Childhood obesity in a multiethnic society

Early life risk factors and communication with parents

Ingun Toftemo

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1   Abbreviations

BMI: Body mass index

CI: Confidence interval

SD: Standard deviation

WHO: World Health Organization

2 Preface

2.1 Motivation

My motivation for research on childhood obesity came through my work as a general practitioner, responsible for primary care to a given list population, and through my work at the local Child Health Clinic, which is attended by almost all pregnant women and children in the community. I have experienced families struggling with obesity, gestational diabetes, and type 2 diabetes mellitus across generations. Approximately 12 % of the patients on my list have ethnic minority background (mainly from East Asia and the Middle East) with a higher risk of type 2 diabetes mellitus and gestational diabetes compared with the ethnic Norwegians. In 2010, staff at the local Child Health Clinic and I developed a new initiative to reduce the risk of childhood obesity in our municipality. We offered a program of interdisciplinary group counselling of parents who had 4-5 years old children with overweight. For more than one year, we worked hard to recruit parents, but very few were interested. Our project failed, and we did not understand why. Therefore, I decided to study childhood obesity and gain knowledge on how to identify children at risk, and how to communicate with the families about my concerns.

2.2 Supervisors

Line Sletner (main supervisor)

Anne Karen Jenum

Per Lagerløv

2.3 Acknowledgements

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2.4 Funding

3   Norwegian summary / Norsk sammendrag

Bakgrunn: Det er økende kunnskap om at forhold tidlig i livet har betydning for senere helse, blant annet risiko for overvekt, fedme og type 2 diabetes. Vi trenger mer kunnskap om hvilke faktorer som påvirker utvikling av overvekt hos barn, og om hvordan helsepersonell best skal kommunisere med foreldre for å fremme sunn vekst.

Formål: Målet med denne studien var å undersøke etniske forskjeller i forekomst av overvekt og tynnhet hos førskolebarn, og hvordan mors vektstatus før svangerskapet, vektoppgang i svangerskapet og svangerskapsdiabetes påvirket barnets kroppsmasseindeks (KMI, kg/m^2) fra fødsel og fram til førskolealder i en multi-etnisk populasjon. Vi ønsket også å utforske foreldres opplevelse rundt at barnet deres ble identifisert som overvektig på helsestasjonen.

Materiale og metode: For å svare på forskningsspørsmålene har vi valgt å bruke både kvantitativ og kvalitativ metode. I den <u>kvantitative delen</u> av prosjektet inkluderte vi 734 barn fra en multietnisk populasjonsbasert kohort av gravide og deres barn. Kvinnene ble inkludert tidlig i svangerskapet, og alle ble screenet for svangerskapsdiabetes. De etniske hovedgruppene hadde opprinnelse i Europa, Sør- Asia, Øst- Asia og Midtøsten/Nord-Afrika. I *artikkel I* brukte vi multinomial logistisk regresjonsanalyse for å utforske etniske forskjeller i tynnhet og overvekt ved 4-5 års alder, samt assosiasjoner med maternelle, pre- og postnatale faktorer. I *artikkel II* undersøkte vi ved hjelp av «linear mixed models» assosiasjoner mellom mors KMI før svangerskapet, vektoppgang i svangerskapet og svangerskapsdiabetes, og barnas KMI-utvikling etter fødselen. I den <u>kvalitative delen</u> av avhandlingen, *artikkel III*, intervjuet vi foreldrene til 10 førskolebarn om erfaringene de gjorde da barnet deres nylig hadde blitt indentifisert med overvekt på helsestasjonen. Data ble analysert ved hjelp av metoden «Systematisk tekstkondensering».

Resultater: Sammenliknet med barn med europeisk opprinnelse, hadde barn med opprinnelse fra Midtøsten/Nord-Afrika dobbelt så høy risiko for overvekt ved 4-5 års alder, mens barna med Sørasiatisk opprinnelse hadde dobbelt så høy risiko for tynnhet. Da vi justerte for at Sørasiatiske barn har en relativt økt fettmasse, steg prevalensen av overvekt,

og prevalensen av tynnhet sank i denne gruppen. Overvekt hos mor var sterkt assosiert med overvekt hos barnet ved 4-5 års alder. Barn som ble eksponert for svangerskapsdiabetes i svangerskapet hadde lik gjennomsnittlig KMI som barn som ikke hadde blitt eksponert både ved fødsel og ved 4-5 års alder. Fra fødsel og fram til seks måneders alder hadde de imidlertid en langsommere KMI vekst, mens de fra seks måneder til 4-5 år hadde en raskere KMI vekst. Barna som var eksponert for pregravid fedme hos mor hadde en høyere KMI ved fødsel og videre en stabilt høyere KMI, sammenliknet med barna som ikke var eksponert. Høy vektøkning hos mor i svangerskapet var assosiert med raskere KMI vekst fra seks måneder til 4-5 år.

I den kvalitative delen av prosjektet presenterte foreldrene som nylig hadde fått vite at førskolebarnet deres var overvektig både seg selv og barnet som sårbare. De var engstelige for at barnet skulle får dårlig selvtillit eller spiseforstyrrelse, og ønsket derfor en samtale med helsepersonell uten at barnet var til stede. Foreldrene snakket gjerne om sine egne vekterfaringer, og disse kunne representere både en barriere og en motivasjon for å hjelpe barnet. Alle stolte på og ønsket hjelp i barnehagen, mens noen opplevde at besteforeldrene motarbeidet foreldrenes forsøk på å bedre barnets livsstil.

Konklusjon og betydning: I en multietnisk kohort av gravide og deres barn fant vi tydelige etniske forskjeller i overvekt og tynnhet hos barna ved 4-5 års alder. Vi fant videre at fedme hos mor før svangerskapet, vektøkning i svangerskapet og svangerskapsdiabetes påvirket barnets KMI utvikling fra fødsel til 4-5 års alder, men på litt ulike måter. I samtaler med foreldre om overvekt hos førskolebarn bør helsepersonell vurdere om man skal ha en første konsultasjon uten barnet til stede og om barnehage og besteforeldre kan bidra med hjelp til familien.

Våre funn setter fokus på betydningen av et livsløps- og generasjonsperspektiv for å kunne forstå og redusere utviklingen av overvekt og fedme hos barn. Resultatene synliggjør et behov for tverrfaglig samarbeid i helsetjenesten, og kan brukes når nye retningslinjer for forebygging av overvekt hos barn og unge nå skal revideres. I tillegg kan våre funn ha betydning for planlegging av bredere folkehelseprogrammer der samarbeidspartnere som skoler og barnehager er viktige for å forebygge at overvekt blir overført fra en generasjon til den neste.

4   List of papers

I.

Contrasting patterns of overweight and thinness among preschool children of different ethnic groups in Norway. Associations with maternal and early life factors

Ingun Toftemo, Anne Karen Jenum, Per Lagerløv, Pétur B. Júlíusson, Ragnhild Sørum Falk, Line Sletner

(BMC Public Health. 2018 Aug 23;18(1):1056. doi: 10.1186/s12889-018-5952-1.)

II.

BMI trajectories up to preschool age in a multi-ethnic population; relations with maternal gestational diabetes, BMI and gestational weight gain

Ingun Toftemo, Anne Karen Jenum, Line Sletner

(Acta Paediatrica. 2020 Oct 24. doi: 10.1111/apa.15637.)

III.

Parents' experiences when their preschool child is identified as overweight: a qualitative study in primary care.

Ingun Toftemo, Kari Glavin, Per Lagerløv

(Family Practice. 2013 Dec;30(6):719-23. doi: 10.1093/fampra/cmt056. Epub 2013 Oct 9.)

5   Introduction

5.1   Childhood obesity – a global and national public health challenge

Childhood obesity is one of the most serious public health issues of our time (1). The global prevalence of overweight and obesity in children has increased by tenfold over the past 40 years. Pooled data from 31.5 million children and adolescence aged 5-19 years show that the global proportion of children with obesity has increased from 0.7 % (girls) and 0.9 % (boys) in 1975 to 5.6 % and 7.8 % in 2016 (2). In Norway, the Bergen Growth Study showed that the overall prevalence of obesity in children aged 2-19 years was 2.3 %, with no significant difference between boys and girls (3). At eight years of age, about 15% of Norwegian children are overweight or obese (17% of girls and 13% of boys) (4). While the prevalence of obesity among Norwegian children aged 8-9 years seems to have reached a plateau, it is rising in the adolescent population (4, 5) . Data collected in 2019 by the Trøndelag Health Study (Ung-HUNT4) showed that in the age group 13-19 years, 6.5 % of girls and 7 % of boys were affected by obesity (6). In total, approximately one out of four adolescence were overweight or obese. This is worrisome, as children with obesity are very likely to remain obese as adults, and are at risk of chronic illnesses that can decrease the life expectancy (7). Today, the majority of Norwegian adults are overweight or obese (5). There is a greater proportion of men (about 25%) than women (about 21%) affected by obesity, and the prevalence has been rising continuously over the last 40-50 years (8).

Worldwide, obesity has nearly tripled since 1975, and there are now more people who are obese than underweight (9). In 2016, 39% of adults were overweight and 13% were obese. While obesity previously was a public health problem mostly in high-income countries, it is now also present in low- and middle-income countries. Many of these countries are facing a "double burden" of malnutrition coexisting with undernutrition (predominantly in new-borns, children and young adults of reproductive age) and obesity (predominantly in adults) (10). Risk factors for non-communicable diseases, such as obesity, are particularly increasing in urban settings of low- and middle-income countries.

5.2   Mechanisms and risk factors for childhood obesity

Childhood obesity arises from a complex interplay between an obesogenic environment and inadequate behavioural and biological responses to the unhealthy environment (1). This combination causes energy imbalance with increased adipose tissue. Responses vary among individuals and are strongly influenced by early life exposures already from conception. Furthermore, social disparities, cultural norms, and genetic predisposition are among factors associated with childhood obesity.

Obesogenic environment

Over the last decades, children worldwide spend more time on screen-based and sedentary leisure activities, and they spend less time on physical activity when playing, and for transportation (11). With urbanization, there has been a transition of food types from traditional diets to processed and high-energy food being available, affordable, and marketed. This pattern is seen in both low- and high-income countries. A Global strategy for Prevention and Control of Non-communicable Diseases was ratified by the World Health Organization (WHO) in 2012, setting nine goals to improve public health (12). In Norway, we are about to a achieve goals as reduction of smoking and alcohol abuse. However, for two goals the arrows are pointing in the wrong direction; (i) Prevalence of obesity and type 2 diabetes is increasing, and (ii) Amount of physical activity for children and adolescents is decreasing (5).

Social disparities

In high-income countries, social disparities is a public health challenge, reflecting higher prevalence of obesity among the poor, those with low education, indigenous people, and in ethnic groups from low-income countries (13, 14). These groups often have limited health literacy, which includes a set of skills needed to navigate the health care system and make good decisions concerning their health; reading, writing, communication, numeracy, and the use of electronic technology (15). In Norway, the risk of childhood overweight and obesity is 30 % higher in children of mothers with primary compared to tertiary education, and 50 % higher for those with divorced parents than for those with married parents (16).

Ethnicity

Findings indicate that in Europe, ethnic minority children with Middle East and North-African origin may be disproportionally affected by obesity, while South Asian children may have lower risk (13, 17-19). Ethnic minority groups in high-income countries are prone to rapid acculturation and poor access to public health information, due to a low level of integration and language difficulties (1, 20). In low- and middle- income countries, the risk of childhood obesity is greatest in higher income groups. However, a changing pattern is emerging as these countries undergo rapid societal and nutritional changes. To date, more children (absolute numbers) with obesity live in low- and middle-income countries than in high-income countries (21). Ethnic minority groups with non-Western origin have increased susceptibility to type 2 diabetes and are diagnosed 8-15 years younger than those of European origin (22, 23). According to data from the Norwegian Childhood Diabetes Registry, very few children are diagnosed with type 2 diabetes, but those who are often have ethnic minority background. In Europe, physical inactivity is more prevalent in children of African, Middle-Eastern and South Asian origin compared with the majority population (19, 24).

Body composition

Body mass index (BMI) is defined by a person's weight divided by the square of body height and expressed by kg/m^2. As BMI is based on weight, it does not differentiate between fat and lean mass. BMI underestimates body fat in South Asian populations who have more fat mass and less fat-free mass compared with Europeans (25). In a study of British children aged 4-5 years, the prevalence of overweight and obesity increased from 19 % in boys and girls to 39 % in boys and 35 % in girls after having applied suggested BMI adjustments that took into account the body composition in children with South Asian background (26). Thus, parents with South Asian background may be inappropriately reassured by health professionals that their children have a normal weight, and may not seek advice about tackling childhood overweight and obesity (27).

Perceptions of obesity in childhood

Perception of childhood overweight and obesity may vary between cultures and over time. However, parents with overweight or obese children often do not perceive that their child has a weight problem. A meta-analysis found that 50 % of parents of children aged ≥ 2 years underestimated their child's overweight or obesity (28). Parents were more likely to recognize overweight in children with higher BMI and in older children. A Norwegian study found that **i**n 2- to 5-year-old overweight children, 91.2% were considered to have normal weight by their parents (29). Socio-cultural factors may contribute to an underestimation of overweight, as big babies and children might be considered signs of wealth, health and good caregiving in some cultures (30). Furthermore, as an increasing proportion of the population is affected by obesity, there might be a transition of people's perception of thresholds for overweight and obesity. In this way, social norms may change. Of adolescents with obesity, 12 % in the Norwegian Ung-HUNT4 study considered themselves to have a normal weight (6). Such a shift in social norms may be an obstacle to prevention of childhood obesity.

Parenting, children's eating behaviour and sleep

As new-born, children have the ability to self-regulate their food intake. As they grow, their eating pattern is increasingly shaped by external factors, and self-regulation of eating seems to decrease with age (31). Young children are dependent of their parents and caregivers for food, including choice of food type, portion size, when feeding occurs, and parent's responsiveness to children's indication of hunger or distress (32). There is a complex interplay between children and their parents regarding eating, and both children's eating behaviour and parents' feeding practices are associated with children's BMI (31, 33). There is substantial causal evidence that parenting affects children's eating. At the same time, children's weight, and perhaps also their eating behaviour, may influence parenting (32). For instance, parental pressure on children to eat is negatively related to their BMI, while parents' restriction regarding food intake is positively related to offspring BMI.

The Children's Eating Behaviour Questionnaire is a parent-report questionnaire designed to assess children's eating styles related to obesity risk (34). Among behaviours positively associated with overweight are (i) Food responsiveness (tendency to eat in response to food cues as sight and smell), (ii) Enjoyment of food (general interest in food and desire to eat),

and (iii) Emotional overeating (35). On the other hand, "satiety responsiveness" (ability to recognize and adjust eating in response of fullness) and "slowness in eating" are behaviours associated with lower weight. "Feeding practices" is a term referring to strategies parents and caretakers apply to control what, when, and how much their children eat. A prospective study showed that parental feeding practices may predict children's eating behaviour (31). Parental use of food as reward at age 6 years (Instrumental feeding) predicted food responsiveness and emotional overeating at eight years of age, while parental encouragement to eat predicted more enjoyment of food two years later.

Furthermore, a recent review and meta-analysis indicated that short sleep duration increases the risk of childhood obesity (RR: 1.30; 95% CI: 1.20–1.42) (36) ref. Focus on parental feeding practices and children's sleeping habits may therefore be of importance in strategies to prevent and treat childhood obesity (31).

<u>Heritability</u>

Genetics play an important role predisposing individuals to obesity. *Monogenic disorders*, like congenital leptin deficiency, are rare (37). Leptin is a hormone, predominantly produced in adipose tissue that regulates energy balance by inhibiting hunger. Lack of leptin causes extensively increased food intake, resulting in early childhood obesity (38). However, the genetic risk of *common obesity* reflects the accumulation of several loci that each contributes to a small proportion of the total risk (39). Obesity susceptibility is linked to genes controlling signals of hunger and satiety, as well as insulin secretion, energy metabolism, lipid biology and formation of fat cells from stem cells (adipogenesis) (40). A genome-wide association study and meta-analysis of BMI identified 97 gene loci associated with BMI (41). The study suggests that common variation in these genes accounts for more than 20 % of the variation in BMI. Longitudinal studies following children over time indicate that genetic risk variants influence the development of obesity, partly by accelerating weight gain during infancy and childhood (42).

Epigenetics involve functionally relevant changes to the genome without modification to the underlying DNA sequence (43, 44). While the genome is the same in all somatic cells in an organism, there are specific structures and functions that distinguish one type of cell from another type. These differences are due to the cell type's unique gene expression patterns

determined during cellular differentiation, as in adipogenesis. These cell-specific gene expression patterns can be affected by an organism's environment throughout lifetime leading to phenotypical changes that have the potential of altering risk of some diseases. Epigenetic modifications may be heritable and affect gene expression and activity in the offspring. Gene-environment and life style interaction studies have found that an obesogenic environment might be amplifying genetic risks for obesity (39). There are reasons to hope that the identification of genes predisposing to obesity may lead to targeted preventive and therapeutic agents for obesity in the future. Importantly, individuals with high genetic risk may lower their risk by increasing physical activity and improving their diets (40).

<u>The importance of early life exposures</u>

The "Developmental origin of Health and Disease" concept
Early life exposures, such as under- and overnutrition, play a powerful role influencing later susceptibility to non-communicable diseases (45, 46). This in known as the "Developmental origin of Health and Disease" concept. One of the pioneers exploring this theory was Anders Forsdahl, previously a general practitioner. He reported in the 1970ies a higher risk of death from arteriosclerotic heart disease in men aged 40-69 years with poor living conditions and high infant mortality rates during early childhood in the far northern part of Norway, compared with men from more affluent counties (47, 48). In the 1970ies, overall living conditions were good throughout the country, and Forsdahl postulated that poverty during childhood and adolescence followed by prosperity was a risk factor for cardiovascular disease. In 1986 Barker and Osmond reported similar findings with a strong geographical relation between ischaemic heart disease mortality rates in England and Wales in 1968-78 and infant mortality rates in 1921-25 (49). They postulated that also *intrauterine* nutritional factors might cause adverse lifelong effects on health, and Barker later described this as "In utero programming of chronic diseases" (45). Several cohort studies with long-term follow-up indicate that thinness at birth and early childhood may be a risk factor for type 2 diabetes and cardiovascular disease, in particular if followed by an increase in BMI later in life (50-53). This growth pattern most likely represents low lean mass from birth,

which tracks throughout adult life, followed by accumulation of fat mass later in life (50, 52).

The Second World War Dutch famine

Men and women exposed to the Second World War Dutch famine (November 1944 – April 1945) during gestation were found to have increased rates of cardiovascular- and metabolic diseases, as well as breast cancer and obesity (54). Studies of these individuals support the developmental origin of disease hypothesis and have revealed that these men and women had epigenetic changes, compared with their unexposed siblings. Those exposed to famine in early gestation had doubled rates of cardiovascular disease, while those exposed in mid-pregnancy (a critical period for renal development) had higher risk of renal dysfunction (55, 56). The Dutch famine birth cohort study has also assessed transgenerational effects. Although grand-maternal exposure to famine during gestation did not affect grandchildren's birth weight, offspring of mothers who themselves had been exposed to famine in utero had increased neonatal adiposity and poorer health later in life (57).

The life course concept; obesity and gestational weight gain

A causal pathway for obesity is captured through the life-course concept, which offers a model stating that early life environment may modify genetic information, thereby modulating the risk of non-communicable diseases that may persist throughout the child's development and adult life (58, 59). Exposures may affect the physiological systems controlling appetite, stress responses, food preferences, fat deposition, body composition and metabolism. These processes may favour short time survival, fitness and reproduction, but may place a large number of children on the pathway to obesity, type 2 diabetes and cardiovascular disease when faced with cumulative exposures through an unhealthy diet and low physical activity (60). Today, a large proportion of young girls and women are obese, which might have adverse consequences not only for the next, but potentially also for future generations (61, 62). According to the Medical Birth Registry of Norway, 35.4 % of all women giving birth in Norway in 2018 had prepregnant overweight or obesity (63). Intrauterine exposures to maternal prepregnant obesity and excessive weight gain during pregnancy are associated with childhood obesity and subsequent cardio-metabolic risks, tracking from childhood into adult age (61, 64-66). This pattern may cause a "vicious

circle" of overweight and obesity being transferred from one generation to the next. The concordance between maternal and offspring obesity can stem from genetics, shared environment and lifestyle, as well as intrauterine exposure (62). Suggested mechanisms of intrauterine effects are maternal insulin resistance, epigenetic changes, inflammation, placental dysfunction, and stem cell differentiation (61, 64). After large maternal weight loss from surgery, prevalence of obesity in offspring is reported to be decreased by 52%, compared with siblings born before their mother went through weight loss surgery (67). By using a within-family study design, confounding by genetic and environmental factors may be reduced.

Gestational diabetes mellitus

Gestational diabetes mellitus is defined as hyperglycemia with onset or first recognition during pregnancy (68). During normal pregnancies, insulin resistance increases (insulin sensitivity declines) with advancing gestation, and a compensatory increase in insulin secretion maintains a normal glucose homeostasis. gestational diabetes occurs if pancreatic β-cells are unable to face the increased insulin demand during pregnancy. Women with gestational diabetes are at increased risk of complications during pregnancy and delivery, such as cesarean section and shoulder dystocia, and they have nearly a 10-fold higher risk of developing type 2 diabetes than those with a normoglycaemic pregnancy (69). The risk of gestational diabetes increases by age, obesity, and a family history of gestational diabetes and type 2 diabetes. Ethnicity has a great impact on the prevalence of gestational diabetes, and women of Asian origin have 2- to 7-fold greater risk of developing gestational diabetes than their European counterparts (23, 70, 71). This difference may partly be explained by heterogeneity in the gestational diabetes phenotypes between the populations, which can only partially be explained by genetic difference (72).

Effects of in utero exposure to hyperglycemia on offspring BMI and the development of adiposity vary between observational studies (73-75). While many studies have reported strong and independent associations between maternal gestational diabetes and large babies (macrosomia) at birth, few have found associations with obesity in early childhood (75, 76). However, evidence is growing that the effects of intrauterine exposure to hyperglycemia can emerge later in childhood (77, 78). As gestational diabetes is associated with maternal

overweight/obesity and excess gestational weight gain, it is difficult to disentangle the independent effect of hyperglycemia from the two other weight-related factors (62, 70, 79).

5.3 Consequences of childhood obesity

Childhood obesity can affect a child's immediate health, quality of life and educational attainment, and is a risk factor for adult obesity and non- communicable diseases, and the associated social and economic costs for individuals and the society are immense (1).

Burden of disease in childhood

Compared with peers with normal weight, children with obesity are more likely to have chronic health conditions as asthma, obstructive sleep apnoea and polycystic ovary syndrome, as well as cardiovascular risk factors as high blood pressure and blood lipids (1, 80). Childhood obesity is also associated with low self-esteem and psychological- and social problems (81). An inverse linear relationship between BMI and quality of life has been reported (81, 82). A systematic review and meta-analysis showed that compared with normal-weight girls, *obese girls* had a 44% increased odds of depression (83). No association with depression was found for *overweight* children, or among *obese or overweight boys*. However, it remains unclear if psychiatric disorders and psychological problems are a cause or a consequence of childhood obesity, or whether common factors promote both obesity and psychiatric disturbances in susceptible children and adolescents (84). Although most children with obesity do not have a psychiatric diagnosis, psychiatric symptoms are more prevalent in clinical samples of children with obesity, compared with normal weighted children (85). Furthermore, youths with overweight and obesity have a higher risk of being teased or bullied (86).

Tracking of childhood obesity

Childhood overweight and obesity often persists into adulthood, which increases the potential for morbidity and premature mortality across the lifespan (87). The risk of overweight in adulthood is at least twice as high in children with overweight, compared with normal-weight children (88). For obese children the risk is generally higher. The likelihood of childhood obesity tracking into adulthood is higher as the child gets older, and is affected by the severity of obesity, and the presence of parental obesity (80). Glavin et. al.

showed that important landmarks for the onset of being overweight at age 8 years are high birthweight, an increasing BMI standard deviation score during first nine months, and high BMI from 2 years of age (89).

Non- communicable diseases

Childhood obesity is linked with premature mortality and cardio-metabolic morbidity as gestational diabetes, type 2 diabetes, hypertension, coronary heart disease, and stroke (90, 91). This is worrisome, as the burden of cardio-metabolic diseases are immense globally. Type 2 diabetes is emerging as a worldwide leading cause of disability, and is observed across all levels of economic development (92). Coronary heart disease and stroke are the two leading causes of mortality worldwide (WHO, 2016). Data also show a strong association between higher BMI in adolescence and increased risk for several cancers, as leukaemia, Hodgkin's disease, colorectal cancer, breast cancer and others in adulthood (90).

Monetary costs

In addition to the individual's burden of disease and reduced life expectancy, obesity causes substantial monetary costs for the society (93). Overweight- and obesity-related diseases lead to medical costs (direct costs) covering treatment, diagnosis, rehabilitation and care. Temporary and permanent work loss related to obesity results in a substantial burden for national health and insurance pension system (indirect costs) (93). In Norway, employees with BMI > 30 kg/m^2 have three times more sick-leave days from work than do employees with BMI < 30 kg/m^2 (8, 94). They are also at higher risk of long-term inability to work

5.4   Prevention and treatment

In theory, weight loss is simple; intake of less energy than used. However, in real life and clinical settings, weight loss and weight maintenance are recognized as very challenging. To understand an individual's struggle to achieve and maintain weight loss, it is important to be aware that energy restriction, irrespective of starting weight, triggers biological adaptations, designed to preserve weight in situations with starvation/hunger (95). With the development of obesity, a life-long biological pressure to preserve an individual's highest sustained body weight may occur.

5.4.1 *Treatment of obesity in adults*

According to the Norwegian national guidelines on treatment of adults with overweight and obesity in primary care, basic interventions are improvement of diet and increased physical activity (96). If lifestyle changes are insufficient, pharmacological or surgical treatment should be considered. Lifestyle intervention and weight loss programmes designed for adults with obesity frequently report low effectiveness, especially in long-term follow-up trials (97). General practitioners have reported that dealing with obesity is frustrating, and that treatment for obesity often is ineffective (98). However, behaviour-based weight loss trials do indicate greater weight loss from interventions compared to control conditions (97). Pooled data from a recent review showed that individuals receiving lifestyle interventions had a greater mean weight loss (-2.4 kg) after 12-18 months, compared with controls (99). Of note, a recent cluster-randomised, primary care-led trial has shown promising results. Individuals (n=306) affected by type 2 diabetes and overweight or obesity were randomized to a dietary and lifestyle intervention or usual care (100). After initial 3-5 months with total diet replacement (phase one), the intervention group received structured support for long-term weight maintenance (phase two). Mean weight loss was 10 kg in the intervention group (1 kg in controls) after one year. After two years, 36% of patients in the intervention group (3% of controls) had sustained remission of diabetes (101).

Nevertheless, as treatment of obesity in adult life is difficult, the best and logical strategy is to prevent the development of obesity in the first place. Clinicians should therefore probably be more proactive in addressing obesity prevention in patients who are gaining weight or are overweight (95).

5.4.2 *Treatment of childhood obesity*

Lifestyle interventions

According to Norwegian and international guidelines, the main strategy for treating childhood obesity should be family-focused lifestyle interventions, including diet modifications, increased physical activity, less screen time and good sleep hygiene (80, 102). Unfortunately, behavioural interventions for treating overweight and obesity in children and youth are associated with low to moderate effect and high dropout rates (103).

However, two Cochrane reviews do conclude that multi-component behaviour-change interventions incorporating diet and physical activity may be beneficial when treating children aged 6 months to 17 years for overweight and obesity, but data from long-time follow-up is sparse. (103, 104).

Results concerning the importance of physical activity and exercise when treating childhood obesity are diverging, and have recently been a matter of debate in Norway (11, 105). One systematic review indicates that complex lifestyle interventions (including physical activity and diet), interventions on diet only, as well as interventions on physical activity only might induce a significant reduction in BMI in children and adolescents with overweight or obesity during a follow-up period of at least six months (106). On the other hand, another systematic review of systematic reviews with longer follow-up (two years or more) on treatment of pediatric obesity (age group 6 months-18 years) showed that there was no significant differences in BMI reduction between interventions with diet only, compared with combined interventions with diet and exercise (107). According to this review, interventions involving exercise alone (without calorie restriction or education) did not cause weight loss. Further, interventions involving parents and children were not superior to those involving parents only. In a Norwegian study, there were no additional long-term effect of group interventions (including weekly physical activity sessions), compared with individual counselling (108). After three years follow-up, both groups had a sustained decrease in BMI standard deviation score.

Communication with parents

Discussing a child's obesity with parents represents a health communication dilemma, and a good therapeutic relationship between parents and health professionals is essential (109). The noble intention of a health professional to help an overweight or obese child may leave parents feeling criticized for poor parenting, and especially the initial communication might be crucial (110, 111). Many parents do not perceive that their child is at risk, and parental ability to recognize overweight in their offspring is particularly poor concerning smaller children (28, 29). Knowledge on how parents' can be motivated to make and maintain a substantial change in lifestyle s of the whole family is limited. Parents' may perceive that they are already following a healthy lifestyle and stigma about excess weight and denial for

the issue might be barriers to engage in lifestyle programmes (111, 112). Motivational interviewing is a non-judgmental counselling technique, which involves reflective listening and addresses patients' ambivalence to change. This technique may be a useful tool for health professionals treating paediatric obesity (80).

Pharmacological and surgical treatment

The role of pharmacological and surgical treatment of childhood obesity is limited. In adolescents, medical interventions with orlistat and metformin may give modest effects (106). In one-year placebo controlled studies, BMI was reduced less than one kg/m^2 by orlistat, and 1.1-1.4 kg/m^2 by metformin (80). There are ongoing trials for medical treatment of adolescents with glucagon-like peptide 1 receptor agonists (GLP-1), and the antidepressant buprion in combination with naltrexone (113).

Bariatric surgery to induce weight loss includes a variety of technical procedures performed in people who are obese despite conventional treatment. Some procedures effectively reduce the amount of food a person can eat per meal, other work by reducing the absorption of nutrients, while others do both. Bariatric surgery also alters the gut hormone levels that are responsible for hunger and satiety, leading to a new hormonal weight set point.

In adolescents with severe obesity, bariatric surgery may be used for treatment when conventional treatment has failed (102). Consensus guidelines on bariatric surgery in adolescents have been established by various medical professional bodies in several countries (113). In terms of patient characteristics the recommendations generally cover: a minimum age, usually in mid-adolescence; the patient should have reached sexual maturity; the presence of severe obesity, usually BMI >35 kg/m^2 with an obesity-associated complication, or BMI >40 kg/m^2; the persistence of obesity despite participation in a formal multidisciplinary programme of lifestyle modification and pharmacotherapy; the patient being able to give informed consent; and the adolescent and family being willing to participate actively in the treatment programme and agreeing to sustained follow-up after the operation. In an Australian prospective, randomized controlled trial, 50 adolescents between 14 and 18 years with a body mass index (BMI) higher than 35 kg/m^2 were assigned either to a supervised lifestyle intervention or to undergo gastric banding, and followed up for 2 years (114). Overall, the mean weight loss in the gastric banding group was 34.6 kg,

representing 12.7 BMI units. The mean loss in the lifestyle group was 3.0 kg, representing 1.3 BMI units. In Norway, the randomized, controlled 4XL-study is now including adolescents between 13-18 years with morbid obesity (115). The main aim of the study is to determine whether laparoscopic gastric bypass surgery gives more health benefits than standard conservative treatment, and whether the method has high safety and a low complication rate.

5.4.3   Prevention of childhood obesity

Primary prevention of obesity in childhood is likely to be more effective and less costly than interventions to restore normal weight in individuals who already have developed overweight and obesity (116). Parents are the closest to nurture their children and provide their basic need. As obesity has physical and psychological health consequences for children and adolescents, not only parents, but also governments and societies can be considered to have a moral responsibility to reduce the risk of childhood obesity (1, 2). This resonates with the UNICEF "Convention of the Rights of a Child", which states that children have a right to a healthy life (117). Basic elements of prevention are i) a healthy diet including daily intake of fruits and vegetables, less energy-dense and processed foods and sugar-sweetened beverages, ii) daily physical activity in various forms, iii) limited sedentary screen time, and iv) good sleep hygiene (80, 102).

In 2019, WHO published guidelines on physical activity, sedentary behaviour and sleep for children under 5 years of age (118). The recommendations are age-specific, and as an example; for children of 3-4 years of age, sedentary screen time should not exceed 60 minutes, children ought to spend at least 180 minutes in physical activity, of which at least 60 minutes should be of moderate to vigorous intensity, and recommended sleep duration is 10-13 hours.

Parents and other primary caretakers have the main responsibility for children's lifestyle. Given the complexity of causation, public health scientists recognize that the solution for childhood obesity cannot solely be found in individual-level actions, but requires a whole-of-society systems approach (119-121). Further, reviews have not found convincing beneficial effects of home only-based interventions to prevent childhood (122, 123).

Importantly, the global smoking cessation campaign showed the positive effects of multisystem and multilevel actions on public health (124). To prevent childhood obesity, societal, social, cultural and biological factors should be taken into account, including arenas for an active lifestyle for families and children, availability of healthy foods, and population-based strategies as regulation of unhealthy food marketing targeting children (119).

The life-course approach

The WHO report *Ending Childhood Obesity*, recommends a systems approach that strongly recognizes the importance of early life (Figure 1) (1). Antenatal care and family-oriented preventive health services for mothers and children are critical to promote a healthy nutrition and active lifestyle (62).

Figure 1.
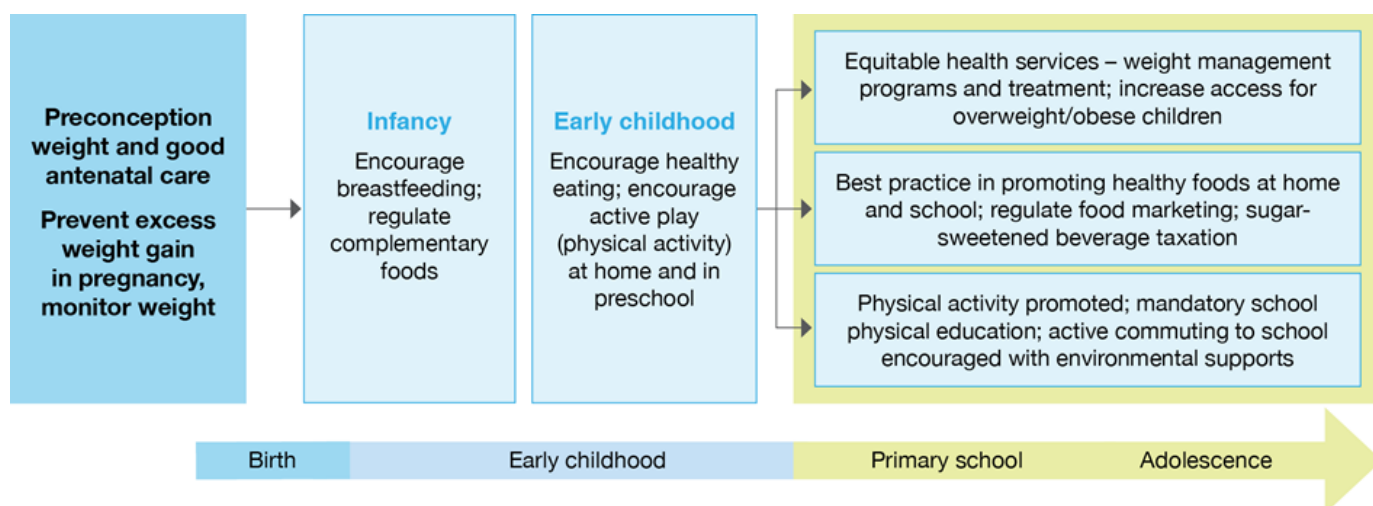
Source: Adapted from WHO Report of the Commission on Ending Childhood Obesity[14]

Interventions

Two recent systematic reviews on interventions to prevent overweight and obesity in children conclude that school-based interventions with combined diet and physical components, as well as a home-element had the greatest effectiveness for children and youth aged 5-19 years (122, 123). However, the effect size of the intervention might be small; about -0.25 kg/m^2 BMI units, and -0.05 in z-score (122). Another review found that for children aged 6-18 years, interventions with physical activity only can reduce the risk of obesity, while diet only interventions showed no effect, and that combined interventions

may be effective (116). In preschool children (0-5 years), interventions that included diet combined with physical activity reduced the risk of obesity by -0.07 kg/m^2 BMI units and -0.11 in z-score (116). Results concerning dietary-only and physical-activity-only interventions in this age group differ among reviews, and firm conclusions are hampered by poor methodological quality in the few published preschool-based randomized controlled trials (116, 122, 123).

Consorted actions against the obesogenic environment

WHO suggests a whole-society approach to improve the obesogenic environment surrounding most children in the world today (1). This system approach includes

1. Urban and transport planning to give opportunities for physical activity,
2. Health sector engagement already from, or even before, conception,
3. Education sector providing health education, nutrition and opportunities for physical activity,
4. Agricultural and trade policies providing affordable, available, and high-quality food,
5. Governments providing equity with support of marginalized and vulnerable population groups,
6. Private sector taking responsibility of food marketing which stimulate the consumption of unhealthy food and sugar-sweetened beverages as digital campaigns accessed through smart-phones, food packaging attracting children, and in-store marketing in children's eye-level, as well as
7. Parents, caregivers, volunteers, academic institutions (117, 119, 125).

The progress setting up coordinated actions globally and at national levels has been slow, although there have been some local trans-sectoral initiatives showing promise (2).

The "Amsterdam Healthy Weight Programme" is a public health programme set up to prevent and treat childhood obesity (126). The programme recognizes that healthy weight is a collective responsibility, and the healthy choice should be the normal choice. Schools, volunteers, food industry, supermarkets, restaurants, midwifes, maternity care, youth healthcare, and policy makers are among groups contributing to this project that show promising results. From 2012-2017 the proportion of children with overweight and obesity

in Amsterdam went down. The downward trend was particularly noticeable among children from disadvantaged neighbourhoods and families with low socio-economic status, which are among the hard to reach groups. To date, children in Amsterdam are breastfed for a longer period than in 2012, and they consume less sugar-sweetened beverages. However, goals set to increase physical activity and decrease sedentary behaviour have not yet been achieved.

5.4.4 *Preventive services for children and pregnant women in Norway*

Organization

All municipalities in Norway are obliged by law to offer preventive services for all children and youth aged 0-20 years, as well as follow-up of all pregnant women (127). Services are located at Child Health Clinics, schools, ant at the general practitioners' offices. Everyone registered in the National Registry as resident in a Norwegian municipality is entitled to have a regular general practitioner, who is obliged to provide primary health care (preventive and curative) for people on his/her list. At the Child Health Clinics, staff is made up by public health nurses, doctors and midwifes, and often by physical therapists and psychologists as well. Services are free of charge and include regular health controls and follow-up, preventive health services, information, home visits, and a vaccination programme. Services should cooperate with general practitioners, secondary health care services, as well as other municipal services and institutions. Is voluntary for the population to make use of these municipal services, but practically all children attend Child Health Clinics and schools for regular follow-up. Most pregnant women are followed by midwives that are connected to the Child Health Clinics.

Preschool children are invited for routine check-ups at age 6 weeks, at 3, 6, 12, and 15 months, and at 2 and 4 years (128). Public health nurses routinely measure children's weight and height at each control, and data are stored in electronic health records. Healthy pregnant women in Norway are offered at least eight controls in primary health care, including one ultrasound scanning (128). Women can choose if they want follow-ups at the Child Health Clinic, at their regular general practitioner's office or a combination of the two. If serious complications occur, women are referred to specialized secondary health care.

According to the current Norwegian clinical guidelines, prevention and treatment of childhood overweight and obesity is mainly a primary care responsibility (102). If complications develop, the general practitioner has the main responsibility. The threshold is high for referring the child to specialist health care; when severe obesity or serious complications occur. At present, services are fragmented, children may fall out of follow-up and actions are taken too late in the course of obesity development. Follow-up of pregnant women with prepregnant obesity (BMI 30-35), screening for gestational diabetes and treatment of mild gestational diabetes, as well as monitoring gestational weight gain is also a primary health care responsibility in Norway (129). The current *Norwegian guidelines on prevention and treatment of childhood overweight and obesity* were published in 2010 (102). They are outdated and do not reflect the systems- and life course approach strategies outlined by WHO (1), nor do they give guidance on how to organize local interdisciplinary and trans-sectoral efforts. The current *Norwegian guidelines for primary health obstetric care* offers minimal information on how to prevent gestational weight gain, gestational diabetes and postpartum weight retention (129). This makes it difficult for health professionals to help families struggling with overweight and obesity across generations, leaving us with the feeling of helplessness.

5.5   Concepts and definitions

5.5.1   Ethnicity

The term *ethnicity* is complex and refers to the social group a person belongs to, or are perceived to belong to, as a result of a mix of cultural and other factors (130). These factors may include one or more of language, diet, religion, geographical and ancestral origins, as well as physical features traditionally associated with race. Characteristics defining ethnicity are not fixed or easily measured, and there is no standard, widely accepted protocol for the collection of ethnicity or ethnic group data (131). Ethnicity is not the same as nationality, race, religion or migrant status, but may include facets of these concepts (132). The term *nationality* is defined as the country a person belongs to by citizenship, while the term *race* implies common hereditary physical/genetic characteristics. Ethnicity has often, especially

in the United States, been used as a proxy for socioeconomic position as valid data on education and income often are missing (133). This is problematic as not all members of an ethnic minority may be economically disadvantaged or deprived, and there may be socioeconomic differences in health within ethnic minority populations. Further, common interests, like for instance the rights of the working class, may be shared between, rather than within ethnic groups. In health research, it is common to use country of birth or mother's country of birth as a proxy for ethnicity (132). However, some ethnic minority groups hold on to their culture and traditions over several generations. In that way, grand- and great grandchildren of immigrants may keep lifestyle patterns different from the major population, concerning diet, physical activity, parity and other factors (131). Further, if they do not marry outside their ethnic minority group, possible differences in genetics and epigenetics compared with the major population may persist.

5.5.2   *Migration, minorities and health*

Norway is becoming more and more a multicultural society. In 1970, less than 2% of the population were immigrants. In 2020, almost 18 % of the Norwegian population are immigrants or persons born of immigrant parents (Statistics Norway, figure 2). In Oslo, this proportion is 33 %. In Norway, 48% of immigrants and persons born of immigrant parents, come from Europe, while 35 % come from Asia, and 14 % from African countries.

Figure 2. Immigrants and persons born of immigrant parents; country of origin (Statistics Norway, 2020)

Legend:
- Nordic countries
- Western Europe outside Nordic countries
- Eastern Europe (European Union)
- Eastern Europe (outside European Union)
- Africa
- Asia
- South- and Central America
- North America and Oceania
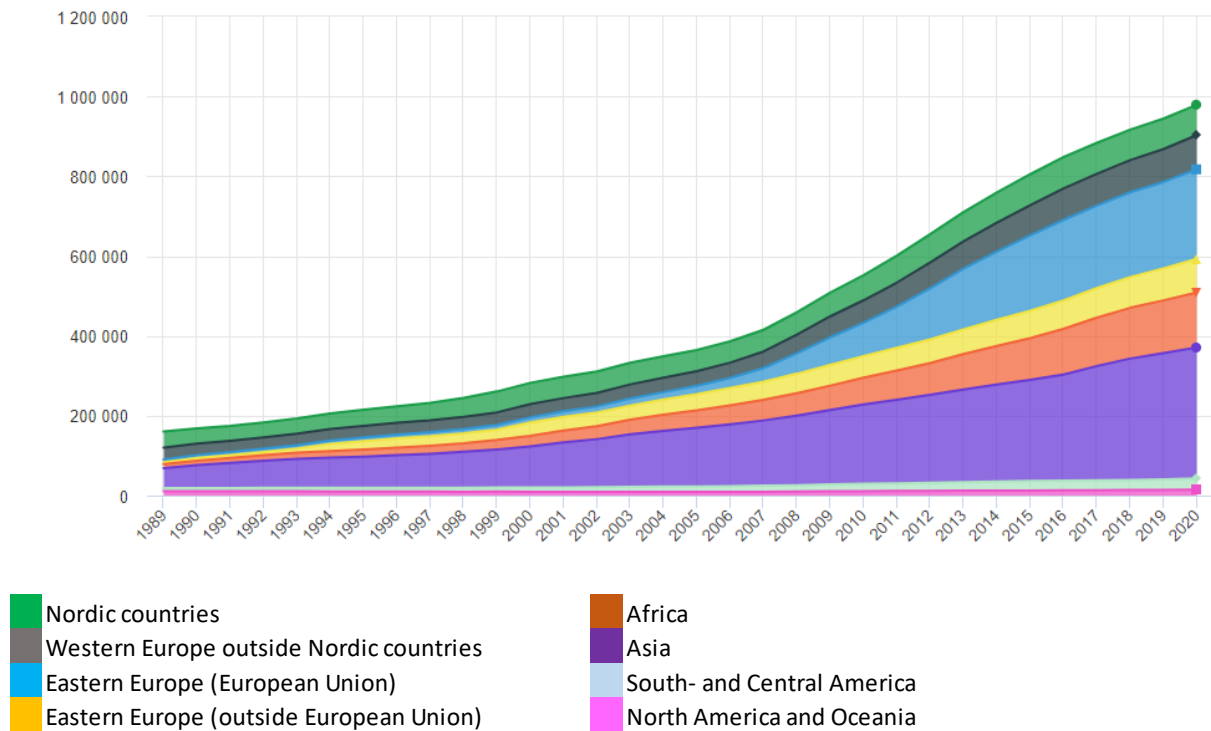
Across city districts in Oslo, the prevalence of overweight /obesity among women is more than the double in Stovner, where approximately 60% of the population has ethnic minority background from countries outside Europe, compared with St. Hanshaugen (134, 135). This underlines the importance of effective preventive measures in minorities and «high-risk neighbourhoods».

Figure 3. Population (%) of immigrants and persons born of immigrant parents in different city districts of Oslo (Municipality of Oslo)
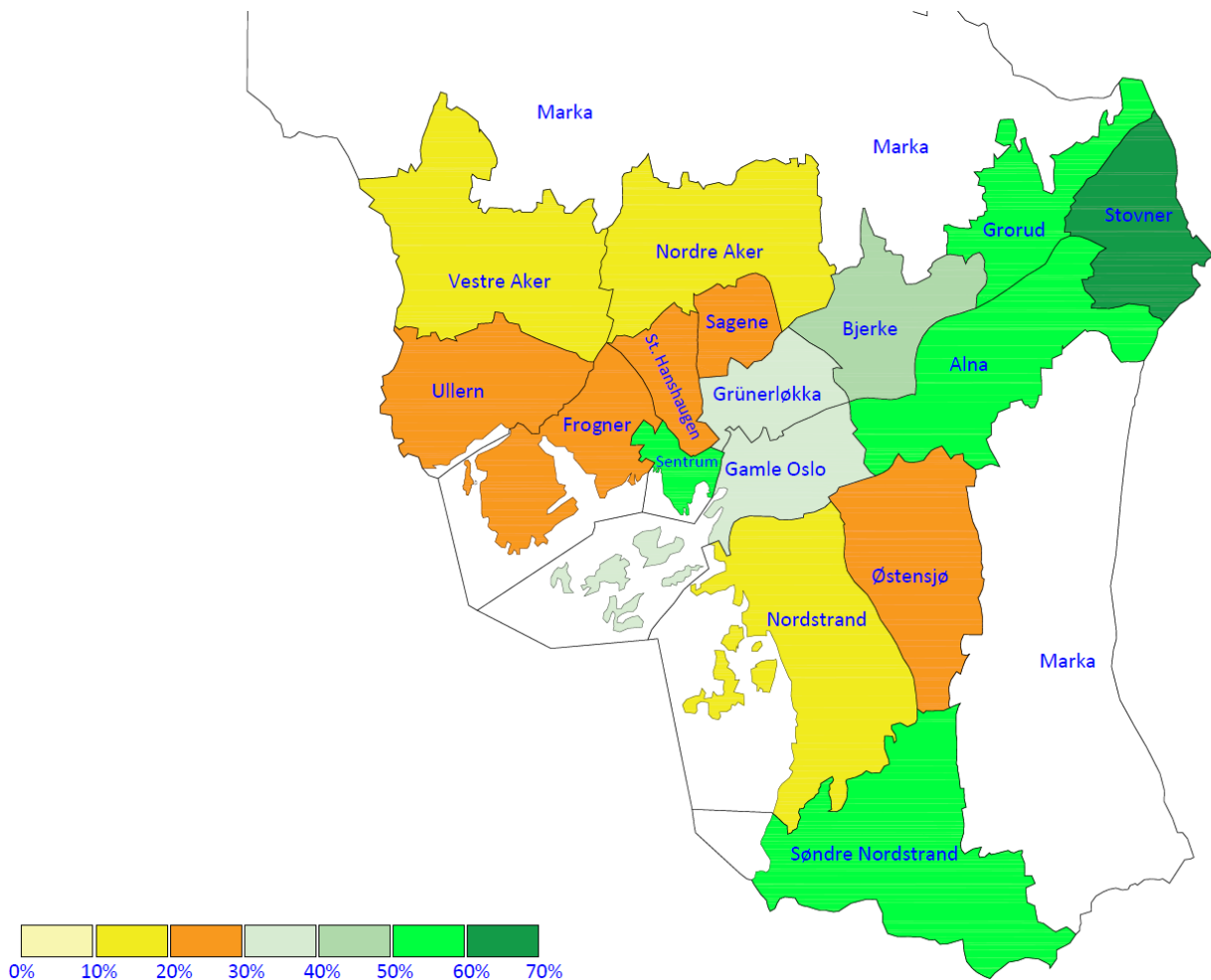
Immigrants moving from low-income to high-income countries often have a lower overall mortality compared with the major population in the new country (136, 137). This is referred to as the "healthy migrant effect". Since they have the ability, resources, and opportunity to migrate, immigrants are commonly not representative of the population they travel from (138). However, with prolonged lengths of stay, the mortality of immigrants may increase and approach that of the majority population (136). Among suggested explanations are disadvantageous acculturation, as adopting unhealthy behaviours, and burden of chronic stress related to migration. Further, these groups may have a lower health literacy compared with the host population (15). In Europe, ethnic minorities from South Asia are diagnosed with type 2 diabetes 10 years earlier and at a lower BMI than the

majority population (139-141). They have a higher prevalence of cardiovascular diseases, including stroke, but a lower prevalence of cancer. In Norway, higher rates of overweight and obesity is specially reported among Turkish and Pakistani adult immigrants, and boys with Western and Middle East /North African backgrounds (137). Unhealthy dietary intake and less physical activity may be main risk factors. Further, the Norwegian immigrant population is more affected by some obstetric-related complications, perinatal mortality, and musculoskeletal disorders and disability pensions.

5.5.3 *Childhood BMI development*

5.5.3.1 BMI growth in childhood and definitions of overweight and obesity

Body mass index (BMI) is a key index for obesity, relating weight to height. An individual's BMI is calculated by weight in kilograms divided by height in meters squared (kg/m^2). In adults, and according to WHO, BMI $>= 25$ kg/m^2 is usually defined as overweight, and BMI $=>30$ kg/m^2 is defined as obesity (142), although definitions suggested for Asians are lower (143). BMI evolves naturally as children, therefore definitions of normal BMI in children varies according to the child's age and sex (1). Children have a rapid increase in BMI during the first year of life and reaches a maximum (adiposity peak) at age 6-8 months (144). BMI then declines and reaches a minimum (adiposity rebound), on average, at 5 to 6 years of age, before a gradual increase through adolescence. In order to monitor healthy BMI growth in children, growth standards (or growth charts) are developed. (Figures 5 and 6). However, BMI is based on weight, and does not differentiate between fat mass and lean mass (145). This problem is further discussed thoroughly in the discussion section (chapter 10.1.1.2). The relation between BMI and percentage of body fat depends on age and sex, and differs across ethnic groups (143).

Terms (Figure 4)

1) *z-scores* is a measure used in statistics, indicating the number of standard deviations (SD) an observation is above or below the mean of the reference population. Z-score (z) is calculated $z = (x-\mu) / \sigma$, where x is the raw score, μ is the reference population mean, and σ is the population standard deviation.

2) *Growth standards (or growth charts)* are constructed using longitudinal length and weight data measured at frequent intervals in a healthy population of children, i.e. WHO's growth standard

3) *Percentile (or centile)* is a measure used in statistics, indicating the value below which a given percentage of observations in a group of observations falls. I.e. if a child's BMI value corresponds to the 75th percentile, 75% of children in the reference population (on which the growth chart is based) will have a BMI below this particular child's BMI

Figure 4. Illustration of percentiles, SD, and z-scores

33
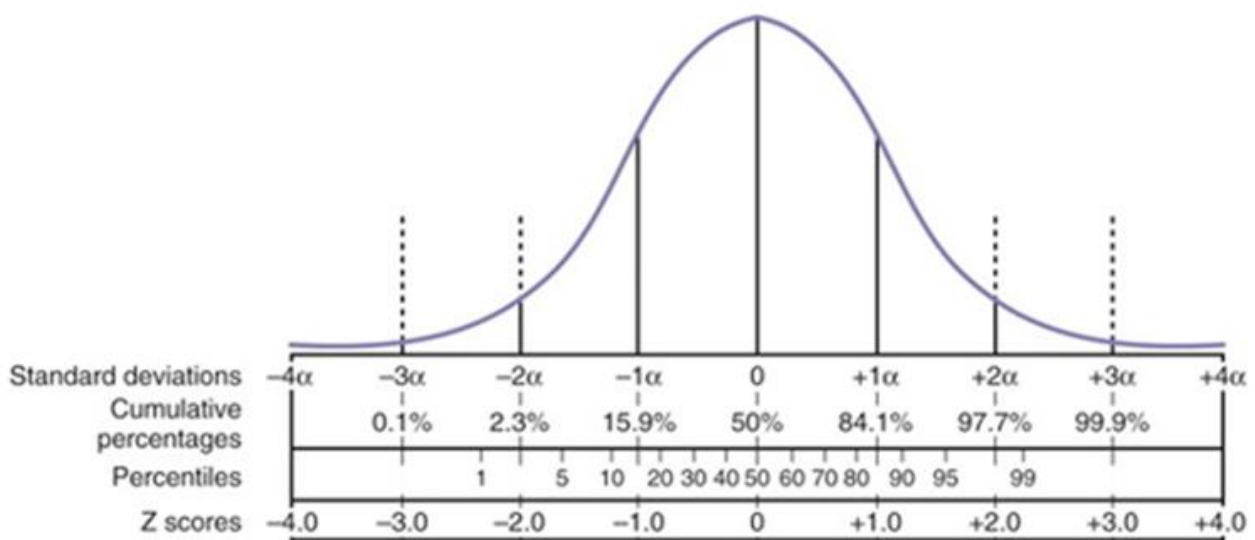
Figure 5.

Growth chart example; BMI for age, girls from birth to5 years (WHO Child growth standards)
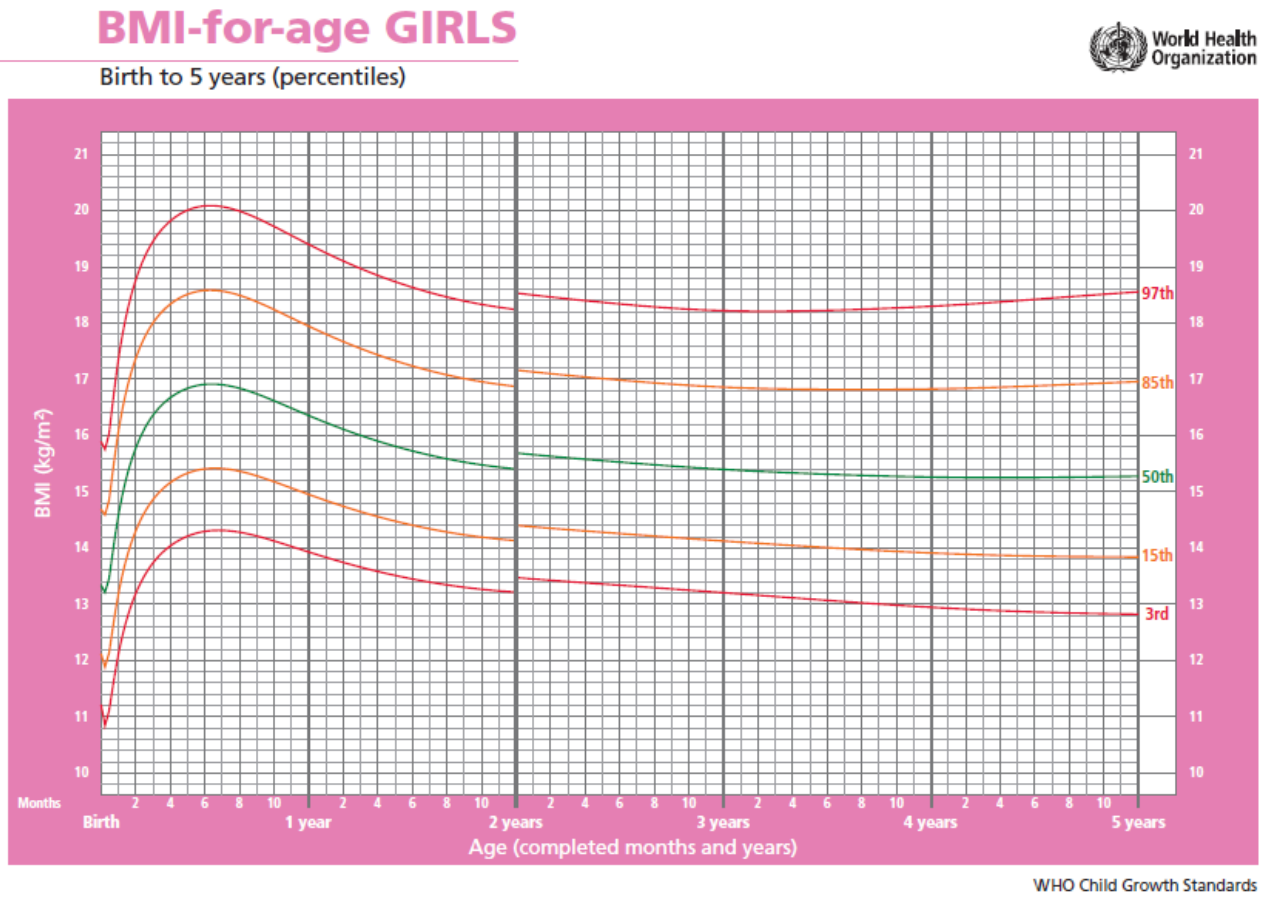
Adiposity peak

Figure 6. Growth chart example; BMI (KMI) for age, girls 2-19 years. (Juliusson et. al., Bergen Growth Study)
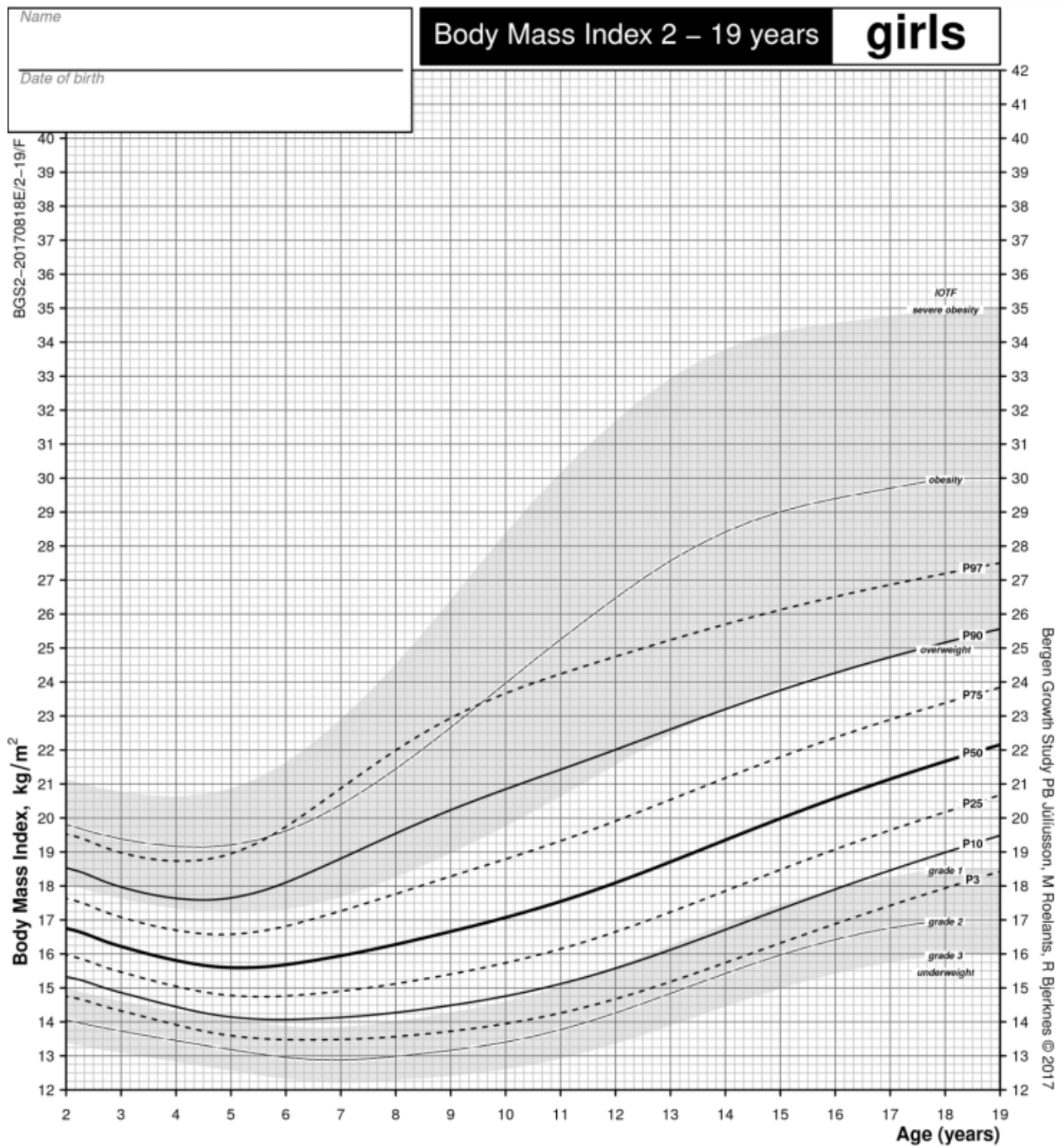
Adiposity rebound

5.5.3.2 The South Asian "thin-fat" phenotype

To promote healthy growth in children in today's multiethnic societies, knowledge about ethnic differences in BMI is important (13, 22). The body composition of populations with South Asians origin differs from those with European and African origin, as South Asians already from birth have a relatively higher ratio of fat to lean mass for any given level of BMI (22, 146). This body composition has been referred to as the "thin-fat phenotype", associated with lower insulin production (reduced beta-cell function) and muscle clearance, and higher insulin resistance, contributing to a "low metabolic capacity" among South Asians (146, 147). Among suggested underlying causes for the "thin-fat" body composition are evolutionary adaptations to ecological stresses linked to increasing population, undernutrition and malnutrition, favouring the survival of babies with relatively preservation of the head and subcutaneous fat at the expense of muscle tissue and inner organs (51, 147). Exposed to a "high metabolic load" imposed by westernized lifestyles, individuals with South Asian origin develop diabetes at a younger age and a lower BMI compared with Europeans (10, 22). Further, toxic substances from high heat and reheated cooking are suggested to harm the beta-cells (130).

6   Aims of the thesis

The overall goal of this thesis was to improve the knowledge base on how to promote healthy growth in pre-school children, first, by improved early identification of preschool children at risk of developing obesity in a multiethnic population, and second, on how to improve the communication between parents and health professionals about their preschool child being overweight.

The specific aims were to:

- **Paper I:** investigate ethnic differences in overweight and thinness in a multi-ethnic population-based cohort of preschool children in Norway, and associations with maternal and early postnatal factors.

- **Paper II:** investigate the independent association between maternal gestational diabetes and children's BMI trajectories from birth to 4-5 years of age, and the effects of prepregnant overweight/obesity and gestational weight gain not mediated through gestational diabetes, in a multiethnic population of pregnant women universally screened for gestational diabetes, and treated accordingly.

- **Paper III:** Explore parents' experiences when a health professional identifies their preschool child as overweight

7   Materials and methods

<u>Principal choices and study design</u>

For this thesis, I have used a mixed methods design to address my research questions at different levels, increase the validity of the results, and broaden the understanding of my findings. In the following, I will provide an outline of materials and methods for the three papers. In the quantitative studies (Paper I and II), I used a population-based cohort design, based on data from the STORK Groruddalen Research Program. Therefore, these papers will be presented together. In Paper III, a qualitative approach was used.

7.1   The quantitative studies; Paper I and II

7.1.1   Context and recruitment: The STORK Groruddalen study

The STORK Groruddalen Research Program was a population-based cohort study set up to identify predictors for gestational diabetes mellitus and foetal growth in a multiethnic population in Oslo, and to improve the identification of high-risk pregnancies and reduce adverse short and long-term outcomes for mothers and children (148). The data collection took place from 2008 to 2011, at Child Health Clinics in the administrative city districts of Stovner, Grorud and Bjerke in the North Eastern part of Oslo. These districts covered a population of 82,500 inhabitants with a wide range in socioeconomic status. A large proportion of the population had non-Western origin. The majority (75-85 %) of pregnant women in the study districts attended antenatal care at the local Child Health Clinics, and virtually all children were followed regularly for free routine check-ups and vaccinations (148).

Before and during data collection, information about the study was widely distributed in the participating city districts, and general practitioners were asked to refer pregnant women early in pregnancy to the local Child Health Clinics. The study intended to include all eligible pregnant women living in the study districts. Information material and

questionnaires were available in eight languages, covering the largest ethnic groups; Arabic, English, Sorani, Somali, Tamil, Turkish, Urdu, and Vietnamese (148).

Eligibility criteria:

Pregnant women were eligible for the study if they were

- living in one of the three city districts (Stovner, Grorud and Bjerke)
- planning to give birth at one of the two study hospitals; Akershus University Hospital or Ullevål University Hospital
- pregnant and ≤20 weeks' gestation
- not having diabetes diagnosed before pregnancy or other diseases requiring intensive hospital follow-up during pregnancy
- able to communicate in Norwegian or any of the eight languages to which all the information materials and questionnaires were translated
- able to give informed written consent.

Inclusion

During the inclusion period from May 2008 to May 2010, 1918 women attended the Child Health Clinic for antenatal care. Of these, 1114 (58%) women were eligible for invitation to participate in the study (148). The main reason for exclusion was late attendance (second half of pregnancy) at the Child Health Clinics. Of the invited women, 291 (26 % of the invited) refused to participate. The main reasons were lack of time due to work obligations or care of small children. To facilitate the inclusion of Pakistani and Somali women, they were allowed to be included until gestational week 25.

Altogether, 823 healthy pregnant women were enrolled at mean week 15.1 (SD 3.4) of gestation. These women represented 73.9% of invited women and 43% of all women who attended the clinics during the inclusion period. They gave birth to 783 live-born singleton neonates (Figure 7, Flow chart). Of the study sample, 59 % had non-Western origin.

Figure 7.
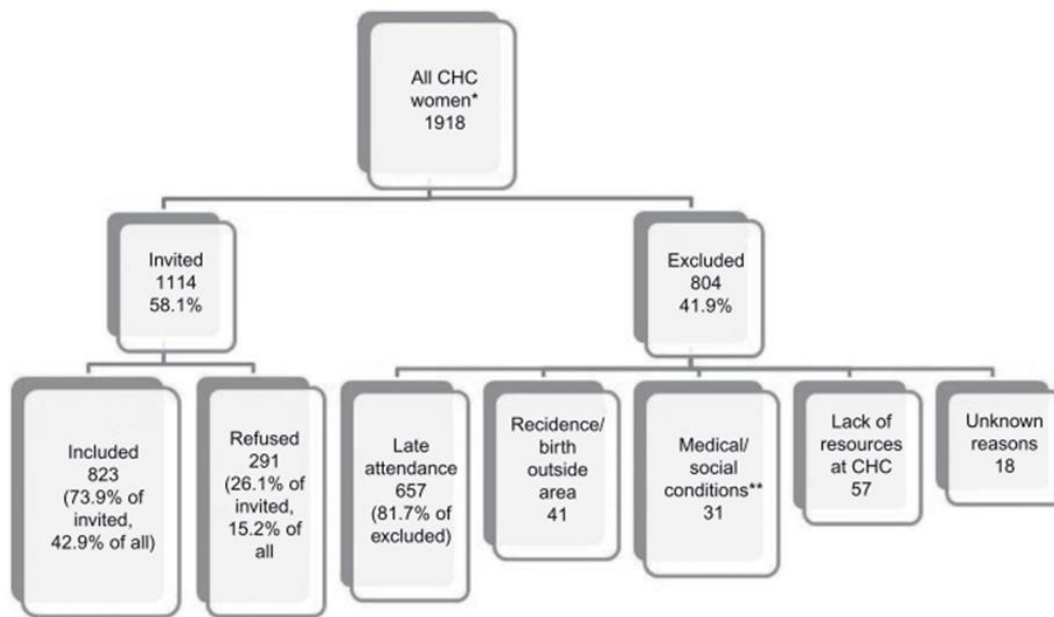
Flow chart, the STORK-Groruddalen cohort study

7.1.2 Data collection

7.1.2.1 Mothers

Trained and certified study staff members collected data from mothers at the Child Health Clinics at two visits during pregnancy and one visit at about 14 weeks postpartum. Study midwifes collected questionnaire data by interviews, supported by professional interpreters when needed (148). The questionnaires were validated and pilot-tested for clarity and feasibility (see Appendix for questionnaires). Study midwifes also performed anthropometric measurements of the mothers. Study personnel collected fasting blood samples and morning urine samples. At week 28 of gestation, all mothers were offered a standard 75g oral glucose tolerance test to identify those with gestational diabetes.

7.1.2.2 Fathers

At the second visit (28 weeks of gestation), mothers received information material and a two-sided questionnaire for the father to fill in, together with a consent form. The questionnaire included demographic variables, self-reported height, weight, ethnicity,

education, occupation, birthweight, and information about their health and cardiovascular disease and diabetes among family members.

7.1.2.3 Children

Ultrasound examinations of the foetus were performed at 24, 32 and 37 weeks' gestation at one of the local Child Health Clinics by experienced foetal ultra-sonographers. At birth, children's birthweight and crown-heel length were routinely measured. The last child was born in October 2010.

During 2014-2015, we retrospectively collected information about children's postnatal growth using routine data (height, weight and head circumference) up to 4-5 years of age from health records collected at the local Child Health Clinics. By 2015, all children had turned 5 years, and we had access to electronic health records of the 592 children still living in the city of Oslo. However, 192 families had moved out of Oslo. To get hold of anthropometric data on these children, we sent letters including mothers' signed consent to 62 Child Health Clinics throughout the whole country. We received data from 50 Child Health Clinics on 168 additional children.

7.1.3 Variables

We used different sets of variables, as well as different criteria for the diagnosis of gestational diabetes. Table 1 gives an overview of variables.

Table 1. Overview of variables paper I and II

	Study I	Study II
Primary outcome variables	Thinness, overweight or obesity at age 4-5 years	Children's BMI and BMI growth from birth to 4-5 years
Main exposure variables	Ethnicity, 3 main ethnic groups; European, South Asian, Middle East/North African	Gestational diabetes (WHO 1999 criteria) Maternal prepregnant BMI Maternal gestational weight gain
Covariates		
Mothers	Age	Age
	Parity	Parity
	Education	
		Socioeconomic position
		Ethnicity, 5 main ethnic groups; European, South Asian, East Asian Middle East / North African, Others
	Prepregnant physical activity	
	Mother's dietary pattern	
	Gestational diabetes (WHO 2013 criteria)	
Children	Sex	Sex
	Breastfed 14 weeks postpartum (subsample)	Breastfed 14 weeks postpartum (subsample)
	Birthweight	Gestational age

7.1.3.1   Anthropometric measurements of children

All anthropometric data collected on children were derived from the two birth wards, and routine measurements at the local Child Health Clinics where children met for check-ups.

At birth

Immediately after birth, staff at the birth wards of the two study hospitals routinely measured birthweight (without a diaper) to the nearest gram on electronic scales, calibrated by study staff (maximum difference: 5 g) (148, 149). Within 72 hours after birth, crown-heel length was measured without a diaper to the nearest 0.1 cm by using a measuring rod, with the head firmly held while stretching the legs, along with other study-specific anthropometric measurements, in the majority of children (unless contraindicated due to medical conditions restricting handling of the neonate) (149). In those with missing study-specific measurements, routine measurements were used.

At Child Health Clinics

Local Child Health Clinics are obligated to invite all children for check-ups at age 6 weeks, at 3, 6, 12, and 15 months, and at 2 and 4 years (128). Measurement recommendations are provided (150):

1) Body weight should be measured on digital, regularly calibrated scales. Up to 2 years of age, the child should be weighed naked, without a diaper, to the nearest 10g. From the age of 2 years, weight should be measured in underwear, to the nearest 100g.

2) Up to 2 years of age, crown-heel length should be measured with the child lying, without a diaper, to the nearest 0.1 cm, by using a measuring rod. The child's head should be firmly held while stretching the legs. From the age of 2 years, standing height should be measured in light clothing, without socks and shoes, using a fixed, regularly calibrated stadiometer, to the nearest 0,1 cm.

BMI

We calculated BMI as weight / height^2 (kg/m^2). We further used the age- and sex-specific BMI cut-off values (z-scores) defined by the International Obesity Task Force (151) to classify weight status as an ordinal variable with four levels: "thinness", "normal weight", "overweight" and "obesity" (Table 8). These cut-off values correspond to centile curves passing through BMI 18.5 kg/m^2 (thinness grade 1), BMI 25 kg/m^2 (overweight) and BMI 30 kg/m^2 (obesity) at the age of 18 years. In Paper III, we used the same cut-off values, here referred to as ISO-BMI, i.e. ISO-BMI above 25 kg/m^2 is defined as overweight, and ISO-BMI above 30 kg/m^2 is defined as obesity.

We defined *BMI growth* as changes in BMI units per month.

Table 2. Child anthropometrics at birth and at routine check-ups at Child Health Clinics – numbers with available data

Child anthropometrics	Age (months)							
	Birth	1.5	3	6	12	15	24	48
Length n	680	445	662	693	670	639	639	617
mean length, cm (SD)	49.9 (2.2)	56.4 (2.2)	6108 (2.4)	68.2 (2.7)	76.6 (2.9)	80.4 (3.0)	91.2 (4.2)	107.2 (4.7)
Weight n	734	664	666	693	671	642	655	618
mean weight, g (SD)	3458 (517)	4948 (682)	6374 (842)	8068 (1027)	9948 (1225)	10716 (1305)	13491 (1899)	18043 (2662)
BMI n	680	445	661	692	670	637	638	638
mean BMI, kg/m^2 (SD)	13.87 (1.38)	15.62 (1.46)	16.67 (1.53)	17.30 (1.61)	16.90 (1.48)	16.56 (1.40)	16.15 (1.50)	16.15 (1.50)

7.1.3.2 Explanatory variables

Main exposure variables

(see table 1 for overview)

Ethnicity

At inclusion, study midwifes asked the women about their and their parent's country of birth. The country of birth of child's mother, or the maternal grandmother's country of birth if this country was outside Europe, defined the child's ethnicity. We had information on 81 % of fathers, and very few children (<5%) had mixed ethnicity, and most of them had mothers from East Asia.

We grouped ethnic origin into five categories:

1) Ethnic Europeans (primarily from Norway and other Scandinavian countries)
2) South Asians (primarily from Pakistan and Sri Lanka)
3) Middle Easterners / North Africans (primarily from Iraq, Turkey, Afghanistan, Morocco, Somalia, and Ethiopia)
4) East Asians (primarily from Vietnam, Thailand and the Philippines)
5) Others (from Sub-Saharan Africa, Central-, and South America)

Maternal prepregnant BMI

At inclusion, height was measured to the nearest 0.1 cm using a fixed stadiometer (checked against a standard meter before study start and twice yearly) (148). Weight was measured in light clothes to the nearest 100g using medically approved scales (Tanita, Tokyo, Japan). Women were thereafter asked about their weight prior to conception, and prepregnant BMI was calculated based on measured height and self-reported pre-pregnancy weight. This variable was categorized according to WHO as; normal- or underweight (BMI < 25 kg/m^2), overweight (BMI 25-30 kg/m^2), and obesity (BMI ≥ 30 kg/m^2).

Maternal gestational weight gain

was calculated as the difference between her weight measured at week 28 during pregnancy (at the time of the glucose tolerance test) and self-reported prepregnancy weight at inclusion (152). We divided this variable into tertiles, using the middle tertile as reference.

Gestational diabetes

The diagnosis of gestational diabetes was based on a standard oral glucose tolerance test, offered to all women at week 28 of pregnancy (148). Pregnant women's fasting glucose was measured on site before they received 75 g glucose dissolved in water (153). Two hours thereafter, the glucose measurements were repeated. According to the protocol, glucose was measured in venous EDTA-blood on site within five minutes after vein puncture, with a patient-near method (HemoCue 201+, Angelholm, Sweden) calibrated for plasma. This method was used to (i) avoid preanalytical glucose reduction due to blood cells consuming glucose, before analyses at the hospital laboratory at Akershus University Hospital; and (ii) receive immediate results to allow optimal patient information and necessary actions if gestational diabetes was diagnosed.

Worldwide, there are several definitions of gestational diabetes diagnosis. We have used different criteria in the two quantitative papers; by the time the children were 4-5 years old (Paper I), we used the new WHO$_{2013}$ criteria. In paper II, we used the WHO$_{1999}$ criteria that were used when the study was performed (2008-2010), and hence reflecting the women that were diagnosed with gestational diabetes and received information and treatment.

Table 3. WHO Diagnostic criteria for gestational diabetes used in papers I and II

Diagnosis criteria	Plasma glucose (mm/l) Fasting	and / or	Plasma glucose (mm/l) 2-hour following a 75g oral glucose load	Women[*] diagnosed with GDM n (%)
WHO 1999 (Paper II)	≥7.0		≥7.8	99 (13.0)
WHO 2013 (Paper I)	5.1 - 6.9		8.5 - 11.0	239 (31.5)

[*]of the total sample of 759 women with information from oral glucose tolerance test, or diagnosed with GDM before the test complete oral glucose tolerance test

Treatment of gestational diabetes

National guidelines at that time used the WHO$_{1999}$ criteria for the diagnosis of gestational diabetes. Women with gestational diabetes were informed about the diagnosis on site, and given oral and written lifestyle advice, and treated according to the study protocol:

(1) Women with fasting glucose ≥ 7.0 mmol/l or 2-hour values ≥ 9.0 mmol/l were categorized as having "moderate/severe gestational diabetes" and were referred to secondary care by the midwife, who called the hospital to reduce delay.

(2) Women with fasting glucose < 7.0 mmol/l and 2-hour glucose values 7.8–8.9 mmol/l were invited back for post-prandial glucose measurements at the Child Health Clinic one week later. If the post-prandial glucose then was ≥ 8 mmol/l, they were referred to specialized care. If the post-prandial glucose was < 8 mmol/l, women were categorized as having "mild gestational diabetes". These women received a letter to their general practitioner, informing about the diagnosis.

Covariates

Education

Women reported their highest level of education in nine categories, which was merged and categorized as: 1) lower level / primary education or less; 2) middle level /completed high school / upper secondary school (10-12 years of education); and 3) higher level / completed university or university / college education.

Socioeconomic position

We used a variable previously defined based on a principal component analysis score of 11 sociodemographic variables (154). The variables contributing most to this score were individual level data about education, occupational class and employment status, and household variables as own or renting tenure and rooms per person in the household. This principal component analysis score was normally distributed (mean=0, median=0.1, SD=1 range: -2.91 to 2.59).

Prepregnant physical activity

was self-reported, collected by a questionnaire that previously had been validated against a physical activity monitor (155).

Prepregnant physical activity

was defined as moderately intensive activity for 30 min on ≥5 days/week, moderately intensive activity for 2.5 h/week over ≥3 days, vigorous-intensity activity for ≥20 min 3 times per week, or activity of both moderate and vigorous intensity (e.g., vigorous activity once per week and moderate activity twice per week) (156). Prepregnant physical activity < 1 year prior to pregnancy was coded as *never*. Prepregnant physical activity > 1 year prior to pregnancy was coded as *regular*.

Mother's dietary pattern

Information on maternal diet was collected in week 28 of pregnancy (and postpartum) with a new food frequency questionnaire, developed to collect information of food intake that reflected the habitual diet also in the ethnic minority groups (157). It included questions

about the frequency of consumption of 65 different food items and drinks, use of fat for food preparation and on bread, meal patterns during the last two weeks', and changes in food habits during the last 14 days. Pictures of food items facilitated categorization for the participants. Using cluster analyses (Ward`s method), four dietary patterns were derived that included 55 of the variables on frequency of food intake (157). We categorized cluster 1 and 3 as the "least healthy", while clusters 2 and 4 were merged into "most healthy".

Gestational age

Gestational week at birth was derived from the first day of the mother's last menstrual period, unless the last menstrual period was unknown/uncertain, or the last menstrual period derived term differed with more than 14 days from the ultrasound term, or the pregnancy was a result of in vitro fertilization (in total: 7% of pregnancies) (158) .

Breastfeeding

Data on breastfeeding were derived from questionnaires used 14 weeks postpartum. Children who were partly or exclusively breastfed at this time point were categorized as "breastfeeding", the rest as "no breastfeeding".

Sample size

Of the 823 pregnancies, there were 784 live-born singleton neonates (Figure 8). At age 4-5 years, we had data on 570 children from three main ethnic groups that were included in Paper I. For Paper II, we included 734 children from all ethnic groups who had data from at least one check-up after birth. Details about each study sample are provided in the papers (Paper I-II).

Figure 8. Flow chart, study I

Healthy pregnant women included at gestational week 14

n = 823

Excluded, n = 39
-Abortions (n=13)
-Stillbirths (n=7)
-Twins (n=11)
-Lost to follow-up /
no birth data (n=8)

Live-born singleton neonates

n = 784

Excluded, n = 158
-Dead (n=2)
-Premature admitted to newborn intensive care unit (n=10)
-Dead (n=2)
-Refused participation or did not show at visit 2 or 3 (n=36)
-Moved abroad (n=23)
-Not possible to retrieve any information on mother or child (n=26)
-No data from Norwegian child health clinic outside Oslo (n=26)
-Not met at 4-years control, but other data (n=29)
-Not measured at 4-years control (n=6)

Study sample of children with growth data at age 4-5 years

n = 626

Study sample Paper I:

Children 4-5 years old
n = 570

3 main ethnic groups:
-Europe (n = 298)
-South Asia (n = 154)
-Middle East / North Africa (n= 118)

Excluded, n = 56
Small ethnic groups:

-South- and Central America (n=8)
-Africa south of Sahara (n=12)
-East Asia (n=36)

Subsample with complete data 14 weeks postpartum

n = 472

Figure 9. Flow chart Paper II

Healthy pregnant women included at gestational week 14

n = 823

Excluded, n = 40
-Abortions (n=13)
-Stillbirths (n=8)
-Twins (n=11)
-Moved prenatally (n=8)

Live-born singleton neonates

n = 783

Excluded, n = 49
-Refused further participation or did not show at the third trimester visit or at the postpartum visit (n=25)
-Medical conditions likely to substantially affect growth (n=19)
-Premature birth, **and** admittid to newborn intensive care unit (n=5)

Study sample Paper II:

Children with growth data at any time point
n = 734

All ethnic origins, 5 groups:
-Europe (n= 344)
-South Asia (n = 180)
-East Asia (n = 38)
-Middle East / North Africa (n = 150)
-Others (n = 22)

Children in Paper I and II are coded differently. I.e. one child who died immediately after birth was coded as "stillbirth" in the flowchart for Paper II, and "dead" in the flow chart for Paper I.

7.1.4 *Statistical methods*

7.1.4.1 Descriptive analyses

Descriptive statistics are given as frequencies, proportions (%), and means with SD.

7.1.4.2 Main analyses

Paper I

Differences in the prevalence of thinness and overweight between the ethnic groups were assessed by Chi-square tests, and differences in children's age were assessed by one-way ANOVA tests.

I performed multinomial logistic regression analysis to identify factors associated with children's risk of overweight and thinness at the age of 4-5 years. Overweight and thin children were compared with normal weight children as reference. The main exposure variable of interest was ethnic origin (Europeans as reference). I considered the following variables as potential covariates; maternal education, parity, age, prepregnant BMI and physical activity, dietary pattern during pregnancy, gestational diabetes, and children's sex and birth weight. I also considered maternal smoking as a covariate, but this variable was left out in the analyses as very few of the ethnic minority women smoked. After conducting univariate analyses, I selected variables into the multivariate model by a *purposeful selection approach* (159). This pragmatic way of building a model makes good clinical sense (160). All variables with a p-value <0.2 in the univariate analyses were included into the multivariate models. I then removed one variable at a time, the variable with the highest p-value first, until all variables reached the level of significance (p<0.05) in at least one of the models. For each step, I checked if the main effect estimates (for ethnicity) changed more than 15 %. If so, the variable was kept in the model to take into account the potential confounding effect on the outcome. Lastly, I tested for interactions between covariates and ethnicity by entering cross-product terms one-by–one into the final model.

Finally, I performed two *sensitivity analyses;*

1) In a subgroup with data on breastfeeding at 14 weeks postpartum (n=472), I included breastfeeding as a mediator into the multinomial logistic regression analysis.

2) As South Asians are known to have a relatively higher ratio of fat to lean mass for any given level of BMI (22, 146), I repeated the multinomial logistic regression analysis using BMI adjustments ($+ 1.12$ kg/m^2) for children of South Asian origin, according to Hudda et. al. (25). Using this method, the adjusted BMI of British children of South Asian relates to body fat in the same way as for British children of White European origin.

Paper II

First, we used the "DAGitty" browser-based tool for creating and analyzing Directed Acyclic Graphs in the model building process (figure 10). In this way, we intended to visualize the causal network that linked gestational diabetes, prepregnant BMI and gestational weight gain with children's growth, and identify true confounders that should be adjusted for to attain more valid estimates. Gestational diabetes was considered our main exposure variable. Mother's prepregnant BMI and gestational weight gain were considered as secondary exposure variables.

Covariates were ethnicity, maternal socioeconomic status, maternal age, parity and gestational week at birth. We did not adjust for potentially mediating factors between maternal metabolic factors and the children's BMI development, such as breastfeeding. In order to obtain estimates that were more precise, we adjusted for gestational age and sex, as these factors are closely related to growth.

Figure 10. Directed Acyclic Graphs

A.
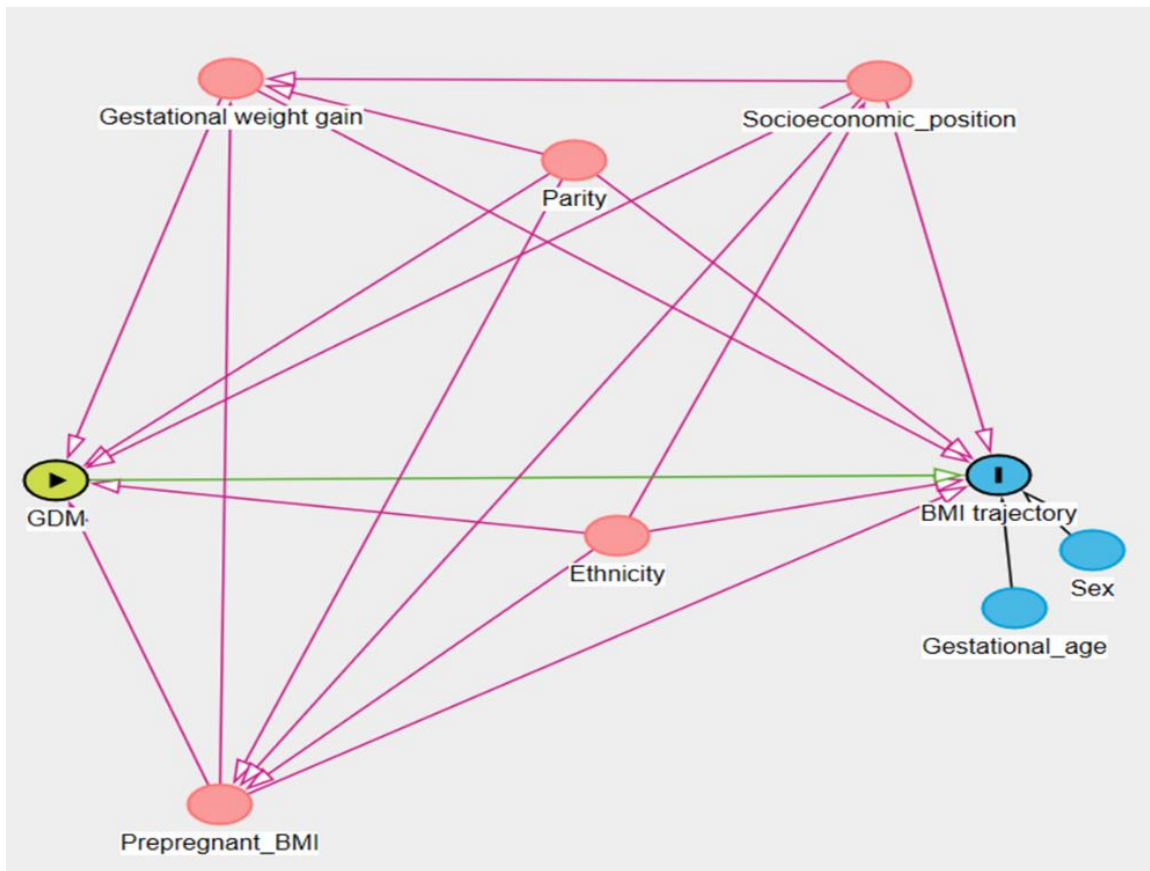
B.
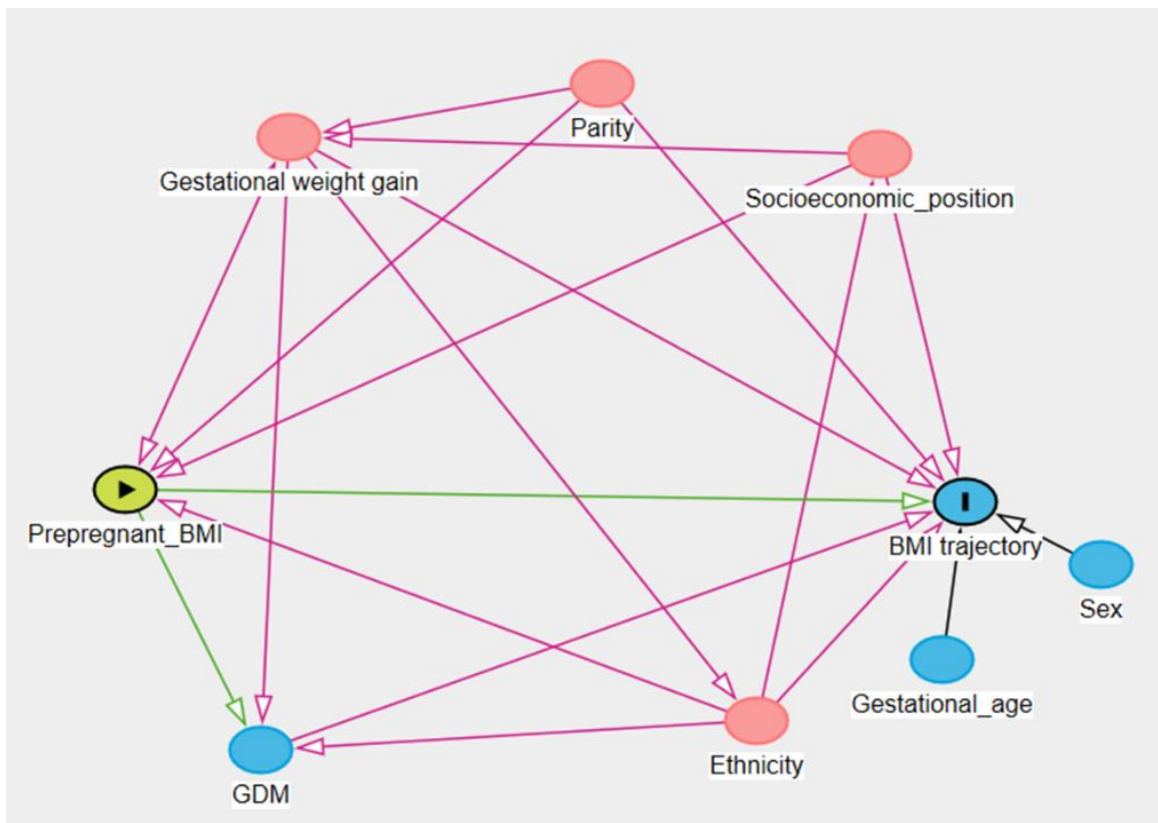
C.
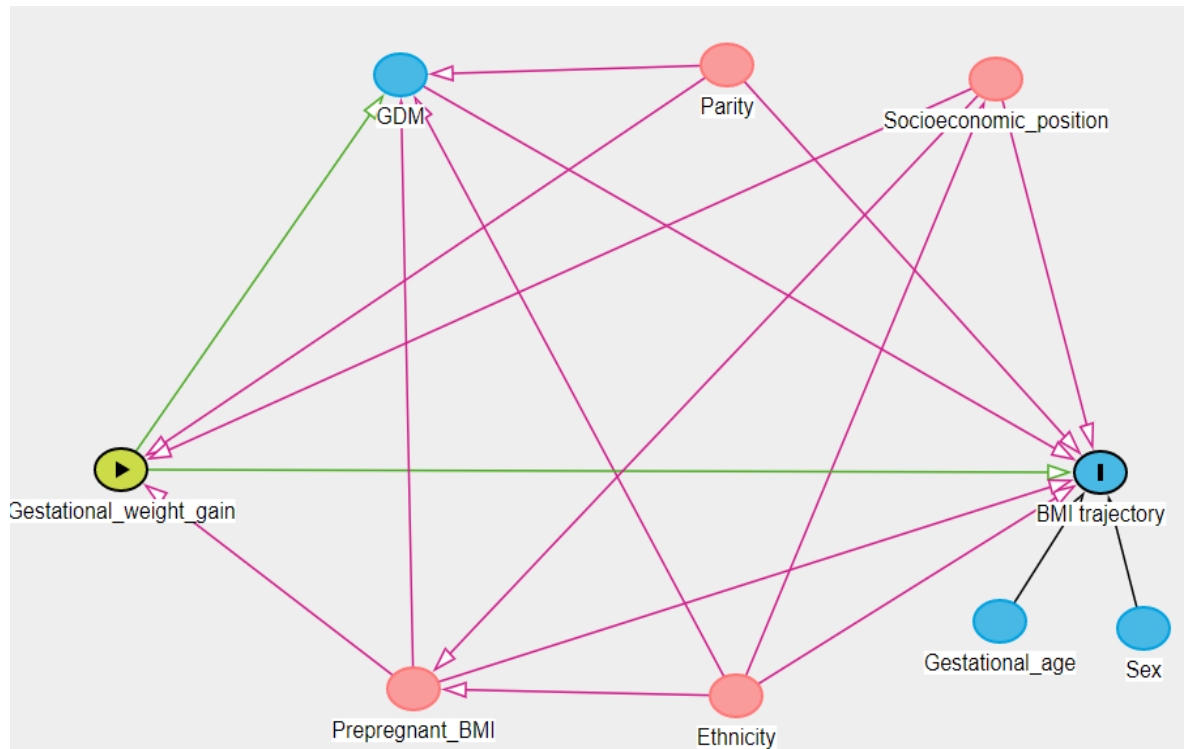
Directed Acyclic Graph of confounders between maternal factors and children's postnatal BMI trajectories by the main exposures; A. Gestational diabetes mellitus (GDM) B. Prepregnant BMI, C. Gestational weight gain

- Green dot; Main exposure
- Red dots; Confounders
- Blue dots; Outcome and ancestors of outcome
- Green arrows; Causal pathways
- Red arrows; Confounding pathways

We performed two types of main analyses; (1) General linear models (linear regression), and (2) linear mixed models

1) I performed *general linear model*s analyses to explore the associations between the predefined exposures, based on the literature and previous studies from the STORK-Groruddalen cohort, and children's BMI at birth. In model 1, I adjusted for gestational diabetes (by the WHO_{1999} criteria) as the main exposure, and covariates related to maternal characteristics; ethnicity, age, parity, and socioeconomic position. In model 2, I

added gestational week at birth and children's sex. In the final model 3, I added the secondary exposure variables; mother's prepregnant BMI and gestational weight gain.

2) To explore associations between the predefined exposures and children's BMI and BMI-trajectories from birth to 4-5 years of age, my main supervisor, Line Sletner, first built a model for *linear mixed models* analyses, which was quality checked by Professor Magne Thoresen, a statistician. This method can handle the strongly correlated, repeated measurements for each child, and overcome the problems of missing outcome data (160). Unlike many other methods for analyzing longitudinal data, this method does not require complete data, and it allowed us to include participants with missing growth data at certain time points.

We were primarily interested in the independent association between gestational diabetes and offspring BMI trajectories, but also the direct and independent effects of maternal prepregnant BMI, and gestational weight gain, i.e. effects not mediated trough gestational diabetes. The natural BMI growth increases rapidly from birth to 6-8 months of age, then declines until 4-5 years of age, followed by a gradual increase. This represents a complex trajectory for statistical modelling. As more than 95% of all children in our cohort had their highest BMI value at the visit at 6 months of age, we stratified the analyses; 1) from birth to 6 months and 2) from 6 months to 4-5 years. All available BMI measurements from any time point were entered into the models. We built basic random intercept, random slope models, and the final basic models included children's age (in months), entered both as fixed effects (age and age*age), and as a random effect (age). Maternal and children's factors were then included, both as fixed factors and as interaction terms with children's age (representing BMI growth). Restricted maximum likelihood was used to estimate the different parameters. Models (each step of the model building process, to avoid overfitting) and covariance structures were compared using Akaike and Bayesian information criteria. An unstructured covariance structure was chosen. In the "univariate models", explanatory variables were added one by one to the basic model, including the interaction term with age. In the first adjusted model (model 1), we entered gestational diabetes, ethnicity, maternal age, parity, and socioeconomic position. In model 2, we further added gestational age and

sex. Finally, in the full model 3, prepregnant BMI and maternal gestational weight gain were also added.

In a supplementary model for growth between 6 months and 4-5 years of age, we also adjusted for BMI-growth between 0-6 months.

Of the 734 children, the majority (671 mother/child pairs) had data on all three main exposure variables as well as all covariates. We have therefore not tested for potential selection bias.

For the quantitative analyses, we used SPSS version 22 (IBM SPSS statistics, NY, USA).

7.2 Qualitative Study; Paper III

7.2.1 Design and choice of method

In this study, we wanted to obtain new knowledge on how to improve the initial communication between parents and health professionals about their preschool child being overweight. More specifically, we wanted to explore parents' experiences of the first conversation when a health professional identifies their child as overweight. At this age, most parents do not recognize their child as overweight (29), and health professionals need to cooperate closely with parents. Therefore, this first conversation might be crucial.

We decided that a qualitative method was most suitable to answer our research questions (161). Qualitative methods involve systematic collection, processing, and interpretation from conversations, observations or written text, and are used to gain understanding of a phenomenon as experienced from those involved (162). We chose individual semi-structured interviews as the data collection method. This method is particularly suitable when the theme is sensitive (163), and we expected the participants to find themselves in a vulnerable position talking about a difficult conversation. We considered focus group interviews to be less suitable, as participants lived in small communities where sensitive information could potentially be spread. Further, we chose this method for practical reasons,

as the Child Health Clinics were situated geographically far apart, and we expected that it might be hard to recruit informants.

In quantitative studies, power calculation determines how large the sample size (n) has to be to make significant estimates. However, for qualitative interview studies there are no similar assessments of sample size. Malterud et. al. have introduced the term "information power" as a concept for sampling strategy for qualitative research (164). Sufficient information power depends on the aim of the study, sample specificity, established theory, and analysis strategy. The more information the sample holds relevant for the present study, the lower number of participants is needed. We stopped data collection at a time point when we decided that the information power was sufficient.

There are different ways of analysing qualitative data, and the research questions may guide the choice of methods (162). After exploring various methods for analyses, we decided on Malterud' s method of *Systematic text condensation* (165). This method was well suited for our study, and I especially found the use of text condensates helpful and understandable. I will describe the specific procedures below.

7.2.2 *Recruitment and participants*

We wanted to recruit parents having a preschool child who had been classified as overweight or obese, defined by the International Obesity Task Force (151), at a recent routine check-up at their local Child Health Clinic.

I visited eight Child Health Clinics in Oppland County, asking the staff to help me recruit informants for my study. Oppland is a mostly rural part of southeaster Norway, and as I live there myself, recruitment from this area was convenient. Public health nurses and general practitioners employed at Child Health Clinics in six different municipalities agreed to participate. These Child Health Clinics were located within a 100 km distance apart from each other. The staff informed parents about the study and handed out written invitations. It was somewhat hard to recruit parents, but after a six months' period (September 2011-March 2012), mothers of nine children and both parents of one child had agreed to participate.

The families were ethnic Norwegian and had at least one grandparent living in the same municipality (Table 4). None of the authors knew any of the families in forehand. Children were between 2.5 - 5.5 years old. Seven were classified as overweight, and three as obese. All children were otherwise healthy and attended kindergartens. None of the children had yet been referred to a paediatric ward. All parents considered themselves and / or their partner as overweight.

Parents were between 22-46 years old, and only one parent was single. In the published paper, there is a misprint in table 2 (Family characteristics) stating that parents were 22-26 years old. Unfortunately, we did not spot this error until it was too late to correct the printed article. The original and correct table is shown below (Table 4).

Table 4 Paper III. Family characteristics, corrected table

Parents' characteristics	n=11
Age range (years)	22-46
Relationship status	
Single	1
Married / living with partner	10
Education level	
Higher (more than 12 years)	5
Lower (12 years or less)	6
Current employment status	
Working	8
Reported sick	3
One or both parents considered themselves as overweight	11

Children's characteristics	n=10
Age range (years)	2.5-5.5
Sex	
Boys	5
Girls	5
Weight status*	
Normal- or underweight	0
Overweight	7
Obesity	3

*According to IOTF definitions

7.2.3   *Preparing and data collection*

Before conducting the interviews, all authors cooperated to develop an interview guide with open-ended questions, based on a systematic literature search in the PubMed database. Further, the authors' broad experiences in the field, as Public health nurse (KG), doctors at Child Health Clinic (PL and IT), Paediatrician (PL), and general practitioner (IT) came well in hand. We also made use of the communication tool of Motivational Interviewing (166) when planning interviews. Motivational Interviewing is based on a person's feelings, taught and believes, and explores resistance, ambivalence, and motivation for change; i.e. just what we were interested in elaborating during the interviews.

In order to achieve a better quality of the study, I initially conducted a pilot interview with a colleague who had a 5-year old child. This gave me the opportunity to rehearse the interviewing, and my colleague gave valuable feedback on the interview guide, as well as my interviewing skills.

The main topics included in the final interview guide were;

1) Parents' experiences at the Child Health Clinics
2) Parents' perceptions of their child's weight and their own weight
3) Parents' experiences concerning lifestyle and their child's weight within the extended family and with kindergartens.

During March–September 2012, I conducted semi-structured, in-depth interviews at the families' local Child Health Clinic, less than four weeks after the initial consultation there. The interviews lasted 45 to 75 minutes. I recorded the interviews digitally and transcribed them verbatim shortly after the interview. In my experience, all parents talked freely, and they did not seem to be offended by any of the topics of the interview guide. Therefore, the original interview guide was kept throughout the period of data collection.

7.2.4   *Data analysis*

All authors cooperated in analysing the qualitative data. According to the method of systematic text condensation, there are four main phases of analyses (161, 165), as described below and in table 5.

1. To get a <u>general impression</u> and to look for <u>preliminary themes</u>, all authors listened to all soundtracks and read all transcripts. After discussions, we agreed on preliminary themes, which were not based on the interview guide, but evolved from the collected data:
   (a) Concerns about the child catching some of the conversation at the Child Health Clinic;
   (b) Consequences of overweight for the child;
   (c) Parents' perceptions of the child;
   (d) Health professionals could be helpful or rude;
   (e) Parents' own weight experiences;
   (f) Experiences with grandparents and kindergartens

2. I imported the transcripts into the software package ATLAS.ti (atlasti.com), which helps to organize, but not analyse the data. I identified <u>meaning units,</u> which are text fragments containing some information about the research question. Example of a meaning unit; *"To celebrate your birthday in kindergarten – that is an experience, an activity. It is nothing you should eat! And then you might get some help"*. This quote may tell us that parents appreciated the kindergarten setting a good example by celebrating birthdays by arranging activities, rather than serving cakes etc. Further, these meaning units were <u>coded,</u> by identifying, classifying and sorting them into the previously negotiated themes. I coded the previous example of a meaning unit (f) into the theme; Experiences with grandparents and kindergartens. Particularly illustrating codes were marked as <u>golden quotes</u> (161).

3. During text <u>condensation,</u> I selected meaningful units within the coded groups and put the quotes into an artificial quotation, which was written in first-person format. Through this systematic abstraction, I intended to <u>summarize</u> the contents of the quotes into a condensate. As some meaningful units were not possible to fit into a condensate, they were at first put aside, and sometimes a new theme emerged. Example of an artificial quote; *"We have a good relationship with the kindergarten, where they know our child well and thereby have a unique competence. They spend much time being active outside. Although they have economic limitations, they eat healthy food. When celebrating birthdays, they focus on the experience, not on food"*

4. In the last step, all the authors together <u>synthesized</u> the contents of the condensates, and developed <u>description and concepts</u>. These were summarized into subcategories and then into main themes. We put subgroups forward and selected a relevant golden quote to illustrate each subgroup. This final text was written in third person, reminding the reader and ourselves at we are researchers and responsible for our interpretations.

Table 5. Illustration of systematic text condensation for the preliminary theme "Experiences with grandparents and kindergartens

Phase 1	Phase 2	Phase 3
A. *General impression* B. Identification of *preliminary themes*	A. Identification of *meaning units / quotes / text fragments* B. *Coding of meaning units* (classifying, sorting etc.) (first person)	Construction of a *condensate* ("artificial quote") summarizing several meaning units (first person)
Vulnerable parents & children	*"To celebrate your birthday in kindergarten – that is an experience, an activity It is nothing you should eat! And then you might get some help".* *"We have a good relationship with the kindergarten, and feel that we can bring up weight problems there. They have a unique competence, and give us support. They focus healthy food and spend a lot of time outside"*	*We have a good relation with the kindergarten where they know our child well and thereby have a unique competence They spend much time being active outside Although they have economic limitations, they eat healthy food. When celebrating birthdays, they focus on the experience, not on food*
Preliminary theme: Experiences with grandparents and kindergartens		

Phase 4
Synthetization of contents of condensates into   A. *Main themes* &   B. *Subcathegories*   Decide on *"golden quotes"*   (third person)
All parents trusted the staff in the kindergartens and felt that they had unique understanding of the child and general competence about childrens health….
"Golden quote":   *"To celebrate your birthday in kindergarten – that is an experience, an activity It is nothing you should eat! And then you might get some help".*
Main theme : Motivational factors   *Subcathegory* : Relationship with significant others

8   Results

8.1   Paper I

<u>Ethnic differences in overweight and thinness</u>

We found strikingly different patterns of thinness and overweight among children of different ethnic groups at age 4-5 years. Using the International Obesity Task Force cut-off values for weight classification (167), the overall prevalence of overweight and obesity was 12.6 %. Children of Middle East / North African origin had a higher prevalence of overweight (22.0%) compared to European children (12.8%), and in adjusted logistic regression analyses almost the double risk (OR 1.98; 95% CI: 1.08-3.63). The prevalence of overweight was lower in children of South Asian origin (5.2%). Children with South Asian background had higher prevalence of thinness (26.0%) compared to ethnic Europeans (10.4%), and the double risk (OR 2.20; 95%CI: 1.25-3.87) in adjusted models.

However, taking into account the relatively increased adiposity in South Asian children by applying BMI adjustments (25), their prevalence of overweight increased substantially, and their prevalence of thinness was reduced (Table 6).

Table 6. Mean age and prevalence of thinness, overweight and obesity at age 4-5 years.

	Ethnic origin, n (%). Main ethnic groups					
	Total	Europe	Middle East / North Africa	South Asia unadjusted BMI	p-value	South Asia adjusted BMI[***]
	n=570	n=298	n=118	n=154		n=154
Age in years at Child Health Clinic control, mean (SD)					p=0.60*	
	4.38 (0.28)	4.37 (0.28)	4.39 (0.31)	4.40 (0.26)		
BMI category, n (%)					p<0.001**	
Thinness	86 (15.1)	31 (10.4)	15 (12.7)	40 (26.0)		6 (3.9)
Normal weight	412 (72.3)	229 (76.8)	77 (65.3)	106 (68.8)		126 (81.8)
Overweight including obesity	72 (12.6)	38 (12.8)	26 (22.0)	8 (5.2)		22 (14.3)

*One-way ANOVA analysis

** Chi-square test

*** positive BMI adjustments of + 1.12 kg/m^2 to account for greater relative adiposity in tjis population

Associations with maternal factors

Children's birthweight and maternal prepregnant overweight were both strongly, positively associated with overweight at age 4-5 years, and inversely associated with thinness. Lower maternal age was associated with overweight only. Gestational diabetes was not associated with children's weight status at age 4-5 years. No significant interactions between covariates and ethnicity were observed.

8.2 Paper II

Sample

Of the 734 mothers, 14.9 % had prepregnant obesity, 12.5 % had gestational diabetes, and 34.2 % had weight gain in the highest tertile. In total, 50 children were exposed to two of the three maternal factors; while only three children were exposed to all three factors (Figure 11, Venn diagram). At 14 weeks postpartum, more than 85 % of children were breastfed, most of them (69%) were exclusively breastfed.

Figure 11. Venn diagram paper II
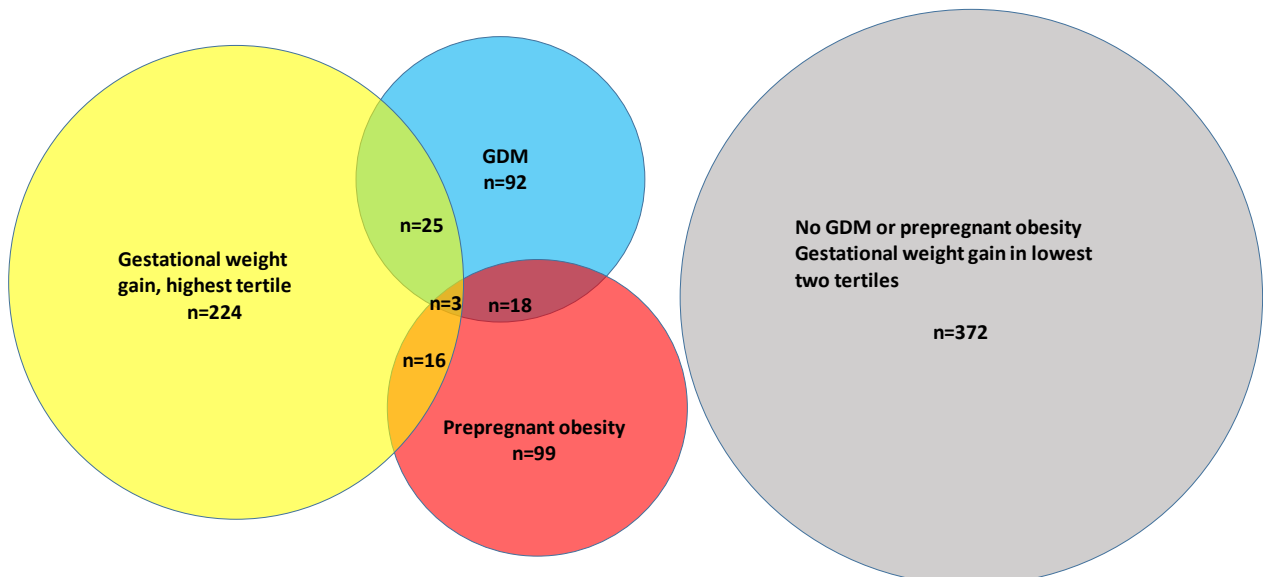
Associations between maternal factors and children's BMI and BMI growth

At birth, BMI of children of mothers diagnosed with, and treated for gestational diabetes, did not differ significantly from BMI of children not exposed to gestational diabetes. However, children exposed to gestational diabetes had a slower BMI growth (B=-0.106 (BMI units / month) SD; 95%CI: -0.17, -0.04) during the first 6 months, and a faster BMI growth from 6 months to 4-5 years of age (B=0.009 (BMI units / month) SD; 95%CI: 0.002-0.02). At age 4-5 years, BMI was similar in both groups.

In contrast, maternal prepregnant obesity was associated with a higher BMI at birth, and a persistently higher BMI up to 4-5 years. Maternal gestational weight gain (the highest tertile) was associated with a faster BMI growth from 6 months to 4-5 years (B=0.007 (BMI units / month) SD; 95%CI: 0.001-0.01).

Associations between ethnicity and children's BMI and BMI growth

The relations between maternal gestational diabetes, prepregnant obesity and maternal gestational weight gain and children's BMI trajectories were similar in all ethnic groups. However, we found substantial ethnic differences in the BMI trajectories. Compared with children with European origin, ethnic minority children had a lower birth weight. During the first 6 months, children with Middle Eastern and North African origin had a faster BMI growth than children with European origin (B=0.069 (BMI units / month) SD; 95% CI: 0.004-0.13) (Paper II, figure 2d). Compared with European children, children with East- and South Asian origin had a persistently lower BMI during the whole period from birth to age 4-5 years. However, the BMI growth in children with East- and South Asian origin did not differ from the BMI growth of children with European origin. Thus, the growth trajectories of children with Asian and European origin were parallel.

8.3   Paper III

When listening to the interviews about parents' experiences when a health professional identified their preschool child as overweigh, the overshadowing impression was how vulnerable the parents presented themselves and their toddlers in this situation. Two principal themes arose from the interviews; (a) Parents feelings and concerns when being told that their child was overweight; (b) and motivational factors that could help them change their lifestyle.

The parents' feelings and concerns

The parents told that their child's possible overweight was a thoroughly difficult issue to discuss. Some felt that the health professionals had been offensive and prejudiced in this situation. Most parents presented themselves and their children as being easily hurt. They were afraid that the toddler would develop low self-esteem and even eating disorders if talking about and dealing with overweight. Therefore, they would prefer to have this conversation without the child being present. Only one parent expressed concerns about by future health consequences like cardiovascular diseases if their children were affected by persistent overweight.

Motivational factors

Although most parents did not consider their child as overweight, they would like health professionals to express their concerns and give support in a sensitive manner. They considered growth charts as useful. Some told that they felt relieved when explained that their child did not have to lose weight, but could grow into a normal weight group. Parents expressed trust in kindergartens, but not all grandparents were considered as helpful. Most parents described themselves as overweight. Surprisingly to us, all parents talked readily about their own weight experiences. Being overweight represented both a barrier to, and motivation for dealing with their toddler's overweight. Talking about the short-term consequences of overweight, such as low self-esteem, poor physical performance, and being bullied, may have given parents motivation for dealing with their child's overweight. However, many parents had experienced that losing weight was very difficult, or even impossible.

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9 Discussion of main findings

9.1 Quantitative papers I and II

9.1.1 Ethnic differences in overweight and thinness

9.1.1.1 Overweight and socioeconomic position

The overall prevalence of overweight at age 4-5 years (12.6%) in our cohort was comparable to another Norwegian study (11.8%) of predominantly ethnic Norwegian children (168). We are not aware of any other studies assessing ethnic differences in overweight or thinness in Norway. However, in line with our findings, multiethnic studies from The Netherlands and the United Kingdoms (UK) also report higher prevalence of overweight in children with origin in the Middle East and Africa compared with their European counterparts (19, 169, 170).

When studying childhood obesity, it may be difficult to disentangle associations with ethnic minority status from associations with low socioeconomic status. A recent study from the UK found ethnic group differences in mean BMI z-scores at age 4-5 years that were similar to our findings (170). These ethnic differences could not be explained by area-level deprivation. On the other hand, a recent multiethnic cohort study (n=3714) from The Netherlands found that the BMI growth patterns leading to overweight at age 5-6 years differed according to maternal socioeconomic position (defined by education level), but not according to ethnicity (171). In this study, children with overweight and low socioeconomic position had a lower BMI during the 2 first years of life, followed by an earlier adiposity rebound, compared with children with overweight and high socioeconomic position. As all children in this study were overweight, the results are not directly comparable to ours. It is interesting that the striking ethnic differences both in the prevalence of overweight and in the growth trajectories in our cohort could not be explained by socioeconomic position. This may at least partly be related to limited power to show associations with socioeconomic position in addition to the ethnic differences. Further, the study from The Netherlands indicated that some of the differences in growth patterns may be explained by a high prevalence of smoking (35%) among mothers with low socioeconomic position. We did not adjust for smoking as a confounder in our cohort, as few mothers, and very few from the ethnic minorities, smoked. In our cohort, the accelerated growth of children with origin in

the Middle East/North Africa (who had the highest risk of overweight at age 4-5 year) appeared during the period from birth to 6 months, compared with children of European origin. This pattern can perhaps be related to ethnicity, as children with origin in the Middle East/North Africa had a lower BMI at birth. Ethnic differences in feeding patterns may also contribute to some of the early accelerated growth (172).

In a multiethnic cohort study from the UK, 5-year old children with Pakistani origin had lower odds of obesity compared with White British children, while Black African children were more likely to be overweight (13). Analyses were adjusted for socioeconomic variables and maternal BMI. Findings from this cohort were compared with a similar cohort from the USA showing diverging results. The US cohort revealed no ethnic disparities in overweight or obesity after adjusting for socioeconomic disadvantage. These differences may illustrate variations from country to country in the total prevalence of obesity, as well as the absolute and relative inequalities in the socioeconomic position and health measures within and between populations. Further, the association between ethnic minority status and low socioeconomic position may differ from country to country based on contextual differences in integration policies, and the ethnic composition differs between countries.

In our studies, we merged countries into larger ethnic groups, which may have concealed variations within these broader groups. A study of a multiethnic population of children living in the UK, showed that 6 years old boys with origin from Bangladesh had higher mean BMI z-scores, compared with their counterparts with origin in India or Pakistan (170). Suggested explanations for this difference were lower socioeconomic position and low dietary acculturation in Bangladeshi migrants.

Reports on the prevalence of childhood obesity in the regions where our ethnic minority groups originate generally show more overweight and obesity in children living in the Middle East and North Africa, compared with children living in the South Asian region (173, 174). Observed differences in the prevalence of childhood overweight and obesity between the country of origin and the country of residence may be due to families changing their life style after emigration (170). However, direct comparisons can be difficult as there may be heterogeneity within these rather large ethnic groups, and definitions of childhood obesity may vary between studies.

9.1.1.2 Ethnic differences in thinness and in body composition

We showed that children with South Asian origin in our cohort had the double risk of being classified as thin, compared with children with European origin. A cross-sectional, population-based study of a multiethnic sample of 2 -15 years old children from England, found that the overall prevalence of thinness was considerably lower (5.7 %) compared with our cohort (15.1 %) (175). The difference in the prevalence between these two studies may be due to differences in the age groups studied, as well as different proportions of ethnic groups in the cohorts (80% White and 14 % Asian in the cohort from England). However, in line with our findings, this study showed that children with Asian origin had a more than 3-fold risk of being classified as thin, compared with children with White European background. The strongest predictor of thinness was parental weight status. The authors concluded, in line with our conclusions, that many cases of thinness are likely to have a genetic origin, and are likely to represent the lower end of a healthy distribution of BMI values.

The cut-off values for thinness, defined by the International Obesity Task Force, are based on calculations of BMI, which underestimate the total amount of body fat in children with South Asian origin (25, 176). A recent study of a multiethnic historical cohort in The Netherlands, showed large differences in the prevalence of thinness and overweight in South Asian children, after applying the specific BMI cut-off values for South Asians (169, 177). When applying the universal BMI reference (167), South Asian children had the highest prevalence of thinness (16.2%) at age 3-4 years. However, when applying the specific BMI cut-offs for South Asians, the estimated prevalence of thinness in children with South Asian origin was comparable to that in the other ethnic groups. Of importance, when using the ethnicity specific BMI cut-off values, children with South Asian origin had the highest prevalence of overweight (26.7%) of all ethnic groups studied (Dutch, Turkish, Moroccan, and South Asian).

After applying the positive BMI adjustments to children of South Asian origin in our cohort, the sensitivity analysis showed that South Asian children had a lower risk of thinness compared with children with European origin. However, the factors positively associated

with overweight (Middle East/North African origin, maternal age, maternal prepregnant BMI, female sex, and birth weight) remained significant even after applying the BMI adjustments for the children with South Asian origin. Thus, prevalence estimates after applying the BMI adjustments for the South Asian population did not seem to change as much in our study as in the study from The Netherlands. One explanation for this might be that the method applied to adjust BMI in the two studies were different. We added 1.12 kg/m^2 to the BMI values of the South Asian children, as suggested by Hudda et.al. (25), while the other study used the calculation of BMI centile curves passing through the cut-off points of 15 (thinness), 23 kg/m^2 (overweight), and BMI 25 kg/m^2 (obesity) at age 18 years, derived from adult Asian Indian cut-off values, as suggested by de Wilde et. al. (143, 177) (Discussion, chapter 10.1.1.2).

9.1.2   Sex

In our cohort, girls were more affected by overweight than boys were at 4-5 years of age. Some multiethnic cohort studies have reported results in line with ours (171), while others report no differences between boys and girls in preschool age (170, 178). In our study, the mean BMI did not differ between boy and girls at birth, but from birth to age 6 months, the boys had faster BMI growth, compared with girls. From age 6 months to 4-5 years, the girls had the fastest BMI growth. We were not able to conduct interaction analyses with sex, due to lack of statistical power. Such analyses would have given additional information, as interestingly, one study reported that maternal hyperglycaemia in pregnancy had an effect on offspring adiposity at 7 years in girls, but not in boys (179).

9.1.3   Relations with maternal factors

9.1.3.1   Gestational diabetes

Children's age

The large Hyperglycaemia and Adverse Pregnancy Outcomes study, blinded for glucose levels in pregnancy, showed a strong, linear association of maternal glucose levels with increased birth weight in the offspring (180). Contrary to our results, most observational studies have shown that gestational diabetes is associated with children's higher weight and adiposity at birth (74, 180, 181). These different findings may be related to the fact that

mothers diagnosed with gestational diabetes in our study were informed and referred for treatment (148). Further, many women in our cohort had a relatively mild gestational diabetes, as our sample represents a generally healthy population of pregnant women that was universally screened for gestational diabetes. This is supported by two other observational studies, which reported similar birth weight and infant adiposity measures in children of mothers with well-treated gestational diabetes, compared with children of mothers without gestational diabetes (182, 183).

Follow-up studies of the children in the Hyperglycaemia and Adverse Pregnancy Outcomes study did not find effects of gestational diabetes on overweight and adiposity at age 2 years, but effects were emerging at 11 years age (77, 184). A meta-analysis of observational studies showed similar results, but the quality of evidence was considered to be low, as most studies did not adjust for other maternal factors, and different definitions of gestational diabetes were used (73). At age 4-5 years, we did not find associations between gestational diabetes and children's BMI in either of our studies. Contrary to our findings, a recent study from the US showed that children exposed to gestational diabetes had a higher risk of obesity at age 4 years, compared with unexposed children (185). Explanations for this discrepancy may be that the latter study did not include mothers with Asian origin, which would probably have affected the prevalence of gestational diabetes as well as the children's BMI. Furthermore, women were not screened for gestational diabetes, and the prevalence of gestational diabetes was relatively low (5.4%). Thus, women with gestational diabetes were probably affected by more severe gestational diabetes that might have a greater impact on children's BMI at 4 years of age.

"Catch-down" growth

Although the mean BMI of children exposed to gestational diabetes in our studies did not differ at birth or at age 4-5 years, we show that they had a "catch-down" BMI growth from birth to 6 months, followed by a faster BMI growth up to 4-5 years, compared with children not exposed to gestational diabetes. Earlier studies have also observed an early "catch-down" growth during the first 6 months of life in children exposed to gestational diabetes (181, 186, 187). However, in these studies, the mean birthweight of children exposed to gestational diabetes was higher than in the non-exposed children. Further, these studies also

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included women with previously or recently diagnosed type 2 diabetes (i.e. more severe gestational diabetes). A study comparable to ours, did not find an early "catch-down" BMI growth in children exposed to pre-existent diabetes or gestational diabetes (188), but this study lacked measuring-points between birth and 8 months of age, and thus, a possible "catch-down" growth could have been missed. Further, this study also included women with type 2 diabetes, and did not adjust for gestational weight gain.

Treatment of gestational diabetes

Randomized control trials have shown that treatment of gestational diabetes reduces the risk of macrosomia at birth, as well as perinatal and neonatal complications (189, 190). A randomized control trial on treatment of mild gestational diabetes showed a reduced risk of caesarean delivery, macrosomia, and shoulder dystocia at birth in offspring in the treated group, compared with controls (191). A follow-up study of these children at age 5-10 years showed no reduction in childhood obesity or metabolic dysfunction in the offspring of treated women, compared with the offspring of mothers who were not treated (192). As most researchers would consider it unethical to perform a new randomized control trial on the effect of treatment of gestational diabetes, it is interesting that we did not find any differences in BMI at age 4-5 years between children exposed and not exposed to gestational diabetes that was treated. A recent study of BMI growth trajectories between age 2 and 6, years found that gestational diabetes requiring glucose-lowering medication was associated with high and increasing BMI trajectory in the children, while the effect of gestational diabetes not requiring medication was small (193). These results indicate that the severity of gestational diabetes may be of importance to BMI growth during early childhood.

9.1.3.2 Prepregnant obesity and gestational weight gain

We found that maternal prepregnant obesity was strongly associated with offspring overweight at age 4-5 years (Paper I), and with higher BMI at all measuring points from birth to age 4-5 years (Paper II). Other studies of multiethnic cohorts have also shown that maternal prepregnant obesity is associated with obesity in their children (19, 24). A recent meta-analysis showed that maternal prepregnant obesity and excess gestational weight gain

were independently associated with childhood overweight at age 2-18 years, with the strongest effects in later ages, and with prepregnant obesity as the most important factor (194). Although this study did not consider gestational diabetes as a confounder, the findings are in line with ours.

Another study assessing children's growth trajectories and maternal obesity, diabetes during pregnancy, gestational weight gain and breastfeeding, also found that maternal obesity had the strongest association with high and increasing BMI from age 2 years to age 6 years (79). The independent effect of excess gestational weight gain was modest and was mainly mediated through birthweight and BMI at age 2 years. Hu et. al. found that excess gestational weight gain and prepregnant obesity were associated with the children's growth trajectories, characterized by an average birth weight, followed by rapid weight gain during first year of life, and a persistently high BMI until age 4 years (185). As this study had no measurements between birth and age 1 year, it could not tell whether the accelerated growth associated with gestational weight gain started at the same age as we show (6 months).

The concordance between maternal and offspring obesity can stem from genetics, shared environment and lifestyle, as well as from intrauterine exposures (62). In a population-based cohort study, Ludwig et. al. examined how differences in gestational weight gain that occurred during two or more pregnancies for each woman, predicted her children's BMI and the odds ratio for being overweight or obese at 12 years age (195). As this study used a within-family study design, confounding by genetic and environmental factors was probably reduced. In line with our finding, this study (adjusting for maternal prepregnant BMI and birthweight) showed that maternal gestational weight gain was positively associated with the children's BMI and an increased risk of overweight/obesity. However, this study differs from ours, as mothers with diabetes during pregnancy were excluded, and 75% of participants were of White European background.

Knowledge about the adverse effects of prepregnant obesity and excess gestational weight gain is of importance for clinical practice. However, midwifes working at 45 Child Health Clinics in Norway reported that discussing gestational weight gain with pregnant women was considered to be difficult, and only 3 % reported discussing the mothers' weight retention at the postpartum control (196).

9.2   Paper III

9.2.1   Parents' feelings and concerns

Parents in our study experienced negative feelings such as sadness, anger and guilt when health professionals raised concerns about their child being overweight. In line with other studies, some parents felt stigmatized and that their parenting skills were being questioned (197).

Parents in our study were also seriously concerned about their child being vulnerable. They feared that their child would develop low self-esteem and eating disorders if overweight was brought up as an issue. Research supports an association between childhood obesity and increased risk of later eating disorders, especially if the child is teased by peers or family (198). However, it is more likely that body dissatisfaction, rather than the focus on healthy eating habits, is the primary cause of eating disorders (198). Research supports the concept that overweight children have lower self-esteem (199) than do children of normal weight. Steinsbekk et. al. reported that children with obesity, seeking treatment, had impaired parent-reported quality of life, which was attributed to the children's elevated levels of psychopathology (85).

In order to protect their child, parents preferred to discuss overweight without the child being present. Similar findings were reported from a larger qualitative study asking parents of children aged 2-5 years how physicians should approach diet and weight-related advice, if parents or the doctor had concerns about the child's weight (200). This study differs from ours, as the index children were not necessarily affected by overweight, and parents may not have experienced the vulnerable situation discussing weight concerns. However, being asked a hypothetical question, parents still did not want their preschool child to be present during discussions about their child's weight.

9.2.2   Motivational factors

All parents thought that the conversation at the Child Health Clinic when their child was identified as overweight had been difficult, but still they expected the health professionals to

inform parents if concerned. A recent qualitative study argues that the initial communication between the health professionals and the parents about their child's BMI is crucial (111). As in our study, many parents consider their child as having a normal weight, and parents would like a personal and tailored approach regarding this initial communication in order to show an interest in attending an obesity management programme. Our informants looked upon growth charts as objective and useful tools. Other studies also suggest that visual information as pictures of portion sizes and growth charts may be helpful (112, 200). Consistent with the works of some others (201-203), parents in our study wanted health professionals to address childhood obesity in a sensitive and respectful manner, avoid blame and negative words like "fat" and "big belly".

All children in our study had at least one parent with overweight. Parenting an overweight child and being overweight oneself may be felt as a double stigma. One study showed that daily experiences with obesity stigma were associated with decreased motivation to diet, exercise, and lose weight (110). In a study from Australia, public health nurses found it especially difficult to raise children's weight issues if parents were overweight (204). Our findings suggest the contrary, that parents consider their own weight experiences to be relevant for health professionals to explore, and that parents freely shared their own experiences on being overweight and dieting. These experiences were presented as a barrier to, but also a motivation for dealing with their child's overweight.

Some parents with overweight themselves had experienced that losing weight was almost impossible, and that the health care system was not helpful. However, during the interview, some parents realized that they did not want their child to experience the negative consequences of overweight in adolescence and as adults. Parents who consider themselves overweight may be more ready to make behaviour changes to help their child lose weight, than parents who have a normal weight (205). Parents told that they wanted to be asked for their opinions and ideas. Our findings suggest that parents, despite of feeling vulnerable and worried about their child's feelings, may accept the communication method of Motivational interviewing (166). A randomized controlled trial showed that a two-year intervention of Motivational Interviewing resulted in a significant reduction in BMI percentiles for children aged 2-8 years with overweight (206).

Systematic reviews on the prevention of childhood obesity show that school-based interventions with combined diet and physical components, as well as a home-element, had the greatest effectiveness (116, 122, 123). This would probably be a fruitful strategy for the families in our study, as parents trusted and appreciated support from kindergartens that promoted healthy eating habits and physical activity. In a study from The Netherlands, parents considered physical activity and nutrition of children aged 10–12 years a responsibility that should be shared with schools (207).

Stewart et al. reported that the extended family often undermines and fails to support lifestyle changes initiated by parents and the health care system (208). This is in line with some of our informants telling that grandparents would give their grandchildren sugar-sweetened beverages, ice cream, and perhaps an extra dinner every day. However, an anonymous online questionnaire study showed that grandparents were more willing to receive information on preschool children's risk of overweight than parents were (203). As for most children in our study, grandparents can play an important role in the feeding of preschool children. Thus, health professionals may consider involving grandparents in a positive manner when appropriate.

9.2.3   *Communication with ethnic minority parents*

This study shows that communication between health professionals and ethnic Norwegian parents about overweight in preschool children may be challenging. We do not know for sure how ethnic minority parents would experience the conversation with the public health nurse or their general practitioner if their preschool child was identified as overweight at the Child Health Clinic. Ethnic minority parents comprise a diverse group, and their experiences would probably differ according to ethnic group and culture, as well as language skills and the level of integration**.** A recent meta-synthesis of qualitative studies found that health professionals working with families with overweight or obese children often reported lack of knowledge and confidence in their communication skills (209). Counselling immigrant parents and groups with low health literacy may be extra challenging (210, 211). Public health nurses in Norway reported in a study that they often regarded immigrant families as a generic group and rarely enquired about the family food or parents' education level and health concepts to adjust their counselling strategy (210).

Public health nurses may experience that the extended family undermine and fail to support lifestyle changes initiated by parents and the health care system (210, 212). Perceived social pressure and social norms, like preference of chubby infants, may influence the mothers' feeding practices in ethnic minority groups (213). In Oslo, mothers with Pakistani background told that they were eager to please the whole family, and served traditional meals with oil and butter in addition to Western food items with sweets and soft drinks (214). In a recent qualitative study from the UK, English speaking black African parents of children aged between 6 months and 5 years told that prices influenced the choices they made for their children's diet more than health messages (211). Further, language barriers, immigration status and discrimination were also identified as central factors that underpinned the families' struggles for a healthy diet and weight maintenance in early childhood. Interviewing immigrant parents less integrated and not speaking English may have given more, and perhaps, different information.

To the best of our knowledge, there are no studies exploring ethnic minority parents' encounter with the Norwegian health care system and their suggestions for useful support from health care professionals, schools, kindergartens and population-based initiatives to promote healthy weight in children.

10 Methodological considerations

The scientific quality of both quantitative and qualitative studies must be available for assessments according to broadly accepted criteria (163). Inadequacies in the design, conduct, or analysis of a study will very likely result in biased estimates (160). It is also important to consider ethical issues. In this section, I will discuss three main essential themes regarding methodological issues;

1) *Internal validity*; whether the data were collected, analysed and interpreted without, or with little, bias. The main potential biases discussed are
   - Selection bias
   - Measurement bias (or information bias), which can be systematic or random
   - Confounding
   Internal validity is a prerequisite for external validity
2) *External validity;* whether the results can be generalized to subjects outside the study sample (in Oslo, nationally, and internationally).
3) *Reflexivity;* how my position and preconceptions as a general practitioner and doctor at Child Health Clinics may have influenced the studies. This applies mainly to Paper III.

10.1 Quantitative papers

10.1.1 Internal validity

10.1.1.1 Selection bias

Selection bias concerns the representativeness of study participants in relation to the source population. It is not possible to make valid generalizations from unrepresentative or biased samples (215). This is of uttermost importance in paper I, which describes prevalence estimates of obesity and thinness. The population-based prospective cohort design itself may counteract selection bias. We attempted to include a representative sample of the source population of healthy pregnant women living in Groruddalen, Oslo. In the following, I will describe how we worked with the intention to achieve a representative sample.

Mothers

<u>Ethnicity</u>

Ethnic minority populations are often underrepresented in research, as they can be hard to reach due to barriers represented by culture, language, and literacy (216). Recruitment strategies to such studies therefore require extra efforts and costs. All written information to eligible women about the STORK Groruddalen study, including the questionnaires, were translated into eight different languages, and minority counsellors helped reaching out to minority women (148). Generally, most mothers already knew and trusted the staff at their local Child Health Clinics. Of the ethnic minority participants, 21 % reported that they needed an interpreter during doctor's visits. The staff members were flexible and used interpreters extensively. This indicates that we were able to recruit a large number of otherwise hard-to-reach women.

We found that the ethnicity of the study participants was fairly comparable to all women attending the study Child Health Clinics regarding age, parity and ethnicity, and their age comparable to the mean age of all Norwegian women in Norway giving birth in 2008 (148). This indicates that we were able to recruit a representative sample. However, we cannot rule out that there might have been some differences between our sample and the source population, as we did not have approval to collect detailed information about the women that were not included.

<u>Choice of antenatal care setting</u>

Data monitoring activity at the Child Health Clinics prior to the study showed that the majority (75-85 %) of pregnant women in the study districts attended antenatal care at their local Child Health Clinic (148). The rest probably had follow-ups by their general practitioners o or by specialized hospital care. General practitioners in the study districts were asked to refer pregnant women early in pregnancy to the local Child Health Clinics. This probably increased the number of women attending the Child Health Clinics during the study period, compared with previous periods. We have reasons to believe that only very few healthy women attended a private obstetrician for prenatal care. Women with medical complications such as pre-existing diabetes and epilepsy were followed by hospital

obstetricians from early pregnancy. Thus, we believe that our sample mainly represents healthy women who lived in the study district who were expected to have a normal pregnancy and birth.

Attendance rate

Compared with similar prospective population-based mother-child cohorts, the participation rate of 74% in the STORK-Groruddalen study is high. Although the attendance rates were slightly lower in Africans and Asians, the attendance rates were still much higher than in most other studies (217, 218). The main reason for exclusion in our study was late attendance (second half of pregnancy) at the Child Health Clinic. Several reasons for late attendance were spontaneously reported by the women and the staff:

1) Many women had not heard of the study in time, partly because their general practitioner did not know or had probably forgotten the ongoing project.
2) More women than anticipated attended general practitioners outside the study districts.
3) Some women did not speak any of the languages covered by information material.
4) Antenatal services were periodically less available at two of the Child Health Clinics due to long-term sick-leave among staff and a prolonged reorganisation with fusions of Child Health Clinics in two of the study districts.

Of the invited women, 291 (26 % of invited) refused to participate. The main reasons spontaneously reported were lack of time due to work obligations or care of small children. As inclusion demanded extensive resources concerning administration, study staff, interpreters, and information material, we had to stop the inclusion period after two years, limiting the number of participants.

Comparisons with other prospective mother-child cohorts

Below, I will mention some other prospective mother-child cohorts that may be compared with ours. Besides a high attendance rate, other strengths of our cohort include the population-based design, inclusion of multiethnic groups, rich information, and detailed

anthropometric measurements of the mother and child. However, the total number of participants is smaller than in some other comparable cohorts, from reasons given above.

The *Norwegian Mother and Child Cohort study (MoBa)* pregnancy cohort study invited about 70% of all pregnant women in Norway from June 1999 to December 2008 (219). The total participation rate for all invited pregnancies was 41%, and the cohort includes more than 95,000 mothers and 114,000 children. However, a weakness of this study was selection bias with a socioeconomic gradient (219). The participation of ethnic minority women was very low, as participants had to fill out an extensive questionnaire in Norwegian, which probably contributed to this. Selection will bias the estimates of prevalence of exposures and outcomes, but not necessarily estimates of associations between exposures and outcomes (220). Of note, in the MoBa study, detailed clinical examinations of participants are not available. Follow-up studies of the MoBa cohort are still ongoing, and its major strength is the large sample size.

The longitudinal *Hyperglycaemia and Adverse Pregnancy Outcomes* (HAPO) multicentre study was set up to explore the risks associated with various degrees of maternal glucose intolerance less severe than type 2 diabetes mellitus (180). This is a large cohort of about 25,000 women from nine different countries and all continents. However, this study was not population-based, and the response rate was lower (53.6 % of the eligible pregnant women), which may have given a less representative sample and weakened the external validity. Further, women were not included until week 28 of pregnancy.

In the *Amsterdam Born Children and their Development study* (ABCD study), 7043 pregnant women (response rate 67%) granted the permission to use her own and her child's medical files (218). This cohort resembles our, as it is prospective, population-based, but comprises a larger study of ethnic minority mothers. Further, it investigates health and ethnic disparities in children at birth and later in life, as well as maternal lifestyle, medical, psychosocial and environmental conditions during pregnancy, and how exposures in pregnancy and early life conditions can explain children's health. Of note, as women were not screened with oral glucose tolerance tests in the ABCD study, it does not explore the effects of gestational diabetes. However, their results from other ethnic minority groups complement our findings.

Another prospective, population-based cohort study in a multiethnic population from The Netherlands is the *Generation R Study* (n=9778), with a response rate of 61% (221). In this study, women were included in early and in late pregnancy, as well as after birth, and oral glucose tolerance tests during pregnancy were not offered. Further, questionnaire data were collected by telephone and by mail (222). Thus, this study does not have as extensive information from pregnancy as our study, and especially not on hyperglycaemia. However, this study is big with a large statistical strength, as well as a great variety of data collected from children after birth from several waves of follow-up.

Children

In paper I, we have some missing anthropometric data on children (Table 2, Figure 8 Flow chart) We had ethical approval for and access to Child Health Clinic records for children still living in Oslo. However, when the children were 4-5 years old, many families had moved out of Oslo and some had moved abroad. Still, we managed to retrieve data on 88 % of the children that had moved from Oslo to other districts in Norway; 168 out of 192 children. One might expect that this high mobility could cause a systematic bias due to selective loss of certain ethnic- or socioeconomic groups. However, the distribution of basic characteristics was remarkably similar in the original cohort of pregnant women and the cohorts of children (Table 7). Further, missing data are more likely due to factors at the Child Health Clinics, rather than factors related to participants. This may indicate that we managed to get representative samples of children in the STORK-Groruddalen study cohort. In Paper II, we have anthropometric data from two or more time points from birth to 4-5 years on 93.7 % of the live-born singleton children. Assuming that the STORK Groruddalen cohort was representative for the source population, our sample of children could be considered a fairly representative sample with good internal validity.

Table 7.
Baseline characteristics of study cohorts

Baseline characteristics	Total cohort of pregnant women	Cohorts of children	
		Study 1	Study II
Ethnicity (%)			
3 ethnic groups			
South Asia	26.6	27	
Middle East / North Africa	22.9	20.7	
Europe	50.5	52.3	
5 ethnic groups			
South Asia	24.3		24.5
East Asia	5.2		5.2
Middle East / North Africa	20.9		20.4
Europe	46.1		46.9
Others	3.5		3
Maternal age, mean	29.9	29.9	29.8
Maternal prepregnant BMI, mean	24.6	24.5	24.6
Maternal education (%)			
Primary education or less	16.2	16.8	16.5
Completed high school	39.4	37.8	39.2
Completed university/college	43.7	45.4	44.3
Child birth weight, mean (grams)	3420	3431	3459

Fathers

Paternal data were missing for 23% of the total study cohort, which may have introduced some selection bias. Fathers' questionnaires were self-administered, and selection related to language skills may be operating, as interpreters were not offered to fathers. Therefore, data on fathers were not included in our analyses, also because this would have reduced the sample in the multivariate analyses. We cannot rule out that our results would have been somewhat different if we had included paternal data about education, and BMI. However, we do know that less than 5% of the children having data on fathers were of mixed ethnicity. Further, fathers not answering the questionnaires were probably not likely to be of a different ethnicity than the mothers were.

Systematic errors

Ethnicity and country of origin

The term ethnicity is complex and refers to the social group a person belongs to, or is perceived to belong to, as a result of a mix of cultural and other factors (130). As discussed in the introduction characteristics defining ethnicity are not fixed or easily measured, and there is no standard or widely accepted protocol for the collection of ethnicity or ethnic group data, although important factors, as food traditions and inheritable factors, may often last over generations (131). In the Stork Groruddalen study, ethnicity was defined by the participating mother or her mother's country of birth if this country was outside Europe (148). In this way, the maternal line was chosen, recognizing the important transfer of dietary and cultural habits through the mother. By using this definition, some women who were born and raised in Norway were included into ethnic minority groups. This may be controversial, as these women may define themselves as Norwegian. However, few were second-generation immigrants, except among Pakistani women. Neonatal anthropometric measurements of neonates in first and second-generation Pakistani immigrant mothers did not differ significantly (149).

Prior to the analyses, we merged women with origin in 65 different countries into five defined ethnic groups. There are several ways we could have defined these groups. In order to make ethnic groups that could cover our cohort, and include the largest immigrant populations in Norway, we had to make some pragmatic decisions, and chose to group populations that share common cultural factors and/or a body composition that are related to type 2 diabetes.

Children with ethnic origin in Pakistan (n=127), India (n=11), and Sri Lanka (n=60) were defined as having South Asian origin. This ethnic groups share a "thin-fat" body composition with a relatively higher ratio of fat to lean mass for any given level of BMI, and a high prevalence of type 2 diabetes, compared with populations with European and African origin (22, 146). However, these merged categories may hide substantial differences regarding culture, religion and language.

The East Asian populations are heterogeneous concerning culture as well as body composition. The children of East Asian origin in our cohort also comprise a smaller and heterogeneous group, with the Philippines (n=13), Vietnam (n=18), Thailand (n=5), and "Other" (n=7) as countries of origin. However, preliminary analyses for Paper II with South- and East Asia grouped both together and apart, showed similar results for these two groups.

Of the ethnic group defined as "European", there may be differences in culture between the "East Europeans" (n=43) and the rest; "Western Europeans" (n=346).

We chose to merge women with origin in the Middle East together with women from North Africa, including the Horn of Africa (mainly Somalia and Ethiopia). This decision might be questioned, but was based on several considerations. First, these regions share many cultural factors. Geographically they are not far apart, and through history, there has been a close contact. Trade of cinnamon from Somalia to Egypt dates back to the 25th century Before Christ (223). Merchants and sailors from the Horn of Africa came, during the 7th century, under the influence of Islam through their Muslim trading partners living on the Arabian Peninsula (224). Second, preliminary analyses showed that children with origin in The Middle East and North Africa, including the Horn of Africa had similar results concerning our outcomes. Third, there are mainly East-Africans in our cohort, and they differ from West-Africans in several ways, including body composition.

Socioeconomic position

We have used two different variables to define socioeconomic position: maternal education level (Paper I) and a principal component variable of 11 sociodemographic variables, including maternal education (Paper II). There is no ideal way to define socioeconomic position, but *education* is a frequently used marker of socioeconomic position in medical research (225). Education is relatively easy to measure in self-administered questionnaires, and the response rate to questions about education is often high and similar for different ethnic groups (226). However, this presumes that participants are able to read and understand the questions. As the questionnaires data in the STORK Groruddalen cohort were collected through interviews with study staff, and assisted by interpreters, we expect to have valid data on education. Nevertheless, limitations exist when education is used as a

proxy for socioeconomic position in multiethnic populations. Education might have different implications if it is obtained outside the country of residence, and proper jobs for a certain education might be hard to find (225). Thus, a high education may not compensate for the stress of being unemployed, underpaid or at a low level of the social hierarchy. Another limitation is that individuals may report the education level before it is fulfilled. As only 31 mothers reported currently being a student, we do not expect this to represent a considerable bias in our cohort.

The *principal component analysis variable of socioeconomic position* comprises more facets than education alone. However, previous research has shown that different socioeconomic indicators may not be equally applicable to all ethnic groups (133, 227). There may be cultural and social differences in economic priorities and opportunities, and there might be differences in the quality of assets (car, house, TV, etc.) between ethnic groups (226). Thus, the principal component variable of socioeconomic position may still be a proxy for other unmeasured health indicators.

Gestational weight gain

Gestational weight gain is most often calculated as the difference between maternal weight at the end of pregnancy and prepregnant weight. We chose to use the estimated gestational weight gain from conception to week 28 of pregnancy. There were three reasons for this choice. First, at week 28, the relative weight of the foetus, the placenta, amniotic fluid and body water is less than later in pregnancy. Thus, this weight gain may give a better estimate of maternal fat gain. Second, using weight gain up to the measurement of maternal glucose levels allowed us to comply with assumptions of temporality, as women diagnosed with gestational diabetes were given life style advice and hence may gain less weight after being diagnosed, affecting the total gestational weight gain measured at birth. Third, early-gestation gestational weight gain has a stronger effect on offspring BMI at age 5 years and adiposity at age 9 years than mid- and late-gestation gestational weight gain (59).

We divided the variable gestational weight gain into tertiles in order to be able to illustrate our findings with a figure of the effect of gestational weight gain. Analyses with gestational weight gain as a continuous variable gave similar results as using the categorized variable, suggesting a continuous dose-effect. Using the recommended optimal gestational weight

gain for women according to their prepregnant weight might have given additional clinical information, but could also cause problems when both adjusting for maternal BMI and taking maternal BMI into account in the gestational weight gain variable.

Definitions and treatment of gestational diabetes

Gestational diabetes, defined as any degree of glucose intolerance with onset or first recognition during pregnancy, was first described about half a century ago (228). Worldwide, there are several criteria for diagnosing gestational diabetes, and some studies even include pre-existing type 2 diabetes. This makes it very difficult to directly compare studies. Prevalence rates of gestational diabetes reported worldwide range from 1-45% of pregnancies (229). This diversity reflects not only which definition of gestational diabetes that is used, but also differences in study populations concerning ethnicity, age, and obesity (153). Further, if a whole population of pregnant women is screened by universal oral glucose tolerance test, more women with gestational diabetes are identified, compared with a population in which screening is not a routine, or is offered to high-risk groups only (230).

As described in the methods section (Table 3), we used different criteria for the diagnosis of gestational diabetes in the two quantitative papers; by the time that the children were 4-5 years old (Paper I), the WHO_{2013} criteria were applied, as we wanted use the new definition based on the HAPO study. In paper II, we used the WHO_{1999} criteria that were used during data collection, reflecting which mothers were actually diagnosed and received information and treatment. Compared with the WHO_{1999} criteria, the WHO_{2013} criteria demand a *lower threshold* for fasting glucose but a *higher threshold* value of 2-hours glucose at the glucose tolerance test, for the diagnosis of gestational diabetes. However, both papers conclude that at age 4-5 years, gestational diabetes (regardless of its definition) was not associated with children's BMI. Further, sensitivity analyses in Paper II with the WHO_{2013} criteria gave similar results as using the WHO_{1999} criteria. All women diagnosed with "mild gestational diabetes" were given oral and written lifestyle advice, and were advised to make an appointment with their general practitioner for further follow-up. However, we do not know the extent to which these women actually received the recommended treatment, and if they met the treatment goals in primary care. This may have influenced our results.

Definitions of thinness, overweight and obesity in children

We have used the BMI cut-off values according to the International Obesity Task Force (167). Internationally, there are several definitions of thinness, overweight and obesity in children. This complicates the comparison of studies. Many countries use their own national growth standards and cut-off values. WHO's definitions of childhood overweight and obesity are different in children under 5 years of age, and in children 5 years of age and older (1): Under 5 years of age, WHO's definition of overweight is more than *two* SD above the WHO growth reference median weight-for-height. From the age of 5 years, overweight is defined as more than *one* SD above the WHO growth reference median BMI-for-age. This distinct change in cut-off values at 5 years of age can make it challenging model growth trajectories in preschool children. As the children in our sample were between 0 and 5 years of age, we chose to use the International Obesity Task Force, which has continuous cut-off values.

Table 8. Weight classification of children, cut-off values

	International Obesity Task Force	**WHO**	
Established	2000 (obesity), 2007 (thinness)	2006	
Based on	Nationally representative cross-sectional growth studies	Healthy, breast-fed infants of non-smoking mothers	
Countries	Brazil, Great Britain, Hong Kong, the Netherlands, USA, Singapore	Brazil, Ghana, India, Norway, Oman, USA	
Cut-off	"iso-BMI" corresponding to centile curves passing through; BMI 18.5 kg/m^2 (thinness grade 1), 25 kg/m^2 (overweight) and 30 kg/m^2(obesity) at age 18 years;	BMI Z-score standard deviations (SD) from the WHO growth reference median weight-for-height	
Age of children	2-18 years	under 5 years	5-19 years
Thinness	iso-BMI below 18.5 kg/m^2	-2 SD	-2 SD
Overweight	iso-BMI above 25 kg/m^2	+2 SD	+1 SD
Obesity	iso-BMI above 30 kg/m^2	+3 SD	+2 SD

The relation between BMI and the percentage of body fat depends on age and sex, and differs across ethnic groups (143). As BMI is based on weight by height, it does not differentiate between fat and lean mass. In children, BMI correlates more strongly with fat mass at the upper end of the adiposity spectrum, where a larger proportion of weight consists of fat mass. In thin children, BMI is a better predictor of lean mass (231). Measures of skin folds, waist circumference, Magnetic Resonance Imaging and other indirect methods would have estimated adiposity more accurate. However, a substantial proportion of our participants would probably not attend follow-up studies with MRI and other more valid methods for measurement of body fat that are difficult to apply (in local study sites) outside hospitals. Importantly, by using routine measurements of height and weight from the Child Health Clinics, we were able to use representative data from a large group of children.

I. We used the positive BMI adjustments of $+ 1.12$ kg/m^2 (25) for children with South Asian origin as in the sensitivity analyses in Paper I. Among other suggested adjustments for South Asian children is the calculation of BMI centile curves passing through the cut-off points of 15 kg/m^2 (thinness), 23 kg/m^2 (overweight), and BMI 25 kg/m^2 (obesity) at age 18 years, derived from adult Asian Indian cut-off values (177). To date, WHO and the International Obesity Task Force do not suggest specific cut-offs for South Asian populations, as both standards are based on populations with different ethnic backgrounds and cultural settings (143, 151, 232).

Preliminary analyses using BMI z-scores

In preliminary analyses we created BMI z-scores (Figure 12) based on the WHO-standards (232) and the Norwegian national references (233). This would be of value to compare the BMI-trajectories in our cohort with standardized growth charts, and our findings would perhaps be easier to interpret on a global scale.

However, when plotting our data, we found that the BMI-development in our cohort differed somewhat from both WHO-standards, as well as from the national references. There are several reasons for this, including for example that the BMI z-score standards from birth are not calculated from the same study sample as postnatal BMI z-scores. When building the mixed models, these differences from the references would also have to be

taken into account. This would increase the complexity and have implications for interpretations. After careful considerations, and advice from an experienced statistician with strong competence in growth modelling using mixed models, we therefore chose to use BMI and not BMI z-scores as our outcome measure.

Figure 12. Preliminary analyzes:

BMI z-score for European children exposed to gestational diabetes (red line) and not exposed to gestational diabetes (blue line).

A. According to WHO standards
B. According to Norwegian national reference (Juliusson)

A.   ·   B.
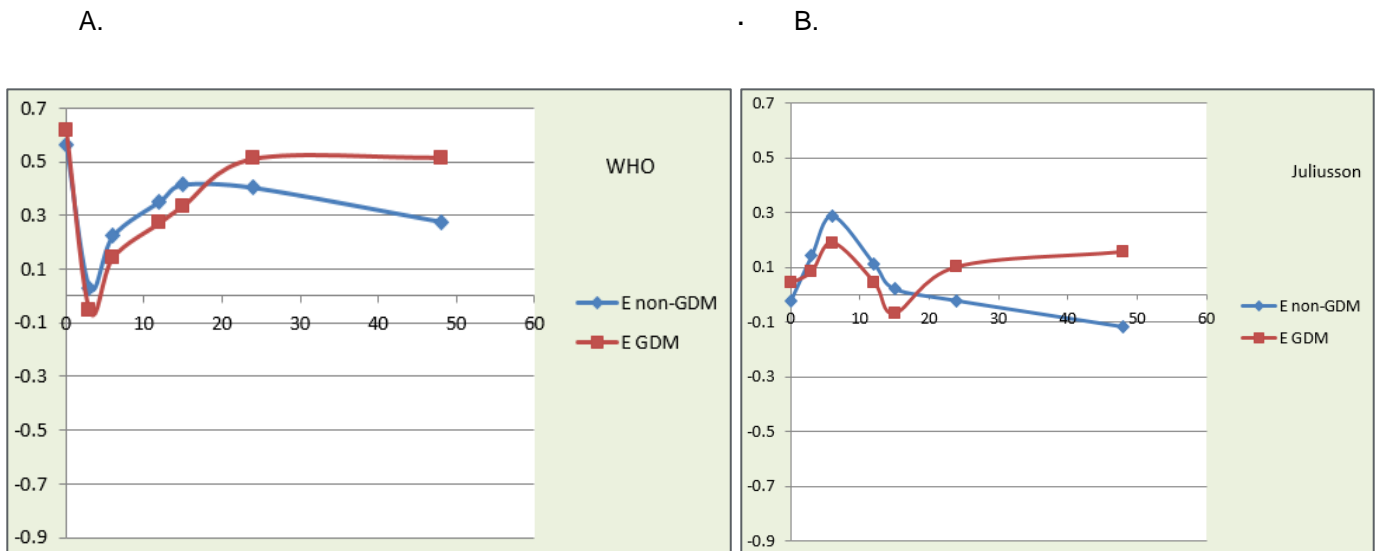
Random errors

Maternal prepregnant weight

Prepregnant weight was self-reported. This variable is sensitive to recall bias. To counteract recall bias, women's weight was measured at inclusion before they were asked about their prepregnant weight. There was a strong correlation between the prepregnant body weight and the measured body weight at enrolment at mean gestational week 15 (r=0.97), with no significant differences across ethnic groups. This indicates fairly god internal validity (234). A recent systematic review and meta-analysis showed that among women of reproductive age, mean self-reported weight was underestimated by -0.94 kg, compared with directly

measured values (235). The review concluded that this magnitude of underestimation was negligible, regarding the use of self-reported weight in clinical work and research.

Calculation of gestational age

For the last decades, the final calculation of gestational age in Norway has been based on routine ultrasound scans at week 17-20 of pregnancy (236). This calculation method assumes that foetuses have similar growth up to 17-19 weeks of pregnancy (237). As the Stork Groruddalen study wanted to analyse ethnic differences in early foetal growth, we chose to calculate gestational age based on days from the first day of the last menstrual period. This method may be sensitive to recall bias. As the study staff used interpreters, misunderstanding related to language would probably not be an issue. Thus, recall bias will most likely apply equally to all ethnic groups. Further, as the most premature children were excluded from further analyses, bias in calculation of gestational age may not be of considerable importance to other measures than birthweight.

Physical activity

Indirect calorimetry is considered the gold-standard method for measuring energy expenditure from physical activity (238). However, methods used for indirect calorimetry are expensive and time consuming and may involve invasive procedures. Therefore, they are inconvenient for epidemiological research. Objective recordings of physical activity with electronic motion sensors are more feasible in clinical studies (239). However, these methods may also be time consuming. They are limited in their ability to discriminate specific modes of and arenas for physical activity, and require the participants being willing to wear a measuring device. In our study, the women were asked to wear a multi-sensor recorder of physical activity for 4-7 days following each visit, and we collected valid recordings from 82.4% of the women (240). Analyses of these data showed that in week 28 of pregnancy, only one quarter of the women compiled with the guidelines for physical activity in pregnancy (241).

Although information about the level of physical activity prior to pregnancy was collected by a validated questionnaire (155), this variable is sensitive to bias. Physical activity levels based on self-reports are often overestimated and may hold recall- and social desirability

bias (156). This variable would probably have been more accurate if measured objectively. Although we have collected objectively measured physical activity during pregnancy, we choose not to adjust for this variable as this was considered a secondary exposure variable, and for almost 20% of the mothers objectively recorded data in pregnancy was missing.

The mothers' dietary pattern

Multiple days of weighed food records is considered the gold standard methods for dietary assessments. However, this method is seldom used in large studies, as it is time consuming for both participants and researchers, and it is still not very accurate (242). Our dietary pattern variable was based on a food frequency questionnaire asking for data from the last two weeks (157). The food questionnaire was developed by experienced researchers, but not validated before the study. Very few of the ethnic minority women were represented in the "most healthy" cluster. In order to have all ethnic groups represented in two main clusters, the two "least healthy" and the two other cluster were merged. We assume that the food questionnaire reflects the mothers' regular dietary patterns, but we cannot rule out that some of the women may have changed their diet during pregnancy. Limitations of food frequency questionnaires are recall bias, inaccurate estimation of portion samples, and socially desirable answers (242). Thus, perfect data on diet are hard to get, and our data on dietary patterns are probably inaccurate.

Anthropometric measurements of children

Inaccuracy in anthropometric measurements may cause less precise estimates. Therefore, it is important to avoid measurement bias.

The main STORK-Groruddalen study: Although the data collection was performed by a limited number of trained personnel during standardized conditions, we cannot rule out minor inaccuracies. However, this probably applies equally to different ethnic groups. Study staff regularly tested the scales at the hospital birth- and maternity wards. The maximum differences ever observed between the scales were 5g (149). This indicates reliable instruments at the study hospitals.

At the Child health clinics, many public health nurses using different scales performed anthropometric measurements of children. According to national guidelines, it is recommended to test scales twice a year (128). However, we do not know if all Child Health clinics followed these guidelines, and it is not mandatory to use medically approved scales (243). Further, we do not know how strictly public health nurses followed the national guidelines for measuring weight, length and height. Looking at data on height for children from the age of 2 years, we may conclude that some public health nurses measured height to the nearest 0.5 cm, and not to the nearest 0.1 cm, as recommended. Thus, children's anthropometric data may have been be more accurate if performed under standardized conditions. However, this measurement bias probably applies equally to all ethnic groups.

Unmeasured confounders

We must assume that there are unmeasured confounders to the relationships of interest in paper I and II, especially concerning lifestyle and socioeconomic factors. Another possible confounder is parental size at birth, especially for the associations with maternal BMI. However, the STORK Groruddalen cohort consists of almost 60% ethnic minority women, primarily born in low- and middle-income countries. Although we asked about parental birth weight, we experienced that many did not know their own birth weight or whether it was outside the "normal" range (many were born at home, birth weight was not always measured, or they did not have access to birth records). This was for example the case for approximately 50% of South Asians and 70% of Africans. We were therefore not able to adjust for maternal birth weight as a confounder. We excluded 19 children from our study sample due to severe medical conditions obviously affecting growth. This included conditions discovered at birth, but also based on information retrieved from local health clinic records. However, we did not have ethical permission to retrieve detailed data on all diseases or medication use that could potentially have influenced children's growth during the entire age span or during specific periods. Although this is a limitation, adding such information to the dataset would also add tremendous complexity to the data, and to the interpretation of findings.

The power of a test is the probability that a study of a given size would detect a real difference of a given magnitude as statistically significant (215). Compared with some other prospective mother-child cohorts, the STORK Groruddalen study had a limited number of participants. This was mainly due to the relatively short and resource demanding inclusion period (2 years). With a larger study cohort, our statistical power would have been larger, and our findings generally more robust. Perhaps we would also have been able to show statistically significant results for more ethnic groups and to study interactions in more details.

10.1.2   *Confounding factors and causality*

A confounder is a variable, which is a common cause for the exposure and the outcome. Confounding can lead to over- or underestimation of an effect, and even change the apparent direction of an effect (215). A collider is a variable, which is a common cause of both the exposure and the outcome. A mediator is a factor that explains the relationship between the exposure and the outcome (i.e. is on the causal pathway).

Figure 13.
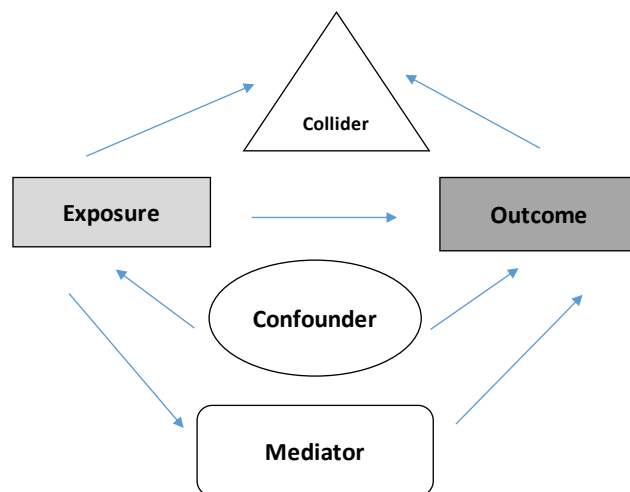
When analysing data from epidemiological studies, one attempts to look systematically at causal connections. Our approach when handling confounding is similar to other comparable mother child-cohort studies, like the ABCD study (218). The randomized controlled trial has long been considered as the gold standard for causal inference in

medical research (160). In an ideal randomized controlled trial, association is causation because of the randomization. However, it is not feasible to investigate all research questions through randomized controlled trials (215). We could for instance not randomize individuals to a certain prepregnant weight class. Other factors such as age and ethnicity are not controllable by the individual. Furthermore, it is not always ethical to randomize individuals.

A prospective observational study design is suitable for studying the distribution of diseases, symptom load, or risk factors among populations and subgroups. Exposures can be measured before the outcome, and this offers a possibility of studying associations and potentially causal relationships (215). Observational studies may have the same goal as randomized controlled studies, but here the investigator cannot control the exposures or treatments. In this case, associations can generally not be directly interpreted to represent causation. However, observational studies are often the only way to explore causal inference (160).

Recently, *causal modelling* with emphasis on graphical models have been used to explore causal connections also in observational studies (160). In paper II, we used Directed Acyclic Graphs in the model building process (Figure 10). In this way, we could visualize the causal network that linked the main exposures (gestational diabetes, prepregnant BMI and gestational weight gain) with the outcome (children's growth). Further, we could identify confounders that should be adjusted for (ethnicity, maternal socioeconomic status, maternal age and parity), as well factors that were strongly associated with child growth (sex and gestational week at birth) to attain more valid estimates. We could also identify potentially colliding and mediating factors (such as breastfeeding) that usually should not be adjusted for.

In paper I, ethnicity was the main exposure variable. However, as ethnicity does not have any relevant confounders (as no factors can influence your ethnicity), we have in this paper primarily adjusted for mediators. It is therefore important to keep in mind that the total effect of ethnicity in this study population are represented by the univariate effect estimates, while adjusted effects represents the effects that are not mediated through other factors (i.e. the direct effect).

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10.1.3   *External validity*

Generalizability of study results beyond the study sample reflects the external validity of the study. Internal validity is a prerequisite for external validity. In chapter 10.1.1.1, representativeness is discussed, and we suppose that our STORK-Groruddalen cohort included a representative sample of pregnant women living in the three study districts. From Statistics Norway we know the composition of the populations of immigrants and the largest groups at the time of the study. The study districts comprise a large amount of the non-Western population in Norway. We therefore assume that the ethnic minority groups included in our sample (i.e. origin in South Asia, Middle East and North Africa, and Europe) were representative for the largest ethnic minority groups of women in childbearing age in Norway.

When comparing characteristics in our total cohort sample with data from the Norwegian Medical Birth Registry on women living in Oslo and Norway at the same period, we found fairly similar results concerning maternal age, parity, prepregnant BMI, and Caesarean section (63, 148). This supports the notion of a representative sample. However, our cohort had a lower smoking rate; of the STORK-Groruddalen pregnant women, 4.3% smoked, compared with 12.4 % of the total population of pregnant Norwegian women. This probably reflects the lower generally lower rate of smoking in Oslo, not least, among ethnic minority women. In line with our findings, international studies report that birthweights of babies born to ethnic minority women are lower than those of babies born to European women (244). A study based on data from the Norwegian birth registry on all single births from 1980 to 1995, showed, in line with our findings, that children of Vietnamese and Pakistani origin had lower mean birthweight, compared with ethnic Norwegian children. However, this study also partly differs from ours, showing that children of North African origin had the highest mean birthweight (245)

The degree, to which the ethnic minority women in our cohort are representative of migrant women in other parts of the world, is unknown. First, the host country policies influence which groups of immigrants that are allowed to come and to settle. Second, the degree of socioeconomic disadvantage, including discrimination, poverty and racism, often experienced by immigrants, may differ from one host country to another, and this may

influence the generalizability of our results. Interestingly, a recent study using multi-country data for more than 31 million term births in 10 high-income countries, showed that migrants from a given origin had heavier new-borns in countries where the mean birthweight of native-born was higher and vice versa. Hence, mean birthweight differences between migrants from the same origin and the native-born varied substantially across destinations (246).

10.2   Paper III

10.2.1   Internal validity

<u>The interaction between the researcher and participants</u>

A mutual understanding between the participant and the interviewer is a prerequisite for the internal validity in an interview study (161); does the interviewer capture the essential meaning of what the informants are telling during the interviews? This depends on the atmosphere during the interview, as well as the interviewing technique of the researcher. Does he or she make the informant feel comfortable and understood? Does the informant understand the concept of the interview and what the interviewer is really asking about? I experienced that informants seemed relaxed, and they all contributed with a large amount of information. In order to prevent response bias, we chose open-ended questions in the interview guide. However, most of the time I did not use the interview guide, but it helped ensure that we had covered all themes in each interview. As a general practitioner, I frequently have conversations with my patients on sensitive topics. This was probably an advantage when conducting research interviews.

<u>Information power</u>

Malterud et. al. have introduced the term "information power" as a concept for sampling strategy for qualitative research (164). The more information the sample holds relevant for the present study, the lower number of participants is needed. One strength of our study is that all parents were interviewed shortly after they had experienced the difficult conversation concerning their child at the Child Health Clinic. In this way, parents were probably able to remember the conversation, as well as the feelings and thoughts that it brought up. We experienced that our informants shared a large amount of information. We

therefore stopped data collection at a time point when all authors decided that the information power was sufficient.

Choice of method for data collection

There are several scientific methods to explore people's opinions. For example, by using an anonymous questionnaire instead of a qualitative interview design, we had probably been able to include more participants. A questionnaire would have taken less time for parents to fill out, and some would perhaps feel more confident about protection of their privacy. Questionnaire data may be controlled, measured, counted and analysed by statistical methods. However, such data would perhaps not have given as broad information as a qualitative design did. For instance, we may not have experienced parents' willingness to talk and elaborate on their own experiences of being overweight. In this way, we would probably not have found that parents' own experience of overweight acted as both a barrier and a motivation to deal with their toddler's risk of overweight.

Focus group interviews can be a good approach when exploring different points of view. The interactions between participants can make it easier to bring up sensitive information that, for instance, are set under a taboo (162). On the other hand, informants living in small communities might be concerned that sensitive information could spread to people they do not trust.

Choice of method for data analysis

The results of a qualitative study may also rely on which methods that are being used in the analyses of the data. However, regardless of methods used, transparency throughout the whole process of data collection, analysis and interpretation, is a prerequisite for internal validity (161). By using an acknowledged and earlier described method, it will make it easier for the reader to follow and evaluate this process. As we wanted to gather knowledge on experiences, feelings, and believes from several participants, a cross-case analysis was suitable. We chose to use the method of *Systematic text condensation (165),* as we found this method to be thoroughly explained and easy to understand. One alternative analytic method is *Thematic content analysis* described by Braun and Clarke, which is believed to be a pragmatic and straightforward process of thematic cross-case analysis of qualitative data

(247). Another method of cross case analyse of qualitative data is *Grounded theory* (161). However, this method focuses more on developing new models and theories, which was not our aim this time.

10.2.2   *External validity*

Parents in our sample were in many ways homogenous. They were all ethnic Norwegian, lived in rural areas with grandparents close by, and 10 out of 11 informants were mothers. By interviewing a larger group of informants, we may have obtained an even broader spectre of information, and perhaps increased external validity. It would particularly be interesting to interview more fathers, as well as ethnic minority parents. Fathers would perhaps have focused more on physical activity. Ethnic minority parents might have expressed different attitudes towards the responsibility for feeding children, and their relationship with grandparents might have been different. Further, perception of a healthy weight for children and adults may vary between different cultures. However, it was hard to recruit parents during a limited time period, and we did not have the resources to translate information materials, neither to hire interpreters.

One strength of the study, which may have increased the external validity, is that we managed to recruit parents with different views. Most parents did not perceive their child as being overweight, but a few had given it a thought when buying clothes for their children. Some were upset and dissatisfied with the information they had received at the Child Health Clinic, while others appreciated health professionals expressing concerns about children's weight.

10.2.3   *Reflexivity*

When performing qualitative research, it is inevitable that the researcher has his or her own background, motives, perspectives, and preliminary hypothesis. These preconception are not synonymous to bias (163). It is, however, crucial to deal thoroughly with these issues. During our work in primary health care, all authors of paper III have had many conversations with parents, addressing our concerns about their child becoming overweight. Our preconceptions could lead us to find "just what we were looking for". To reduce the effects of our preconceptions, we chose a qualitative design with in-depth interviews where

informants could speak freely, rather than a survey with fixed questions. Having a non-health professional person conducting the interviews may have given other, and perhaps more negative information concerning the staff at the Child Health Clinics. However, the authors' professions as health care providers may also be considered as an advantage. This has given us motivation and qualifications for exploring the field of childhood obesity. An experienced clinician can facilitate informants to open up and talk freely. Although all authors are health professionals with experience from Child Health Clinics, our backgrounds as public health nurse, paediatrician, and general practitioner are somewhat different. Conducting analysis in close cooperation, we might have reduced the influence of the different researchers' preconceptions and unveiled blind spots.

11 Ethical considerations

Ethical considerations were integrated all the way from the planning of the three papers, collecting data, handling and analysing the data, and when writing the papers. All studies were conducted in agreement with the Declaration of Helsinki's ethical principles for medical research involving human subjects (248).

11.1   Paper I and II

At the three Child Health Clinics participating in the STORK Groruddalen Research Project, all invited women received detailed oral and written information in their preferred language about the study. The study protocol and consent forms were approved by the Regional Committee for Medical and Health Research Ethics for South-Eastern Norway (REK reference number: 2007.894). At inclusion, women willing to participate signed written consents on behalf of themselves and their child, including a consent to collect routine growth data at the local Child Health Clinics, specified in a separate approval (July 1st, 2010).

For the quantitative studies (Papers I and II), we have worked with sensitive data that can be traced back to the study participants. All the data were stored according to standards approved by the Norwegian Data Inspectorate (reference number: 07/ 01355-4/CGN), first at a secure server at Oslo University Hospital, and from 2017 at the University of Oslo's Service for Sensitive Data (TSD). This is a platform for collecting, storing, analysing, and sharing sensitive data in compliance with the Norwegian privacy regulations. Prior to all analyses, we removed all identifying information as social security numbers, addresses and telephone numbers.

For the STORK Groruddalen study, fathers were not asked for their consent to collect data on the children. Today, the Regional Ethics Committee probably would have demanded written consents from fathers as well, but this was not an established practice at the time of inclusion.

The children's growth data after birth are from routine measurements that would have been carried out irrespectively of the study. No children or mothers were exposed to potentially harmful or unpleasant procedures related to the studies.

Ethnicity can be a delicate issue. When presenting our findings on ethnic differences, we have intended to avoid any stigmatization of groups, and do not present ethnicity as a decisive factor.

12 Implications

12.1 Implications for health care professionals

The findings from this thesis may have implications for the approach that health care professionals use to promote healthy growth in children. Primary health care in Norway has a unique opportunity to take actions to prevent childhood obesity, as almost all children and pregnant women receive sustained care at Child Health Clinics, and by their regular general practitioner. Furthermore, our findings may also be relevant for specialized health care, as well as for health professionals outside Norway.

First, we highlight the importance of a *life-course- and transgenerational perspective* to understand the development of childhood obesity and to identify potentially crucial entry points for preventive actions. Maternal prepregnancy obesity and high gestational weight gain are intrauterine exposures associated with increased risk of childhood obesity. Young women may enter pregnancy affected by overweight, and a vicious circle of obesity, passed on from one generation to another might be created. Pregnancy and the postpartum period provides a window of opportunity for health professionals to promote healthy behaviour changes to prevent excess weight gain in pregnant women and subsequent postpartum weight retention. Our findings suggest that during infancy, children exposed to gestational diabetes that is treated may have a natural catch-down growth, and extra formula feeding might not be necessary.

Second, we report *ethnic disparities in childhood obesity*. The growth of children with origin in the Middle East/ North Africa should be monitored closely, as they have an increased risk of obesity, compared with the general population. Further, children with South Asian origin should receive extra attention if they display a growth pattern for weight/BMI with an upwards crossing of growth percentiles also before they reach the cut-offs for overweight, due to their increased relative adiposity.

Third, as parents and children report to feel vulnerable when *discussing the child being overweight*, health care professionals should be aware of this and cautious during such conversations, especially if the child is present. Our findings point to a strategy setting up a new appointment with one or both parents without the child. Fruitful subjects to discuss

may include parents' own weight experiences, as well as fear of eating disorders and low self-esteem in their children. Grandparents and kindergartens may also play an important part in feeding children, and should be included if relevant.

12.2   Implications for public health policy

The findings of this thesis may also have implications for public health policies.

First, the current *Norwegian guidelines* for preventing and treating childhood obesity (102) are outdated and do not cover the life-course- and transgenerational perspective (102). Nor do the guidelines suggest how interdisciplinary collaboration in primary care should be organized to prevent early childhood obesity. Our findings may contribute to the planned revision of these guidelines, and may also be of importance for revisions of guidelines for the preventive health services for children (128), as well as guidelines for antenatal care (129). To date, there are no formal, evidence-based guidelines on recommended gestational weight gain from European countries (196). Our findings may contribute to international strategies to prevent childhood obesity in a life-course- and transgenerational perspective.

Second, pointing out the importance of life-course- and transgenerational perspective, we advocate for *consorted actions and broad public programmes* to prevent obesity across generations. The whole community should be considered a target for population-based intervention, including grandparents, kindergarten- and school professionals. As we know that some ethnic minorities have higher risk of gestational diabetes, childhood obesity, and cardio-metabolic disease, these groups may need extra attention.

Third, *research* is needed on how to provide evidence-based interventions and improve trans-sectoral and interdisciplinary cooperation to prevent and treat childhood obesity.

12.3   Paper III

The Regional Committee for Medical and Health Research Ethics for South-Eastern Norway assessed the study protocol (reference number: 2011/1753). The Norwegian Data Inspectorate (NSD) and the University of Oslo approved of handling data anonymously.

According to Brinkmann and Kvaale, ethical considerations must be given throughout the entire qualitative research process (162). They argue that the researcher should particularly pay attention to four items; informed consent, confidentiality, consequences, and the role of the researcher.

1) *Informed consent.* Before I conducted the interviews, all informants received oral and written information about the aims of the study, who would get access to the data, who would perform analyses, and that they could withdraw themselves from the study within three months without any questions or consequences. Parents signed written consents on behalf of themselves.

2) *Confidentiality.* Before conducting interviews, I ensured parents that collection of data and results would be anonymized so that their confidentiality was ensured. Information on parents and children (Table 1, Paper II) was collected anonymously. I deleted soundtracks after transcription, and transcripts were deleted after analyses. During transcription, I gave participants numbers instead of names, and "the child", "mother in law" etc. replaced given names in transcripts. Further, in the text I tried to secure confidentiality by anonymizing information and choosing quotes that may not lead back to specific parents or children. However, we cannot rule out that parents later felt insecure and regretted on participation.

3) *Consequences.* There is a risk of stressful emotions coming up during research interviews discussing sensitive themes. Openness and intimacy during interviews can lead the informants to share information that they later will regret on giving. Knowing about ethical pitfalls, I tried to conduct interviews in a professional and sensitive manner. No parents wanted to withdraw from the study. They all gave positive feedback after the interview, which may have given them a good opportunity for reflection. However, we do not know if any of the parents felt that they had to participate to keep a good relationship with their public health nurse.

4) *The role of the researcher.* During interviews, researchers should be aware of the asymmetrical balance of power between them and the informants (162). Private information is given in a one-way dialogue and the scientists gets to interpret the statements. Further, the researcher must refrain from entering the informants' private zone and push them to share more than they want to. The researcher should keep a

professional distance and refrain from a therapeutic position. Thus, during interviews, I had to set aside my usual role as a doctor and helper in order to become a neutral researcher.

13 Conclusions

<u>Identification of children and families at risk</u>

- Compared with European children, children of Middle Eastern and North African origin had almost the double risk of overweight, while children of South Asian origin had the double risk of thinness at 4-5 years of age.

- After applying BMI adjustments for the South Asian children, taking into account their body composition with relatively increased adiposity, their prevalence of overweight markedly increased, and their prevalence of thinness decreased.

- Maternal prepregnant obesity was associated with persistently higher child BMI from birth to age 4-5 years, and with overweight at 4-5 years of age.

- Intrauterine exposures to gestational diabetes, prepregnant obesity, and high gestational weight gain were all independently associated with children's BMI trajectories from birth to preschool age, but the effects differed in relation to their effect size, timing and direction.

<u>Communication with parents of preschool children at risk</u>

- To protect their child from low self-esteem and eating disorders, some parents preferred to have the conversation about overweight without the child being present

- Parents' own weight- and dieting experiences represented a barrier, but also as a motivation for dealing with their toddler's weight

- Trying to achieve a healthy lifestyle, parents appreciated support from kindergartens, while grandparents often undermined parents' efforts.

14 Future perspectives

Most ethnic minorities in Norway with origin outside Europe have a higher risk of cardiovascular disease, type 2 diabetes and GDM, compared with the majority population (70). Pregnant ethnic minority women participating in the STORK Groruddalen study had a higher risk of depression, vitamin D- and iron deficiency, compared with their counterparts of European origin (249-251). At birth, we found ethnic differences in neonatal body composition (149). This thesis shows that ethnic disparities in childhood BMI development was also present in the same cohort.

More research is needed to gain knowledge on how to reduce ethnic and social disparities in health for pregnant women and their children. Little is known about how ethnic minority parents experience discussing their children's weigh with health professionals.

The population-based STORK-G cohort study has so far given valuable information of biological and social determinants of children's growth and development from early pregnancy to postpartum, as well as child growth data through infancy to preschool age. There is an ongoing collection of follow-up data from the mothers (STORK-G2). The children have now visited the public health school nurse for their six- and nine year's routine check-up. Data on weight and height measured at these controls are stored in digital health records, and we have gained ethical approval to collect such data. Using these data may give us an opportunity to explore whether the associations between the adverse intrauterine exposures and children's' BMI development, as well as the ethnic differences in overweight and thinness persist later in childhood.

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16 Appendix

Case report forms

Informed consent forms

FORM 1.1 (CRF 1.1)

(For information: If*: The interviewer must fill in the right category/code)

1. What is your current marital status?

☐ Married ☐ Partnership ☐ Cohabitant ☐ Single ☐ Divorced/separated ☐ Widow ☐ Other

2. What is your level of education?

	Completed	Attending now	No. of years
Less than 7 years' schooling	☐	☐	☐☐
Primary school (7-9 years' schooling)	☐	☐	☐☐
1-2 years' upper sec./vocational school (10-11 yrs)	☐	☐	☐☐
3-year upper sec./vocational school (12 years)	☐	☐	☐☐
District college, university, up to 4 years (Nurse, teacher, Bachelor's degree)	☐	☐	☐☐
University college, university, more than 4 years (Master's, PhD)	☐	☐	☐☐

3. What was your work situation when you became pregnant?

☐ Attending educational institution

☐ Housewife

☐ Job-seeker/laid off

☐ Rehabilitation/disabled

☐ Employed in the public sector

☐ Employed in the private sector

☐ Other   If other, what?:……………………

4. What is your occupation? State occupation/job title*   …………………………….
(Answer even if you are temporarily not working due to illness/leave)

5. Which religious community/religion do you belong to?* ………………………...............

6. Which country were you born in? Indicate which country*………………......................
If Norway:

☐ Born in Norway of two Norwegian parents

☐ Born in Norway of two foreign-national parents

☐ Born in Norway of one Norwegian + one foreign-national parent

7. Citizenship in which country? Indicate which country*………………………………..

8. *(If the country of birth and ethnic group do not appear to agree (e.g. "Indian" but born in Kenya, Uganda, South-Africa)* **Which ethnic group (common language, culture, history) do you feel you belong to?: ……………………………………………………………………..**

9. What is your native language? State language* …………………………………

10. How do you rate your Norwegian language skills?
□ Very good □ Good □ Fair □ Not very good □ Poor

11. Do you normally use an interpreter for doctor's appointments?
□ Yes, professional □ Yes, family/friend □ No

12. Have you been pregnant before? (Also consider pregnancies that ended in miscarriage/ abortion or with a stillbirth)
□ No □ Yes   If yes:

Number born alive: □□ Number stillborn: □□   Number of spontaneous miscarriages: □□

Number of induced abortions: □□   Number of ectopic pregnancies (outside the uterus): □□

13. I am now going to ask you about earlier pregnancies that have lasted more than 22 weeks.
(If more than 1 child per pregnancy, count twin 1, twin 2.)

(For each child)
Year of birth: □□□□   Pregnancy week for birth □□   Baby's weight in grams □□□□
Gender: Boy □ Girl □   Place of birth: □ Norway   □ Own native country □ Other
Method of delivery: □ Normal vaginal   □ Forceps   □ Vacuum   □ Caesarean section
If multiple birth: □ Twins □ Triplets
Healthy the first week?: □ Yes □ No   If no: □ Healthy now   □ Ill now   □ Dead

14. Do you have/have you had any of the following illnesses?
(Some diagnoses will mean that the woman cannot take part in the study)
(If yes, state the year the diagnosis was made).

		Year
Diabetes type 1	□ Yes □ No	□□□□
Diabetes type 2	□ Yes □ No	□□□□
Asthma	□ Yes □ No	□□□□
Allergy	□ Yes □ No	□□□□
Repeated urinary tract infections	□ Yes □ No	□□□□
Chronic liver disease	□ Yes □ No	□□□□
Prolonged high blood pressure	□ Yes □ No	□□□□

Heart disease	☐ Yes ☐ No	☐☐☐☐
Arthritis/Bechterew's disease	☐ Yes ☐ No	☐☐☐☐
Epilepsy	☐ Yes ☐ No	☐☐☐☐
Disease of the uterus/operation	☐ Yes ☐ No	☐☐☐☐
Involuntary infertility more than 1 year	☐ Yes ☐ No	☐☐☐☐
Mental illness	☐ Yes ☐ No	☐☐☐☐
Abdominal/intestinal disorder	☐ Yes ☐ No	☐☐☐☐
Metabolism disorder	☐ Yes ☐ No	☐☐☐☐
Other: ………………	☐ Yes ☐ No	☐☐☐☐

15. How old were you when you menstruated for the first time? State age in years: ☐☐

16. Have you had pregnancy diabetes during a previous pregnancy?
If yes - which pregnancy? In which pregnancy week were you diagnosed? Did you use insulin?

	Pregnancy week	Insulin
1st pregnancy	☐☐	☐ Yes ☐ No
2nd pregnancy	☐☐	☐ Yes ☐ No
3rd pregnancy	☐☐	☐ Yes ☐ No
4th pregnancy	☐☐	☐ Yes ☐ No
5th pregnancy	☐☐	☐ Yes ☐ No
6th pregnancy	☐☐	☐ Yes ☐ No
7th pregnancy	☐☐	☐ Yes ☐ No
8th pregnancy	☐☐	☐ Yes ☐ No

17. Are there any inheritable diseases in the family?
☐ None I know of   ☐ Yes   If yes, tick the appropriate box/boxes:

☐ Cardio-vascular disease   ☐ Diabetes
☐ Cancer   ☐ Neurological disease
☐ Mental illness   ☐ Arthritis
☐ Muscular disorder   ☐ Other   If other, state:………………..

18. Are you and the father of the child related?
☐ Yes ☐ No

If yes, is the father of the child your:

□ Cousin   □ 3rd cousin   □ 4th cousin   □ Uncle   □ Nephew   □ Other

19. Have you ever smoked/used snus?

Smoked: □ Never   □ Sometimes   □ Yes, daily

Snus: □ Never   □ Sometimes   □ Yes, daily

If the answer is never to both, go to question 23.

20. Did you smoke/use snus during the last 3 months before this pregnancy?

Smoking: Snus:

□ Never   Number of cigarettes/daily   □ Never

□ Yes, sometimes   □□   □ Yes, sometimes

□ Yes, daily   □□   □ Yes, daily

21. Do you smoke/use snus now?

Smoking: Snus:

□ Never   Number of cigarettes/daily   □ Never

□ Yes, sometimes   □□   □ Yes, sometimes

□ Yes, daily   □□   □ Yes, daily

22. How old were you when you started to smoke? State age: □□

If you have smoked previously, but do not smoke now, how old were you when you quit?

State age: □□

23. Your alcohol consumption:

Last 3 months before pregnancy:

□ Never   □ Sometimes   □ Yes, daily   Amount of alcohol units, normally: □□

Now: □ Never   □ Sometimes   □ Yes, daily   Amount of alcohol units, normally □□

(Number of alcohol units – 1 unit is: 1 glass of wine, 0.33 litres of beer, 1 glass of liquor)

24. Last menstruation's 1st day of bleeding:

Date:….. ….

25. Term before ultrasound:

Date:……….   □ Certain □ Uncertain

26. Estimate your weight in kilos:

Right before you became pregnant: □□□   25 years old: □□□   18 years old: □□□

27. Estimate your highest and lowest weight (in kilos), not including pregnancies, after you turned 18 years of age.

Highest: ☐☐☐   Lowest: ☐☐☐

Comment if the difference as greater than 20 kilos …………………………………

THANKS FOR TAKING THE TIME TO ANSWER THESE QUESTIONS!

Case Record FORM 1.2

31. If you are in paid employment – how large a percentage of fulltime employment did you have during the last three months before you became pregnant? What percentage do you have now?
(Applies regardless of any sick leave)

Before pregnancy: □□□ %   Now: □□□ %

32. If you are in paid employment – are you currently absent from your normal job?

□ Yes   □ No   □ Partly

33. (If your answer to question 32 was "Yes" or "Partly") **What is the reason for your absence?**

□ Sick leave   □ Leave   □ Sick child   □ Other

34. If you are in paid employment – have you been on sick leave for more than two weeks during this pregnancy?

	Full sick leave:	Partial sick leave:

If yes, state the approx. number of weeks: □□   If yes, state the approx. number of weeks: □□

36. Think back to when you were 10 years old. What occupation did your mother/father have?
MOTHER…………….   FATHER…………..

37. Think back to when you were 10 years old. How many rooms did your flat/dwelling have?

(Don't count kitchen and bathroom).   State number of rooms: □□

How many people lived in the flat/dwelling?   State number of people: □□

Did your mother/father/guardian own a car?   □ Yes   □ No

38. How old was your mother when you were born?   □□ years of age

39. How many brothers and sisters (siblings) do you have?   □□ (With the same mother)

40. Which number were you among your siblings?   □□ (With the same mother)

Any half-siblings? State number, if any □□

41. How long have you lived in: (State the number of years)

The city district you currently live in: □□   Oslo: □□

42. Where did you live for most of the time before you turned 16 years of age?

□ In the same city district as now □ In another city district/area of Oslo □ In another county in Norway

□ Outside Norway

State any previous city districts:……………..

If outside Norway: ☐ In own country of origin ☐ Other

43. Who do you share your household with?

☐ Spouse/cohabitant ☐ Parents ☐ Parents-in-law ☐ Child/children ☐ No one

☐ Other(s), describe:……………

44. How many persons are there in your household? Count yourself as well

Number of persons 18 or older: ☐☐ Number of persons 12-17 years of age: ☐☐

Number of persons 6-11 years of age: ☐☐ Number of persons under 6 years of age: ☐☐

45. How many rooms are there (don't count kitchen and bathroom) in the flat/dwelling where you live? State number of rooms: ☐☐

Type of dwelling:

☐ Flat in a block of flats/house with several housing units, e.g. quadruplex (four units)

☐ Terrace/row house

☐ Detached house ☐ Other

Do you own or rent your dwelling? ☐ Own ☐ Rent

46. If you are a first generation immigrant: How long have you lived in Norway?

State number of years: ☐☐

47. Are you the descendant of immigrant parents/parents who were not born in Norway?

☐ Yes ☐ No

If yes:

☐ Born in Norway, but both parents born abroad

☐ Born abroad with one parent born in Norway

☐ Born in Norway with one parent born abroad

☐ Born abroad of foreign-national parents

If you were born in Norway, with both parents born abroad, state the country of origin of your parents:
Country of origin for: your mother:……………… your father:………………

48. On what grounds did you come to Norway?

☐ Work

☐ Married a Norwegian

☐ Family reunification

☐ Refugee

☐ Residence on humanitarian grounds

☐ Other

49. How often in the course of the last year have you:

Read a newspaper in your own language/parents'

native language: ☐ Daily ☐ Weekly ☐ Less than weekly ☐ Never

Been visited by at least one Norwegian:

Read a Norwegian newspaper/watched
Norwegian TV:

Received help/support from at least one
Norwegian:

Participated in a meeting arranged by your
own/parents' countrymen:

50. Have you here in Norway experienced being denied a chance to rent or buy a dwelling because of your immigrant background?

☐ Yes, definitely ☐ Yes, I suspect so ☐ No ☐ Don't know

51. During the last five years in Norway have you experienced being denied a job you applied for due to your immigrant background?

☐ Yes, definitely ☐ Yes, I suspect so ☐ No ☐ Don't know

52. What was your state of health the last three months before your pregnancy?

☐ Poor ☐ Not too good ☐ Good ☐ Very good

53. Was this pregnancy planned?

☐ Yes ☐ No ☐ Partially Any comments:………………

54. If planned, how long have you been trying to get pregnant? State number of months: ☐☐

55. Have you had any pain in any of the following parts of your body during your pregnancy?

	☐ No pain	☐ Some pain	☐ Much pain
In the lower back <u>not</u> radiating to the leg(s)	☐ No pain	☐ Some pain	☐ Much pain
In the lower back <u>with</u> it radiating to the leg(s)	☐	☐	☐
In the front of the pelvic bone, over the pubic bone (symphysis)	☐	☐	☐
Back, over <u>one</u> pelvic joint	☐	☐	☐
Back, over <u>both</u> pelvic joints	☐	☐	☐
Front and back of <u>one side</u> of the pelvic bone	☐	☐	☐
Front and back of <u>both sides of the</u> pelvic bone	☐	☐	☐

56. Think back over the last 14 days. Have you taken cod-liver oil/cod-liver oil capsules/pills (*tran*) and/or other dietary supplements during this time? If yes, state the number of capsules/pills/spoons per day and the correct frequency.

Cod-liver oil/Cod-liver oil capsules: ☐ Never   ☐ <Once a week   ☐ 1-2 times a week   ☐ 3-4 times a week   ☐ 5-6 times a week   ☐ Every day
Fish oil capsules:
Seal oil capsules:
Folate (vitamin B):
Iron supplement:
Multi-vitamins with minerals (e.g. *Vitamineral, Kostpluss, Solaray Spektro* etc.):
Multi-vitamins without minerals: (e.g. *Sanasol, BioVit, Vitaplex* etc.)
Other dietary supplement:

State the name of the dietary supplement:…………….
State the name of any iron supplements:………………

57. Have you taken medication regularly, including birth-control, the last three months before your pregnancy?
State the name of the medication…………. – and the illness/disorder, if any…………………..

☐ The pill   ☐ Mini-pill   ☐ IUD/coil   Brand/name:…………….

58. Have you taken medication regularly during this pregnancy?
State the name of the medication…………. – and the illness/disorder, if any…………………..

59. Have you experienced any of the following events or problems in your life during the last six months?

You have been stricken with a serious illness, been injured or assaulted   ☐ Yes ☐ No

One of your closest family members (mother or father, spouse/cohabitant, children or brothers/sisters) has been seriously ill, injured or the victim of an assault   ☐ Yes ☐ No

One of your closest family members (mother or father, spouse/cohabitant, children or brothers/sisters) has died   ☐ Yes ☐ No

You have separated/divorced, or have broken off a long-term relationship   ☐ Yes ☐ No

You have had problems/major concerns about your children (upbringing, school, discipline)
          ☐ Yes ☐ No

You have become unemployed or been searching in vain for a job for more than one month
          ☐ Yes   ☐ No

You have experienced other difficult circumstances, e.g. a serious problem with a close friend, neighbour, relative or partner, serious financial concerns, something you valued dearly has been lost or stolen, death of someone close to you, or have major problems at work

☐ Yes   ☐ No

ANY IMPORTANT SUPPLEMENTAL COMMENTS ON YOUR ANSWERS TO THE QUESTIONS:

Question number: ☐☐   Comment……………………
You can also add more detailed comments here: ………………………………

THANKS FOR TAKING THE TIME TO ANSWER THESE QUESTIONS!

FORM CRF 2

1. What is your current marital status?
□ Married □ Partnership □ Cohabitant □ Single □ Divorced/separated □ Widow □ Other

2. Term based on ultrasound:
Date: □□.□□.□□□□ (day/month/year) □ Certain □ Uncertain

3. If you are in paid employment – are you currently absent from your normal job?
□ Yes □ No □ Partly

4. (If your answer to question 32 was "Yes" or "Partly") **What is the reason for your absence?**
□ Sick leave □ Leave □ Sick child □ Other

5. If you are in paid employment – have you been on sick leave for more than two weeks during this pregnancy?

Full sick leave: Partial sick leave:

If yes, state the approx. number of weeks: □□   If yes, state the approx. number of weeks: □□

6. What is your current state of health?
□ Poor □ Not too good □ Good □ Very good

7. Have you had any pain in any of the following parts of your body during your pregnancy?

	No pain	Some pain	Much pain
In the lower back <u>not</u> radiating to the leg(s)	□	□	□
In the lower back <u>with</u> it radiating to the leg(s)	□	□	□
In the front of the pelvic bone, over the pubic bone (symphysis)	□	□	□
Back, over <u>one</u> pelvic joint	□	□	□
Back, over <u>both</u> pelvic joints	□	□	□
Front and back of <u>one side</u> of the pelvic bone	□	□	□
Front and back of <u>both sides of the</u> pelvic bone	□	□	□

8. Have you had any of the following illnesses since you joined the project?

Diabetes type 1	□ Yes	□ No
Diabetes type 2	□ Yes	□ No

Metabolism disorder	☐ Yes ☐ No
Asthma	☐ Yes ☐ No
Allergy	☐ Yes ☐ No
Repeated urinary tract infections	☐ Yes ☐ No
Chronic liver disease	☐ Yes ☐ No
Prolonged high blood pressure	☐ Yes ☐ No
Arthritis/Bechterew's disease	☐ Yes ☐ No
Heart disease	☐ Yes ☐ No
Epilepsy	☐ Yes ☐ No
Abdominal/intestinal disorder	☐ Yes ☐ No

Have you had acute
morning sickness with vomitting
(*hyperemesis gravidarum*)   ☐ Yes ☐ No   If yes, from week: ☐☐ to week: ☐☐

Other: ………………   ☐ Yes ☐ No

9. Does one or more of <u>your</u> immediate relatives (mother, father, siblings, children) have diabetes?
Tick as many boxes as necessary

Mother	☐ No	☐ Yes, type 1 diabetes	☐ Yes, type 2 diabetes	☐ Yes, diabetes (unknown type)
Father	☐ No	☐ Yes, type 1 diabetes	☐ Yes, type 2 diabetes	☐ Yes, diabetes (unknown type)
Own sibling	☐ No	☐ Yes, type 1 diabetes	☐ Yes, type 2 diabetes	☐ Yes, diabetes (unknown type)
Own child	☐ No	☐ Yes, type 1 diabetes	☐ Yes, type 2 diabetes	☐ Yes, diabetes (unknown type)

_____

10. Do you know if one or more of <u>your</u> immediate relatives (mother, father, siblings) have had cardiovascular disease (heart attack, angina, stroke) before they were 55 years of age, for men, and 65 years of age, for women? *Tick as many boxes as necessary*

Mother	☐ No	☐ Yes	Comments:……………………………………..
Father	☐ No	☐ Yes	Comments:……………………………………..
Own sibling	☐ No	☐ Yes	Comments:……………………………………..

Eating disorders

11. Hvilken av følgende påstander passer best på deg?
☐ Vekt eller kroppsform påvirker ikke i det hele tatt hva jeg synes om meg selv

☐ Vekt eller kroppsform betyr noe for hva jeg synes om meg selv

☐ Vekt eller kroppsform betyr en del for hva jeg synes om meg selv

☐ Vekt eller kroppsform betyr mye for hva jeg synes om meg selv

☐ Vekt eller kroppsform betyr alt for hva jeg synes om meg selv

12. Har du noen gang brukt noen av følgende metoder for å kontrollere vekten?

Fremkalle brekninger for å kaste opp   ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Ta avføringsmidler   ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Trene mer enn to timer per dag   ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Faste eller ikke spise i 24 timer eller mer ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Hvis aldri - gå til sp. 14

13. I dag, bruker du noen av følgende metoder for å kontrollere vekten?

Fremkalle brekninger for å kaste opp   ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Ta avføringsmidler   ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Trene mer enn to timer per dag   ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

Faste eller ikke spise i 24 timer eller mer ☐ Aldri   ☐ En eller to ganger ☐ Ukentlig ☐ Daglig

14. Har du noen gang hatt perioder med overspising, dvs anfall der du har spist store mengder mat i løpet av en kort tid?   ☐ Ja   ☐ Nei

Hvis nei - gå til sp.25

15. Hvis ja, følte du da at du ikke kunne kontrollere spisingen?

☐ Ikke i det hele tatt   ☐ Litt   ☐ Noe   ☐ Mye   ☐ Veldig mye

16. Når du hadde flest episoder med overspising, hvor mange ganger per måned skjedde dette?   ☐☐☐

17. Hvor lenge varte perioden med overspising?

☐ Mindre enn en måned   ☐ 1-2 måneder   ☐ 3-5 mnd   ☐ 6-12 mnd ☐ Lengre enn et år

18. Førte episodene med overspising til at du ble opprørt eller ulykkelig?

☐ Ikke i det hele tatt   ☐ Litt   ☐ Noe   ☐ Mye   ☐ Veldig mye

19. Brukte du noen av metodene nedenfor samtidig som du overspiste?

Fremkalle brekninger for å kaste opp   ☐ Aldri   ☐ En eller to ganger   ☐ Ukentlig   ☐ Daglig

Ta avføringsmidler   ☐ Aldri   ☐ En eller to ganger   ☐ Ukentlig   ☐ Daglig

Trene mer enn to timer per dag   ☐ Aldri   ☐ En eller to ganger   ☐ Ukentlig   ☐ Daglig

Faste eller ikke spise i 24 timer eller mer   ☐ Aldri   ☐ En eller to ganger   ☐ Ukentlig ☐ Daglig

20. I dag, hender det du har perioder med overspising, dvs anfall der du har spist store mengder mat i løpet av kort tid? *Hvis nei - gå til sp.25*

☐ Ja   ☐ Nei

21. Hvis ja, føler du da at du ikke kan kontrollere spisingen?

☐ Ikke i det hele tatt   ☐Litt   ☐Noe   ☐ Mye   ☐ Veldig mye

22. Hvor mange ganger per måned skjer dette?   ☐☐☐

23. Hvor lenge har perioden med overspising vart?

☐ Mindre enn en måned   ☐ 1-2 måneder   ☐ 3-5 mnd   ☐ 6-12 mnd   ☐ Lengre enn et år

24. Fører episodene med overspising til at du blir opprørt eller ulykkelig?

☐ Ikke i det hele tatt   ☐ Litt   ☐ Noe   ☐ Mye   ☐ Veldig mye

25. Spiser du mer når du er engstelig, stresset eller opprørt?

☐ Alltid   ☐ Ofte   ☐ Noen ganger   ☐ Nei, jeg spiser heller mindre

Antepartum depression (depression during pregnancy)

26. In the past seven days I have been able to laugh and see the funny side of things

☐ As much as I alawys could

☐ Rather less than I used to

☐ Definitely not so much now

☐ Not at all

27. In the past seven days I have looked forward with enjoyment to things

☐ As much as I ever did

☐ Rather less than I used to

☐ Definitely less than I used to

☐ Not at all

28. In the past seven days I have blamed myself unnecessarily when things went wrong

☐ Yes, most of the time

☐ Yes, some of the time

☐ Not very often

☐ No, never

29. In the past seven days I have been anxious or worried for no good reason

☐ No, not at all

☐ Hardly ever

☐ Yes, sometimes

☐ Yes, very often

30. In the past seven days I have felt scared or panicky for no good reason

☐ Yes, quite a lot

☐ Yes, sometimes

☐ No, not much

☐ No, not at all

31. In the past seven days things have been getting on top of me

☐ Yes, most of the time I haven't been able to cope at all

☐ Yes, sometimes I haven't been coping as well as usual

☐ No, most of the time I have coped quite well

☐ No, I have been coping as well as ever

32. In the past seven days I have been so unhappy that I have had difficulty sleeping

☐ No, not at all

☐ Not very often

☐ Yes, sometimes

☐ Yes, most of the time

33. In the past seven days I have felt sad or miserable

☐ Yes, most of the time

☐ Yes, quite often

☐ Not very often

☐ No, not at all

34. In the past seven days I have been so unhappy that I have been crying

☐ Yes, most of the time

☐ Yes, quite often

☐ Only occacionally

☐ No, never

35. In the past seven days the thought of harming myself has occurred to me

☐ Yes, quite often

☐ Sometimes

☐ Hardly ever

☐ Never

Urinary incontinence (involuntary leakage of urine)

36. How often do you leak urine?

☐ Never

☐ Around once a week or even less

☐ 2-3 times a week

☐ Approximately once a day

☐ Several times a day

☐ All the time

37. We would like to know how <u>much</u> urine you leak. How much urine do you <u>usually</u> leak (whether you use protection or not)?

☐ Nothing at all

☐ A little bit

☐ A moderate amount

☐ A large amount

38. How much does the urinary incontinence affect your day-to-day life? Here we use a scale from 0-10.

Not at all   Very much

☐ 0   ☐ 1   ☐ 2   ☐ 3   ☐ 4   ☐ 5   ☐ 6   ☐ 7   ☐ 8   ☐ 9   ☐ 10

39. When do you leak urine? Tick as many boxes as necessary

☐ Never, I don't leak urine

☐ I leak before I get to the toilet

☐ I leak when I cough or sneeze

☐ I leak when I'm sleeping

☐ I leak when I'm physically active/exercising

☐ I leak when I am finished passing water and I have got my clothes back on

☐ I leak without any apparent reason

☐ I leak the whole time

40. Do you smoke/use snus now?

Smoking:		Snus:
☐ Never	Number of cigarettes/daily	☐ Never
☐ Yes, sometimes	☐☐	☐ Yes, sometimes
☐ Yes, daily	☐☐	☐ Yes, daily

41. Your alcohol consumption:

☐ Never   ☐ Sometimes   ☐ Yes, daily   Amount of alcohol units, normally: ☐☐

(Number of alcohol units – 1 unit is: 1 glass of wine, 0.33 litres of beer, 1 glass of liquor)

42. Have you experienced any of the following events or problems in your life <u>since you joined the project</u>?

You have been stricken with a serious illness, been injured or assaulted   ☐ Yes ☐ No

One of your closest family members (mother or father, spouse/cohabitant, children or brothers/sisters) has been seriously ill, injured or the victim of an assault   ☐ Yes ☐ No

One of your closest family members (mother or father, spouse/cohabitant, children or brothers/sisters) has died   ☐ Yes ☐ No

You have separated/divorced, or have broken off a long-term relationship   ☐ Yes ☐ No

You have had problems/major concerns about your children (upbringing, school, discipline)
          ☐ Yes ☐ No

You have become unemployed or been searching in vain for a job for more than one month

        □ Yes   □ No

You have experienced other difficult circumstances, e.g. a serious problem with a close friend, neighbour, relative or partner, serious financial concerns, something you valued dearly has been lost or stolen, death of someone close to you, or have major problems at work

        □ Yes   □ No

43. Think back over the last 14 days. Have you taken cod-liver oil/cod-liver oil capsules/pills (*tran*) and/or other dietary supplements during this time? If yes, state the number of capsules/pills/spoons per day and the correct frequency.

Cod-liver oil/Cod-liver oil capsules: □ Never □ <Once a week □ 1-2 times a week □ 3-4 times a week □ 5-6 times a week □ Every day

Fish oil capsules:

Seal oil capsules:

Folate (vitamin B):

Iron supplement:

Multi-vitamins with minerals (e.g. *Vitamineral, Kostpluss, Solaray Spektro* etc.):

Multi-vitamins without minerals: (e.g. *Sanasol, BioVit, Vitaplex* etc.)

Other dietary supplement:

State the name of the dietary supplement:…………….

State the name of any iron supplements:……………….

44. Have you taken medication regularly the last three months?

State the name of the medication…………. – and the illness/disorder, if any………………….. *

THANKS FOR TAKING THE TIME TO ANSWER THESE QUESTIONS!

PHYSICAL ACTIVITY – FORM NO. 1

Information for the interviewer:
The aim of this interview questionnaire is to ascertain the physical activity of the woman before her pregnancy and during the pregnancy, and to ascertain what her attitude is to physical activity. The physical activity shall also be registered objectively with the armband, preferably the week after this interview. Most of the questions refer to the woman's subjective understanding. But the aim of questions 3-5 is to form a picture of her activity level, to find out, among other things, if she is as active as the health authorities recommend (question 6).

Physical activity means:
1. Physical activity in day-to-day life (at work, leisure time and in the home, and how one gets to and from work and leisure activities)
2. Planned exercise activities (such as going for walks, swimming, dancing etc.)
3. Exercising (to improve your physical shape, strengthen muscles and improve other skills)
Text in italics is information for the interviewer and is not to be read to the woman being interviewed.

---

1. How would you rate your physical activity level at present?

☐ Low   ☐ Fairly low   ☐ Average   ☐ Quite high   ☐ High

---

2. Think back over the last three months <u>before this pregnancy</u>. What physical shape were you in compared to other women of your age? Think, for example, about your capacity when you walked up stairs or hills.

☐ Much worse   ☐ A little worse   ☐ The same as other women of my age

☐ A little better   ☐ Much better

---

3. How often were you physically active during the last three months <u>before this pregnancy</u>?
We are especially interested in activity that is moderate (e.g. brisk walking) or more intense. Bicycling or walking to work, and walking on the job can be included if at least 10 minutes' duration each time.

Time used (minutes):

☐ Never   ☐ 1-3 x /month   ☐ 1 x per week   ☐ 2 x per week   ☐ 3-6 x per week   ☐ Daily   ☐☐☐

Run/jog/orienteering
Bicycling
Fitness centre/weight-lifting
Aerobics
Dance (jazz, swing, rock etc)
Ball sports/netball
Swimming
Brisk walking/hiking/skiing
Strolling
Other
If other, what?.......

_____

4. How often have you been physically active <u>the last 7 days</u>?

This question will be used with question 5 to assess if the woman is as active as the health authorities recommend (question 6.) For the activity to be taken into consideration, it must be of moderate (e.g. brisk walking) or hard intensity. The last type of activity (strolling/walking) does not have a high enough intensity to be included, but any activity is better than nothing at all, not least in terms of energy use. Bicycling or walking to work, and walking on the job can be included if of at least 10 minutes' duration each time.

Time used (minutes):

□ Never   □ 1 x per week   □ 2 x per week   □ 3-6x per week □ Daily   □□□

Run/jog/orienteering
Bicycling
Fitness centre/weight-lifting
Aerobics
Dance (jazz, swing, rock etc.)
Ball sports/netball
Swimming
<u>Brisk walking</u>/hiking/skiing
<u>Strolling</u>
Other
If other, what?......

_____

5. If you think back over <u>the last month</u>, was you leisure-time activity level during the last 7 days:

□ Much less than usual   □ A little less than usual   □ The usual   □ A little more than usual

□ Much more than usual

_____

Now we will use your answers to questions 4 and 5 to see if it can be said that you are physically active on a regular basis as we define it here. In this case, some of your activities must be of moderate intensity, as for example brisk walking.

If you have answered "Much less than usual" or "Much more than usual" over the last week, we will ask you to use the activity level from the previous month as the basis for your answers below.

6. Think about your physical activity during this pregnancy. Do you practise:

Moderately intensive activity for 30 minutes at least 5 days of the week?   □ Yes □ No

Moderately intensive activity in total at least 2.5 hours/week over at least 3 days?   □ Yes □ No

Hard activity (e.g. jogging) at least 20 minutes 3 times a week?   □ Yes □ No

Activity of both hard and moderate intensity (e.g. hard activity once a week

and moderately intensive activity twice a week)   □ Yes □ No

_____

If the woman answers "no" to all four alternatives, go to question 7 and let her find the one of the three alternatives that fits her best.

If the woman answers "yes" to at least one of the four alternatives, go to question 8 and let her find the alternative that fits her best.

7. Think about yourself during this pregnancy. To count yourself as regularly physically active, you must have answered yes to at least one of the alternatives under question 6.

☐ I am not regularly physically active (at least moderate intensity) and have no plans for being so

☐ I am not regularly physically active (at least moderate intensity) but I am considering a change

☐ I am somewhat physically active (at least moderate intensity), but less than stated under question 6

8. To be filled in if the woman has answered "yes" to one or more of the alternatives in item 6.

☐ I am regularly physically active, but have been so for less than 6 months

☐ I am regularly physically active and have been so for more than 6 months

If the woman answers "yes" to the first of these two alternatives, go to question 10

9. How long have you been regularly physically active?

☐ Under 1 year   ☐ 1-5 years   ☐ 6-10 years ☐ More than 10 years

10. Have you changed your physical activity level <u>after you became pregnant</u>?

☐ Less active now   ☐ Unchanged   ☐ More active now

11. If you are less active now than before you became pregnant – what is the main reason/reasons for this?
Let the woman answer the question before you present the categories below. Tick up to three boxes

Pregnancy related <u>disorders</u> (fatigue/drowsy, nauseous)..................   ☐ Yes ☐ No

<u>Pain</u> which increases with physical activity.....................................   ☐ Yes ☐ No

New <u>illness</u> connected to the pregnancy...   ☐ Yes ☐ No
Other health problems you have...
Have been advised by friends/family to be less physically active

during your pregnancy..   ☐ Yes ☐ No
Have been advised by health care staff to be less physically active

during your pregnancy...   ☐ Yes ☐ No

Worried about the baby......................................   ☐ Yes ☐ No

Don't have time..   ☐ Yes ☐ No

Other...   ☐ Yes ☐ No

Now I am going to read a number of statements for which I want you to indicate the degree to which you agree with them. We use scales with 3 to 7 points.

The first scale has 7 points ranging from "Not at all" to "Very sure".

12. Think about how things are for you now. Think about all the types of activity. Decide how you would answer each statement: I'm sure that I can carry out the planned physical activity even if:

	Not at all				Very sure		
I am tired	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
I feel depressed	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
I'm worried	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
I'm angry because of something	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
I feel stressed	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7

This scale also has 7 points ranging from "Totally agree" to "Totally disagree".

13. Think about how things are for you now. Think about all the types of activity. For each statement, state the degree to which you agree/disagree.

	Totally agree					Totally disagree	
Whether I am regularly physically active or not, is entirely up to me	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
If I want to, I would have no problems being regularly physically active	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
I would have liked to have been regularly physically active, but I'm not really sure if I can manage	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
I have full control over being regularly physically active	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7
Being regularly physically active is difficult for me	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6	□ 7

Now the scale has 5 points ranging from "Does not fit well" to "Fits well"

14. Think about how things are for you now. To what degree do these statements describe you as a person?

	Does not fit well				Fits well
I see myself as a person who is concerned about being physically active	□ 1	□ 2	□ 3	□ 4	□ 5
I think of myself as a person who is concerned about keeping in good physical shape	□ 1	□ 2	□ 3	□ 4	□ 5
Being physically active is an important part of who I am	□ 1	□ 2	□ 3	□ 4	□ 5

Now the scale has 3 points ranging from "Great effect" to "No effect"

15. To what degree do you feel that daily physical activity can have a beneficial effect in preventing the following illnesses?

If the woman has problems answering this, you can add: If you think this is difficult to answer, you can answer "Don't know".

	□ Great effect	□ Little effect	□ No effect	□ Don't know
Cardio-vascular illnesses				
Muscular/skeletal disorders				
Diabetes type 2				
Cancer				
High blood pressure				
Mental disorders				
Being overweight/obese				
Abdominal/intestinal illnesses				
Asthma and allergies				

In the next statements the scale has 5 points ranging from "Never" to "Very often".
First there are 6 statements about your family, and then 6 statements about your friends.

16. Think about how things are for you now. Have your family (members of your household):

If the woman has problems answering this, you can add: If you think this is difficult to answer, you can answer "Does not fit well".

	□ Never	□ Seldom	□ A few times	□ Often	□ Very often	□ Does not fit well

1. Encouraged you to be physically active?

2. Discussed physical activity with you?

1

3. Changed their plans so that you could take part in physical activity together?

4. Taken over chores for you, so that you have more time to be physically active?

5. Said that physical activity would be good for you health?

6. Talked about how much they like being physically active?

17. Think about how things are for you now. Have your friends/acquaintances/family members outside the household:

If the woman has problems answering this, you can add: If you think this is difficult to answer, you can answer "Does not fit ".

□ Never □ Seldom □ A few times □ Often □ Very often □ Does not fit

1. Suggested that you should take part in physical
   activity together?

2. Encouraged you to be physically active?

3. Given you such helpful reminders about physical
activity as: "Shall we go for a walk tonight?"

4. Changed their plans so that you could take part in
   physical activity together?

5. Said that physical activity would be good
for your health?

6. Talked about much they like
being physically active?

_____
Here the scale has 4 points ranging from "Totally disagree" to "Totally agree"

18. Think about how things are for you now. People who are important to me:

□ Totally disagree □ Slightly disagree □ Slightly agree □ Totally agree

Think I should be physically active
Think it is good if I'm physically active
Want me to be physically active
Think it improper that I'm physically active
Do not like that I'm physically active

_____
19. Here the scale has 5 points ranging from "None" to "Everybody"

Of people you know well – how many are physically active at least 3
times a week? □ None □ A few □ Quite a few □ Just about everybody □ Everybody

Of people your age who you know
well – how many are physically active
at least 3 times a week? □ None □ A few □ Quite a few □ Just about everybody □ Everybody

Of women your age who you know
well – how many are physically active
at least 3 times a week? □ None □ A few □ Quite a few □ Just about everybody □ Everybody

_____

20. How often do you see adults in your neighbourhood in one or another form of physical activity?

☐ Very often ☐ Often ☐ Sometimes ☐ Seldom ☐ Never

_____

21. How often do you see other women your age in your neighbourhood in one or another form of physical activity?

☐ Very often ☐ Often ☐ Sometimes ☐ Seldom ☐ Never

_____

22. How many times a week does your spouse/cohabitant/the child's father take part in a physical activity these days?

☐ More than 3 times a week ☐ 1-3 t/week ☐ 1-3 t/month ☐ Less often ☐ Don't know

_____

23. About how long would it take you to walk from home to:

☐ 1-5 min ☐ 6-10 min ☐ 11-20 min ☐ 21-30 min ☐ > 30 min ☐ Don't know

The grocer's
A recreational area, park or walking/hiking path
Fitness centre, swimming pool

_____

24. Do you find the following in your neighbourhood:

☐ Totally disagree ☐ Slightly disagree ☐ Slightly agree ☐ Totally agree

Safe places to walk (park, recreational area, hiking path, pavement) which is adequately lit

Many places where you can be physically active (outdoor areas, swimming pool etc.)

Several exercise and physical-activity programmes (which could interest you)

Easy to walk to shops (10-15 minutes to walk, pavement along most of the streets)

Easy access to walking or bicycle paths

So much traffic in the streets that it is difficult or unpleasant to walk there

Pedestrian crossings and traffic lights that make it easier to cross the streets

_____

25. Do you have a bicycle you can use? ☐ Yes ☐ No

_____

26. Do you/the child's father own a car? ☐ Yes ☐ No

Are you used to bicycling? ☐ Yes ☐ No

_____

27. My municipality/city district does not do enough to promote physical activity

☐ Totally disagree ☐ Slightly disagree ☐ Slightly agree ☐ Totally agree

_____

THANKS FOR TAKING THE TIME TO ANSWER THESE QUESTIONS!

Initialer intervjuer: ☐☐☐☐

Svangerskapsuke: ☐☐ _____

Undersøkelsesdato: ☐ ☐ ☐ ☐ ☐ ☐

Uker etter fødsel: ☐☐ _____

Kvinnens fødselsdato: ☐ ☐ ☐ ☐ ☐ ☐

Us bydel:

Intervjuers kode: ☐☐ _____

[engelsk – kosthold]

STORK Groruddalen

DIET

1. Think back over the last 14 days. Now I'm going to ask you some questions about what you have drunk or poured on your cereal (such as cornflakes, muesli etc.) during this time.
Example: Have you drunk any cola with sugar during this period? If yes, how often have you drunk this? How much did you drink each time?

Tick (X) for how often (frequency) and how much each time (in litres) where applicable.

	Have not drunk	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily	Amount per time 1/5l, 1/3 l, ½ l and 1l or over			
							[1/5 l]	[1/3 l]	[1/2 l]	1 l+
Cola with sugar	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other soda pop/fizzy drink with sugar	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sugar-free cola	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other sugar-free soda pop/fizzy drink	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Squash/juice and other drinks with sugar (including nectar)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sugar-free squash/juice and other drinks	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fruit juice	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Whole milk, kefir, sour milk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
2% milk, Cultura, Biola, chocolate milk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
0.7% milk (green)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Skimmed milk, skimmed sour milk, Biola berry	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Tea	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Coffeemaker coffee, instant coffee	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Coffee press, percolated coffee	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other coffee	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other drinks _____	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Comments _____

_____

2. If you drink tea or coffee, how many teaspoons of sugar and/or honey do you use per cup?

	Don't use sugar/honey	1 tsp	2 tsp	3 tsp	4 tsp	≥ 5 tsp
			Tick box			
How many tsp sugar/-honey in tea	☐	☐	☐	☐	☐	☐
How many tsp sugar/-honey in coffee	☐	☐	☐	☐	☐	☐

3. Think back over the last 14 days. How often have you eaten/used yoghurt (from cups, with cereal and/or in cooking)?

	Have not eaten	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Natural yoghurt	☐	☐	☐	☐	☐	☐
Greek/Turkish yoghurt	☐	☐	☐	☐	☐	☐
Yoghurt with fruit/berries	☐	☐	☐	☐	☐	☐
Light yoghurt with fruit/berries	☐	☐	☐	☐	☐	☐

4. Think back over the last 14 days. How often have you eaten fruit and/or berries?

	Never or < once a week	1-2 t/week	3-4 t/week	5-6 t/week	Once a day	2 t/day	≥ 3 t/day
Fresh fruit, berries, fruit-salad/fruit chart etc.	☐	☐	☐	☐	☐	☐	☐

5. Think back over the last 14 days. How often have you eaten vegetables?

	Never or < once a week	1-2 t/week	3-4 t/week	5-6 t/week	Once a day	2 t/day	≥ 3 t/day
Raw vegetables, mixed vegetables/vegetable chart, salads	☐	☐	☐	☐	☐	☐	☐
Fried/wok vegetables, boiled/steamed/baked vegetables, vegetables in stews (e.g. curry)	☐	☐	☐	☐	☐	☐	☐

6. **Think back over the last 14 days. How often have you eaten potatoes?**

	Never or < once a week	1-2 t/week	3-4 t/week	5-6 t/week	Once a day	2 t/day	≥ 3 t/day
Potato (boiled, baked, roasted in the oven)	☐	☐	☐	☐	☐	☐	☐
Gratinated potatoes	☐	☐	☐	☐	☐	☐	☐
French fries/chips (deep-fried, fast-food)	☐	☐	☐	☐	☐	☐	☐

7. **Think back over the last 14 days. How often have you eaten beans, lentils, peas, chickpeas etc?**

	Never or < once a week	1-2 t/week	3-4 t/week	5-6 t/week	Once a day	2 t/day	≥ 3 t/day
Baked beans, other beans, creamed peas/beans, dahl, lentil/pea soup, chickpeas, lentil cakes, falafel (etc.), hummus	☐	☐	☐	☐	☐	☐	☐
Other _____	☐	☐	☐	☐	☐	☐	☐

8. Think back over the last 14 days. How often have you eaten meat and/or sausage products (does not refer to sandwich meats)?

	Have not eaten	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Chicken, turkey, other lean meat such as trimmed roast beef, pork (fried, boiled, grilled, in stews etc.)	☐	☐	☐	☐	☐	☐
Dishes with lean minced beef, hamburger, hot dog/sausage meat of chicken/turkey, light hot dogs, chops trimmed of fat	☐	☐	☐	☐	☐	☐
Chops not trimmed of fat, lamb, hot dogs, fricadelles, kebab, other dishes with minced meat/other forcemeat products	☐	☐	☐	☐	☐	☐
Pizza, fast-food (purchased outside the home)	☐	☐	☐	☐	☐	☐
Other _____	☐	☐	☐	☐	☐	☐

9. **Think back over the last 14 days. How often have you eaten fish and/or fish products (does not refer to sandwich spread etc.)?**

	Have not eaten	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Lean fish (fillets, whole fish) of cod, saithe/pollock, flounder, haddock, tuna, other lean fish (fried, steamed, boiled, grilled etc.)	☐	☐	☐	☐	☐	☐

	Have not eaten	Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Fat fish such as salmon, trout, halibut, mackerel, herring, other fat fish (fried, steamed, boiled, grilled etc.)	☐	☐	☐	☐	☐	☐
Fish products (fish cakes, fish pudding etc.)	☐	☐	☐	☐	☐	☐
Fish fingers or similar products (deep-fried or fried)	☐	☐	☐	☐	☐	☐
Other _____	☐	☐	☐	☐	☐	☐

10. Think back over the last 14 days. How often have you eaten food that has been:

	Have not eaten	Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Pan-fried (with butter, margarine, oil etc.), fried in a wok/haandi	☐	☐	☐	☐	☐	☐
Deep-fried	☐	☐	☐	☐	☐	☐

11. Think back over the last 14 days.
    What type of fat have you used on bread?
    What type of fat have you used for frying?
    What type of fat have you used for deep-frying?
    What type of fat have you used for other types of cooking, for example baking?

After each question tick the box for one or more correct alternatives. First ask about fat used on bread, then for frying, deep- frying and other cooking. Use chart/pictures

	On bread	For frying	For deep-frying	For other types of cooking
Not used fat	☐	☐	☐	☐
Butter (dairy butter)	☐	☐	☐	☐
Melange (margarine), Bremyk	☐	☐	☐	☐
Brelett	☐	☐	☐	☐
Soft margarine (Soft Flora, Soft Ekstra, Soft Oliven, Vita, Soya etc.)	☐	☐	☐	☐
Light vegetable margarine (Soft light, Vita Lett etc.), ProVita/ProActiv (Becel)	☐	☐	☐	☐
Liquid margarine (Melange, Olivero, Vita, Bremyk)	☐	☐	☐	☐
Vegetable oils (sunflower/corn oil, soya oil, olive oil, rape-seed oil etc.)	☐	☐	☐	☐
Coconut/palm oil	☐	☐	☐	☐
Other margarine _____	☐	☐	☐	☐
Other oil _____	☐	☐	☐	☐
Ghee/nej/purified butter	☐	☐	☐	☐

12. Think back over the last 14 days. How often have you eaten the following food?

	Have not eaten	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
White bread (French bread, nan, breakfast crispbread etc.) and/or semi-whole-wheat bread (*kneipp*/rolls/buns/crispbread)	☐	☐	☐	☐	☐	☐
Whole-wheat and dark-brown bread/buns/crispbread, chapati	☐	☐	☐	☐	☐	☐
Cereals/muesli with no or little sugar added (oatmeal, four-grain etc) *Use chart/pictures*	☐	☐	☐	☐	☐	☐
Cereals/muesli with much sugar added *Use chart/pictures*	☐	☐	☐	☐	☐	☐
White rice, macaroni, pasta/spaghetti, couscous	☐	☐	☐	☐	☐	☐
Whole-wheat pasta or natural rice/long-grain rice/whole-grain rice, millet	☐	☐	☐	☐	☐	☐

13. Think back over the last 14 days. How often have you used the following on sandwiches?

	Did not use	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
White cheese (Norvegia, Gulost, Nøkkelost, smøreost) brown cheese, other fat cheese	☐	☐	☐	☐	☐	☐
Lighter/lean white cheese/cheese spread, light brown cheese, whey cheese spread, other light/lean cheese	☐	☐	☐	☐	☐	☐
Liver pâté, salami, saveloy/baloney, mutton sausage etc.	☐	☐	☐	☐	☐	☐
Liver pâté with less fat, liver pâté with "healthy" fat, ham, turkey slices, light saveloy/baloney	☐	☐	☐	☐	☐	☐
Fish (mackerel in tomato sauce, other mackerel, salmon, trout, sardines, anchovies, herring, caviar etc.)	☐	☐	☐	☐	☐	☐
Jam, marmalade	☐	☐	☐	☐	☐	☐
Light jam	☐	☐	☐	☐	☐	☐
Chocolate or nut spread, Sunda, syrup, honey etc.	☐	☐	☐	☐	☐	☐
Mayonnaise salads (Italian salad, shrimp salad etc.)	☐	☐	☐	☐	☐	☐
Egg	☐	☐	☐	☐	☐	☐
Other _____	☐	☐	☐	☐	☐	☐

14.   Think back over the last 14 days. How often have you eaten the following food?

	Have not eaten	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Cakes, angel-food cake, muffins, doughnuts, Danish pastries	☐	☐	☐	☐	☐	☐
Sweet cookies (cream cookies, chocolate cookies; Balerina, Bixit, wafer cookies, Mariekjeks, Kornmo (graham wafers) etc.)	☐	☐	☐	☐	☐	☐
Sweet buns, *skolebrød* (bun with custard and icing), other sweet yeast-baked products	☐	☐	☐	☐	☐	☐
Waffles, pancakes, sweet pancakes etc.	☐	☐	☐	☐	☐	☐
Chocolate/sweets/boiled sweets, snacks with sugar (Jell-O, Turkish delight)	☐	☐	☐	☐	☐	☐
Foreign sweet snacks (mithai, jalebi, halwa, zarda, la'du, baklava etc.)	☐	☐	☐	☐	☐	☐
Ice-cream	☐	☐	☐	☐	☐	☐
Desserts/pudding/creamed rice	☐	☐	☐	☐	☐	☐
Dried fruit	☐	☐	☐	☐	☐	☐
Other sweet food/snacks	☐	☐	☐	☐	☐	☐

15.   Think back over the last 14 days. How often have you eaten the following food?

	Have not eaten	< Once a week	1-2 t/week	3-4 t/week	5-6 t/week	Daily
Salt snacks (crisps/potato chips with various flavours, tortilla chips), other fattening snacks, Bombay mix etc.	☐	☐	☐	☐	☐	☐
"Light" snacks (pretzels, popcorn etc.)	☐	☐	☐	☐	☐	☐
Nuts	☐	☐	☐	☐	☐	☐

16.   Think back over the last 14 days. How often do you eat the following meals during one week?

Tick a box for each meal

	Never/ rarely	Once a week	Twice a week	Three times a week	Four times a week	Five times a week	Six times a week	Every day
Breakfast	☐	☐	☐	☐	☐	☐	☐	☐
Lunch	☐	☐	☐	☐	☐	☐	☐	☐
Dinner	☐	☐	☐	☐	☐	☐	☐	☐
Supper	☐	☐	☐	☐	☐	☐	☐	☐
Midnight snack	☐	☐	☐	☐	☐	☐	☐	☐

17. Think back over the last 14 days. How often do you eat or drink one or more of the following in-between meals during the course of the day?

	Rarely	Once a day	Twice a day	Three times a day	Four times a day	More than four times a day
Chocolate, sweets, snacks, soda pop/fizzy drink etc.	☐	☐	☐	☐	☐	☐
Fruit, slice of bread/crispbread etc.	☐	☐	☐	☐	☐	☐

18. Think back over the last 14 days. Have you had a special diet during this period?   ☐ No   ☐ Yes

If **yes**, what has been special about it?

_____

_____

_____

**19.   Have you changed your diet after you became pregnant/after you gave birth?
(cross out the alternative that does not fit)**   ☐ No   ☐ Yes

If **yes**, what changes have you made and when did you make these changes?

_____

_____

_____

20.   How would you describe your diet? (tick the box for the closest alternative)

My diet includes meat and fish ☐

I avoid meat, but eat fish ☐

I avoid fish, but eat meat ☐

I am a vegetarian and include milk products and eggs in my diet (ovo-lacto vegetarian) ☐

I am a vegetarian and include milk products but not eggs in my diet (lacto vegetarian) ☐

I am a vegetarian and exclude all milk products and eggs from my diet (vegan) ☐

THANKS FOR TAKING THE TIME TO ANSWER THESE QUESTIONS

Til deg som er gravid og bor i bydelene Stovner, Grorud og Bjerke

Forespørsel om å delta i forskningsprosjektet "STORK Groruddalen"

Hensikten med studien

Formålet er å videreutvikle svangerskaps- og fødselsomsorgen og helsestasjonstjenesten slik at vi bedre kan forebygge og behandle nye helseproblemer som overvekt, inaktivitet og diabetes. Kvinner med diabetes og deres barn har noe høyere risiko for komplikasjoner i svangerskapet. Noen ganger oppstår diabetes i svangerskapet. Selv om tilstanden vanligvis går over etter fødselen, er det økt risiko for å få type 2 diabetes senere.

Vi som arbeider på helsestasjonene i bydelene Stovner, Grorud og Bjerke, ønsker i samarbeid med universitetssykehusene i Osloområdet å kartlegge disse problemene, hvordan de påvirker helsetilstanden for mor og barn på kort og lang sikt, og finne årsakene til at svangerskapsdiabetes og type 2 diabetes øker. Aker universitetssykehus er ansvarlig for studien.

Hva innebærer studien?

Du vil på helsestasjonen få en ekstra og en utvidet kontroll i svangerskapet og en ekstra undersøkelse etter fødselen. Vi vil ta noen ekstra blod- og urinprøver og målinger, og be deg svare på spørsmål om din helse, fysiske aktivitet og kosthold. Du vil også få ekstra ultralyd-undersøkelser i svangerskapet. Det tas noen blodprøver fra barnets navlesnor og morkaken ved fødselen, og noen undersøkelser og blodprøver av barnet senere.

Hva skjer med prøvene og informasjonen om deg?

Dette skal kun brukes slik som beskrevet i hensikten med studien. Alle opplysningene og prøvene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. En kodeliste som knytter sammen studieresultatene og deltakernes navn, er kun tilgjengelig for autorisert helsepersonell knyttet til studien, og denne vil bli slettet ved studieslutt, senest i 2030. Det vil selvsagt ikke være mulig å identifisere deltakerne når resultatene av studien offentliggjøres. For å finne ut hvor mange som får diabetes og/eller hjerte- og karsykdom i fremtiden, ønsker vi å kunne hente ut disse opplysningene fra din fastlege og fra sykehus der du får behandling, og fra Norsk Diabetesregister for voksne, Norsk Pasientregister, Reseptregistret, Dødsårsaksregistertet og Medisinsk fødselsregister.

Frivillig deltakelse

Det er frivillig å delta i studien, og alle har rett til betenkningstid før man bestemmer seg. De som ikke ønsker å delta, trenger ikke å oppgi grunn, og det får ingen konsekvenser for den videre behandlingen. Hvis du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Du kan senere trekke tilbake samtykket uten at det påvirker behandlingen, ved å kontakte prosjektleder dr. med. Anne Karen Jenum (telefon 91 18 14 16).

Mer informasjon om studien finnes i *Kapittel A*
Mer informasjon om biobank, personvern økonomi og forsikring finnes i *Kapittel B*

Kapittel A- utdypende forklaring om hva studien innebærer

Alle gravide som går til svangerskapskontroll i bydelene Stovner, Grorud og Bjerke, vil bli invitert til å delta i studien. Hvis du sier ja til å delta, vil vi be deg møte til en ekstra undersøkelse på helsestasjonen omkring uke 12, en utvidet undersøkelse omkring uke 28 og en ekstra undersøkelse omkring 12 uker etter fødselen.

Alle gangene vil vi stille deg spørsmål om din helse og intervjue deg om kostholdet ditt og din fysiske aktivitet, og kartlegge din fysiske aktivitet med et armbånd som bæres på overarmen. Vi vil også stille noen spørsmål om helseforhold og sykdom i familien, veie deg med en spesialvekt som angir fettinnholdet i kroppen, og måle hudtykkelsen med en enkel ytre målemetode.

Alle disse tre gangene må du møte fastende på grunn av de ekstra blodprøvene som skal tas sammen med rutineprøvene i svangerskapet. Det betyr at du ikke skal spise, drikke eller røyke etter kl 24 kvelden før disse timene hos oss. Rutineprøvene og noen av de andre blodprøvene vil du få svar på ved neste undersøkelse. Andre blir sendt til sykehus for nedfrysing for senere analyser. Det gjelder også urinprøvene.

Omkring uke 28 vil vi ta en blodsukkerbelastning som gjøres på følgende måte: Etter den fastende blodprøven drikker du 75 gram druesukker. Etter 2 timer tas en ny blodprøve. Det er lurt å ta med matpakke som du kan spise etterpå. Prøvene analyseres på helsestasjonen. Du får med en gang vite om du har fått svangerskapsdiabetes. Da får du ekstra oppfølging/behandling.

Du vil også bli tilbudt 3 ekstra ultralydundersøkelser for å se på barnets vekst. Etter fødselen vil vi i samarbeide med sykehuset du føder på innhente journalopplysninger fra svangerskapet om resultatene av ultralydundersøkelsene, fødselsforløpet, din helse og barnets lengde, vekt, hodeomkrets, fordeling av kroppsfett og helsetilstand. Det tas også blodprøver fra barnets navlestreng og morkake ved fødselen.

Etter fødselen ønsker vi å kartlegge hvor lenge barnet får morsmelk, og hvordan barnet vokser (lengde og vektutvikling) i barnealderen. Dette skjer ved den vanlige barnekontrollen på helsestasjonen. Vi ønsker å kunne gjøre noen tilleggsundersøkelser av barnet ved 6 og 10 års alder (kost, fysisk aktivitet og blodprøver). *Kvinner som får påvist svangerskapsdiabetes* vil få utført ny blodsukkerbelastning ca.3 måneder etter fødselen. Vi vil senere også innkalle dem en gang i året i 5 år, og så hvert 5. år for nye prøver for å avklare om de har fått type 2 diabetes.

Ved oppfølgingsstudiene vil vi komme tilbake med ny henvendelse med spørsmål om å delta.

Mulige fordeler og ubehag/ulemper

- Økt kunnskap om fysisk aktivitet, sunn kost og helse.
- Ekstra nøye oppfølging av de som får påvist svangerskapsdiabetes
- Blodsukkerbelastningen (uke 28 og 3 mnd etter fødsel) kan utløse kvalme
- Ingen av de andre undersøkelsene gir ubehag eller risiko utover vanlig blodprøvetakning.
- Ved blodprøver av barnet ved 6 og 10 års alder vil barnet på forhånd kunne få lokalbedøvende salve på huden.

Kapittel B - Personvern, biobank, økonomi og forsikring

Personvern

Opplysninger som registreres om deg er journalopplysninger fra helsekort for gravide, sykehusjournal fra mors og barnets journal i forbindelse med svangerskap og fødsel hentet ut av autorisert helsepersonell, fra intervjuene om kost og fysisk aktivitet, og resultater fra de innsamlede målinger og blod- og urinprøver. For barnet ditt gjelder det opplysninger fra sykehuset fra fødsel og nyfødtperiode, og helsestasjonsdata om amming, vekstutvikling, samt tilleggsundersøkelsene om kosthold, fysisk aktivitet og blodprøver ved 6 og 10 års alder. For å finne mer ut om helsekonsekvensene av fysisk inaktivitet, overvekt og diabetes og senere helse for mor og barn, spesielt hvorfor noen får diabetes og hjerte- og karsykdom, ønsker vi å kunne hente ut opplysninger om disse diagnosene fra din journal hos fastlege og sykehus der du får behandling, og kunne koble opplysningene fra "STORK Groruddalen" med data fra Norsk Pasientregister, Norsk Diabetesregister for voksne, Reseptregisteret, Dødsårsaksregistertet og Medisinsk fødselsregister Alle som får innsyn, har taushetsplikt. Opplysninger om fars helse og forekomst av hjerte- og karsykdom og diabetes i familien vil også bli samlet inn. For at det skal være mulig å følge med din og barnets medisinske utvikling over lengre tid, slettes opplysninger og prøver først i 2030. Aker universitetssykehus ved administrerende direktør er databehandlingsansvarlig for studien.

Behandling av materiale og opplysninger hos andre

Hvis du sier ja til å delta i studien, gir du også ditt samtykke til at avidentifiserte opplysninger og prøver kan oppbevares og behandles hos ulike forskere og samarbeidspartnere tilknyttet prosjektet, i Norge og i utlandet. Dette er nødvendig for å oppfylle formålet med studien. Vi vil stille samme strenge krav til beskyttelse av informasjonen til våre samarbeidspartnere, også i land med lover som ikke gir like god personvernbeskyttelse som her.

Biobank

Blod- og urinprøvene og vev fra morkake som blir tatt og informasjonen utledet av dette materialet, vil bli lagret i en forskningsbiobank ved Aker universitetssykehus. Hvis du sier ja til å delta i studien, gir du også samtykke til at det biologiske materialet og analyseresultater inngår i biobanken. Prof. dr. med. Kåre Birkeland er ansvarlig for biobanken. Biobanken planlegges å vare til 2030. Etter dette vil materialet og opplysninger bli ødelagt etter interne retningslinjer.

Rett til innsyn og sletting av opplysninger om deg og sletting av prøver

Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har også rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

Økonomi, prosjektleders rolle og forsikring

Studien og biobanken er finansiert gjennom forskningsmidler fra Forskningsrådet og Helse Sør-Øst. Vi vil senere kunne søke også andre kilder, som farmasøytisk industri. Prosjektleder har ingen personlige økonomiske interesser i prosjektet. Norsk pasientskadeerstatningsordning gjelder ved deltagelse i studien.

STORK
Groruddalen

Samtykke til deltakelse i studien

Jeg er villig til å delta i studien

_____

(Signert av prosjektdeltaker, dato)

Jeg bekrefter å ha gitt informasjon om studien

_____

(Signert, rolle i studien, dato)

10.05.09

Forespørsel om deltakelse i forskningsprosjektet

Foreldres erfaringer med at helsepersonell tar opp bekymring for overvekt hos 4-åringen deres.

Bakgrunn og hensikt
Mitt navn er Ingun Toftemo, og jeg har siden 2000 jobbet som fastlege og helsestasjonslege på Lillehammer. Ved siden av dette forsker jeg nå på vekst utvikling hos barn.

Dette er et spørsmål til deg om å delta i en forskningsstudie for å skaffe kunnskap som kan gi helsepersonell innsikt i hvordan de på et tidlig stadium skal bevisstgjøre foreldre for å forhindre varig overvekt hos barn. Da du nettopp har vært på helsestasjonen med barnet ditt og fått beskjed om barnets vekt, har du en spesiell erfaring jeg ønsker å få del i.

Hva innebærer studien?
Studien innebærer intervju med flere foreldre som nylig har vært til 4års kontroll med barnet sitt. I samtalene vil jeg vite hvordan du opplevde 4års kontrollen og vekstmålingene av barnet ditt. Vi vil sammen se på vekstkurven til 4åringen din. Samtalen med meg er individuell. Den vil vare ca. en time, og foregå her på helsestasjonen. Samtalen blir tatt opp på lydmedium. På den måten kan vi ha oppmerksomhet om samtalen, og i etterkant lettere finne ut hva som ble sagt. Deltagelse i studien vil ikke ha noen betydning for ditt forhold til helsestasjonen.

Mulige fordeler og ulemper
Din innsats kan sette helsepersonell i bedre stand til å forebygge varig overvekt hos barn. Jeg ser ingen ulemper ved samtalen, annet enn at vi bruker din tid.

Hva skjer med informasjonen om deg?
Intervjuet vil bli skrevet ned. Både lydfilen og det skriftlige dokumentet vil bli slettet når undersøkelsen er ferdig. Informasjonen som registreres om deg og barnet ditt skal kun brukes slik som beskrevet i hensikten med studien. Alle opplysningene vil bli behandlet uten navn og fødselsnummer eller andre gjenkjennende opplysninger. Det vil ikke være mulig å identifisere deg eller barnet ditt i resultatene av studien når disse publiseres.

Frivillig deltakelse
Det er frivillig å delta i studien. Du kan uten å oppgi noen grunn trekke ditt samtykke til å delta. Dette

Foreldreerfaring Barn og vekt

vil ikke få konsekvenser for din oppfølging ved helsestasjonen. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Etter intervjuet er gjort kan du innen 3 måneder trekke deg fra undersøkelsen. Dersom du ønsker å trekke deg eller har spørsmål til studien, kan du kontakte prosjektleder Ingun Toftemo på telefon 91610528.

Personvern
Opplysninger som registreres er navnet ditt slik at jeg kan ta kontakt, samt anonyme data fra barnets vekstkurve. Ditt og barnets navn er kun tilgjengelig for Ingun Toftemo. Selve intervjuet er uten slike opplysninger, og vil kun være tilgjengelig for forskerteamet som har taushetsplikt.

Rett til innsyn og sletting av opplysninger om deg
Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få opplysninger slettet, med mindre opplysningene allerede er brukt i vitenskapelige publikasjoner.

Økonomi
Studien er finansiert gjennom forskningsmidler fra Den norske Legeforening

Informasjon om utfallet av studien
Du har rett til å få informasjon om resultatene fra studien.

Samtykke til deltakelse i studien

Jeg er villig til å delta i studien

(Signert av prosjektdeltaker, dato)

og kan kontaktes på telefon: _____

Jeg bekrefter å ha gitt informasjon om studien

(Signert, rolle i studien, dato)

17 Papers I-III

I

BMC Public Health

Contrasting patterns of overweight and thinness among preschool children of different ethnic groups in Norway, and relations with maternal and early life factors

Ingun Toftemo[1*] , Anne Karen Jenum[2], Per Lagerløv[1], Pétur B. Júlíusson[3], Ragnhild Sørum Falk[4] and Line Sletner[5]

Abstract

Background: Childhood obesity is a worldwide health challenge and risk factor for adult life obesity, which predisposes to development of type 2 diabetes and cardiovascular diseases. However, also thinness in early life has been related to these diseases, especially if followed by fat gain. In European countries, susceptibility to cardio-metabolic diseases varies considerably between ethnic groups. We investigated ethnic differences in overweight and thinness in a multi-ethnic, population-based cohort of preschool children in Norway, and associations with maternal and early postnatal factors.

Methods: Participants were children aged 4–5 years ($n = 570$) drawn from the population-based STORK Groruddalen cohort of healthy women and offspring followed from early pregnancy. Ethnic groups were: European ($n = 298$), South Asian ($n = 154$), and Middle East/North African ($n = 118$). Children's growth data were provided from routine visits at local Child Health Clinics. Weight status was defined by the International Obesity Task Force. Using multinomial logistic regression analysis, we explored ethnic differences in overweight and thinness, and associations with maternal-, pre, – and postnatal factors.

Results: Children of Middle East/North African origin had higher prevalence of overweight (22.0%) compared to European (12.8%) children, and in adjusted logistic regression analysis almost the double risk (OR 1.98; 95%CI: 1.08–3.63). Prevalence was lower in children of South Asian origin (5.2%). Children with South Asian background had higher prevalence of thinness (26.0%) compared to ethnic Europeans (10.4%), and the double risk (OR 2.20; 95%CI: 1.25–3.87) in adjusted models. Applying newly suggested BMI adjustments in South Asian children, taking into account their relatively increased adiposity, markedly increased the prevalence of overweight, and decreased the prevalence of thinness in this subgroup. Birthweight and maternal prepregnant overweight were strongly, positively associated with overweight, and inversely associated with thinness. Lower maternal age was associated with overweight only.

Conclusions: In a multi-ethnic cohort we found strikingly different patterns of overweight and thinness among children of different ethnic groups at age 4–5 years, and a strong association between maternal BMI and their children's weight status. More knowledge is needed on what characterizes and what promotes healthy growth patterns in multi-ethnic populations.

Keywords: Ethnicity, Preschool, Overweight, Obesity, Thinness, BMI

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Full list of author information is available at the end of the article

Background

An increase in the prevalence of overweight and obesity among preschool children has been widely reported over the last decades. This represents a serious public health challenge [1]. Currently, the majority of children with overweight live in low- and middle-income countries where the overall prevalence is still increasing [1]. In European preschool children, the prevalence of overweight seems to have reached a plateau during the last years, although the prevalence varies considerably between countries. The rates tend to be higher in girls than in boys [2–4]. Findings indicate that some ethnic minority children with Middle East or North-African origin may be disproportionally affected by childhood overweight [4, 5], while other ethnic minorities, such as South Asian children, may have lower risk [6].

Childhood overweight and obesity have been associated with maternal factors such as prepregnant overweight and obesity, low socioeconomic position, and gestational diabetes mellitus [7–9]. Further, high birth weight and rapid weight gain in infancy also increase the risk [9]. Childhood overweight is a strong predictor for adult obesity and increases the risk of insulin resistance, type 2 diabetes, and cardiovascular diseases [10, 11].

However, cohort studies with long-term follow-up indicate that thinness at birth and early childhood may also be a risk factor for type 2 diabetes and cardiovascular disease, in particular if followed by an increase in body mass index (BMI) later in life [12–15]. This growth pattern most likely represents low lean mass from birth, which tracks throughout adult life, followed by accumulation of fat mass later in life. This "thin-fat phenotype" increases the risk of later cardio-metabolic diseases, but is not necessarily linked to adult overweight as defined by a high BMI [15–17]. Some ethnic groups, particularly South Asian populations, have an increased risk for type 2 diabetes and cardiovascular disease for a given BMI compared to populations with European or African origin. The susceptibility of South Asians is believed to be partly explained by their predisposition to a thin-fat phenotype already from birth [13, 18, 19]. By measuring body fatness in a multi-ethnic population of children in UK, Hudda et al. showed that BMI underestimated body fat in South Asian children, and suggested a positive adjustment of BMI of + 1.12 kg/m^2 to account for greater relative adiposity in this population [20].

Pregnancy, the postpartum period, and early childhood are increasingly recognized as underused windows of opportunity to improve public health in the short and longer term [21]. In Norway, as in many other countries, pregnant women and their children are followed up regularly at Child Health Clinics. Increased knowledge about factors influencing overweight and thinness in multi-ethnic child-populations could contribute to an improved identification of children at risk, who could be offered targeted interventions in early life with the potential to reduce ethnic and social differences in health [22]. The aim of the current study was to investigate ethnic differences in overweight and thinness in a multi-ethnic, population-based cohort of preschool children in Norway, and associations with maternal and early postnatal factors.

Methods

Design and study population

The data were drawn from a prospective, population based cohort study, STORK Groruddalen, of 823 healthy pregnant women living in North-East Oslo [23]. Pregnant women were eligible for the study if they were 1) living in one of 3 city districts in Groruddalen; 2) planning to give birth at one of the 2 study hospitals; 3) at ≤20 weeks' gestation; 4) not having pre-pregnancy diabetes or other diseases requiring intensive hospital follow-up during pregnancy; 5) able to communicate in Norwegian or any of the 8 languages to which all the information materials and questionnaires were translated (Arabic, English, Sorani, Somali, Tamil, Turkish, Urdu and Vietnamese); and 6) able to give informed written consent. While attending prenatal care at the Child Health Clinic, women were enrolled at mean 15 weeks of gestation, from 2008 to 2010. The Groruddalen area has a population that covers a large span in socioeconomic status, and has a high proportion of ethnic minorities. Of the study population, 59% had ethnic minority background. Questionnaire data were collected by specially trained midwifes through interview, supported by a professional interpreter when needed.

Outcome variable

Virtually all children in Norway attend a Child Health Clinic regularly for vaccinations and check-ups. For this study we used children's growth data from the routine preschool visit performed at age 4–5 years at the local Child Health Clinic. Trained child health care nurses measured weight, to the nearest 100 g and height, to the nearest 0.1 cm. BMI was calculated as weight/height^2 (kg/m^2). Our outcome variable was weight status at age 4–5, classified as an ordinal variable with 4 levels: "thinness", "normal weight", "overweight" and "obesity" using the age- and sex-specific BMI cut-off values (z-scores) defined by the International Obesity Task Force [24]. These cut-off values correspond to centile curves passing through BMI 18.5 (thinness grade 1), 25 (overweight) and 30 (obesity) at age 18 years. In the regression analysis, the overweight and obesity group were merged and referred to as "overweight".

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Exposure variables

We chose exposure variables according to the literature and availability. The following variables were collected by interviews and questionnaires at inclusion:

Ethnicity was considered the main exposure variable and defined by the child's mother or maternal grand-mother's country of birth if this country was outside Europe. We had information on 81% of fathers. Based on these data and clinical experience, there were very few children of mixed ethnicity (< 5% of ethnic European, South Asians and children with Middle East / North African origin). For this study we chose to focus on results for 3 main ethnic groupings: 1) Europeans (primarily from Norway and other Scandinavian countries); 2) South Asians (primarily from Pakistan and Sri Lanka); 3) Middle East/North Africans (primarily from Iraq, Turkey, Morocco, Afghanistan, Somalia, and Ethiopia).

Mother's pre pregnant BMI was used as a categorical variable based on measured height and self-reported pre-pregnancy weight at inclusion [23], divided into three levels: Thinness (BMI < 18.5 kg/m^2), normal weight (BMI 18.5–25 kg/m^2) and overweight including obesity (BMI > 25 kg/m^2).

Education was defined as an ordinal variable: Lower level (primary education or less), middle level (completed high school/upper secondary), and higher level (completed university/university-college education ≥4 years).

Prepregnant physical activity was self-reported and collected by a questionnaire that is previously validated against a physical activity monitor [25]. This variable is defined as moderately intensive activity for 30 min for ≥5 days/week, moderately intensive activity for 2.5 h/week over ≥3 days, vigorous-intensity activity for ≥20 min 3 times per week, or activity of both moderate and vigorous intensity (e.g., vigorous activity once per week and moderate activity twice per week). Prepregnant physical activity < 1 year prior to pregnancy was coded as *never*. Prepregnant physical activity > 1 year prior to pregnancy was coded as *regular*.

Mother's dietary patterns were collected in week 28 of pregnancy, through a food frequency questionnaire, and defined by cluster analysis on 55 variables of intake and dichotomized into healthy and unhealthy [26].

Gestational diabetes mellitus was for this study defined according the WHO$_{2013}$ criteria; fasting glucose ≥5.1 mmol/l and/or 2-h plasma glucose ≥8.5 mmol/l. A glucose tolerance test was performed in gestational week 28.

Other variables

Age at enrolment was used as continuous variable, and parity dichotomized as nulliparous and parous. Child birthweight to the nearest gram was routinely collected at birth on calibrated electronic scales [27]. Breast feeding was based on self-reports at 14 weeks postpartum and categorized as any and no breastfeeding. Maternal smoking status was also considered as covariate, but as very few of the ethnic minority women smoked, this variable was left out from the analyses.

Sample size

Of 823 women enrolled in early pregnancy, 784 singleton live born neonates with valid birth data were born (Fig. 1, flow chart). Of these neonates, 158 were excluded due to prematurity, postnatal death, refused further participation, family moved abroad or mother changed social security number, no data from Child Health Clinics outside Oslo, or missing data at Child Health Clinic at age 4–5 years. At last, 56 children from smaller and heterogeneous ethnic groups (East Asia, South- and Central America, and Sub- Saharan African countries) were excluded. The final study sample consisted of 570 children from 3 ethnic groups; 298 (52.3%) ethnic Europeans, 154(27.0%) ethnic South Asians, and 118 (20.7%) with Middle East/North African ethnicity.

Statistical analysis

Descriptive statistics are given as frequencies, proportions (%), and means with standard deviation (SD). Differences in prevalence of thinness and overweight between the ethnic groups were assessed by Chi-square tests. Differences in children's age were assessed by one-way ANOVA tests.

To investigate the impact of maternal and postnatal determinants on the child's risk of overweight and thinness at the age of 4–5 years, we performed multinomial logistic regression analysis. Overweight and thinness were compared with normal weight children. Ethnic origin was the main exposure variable of interest (Europeans as reference). The following variables were considered potential covariates; maternal education, parity, age, prepregnant BMI and physical activity, diet during pregnancy, gestational diabetes mellitus, and child sex and birth weight. We selected variables into the multivariate model by a purposeful selection approach [28]. Variables with a *p*-value < 0.2 in the univariate analysis were included into the multivariate model. We removed one variable at a time, the variable with the highest p-value first, until all variables reached the level of significance ($p < 0.05$) in at least one of the models. For each step we checked if our main effect estimates (ethnicity) changed more than 15%. If so, the variable was kept in the model to take into account the potential confounding effect on the outcome. In the final model we tested for interactions with ethnicity by entering cross-product terms one-by–one. No significant interactions were observed. Results are shown as odd ratios (ORs) with 95% confidence intervals (CI). We included breast feeding into the multinomial logistic

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Fig. 1 Flow chart
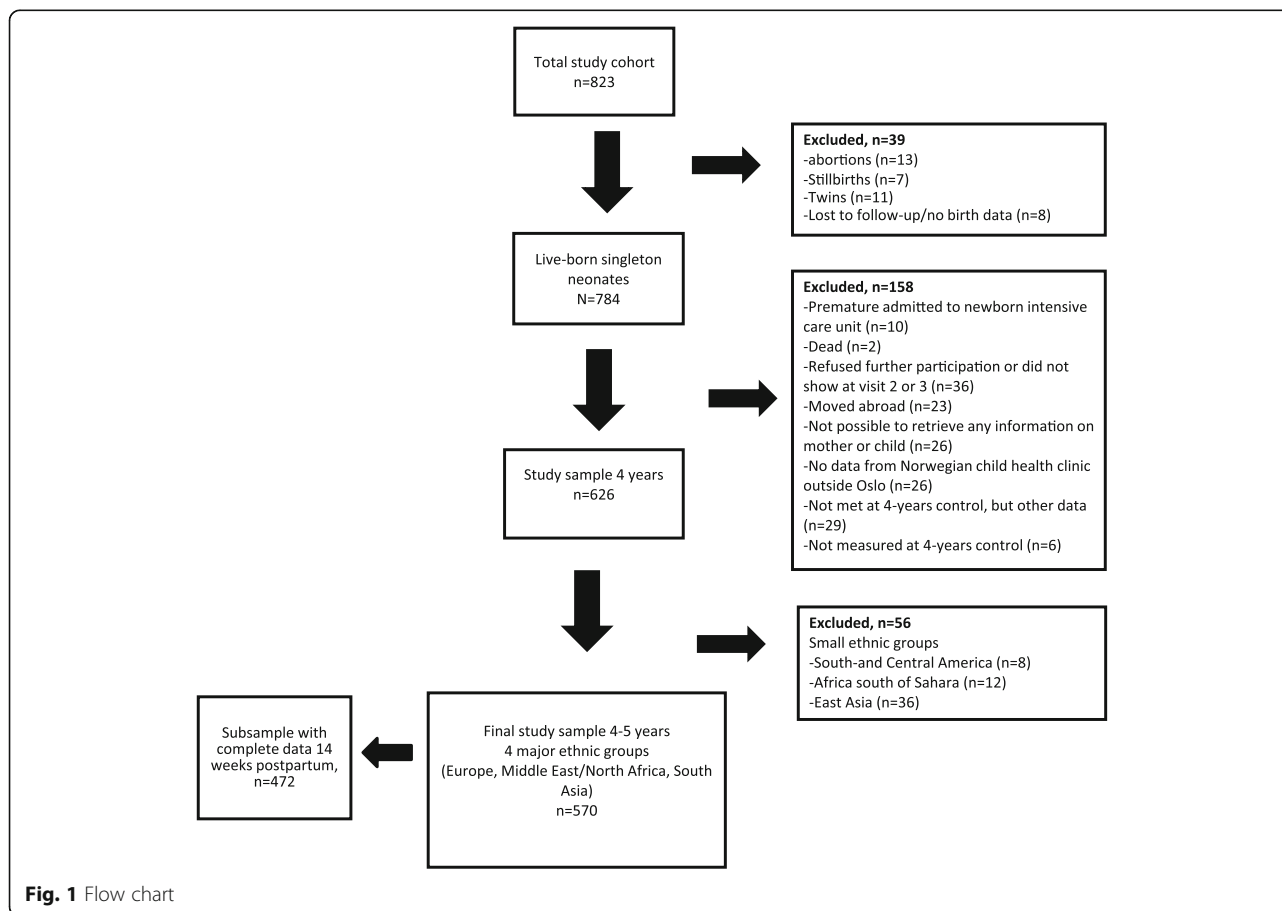
regression analysis of a subgroup with complete data at 14 weeks postpartum.

As it has been reported that children with South Asian origin have a higher fat mass for a given BMI than the other ethnic groups, we also performed positive BMI adjustments of + 1.12 kg/m2 for this group [20]. We then made a sensitivity logistic regression analysis using this adjusted classification of weight status for South Asians as outcome variable.

For all analyses we used SPSS version 22 (IBM SPSS statistics, NY, USA).

Results

Characteristics

Compared with European women, ethnic minority mothers were younger, had lower education, and higher parity. They were less physically active before pregnancy, and smoked less (Table 1). Prepregnant BMI was lower in South Asian women and higher in women from Middle East/North Africa. The prevalence of gestational diabetes mellitus was higher in ethnic minority mothers, and more minority women had a less healthy diet compared to ethnic European mothers. European children

had higher birthweight than children in the other ethnic groups.

Prevalence of overweight and thinness at age 4–5 years

The prevalence of overweight was significantly higher in children of Middle East / North African origin (22.0% (14.5–29.6)) compared to children with European origin (12.8% (8.9–16.6)), while the prevalence in children with South Asian origin was significantly lower (5.2% (2.2–8.0)) (Table 2). In contrast, the prevalence of thinness was significantly higher in children with South Asian origin (26% (19.9–32.0) compared to children with European origin (10.4% (6.9–13.9)) and Middle East/North Africa origin (12.7% (6.6–18.8)).). Taking account of the South Asian's relatively increased adiposity, we made positive adjustments of BMI. After adjustments, prevalence of thinness changed from 26 to 3.9%, and prevalence of overweight changed from 5.2 to 14.3% (Table 2).

We found strong relations between mothers' prepregnant weight status and the prevalence of overweight and thinness in the offspring. Of mothers with prepregnant overweight, 34% of Middle East/North African- and 19% of European mothers had children with overweight (Fig. 2).

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Table 1 Baseline characteristics

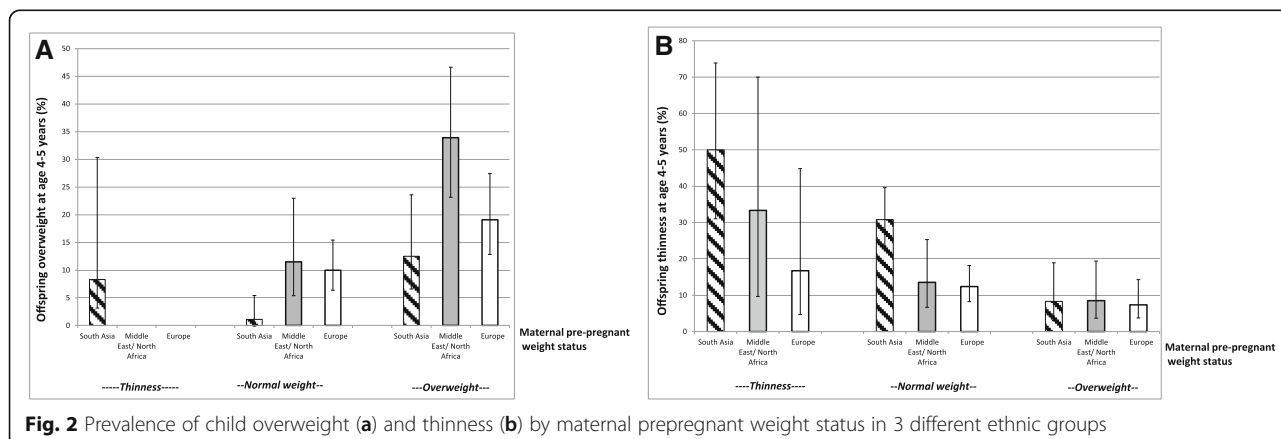| Variables	Ethnic origin n (%)			
	Total	Europe	Middle East/North Africa	South Asia
	$n = 570$	$n = 298$ (52.3)	$n = 118$ (20.7)	$n = 154$ (27.0)
	n (%)	n (%)	n (%)	n (%)
Mothers				
Maternal age, mean (SD)	29.9 (4.8)	30.7 (4.5)	29.3 (5.5)	27.8 (4.5)
Prepregnant BMI, kg/m2 (SD)	24.5 (4.8)	24.6 (4.7)	26.1 (5.7)	23.7 (4.3)
Prepregnant weight status				
Thinness, BMI < 18.5	30 (5.4)	12 (4.1)	6 (5.1)	12 (7.9)
Normal weight, BMI 18.5–25	313 (55.9)	170 (58.2)	52 (44.4)	91 (60.3)
Overweight, BMI > 25	217 (38.8)	110 (37.7)	59 (50.5)	48 (31.8)
missing	10	6	1	3
Nulliparous	267 (46.8)	161 (54.0)	41 (34.7)	65 (42.2)
Education				
Primary education or less	95 (16.8)	15 (5.1)	52 (44.1)	28 (18.5)
Completed high school/upper secondary	214 (37.8)	90 (30.5)	47 (39.8)	77 (50.3)
Completed university/college education	257 (45.4)	190 (64.4)	19 (16.1)	48 (31.4)
missing	4	3	0	1
Prepregnant regular physical activity[a], $n = 570$	174 (30.6)	132 (44.3)	18 (15.3)	24 (15.6)
Gestational diabetes mellitus[b], $n = 556$	180 (32.4)	72 (24.7)	41 (36.3)	67 (44.1)
Non-healthy diet[c], $n = 551$	86 (15.6)	15 (5.2)	27 (25.0)	44 (28.9)
Smoking 3 months before pregnancy, $n = 478$	104 (21.8)	90 (32.1)	11 (12.5)	3 (2.7)
Children				
Sex, boy (%)	284 (49.8)	157 (52.7)	52 (44.1)	75 (48.7)
Birth weight (mean), g (SD)	3431.1 (548.6)	3577.9 (527.1)	3413.4 (509.5)	3226.1 (526.8)
Subsample with data 3 months postpartum				
Breast feeding 14 weeks postpartum, $n = 472$	404 (85.6)	214 (84.9)	80 (88.9)	110 (84.6)

[a] Self-reported, based on validated questionnaire
[b] Defined by WHO 2013 criteria; fasting glucose ≥5.1 mmol/l and/or 2-h plasma glucose ≥8.5 mmol/l
[c] Collected by food frequency questionnaire in week 28 of pregnancy, and defined by cluster analysis on 55 variables of intake

Table 2 Age and prevalence of thinness, overweight and obesity in children aged 4–5 years

	Ethnic origin, n (%). Main ethnic groups					
	Total	Europe	Middle East/North Africa	South Asia unadjusted BMI	p-value	South Asia adjusted BMI[***]
	$n = 570$	$n = 298$	$n = 118$	$n = 154$		$n = 154$
Age in years at Child						
Health Clinic control, mean (SD)					$p = 0.60$[*]	
	4.38 (0.28)	4.37 (0.28)	4.39 (0.31)	4.40 (0.26)		
BMI category, n (%)					$p < 0.001$[**]	
Thinness	86 (15.1)	31 (10.4)	15 (12.7)	40 (26.0)		6 (3.9)
Normal weight	412 (72.3)	229 (76.8)	77 (65.3)	106 (68.8)		126 (81.8)
Overweight including obesity	72 (12.6)	38 (12.8)	26 (22.0)	8 (5.2)		22 (14.3)

[*] One-way ANOVA analysis
[**] Chi-square test
[***] positive BMI adjustments of + 1.12 kg/m2 to account for greater relative adiposity in this population

Fig. 2 Prevalence of child overweight (**a**) and thinness (**b**) by maternal prepregnant weight status in 3 different ethnic groups

In contrast, 50% of South Asian mothers with thinness had children with thinness.

Associations between maternal and early postnatal factors and overweight at age 4–5 years

Unadjusted multinomial logistic analysis (Table 3) showed that children of South Asian origin had 54% lower risk of overweight compared to European, while Middle East/ North African origin had 2-fold (higher) risk. Maternal pre pregnancy overweight, low education, gestational diabetes mellitus, and child's birthweight were also positively associated with overweight.

In the adjusted model, Middle East/North African origin still nearly doubled the risk of being overweight (OR1.98; 95% CI:1.08–3.63). Maternal prepregnant overweight (OR 2.56; 95% CI: 1.52–4.61) and age (OR 0.93; 95% CI: 0.88–0.99), and child birthweight (OR1.11; 95% CI: 1.05–1.18) were independently and positively associated. Female sex was borderline significantly associated (OR1.73; 95% CI: 0.99–3.01). However, the negative effect estimate for South Asian origin was no longer statistically significant when adjusting for other factors. The association between maternal gestational diabetes and overweight in the offspring was more apparent in ethnic Europeans than in the ethnic minority groups. When formally testing for an interaction between ethnicity and gestational diabetes, however, the interaction was only borderline significant.

Associations between maternal and early postnatal factors and thinness at age 4–5 years

Unadjusted multinomial logistic analysis (Table 3) showed that children with South Asian origin had a 2.8-fold higher risk of thinness compared to those with European origin. Maternal pre pregnancy overweight and the child's birth weight were negatively associated with thinness, while unhealthy diet was positively associated. The adjusted model showed that South Asian origin still doubled the risk of thinness (OR 2.20; 95% CI: 1.25–3.87). Prepregnant

maternal overweight (OR0.50; 95% CI: 0.28–0.90) remained independently negatively associated with thinness at 4–5 years.

The sensitivity logistic regression analysis using the adjusted classification of weight status for South Asians (Additional file 1) shows that the South Asian children have a lower risk of thinness (OR 0.22; 95% CI: 0.08–0.60) compared to the European. This result contradicts the logistic regression analysis using unadjusted BMI for South Asian children (Table 3). However, the factors positively related to overweight (Middle East/North African origin, maternal age, prepregnant overweight, female sex and birth weight) remain significant in the sensitivity model.

Discussion

To the best of our knowledge, this study is the first population based study to assess both thinness and overweight in a multiethnic sample of children in Europe. We found strikingly different patterns of overweight and thinness among children of different ethnic groups at age 4–5 years. Compared to European children, children of Middle East/North African origin had almost the double risk of being overweight at 4–5 years of age. In contrast, South Asian origin almost doubled the risk of thinness. Applying newly suggested BMI adjustments in South Asian children, taking into account their relatively increased adiposity, markedly increased the prevalence of overweight and decreased the prevalence of thinness in this subgroup. Factors increasing the risk of overweight were maternal prepregnant overweight, lower maternal age, higher birthweight, and female sex. Maternal prepregnant overweight and higher birthweight reduced the risk of thinness. Our findings may be of great relevance for public health.

The overall prevalence of overweight and the prevalence in ethnic Europeans in our study were comparable with findings from another study of mainly ethnic Norwegian children. These data were collected in 2003– 2006, and showed that in children aged 2–5 years, the

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Table 3 Multinomial logistic regression for child overweight including obesity (A), and thinness (B), compared to normal weighted at age 4–5 years

Candidate factors	A. Overweight						B. Thinness					
	Unadjusted OR			Final model			Unadjusted OR			Final model		
	OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Ethnic origin												
Europe (reference)												
South Asia, unadjusted	0.46	0.21–1.0	0.53	0.55	0.24–1.27	0.2	2.79	0.74–4.7	< 0.001	2.20	1.25–3.87	0.006
Middle East/North Africa	2.04	1.16–3.57	0.01	1.98	1.08–3.63	0.03	0.29	0.74–20.8	0.3	1.27	0.63–2.57	0.5
Age	0.96	0.91–1.02	0.2	0.93	0.88–0.99	0.02	0.97	0.92–1.02	0.2	1.00	0.94–1.05	0.9
Prepregnant BMI, categorized												
Normal weight (reference)												
Underweight	0.44	0.06–3.40	0.4	0.45	0.06–3.72	0.5	1.80	0.82–4.0	0.1	1.89	0.81–4.43	0.1
Overweight including obesity	3.01	1.78–5.09	< 0.001	2.65	1.52–4.61	0.001	0.47	0.27–0.83	0.009	0.50	0.28–0.90	0.02
Parity												
Para 0 (reference)												
Para 1 or more	1.11	0.68–1.83	0.7				0.91	0.58–1.42	0.7			
Education												
University/college (reference)												
High School/Upper secondary school	1.58	0.90–1.79	0.1				1.24	0.76–2.05	0.4			
Primary school or less	2.00	1.04–4.00	0.04				1.26	0.66–2.41	0.5			
Prepregnant regular physical activity[a]												
Yes (reference)												
No	1.67	0.93–3.02	0.09				1.26	0.76–1.08	0.4			
Gestational diabetes mellitus[b]												
No (reference)												
Yes	1.69	1.01–2.84	0.047				1.09	0.67–1.78	0.7			
Cluster healthy/non-Healthy nutrition[c]												
Healthy (reference)												
Non-healthy	0.76	0.35–1.66	0.5				1.88	1.07–3.28	0.03			
Sex, child												
Male (reference)												
Female	1.52	0.92–2.52	0.1	1.73	0.99–3.01	0.05	0.74	0.47–1.16	0.2			
Birth weight (per 100 g)	1.12	1.07–1.18	< 0.001	1.11	1.05–1.18	< 0.001	0.93	0.90–0.97	0.001	0.96	0.92–1.01	0.1
Breast-feeding 3 months postpartum												
Yes (reference)												
No	1.58	0.79–3.18	0.2				1.07	0.52–2.17	0.9			

[a] Self-reported, based on validated questionnaire
[b] Defined by WHO 2013 criteria; fasting glucose ≥5.1 mmol/l and/or 2-h plasma glucose ≥8.5 mmol/l
[c] Collected by food frequency questionnaire in week 28 of pregnancy, and defined by cluster analysis on 55 variables of intake

prevalence of overweight was 12.7% [8]. Other European studies have only assessed either overweight or thinness. In two Dutch studies investigating overweight only, Turkish and Moroccan children had higher odds of being overweight compared to native Dutch children [4, 5]. In a British cross-sectional study investigating thinness only, the prevalence of thinness in British preschool children was 5.7%, while ethnic Asian children had an OR

of 3.6 compared to white children [29]. The results from both these studies were in line with our findings.

In accordance with findings from other studies [5, 7, 30], maternal prepregnant overweight was a very strong predictor of childhood overweight. Nevertheless, it is largely unknown how much of this effect can be explained by a direct, causal relationship, for example mediated through maternal insulin resistance and inflammatory signals during

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pregnancy, and how much is mediated through genetics or shared life style factors [21].

Sex differences in childhood overweight vary in a global perspective [31]. In our study girls had more overweight than boys at age 4–5 years. Earlier Scandinavian studies show similar results at preschool age, but in older age groups there are more overweight in boys than in girls [8, 32].

Low socioeconomic status and low parental education have also been associated with childhood overweight [8, 30, 33]. We found no significant associations between maternal education and overweight at age 4–5 years. This might partly be due to lack of power, and that ethnicity also carries information about socioeconomic status [34]. However, other socioeconomic factors may be more important. A recent Swedish study showed that neighborhood purchasing power was more strongly related to overweight at age 4–5 years than parental educational levels [32]. This could be especially relevant in an immigrant population.

At the age of 4–5 years we found no association between gestational diabetes and offspring overweight. Studies investigating the possible link between gestational diabetes and offspring overweight show inconsistent results [35–38]. In a recent study from Hong Kong, maternal hyperglycemia in pregnancy was associated with adiposity in girls at age 7, but not in boys [36]. Our findings could suggest that also ethnicity may differentially affect this relation. Larger samples may be needed to study such effect modifications.

Other explanations of ethnic differences in overweight have been suggested. Perception of ideal BMI may vary between ethnic groups. Fatness may be associated with wealth, good health, beauty, strength, and happiness [39, 40]. In many countries, and in immigrant populations from Asia and the Middle East/North Africa, a shift from traditional food habits and entering a more sedentary life style has dramatically increased the prevalence of obesity and other risk factors for type 2 diabetes and cardiovascular diseases [41, 42].

The prevalence of thinness in the South Asian children at age 4–5 years in our study was significantly higher than in the other ethnic groups. Importantly, our definition of thinness is based on BMI z-score and does not necessarily reflect the children's body composition. This was demonstrated in two UK studies showing that the adiposity levels were markedly higher in South Asian children than in white European children, despite lower BMI [20, 43]. In adulthood, the proportion of Asian people with a high risk of cardiovascular diseases and type 2 diabetes is substantial at BMIs lower than the WHO cut-off point for overweight [44]. However, estimated and suggested cut-off points for observed risk vary even between Asian populations and by outcomes

[45]. As previously also shown in our cohort, neonates with South Asian ethnic origin are smaller and "thinner" at birth with smaller abdomen and less fat-free mass, but have relatively preserved fat mass [27]. This phenotype may result from in-utero programming after multigenerational malnutrition, tracks through life, and increases the risk of type 2 diabetes and cardiovascular disease [17, 18, 46]. Most of the South Asian children in our cohort had moderate thinness (clinically classified as grade 1 underweight). Hence, this is probably not a sign of illness, but rather due to lower lean mass. These children may be especially vulnerable if experiencing an increase in BMI; mainly representing an absolute and relative increase of fat tissue.

BMI underestimates total body fatness in South Asians. Positive BMI adjustments are suggested to make a more clinically relevant classification of weight class in children with South Asian origin [20] A recent UK study showed that when applying positive BMI adjustments in South Asian children living in England, the prevalence of thinness and overweight increased considerably in this group [47]. When applying the same BMI adjustments in our cohort of South Asian children, we also found marked changes in prevalence of thinness and overweight.

Our study suggests that health professionals should pay more attention to ethnic minority groups that are especially vulnerable to excess weight gain. On one hand, children of Middle East/North-African origin seem to have increased risk of developing overweight from early age. Overweight may persist into adult life and increase their risk of cardio-metabolic disease. On the other hand, children with South Asian origin have higher risk of thinness, probably reflecting low lean mass. This may increase their vulnerability to cardio-metabolic disease, especially if they experience excessive fat gain later in childhood or in adult life. Therefore, children who are thin from birth and in early childhood should receive earlier attention if they start crossing percentiles, and importantly, before they reach the "overweight threshold". More research is needed before applying adjusted BMI classification based on ethnicity in a clinical setting. At Child Health Clinics and in general practice almost all children and pregnant women are followed over time from birth to adolescence and through pregnancies. This gives health professionals unique opportunities to promote healthy growth in children. Young girls and women in reproductive age can be targeted before, in, and in between pregnancies to prevent maternal overweight.

Strengths of the present study include the population-based cohort design, the large proportion of ethnic minorities that are often excluded in research, a very extensive and high quality data set, high attendance rate, and minor loss to follow-up. Limitations to this study should also be noted: Although anthropometric data on children were collected by trained Child Health Clinic personnel, they may be more inaccurate than if

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performed under standardized conditions. However, this probably applies equally to all ethnic groups. With 570 children in our cohort, power was limited, especially related to exploring potential effect modifications. Lifestyle characteristics, as diet and physical activity, are difficult to measure, and our variables may not represent all relevant variation. Due to small numbers for many countries of origin, we merged women into three larger ethnic groups. It cannot be ruled out that this may cause some bias because of within-group heterogeneity.

Conclusion

We found strikingly different patterns of overweight and thinness among children of different ethnic groups at 4–5 years of age; a finding with a great relevance to public health. Compared to the European children in our cohort, children with Middle East/North African background had almost the double risk of being overweight at age 4–5 years. Maternal prepregnant overweight and younger age, and higher child birthweight also increased this risk. In contrast, children with South Asian origin had almost the double risk of being classified as thin. However, applying suggested BMI adjustments in South Asian children, taking into account their relatively increased adiposity, markedly increased the prevalence of overweight, and decreased the prevalence of thinness in this group. The deviant and contrasting weight development in both of our ethnic minority groups could represent an increased vulnerability to contract cardiovascular diseases and type 2 diabetes in adult life. This adds to the complexity health workers need to be aware of. More knowledge is needed on how to tailor culturally sensitive health care for mothers and their children to promote healthy growth and prevent disease over the life course.

Additional file

Additional file 1: Multinomial logistic regression for child overweight including obesity and thinness, compared to normal weighted at age 4–5 years. Positive BMI adjustments for the South Asian children. (DOCX 26 kb)

Authors' contributions
LS, AKJ and IT conceived and designed the study. IT and LS collected data at the Child Health Clinics and plotted them. IT preformed the statistical analysis supervised by LS and RSF. All authors (IT, AKJ, PL, PBJ, RSF, LF) contributed to interpreting and discussing the results, writing and reviewing the manuscript. All authors read and approved the final manuscript.

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II

REGULAR ARTICLE

ACTA PÆDIATRICA
NURTURING THE CHILD

WILEY

Body mass index trajectories up to preschool age in a multi-ethnic population; relations with maternal gestational diabetes, BMI and gestational weight gain

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Abstract

Aim: Independent effects of gestational diabetes (GDM), maternal prepregnant obesity and gestational weight gain on offspring BMI and obesity are scarcely documented. We examined associations between GDM and children's BMI trajectories from birth to 4-5 years age, and effects of prepregnant obesity and gestational weight gain not mediated through GDM.

Methods: We included 734 children from a population-based, multi-ethnic cohort of women and their offspring followed from early pregnancy. All women were screened for GDM. Using linear mixed models, we explored associations between maternal factors and children's BMI development through seven serial measurements.

Results: At birth and age 4-5 years, BMI of children exposed to GDM was similar to those not exposed. However, they had slower BMI growth (B = −0.1 BMI units/month (95% CI: −0.17, −0.04)) during first 6 months, and faster BMI growth from 6 months to 4-5 years. Maternal prepregnant obesity was associated with higher child BMI at birth, and thereafter persistently higher BMI. High gestational weight gain was associated with faster BMI growth from 6 months to 4-5 years.

Conclusion: Effects of maternal GDM, prepregnant obesity, and gestational weight gain on children's BMI and BMI trajectories from birth to preschool age differed in relation to effect size, timing and direction.

KEYWORDS
childhood obesity, gestational diabetes, gestational weight gain, growth trajectory, prepregnant obesity

1 | INTRODUCTION

While many studies have reported strong associations between maternal gestational diabetes (GDM) and macrosomia and adiposity at birth, few have found associations with adiposity in early childhood.[1] However, evidence is growing that the effects of intrauterine exposure to hyperglycaemia can emerge later in childhood[2,3] Groups in Europe with low socioeconomic position and ethnic minorities have a higher burden of GDM and are diagnosed with type 2 diabetes at a far younger age and at lower BMI.[4] Effects of in utero exposure to hyperglycaemia on offspring BMI and development of adiposity vary between observational studies.[5,6] Inconsistencies may partly relate to

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methodological issues; First, most studies are cross-sectional,[1,3] second, different definitions of GDM are used (from relatively mild GDM to overt type 2 diabetes),[5] third, in some studies, GDM is treated, in others not.[7,8] Randomised control studies and meta-analyses have shown an effect of treatment of relatively mild GDM on the risk of a large for gestational age baby, but information on long-term effects on overweight and adiposity is so far limited.[9]

Intrauterine exposures to maternal prepregnant obesity and to excessive maternal gestational weight gain (GWG) are strongly associated with obesity and cardio-metabolic risk factors in the offspring.[10] We have previously reported striking ethnic differences in overweight and thinness at 4-5 years of age and also found that maternal prepregnant overweight was strongly associated with children's overweight, while GDM was not.[11] As GDM is associated with maternal overweight/obesity and GWG, it is often difficult to disentangle the independent effect of hyperglycaemia from the two other weight-related factors.[4,12]

Our aim was therefore to investigate the independent association between maternal GDM and children's BMI trajectories from birth to 4-5 years of age, and the effects of prepregnant overweight/obesity and GWG not mediated through GDM, in a multi-ethnic population of pregnant women universally screened for GDM and treated accordingly.

2 | METHODS

2.1 | Design and study population

We drew data from the prospective, population-based STORK-Groruddalen cohort, in which 823 healthy pregnant women living in an ethnically and socioeconomically diverse area in Oslo, Norway, were enrolled in early pregnancy.[13] Pregnant women were eligible if they were (a) living in one of three city districts in Groruddalen, Oslo; (b) planning to give birth at one of the 2 study hospitals; (c) at ≤20 weeks' gestation; (d) not having pre-pregnancy diabetes or other diseases requiring intensive hospital follow-up during pregnancy; (e) able to communicate in Norwegian or any of the eight languages to which all the information materials and questionnaires were translated (Arabic, English, Sorani, Somali, Tamil, Turkish, Urdu and Vietnamese); and (f) able to give informed written consent. Women attending antenatal care at three child health clinics were enrolled at mean 15 weeks' gestation, from 2008 to 2010. The participation rate was 74% and 59% had ethnic minority background. The cohort was representative for women attending the child health clinics with respect to ethnicity and age. Specially trained midwifes collected questionnaire data through interviews, supported by a professional interpreter when needed.

2.2 | Sample size

From the 823 women enrolled in early pregnancy, 783 gave birth to a singleton, live born neonate with valid birth data (Figure 1,

Key Notes

- In a multi-ethnic cohort screened for gestational diabetes (GDM), we explored associations between children's BMI trajectories from birth to preschool age and intrauterine exposure to GDM, prepregnant obesity and gestational weight gain.
- Effects of these maternal exposures differed in relation to effect size, timing and direction.
- Children exposed to GDM had a catch-down BMI growth during first 6 months, followed by a catch-up growth 6 months to 4-5 years.

flow chart). Of these, 49 (6.7%) were excluded because the mother actively refused further participation, did not attend at study visits after enrolment, the child had severe medical conditions likely to substantially affect growth (information retrieved either from birth records or local health clinic records) or due to pre-term birth (otherwise healthy but premature and needed to be admitted to neonatal intensive care unit). The final study sample consisted of 734 children (all had at least one postnatal BMI measure), of which 344 (46.9%) had European, 180 (24.5%) South Asian, 38 (5.2%) East Asian and 150 (20.4%) Middle East/North African origin. A small heterogeneous group of 22 mother-child pairs (3%) had 'other' origin.
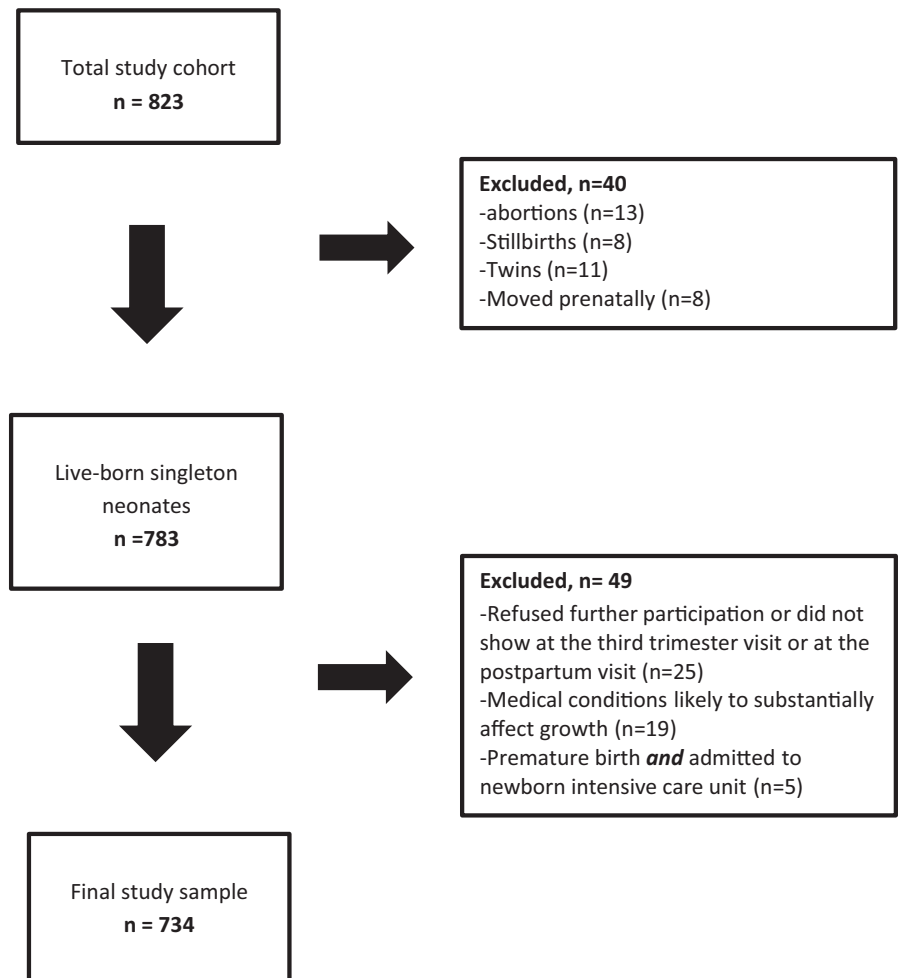
2.3 | Outcome variables

Child birthweight to the nearest gram (without diaper) was routinely collected at birth on calibrated electronic scales.[14] Crown-heel length was measured to the nearest 0.1 cm, by a measuring rod, with the head firmly held, while stretching the legs. We collected children's growth data retrospectively from the local child health clinics which virtually all children in Norway attend regularly, from all the routine check-up visits at 6 weeks, and 3, 6, 12, 15, 24 and 48 months of age.[11] According to national guidelines, public health nurses measure weight, to the nearest 10 g without diaper for children aged 0-15 months, and to the nearest 100 g in light clothing for children aged 24-48 months. Height is measured to the nearest 0.1 cm (crown-heel-length from birth to 15 months and standing height from 2 years of age). Our outcome variables were BMI calculated from all time points as kg/m^2 and BMI growth (changes in BMI units per month) from birth to 4-5 years of age.

2.4 | Exposure variables

GDM was our primary exposure variable, defined according the WHO_{1999} criteria (fasting glucose ≥ 7.0 mmol/L and/or 2-hour

FIGURE 1 Flow chart

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            ┌─────────────────────┐
            │  Total study cohort │
            │      n = 823        │
            └─────────────────────┘
                      │
                      │         ┌──────────────────────────────────┐
                      │────────▶│ Excluded, n=40                   │
                      │         │ -abortions (n=13)                │
                      ▼         │ -Stillbirths (n=8)               │
            ┌─────────────────────┐ │ -Twins (n=11)                 │
            │ Live-born singleton │ │ -Moved prenatally (n=8)       │
            │      neonates       │ └──────────────────────────────────┘
            │      n =783         │
            └─────────────────────┘
                      │
                      │         ┌──────────────────────────────────┐
                      │         │ Excluded, n= 49                  │
                      │         │ -Refused further participation or│
                      │────────▶│ did not show at the third        │
                      │         │ trimester visit or at the        │
                      ▼         │ postpartum visit (n=25)          │
            ┌─────────────────────┐ │ -Medical conditions likely to │
            │  Final study sample │ │ substantially affect growth   │
            │      n = 734        │ │ (n=19)                        │
            └─────────────────────┘ │ -Premature birth *and*        │
                                   │ admitted to newborn intensive │
                                   │ care unit (n=5)               │
                                   └──────────────────────────────────┘
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plasma glucose ≥ 7.8 mmol/L) after an oral glucose tolerance test in gestational week 28[15,16]; women diagnosed with mild GDM (fasting glucose < 7.0 mmol/L and 2-hour values 7.8-8.9 mmol/L) were given oral and written lifestyle advice and referred to their general practitioner for follow-up and women with 'moderate/severe GDM', (fasting glucose ≥ 7.0 mmol/L or 2-hour values ≥ 9.0 mmol/L) were referred to secondary care according to national guidelines. Secondary exposure variables were mother's prepregnant BMI and maternal GWG *Mother's prepregnant BMI* was based on measured height and self-reported pre-pregnancy weight at inclusion,[13] categorised as follows: normal- or underweight (BMI < 25 kg/m^2), overweight (BMI 25-30 kg/m^2) and obesity (BMI ≥ 30 kg/m^2). *Maternal GWG* was calculated as the difference between weight measured at gestational week 28 (the time of the oral glucose tolerance test) and self-reported pre-pregnancy weight at inclusion, allowing us to comply with assumptions of temporality, and because the relative weight of the foetus, the placenta, amniotic fluid and body water will be less in week 28 than later in pregnancy. In addition, early-gestation GWG has a stronger effect on offspring BMI and adiposity than mid- and late-gestation GWG.[17] We divided GWG into the lowest, middle (reference) and highest tertiles.

2.5 | Covariates

Prior to analyses, we used Directed Acyclic Graphs in the model building process to visualise the causal network linking GDM, prepregnant BMI and GWG with children's BMI growth (Figure S1) and to identify confounders to adjust for maternal ethnicity, socioeconomic status, age and parity. In order to obtain more precise estimates, we also adjusted for gestational age at birth and child sex as competing exposures, as they are strongly associated with growth. *Ethnicity* was defined by the child's mother or maternal grandmother's country of birth if was outside Europe and merged into five ethnic groups (Table 1). We had information on 81% of the fathers. Very few children were of mixed ethnicity (<5%).

Maternal socioeconomic status was a principal component score of 11 sociodemographic variables, dominated by the individual level variables education, occupational class, employment status, and household variables own or renting tenure, rooms per person in the household.[18] *Maternal age in years* was used as a continuous variable. *Parity* was categorised as Nulliparous, para 1 and para ≥ 2.

Gestational week at birth was derived from the first day of the mother's last menstrual period, unless last menstrual period was unknown/uncertain, last menstrual period derived term

TABLE 1 Characteristics of 734 mother and child pairs

Ethnic origin[a]	Total n = 734 (100%)	Europe n = 344 (46.9%)	Middle East/ North Africa n = 150 (20.4%)	South Asia n = 180 (24.5%)	East Asia 38 (5.2%)	Others 22 (3.0%)
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Mothers						
Age, mean (SD)	29.8 (4.8)	30.6 (4.5)	29.3 (5.4)	28.8 (4.6)	30.9 (4.5)	29.4 (6.5)
Education						
Primary education or less	120 (16.5)	14 (4.2)	61 (40.9)	34 (19.0)	8 (21.1)	3 (13.6)
Completed high school/ upper secondary	286 (39.2)	108 (31.7)	64 (43.0)	87 (48.6)	16 (42.1)	11 (50.0)
Completed university/ college education	323 (44.3)	219 (64.2)	24 (16.1)	58 (32.4)	14 (36.8)	8 (36.4)
Missing	5	3	1	1	0	0
Prepregnant BMI (SD)	24.6 (4.8)	24.6 (4.8)	25.9 (5.4)	23.7 (4.1)	22.0 (2.8)	26.6 (5.7)
Prepregnant weight status						
Normal- or underweight	420 (60.8)	198 (61.54)	74 (50.7)	109 (65.3)	30 (83.3)	9 (45.0)
Overweight	172 (24.9)	78 (24.2)	39 (26.7)	42 (25.1)	6 (16.7)	7 (53.0)
Obesity	99 (14.3)	46 (14.3)	33 (22.5)	16 (9.6)	0 (0)	4 (20.0)
Gestational weight gain (prepregnancy to 28 wk of pregnancy), kg (SD)	8.64 (4.8)	8.93 (4.6)	8.86 (5.1)	8.20 (4.7)	8.24 (4.3)	6.93 (6.6)
Gestational weight gain, tertiles						
Lowest third	213 (32.5)	102 (30.3)	47 (32.6)	64 (36.8)	17 (44.7)	9 (40.9)
Middle third	218 (33.3)	114 (33.8)	47 (32.6)	57 (32.8)	11 (28.9)	6 (27.3)
Highest third	224 (34.2)	121 (35.9)	50 (34.7)	53 (30.5)	10 (26.3)	7 (31.8)
Gestational diabetes mellitus[b]	92 (12.5)	38 (11.0)	22 (14.7)	26 (14.4)	6 (15.8)	0 (0)
Daily smoking 3 mo before pregnancy	81 (11.1)	69 (20.4)	8 (5.3)	1 (0.6)	2 (5.3)	1 (4.5)
Parity						
Nulliparous	335 (45.6)	183 (53.2)	52 (34.7)	74 (41.7)	13 (42.1)	9 (40.9)
Para 1	251 (34.2)	125 (36.3)	46 (30.7)	57 (31.7)	15 (39.5)	8 (36.4)
Para ≥ 2	148 (20.2)	36 (10.5)	52 (34.7)	48 (26.7)	7 (18.4)	5 (22.7)
Married (at first visit)	696 (94.8)	330 (95.9)	140 (93.3)	178 (98.9)	31 (81.6)	17 (77.3)

(Continues)

TABLE 1 (Continued)

	Total n = 734 (100%)	Europe n = 344 (46.9%)	Middle East/ North Africa n = 150 (20.4%)	South Asia n = 180 (24.5%)	East Asia 38 (5.2%)	Others 22 (3.0%)
Ethnic origin[a]	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Children						
Sex, boy	374 (51.0)	181 (52.6)	63 (42.0)	96 (53.3)	22 (57.9)	12 (54.3)
Gestational age, days (SD)	280 (11)	281 (11)	279 (11)	278 (11)	277 (12)	278 (10)
Birth weight, g (SD)	3459 (518)	3602 (494)	3455 (526)	3270 (472)	3205 (570)	3217 (330)
Breastfeeding 14 mo postpartum	538 (87.5)	258 (86.6)	105 (91.3)	129 (85.4)	29 (85.3)	17 (100)

[a]Europeans (primarily from Norway and other Scandinavian countries); Middle East/North Africans (includes Horn of Africa, primarily from Iraq, Turkey, Morocco, Afghanistan, Somalia, and Ethiopia); South Asians (primarily from Pakistan and Sri Lanka); East Asians (primarily from Vietnam, Thailand and the Philippines); and Others (from Sub-Saharan Africa, Central-, and South America).

[b]Defined by WHO 1999 criteria; Fasting glucose ≥ 7 or OGTT ≥ 7.8.

[c]OGTT 7.8-8.9.

[d]OGTT > 9.

differed > 14 days from ultrasound term, or the pregnancy was a result of in vitro fertilisation (in total: 7% of pregnancies[16]).

Maternal smoking status was not included as a covariate, as very few, and in particular ethnic minority women, smoked. We did not include variables that were considered to be on the causal pathway between the three maternal factors of interest (ie mediators) and the child's BMI. Breastfeeding, which may be negatively affected by GDM and prepregnant obesity,[19,20] was therefore not included in the mixed models analyses.

2.6 | Statistical analysis

Descriptive statistics are given as frequencies, proportions (%) and means with standard deviation (SD).

We first used linear models to explore associations between exposures and children's BMI at birth. Further, in our primary analyses, we used linear mixed models to explore associations with BMI trajectories from birth to 4-5 years of age. We were primarily interested in the independent association between GDM and offspring BMI trajectories, but also the direct effects of maternal prepregnant BMI, and GWG, that is effects not mediated trough GDM. BMI increases by nature rapidly from birth to 6-8 months of age and then declines until 4-5 years of age, followed by a gradual increase. This represents a complex trajectory for statistical modelling. As more than 95% of all children in our cohort had their highest BMI value at the 6 months visit, we stratified the analyses: (a) from birth to 6 months and (b) from 6 months to 4-5 years. BMI from all time points was entered into the models. We built basic random intercept, random slope models, and the final basic models included children's age (in months), entered both as fixed effects (age and age*age) and as a random effect (age). Maternal and child factors were then included, both as fixed factors and as interaction terms with child age (representing BMI growth). Restricted maximum likelihood was used to estimate the different parameters. Models (each step of the model building process, to avoid overfitting) and covariance structures were compared using Akaike and Bayesian information criteria. The two information criteria were in agreement at all times. An unstructured covariance structure was chosen. In the 'univariate models', explanatory variables were added one by one to the basic model, including the interaction term with age. In the first adjusted model (model 1), we entered GDM, ethnicity, maternal age, parity and socioeconomic position. In model 2, we added gestational age and sex. Finally, in the full model 3, prepregnant BMI and maternal GWG were added. In a supplementary model for growth between 6 months and 4-5 years of age, we also adjusted for BMI growth between 0 and 6 months. Of the 734 children, the majority (671 mother/child pairs) had data on all three main exposure variables as well as all covariates. We have therefore not tested for potential selection bias. In the mixed models analyses estimating mean BMI in relation to one maternal exposure at a time, the two other maternal exposures were adjusted for, thus acting as confounders.
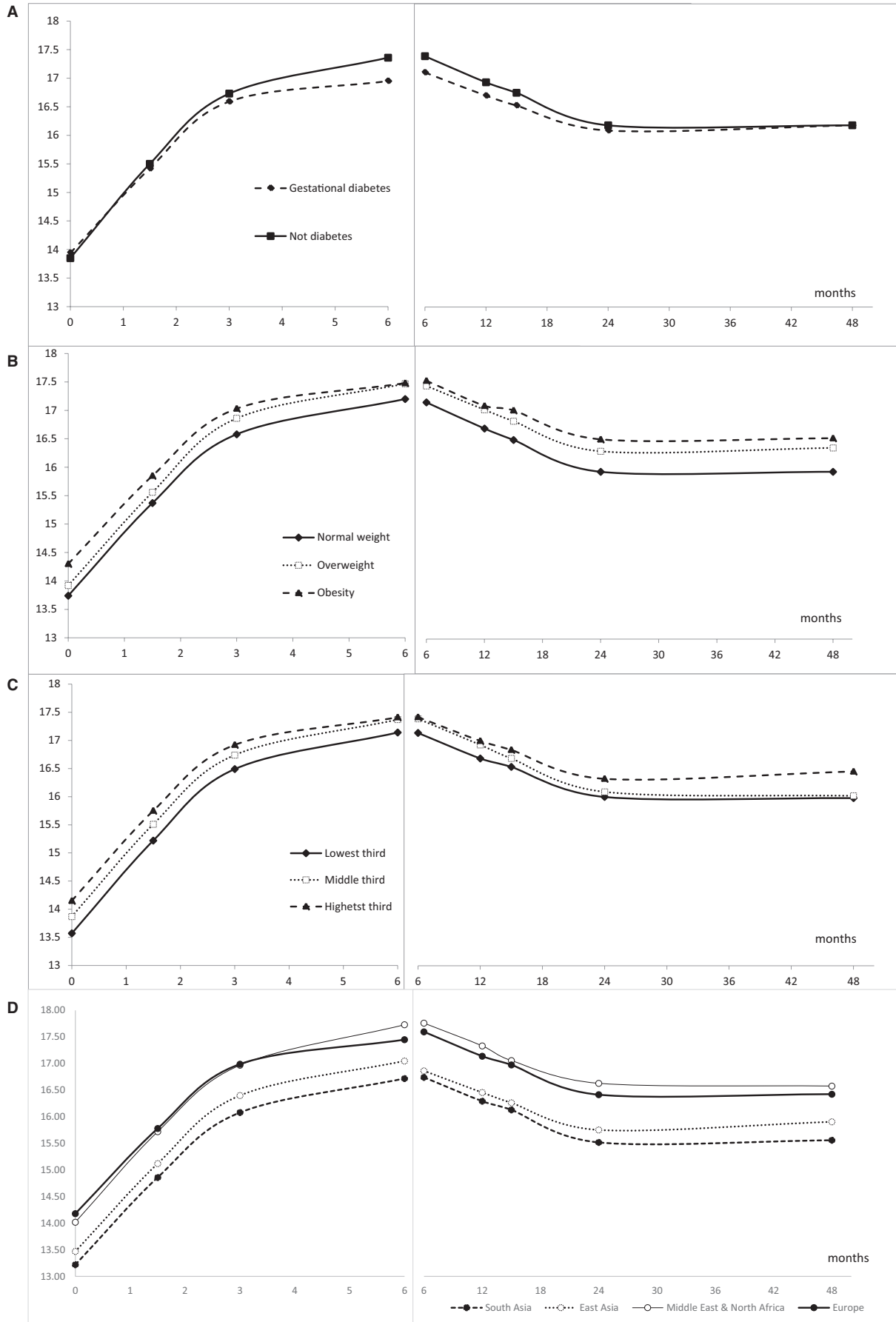
A

B

C

D

FIGURE 2 Children's estimated mean BMI from birth to 4-5 y of age extracted from fully adjusted mixed models in relation to maternal exposures; (A) mothers with and without GDM, (B) maternal prepregnant BMI status and c) maternal gestational weight gain in tertiles. Curves represent estimated mean BMI from eight time points from Mixed models (separate models for 0-6 mo and 6-48 mo), with the following variables included: GDM, ethnicity, maternal age, parity, socioeconomic position, gestational age, sex, maternal prepregnant BMI and maternal gestational weight gain. a. Maternal GDM diagnosed by WHO_{1999} criteria (fasting glucose ≥ 7.0 mmol/L and/or 2-h plasma glucose ≥ 7.8 mmol/L); GDM (n = 92) vs not GDM (n = 624). B, Maternal prepregnant BMI category; normal weight (n = 420), overweight (n = 172), obesity (n = 99). C, Maternal gestational weight gain category in tertiles; highest third (n = 224), middle third (n = 218), lowest third (n = 213). D, Ethnic group; Europe (n = 344), Middle East/North Africa (n = 150), South Asia (n = 180), East Asia (n = 38)

For all analyses, we used SPSS version 22 (IBM SPSS statistics, NY, USA).

3 | RESULTS

3.1 | Sample characteristics

Details for mean BMI (SD) and numbers of children at each check-up visit are given in Table S1. Children's mean age at last follow-up was 4.38 (SD 0.28) years. Of all women, 45.6% were nulliparous, and 16.5% had only primary education or less, although this differed between ethnic groups (Table 1). In total, 12.5% were diagnosed with GDM. Prior to pregnancy, 24.9% of the women were affected by overweight and 14.3% by obesity. Of the 734 children, 50 children were exposed to two of the three maternal factors; GDM, prepregnant obesity or GWG in the highest tertile, while only three were exposed to all three factors. At 14 weeks postpartum, more than 85% of children were breastfed, most of these (69%) exclusively.

3.2 | Associations between maternal factors and children's BMI and BMI growth

General linear models analyses (Table S2) and linear mixed models analyses (Figure 2A, Tables S3 and S4) showed in univariate, and in fully adjusted models, that children exposed to GDM that was treated accordingly, had a similar BMI at birth, compared with non- exposed children (B = 0.227 (BMI units) 95% CI: −0.07, 0.52). However, they had a slower BMI growth (B = −0.106 (BMI units/month) 95% CI: −0.17, −0.04) during the first 6 months, and a faster BMI growth from 6 months to 4-5 years (0.009 (BMI units/month) (0.002, 0.02)). At age 6 months, their BMI was lower (B = −0.489 BMI units (−0.85, −0.13)). At the end of follow-up at 4-5 years of age, their BMI was similar to the non-exposed children (B = −0.013 BMI units (−0.34, 0.32)). In the supplementary model for BMI growth from 6 months to 4-5 years, adjusted for growth 0-6 months, the effect of GDM was no longer significant (data not shown). Thus, a slower BMI growth during the first 6 months was followed by an accelerated BMI growth after 6 months.

Maternal prepregnant obesity was associated with higher offspring BMI at birth (0.42 (0.19, 0.67)) and persistently higher BMI up to 4-5 years (Figure 2B, Tables S2, S3 and S4). Hence, maternal prepregnant obesity was not associated with BMI growth. Compared with the reference (middle tertile), maternal GWG in the highest

tertile was associated with a faster BMI growth from 6 months to 4-5 years (0.007 (0.001, 0.01)) (Figure 2C), but not with BMI growth between birth and 6 months. Maternal GWG (lowest tertile) was associated with a lower BMI at birth (−0.23 (−0.42, −0.05)) (Figure 2C, Tables S2, S3 and S4). We did not have statistical power to perform formal tests for interaction between ethnicity and the maternal factors. However, stratified analysis showed that the relations of children's BMI growth with GDM, maternal prepregnant obesity and GWG were similar in all ethnic groups (ie similar effect estimates, although they did not reach statistical significance).

3.3 | Associations with ethnicity

Both in univariate analyses, and when taking all other maternal factors into account, children with Middle Eastern or North African origin had a slightly lower BMI at birth, but a faster BMI growth from birth to 6 months, compared with ethnic European children (Figure 2D, Tables S2, S3 and S4). Children with South Asian origin had a substantially lower BMI at birth, and their BMI development did not differ significantly from children with ethnic European origin. Thus, their mean BMI remained lower up to the age of 4-5 years. Ethnic East Asian children's growth patterns were similar to the ethnic South Asian's with a lower BMI at birth, followed by a stable lower BMI.

4 | DISCUSSION

To the best of our knowledge, this is the first longitudinal study exploring the associations between the maternal exposures GDM, prepregnant obesity, GWG and early childhood BMI trajectories in a multi-ethnic cohort from Europe. We found that these three factors were all associated with offspring BMI and with BMI development from birth to 4-5 years. However, the timing, direction and size of these effects differed. Although children exposed to GDM that was treated did not differ in BMI at birth, they had a catch-down BMI growth from birth to 6 months, followed by a faster BMI growth up to 4-5 years, compared with children not exposed to GDM. In contrast, maternal prepregnant obesity was associated with a persistently higher BMI from birth to 4-5 years, while high maternal GWG was associated with a faster BMI growth from 6 months to 4-5 years. These effects were similar in all ethnic groups.

We observed an early catch-down BMI development during the first 6 months of life in GDM exposed children, in accordance

with some earlier studies.[21,22] However, these studies also included women with previous or newly diagnosed type 2 diabetes (ie more severe GDM), and their children's birthweights were higher than unexposed children. We demonstrate that a catch-down development was also present in a cohort of children who were predominantly exposed to relatively mild, treated GDM, with a birthweight similar to non-exposed children. Contrary to our results, most observational studies have shown that GDM is associated with higher weight and adiposity at birth in the offspring.[8,23] This discrepancy is probably related to the fact that mothers diagnosed with GDM in our study were informed and referred for treatment.[13] Furthermore, many had a relatively mild GDM, as our sample represents a generally healthy population that was universally screened with oral glucose tolerance test.

Randomised control trials have shown that treatment of GDM reduces the risk of macrosomia at birth, as well as perinatal and neonatal complications.[24,25] A recent study found that GDM requiring medication treatment was associated with children's higher and increasing BMI trajectory, while GDM not requiring medication had little association.[26] A meta-analysis of observational studies showed that children exposed to GDM in utero had a higher risk of being affected by overweight and obesity and a higher BMI z-score from 10 to 11 years of age, compared with children not exposed.[5] This association was not found in the younger age groups. However, the quality of evidence was considered as low as most studies did not adjust for other maternal factors, and different definitions of GDM were used. Follow-up studies on data from the children participating in the large Hyperglycaemia and Adverse Pregnancy Outcomes study, blinded for glucose levels in pregnancy, did not find effects of GDM on overweight and adiposity at age 2 years, but emerging at 11 years age.[2,23] Our results, showing a catch-down BMI growth from birth to 6 months, followed by a faster BMI growth up to 4-5 years, could provide a possible explanation for this inconsistency, if the observed acceleration in BMI continues into school years and adolescence.

In contrast to the effect of GDM, maternal prepregnant obesity and excessive GWG have been shown to independently increase the risk of adiposity at all life stages in the offspring.[27] A recent study showed that prepregnant obesity and excessive GWG were associated with child growth trajectory characterised by rapid weight gain during first year of life, and stable high BMI until age four years.[28] GDM was not significantly associated with this trajectory. As this study had no measurements between birth and age 1 year, it could not tell when children exposed to excessive GWG started their accelerated growth during their first year of life. Our findings of a different timing, direction, and size of the effects of GDM, prepregnant obesity and GWG may shed light on the mechanisms underlying the varying observations across observational studies. In line with other previous studies,[29,30] we found striking ethnic differences in BMI development. However, the associations with prepregnant obesity, GWG and GDM persisted after adjustments for ethnicity and were similar in all ethnic groups. We have not found any studies on children's BMI development that assess the effects of all these three factors, and with the same ethnic groups as ours.

4.1 | Strengths and limitations

Strengths of the present study include the population-based cohort design, the large proportion of ethnic minorities, otherwise often excluded in research, and a high attendance rate. The data set for maternal exposures and confounders is extensive, including universal oral glucose tolerance tests and various measures of socioeconomic position. We have measures of the children's BMI at birth and at seven time points up to age 4-5 years, with minor loss to follow-up of children. The analytic models included potential confounders identified by DAGs, reducing the risk of biased estimates for the associations between maternal factors and children's BMI development. Limitations should also be noted: Although trained child health clinic personnel collected routine anthropometric data on children, and measurements performed under standardised conditions would have been more accurate, this probably applies equally to all ethnic groups and status for maternal exposures. With 734 children in our multi-ethnic cohort, the power was limited, especially related to exploring potential effect modifications. We merged women into five ethnic groups, due to small numbers for many countries of origin, which may have caused some within-group heterogeneity. Furthermore, BMI has limitations as a measure for body fatness and as predictor of the development of obesity with considerable ethnic differences between BMI and objectively measured fatness in preschool years.[29] Further, we must assume that there are unmeasured confounders to the relationships of interest in this study, especially concerning lifestyle and socioeconomic factors. Another possible confounder is parental size at birth. However, as more than half of the participating mothers in this cohort were women born low- or middle-income countries, many were not aware of their birthweight (were born at home or did not have access to birth records). We were therefore not able to take this possible confounder into account. Also, we excluded 19 children from our study sample due to severe medical conditions obviously affecting growth, but have not collected detailed information on children's diseases and medication use. This could potentially also lead to residual confounding. Last, in this study, we used self-reported prepregnant weight to calculate maternal prepregnant BMI, which might be sensitive to recall bias. However, a strong correlation between the prepregnant body weight and the measured body weight at enrolment at mean gestational week 15 ($r = 0.97$), with no significant differences across ethnic groups, suggest that this is not an important source of bias in the present study.

5 | CONCLUSION

In summary, the effects of intrauterine exposure to GDM, prepregnant obesity and gestational weight gain on offspring BMI trajectories from birth to 4-5 years differed concerning both direction, timing and size. Health professionals following children's growth should be aware that children born of mothers with GDM that is treated might have a natural catch-down BMI development during

the first 6 months of life, also when birthweight is normal. More longitudinal studies exploring additive effects of maternal factors for offspring growth in later childhood, adolescence and into adulthood are important for breaking the intergenerational circle of obesity and diabetes.

CONFLICT OF INTEREST

The authors declare that there is no duality of interests associated with this manuscript.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.