sent out invitations and if necessary reminders by mail or telephone, before the two follow-up visits at the maternity clinic.

#### Weight measurements

The women were weighed by the staff at the maternity clinics on the same scale without shoes and in light indoor clothing to the nearest 0.1 kg. The scales were calibrated in each maternity clinic. The women self-reported their

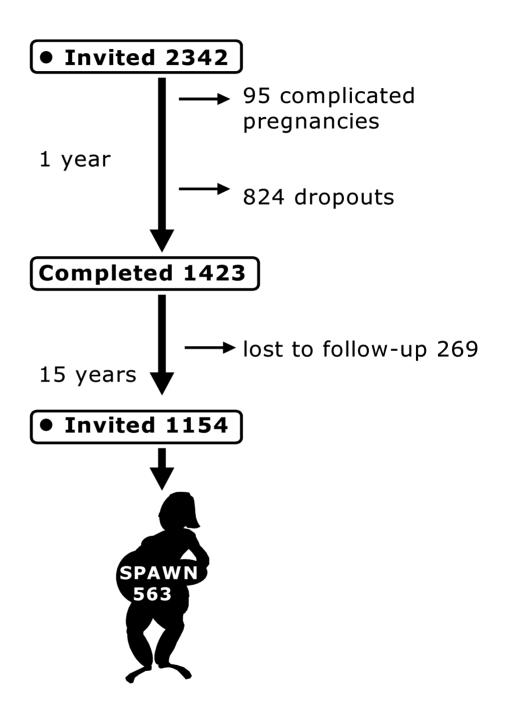


Figure 3. The design of the SPAWN study.

body weight and height before conception on their first visit to the maternity clinic, usually between gestation weeks 8 and 12. The prepregnancy BMI was calculated from self-reported weight and height. Thus, in total, for 1423 women all material was obtained and could be included in the study up to one year post-partum.

#### **Lactation score**

A scoring system was constructed in order to express duration and intensity of breast feeding <sup>55</sup>. Every month with full lactation was given four points, and every month with mixed feeding was given two points. The total sum (0-48) indicated the individual lactation amount. This score was used as a rough estimate of the total energy expenditure for milk production.

## Eating habits and physical activity

Questionnaires were filled out on eating habits and physical activity at 2.5, 6 and 12 months.

The questionnaires included eight questions on diet and two questions about physical activity at work and in leisure time. The questions were retrospective, of multiple choice type and usually with four answer alternatives. The diet questions concerned mealtime regularity, frequency of breakfast, lunch and snacks, number of cooked meals, breakfast components, self-perceived change of meal sizes and/or snacking and adherence to a special diet. Each question was answered for each of the four periods in the study: in the questionnaire used 6 months postpartum questions were asked concerning the situation before pregnancy, during pregnancy and 1-6 months after pregnancy. The 12 months questionnaire concerned the time period 7-12 months postpartum.

#### Socio-demographic factors

Data was collected concerning age, parity, marital status, occupation, living area, and duration of maternity leave.

### Data collection at 15 years of follow-up

All women (n=1423) who participated in the first part of the study and completed the whole study were invited to the follow-up. These women were tracked by the Sema info-group using the individually characteristic national unique twelve digit identity number, which gives access to their current addresses. 269 women could not be found because of incomplete identity number, location out of Sweden or death. 1154 women were identified and letters sent, and eventually 563 women agreed to participate in the follow-up study.

#### Procedures at 15 years of followup

The women who still lived in the Stockholm area (n=363) were asked by letter to attend Huddinge University Hospital for objective measurement of height and weight and to fill in the questionnaires under the supervision of a researcher or a Unit nurse. At the hospital anthropometric measurements were taken; weight was recorded to the nearest 0.1 kg and height measured to the nearest cm. Diastolic and systolic blood pressures were measured in a lying position after 5 minutes of rest. Waist circumference was measured in a standing position halfway between the lowest costal arc and the ileca crest in the normal respiratory position and hip circumferences over the widest part of the hips were measured. Body mass

index (BMI) and waist/hip ratio (WHR) were calculated. Body fat was determined by bioimpedance (Tanya TBF 305, Tokyo, Japan). Blood sample was drow for genetic and metabolic analyses (data not presented in this thesis).

Of these, 200 women lived too far away from our clinic to attend in person. They therefore completed the same questionnaire but weight and height were self-reported rather than objectively measured. There was no difference between these two groups regarding variabels during pregnancy, included clinical parameters, such as fecundity history, high blood pressure, oedema, delivery methods, weight gain during previous pregnancies, dietary advice during pregnancy or age and BMI at follow-up.

Data used in paper IV was also retrieved from the national Swedish Medical Birth Register (MBR) which contains information of all births in Sweden since 1973, including weight gain during pregnancy, illnesses during pregnancy, anthropometric information regarding the newborn child and illnesses in association with delivery 129. This register covers more than 99% of all children born in Sweden.

In the original study protocol, women who developed gestational diabetes mellitus (GDM), were excluded. It was assumed that weight development of this subgroup could differ from that of a normal pregnancy. At follow-up 15 years later, however, it was realised that data from this subsample could provide interesting information about weight development over time in GDM women.

For this study (paper V), all women who were diagnosed with GDM in 1984-85 and gave birth in southern Stockholm were sampled from the Swedish medical register<sup>130</sup>. The diagnose GDM was made after a

general screening of all pregnant women by urine-glucose test. Women with glucosuria went through a two hours oral glucose test with 75g glucose test. The diagnosis GDM was made if the women had a two-hours values over 9 mmol/l. The GDM women were then followed by the maternal specialist clinic. Sixty-four such women were identified, six of them as type 1 diabetes mellitus and were thus excluded. From the SPAWN study 150 women at random (every 5<sup>th</sup> woman) were identified to form a control group. Twenty-eight subjects in the GDM group who volunteered to participate in our study did not differ significantly in age compared to the 30 GDM women, identified through the national register, who did not wish to participate in the study. Fifty-two SPAWN control women who volunteered to participate were not significantly different in age, weight before pregnancy or weight gain during pregnancy compared with the 98 who did not wish to participate in the study.

All women were invited to the Obesity Unit and came for an oral glucose tolerance test with 75 g glucose after a night of at least 8 hours of fasting. The diagnosis diabetes mellitus was made if the women had an two-hours bloodglucose value over 10 mmol/l. Blood was sampled immediately before the glucose load for fasting glucose, insulin and liver tests and other routine lab samples. Glucose was sampled every 30 minutes for two hours; on the first and last occasion. Blood insulin was also measured. Subjects were asked to fill in questionnaires about their eating behaviour, exercise habits, social conditions and medical history and questions about their knowledge of risk for type 2 DM.

Case records for the index pregnancy supervision were obtained from all maternity units after permission from all participating women had been obtained.

Cariogenic microorganisms was measured by using paraffin-stimulated whole saliva (paper VI). The saliva samle was collected at the subjects' home during five minutes in the morning (before eating and toothbrushing). Of the 362 women, saliva samples was available from 303 individuals and of these 11 were discarded since these women had used antibiotics during the past two months. One milliliter (ml) of the saliva was transferred to 4.2 ml of VMG transport medium <sup>131</sup>. The sample was sent by mail to the Department of Cariology in Göteborg and was processed within 24 hours. It was dispersed on a Whirlimixer for 20 seconds, serially diluted in 0.05 M phosphate buffer and plated on MSB agar<sup>132</sup> to estimate number of mutans streptococci and in Rogosa SL agar to estimate lactobacilli. The MSB agar plates were incubated in 95%  $N_2$  and 5%  $CO_2$  at  $37^{\circ}$ C for two days and the SL agar plates aerobically at 37°C for three days. The number of colony forming units (CFU) of mutans streptococci on MSB agar were counted and identified by their characteristic colony morphology <sup>25</sup>. All CFU in SL agar were regarded as lactobacilli.

### Questionnaires at 15 years of follow-up

At 15 years of follow-up participants completed a questionnaire containing questions about their eating behaviour, repeating the previous dietary questions about meal pattern and meal quality and questions about physical activity at work and leisure time. Also questions about experience of slimming (methods and kg of weight loss) and unintentional weight lost (reason and kg), a brief medical history and medication, additional births, smoking habits and smoking cessation in generally and

during pregnancy, body and self image, educational levels, occupation, working hours, total family income, marital status were included. The SPAWN women also reported weight before and one year after previous and later pregnancies apart from the index pregnancy, age and sex of the children and furthermore, if any child was adopted.

In paper VI a self-administered questionnaire was used. In the first part, general questions about the menstrual cycle (i.e. cycle regularity and length), hormone treatment and intake of sweet foods were included. In the second part, the subjects were asked to quantify both their habitual intake of sweet foods and their intake during the menstrual cycle, when they ate more sweet foods than usual (so-called "extra large" intake). The questions about the intake of sweet foods were adapted from the validated SOS (Swedish Obese Subjects) dietary questionnaire <sup>133</sup> and covered the intake during the last two months. The following sweet food products were included: sweets, chocolate, cake, pastries, biscuits and ice cream. The number of different sweet foods and the sizes and/or weight, eaten per day or per week was registered. Sweets and chocolate were also quantified by using pre-confectioned packages sold in Sweden. The amounts were then converted into grams, from which the daily intake of energy, fat and sugar (mono+disaccharides) was calculated. The nutrient values in the database were obtained from food tables from the Swedish National Food Administration and from the manufacturers. The habitual daily intake of sweet foods was calculated for all subjects. The extra large intakes were calculated for all subjects reporting a higher intake of sweet foods during any part of the menstrual cycle. Since the extra large intakes for the vast majority of the

subjects (85%) were pre-menstrual, extra large intake and pre-menstrual intake are used synonymously.

#### Psychometric variables (Paper VI)

The self-rating Scale for Affective Syndromes (CPRS S-A), derived from the CPRS (Comprehensive Psychopathological Rating Scale), was used. The instrument consists of 19 items in self-report format, which forms three subscales designed to measure symptoms (state aspects) of depressive (MADRS: Montgomery-Asberg Rating Scale), anxiety (BSA; Brief Scale for Anxiety) and obsessive-compulsive (CPRS-OCD scale) syndromes. For each of the 19 variables, the subjects rated the degree of symptoms on a fourstep scale with end-points from "absence of symptoms" (score 0) to "extreme degree of symptoms" (score 3). The state aspect of the instrument is stressed by information to rate the condition for each variable during the recent three days. The CPRS S-A is a useful and reliable instrument for quantitative rating of symptoms in clinical outpatient settings and has previously been validated 134, 135 A score higher than six in the subscales indicates mild symptomatology, with an increased risk to fulfil criteria for a mood disorder according to DSM-IV (Diagnostic and Statistical Manual of Mental Disorders)<sup>136</sup>.

SPAWN study we have made drop-out analyses from two different aspects. One is the drop-out analysis from the pregnancy and the other drop-out analysis performed was to sample information from Statistic Sweden. Result is presented in paper I

#### Statistical analyses

The statistical methods used in the studies were in general parametric statistics. All analyses were performed using SPSS 10.5 for Windows (SPSS Inc). In paper VI non-parametric statistics were used, since data was not normally distributed. Analysis of variance with repeated measurements was used to examine the change in body weight at different time points of assessment. Between group factors based on weight change and parity were included in the separate analyses. Greenhouse-Geisser corrections were used where sphericity could not be assumed. Student's t-test and ANOVA were used for analyses of continues variables, and Chi<sup>2</sup> test was used to examine associations between categorical variables.

#### **Drop-out analyses**

Of the 269 women not available for follow-up 17 women had died during the follow up period and 106 women had emigrated. For the other women data was incomplete or wrong personal number had been given.

In a study with such a long follow-up time as 15 years it is important to perform drop-out analyses. In the

#### **RESULTS**

#### Paper I

### LONG TERM WEIGHT DEVELOPMENT AFTER PREGNANACY

In this paper we present the background for the study, drop-out analyses and some basic results from the SPAWN study. In summary of the background there is some evidence in the literature for pregnancy as a cause of obesity, but there is still no single strong factor that can explain the weight retention after pregnancy.

In a study with such a long follow-up time as 15 years it is important to perform drop-out analyses. In the SPAWN study we have made drop-out analyses from two different aspects. One is the drop-out analysis with variables from the pregnancy where subjects have been divided into four groups, depending on if they dropped out during their index pregnancy or after pregnancy, participated in this study by coming to the clinic or sent in forms. Regarding the clinical parameters, such as infertility history, high blood pressure, oedema, delivery methods, weight gain during previous pregnancy, dietary advice during pregnancy or the social factors like working status, alcohol use and smoking there were no significant differences between the four groups ( $X^2$ -test). The only statistically significant difference observed was for nationality, where fewer Nordic women dropped out. The other drop-out analysis performed was on information from Statistics Sweden<sup>137</sup>. The data was taken out of the LOUISE (longitudinal database on education, income and occupations) register which contains

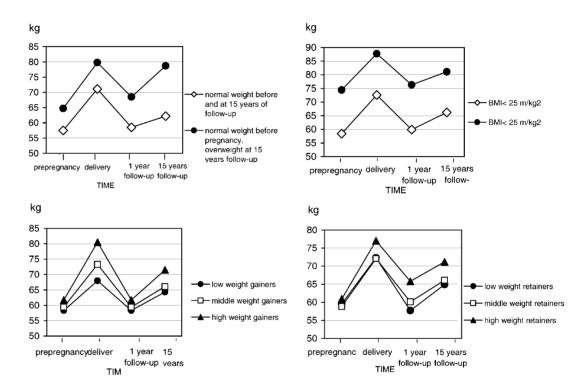
data on social and living status on a yearly basis based on all individuals living in Sweden up till 1999/12 /31. Data indicate that the SPAWN population seems reasonably representative for the entire group initially recruited.

#### Paper II

## WEIGHT DEVELOPMENT OVER TIME IN PAROUS WOMEN- THE SPAWN STUDY - 15 YEARS FOLLOW-UP

In this paper we examine whether pregnancy had any impact on weight gain in women who are shifting from normal weight to overweight during the fifteen years. The sample was divided into two main groups, those who were normal weight before pregnancy and remained normal weight, and those who were normal weight before pregnancy and became overweight at 15 years of follow-up.

Women who became overweight had a higher pre-pregnant BMI (22.3  $\pm$  1.5  $kg/m^2 vs 20.5 \pm 1.6 kg/m^2$ , p<0.001) but both groups were still in the normal range of BMI (20-25). They gained more weight during pregnancy (16.3  $\pm$  $4.3 \text{ kg vs } 13.6 \pm 3.7 \text{ kg}, p < 0.001)$  and had retained more at one year of followup (normal weight 0.8 kg and overweight 3.1 kg). The women who became overweight also gained more from one year of follow-up until 15 years of follow-up  $(11.1 \pm 6.5 \text{ kg vs } 4.5 \text{ kg})$  $\pm$  6.5 kg, p<0.001) (Figure 4a). Consequently these women had a higher BMI at 15 years of follow-up (27.5  $\pm$  $2.6 \text{ kg/m}^2 \text{vs } 22.5 \pm 2.3 \text{ kg/m}^2, p < 0.001$ ).



**Figure 4** a) Change in body weight (kg) in women who remained in the BMI 20-25 range, from pre-pregnancy to 15 years follow-up and women who increased from BMI 20-25 pre-pregnancy to BMI > 25 at 15 years follow-up. b) Change in body weight (kg) in normal weight women (BMI 20-25 kg/m²) before pregnancy and overweight women before pregnancy (BMI over 25 kg/m²), from pre-pregnancy to 15 years follow-up. c) Change in body weight (kg) in low, intermediate and high weight gainers during pregnancy, from pre-pregnancy to 15 years follow-up. d) Change in body weight (kg) in low, intermediate and high weight retainer one year after pregnancy, from pre-pregnancy to 15 years follow-up.

Age, the number of children and other socio-economic factors did not differ between these two groups.

Data show that women who during 15 years increased from normal weight to overweight gained more weight during pregnancy, but they also gained more weight between one and 15 years of follow-up. Some of the weight gain, but not all, could be explained by their pregnancy.

#### Paper III

EFFECTS OF PREGNANCY ON LONG-TERM WEIGHT DEVELOPMENT - A 15 YEARS FOLLOW-UP OF THE SPAWN WOMEN.

In this paper the women in the SPAWN study were divided into groups regarding three presumably critical time periods: 1. Overweight and normal weight before pregnancy based on initial BMI. 2. Low, intermediate and high weight gainers during pregnancy by tertiles and finally 3. Low, intermediate and high weight retainers at one year after pregnancy by tertiles. The results (figure 4) showed that 1. Overweight women did not gain more weight during pregnancy or retain more weight at one year of follow-up. 2. High weight gainers retained more weight at one year of follow-up and at 15 years of follow up. 3. High weight retainers had gained more during pregnancy and retained it at 15 years of follow-up. (Figure 4 b,c,d) Fifty-six percent of the high weight gainers during pregnancy ended up in the high weight retainers group. There was little effect of socioeconomic factors. It seems that overweight women did not gain more weight than the normal weight women during pregnancy, and did not retain more at one year of follow-up. Women with overweight before pregnancy do not have an additional higher risk of

postpartum weight retention than normal weight women. Thus, it is not the initially overweight woman who needs to be the focus of weight control programs after pregnancy since, in fact she can be identified already before conception. Both high weight gainers and high weight retainers had higher BMI at 15 years of follow-up, although only 56% of the high weight gainers during pregnancy end up as the high weight retainers at one year after pregnancy. It seems that weight retention at the end of the postpartum year predicts future overweight.

#### Paper IV

INTERRELATIONSHIPS BETWEEN WEIGHT DEVELOPMENT AND WEIGHT RETENTION IN SUBSEQUENT PREGNANCIES - THE SPAWN STUDY

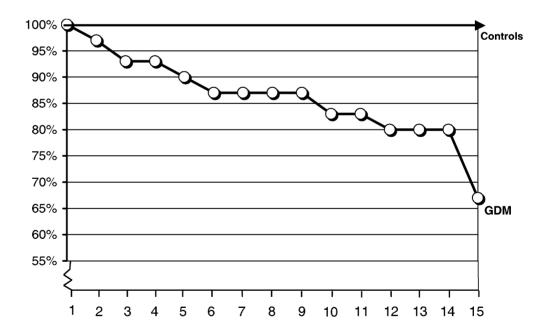
In this paper we studied the effect of one pregnancy on the other. The sample was divided into groups regarding four critical time periods:

- 1. Time interval between first and second pregnancy; less and more than two years.
- 2. Overweight and normal weights before first pregnancy based on initial RMI
- 3. Low and high weight gainers during pregnancy by mean split
- 4. Low and high weight retainers at one year after first pregnancy by mean split. The result show that:
- 1. Pregnancy interval did not effect weight development.
- 2. Initial BMI level did not effect weight development.
- 3. High weight gainers retained more weight at one year after both the first and second pregnancy.
- 4. High weight retainers had gained more during the first pregnancy and retained it into the second pregnancy and also gained more during and

retained more after the second pregnancy. Weight gain during pregnancy and delta weight up to one year after delivery is of some predictive value for the weight development in the second pregnancy, but obviously the environmental variation is an important determinant of the difference in weight increase which follows a pregnancy. Years between pregnancies do not seem to effect weight development. There are practical implications: Women who plan their later pregnancy several years after the first pregnancy have no reason to assume that the weight development during subsequent pregnancies will be worse than before. On the other hand, women who had increased considerably in weight during the first pregnancy or retained weight have a higher risk of doing so in subsequent pregnancies.

# Paper V NATURAL COURSE OF GESTATIONAL DIABETES MELLITUS – LONG-TERM FOLLOW-UP OF WOMEN IN THE SPAWN STUDY

Thirty-three percent of the women with GDM had developed type 2 DM at the follow-up. During the 15 years follow-up period, six women out of 28 (21%) GDM women had been diagnosed with type 2 DM in general practice, and another four where identified as diabetics by the OGTT in our unit, but all in the control group had normal OGTT (p<0.001) and none was diagnosed with diabetes. The cumulative frequency of GDM women who remained free from type 2 DM during the 15 years follow-up is shown in Figure 5.



**Figure 5.** Cumulative frequency of GDM women who remain free from type 2 diabetes mellitus during a 15 years follow-up (p<0.001), Kaplan-Meier plot.

At follow-up the group of GDM women who developed type 2 DM had a significantly higher BMI (p<0.05) and almost significantly higher waist/hip ratio (p=0.06) than GDM women who did not developed type 2 DM. However, blood pressure levels and serum lipoprotein concentrations did not differ between the groups. Fifty-four percent of the GDM women stated that they had never been informed that they had a higher risk of developing type 2 DM than others. However, no statistically significant relationship between weight development since the index pregnancy and the likelihood for type 2 DM was found. The women with type 2 DM had gained more weight since the birth of their first child compared to the other groups (p<0.05). The women who are diagnosed with GDM have a considerably higher risk of developing type 2 DM later in life. Despite a close medical monitoring during the pregnancy the further follow-up within the health care system and information about long-term consequences of GDM for later type 2 DM development seems to be generally lacking. More active strategies for future weight control and lifestyle advice after delivery might therefore be indicated for women with GDM.

# Paper VI

INTAKE OF SWEET FOODS AND COUNTS OF CARIOGENIC MICROORGANISMS IN RELATION TO BMI AND PSYCHOMETRIC VARIABLES IN WOMEN

As a part of the SPAWN study, the intake of sweet foods (habitual and premenstrual intakes) and number of cariogenic microorganisms in saliva was analysed in relation to BMI and psychometric variables. 362 women with a median BMI of 24.2 kg/m<sup>2</sup> (range 17.5-47.8) filled in a dietary

history, and salivary counts of mutans streptococci and lactobacilli were sampled and a self-rating scale on psychometric variables (CPRS-S-A). The results showed that the number of mutans streptococci correlated with BMI (P<0.05), indirectly indicating a higher intake of sweet foods in obese. The intake of sweet foods (more specifically the intake of chocolate), correlated with CPRS scores (P < 0.01), indicating that women with a higher psychiatric symptomathology have higher intakes of sweet foods. This study suggests that women with higher CPRS score have higher intakes of sweet foods, suggesting a link between mood regulation and the intake of sweets.

# **GENERAL DISCUSSION**

To our knowledge SPAWN is the longest follow-up study of women after pregnancy. We have also studied long-term effects of women with gestational diabetes and the intake of sweet food in relation to BMI and psychometric variables.

# When is the optimal time to determine weight retention after pregnancy?

It is complicated to determine the optimal time to monitor the weight development after pregnancy. If this is too soon after delivery, women will not have had enough time to lose the weight they lose naturally after delivery. If on the other hand it is measured much later, other factors will take over or the women may get pregnant again. A high BMI before pregnancy is associated with higher rates of pregnancy and delivery complications<sup>13</sup>. but in our study a high initial BMI does not seem to be a risk for retaining more weight after pregnancy in the long-term perspective. The same results have been shown in another parallel long-term follow-up study by Ronney et al<sup>38</sup>. These overweight women gained less than the normal weight women during pregnancy, and did not retain more at one year of follow-up. Thus, it is not the initially overweight women who need to be in focus for weight control programs after pregnancy. These women could easily be identified before pregnancy as a risk group for complicated pregnancy and delivery.

Women who gained most weight during pregnancy had higher BMI before pregnancy, although all groups were

within normal BMI ranges (paper III). They retained more at one year of follow-up. The same results are shown in paper IV. This is understandable; if more weight is gained it may be harder to lose. These women reported more oedema, which does however not explain all the weight gain, since this weight component is immediately lost after delivery.

Only half of the women who gained the most during pregnancy ended up in the highest category of weight retention at one year of follow-up. This is in line with the study of Ronney et al<sup>38</sup>. It remains controversial whether energy restriction should be advised to pregnant, overweight women to control their potential risk to increase excessively in weight during pregnancy 138, when a restrictive diet could effect the growth of the fetus 139. If intervention would address weight gain during pregnancy, half of the women would be overtreated. Thus, one year post-partum seems to be a better point in time to predict later weight problems after pregnancy then during the pregnancy itself. These facts have also been illustrated in paper IV, but selected for two consecutive pregnancies.

# Group formation of the SPAWN women

For a study like SPAWN there are some naturally dividing points. Women before pregnancy were divided into groups by the WHO definition for overweight and normal weight <sup>1</sup>. For the weight-gain during pregnancy and the weight retention after pregnancy, there

is no such natural division. We carried out these analyses by dividing these groups of women into different ways; into tertiles, as mean  $\pm$  SD and as a predefined weight gain of  $\pm$  3 kg and a weight gain of 4 kg. We chose to form our groups and present our results as tertiles and in paper IV by mean split, the latter because the material in study IV was smaller.

#### Which women are at risk?

It is still unclear what causes women to gain and retain weight after pregnancy. Further studies are obviously needed to identify additional factors, since several of the most obvious candidates, like breastfeeding, have actually been addressed and not found to be powerful predictors. Obviously, we need to develop a deeper understanding for the changes in physiology, genetics and psychology, eventually determining the situation of the pregnant and lactating woman to prevent a weight development with potentially harmful consequences.

In the SPAWN study we have focused on different time periods as factors for predicting weight development. It is obvious that weight retention after pregnancy is hard to predict before or during pregnancy by any methods used so far.

#### **General limitations**

There are some limitations of studies of weight development after pregnancy.

1.Usually they use self-reported weight before pregnancy since most women contact the maternity clinic when they know they are pregnant. It is a well know phenomenon that people tend to underestimate their weight<sup>140</sup>, especially among overweight and obese <sup>141</sup>.

2. Few studies have representative control groups. To recruit a group of women who will not have children,

living in relationships and with the same socio-economic, education level is obviously extremely difficult, especially in the long-term perspective. Only two short-term studies have a control group <sup>19, 64</sup>.

- 3. Interaction with other factors such as age as well as numerous socio-economic variables are as always hard to rule out.
- 4. It is still unclear when to perform the follow-up, at six months, one year or later? Since different studies use different time periods, this makes comparisons even more difficult. Most studies have actually settled for a mean follow-up time of one year, but sometimes with a wide range.

#### **Specific limitations of SPAWN**

There are some specific limitations of the SPAWN study in addition to the general problems.

- 1. The quality of the data can always be discussed with a 24% follow-up after 15 years. However, in a general perspective this seems to be a respectable recovery.
- 2. There is no information between one year and 15 years of follow-up and so we do not know if the changes observed represent a linear trend.
- 3. Also in SPAWN, we have had to rely on self-reported data. In particular this represents a problem in paper IV, where weights are self-reported before as well as one year after each of the two pregnancies.
- 4. It could be argued that a multiple regression analysis might disentangle the underlying causes for weight change. However, although sound in principle, this is complex in reality since so few factors turn out to be significant predictors. Such an analysis would anyhow leave us with the information that initial weight and increase during pregnancy remain of

predictive value.

# Limitations of the study of intake of sweet food

A limitation of the questionnaire on the sweet food intake was that it did not cover the total dietary intake, since we wanted to make the questionnaire as short as possible in order to increase the compliance and make subjects to focus on sweet foods to prevent underreporting due to forgetfulness. Since the salivary counts of mutans streptococci and lactobacilli reflect the intake of all fermented carbohydrates, higher correlations might have been found if a complete dietary history covering the total food intake had been included in the study. This would still presume that underreporting was not a problem and that the subjects had good memory.

#### **Drop-out analyses**

In the SPAWN study we re-recruited 24% of all subjects initially recruited. This number for restudy after 15 years is a fairly high recovery. To analyse the implications with drop-outs, we have made several drop-out analyses to determine if our material is representative. We know from previous studies that the subjects who drop out are usually people with low income and low socio-economic status<sup>143</sup>. These variables also correlate with higher body weight<sup>90</sup>. So, if anything, SPAWN is probably underestimating the problem with weight development after delivery.

### Gestational diabetes

Type 2 DM is a growing problem over the world. Thirty-three percent of the women with GDM developed type 2 DM (paper V). Women who were diagnosed with GDM had developed type 2 DM in about one third of all subjects in a more or less linear fashion over more than a 15 years period. We found 28 out of 58 GDM women for follow-up. Even if each and every of the 58 women had participated in our study, and none of the remaining women would have turned out to have type 2 DM, the percentage of the type 2 DM had still been 17% versus 0% in the control group. In the literature, incidence data vary from 3% up to 50% with follow-up periods ranging from a few months up to twelve years 10,16,17,18 Numerous factors can explain this variation, and in addition to design differences, genetic variation or diagnostic criteria and clinical treatment policies may play a part. This makes a critical comparison between results difficult. GDM women who developed type 2 DM had a significantly higher BMI and almost significantly higher waist/hip ratio at follow-up which indicates an association with a metabolic syndrome<sup>19,20</sup> Women who develop GDM during their pregnancy will be monitored in close

Women who develop GDM during their pregnancy will be monitored in close contact with their maternity clinic and under specialist supervision. The advice given about diet control and need to prevent excessive weight gain may result in a lower weight gain during pregnancy than in other pregnant women <sup>23,24</sup>.

However, when the child is delivered, most GDM women will not be followed-up, neither at the maternity units nor in the primary health care system, once blood glucose levels have returned to normal. It has been argued however that screening for GDM is not justified, mainly because it causes psychosocial problems and will not result in any change in behaviour<sup>25</sup>. Although weight control of GDM women during pregnancy is well monitored, our study demonstrates that GDM women will gain more weight over long time than controls, once the child has been delivered. For these women it is unlikely that they will

contact the medical health care system until they develop overt signs of type 2 DM. Although all GDM women were probably well informed about their diabetic condition during the pregnancy, 54% of them claimed that they had no idea that they might run a higher risk than others to develop clinically manifest type 2 DM at any time after delivery. We do not know whether this represents a selective memory, inadequate follow-up within the health care system or lack of appropriate clinical routines. In some countries, such as Sweden, there are recommendations that these women should be followed-up annually by glucose tests, but in clinical reality this does not seem to happen often, since most women will not seek medical care until they develop clinical symptoms and rapidly develop other priorities, once their child has been born. It is obvious that a strict program to identify women at high risk to develop type 2 DM, as indicated by their GDM, could be of importance to postpone the well-known complications of type 2 DM. Two recent studies indicate that a modest weight reduction has profound effect in prevention the development of clinical manifest type 2 DM <sup>26,27</sup>. The diabetic women in our study had a mean BMI of 27.4 kg/m<sup>2</sup>, a level which already indicates a pronounced risk for obesity related diabetic complications. Preventive long-term follow-up programs for GDM women thus seem essential to develop<sup>10</sup>.

### Intake of sweet food

In the study of sweet intake (Paper VI) there was no correlation between the intakes of sweet foods (habitual as well as pre-menstrual) and BMI, although 50% of the obese and 33% of the overweight women considered their intake to contribute to their weight problems. However, there was a

correlation between the number of mutans streptococci and BMI. Thus, obese women had higher number of mutans streptococci compared to normal weight women, which could indicate that the intake of sweet foods is larger in obese women <sup>126</sup>. The lack of relationship between the reported habitual intake of sweet foods and BMI is most likely due to bias in reporting the dietary intake. Under-reporting may not be random, i.e. subjects under-report to different extent.

Moreover, overweight and obese subjects are more likely to under-report than normal weight subjects<sup>144</sup>. We need to find more objective tests when measuring food intake and the saliva test could be one of them, since it is relatively cheap and easy to use. The habitual intake of sweets correlated with the CPRS score, indicating that higher intakes of sweet foods are associated with more psychiatric symptoms. Interestingly, chocolate was the only single group of sweet foods which was associated with the CPRS score. The reason why subjects with more psychiatric symptoms have higher intakes of sweet foods (especially chocolate), could be due to attempts to "self medicate" if the sweet foods eaten have potent psycho-pharmacological compounds. It has been suggested that the liking and craving for chocolate is related to the presence of psychoactive compounds in chocolate. However, up to date, there is no conclusive scientific support for this theory. The compounds in chocolate which could have psychopharmacological effects are either too weak to exert any effect or exist in much higher concentrations in other foods with less appeal than chocolate. Mela and Rogers <sup>121</sup> have instead suggested that chocolate has a special role as a treat. It is highly desirable but according to social norms due to the nutritional value (high in sugar and fat), it should be eaten with restraint, i.e.

"nice but naughty". The attempt to resist "naughty" foods may increase the attraction for the actual food. The relationship between mood and the craving for specific macronutrients, such as carbohydrate-rich foods, has previously been studied repeatedly. In seasonal affective disorder (SAD), a high frequency of atypical depressive symptoms, including hyperphagia and weight gain, has been reported 145 Increased appetite and carbohydrate craving in SAD has been observed to predominate in women<sup>146</sup>. Wurtman et al <sup>147</sup> have claimed that subjects with depressive moods linked to deficient serotonergic function, such as SAD and pre-menstrual syndrome (PMS), "selfmedicate" by consuming carbohydraterich foods in order to increase brain serotonin activity. However, the evidence against that theory is prominent. For example, the presence of small amount of protein (about 4%) in a high-carbohydrate meal is sufficient to block the meal-induced increase serotonin activity in the brain, and the foods craved by these subjects have thereby a too high protein content to have an impact on brain serotonergic function <sup>148, 149</sup>. The foods craved are rather sweet foods with a high fat and carbohydrate content, such as chocolate, cakes and ice cream, than pure carbohydrate-rich foods. Drewnowski 150 has instead suggested that cravings for foods high in sugar and fat is linked to the activity of the endogenous opioid "rewarding" system and that such cravings have similarities to opiate drug addiction. It has been suggested that pre-menstrual intakes of sweet foods is a sort of "self-medication" due to premenstrual hormonal changes affecting the state of mood. If so, one would expect that women with pre-menstrual intakes of sweet foods would have higher CPRS scores. However, there was no difference in the CPRS score

between women with and without premenstrual intakes in the present study. Moreover, the menstrually-related changes in mood and food intake, the so called PMS, are a well-known phenomenon for women in the Western society but are not experienced by women in tribal societies 151. This indicates that PMS could be a culturebound phenomenon, suggesting that menstrually-related overeating can only partly be attributed to hormonal changes. For some subjects, instead the awareness of cycle stage may condition overeating unrelated to hormonal changes or state of mood.

#### **Future**

There are still questions witch could be at focus for further SPAWN analyses. More data is available on psychological factors. Genetic analyses have not been carried out in this study, although such material has been stored. This could give us useful information about factors affecting the weight retention during and after pregnancy.

As an off-spring of the SPAWN study, there is a ongoing study at the Obesity Unit monitoring SPAWN mothers and children now about 16 years old: Stockholm WEight DEvelopment Study (SWEDES). Predictors for childhood obesity and interaction between mothers and children may be detected. Pregnancy does not only affect the mothers but the conditions around the pregnancy obviously constitute an important first environment for the fetus. Recent studies have set the focus on the impact of infant growth on later health 138. SWEDES with its information on weight development from conception to the follow up will give us a unique opportunity to address these issues of mother-child intererelationships at a later stage.

# **CONCLUSIONS**

The SPAWN study suggests that pregnancy is a risk factor for some women to retain weight after pregnancy. Although numerous factors contribute to this weight retention, our study cannot fully explain more than part of this variability.

Both high weight gainers and high weight retainers had higher BMI values at 15 years of follow-up, although only 56% of the high weight gainers during pregnancy ended up as the high weight retainers one year after pregnancy. Weight retention at the end of the postpartum year however predicts future overweight.

Contrary to popular belief and official recommendations, high BMI before pregnancy is not a predictor for weight retention. Women with overweight before pregnancy do not have a higher risk of postpartum weight retention than normal weight women.

Women who are diagnosed with gestational diabetes have a considerably higher risk of developing type 2 DM later in life. More active strategies for future weight control and lifestyle advice after delivery might therefore be indicated for women with GDM.

Our SPAWN data furthermore suggests that women with depressive traits, as evaluated by higher CPRS scores, have higher intakes of sweet foods, suggesting a link between mood regulation and consumption of such sweets. Salivary counts of mutans streptococci and lactobacilli are simple, cheap and chair-side tests known to mirror the consumption of fermented carbohydrates and may thus be used as an objective indicator of the sweet food intake in dietary surveys.

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# **REFERENCES**

- 1. WHO. Obesity:Preventing and managing the global epidemic. Report of a WHO Consultation on Obesity, 3-5 June. Geneva: WHO, 1997.
- 2. Glowinska B, Urban M, Koput A. Cardiovascular risk factors in children with obesity, hypertension and diabetes: lipoprotein(a) levels and body mass index correlate with family history of cardiovascular disease. Eur J Pediatr 2002; 161:511-8.
- 3. Meigs J. Epidemiology of the metabolic syndrome, 2002. Am J Manag Care 2002: 8:S283-92.
- 4. Lissner L, Johansson SE, Qvist J, Rossner S, Wolk A. Social mapping of the obesity epidemic in Sweden. Int J Obes Relat Metab Disord 2000; 24:801-5.
- 5. Arner P. Hunting for human obesity genes? Look in the adipose tissue! Int J Obes Relat Metab Disord 2000; 24 Suppl 4:S57-62.
- 6. DiPietro L. Physical activity, body weight, and adiposity: an epidemiologic perspective. Exerc Sport Sci Rev 1995; 23:275-303.
- 7. Blundell J. Appetite control and energy (fuel balance). Nutr Res Rev 1995; 8:225-42.
- 8. Fernström M. Drugs that cause weight gain. Obes Res 1995; 3 Suppl 4:435S-439S.
- 9. Rössner S. Pregnancy, weight cycling and weight gain in obesity. Int J Obes Relat Metab Disord 1992; 16:145-7.
- 10. Öhlin A, Rössner S. Maternal body weight development after pregnancy. Int J Obes 1990; 14:159-73.
- 11. Hyattsville M. National Center for Health Statistics. Vol. 183. United States, 1996.
- 12. Bongain A. Obesity in obstetrics and gynaecology. Eur J Obstet Gynecol and Reprod Biol 1998; 77:217-28.
- 13. Baeten J, Bukusi E, Lambe M. Pregnancy complications and outcomes among overweight and obese nulliparous women. Am J Public Health 2001; 91:436-40.
- 14. Ramsay J, Ferrell W, Crawford L, Wallance A, Greer I, Sattar N. Maternal obesity is associated with dysregulation of metabolic, vascular, and inflammatory pathways. Clin Endocrinol Metab 2002; 87:4231-7.
- 15. Cnattingius S, Lambe M. Trends in smoking and overweight during pregnancy: prevalence, risks of pregnancy complications, and adverse pregnancy outcomes. Semin Perinatol 2002; 26:286-95.

- 16. Greene J. Clinical stydy of the etiology of obesity. Ann Intern Med 1939; 12:1797-1803.
- 17. Newcombe RG. Development of obesity in parous women. J Epidemiol Community Health 1982; 36:306-9.
- 18. Brown JE, Kaye SA, Folsom AR. Parity-related weight change in women. Int J Obes Relat Metab Disord 1992; 16:627-31.
- 19. Williamson DF, Madans J, Pamuk E, Flegal KM, Kendrick JS, Serdula MK. A prospective study of childbearing and 10-year weight gain in US white women 25 to 45 years of age. Int J Obes Relat Metab Disord 1994; 18:561-9.
- Lowe G, JR G. Change in body weight associated with age and marital status. BMJ 1955:1006-8.
- 21. Hytten F, Chamberlerlein G. Clinical physiology in obstetrics. Oxford: Blackwell Scientific Publications, 1980.
- 22. Chesley L. Weight changes and water balance in normal and toxic pregnancy. Am J Obstet Gynecol 1944; 48:565-93.
- 23. Abrams B, Laros R. Prepregnancy weight, weight gain, and birth weight. Am J Obstet Gynecol 1986; 154:503-9.
- 24. Abrams B, Newman V, Key T, Parker J. Maternal weight gain and preterm delivery. Obstet Gynecol 1989; 74:577-83.
- 25. Abrams B, Parker J. Maternal weight gain in women with good pregnancy outcome. Obstet Gynecol 1990; 76:1-7.
- 26. Taffel SM. Maternal weight gain and the outcome of pregnancy: United States, 1980. Vital and Health Statistics, Series 21, No 44 DHSS Publ. No (PHS) 86-1922. National Center for Health Statistics, Public Health Service, U.S. Department of Health and Human Services, Hyattsville, Md: 1986: 25.
- 27. Johnston E. Weight changes during pregnancy and postpartum period. Prog Food Nutr Sci 1991; 15:117-57.
- 28. Dieckman W. The Toxemias of Pregnancy. In: ed e, ed. St Louise: Mosby, 1952.
- 29. Prentice A, Spaaij C, Goldberg G, et al. Energy requirements of pregnant and lactating women. Eur J Clin Nutr 1996; 50:S82-S110.
- 30. Spaaij C, van Raaij J, de Groot L, van der Heijden L, Boekholt H, Hautvast J. Effect of lactation on resting metabolic rate and on diet- and work- induced thermogenesis. Am J Clin Nutr 1994; 59:42-7.
- 31. Forsum E, Sadurskis A, Wager J. Resting metabolic rate and body composition of healthy Swedish women during pregnancy. Am J Clin Nutr 1988; 47:942-7.

- 32. Parham ES, Astrom MF, King SH. The association of pregnancy weight gain with the mother's postpartum weight. J Am Diet Assoc 1990; 90:550-4.
- 33. Janney CA, Zhang D, Sowers M. Lactation and weight retention. Am J Clin Nutr 1997; 66:1116-24.
- 34. Harris HE, Ellison G, Clement S. Relative importance of heritable characteristics and lifestyle in the development of maternal obesity. J Epidemiol Community Health 1999; 53:66-74.
- 35. Thorsdottir I, Birgisdottir BE. Different weight gain in women of normal weight before pregnancy: postpartum weight and birth weight. Obstet Gynecol 1998; 92:377-83.
- 36. Greene GW, Smiciklas-Wright H, Scholl TO, Karp RJ. Postpartum weight change: how much of the weight gained in pregnancy will be lost after delivery? Obstet Gynecol 1988; 71:701-7.
- 37. Schauberger C, Rooney B, Brimer L. Factors that influence weight loss in the puerperium. Obstet Gynecol 1992:424-9.
- 38. Rooney B, Schauberger C. Excess pregnancy weight gain and long-term obesity: one decade later. Obstet Gynecol 2002; 100:245-52.
- 39. Harris H, Ellison G, Clement S. Do the psychosocial and behavioral changes that accompany motherhood influence the impact of pregnancy on long-term weight gain? J Psychosom Obstet Gynaecol 1999; 20:65-79.
- 40. Thomson A, Billewicz W. Maternal weight for height. Proc Nutr Soc 1965; 24:14-20.
- 41. Cederlöf R, Kaij L. The effect of childbearing on body weight: a twin control study. Acta Psychiatry Scand Supp 1970:47-9.
- 42. Beacke J, Burema J, Frijters J, Hautvast J, Van der Wiel-Wetzels W. Obesity in young Dutch adults: I, socio-demographic variables and body mass index. Int J Obes 1983; 7:1-12.
- 43. Lee-Feldstein A, Harburg E, Hauenstein L. Parity and blood pressure among four race-stress groups of females in Detroit. Am J Epidemiol 1980; 111:256-366
- 44. Noppa H, Bengtsson C, Wedel H, Wilhelmsen L. Obesity in relation to morbidity and mortality from cardiovascular disease. Am J Epidemiol 1980; Jun;111:682-92.
- 45. Heliövaara M, Aromaa A. Parity and obesity. J Epidemiol Community Health 1981; 35:197-99.
- 46. Prentice AM, Whitehead RG, Roberts SB, Paul AA. Long-term energy balance in child-bearing Gambian women. Am J Clin Nutr 1981; 34:2790-9.

- 47. Forster J, Bloom E, Sorensen G, Jeffery R, Prineas R. Reproductive history and body mass index in black and white women. Prev Med 1986; Nov;15:685-91.
- 48. Rona R, Morris R. National Study of Health and Growth: social and family factors and overweight in English and Scottish parents. Ann Hum Biol 1982; 9:147-56.
- 49. Caan B, Horgen D, Margen S, King J, Jewell N. Benefits associated with WIC supplement feeding during the interpregnancy interval. Am J Clin Nutr 1987; 45:29-41.
- 50. Chowdhury A. Change in maternal nutritional status in a chronically malnourished population in rural Bangladesh. Ecol Food Nutr 1987; 19:201-11.
- 51. Kumanyika S. Obesity in black women. Epidemiol Rev 1987; 9:31-50.
- 52. Kritz-Silverstein D, Barrett-Conorr E, Wingard D. The effects of parity on the later development of non-insulin dependent diabetes mellitus or impaired glucose tolerance. N Engl J Med 1989; 321:1214-19.
- 53. Rondin J, Radke-Sharpe N, Rebuffé-Scrive M, Greenwood M. Weight cykling and fat distribution. Int J Obes 1990; 14:303-10.
- 54. Manson J, Rimm E, Colditz G, et al. parity and incdence of non-insulindependent diabetes mellitus. Am J Med 1992; 93:11-18.
- 55. Öhlin A, Rössner S. Factors related to body weight changes during and after pregnancy: the Stockholm Pregnancy and Weight Development Study. Obes Res 1996; 4:271-6.
- 56. Hunt SC, Daines MM, Adams TD, Heath EM, Williams RR. Pregnancy weight retention in morbid obesity. Obes Res 1995; 3:121-30.
- 57. Gunderson E, Abrams B, Selvin S. Does the pattern of postpartum weight change differ according to pregravida body size? Int J Obes 2002; 25:853-62.
- 58. Soltani H, Fraser RB. A longitudinal study of maternal anthropometric changes in normal weight, overweight and obese women during pregnancy and postpartum. Br J Nutr 2000; 84: 95-101.
- 59. Beazley J, Swinhoe R. Body weight in parous women: Is there any alteration between successive pregnancies? Acta Obstet Gynecol Scand 1979; 58:45-7.
- 60. Olsen F, Frische G, Poulsen AO, Kirchheiner H. Changing smoking, drinking, and eating behaviour among pregnant women in Denmark. Evaluation of a health campaign in a local region. Scand J Soc Med 1989; 17:277-80.
- 61. Muscati SK, Gray-Donald K, Koski KG. Timing of weight gain during pregnancy: promoting fetal growth and minimizing maternal weight retention. Int J Obes Relat Metab Disord 1996; 20:526-32.
- 62. Scholl TO, Decker E, Karp RJ, Greene G, De Sales M. Early adolescent

- pregnancy: a comparative study of pregnancy outcome in young adolescents and mature women. J Adolesc Health Care 1984; 5:167-71.
- 63. McKeown T, Record RG. The influence of reproduction on body weight in women. J Endocrinol 1957; 15: 393-409.
- 64. Rookus MA, Rokebrand P, Burema J, Deurenberg P. The effect of pregnancy on the body mass index 9 months postpartum in 49 women. Int J Obes 1987; 11:609-18.
- 65. Breadey P. Does pregnancy cause obesity? Med J Aust 1989; 151:543-4.
- 66. Keppel K, Taffel S. Pregnancy-related weight gain and retention: Implications of the 1990 Institute of medicin Guidelines. Am J Public Health 1993:1100-3.
- 67. Smith DE, Lewis CE, Caveny JL, Perkins LL, Burke GL, Bild DE. Longitudinal changes in adiposity associated with pregnancy. The CARDIA Study. Coronary Artery Risk Development in Young Adults Study. JAMA 1994; 271:1747-51.
- 68. Walker LO. Predictors of weight gain at 6 and 18 months after childbirth: a pilot study. J Obstet Gynecol Neonatal Nurs 1996; 25:39-48.
- 69. Harris H, Ellison G. Practical approaches for estimating prepregnant body weight. J Nurse Midwifery 1998; 43:97-101.
- 70. Harris HE, Ellison GT, Holliday M. Is there an independent association between parity and maternal weight gain? Ann Hum Biol 1997; 24:507-19.
- 71. Harris H, Ellison G, Holliday M, Lucassen E. The impact of pregnancy on the long-term weight gain of primiparous women in England. Int J Obes Relat Metab Disord 1997: 21:747-55.
- 72. Harris HE, Ellison GT, Holliday M, Lucassen E. Methodological considerations in the design of an obstetric database abstracted from medical records. Methods Inf Med 1997; 36:191-200.
- 73. Harris HE, Ellison GT, Holliday M, Nickson C. How accurate are antenatal weight measurements? A survey of hospital and community clinics in a South Thames Region NHS Trust. Paediatr Perinat Epidemiol 1998; 12:163-75.
- 74. Harris HE, Ellison GT, Holliday M. Is there an independent association between parity and maternal weight gain? Ann Hum Biol 1997; 24:507-19.
- 75. Harris HE, Ellison GT, Richter LM, de Wet T, Levin J. Are overweight women at increased risk of obesity following pregnancy? Br J Nutr 1998; 79:489-94.
- 76. Öhlin A, Rössner S. Cessation of smoking during pregnancy can cause heavy weight gain. Läkartidningen 1989; 86:4443-6.
- 77. Öhlin A, Rössner S. Trends in eating patterns, physical activity and sociodemographic factors in relation to postpartum body weight development. Br J

Nutr 1994; 71:457-70.

- 78. Hallberg L, Bengtsson C, Grby L, Lennartsson J, Rossander L, Tibblin E. An analysis of factors leading to a reduction in iron deficiency in Swedish women. Bull World Health Organ 1979; 57: 947-54.
- 79. Ash S, Fisher C, Truswell A, Allen J, Irwig L. Maternal weight gain, smoking and other factors in pregnancy as predictors of infant birth-weight in Sydney women. Aust NZ J Obstet Gynsecol 1989; 29:212-19.
- 80. Mitchell M, Lerner E. A comparison of pregnancy outcome in overweight and normal weight women. J Am Coll Nutr 1989; 8:617-24.
- 81. Brown J, Jacobson H, Askue L, Peick M. Influence of pregnancy weight gain and the size of infants born to underweight women. Obstet Gynecol 1981; 57:13-7.
- 82. Kleinman J. Maternal weight gain during pregnancy: determinants and consequences. NCHS Working Paper Series No. 33. National Center for Health Statistics, Public Health Service, U.S. Department of Health and Human Services, Hyattsville, Md. pp 24.
- 83. Theron G, Thompson M. A centile chart for weight gain in pregnancy for the urban population of the Western Cape, South Africa. Int. J Gynaecol. Obstet 1990; 33:127-33.
- 84. Walker LO. Weight and weight-related distress after childbirth: relationships to stress, social support, and depressive symptoms. J Holist Nurs 1997; 15:389-405
- 85. Crowell DT. Weight change in the postpartum period. A review of the literature. J Nurse Midwifery 1995; 40:418-23.
- 86. To WW, Cheung W, Kwok JS. Paternal height and weight as determinants of birth weight in a Chinese population. Am J Perinatol. 1998;15(9):545-8.
- 87. Hofstetter A. Current chemotherapy in urogenital infections. 6: antibiotics during pregnancy and lactation in urethro-adnexitis, vulvovaginitis and cervicitis. Fortschr Med 1992; 30;110; 60- 63-4.
- 88. Helt EH. Economic determinism: a model of the political economy and medical care. Int J Health Serv 1973; 3(3):475-85.
- 89. Johnston E, Hyson D, Blackmer E. Weight gain and related characteristics of pregnant Nova Scotia women. J Can Diet Assoc 1985; 46:45-9.
- 90. Stunkard A, Sorensen T. Obesity and socioeconomic status: a complex relation. N Engl J Med 1993; 329:1036-7.
- 91. Parker JD, Abrams B. Differences in postpartum weight retention between black and white mothers. Obstet Gynecol 1993; 81:768-74.

- 92. Rössner S, Öhlin A. Pregnancy as a risk factor for obesity: lessons from the Stockholm Pregnancy and Weight Development Study. Obes Res 1995; 3 Suppl 2:267s-275s.
- 93. Kramer F, Stunkard A, Marshall K, McKinney S, Liebschutz J. Breast-feeding reduces maternal lower-body fat. J Am Diet Assoc 1993; 93:429-33.
- 94. Butte N, Hopkinson J. Body composition changes during lactation are highly variable among women. J Nutr 1998; 128:381S-385S.
- 95. Waaler H. Height, weight and mortality. The Norwegian experience. Acta Med Scand Suppl 1984; 679:1-56.
- 96. Potter S, Hannum S, McFarlin B, Essex-Sorlie D, Campbell E, Trupin S. Does infant feeding method influence maternal postpartum weight loss? J Am Diet Assoc 1991; 91:441-6.
- 97. Brewer MM, Wozniak P, Bates M, Vonnoy L, Mangum M. Assessment of postpartum body fat change from underwater weight and skinfold thickness measurements. Nutr Res 1989; 9:1331-7.
- 98. Rössner S. Physical activity level and weight control during pregnancy. In: Bouchard C (ed.). Physical activity and obesity. Windsor, Ont., Canada: Human Kinetics Publishers Inc, 2000: 279-94.
- 99. Sampselle C, Seng J, Yeo S, Killion C, Oakley D. Physical activity and postpartum well-being. J Obstet Gynecol Neonatal Nurs 1999; 28:41-49.
- 100. Miller W, Lindeman A, Wallace J, Niederpruem M. Diet composition, energy intake, and exercise in relation to body fat in men and women. Am J Clin Nutrition 1990; 52:426-30.
- 101. Sheldon J. Metarnal obesity. Lancet 1949; ii:869-73.
- Harris HE, Ellison GT, Richter LM, de Wet T, Levin J. Are overweight women at increased risk of obesity following pregnancy? Br J Nutr. 1998 Jun;79(6):489-94
- 103. Boardley DJ, Sargent RG, Coker AL, Hussey JR, Sharpe PA. The relationship between diet, activity, and other factors, and postpartum weight change by race. Obstet Gynecol 1995; 86:834-8.
- 104. Wolf W, Sobal J, Olson C, Frongillo E, Williamsson D. Parity-associated weight gain and its modification by sociodemographic and behavioural factors: a prospective analysis in US women. Int J Obes 1997; 1997:802-10.
- 105. Friis H, Gomo E, Kaestel P, Nyazema N, Ndhlovu P, Fleischer Michaelsen K. Does the first pregnancy precipitate age-related fat deposition? Int J Obes Relat Metab Disord 2002; 26:1274-6.
- 106. Lederman S. The effect of pregnancy weight gain on later obesity. Obstet Gynecol 1993; 82:148-55.

- 107. Jenkin W, Tiggemann M. Psychological effects of weight retained after pregnancy. Women Health 1997; 25:89-98.
- 108. Gabbe S. Gestational diabetes mellitus. N Engl J Med 1986; 315:1025-26.
- 109. Guttorm E. Practical screening for diabetes mellitus in pregnant women. Acta Endocrinol (Copenhagen) 1974; 75:11-24.
- Metzger B, Coustan D. Summary and recomendations of the fourth international workshop-conference on gestational diabetes mellitus. Diabetes Care 1998; 21:B 161-7.
- 111. Dornhorst A, Rossi M. Risk and prevention of Type 2 diabetes in women with gestational diabetes. Diabetes Care 1998; 21:B43-9.
- Sepe S, Connell F, Geiss L, Teutsch S. Gestational diabetes: Incidence, maternal characteristics and perinatal outcome. Diabetes 1985; 34:13-6.
- 113. Catalano P, Roman N, Tyzbir E, Merritt A, Driscoll P, Amini S. Weight gain in women with gestational diabetes. Obstet Gynecol 1993; 81:523-8.
- Jocobson J, Cousins L. A population-based study of maternal and perinatal outcome in patients with gestational diabetes. Am J Obstet Gynecol 1989; 161:981-6.
- Bolton-Smith C, Woodward M. Dietary composition and fat to sugar ratios in relation to obesity. Int J Obes 1994; 18:820-8.
- 116. Drewnowski A. Food preferences and the opioid peptide system. Trends in Food Science and Technology 1992; 3:97-9.
- 117. Gibson S. Are high-fat, high sugar foods and diets conductive to obesity? International Journal of Food Sciences and Nutrition 1996; 47:405-415.
- 118. Stam-Moraga M, Kolanowski J, Dramaix M, De Backer G, Kornitzer M. Sociodemographic and nutritional determinants of obesity in Belgium. Int J Obes 1999; 23:1-9.
- 119. Macdiarmid J, Vail A, Cade J, Blundell J. The sugar-fat relationship revisited: differences in consumption between men and women of varying BMI. Int J Obes 1998; 22:1053-61.
- 120. Macdiarmid J, J.E. B. Dietary under-reporting: what people say about recording their food intake. Eur J Clin Nutr 1997; 51:199-200.
- 121. Mela D, Aaron J. Honest but valid: what subjects say about recording their food intake. Journal Am Diet Assoc 1997; 97:791-793.
- Macdiarmid JI, Blundell JE. Assessing dietary intake: who, what and why of under-reporting. Nutr Res Rev 1998; 11:231-53.

- 123. Andréen I, Köhler B. Effects of Weight Watchers' diet on salivary secretion rate, buffer effect and number of mutans streptococci and lactobacilli. Scand J Dent Res 1992; 100:93-7.
- 124. Kristoffersson K, Birkhed D. Effects of partial sugar restrictions for 6 weeks on number of Streptococcus mutans in saliva and interdental plaque in man. Caries Res 1987; 21:79-86.
- 125. Scheie A, Arneberg P, Orstavik D, Afseth J. Microbial composition, PH-depressing capacity and acidogenicity of 3-week smooth surface plaque developed on sucrose-regulated diets in man. Caries Research 1984; 18:74-86.
- 126. Barkeling B, Andersson I, Lindroos A-K, Birkhed D, Rössner S. Intake of sweet foods and counts of cariogenic microorganisms in obese and normal-weight women. Eur J Clin Nutr 2001; 55:850-5.
- 127. Rogers P, Smit H. Food craving and food "addiction": A critical review of the evidence from a biopsychosocial perspective. Pharmacol Biochem aBehav 2000; 66:3-14.
- 128. Rooth P, Hyll M, Rössner S. High prevalence of anxiety and depressive symptoms in obese patients. 1999.
- 129. Garpenby P, Carlsson. The role of national quality registers in the Swedish health service. Health Policy 1994; 29:183-95.
- 130. Cnattagus S, Ericsson A, Gunnarskog J, Kallen B. A quality study of a medical birth registry. Scand J Soc 1990; 18:143-48.
- 131. Möller AJR. Microbiological examination of root canals and periapical tissues of human teeth. Thesis. Ondontol Tidskr 1966; 74: 1-380.
- 132. Gold O, Jordan H, Van Houte J. A selective medium for Streptococcus mutans. Arch Oral Biol 1973; 18:1357-64.
- 133. Lindroos A-K, Lissner L, Sjöström L. Validity and reproducibility of a selfadministered dietary questionnarie in obese and non-obese subjects. Eur J Clin Nutr 1993; 47:461-81.
- 134. Mattila-Evenden M, Svanborg P, Gustavsson P, Åsberg M. Determinants of self-rating and expert rating concordance in psychiatric out-patients, using affective subscales of the CPRS. Acta Ped Scand (Copenhagen) 1996; 94:386-396.
- 135. Svanborg P, Åsberg M. A new self-rating scale for depression and anxiety states based on Comprehensive Psychopathological Rating Scale. Acta Psychiatr Scand (Copenhagen) 1994; 89:21-8.
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders - DSM-IV 1994.
- 137. Hedman B, Herner E. Women's health and women's work in health

- service:what statisics tell us. Women Health 1988; 13:9-34.
- Barker DJ, Godfrey KM, Osmond C, Bull A. The relation of fetal length, ponderal index and head circumference to blood pressure and the risk of hypertension in adult life. Paediatr Perinat Epidemiol 1992; 6:35-44.
- Wadsworth ME, Kuh DJ. Childhood influences on adult health: a review of recent work from the British 1946 national birth cohort study, the MRC National Survey of Health and Development. Paediatr Perinat Epidemiol 1997; 11:2-20.
- 140. Stewart A. The reliability and validty of self-repored weight and height. J Chron Dis 1982: 35:295-309.
- 141. Rowland M. Self-reported weight and height. Am J Clin Nutr 1990; 52:1125-33.
- Kaul L, Nidiry JJ. Management of obesity in low-income African Americans. J Natl Med Assoc 1999; 91(3): 139-43.
- van Lenthe FJ, Boreham CA, Twisk JW, Savage MJ, Murray L, Smith GD. What determines drop out in prospective studies of coronary heart disease risk factors between youth and young adulthood: the Young Hearts Study. J Epidemiol Community Health 2001; 55(9): 681-2.
- 144. Macdiarmid J, Blundell J. Assessing dietary intake: Who, what and why of under-reporting. Nutr Resh Rev 1998; 11:231-53.
- Rosenthal N, Sack D, Gillin J. Seasonal affective disorders. A description of the syndrome and preliminary findings with light therapy. Arch Gen Psychiatry 1984; 41:72-80.
- Takahashi K, Asano Y, Kohsaka M. Multi-center study of seasonal affective disorders in Japan. A preliminary report. J Affect Disord 1991; 21:57-65.
- Wurtman R. Dietary treatments that affect brain neurotransmitters: effects on calorie and nutrient intake. 1987.
- 148. Christensen L. The effect of carbohydrates on affect. Nutrition 1997; 13:503-14.
- 149. Fernstrom JD. Carbohydrate ingestion and brain serotonin synthesis: relevance to a putative control loop for regulating carbohydrate ingestion and effects of aspartame consumptiion. Appetite 1988; 11(Suppl). 35-41.
- 150. Drewnowski A, Henderson S, Shore A, Fischler C, Preziosi P, Hercberg S. The fat-sucrose seesaw in relation to age and dietary variety of French adults. Obes Res 1997; 5:511-8.
- 151. Paige K, Paige JM. Politics and reproduction rituals. Berkeley, CA: University of California Press, 1981.