Predictors of underweight, overweight and obesity, and effect of treatment of obesity in early childhood

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Thesis for the degree of Philosophiae Doctor (PhD)
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Scientific environment

This thesis was part of "The Oppland health and growth study", which is a cohort study with overweight and obesity in childhood as the main focus. The study was established by Trond Markestad and Jørgen Hurum at the Department for Paediatric and Adolescent Medicine, Innlandet Hospital Trust, Lillehammer.

My main supervisor was Professor Trond Markestad, University of Bergen, and also research advisor at the Department of Research, Innlandet Hospital Trust. Assistant supervisors were Professor Pétur B. Júlíusson and Professor Robert Bjerknes, Department of Clinical Science, University of Bergen. The PhD was funded by Innlandet Hospital Trust, and "The Oppland health and growth study" also received grants from The South-Eastern Norway Regional Health Authority (Helse Sør-Øst). My affiliation was the Department of Paediatric and Adolescent Medicine, Innlandet Hospital Trust, Lillehammer.

Paediatrician Jacob Holter Grundt, who earlier worked at the Department for Paediatric and Adolescent Medicine, Innlandet Hospital Trust, Lillehammer, was co-author in papers I and II. Psychologist Helene Toxe, Clinic of Child and Adolescent Psychiatry, Innlandet Hospital Trust, Otta, was co-author in paper III. Biostatistician Geir Egil Eide, Centre for Clinical Research, Haukeland University Hospital, provided statistical support.

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My colleague and co-author of the first paper, Jacob Holter Grundt, was the first PhD-student in our research group. We worked together with data cleaning, analyses, discussions and writing. I especially remember several evenings in 2013, where we worked with data cleaning and merging files with baby Julian on my lap, and the late night work sessions writing the article together after the kids were in bed. Jacob, I am grateful for all our interesting discussions, our friendship and your enthusiasm.

My good friend and colleague Asborg Aanstad Bjertnæs joined our research group a bit later than me, and started her PhD with studies on the children from OHGS at a later time point. Asborg, I have really appreciated all our scientific discussions, all your support, and most of all, thank you for our friendship.

Psychologist and co-author Helene Toxe wrote her specialist paper about the psychological health of children with overweight and obesity, with data from OHGS. Thank you for choosing this theme, as this was the inspiration for paper III. I have really appreciated our discussions and collaboration, and all your support.

The OHGS has been a large, ongoing study for many years, and the research group has consisted of several people connected to the Department of Paediatric and Adolescent Medicine in Lillehammer and Gjøvik. In addition to the members already mentioned, I am indebted to registered nurses Anne Berit K. Sundby, Ragnhild Gunstad, Turid Skundberg, Liv Thorsen and Randi Asbjørnsen, physiotherapists Karianne Storesund, Randi Bragelien and Merete Welhaven Steen, nutritionists Kristina W. Monsbakken and Eli Nyhagen, preschool teachers Gro Lisbeth Hofstad and Gjertrud Skundberg and paediatrician Halvor Bævre for their invaluable contributions. Anne Berit deserve special thanks for all the numerous hours used on phone calls, letters and data punching, always with a smile.

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Contents

Abstract

Background:

The prevalence of overweight (OW) and obesity (OB) has escalated throughout the world over the last decades, both in children and adults. Childhood OB is a major risk factor for adult OB and the subsequent increased risk of major morbidities secondary to OB. Consequently, research on prevention and early interventions play a key role in order to understand how to curtail this epidemic. To be able to prevent or treat OB in childhood, it is crucial to understand the complexity of causes and risk factors, such as effects of societal and family contributors and psychological mechanisms.

Aims:

The overall aim of this thesis was to investigate associations between the development of unhealthy weights in early childhood and family- and child-related health and behavioural factors, and to test the effect of an intervention program to curtail OB in this age group.

Methods:

The study was based on the Oppland Health and Growth Study (OHGS), which is a cross-sectional study of all the children in the county who met for the school entry health assessment at 5-6 years of age in 2007. Height and weight were measured by midwives at birth, and later by public health nurses and study nurses. Parents of consenting families completed questionnaires on sociodemographic and family- and child-related health and lifestyle factors. They assessed their child's psychological health with the Strength and Difficulties Questionnaire (SDQ). The public health nurses reported age, sex, height and weight anonymously for the children of families who declined to participate.

In two of the papers we explored the associations between the background factors and the weight categories underweight (UW), overweight (OW) and obesity (OB) of the children at school entry. Children with normal weight (NW) were the reference.

In the third paper, we investigated the effect of a three-year multidisciplinary intervention programme to curtail OB. The program was group-based and only addressed the parents. We recruited children with OB, mostly from the OHGS, from eight of the municipalities for the intervention, while the rest of the OHGS cohort with OB served as controls without any interventions. The main outcome was the change in body mass index standard deviation score (BMI SDS) over the three years. Within the intervention group we also measured skinfolds, waist circumference and 6-minute walk test and assessed potential success factors based on the initial measurements and the background variables.

Results:

The prevalence of UW, OW and OB was 7.8 %, 10.6 % and 3.5 %, respectively, at a mean age (standard deviation -SD) of 5.70 (0.49) years. The parents of 1119 of 1895 eligible children (59%) gave consent and provided background information. The prevalence of UW, OW and OB was slightly lower among the participants.

In bivariate analyses, UW was related to weight, weight SDS and BMI SDS at birth, and BMI of parents and siblings, but none of the sociodemographic or behavioural factors. OW and OB were related to low education and high BMI of parents, mother smoking and having no siblings. In addition, OB was related to exclusive breastfeeding less than 4 months, dental caries, less physical activity than peers, TV in the child's bedroom, father not working, non-western ethnicity, and living with one caretaker.

In adjusted analyses, UW was only related to the children's crown-heel-length-SDS and the BMI of the father. OW was associated with birthweight SDS, parental BMI, having no siblings, low maternal education and maternal smoking, and OB to maternal BMI, low maternal education and maternal smoking. The relative risk of overweight or obesity (OWOB) increased with increasing strata of low education and OWOB in the parents.

Psychological symptoms, as assessed with the SDQ, had curvilinear associations between mean scores on the SDQ subscales Emotional Problems and Peer Problems,

with higher scores for UW and OB and nadir for normal weight (NW). Furthermore, the Total Difficulties score (TDS) and Total Difficulties above the 90th percentile (TDS90) had similar patterns, and with significantly higher scores for children with UW and OB than NW. However, TDS90 was only significantly associated with UW after adjustments for the socioeconomic and lifestyle variables, and for the children's difficulties with sleep or fine motor, language or social skills.

In the intervention study, 31 children completed the intervention and 33 the control period. The median decline in BMI SDS was the same in both groups (0.19 BMI SDS). A higher BMI SDS at entry was similarly associated with a larger decline in BMI SDS in both the intervention and control group. None of the other relevant variables were related to the outcome. Within the intervention group, only age and mean skinfold SDS at entry were significantly related to change in BMI SDS.

Conclusions

Since environmental factors were associated with OW and OB, but not with UW, we suggest that the environmental factors were the most important risk factors for OW and OB in preschool children. UW, but not OW or OB, was associated with psychological symptoms after adjusting for environmental factors. Our interpretation is that psychological symptoms were neither a cause nor a consequence of OW or OB, while psychological difficulties may be a cause or a consequence of UW. A three-year multidisciplinary intervention programme had no effect over no intervention on the development of BMI SDS. Hence, early prevention is of vital importance in order to limit the obesity epidemic.

Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
BMI	Body Mass Index (kg/m^2)
BW	Birth weight
IOTF	International Obesity Task Force
LGA	Large for gestational age (birthweight> 90^{th} percentile)
LMS	Learning and Mastery Service
OHGS	Oppland Health and Growth Study
OB	Obesity
OPD	Oppland Perinatal Database
OW	Overweight
OWOB	Overweight including Obesity
SDS	Standard Deviation Score
SDQ	Strengths and Difficulties Questionnaire
SES	Socioeconomic Status
SGA	Small for gestational age (birthweight < 10^{th} percentile)
TDS	Total Difficulties Score
TDS90	Total Difficulties Score > 90^{th} percentile
UW	Underweight
WC	Waist circumference
WHO	World Health Organization
WHtR	Waist-to-Height Ratio

List of Publications

I Donkor HM, Grundt JH, Júlíusson PB, Eide GE, Hurum J, Bjerknes R, Markestad T. Social and Somatic Determinants of Underweight, Overweight and Obesity at 5 years of Age: A Norwegian Regional Cohort Study. BMJ Open. 2017 Aug 18;7(8):e014548. doi: 10.1136/bmjopen-2016-014548.

II Donkor HM, Toxe H, Hurum J, Bjerknes R, Eide GE, Júlíusson PB, Markestad T. Psychological health in preschool children with underweight, overweight or obesity. Submitted June 2020.

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1. Introduction

1.1 Background for the study

Over the last decades, the rates of overweight and obesity (OWOB) have been increasing throughout the world. The World Health Organization (WHO) have called this an epidemic [1], and OWOB is the cause of death of over 4 million people each year according to the Global Burden of Disease [2, 3]. The OWOB epidemic started in the high-income countries, but over the last years, the problem has been rising in the low- and middle-income countries [4, 5]. The low-income countries have a double burden of both malnutrition and obesity (OB), and it is now estimated that there are more people with OWOB than underweight (UW) in most regions of the world, except parts of Sub-Saharan Africa and Asia [6].

The OWOB epidemic also involves children [4]. During childhood, OWOB is associated with significant somatic and mental health challenges [7-10]. Maybe more important, OWOB during childhood is a major risk factor for adult OB and the subsequent increased risk of major morbidities secondary to OB, such as diabetes, cardiovascular diseases, musculoskeletal and mental diseases, and premature death [10-15].

Since OWOB in childhood is an important risk factor for OWOB in adulthood, research on prevention and early interventions at a time when children develop an unhealthy weight trajectory play a key role in order to understand how to curtail this epidemic. To be able to prevent or treat OWOB in childhood, it is crucial to understand the complexity of causes and risk factors, such as genetic predispositions, effects of societal and family stressors, psychological mechanisms, and mental health issues that are probably important, but poorly understood [16-18].

So far, intervention studies in childhood OB have had limited success [19-23]. Many of the studies had short follow-up periods and high risks of bias. Family-based

interventions addressing combinations of physical activity, nutrition and other behavioural components have shown the most promising results [23].

The significance of UW is extensively studied in relation to anorexia nervosa and bulimia nervosa in older children and adolescents [24-26], but has received little attention in young children except in the context of diseases, undernutrition and malnutrition.

1.2 Definitions of overweight (OW), obesity (OB) and underweight (UW)

OW and OB may be defined as an excessive amount of fat in such a way that it represents a health risk, as stated by WHO [1]. The normal amount of fat mass and fat free mass in the body vary with gender, age, genetic factors and fitness.

There are many different ways to measure fat mass, but there is currently no consensus on the best method. So far, Dual energy X-ray absorptiometry (DXA) has been considered the best method to measure body composition in some studies [27]. Magnetic resonance imaging (MRI), computerized tomography (CT), under-water weighing, bioelectrical impedance analysis, air displacement plethysmography, and several other methods are also being used [28]. These methods are, however, expensive, cumbersome and not easily available, and therefore mainly used in specific research settings. Body fat reference curves have been developed on basis of measurements by bioelectrical impedance, also for children [29], but to my knowledge, it has rarely been used in published in epidemiological studies.

However, the international definitions of OWOB are based on body mass index (BMI) defined as the weight (in kg) divided by the square of the height (in meters, BMI=kg/m^2). In adults, OW and OB are defined as a BMI of at least 25 and 30, respectively [1]. Because children are growing and the body shape is changing throughout childhood, the definitions of OW and OB depend on the age and sex of the child. The International Obesity Task Force (IOTF) has published percentiles for

BMI and cut-offs for OW and OB in children adjusted for age and sex [30, 31], and these limits are now incorporated in the Norwegian BMI percentiles charts [32, 33]. The United States and some other countries use definitions based on percentiles in BMI growth charts, where a BMI above the 85th percentile is defined as OW and a BMI above the 95th percentile as OB [34]. The WHO has also developed a definition of childhood OWOB based on Standard Deviation Scores (SDS) [35], where OW is defined as more than one standard deviation (SD) above mean and OB as above 2 SD. However, the IOTF cut-offs are the most frequently used.

The BMI definitions for OWOB in childhood are internationally accepted and widely used. Nevertheless, BMI does not differentiate between fat and fat free mass, and offers no information on fat distribution. However, high BMIs are closely correlated to high fat mass [36-38], and studies indicate that BMI has high specificity, but low sensitivity to detect excess adiposity [39]. BMI is considered a fairly good measure of OWOB in a population setting, but might be misleading on an individual basis. For instance, children with high muscle and bone mass may have a relatively high BMI without significant adiposity, and tall children have systematically higher BMIs than children of average height [40]. It has also been shown that some ethnicities, like Asians, may have higher fat mass at a lower BMI than Caucasians, questioning if Asian should have their own definitions for OWOB [41]. Despite all these uncertainties, BMI currently appears to be the best available method to define OW and OB, and the international definitions make it easier to collaborate in research. Assessment of BMI is also non-invasive and cheap, and is already a part of the child health care programs in most countries.

Definitions of underweight (UW) in childhood adjusted for age and sex, were published in 2007 [42] and are also incorporated in the Norwegian growth charts [32, 33].

It is crucial to monitor the height and weight in children, as many different diseases and conditions may affect linear growth and weight. Many countries, like Norway, follow almost all children in child health care clinics and through school programs,

and they have their own growth charts. In Norway, OB or UW in childhood were previously assessed by plotting the weight in relation to height and age on a percentile chart [43]. The WHO has constructed percentile charts for 0-5-year-old children based on studies of breastfed children in six countries thought to be representative for children throughout the world [44]. However, there is evidence that the natural growth of children in some countries deviate from this pattern, including Norway [45-48]. Consequently, there is still an ongoing debate on whether to use national or international references. It might be appropriate to use national references to monitor the growth of the individual child, and international references on a population basis and in research.

1.2.1 BMI Standard deviation score (SDS) and BMI increments

The BMI SDS are age- and sex-adjusted values that describe the distance from the mean divided by the standard error. Since BMI SDS is age-adjusted, it is generally accepted as a sensible way to follow a child's growth, and some argue that this parameter is the best available to predict fat loss [49]. On the other side, some studies have shown that the BMI SDS has limitations for the children with severe OB [50]. A certain percentage above BMI limits, like the IOTF limit for OW, has been suggested as an alternative method [51].

BMI increments represent the change in BMI over time, such as yearly. An annual increase of more than two standard deviations have been associated with a rapid increase in body fat mass [52]. Conditional change in BMI SDS might be an alternative method to assess the BMI changes [53].

1.2.2 Waist and skinfold measurements

Waist circumference (WC) represents central adiposity and is closely associated to OB. Some studies have shown that WC is more strongly associated to fat mass and cardiometabolic risk than BMI [54, 55]. WC is a simple and non-expensive examination, but there might be significant inter- and intra-examiner variation. Norwegian WC percentiles with cut-offs for OW and OB were published in 2011 [56].

Waist-to-height ratio (WHtR), the ratio between the WC and height, is also strongly related to OWOB and fat mass [57-60]. In adults, a ratio above 0.5 has been suggested as a definition of OB, while there is no such consensus for children. However, there are Norwegian references for children [56].

Skinfolds, both triceps and subscapular, represent subcutaneous fat, and have been shown to represent body fat mass as well [61]. Nevertheless, skinfold measurements are technically difficult and measurements are prone to be inaccurate. Some studies have shown that BMI is equally precise in order to assess excess body fat [62]. Norwegian references were published in 2013 [63].

1.3 Prevalence

The explosive increase in OWOB in the world is alarming. According to the Global Burden of Disease, the global prevalence of OWOB in adults was 29 % in 1980, rising to 38 % in 2013 [4]. Worldwide, the prevalence of OB has nearly tripled since 1975, and according to the WHO, most of the population in the world live in countries where the mortality from OW and OB exceeds the mortality from UW [64]. In some countries, more than 50 % of the adults have OB [4]. The increase of OWOB in children has been equally alarming. The prevalence of OW or OB among children in high-income countries was 23 % in 2013 compared to 16 % in 1980. For children in low- and middle-income countries the prevalence increased from 8 % to 13 % during the same period [4]. The global prevalence of OWOB in children has increased more than four-fold from 1975 to 2016, i.e. from 4 % to 18 % [6], and the WHO estimated that there are currently 38 million children under the age of 5 years with OWOB in the world [64].

The rapid rise in the prevalence of OWOB started in the high-income countries, but during the last two decades the increase has been more severe in the low- and middle-income countries. The low- and middle-income countries face the double burden of both malnutrition and OWOB, and malnutrition can even affect the persons with OW and OB because of high intakes of energy-dense food that are low on essential nutrients.

In Norway, the prevalence of OB was 5 % for men and 13 % for women in the 1960s [65], but has increased to around 25 % of the adult population during the last decade [66, 67]. The prevalence among Norwegian children has also increased [68], and around 15-20 % of the children are now OW or OB. The prevalence vary with age in that around 15 % of 8-9 year-old children [69] and 20-28 % of adolescents [70, 71] are OW or OB. Norwegian data from the National Institute of Public Health are part of the WHO Childhood Obesity Surveillance Initiative, where we see a North-South gradient with a higher prevalence in the southern part of Europe [72, 73]. In some countries, like Norway, the prevalence of OWOB in childhood might have reached a plateau during the last decade [69, 74].

1.4 Causes

The logical background for OWOB is an excess of calories where the intake of calories over time exceeds both the basic metabolism and the calories burned through physical activity. The reason for this surplus energy is, however, much more complex, with a variety of intertwining risk factors.

1.4.1 Genetics

From twin- and adoption studies it has been estimated that 40-90 % of the variation in BMI is explained by heritable factors [75, 76]. There is also strong evidence for an interaction between genetic and environmental factors.

In rare cases, a single gene disorder is the cause of OWOB. The most common is a mutation in the melanocortin 4 receptor (MC4R) gene. In some studies persons with this genetic characteristic account for up to 5-8 % of adults and children with morbid OB [77-79]. Mutations in the leptin gene, the leptin receptor gene, the proopiomelanocortin (POMC) gene and the prohormonconvertase 1 (PC1) gene account for some of the other known single-gene defects in obesity, and all these possible mutations are involved in the regulation of appetite [80]. Children with these single gene defects usually develop OB at a very young age, as well as taller-than

average height and other more specific symptoms and signs in each of these mutations.

Several monogenic syndromes include OB as one of the clinical findings; the most common of them is the Prader Willi syndrome [81]. OB may also be a part of other and more common syndromes, like Down syndrome (trisomy 21).

However, the main genetic contribution to OWOB involves polygenic inheritance, also called common obesity. The complexity of the polygenic inheritance of OW and OB is not yet fully understood, but some of the genes and mechanisms have been discovered. The FTO-gene (fat mass and obesity) on chromosome 16 is one of the best documented genes associated with OB [82, 83], and studies have estimated an odds ratio of about 1.5 for the homozygote of this gene to be OB [83]. So far, more than 200 genetic loci have been linked to OB [84, 85].

The complex interaction between genes and environment is expressed through epigenetic mechanisms, where environmental influences change the gene expression. There are several studies suggesting that epigenetic changes, like *d*eoxyribo*n*ucleic *a*cid (DNA) methylation, is associated with OB [84, 86].

1.4.2 Obesogenic environment

During the recent few decades, the society has changed markedly in terms of availability of energy dense food and snacks and sugar sweetened beverages, and in terms of decreased needs of physical activity. People drive instead of walk even on short distances, and the majority of work is sedentary. All of these changes are part of the term “the obesogenic environment” [87], where the environment promotes unhealthy choices. Combined with genetic vulnerability, today’s society encourages weight gain.

Daily physical activity has decreased because most work is less physically demanding and most transportation is motorized. Schoolchildren also spend most of their days sitting in the classroom. The Norwegian Directorate of Health recommends one hour of daily physical activity for children and at least 150 minutes of moderate

activity per week for adults [88]. About 80-90 % of Norwegian children in primary school meet the recommendations, as opposed to only 50 % of the 15-year-olds and 30 % of adults [89]. Studies suggest that physical activity during leisure time has increased, but still the majority of the adolescents and adults in the world are mainly inactive [90]. Studies have shown that children with OW and OB are less active than other children [91-93], but to what extent less activity predisposes to OB or is a result of OB is not clear.

As physical acidity has decreased, screen time has increased both at home and in schools. Many municipalities in Norway distribute lap top computers or tablets to all the children in primary school for homework and work during school hours. In addition, an increasing number of children have their own cell phones, many of them as smart phones. These changes encourage screen time, and many studies have shown that screen time is associated with OWOB in children [94-96].

During the last decades, the food intake has shifted to more energy dense food, fast food, snacks and sugar sweetened beverages, and these changes may be one of the main drivers of the obesity epidemic [97-99]. However, during the last couple of decades, the artificially sweetened beverages have gained popularity, and since year 2000 the sugar intake has decreased and subsequently stabilized in Norway [89]. The eating habits differ across Europe [100], and both from comparisons between and within nations the significance of nutritional habits to OB is unquestionable. It is not clear, however, which of the unhealthy eating habits that have the largest impact on weight [101].

1.4.3 Socioeconomic factors

Several socioeconomic factors are associated with childhood OWOB. The parents' level of education is an important indicator of socioeconomic status (SES), and low education is strongly linked to childhood OB in high-income countries [102-104]. Household income and parental employment are other important SES factors, and both are inversely associated with OWOB in high-income countries [102]. In Norway, several studies have documented the association between SES and

childhood OWOB [95, 105-108]. In low-income countries the association is the opposite in that OWOB is related to high SES [109].

The drivers of this strong association between SES and OB are probably complex. In areas with high SES the citizens have purchasing power to make healthy dietary choices [95, 105, 110, 111] and opportunities to engage in physical activities in suitable arenas [105, 112, 113]. Their higher education and economic privileges give them opportunities and create willingness to change behaviour according to medical advice on nutrition, physical activity and on, for instance, limiting screen time [95, 105, 110, 112, 113].

1.4.4 Family relations and anthropometrics

The family is an important basis of growing up. A loving family with healthy eating habits, active living, appropriate boundary settings and good psychological support facilitates the raising of healthy and robust children. Divided and blended families are frequent in today's society, and may cause several challenges for both parents and their children. Single caretakers might have demanding everyday lives, both economically, physically and psychologically. To experience a parental divorce might also cause psychological stress for the children, which may lead to different eating patterns like over- or undereating. Many studies have shown that single parenthood is associated with childhood OWOB [114-116]. Having siblings seem to decrease the risk of being OW or OB [106, 117].

Parent OW and OB are among the strongest risk factors for childhood OWOB [76, 114, 116, 118-120]. The reasons are probably genetic susceptibilities combined with the family's habits in terms of diet and physical activity. The intrauterine environment may also be of significance since being born large for gestational age (LGA) as well as small for gestational age (SGA) seem to increase the risk of later OWOB [121-123].

1.4.5 Urbanity

Norway covers an area of 323 778 km^2, but permanent living quarters and industries cover only 1.7 % of the land area [124]. Of the total population, 82 % live in urban

settlements [125]. Oppland county has only two small cities with a population of 25.000-30.000 in each, but 59.3 % of the people live in densely populated areas [125]. Several studies have found that OWOB in children is more common in rural than urban areas in high-income countries [108, 109, 120]. As for SES, the association between urban living and childhood OWOB in low-income countries is opposite to that of high-income countries [64, 109, 126].

1.4.6 Mental health

Several studies have shown a correlation between psychological health and OW and OB in children and adults, but the direction of the association is unclear. Both depression [7, 17, 127, 128], poorer quality of life [7, 129], lower self-esteem [7], and behavioural and emotional problems [7, 130] seem to be associated with OWOB. Studies on relationships between attention deficit hyperactivity disorder (ADHD) and OWOB are conflicting in that ADHD is associated with OWOB in some [131], but not in other studies [132, 133]. Few studies have explored relationships between psychological symptoms and UW in children.

1.5 Consequences

OW and OB in childhood are a major risk factor for OB in adulthood. This tracking of OWOB into adulthood has been documented in several studies [13, 134-137]. Many of the possible consequences of OWOB may not emerge until adulthood, but an increasing number of children and adolescents have experienced early somatic complications as the OB epidemic has proceeded [8, 9]. Cardiovascular disease is an important complication of OB [10, 12, 14, 135], but some studies have found that the risk of cardiovascular disease for adults who had NW, but had OB in childhood, was similar to the risk for adults who had NW both as adults and in childhood [135]. Diabetes type 2 [15, 138] or reduced insulin sensitivity are also frequent complications of OB, as well as musculoskeletal disorders [139], liver steatosis[138], sleep apnoea [109], asthma [140] and certain types of cancer [109]. Some studies indicate a U-shaped relationship between BMI and morbidity [141]. One study found

that poorer global health and special health care needs were associated with UW at preschool age, but with OB in older children [142].

The psychological health might also be affected by being OW or OB, and reduced psychological wellbeing is often the first consequence of OWOB in childhood [7, 10, 130]. For instance, children with OWOB are at higher risk of being bullied [143]. It is not clear, however, to what extent psychological problems may be a consequence or a precursor of OW and OB [7, 18, 144].

1.6 Prevention and treatment

Treatment of OW and OB is difficult. The tracking from childhood to adult OB, and the potential consequences of OB, make it especially important to try to prevent and curtail OB during childhood. For children with OW or moderate OB it is usually not recommended to lose weight, but rather to stabilize the weight and thereby "grow into" their increasing height. At least theoretically, early childhood ought to be the most important age for prevention and early treatment since the parents are responsible for the food available in the house and for establishing an active and non-obesogenic environment.

Prevention and early treatment are the internationally recommended primary approach to avoid preventable diseases, such as OB and consequences of OB. This is particularly relevant for OB since treatment of OB during late childhood and adulthood by ways of changing habits has proven extremely difficult [145]. Mark Hanson has illustrated this approach with reference to OB (Figure 1). As age and severity of a disease increases, the possibility of obtaining an effect on morbidity and mortality (plasticity) decreases (Figure 1).

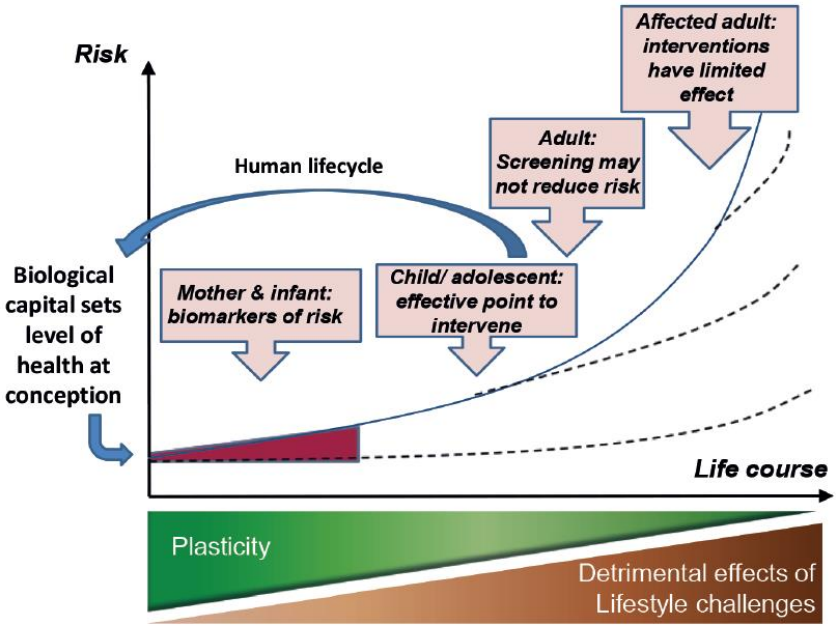

Figure 1. *Mark Hanson. In: Nurturing Human Capital along the Life Course: Investing in Early Childhood Development, World Health Organization, Geneva 2013 [146]. Reprinted with the permission of the author.*

However, interventions to curtail OW and OB, even in childhood, have so far had very limited success [19-22]. Some interventions in childhood and adolescence have had some short-term effect [147], but several Cochrane reviews have concluded that many studies are of poor quality and with too short follow-up periods to be conclusive in situations where some effect was obtained [19-22]. Family oriented interventions have been the most promising strategies, and the most successful interventions target behaviour, in particular related to both diet and physical activity [19, 20, 22]. Some studies show better results of interventions in younger than older children [148], but few studies have addressed very young children, e.g. at preschool age. Nevertheless, there is no consensus on either the content, intensity or duration of the interventions, or on the specific goal for the interventions. Any reduction in BMI

SDS might be clinically beneficial for the individual child, but the necessary BMI SDS reduction to improve comorbidities is not clear [149]. One study showed improvement in metabolic health from a BMI SDS reduction of ≥0.25, but a higher benefit with a BMI SDS reduction of ≥0.50[150]. A Norwegian study reported lower serum cholesterol levels with a BMI SDS reduction of <0.1 units [151].

Pharmacological options to treat severe OB do exist [152, 153]. In Norway, the drug Orlistat, which inhibits lipase and increases faecal loss of triglycerides, is the only of the available drugs approved for children, but only from 12 years of age. Studies have shown moderate positive results, but unwanted side effects are quite common [153, 154]. The treatment appears to be rarely used in children in Norway. A Cochrane review concluded that these drugs might have a short-term benefit, while long term data are non-existing [153]. Bariatric surgery is not an option for the youngest children, but there are countries and studies who include adolescents under the age of 18 [152, 155]. In Norway, the ongoing study 4XL offers bariatric surgery for adolescents aged 13-18 years with morbid OB, but only after extensive investigation, information and selection [156]. So far, studies on bariatric surgery in adolescents have shown promising results on BMI reduction and improvement in comorbidities, but side effects might be concerning, and further research is needed to examine long-term effects [155, 157].

Since childhood OB is difficult to reverse, primary prevention is probably a better strategy. The number of interventions to prevent OB are rapidly increasing. These programs may involve children of all weight groups [158]. The latest Cochrane-review suggested that interventions combining diet and physical activity can reduce the risk of OB in young children (0-5 years), while interventions focusing on physical activity or diet alone had no effect in this age group [158]. For older children (age 6-12 years) and adolescents (age 13-18 years) interventions focusing on physical activity alone, but not on diet alone, seemed to reduce the risk of OB, while there was some evidence that the combination of focus on diet and physical activity reduced the risk of OB. The review found no reports on adverse effects or increasing health inequalities from the interventions.

The WHO has suggested several steps towards halting the rise in childhood OB, and most of these steps are preventive measures [159]. Societies have mostly focused on the individual responsibility and recommended individual changes in behaviour, in particular related to diet and physical activity [160]. However, to curtail this epidemic, the whole society needs to take additional responsibility in promoting national and international actions to tackle the obesogenic environment [160].

2. Aims of the study

The overall aim of the study was to investigate associations between the development of unhealthy weights in early childhood and family- and child-related health and behavioural factors, and to test the effect of an intervention program to curtail OWOB in this age group.

The specific aims were to examine:

- Risks of developing early UW, OW or OB related to prenatal exposures, birth anthropometrics and exposures to socioeconomic and family- and child-related health and behavioural factors.
- The significance of psychological health related to UW, OW and OB in preschool children.
- The effect of a long-term multidisciplinary and family-oriented intervention program to curtail OB in preschool children.

3. Hypotheses

- Most previous studies on potential causes of unhealthy weights have been conducted in older children and often in societies that may have less comprehensive and standardized family- and child-related prophylactic care than Norway. Factors related to the early development of unhealthy weights in Norway may therefore differ from those of other studies. New insights may extend our understanding of the development of unhealthy weight trajectories and new ways of prevention and treatment.

- Since interventions to curtail OB in old children have had limited success, we hypothesized that an intervention before entering school may be more effective since parents have more control of children's behaviour at that age. We further hypothesized that a multidisciplinary long-term program addressing parents in a group-based setting where parents could learn from each other and have a major impact on inputs from the professional team, may be more effective than most previous programs.

4. Methods

4.1. Study populations

4.1.1. The Oppland health and growth study (OHGS)

Oppland County, Norway, was one of 20 counties in Norway in 2007 and had approximately 183 000 inhabitants at the time of the study. The county has two cities with a population of 25000 - 30000 in each (Lillehammer and Gjøvik), and was otherwise rural with towns of variable sizes.

In 2007, the public health nurses were asked to invite all families of children entering primary school in the county to be part of the OHGS at the routine health assessment before entering school. Almost all the children attend this assessment. Oppland consisted of 26 municipalities of variable sizes, and the participation rate varied from 20-85 % in the different municipalities. One of the municipalities had no participants. In total, 1119 of 1895 eligible families (59%) joined the study. The children were 5-6 years old at inclusion. They were born in 2001, except that two were born in 2002 and 47 (4 %) in 2000. For the children of families who declined to participate, the public health nurses reported sex and current height and weight anonymously to the research team.

The parents who agreed to join the study, signed a written consent, which included the completion of a questionnaire addressing family- and child-related factors concerning health and lifestyle, and permission by the public health nurses to report all anthropometric measurements obtained from birth to the current health assessment, and age, height and weight from the scheduled health assessments through primary school. The mothers who gave birth in Oppland county, were also asked for permission to obtain data on maternal health, pregnancy and the newborn child's characteristics from the Oppland Perinatal Database (OPD, see section 4.1.3).

The questionnaire completed by the parents was quite extensive, and contained questions on sociodemographic factors, the family's and child's somatic and mental health, chronic diseases, and lifestyle and habits related to nutrition and physical

activity. Most of the questions were equal to or adapted from variables of various Norwegian and international studies, e.g. the Norwegian HUNT-study [161], the Bergen Growth Study [162], The health inquiry on children in Hedmark and Oppland [163], and the International Study of Asthma and Allergies in Childhood (ISAAC) [164] (See Appendix). Mental health of the child was assessed with the Strengths and Difficulties Questionnaire (SDQ) completed by the parents [165].

4.1.2 The intervention study

Children in the OHGS study with weights of at least 1 kg above the 97th percentile for weight related to height were regarded eligible for the intervention study. At that time, BMI percentiles were not in use at the child health care clinics in Norway, but this criterion almost concur with the IOTF definition of OB [30], although some of the children had a BMI barely below the IOTF OB limit. The public health nurses in the eight municipalities situated closest to the two hospitals in the County (Gjøvik, Lillehammer, Øyer, Østre Toten, Vestre Toten, Gausdal, Gran and Sør-Fron) were asked to invite the families of eligible children to the intervention program while eligible families in the other municipalities served as controls without any interventions. We elected to recruit families on basis of municipality instead of individual randomizations independent of municipality to avoid contamination from overflow of information within municipalities, and also because most municipalities were located far from the hospitals (Gjøvik and Lillehammer) where the intervention program was conducted. The eight municipalities were chosen for practical reasons due to closeness to the two hospitals. The intervention municipalities had approximately 60 % of the population in the county. Children in the other municipalities who fulfilled the inclusion criteria and eligible families in the intervention municipalities who were not referred, served as controls. The control group received no information about the intervention program, and had no scheduled appointments with health care services during the three years of the program.

In addition to the children from the OHGS cohort, some families of children with OB in the intervention municipalities heard about the project and enquired about the possibility to join the intervention program. The families of children with an age

close to the age of the children of the OHGS were accepted to the intervention with the argument that a larger intervention group opened for a better estimate of potential effects.

The comparison of anthropometric measurement between the intervention and control group were based on the measurements performed by the public health nurses before school entry and in third grade.

4.1.3 The Oppland Perinatal Database (OPD)

The Oppland Perinatal Database was established in 1989, and is a register of pregnancy and perinatal data. The pregnant women were included at the routine ultrasound screen at 17-18 weeks of pregnancy. The ultrasound screening was performed at the hospitals in Gjøvik and Lillehammer, and the register contains extensive and prospectively obtained data on maternal health, pregnancy, delivery and health of the newborn on nearly all pregnancies and births in Oppland County.

The parents in OHGS who gave birth in Oppland, were asked for permission to link the OPD data to the OHGS data. Of 1088 families in the OHGS, 749 also provided data from the OPD. From the OPD we included the variables maternal age at birth, pre-pregnancy weight and height, pregnancy weight gain, employment and smoking at 18 weeks of pregnancy, and weight and length of the newborn.

4.2 Intervention program

The group selected for intervention (Paper III) met at the hospitals in Lillehammer or Gjøvik. The program was organized in cooperation with the Learning and Mastery Service (LMS) at the hospitals [166]. The LMSs are part of the specialist health services in Norway, and their role is to promote health through group-based patient education programs with the aim to obtain self-management for people living with chronic health challenges. An important ideology of the LMS is that the group process will identify specific challenges and needs and promote a deeper motivation for change. Each group consisted of 5-7 pairs of parents, and each session lasted

approximately 2.5 hours after working hours. Only the parents participated in these sessions while the children played under the supervision of a preschool teacher in another room.

Nurses trained in providing guidance led the group sessions. Either a paediatrician, nutritionist, physiotherapist, or a psychologist contributed at each group session, either alone or together with some of the other professionals in addition to the nurses. The sessions had partly a predetermined program and partly agendas as needs were identified by the group. The different health care personnel gave practical advice regarding, for instance, nutrition and physical activity, but most importantly they encouraged discussions on different challenges in changing lifestyles.

The intervention was planned to last three years. There were four meetings at the LMS during the first year, twice during the second and one during the third year (Figure 2). Between each of these sessions, the families (both parents and child) were invited to meet once with a trained study nurse for assessments, discussions and guidance.

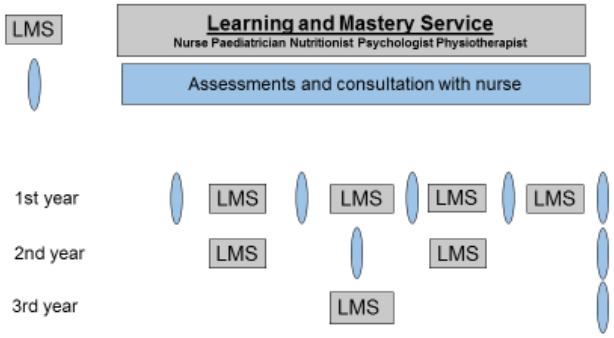

Figure 2. *Plan of the intervention sessions*

4.3 Description of variables

4.3.1 Measurements

Birthweight and crown-heel length of the children were measured by midwives in the hospital at the time of birth. The public health nurses measured height and weight at the school entry assessment, with children wearing light underclothes. Height was measured to the nearest millimetre and weight to the nearest 100 grams, according to national guidelines [167]. The school nurses performed the measurements in 3rd grade, and the children were wearing light clothes.

In the intervention group, measurements at entry and after each of the three years during the follow-up were performed by two specifically trained study nurses, one at each site. The measurements included height, weight, triceps and subscapular skinfold thicknesses, WC and maximum walking distance on a 6 minute walk test [168]. WC was measured to the nearest millimetre, and WHtR was calculated as the waist circumference divided by the simultaneously measured height. For the skinfold measurements, the nurses used the Holtain Tanner/Whitehouse skinfold caliper (Crosswell, Pembrokeshire, UK) according to the same procedure as in the Bergen Growth Study which were published as the Norwegian references [63].

BMI was calculated as weight/height^2 (kg/m^2). The standard deviation score (SDS) for the BMI, waist circumference, waist-to-height ratio and skinfolds of the children were based on current Norwegian growth references [32, 56, 63]. Current age was calculated from date of the school entry or school measurements and date of birth. Birth weight <10th percentile or >90th percentile was based on Norwegian percentiles [32].

The parents' weight and height were self-reported, and the parents also reported the siblings' weight and height. Parental BMI was classified into UW, normal weight (NW), OW and OB according to the WHO definitions [1]. From the OPD, pre-pregnancy BMI was calculated from self-reported weight and height at the beginning

of pregnancy. Gestational weight gain was calculated as the difference between weight at admission for delivery (recall at 6 months post-partum) and pre-pregnancy weight (self-reported).

4.3.2 Mental health

Mental health was evaluated with the Strengths and Difficulties-questionnaire (SDQ, see Appendix), which is a validated questionnaire to detect mental health problems [165]. The SDQ has been formally translated to Norwegian and validated in Norway [169], and it has been used extensively. The SDQ is a 25-item list of statements with five items in each of the following subscales: Emotional problem Scale, Hyperactivity/Inattention Problem Scale, Conduct Problem Scale, Peer Problem Scale, and Prosocial Behaviour Scale. The score on each subscale is ranged from 0 to 10. A sum score called Total Difficulties Score (TDS) sums up the first four subscales giving a range of 0-40. A high score for each of the subscales and TDS indicates more problems, and scores above the 90th percentile are associated with significant mental health problems [165]. On the Prosocial behaviour scale the score has the opposite direction in that lower scores indicate more concerns, and a score below the 10th percentile may indicate a behavioural problem. A TDS above the 90th percentile (TDS90) suggests an increased risk for a psychiatric disorder [165, 170]. On the additional Impact Scale, the parents state whether the child has any difficulties with emotions, concentration, behaviour or in social contact with other people, and if so, what impact it has on everyday life (range 0-10).

4.3.3 Variables in Paper I

The main outcome was the weight category defined as UW, NW, OW and OB based on the IOTF BMI classification at the school entry health assessment [30, 42]. The exposure variables were measurements at birth, pregnancy data from the OPD and sets of relevant variables regarding socioeconomic characteristics, somatic health and lifestyle factors from the parental questionnaire.

Premature birth was defined as birth before 37 completed weeks of pregnancy. Breastfeeding was dichotomized as exclusive breastfeeding for at least 4 months or less. Maternal age at birth was calculated from the mother's date of birth reported in

the questionnaire. Parental higher education was defined as one or both parents having education beyond high school. Physical activity was dichotomized as poorer vs. equal or higher than peers, where poorer meant inferior capabilities than their peers on at least one of the three categories frequency, intensity or endurance.

Binary variables (yes/no) from the questionnaire were: Sex, asthma (current maintenance medication and/or asthma attack requiring medication after 2 years of age), antibiotics >3 times (irrespective of cause), dental caries, kindergarten since two years of age, TV in the child's bedroom, vegetables <5 times/week, fruits <5 times/week, sugar-sweetened beverages >1 time/week, sweets/snacks > 1 time/week, living in a city with > 20 000 inhabitants (urban) or not (rural), parental education after high school, parents working (either full-time, part-time or student), one or both parents from non-Western countries, living with one caretaker, having siblings, and mother smoking.

For families with data in the OPD, we dichotomized gestational weight gain above recommended or not according to guidelines from the Institute of Medicine and National Research Council (USA) [171]. Smoking in pregnancy at the time of the routine ultrasound at week 17-19 was also dichotomized as yes/no.

4.3.4 Variables in paper II

This study was based on data from the OHGS. The main outcome measure was a "yes" or "no" on the Total Difficulties Score above the 90th percentile (TDS90) on the SDQ. Secondary outcome measures were mean scores and scores above the 90th percentile or not on the four subscales that were part of the TDS, and less than the 10th percentile on the prosocial behaviour scale. The weight group of the children was the primary exposure variable, but in the analyses we adjusted for potential confounders which were largely the relevant family- and child-related variables used in Paper I. In addition, we computed a dichotomized variable to describe the child's psychomotor development from several variables in the questionnaire related to fine motor, language, social and behavioural skills, and sleep pattern. The child was defined as having a developmental difficulty if he or she scored lower than their peers

on any of these items, or if the child had received any professional interventions within physiotherapy, speech therapy, psychology or psychiatry, or had received any extra professional support in kindergarten after two years of age. Any physical health problems represented any current or previously chronic illnesses (heart disease, diabetes, coeliac disease, asthma, eczema, hay fever). Low nutritional standard was based on at least three of the following: sweets > 1 day/week, soft drink > 1 day/week, fast food > 1 day/week, breakfast < 4 days/week, vegetables < 2 days/week, and fruit < 5 days/week. We also added a variable on behavioural difficulty among family members, and it was defined as behaviour difficulties when at least one family member other than the child had attention deficit hyperactivity disorder (ADHD), difficulties with attention or other behavioural difficulties.

4.3.5 Variables in paper III

The main outcome measure was the change in BMI SDS from entry to the end of the intervention. The explanatory variables were BMI SDS at entry, the parents' BMI, and basically the same questionnaire-based exposure variables as in Paper I. We also included a variable on current smoking by at least one family member, and the question whether the parents perceived their child as being overweight.

When comparing the intervention and control group we used the height and weight measured by the public health nurses at the routine school entry health assessment and in 3rd grade. We used the measurements performed by the study nurses at entry and after each of the three years when analysing the details on which exposures had positive or negative effects on outcome within the intervention group.

Mean skinfold SDS was calculated as the mean of the sum of the triceps and subscapular skinfolds. The number of attendances at the LMS sessions and meetings with the study nurses was used as a proxy for motivation for change.

4.4 Statistical analyses

Descriptive variables were presented as means with standard deviations (SD) or medians with interquartile range (IQR) for continuous variables and as counts and percentages (%) for categorical variables. Pairwise comparisons were analysed with the Student's t-test, the Mann-Whitney's U test or the Chi-square test, as appropriate. We explored the differences between the four weight groups (UW, NW, OW, OB) by using one-way analysis of variance (ANOVA) or Chi-square tests across all weight groups. When these tests across weight groups showed significant differences, we performed post hoc pairwise testing comparing children in the other weight groups (UW, OW, OB, respectively) to the children with NW.

We subsequently used logistic regression models to explore the significance of main exposures and potential confounders on outcome. We also tested for collinearity and interactions in the models.

Within the intervention group the development of the anthropometric measures were analysed with Related-Samples Wilcoxon Signed Rank Test, and we performed a linear regression analysis with change in BMI SDS from the start to the end of intervention as outcome.

The SPSS Statistics for Windows was used for all analyses. P values ≤ 0.05 were considered statistically significant. The BMI SDS, skinfolds SDS, WC and WHtR were calculated with the R V.2.6.0 (The R Foundation for Statistical Computing, Vienna, Austria) using the Norwegian growth references [32, 56, 63].

4.5 Ethical considerations

The study was approved by the Regional Committee on Medical Research Ethics (REK 1.2006.3491) and the Norwegian Data Protection Official for Research (02-

2006 SI). One of the parents gave written consent. The intervention study was registered at ClinicalTrials.gov (NCT00458224) before recruitment.

5. Summary of results

5.1 Paper I. Social and somatic determinants of underweight, overweight and obesity at 5 years of age: a Norwegian regional cohort study.

Of 1895 eligible families, 1119 (59 %) consented to participate, and the parents completed the questionnaire. However, data on age, height or weight at entry were missing for 31 of the children, leaving 1088 children and their families for further analyses. The participants (n=1088) and those who declined (n=776), did not differ in age, sex distribution or mean anthropometric measures, but there was a slightly higher proportion of children with weight groups other than NW among those who declined.

At entry, the mean (SD) age was 5.71 (0.44) years, the mean BMI 15.87(1.71) and the mean BMI SDS 0.01 (1.03), and 52.3% were girls. The respective prevalence of UW, OW and OB were 7.8%, 10.6% and 3.5%. UW was associated with their anthropometric measures at birth and those of parents, but not with sociodemographic or behavioural characteristics. On the other hand, OW and OB were associated with anthropometric measures of parents and siblings and a variety of unfavourable social characteristics, lack of exclusive breastfeeding for at least four months, sedentary behaviour and dental caries, but not with current dietary habits. In a multivariable logistic regression model, OW and OB were strongly associated with parental OWOB and low education, especially on part of the mother. These effects were still strong after adjusting for the social, environmental and behavioural factors. Our conclusion was that the strong associations between sociodemographic and behavioural factors and OW and OB, but not UW, may suggest that environmental factors are major contributing causes of OW and, in particular, of OB at 5-6 years of age.

5.2 Paper II. Psychological health in preschool children with underweight, overweight or obesity

The aim of the study was to examine if children with UW, OW or OB had more psychological symptoms than the children with a normal weight (NW). The children were the participants in the OHGS. Psychological health was assessed with The Strengths and Difficulties Questionnaire (SDQ) completed by the parents when the children entered the study at the school entry health assessment at 5-6 years of age.

Of the 1895 eligible children, 1119 families consented to participate and for 1088 families (57.4% of eligible children) the database contained all the necessary information for analyses. From data on age, sex, height and weight of participants and eligible children who declined to participate, the participants were probably representative of all eligible children.

The mean scores and the proportion of scores $\geq 90^{th}$ percentile on the SDQ had a curvilinear pattern from children with UW through NW, OW and OB, and with NW as nadir, but the pattern was only statistically significant for the mean score on the Emotional problem, Peer problem and Total SDQ scales, and for the Total SDQ score $\geq 90^{th}$ percentile (TDS90). After adjusting for relevant sociodemographic and behavioural factors in logistic regression analyses, only the association between UW and TDS90 remained significant. The results suggest that psychological challenges may be an independent risk factor related to UW, but not to OW or OB in preschool children.

5.3 Paper III. A family-oriented intervention programme to curtail obesity from five years of age had no effect over no Intervention

The program was completed by 31 children with OB in the intervention program and by 33 controls. The intervention group was recruited from the OHGS cohort and supplemented with other children of similar age from the same eight municipalities. The controls were all from the OHGS cohort, both from municipalities where no intervention was offered and from the intervention municipalities where children who were not recruited for intervention, served as part of the control group. The recruitment is illustrated in Figure 3.

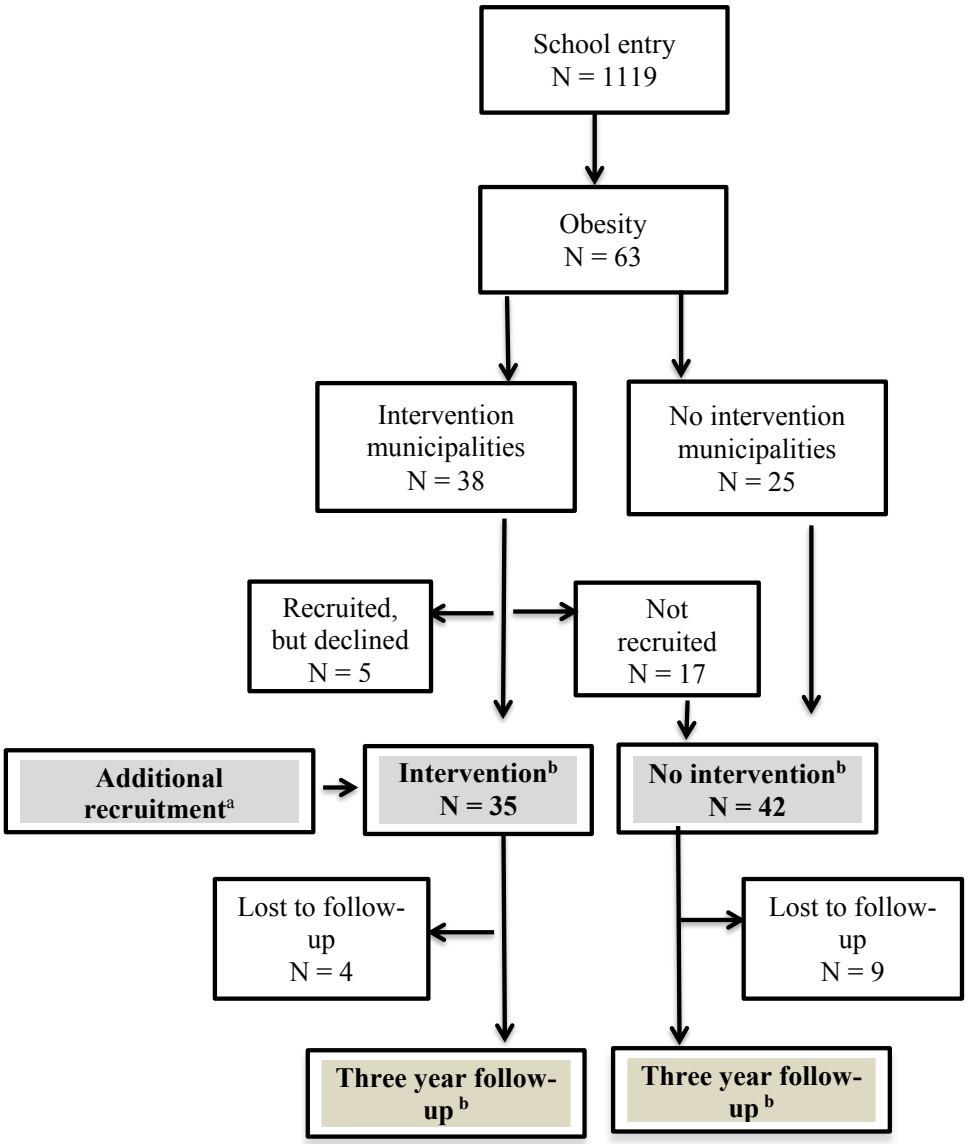

Figure 3. *Recruitment of families of 5-6 year old children with obesity to intervention and no intervention (controls).*

[a]*Included at the request of parents,* [b]*Measurements at public health care clinics*

The median (IQR) age at entry was 5.83 (0.36) years for the intervention and 5.74 (0.66) years for the control group, and the respective median BMI SDS were 2.35 (1.06) and 1.95 (0.49), p=0.012. The difference in BMI SDS at entry was due to the recruitment process in that the children recruited at the request of parents, on average had somewhat higher BMI SDS. The median decrease in BMI SDS over the three years was 0.19 in both groups. The decline increased with increasing BMI SDS at entry, but similarly irrespective of group. Social and behavioural factors were of no significance in this comparison. Within the intervention group, outcome was not related to the behavioural or social factors, waist-to-height ratio or physical ability as measured with the 6-minute walk test, or attendance as a measure of motivation or to attendance. A higher mean skinfold thickness were associated with a larger decline in BMI-SDS, but the BMI SDS and mean skin fold thickness were strongly correlated (r=0.86). Our conclusion was that the skinfold thickness reflected the BMI SDS and that the intervention program had no benefit over no intervention other than the common public attention on avoiding OW and OB during childhood.

6. Discussion

6.1 Overall results

In this regional Norwegian cohort of 5-6-year-old children the distribution of UW, NW, OW and OB for all the 1895 eligible children were 7.8%, 78.1%, 10.6% and 3.5%, and the distribution did not differ significantly between the boys and the girls. The distribution of weight categories for the children of families who elected to participate (n=1119, 59%) differed slightly, but statistically significant from those who declined in that the proportion of all weight categories other than NW was lower (7.8% vs 10.1% for UW, 10.6% vs. 13.5% for OW and 3.5% vs. 4.7% for OB, overall p=0.017).

Current weight category before entering primary school at six years of age was associated with numerous factors, such as BMI of parents and siblings, gestational weight gain and maternal smoking in pregnancy, birthweight, extent of breast-feeding during infancy and physical activity, and sociodemographic factors such as parental education, employment and smoking habits, single vs. two caretakers, and ethnicity. There was no significant association with current dietary habits, but with dental caries.

Compared to children with NW, OW and OB were strongly associated with the BMI of parents and siblings, but also with sociodemographic factors that were likely associated with unfavourable lifestyles, such as maternal smoking during pregnancy and high gestational weight gain, low physical activity, dental caries, low parental education, single caretaker, lack of employment and non-Western ethnicity. UW, however, was associated with birthweight and anthropometric measurements of the parents, but not with the sociodemographic or lifestyle factors. Based on the parents' assessment of the children's psychological health with the SDQ questionnaire, weight category from UW through OW and OB was associated with emotional problems, peer problem and overall psychological problems, and with NW as nadir.

When compared with children with NW after adjustments for potential confounding, OW and OB were only associated with parental BMI, low parental education and maternal smoking. There were no significant association with measures on habits related to physical activity or nutrition, or to psychological difficulties. UW was only associated with parental BMI, birthweight and overall psychological problems.

The three-year multidisciplinary and group-based intervention program had no effect over no intervention on the evolution of BMI. Both the intervention and the no-intervention group had a median decrease in BMI SDS of 0.19, and the change in BMI SDS was not related to any of the registered social or behavioural factors. Within the intervention group, none of the exposure variables had any effect on outcome in terms of decline in BMI SDS.

6.2 Methodological considerations

6.2.1 Participants

In the OHGS, 59 % of all eligible families participated in the study. Compared to most cohort studies that recruit children and parents to follow-up studies by invitation, we suggest that the participation rate was satisfactory [172], in particular since we had reliable data related to BMI and distribution of weight categories on the children of parents who declined. However, we cannot exclude the possibility of selection bias since the proportion of children in weight groups other than NW was slightly higher among the children who declined. Furthermore, the number of participants with weights outside NW in the OHGS cohort was limited. The associations, or in particular lack of associations, between predictors or exposures and outcome in terms of BMI or weight group, should therefore be interpreted with caution since the chance of type I and type II errors may be clinically significant. This is partly illustrated by rather large confidence intervals or standard deviations in many of the data. Nevertheless, we suggest that the number in the OHGS cohort study was large enough to provide robust data on important predictors of an

unhealthy weight development that can be addressed in preventive or therapeutic programs, both on an individual and a group basis. There are arguments why selection bias was not of major importance: 1) Recruitment varied markedly with municipality (0% - 87%) suggesting that the moderate participation rate was mainly caused by variable motivation on part of the public health nurses to recruit families to the study, rather than selectivity on part of the parents, and 2) the differences in anthropometric measures and weight categories between the participants and non-participants were modest.

The intervention study had a complicated recruitment process. Primarily our intention was to divide the OHGS cohort into two groups of similar sizes, where all children with OB and their families in some municipalities were invited to intervention while the children and their families in the other municipalities were not offered any intervention. For practicability, counties closest to the two hospitals were chosen for intervention since it became clear that the program had to be conducted at the hospitals instead of locally in the municipalities. The travel distance would be so long for families from other municipalities that the motivation for participation would probably be low. In fact, even a large number of eligible children from the intervention municipalities were not referred. We do not know to what extent they were not invited or declined to participate. Lack of referral may imply that many families did not perceive their child's weight to be a significant problem or they may have been worried that the intervention would lead to stigmatization. We elected to include these children in the non-intervention group despite living in the intervention municipalities since the parents had given written consent to stay in the project through elementary school.

In general, intervention trials based on individual randomization are considered superior to trials based on convenience sampling. Despite that argument, we chose to divide families into an intervention and control group on basis of residence in municipalities for two reasons; difficulties with attendance due to long traveling distances from many municipalities, and risk of spill-over effects. We elected to invite families from municipalities geographically close to the hospitals for

intervention. The data from the OHGS cohort suggested that the non-random selection of municipalities would not introduce a significant selection bias. Spill-over effects are a well-known concern in studies where one arm of a study may be perceived as preferable [173, 174]. A spill-over effect may be less likely when groups are randomized on basis of geography rather than on an individual basis. Despite this concern we elected to include eligible children in the intervention municipalities who had not been referred for intervention, in the control group for three reasons: 1) It would account for all the eligible children included in the OHGS cohort, 2) the control group would be larger, 3) we considered the risk of spill-over effects to be negligible since we chose to conduct the intervention program in the hospitals rather than in the municipalities, and 4) the municipalities were large with little chance of spreading specific news about the program, in particular because no public information about the intervention study was publicised during the three years of the study and the children did not have scheduled appointments with public health programs between during this time period. Supporting this belief, the control children, who lived in the intervention municipalities, did not differ from the rest of the controls in terms of exposures or outcomes.

We also elected to include some families and children from the intervention municipalities who were not part of the OHGS cohort, but of similar age. They were included at the request of the parents.

These adjustments left us with larger numbers, both in the intervention and control groups, and thereby better statistical power. Due to these recruitment processes, the BMI SDS at entry was somewhat higher in the intervention than the control group. We presumed, however, that weaknesses in terms of possibilities of selection bias could be reduced by the extensive knowledge of social and demographic backgrounds, and health and lifestyles; factors that could be adjusted for in the data analyses. We hypothesized that the higher average BMI in the intervention group could include children and families that were less likely to succeed in BMI reduction, but that this possibility would be counteracted by the fact that these families were particularly motivated to change their lifestyle since they were recruited through their

own initiative. Also, a larger proportion of parents in the intervention than in the control group perceived that their child "looked overweight", which also could indicate a greater motivation for treatment. The intervention and control group also differed somewhat in age at the end of the study, mostly because different municipalities had different routines for when they performed the school measurements during 3rd grade.

In the intervention study, the median change in BMI, BMI SDS and the interquartile ranges for the changes were similar for both groups. It is therefore unlikely that a larger study would have detected clinically meaningful differences. In our power calculation we had estimated that a clinically meaningful difference in BMI after three years ought to be 1 BMI unit (1 kg/m^2), given the extent and duration of the program. Indeed, we did not obtain any significant difference.

6.2.2 Anthropometric measurements

Public health nurses performed all the measurements of the children in the OHGS, both at entry to the study and in 3rd grade. The comparisons of effects on BMI and weight group between the intervention and control group were based on these measurements. Since different nurses performed these measurements in the municipalities, there is a chance of measurement error. However, public health nurses are trained in measuring height and weight of children, and are expected to follow national guidelines on how to do it [167]. We did not do any specific training or validation on how well they did it with reference to the guidelines, or how accurate the measurements were. However, any errors were expected to be random and not systematic, and we therefore expected that such errors did not introduce significant bias.

The anthropometric measurements that were used for analyses of associations between exposures and outcome within the intervention study (height, weight, skinfold thicknesses and waist circumference) were all performed by two specifically trained study nurses, one located at each hospital. At each site they used the same equipment for the measurements. They were trained simultaneously by personnel

who had developed the national references for skinfolds and waist circumference. Measurements of parents and siblings were self-reported by the parents, and therefore at risk of information bias. All measurements were thoroughly checked for possible errors in a data cleaning process. All outliers were manually checked, and obvious inaccurate measurements were removed.

Our outcome measures were BMI SDS and weight groups based on BMI, with all the limitations of BMI measurements (see section 1.2). This may be a weakness of the study, especially the intervention study where we followed children with OB. For children with OB, the BMI may not be an accurate estimate of the proportion and distribution of body fat, and it has been argued that a decrease in BMI SDS with age in children may not necessarily mean a decrease in the degree of OB because the SDS of BMI may not accurately correct for age, sex and degree of OB [50, 51]. However, the intervention study showed that there was a very close and linear correlation between waist-to-height ratio and skinfold thicknesses on one hand and BMI SDS on the other (correlation coefficients in the range of 0.78 -0.86), suggesting that BMI SDS gave a good estimate of body fat on a group basis. Furthermore, BMI SDS is the commonly used measure in intervention studies, particularly since it corrects for variation in age, sex and intervals between entry and end of an intervention.

6.2.3 Questionnaire

The SDQ is a validated form (see section 4.3.2), but the rest of the questionnaire was not. However, most of the questions were used as stated or adapted from other Norwegian studies, like the HUNT- studies [161], The health inquiry on children in Hedmark and Oppland [163] and The Bergen Growth Study [162]. We used the questionnaire from the International Study of Asthma and Allergies in Childhood (ISAAC) to describe respiratory health [164].

Questionnaires carry a risk of recall bias, which may be a more severe problem the further in the past an event occurred. In this study, very few questions pertained to the past, such as duration of exclusive breastfeeding. Most of the questions addressed today's challenges and opinions (see Appendix). Missing data is a frequent problem

with questionnaires, also in this study. The highest proportion of missing data from the parents' information on the questionnaire for the OHGS were: height and/or weight of siblings (42.1 %), mothers (9.8 %) or fathers (15.1 %), and breastfeeding > 4 months (7.8 %). Weight and length of the children at birth were based on measurements performed by midwives at birth and retrieved from the OPD or as reported to the child health care clinics. BMI at birth was missing for 13.6 % of the children because weight and/or length were missing. Current height and weight were provided by the public health nurses, and current child height SDS at entry was missing for 5.7% of the children. Less than 5% were missing for the other variables in the questionnaire and reports from the public health nurses. A rate of missing less than 5 % is often considered acceptable and random [175]. In the OPD, the variables of gestational weight gain above recommended (11.7 %), pre-pregnancy BMI (10.1 %) and gestational weight gain (9.5 %) all had high rates of missing, but the rest of the variables from OPD had only 1-2 % missing. The high rates of missing in some variables affected the statistical power in the multivariate analyses, as cases were excluded list-wise, and only cases with data on all selected variables were included. Variables from the OPD were, however, not included in multivariate analyses.

The questionnaires were only filled out once at inclusion to the study. The OHGS study was cross-sectional, but the intervention study followed the children for three years. Hence, there might have been significant changes in some of the variables during the three years of the intervention, but most of the central variables are considered quite stable, for instance parental education.

6.2.4 Ethical considerations

Clinical research on children is important, but it is equally important to be extra careful not to cause harm. In these studies, the children were at preschool age at inclusion, and the parents consented to join the study. The cohort study did not involve any tests or clinical examinations that were not routine, and the expected results were expected to be of benefit to children in general.

Intervention studies have a potential to cause harm, even without invasive examinations and when performed with the best intention and empathy. The development of an unhealthy weight is a major and increasing personal and societal health risk and need evidence-based measures for prevention and treatment. To continue with an intervention that is not proven effective in sound scientific studies is unethical because lack of effect may leave the child and the family with the psychological consequences of a sense of failure, such as low self-esteem and depression. Our intervention study only involved the parents, and our ideology was that limiting an unhealthy weight trajectory at this young age is the responsibility of parents. The aim of the intervention study was, therefore, to give information and discuss the challenges in changing lifestyles. Moreover, we involved the parents in groups with the hypothesis that it would cause less stigma by emphasizing how common OW and OB is and how difficult it is to manage. We also presumed that involving the parents in groups would promote learning and boost motivation by discussing the challenges as they see them on a daily basis between themselves and with the professionals.

In our intervention study we did not do any systematic evaluation or registration of potential harmful side effects, such as unwanted psychological symptoms. Some families missed many of the scheduled meetings, but we do not know the reason. They may not have felt that the child's weight was a significant problem, feared stigmatization or that the program did not answer to their expectations.

6.3 Interpretations and comparison of results

6.3.1 Prevalence of unhealthy weights

The prevalence of OW and OB in OHGS cohort was in line with other Norwegian studies at the time [68, 69, 176, 177], while other countries in Europe and the rest of the world generally reported higher prevalence [72, 178, 179]. The reasons for a lower prevalence of OWOB in Norway and Northern Europe are unknown and left to

speculations [73]. Some argue that part of the explanation may be that children in the southern part of Europe have a shorter stature [180]. Other suggested explanations are variations in genetic predispositions, environmental factors like differences in diet and physical activity [100, 180], and differences in SES [100, 181]. Norway and the other Nordic countries have smaller disparities between the socioeconomically disadvantaged and advantaged inhabitants than countries further south in Europe, although these differences seem to increase also in the north [182].

The prevalence of UW in our study was similar to a Norwegian study of 6-year-olds [177], and also another Norwegian study with a wider age span (2-19 years) [183]. Many European countries have corresponding prevalence of children with UW [184], however, some countries have a higher prevalence of 15-20 % [141, 179]. The variation in the prevalence of UW is rarely discussed in these papers, as the main focus is usually OWOB.

6.3.2 Predictors of unhealthy weights

6.3.2.1 Predictors of OW and OB

We found associations between OWOB and several unfavourable sociodemographic and behavioural factors, as well as parental BMI and measurements at birth. However, several of the outcomes were mostly related to OB, not OW. Given the limitations of the BMI classification of OW, one could question whether this group really has an unhealthy weight. Indeed, one study showed that 30 % of the children with OW had normal levels of body fat while 50% had moderately increased and only 20 % significantly increased body fat [185]. The OW group is a mixed group, and some of these children might be healthy and muscular, with no increased health risk.

Parental BMI

As expected, we found a strong association between parental BMI and both OW and OB, also after adjusting for relevant socioeconomic and behavioural factors. The siblings' mean BMI SDS was also related to the child's OW and OB, but not in adjusted analyses. The association between parental BMI and childhood OWOB is well known [118, 119], and there is reason to believe that the relation to parental and

siblings' BMI reflect both genetic and environmental impacts on the child [119, 186]. In adjusted analyses, only OW was related to the father's BMI, while both OW and OB was related to maternal BMI. In agreement with our findings, others have also found maternal BMI to be more strongly linked to childhood OB [187, 188]. It is possible that maternal OB is especially important in critical developmental periods, like pregnancy [189]. The stronger association between childhood OWOB and maternal BMI may also reflect both a genetic influence or merely the importance of the mother in establishing and maintaining the family behavioural pattern related to diet and physical activity.

Birthweight (BW)

In our study, increasing BW was associated with increased risk for OW, but only when the mothers had NW or OW (Paper I). However, we found no association between BW and OB, which is in contrast to earlier studies [122]. The relationship between BW and OWOB is complex since both low [121] and high BW [122, 123, 190] are associated with OWOB later in childhood in several studies. Different mechanisms might explain the different associations. High maternal BMI is associated with high BW [191] and also with a higher proportion of fat mass in the baby [189], which may suggest a higher risk for OWOB in childhood and adulthood. Children with OB have a higher body fat percentage than children with OW, which may support such a notion [192, 193]. On the other hand, being born SGA, which usually implies a low fat mass, is also associated with later OWOB [121]. A possible explanation may be that the catch-up growth after growth restriction in foetal life may have long lasting consequences for body composition [194, 195]. SGA is also related to metabolic syndrome in childhood [196].

Smoking

It is well established that smoking in pregnancy leads to reduced BW [197, 198]. This was also reported from the county of our cohort [199]. It is also well known that maternal smoking in general is associated with childhood OB [200, 201]. In paper I, we found an association between maternal smoking and childhood OW and OB, also after adjusting for other socioeconomic and behavioural factors. The mechanism for

the risk of later OB may be the one suggested for children born SGA (see above), since smoking leads to intrauterine growth restriction. It may also be that maternal smoking just reflects unhealthy behaviour with consequences for the children [202]. It is still unknown whether maternal smoking has a direct obesogenic effect on the offspring.

Parental education

Parental education is a strong indicator of the family's SES, and we found a cumulative effect of parental education in combination with parental BMI on the outcome OWOB. Low maternal education was strongly associated with OB, and also OW, even after adjusting for other important factors. Our findings are in line with several other studies [95, 102, 108, 110]. The background for the association between parental education and childhood OWOB is multifaceted, but studies have shown that families with lower education tend to eat fewer regular meals, more unhealthy food and have more sedentary behaviour [95, 105, 110, 112].

Rural living

We did not find an association between rural living and OWOB in our study, which is in contrast to some studies [108, 203]. We suggest that the reason may be that there are relatively small differences in population densities and ways of living between the small cities and more rural areas in this county. Differences may possibly evolve as the children get older when the children in the rural districts are more dependent on transport to secondary schools and leisure activities. A follow-up study of the OHGS cohort in 3rd grade supports this notion since the rate of OWOB was higher in the rural municipalities at that age [204].

Physical activity, sedentary behaviour and diet

We found no effect of physical activity in adjusted analyses. We suggest that young children mainly stay physically active through play rather than through organized activities. Furthermore, 90% of the children had attended kindergarten since two years of age, and probably had similar activities there. The lack of effect of physical activity is in contrast to some studies [92, 93, 105], but most studies have included

older children, and more research is needed to make conclusions on the significance of paying more attention to physical activity in the preschool age group. Similarly, in opposition to earlier studies [95, 205], we found no effect of TV in the child's bedroom or screen time. Again, it may be too early to see an effect of screen time since most children in this age group are equally active in kindergarten, and the differences in screen time may be too small to be of clinical significance.

Eating fruits often was the only dietary factor related to OW in our study, and no dietary factors were related to OB. Other Norwegian studies have also found that children with OWOB eat more healthy food [95, 205, 206], and reverse causality may be a natural explanation in that the parents try to counteract increasing OW by providing healthy food. We found no associations with sugar-sweetened beverages or sweets and OWOB, in contrast to other studies [95, 205]. However, under-reporting of unhealthy food in children with OWOB might be a concern [207].

We found no effect of dental caries, asthma medication or antibiotics in our study. Other studies have found associations with these factors [140, 208-210]. We included dental caries under the assumption that having caries was a significant proxy for unhealthy food habits [211].

Lastly, we found an association with shorter exclusive breastfeeding and OB in unadjusted analyses, but not in adjusted analyses. This is in line with some [204], but in contrast to other studies [123, 212]. In our study, a total of 90 % of the children were breastfed, and 72.8 % exclusively breastfed for more than four months. Hence, there might be a difference we could not detect because of lack of power.

Mental health

We found no relation between OW or OB and psychological problems when adjusting for known risk factors of OWOB, but we did find higher scores of Emotional Problems, Peer Problems, TDS and TDS90 in the children with OB in unadjusted analyses.

Several studies have found associations between OW or OB and reduced quality of life [7, 16, 18], as well as lower self-esteem [7, 16, 18] and depression [7, 16, 17, 128, 213]. Studies have shown that young children, even in kindergarten, are often dissatisfied with their body size [214-216]. Still, the majority of preschool children are content with their physiognomy [214, 217], but amongst older children, children with both UW and OB are dissatisfied with their weight [218]. We suggest that since the majority of the children in our study had been in kindergarten since two years of age, they had little focus on body size, and studies have shown that the anti-obese attitudes in preschool children did not affect playmate selection [219].

The children with OW did not differ from children with NW in terms of psychological difficulties in our study. Most parents of children with OW consider their children to have a normal body size [183], and also the majority of children and adolescents with OW assess their own body size as normal [218]. The similar scores of the OW and NW children on the SDQ questionnaire reflect their similarly perceived normal body shape. Also, as discussed earlier, OW in young children may reflect lean body mass and thereby an appearance associated with a healthy and strong body and not an appearance associated with OB [220].

There is conflicting evidence about the association between ADHD and other behavioural difficulties and OWOB in childhood. Some studies have shown that ADHD is a risk factor for OW and OB in adolescence and adulthood [131, 133]. In childhood, several systematic reviews show no association between ADHD and OWOB [7, 132, 133], while some studies show an association [131, 221]. Donnchadha et al. found that children with ADHD were more likely to have OWOB, but this relationship was largely explained by psychosocial factors [132]. None of these studies included preschool children. In our study, we found no association between OW, OB and behavioural difficulties, which is in congruence with a Norwegian study of children at 18 and 36 months of age [222]. In a study of 6-12-year-old Norwegian children, the mothers rated their children with the Norwegian Hierarchical Personality Inventory of Children [223]. Children with OW or OB were rated lower on energy, optimism, compliance, concentration, perseverance and self-

confidence, and higher on egocentricity, irritability and anxiety than children with NW. There is a possibility that children with OB in our study had psychological symptoms or issues that we could not detect with the SDQ.

6.3.2.2 Predictors of UW

Anthropometric and sociodemographic factors

UW was only associated with birth anthropometrics and paternal BMI, and none of the sociodemographic factors, also in adjusted analyses. Since all weight groups were related to parental BMI, it is reasonable to think that the genetic component was important. However, the size of the UW group was quite small, hence there might have been true effects of environmental factors that we could not find because of lack of power. This view might be enhanced by the linear association of several environmental factors with weight groups, even though they were not significant for UW.

In our study, UW was associated with a low BW in unadjusted analyses, which is supported by other large studies [224, 225]. In adjusted analyses, we found that UW was associated with high crown-heel length at birth, but not with BW. We found no associations with sociodemographic factors. Few studies have looked at UW children and SES in high-income countries. A South-Korean study found that UW in childhood was associated with lower SES in boys, but not in girls. Other risk factors were female sex, living in a metropolitan city (not in the capital or more rurally) and parental body shape [226]. A study of 3-4-year-old children in Scotland found a higher risk of UW in the most deprived part of the population, also when adjusted for BW [227]. On the other hand, a study of adolescents in Germany found that the prevalence of UW increased with increasing occupational status of the parents [228].

In our study, UW seemed to be associated with only anthropometric and potentially genetic factors. However, both parental BMI and birth anthropometrics may be affected by a meticulous parental behaviour in terms of diet and physical activity that we were not able to examine in the scope of this study.

Mental health

We found that UW was related to psychological difficulties, which has rarely been reported in preschool children before. An Italian study showed that children with UW had more internalizing as well as externalizing problems than children with NW at all assessments from 2 to 11 years of age [229]. In adolescence, children with UW are teased almost as often as children with OB, suggesting that UW may have somewhat the same psychological consequences for a child [230]. Bullying behaviour is common already in kindergarten [231]. In addition to the lower muscle mass, the children with UW are also shorter, as shown in both our cohort (Paper I) and in other studies [218]. We therefore propose that one possible explanation for more mental health problems in the UW group could be that they are the physically weakest and least robust part in a kindergarten setting.

It is interesting that the UW children had more mental health problems despite the finding that UW was not associated with SES or other family related or behavioural factors (Paper I). On the other hand, children with OB had no discernible psychological symptoms despite the strong association with unfavourable SES and behavioural factors. Some studies have found premorbid UW in children who later on developed eating disorders, mainly anorexia nervosa [26]. From our data, we cannot exclude that UW in some of the children, and the association with low BMIs among their parents, was partly due to rigorous and controlling behaviour by the parents rather than genetic predisposition, and possibly a problematic relationship with food and physical activity on part of the parents [24, 232].

6.2.3 Effects of prevention and treatment of OW and OB

We found no effect of a multidisciplinary intervention program with the purpose to curtail OB from 5-6 years of age. Both the intervention and the control group had the decline in BMI SMS of 0.19 after three years.

The Cochrane database contains several reviews of interventions to treat OWOB in children and adolescents of different age groups [19-22]. A review for children aged 6 to 11 years, which is the most relevant age group compared to our study, found a

mean difference in BMI SDS of -0.06 (95% CI -0.10 - -0.02) for intervention versus no treatment. However, the quality of evidence was low or very low [22]. The review of Loveman et al concerning parent-only interventions for children with OWOB aged 5 to 11 years found a minimal effect compared to children on a waiting list, but again, the quality of evidence was low, and the maximum follow-up period was 10-12 months [21]. Our results were similar to other Nordic studies. A Danish intervention study of children with OB aged 2-16 years found a mean change in BMI SDS of –0.3 after two years, and better results in the youngest children [148]. However, the Danish study had no control group. A Norwegian intervention study of 7-17-year-old children found an overall BMI SDS reduction of 0.13. Nevertheless, they found improvement in levels of insulin, total cholesterol, LDL and total/HDL cholesterol ratio, even in the group with the smallest decrease in BMI SDS (0.0 - <0.1) [151]. Another Norwegian intervention study compared a multi-family with a single-family intervention of children with OWOB aged 6-12 years [233]. After two years, pooled data showed a borderline difference in BMI SDS of -0.14 in favour of the multi-family group (reduction of 0.20 vs. 0.08, p= 0.046). Neither of these Norwegian studies had control groups who were not offered any intervention.

Within our intervention group, only BMI SDS at entry influenced the decline in BMI SDS in the unadjusted analyses; the decrease in BMI SDS was higher the higher the baseline. None of the other factors suspected of being risk or beneficial factors related to expected failure or success were of significance. However, the significance of BMI SDS at entry was the same in the intervention and control groups suggesting that the decline was not affected by the intervention.

In the adjusted analyses limited to the intervention group, higher mean skinfold SDS at entry was also associated with a larger decrease in BMI SDS. Skinfolds were not measured in the control group, and given the very close correlation between BMI and skinfold thicknesses, it is likely that the same change in skinfolds thicknesses occurred in the control as in the intervention group. We found it particularly interesting that attendance at the meetings in the intervention program was not associated with outcome. One of our hypotheses was that high attendance was a

measure of motivation for changing behaviour related to nutrition and physical activity. However, a similar lack of association was also seen in the Danish study [148]. Neither dental caries, which may imply unhealthy nutrition, parental education or single caretaker were associated with change in BMI SDS. The subscapular skinfold SDS declined over the three years of the intervention, but the triceps skinfold, mean skinfold, WC and WHtR were stable. This suggest that the intervention did not change the body composition of the majority in the intervention group. Nevertheless, the intervention may have had a clinically relevant effect on individual children, and even small declines in BMI SDS can improve cholesterol and other cardiovascular risk factors [151].

The identical average decrease in BMI SDS in the intervention and control group, may suggest that specific intervention programs do not add effects over what is accomplished by intensive public focus on prevention of OW and OB for children and adults from national and local health authorities. There are data to show that the prevalence of OWOB among children in Norway may not have increased during the last years [69],which may be a result of public attention. The national trend and our findings lend support to the notion that public focus on early prevention of OWOB in childhood is the most important approach to diminish the obesity epidemic.

7. Conclusions

OW, and especially OB, at five years of age was strongly associated with several unfavourable sociodemographic and behavioural factors, as well as with parental OW and OB. On the other hand, UW was only associated with birth anthropometrics and parental BMI. Consequently, we suggest that environmental factors are the most important predictors of the development of OWOB in preschool children.

Preschool children with UW and OB, but not OW, had more psychological difficulties in terms of emotional symptoms, peer related problems and total difficulties, than children with NW. After adjusting for several adverse sociodemographic and family related factors, only UW was independently associated with psychological problems. We suggest that psychological difficulties in preschool children with OB are mainly due to other factors than their appearance, but that UW is an independent risk factor for experiencing psychological difficulties, or conversely, that psychological challenges may be an independent risk factor for UW.

A multidisciplinary three-year intervention program targeting 5-6-year-old children with OB had no effect over no intervention on BMI SDS development. The intervention group and control group had an equal decline in BMI SDS. The decline increased with increasing BMI SDS at entry, but the decrease was the same for the intervention and control group. The intervention study confirms most earlier studies that OWOB is difficult to reverse with specific programs, also during early childhood, and lends support for a high and continuous focus on prevention in national and local public health programs.

8. Future perspectives

Children with UW have rarely been studied, other than in relation to diseases, malnutrition and eating disorders. Unexpectedly, we found more psychological problems in children with UW than in children with NW, OW and OB. Further research is needed in order to understand this seeming paradox.

Long-term studies of children of all weight groups should be conducted in order to monitor changes and risk factors for unhealthy weight trajectories as well as physical and psychological consequences of unhealthy weights. In particular, effects of well-intended national and local initiatives to promote healthy weights should be monitored to assess efficacy, cost-effectiveness and risks of untoward side effects, in particular related to psychological health. In Norway, weight and height are currently measured regularly before entering school and in 3rd and 8th grade. These measurements should be collected anonymously in regional and national health registers in order to monitor positive and potentially negative effects of regional and national programs to prevent unhealthy weights.

Despite extensive public programs to prevent OWOB in children, OWOB will continue to be a significant health problem on an individual and a societal basis. Children with OB and their families will continue to need care, either on an individual or on a group basis. However, given the large number of patients and available resources, it is mandatory that programs are tested scientifically for efficacy and cost-effectiveness, preferably in large and good quality studies with a long follow-up period. Scientific evidence of effect of programs is also mandatory because stigma and shame associated with OB may increase with failure to obtain weight control. A lack of intended effect can be perceived as a personal failure due to lack of character and cause severe harm on mental health. It is also important to expand our knowledge on psychological consequences and possible precursors of OWOB, since psychological mechanisms may influence the response to treatment.

In addition to public focus on healthy living and on scientifically proven intervention programs, there is a need of more scientific focus on how a society can prevent an

obesogenic environment. It may be too simple to just argue for cheaper healthy food and more expensive unhealthy food and for creating arenas for physical activity. This is a complex issue because comprehensive restrictions may be perceived as a violation of individual freedom and cause resentment and resistance if they are not well founded. From a political perspective, anti-obesogenic measures have to be balanced against other national and international interests and agreements.

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10. Errata

Paper III, page 6, section 3.2 “The intervention group”, line 17. The corrected text is written in italic.

Corrected text should read: “In the multiple linear regression analysis, a higher *mean skinfold* SDS at entry was associated with a larger reduction in BMI SDS, but none of the other exposures were associated with a change in BMI SDS (Table 3)”.

11. Appendix & Papers

Appendix: Questionnaire

"Growth and health in children in Oppland", Norwegian version [Vekst og helse blant barn i Oppland], including the Strengths and Difficulties Questionnaire.

Paper I

Social and somatic determinants of underweight, overweight and obesity at 5 years of age: a Norwegian regional cohort study.

Supplementary appendix to Paper I (online material, Supplementary tables 1-3)

Paper II

Psychological health in preschool children with underweight, overweight or obesity

Paper III

A family-oriented intervention programme to curtail obesity from five years of age had no effect over no intervention

Vekst og Helse blant barn i Oppland

Samarbeid mellom helsestasjonene og barnepoliklinikkene i Gjøvik og Lillehammer

Skjemaet fylles ut av pårørende og gis til helsesøster.
NB! Skriv tydelig og pass på at kryssene står inne i avkrysningsboksene!

Barnets navn: _____ Fødselsdato: ☐☐☐☐☐☐

Mors navn: _____

Adresse: _____

Mors fødselsnummer: ☐☐☐☐☐☐☐☐☐☐☐

1. Hvor ble barnet født?
☐ Gjøvik sykehus ☐ Lillehammer sykehus ☐ Ringerike sykehus ☐ Annet; Hvor? _____

2. Ble barnet født omtrent til ventet tid (innenfor 3 uker før og 2 uker etter termin)?
☐ Ja ☐ Nei ☐ Vet ikke

Hvis nei, hvor mange uker for tidlig/for sent? ☐☐ Uker for tidlig ☐☐ Uker for sent

3. Hva var fødselsvekten? ☐☐☐☐ gram

4. Har barnet noen gang vært innlagt i sykehus?
☐ Nei
☐ Ja; i så fall: Hvor mange ganger ☐☐ ganger

5. Hvis barnet har vært innlagt i sykehus, skriv årsaken til at barnet har vært innlagt og hvor gammelt barnet da var:

Opphold Nummer	Årsak til innleggelse	Alder i hele år	Ikke skriv her ICP
1			
2			
3			
4			

Spørsmål om lungefunksjonen

6. Har barnet noen gang (etter nyfødtperioden) hatt tung pust piping/surkling/tetthet i brystet? *Kryss av for det alternativet du mener passer best.*

☐ Ja; Hvis ja
- ☐ Bare før, men ikke etter 2 års alder
- ☐ Både før og etter 2 års alder
- ☐ Bare etter 2 års alder

☐ Nei, aldri; *hvis nei, gå til spørsmål 11*

7. Har barnet hatt tung pust eller piping/surkling/tetthet i brystet i løpet av de siste 12 månedene?

☐ Ja
☐ Nei; *hvis nei gå til spørsmål 11*

8. Hvor mange anfall med tung pust eller piping/surkling/tetthet i brystet har barnet hatt i løpet av de siste 12 månedene?

☐ Ingen
☐ 1 til 3
☐ 4 til 12
☐ mer enn 12
☐ har slike plager hele tiden

9. Hvor ofte har barnets søvn i gjennomsnitt blitt forstyrret på grunn av piping/surkling/tetthet i brystet de siste 12 månedene?

☐ aldri våknet
☐ mindre enn 1 natt pr uke
☐ 1 eller flere netter pr uke

10. Har piping/surkling/tetthet i brystet eller tung pust vært så alvorlig de siste 12 måneder at barnet har hatt problemer med å snakke slik at han/hun bare kunne si ett eller to ord mellom hvert pust?

☐ Ja
☐ nei

11. Har barnet noen gang hatt astma?

☐ Ja
☐ nei

12. Har barnet i løpet av de siste 12 måneder hatt tung pust eller piping/surkling/tetthet i brystet under eller etter fysisk trening, aktiv lek eller mosjonering?

☐ Ja
☐ nei

13. Har barnet i løpet av de siste 12 måneder hatt tørr hoste om natten, utenom hoste i forbindelse med forkjølelse eller andre luftveisinfeksjoner?

☐ Nei
☐ Ja, noen ganger
☐ Ja, hele tiden

14. Har barnet noen gang brukt astmamedisiner?
Antibiotika/penicillin ved lungebetennelse og bronkitt regnes ikke med.

☐ Nei, barnet har aldri brukt slike medisiner (se nedenfor)
☐ Ja, barnet brukte slike medisiner før 2 års alder, men ikke senere
☐ Ja, barnet brukte slike medisiner etter 2 år, men ikke nå lenger
☐ Ja, barnet bruker fortsatt medisiner, i så fall hvilke:
  ☐ Inhalasjonssteroider (Flutide, Pulmicort, Becotide, Seretide, Symbicort); i så fall brukes disse fast eller i perioder?
    ☐ fast daglig ☐ bare i perioder med forkjølelse eller tung pust
  ☐ Anfallsmedisiner (Efedrin, Ventolin, Bricanyl, Airomir, Oxis, Serevent, Atrovent)
    ☐ fast daglig ☐ bare ved tung pust eller før anstrengelse
  ☐ Singulair
  ☐ Andre lungemedisiner, skriv ned hvilke: _____

15. Har barnet noen gang hatt episoder med lungebetennelse eller bronkitt som har blitt behandlet med penicillin eller andre antibiotika?

☐ Nei
☐ Ja. I så fall;
Omtrent hvor mange ganger før 2 års alder *(sett 0 for ingen?)*
☐ ganger
Omtrent hvor mange ganger fra 2 års alder til for 12 mndr siden?
☐ ganger
Omtrent hvor mange ganger siste 12 måneder
☐ ganger

16. Hvor mange ganger har barnet fått penicillin eller andre antibiotika for andre sykdommer enn lungesykdommer? *Skriv 0 dersom ingen*
☐ ganger

Spørsmål om andre sykdommer

17. Har, eller har barnet hatt atopisk (kløende) eksem?
☐ Nei ☐ Ja, tidligere ☐ Ja, fortsatt
18. Har, eller har barnet hatt, høysnue?
☐ Nei ☐ Ja, tidligere ☐ Ja, fortsatt
19. Har, eller har barnet hatt, andre allergiske sykdommer?
☐ Nei ☐ Ja; tidligere ☐ Ja, fortsatt

Beskriv i så fall:_____

20. Bruker barnet briller? ☐ Nei ☐ Ja; hva er styrken?_____
21. Skjeler barnet? ☐ Nei ☐ Ja; behandling?_____
22. Er barnet nærsynt eller langsynt? ☐ Nei ☐ Ja; hva?_____
23. Har barnet andre synsproblemer? ☐ Nei ☐ Ja; hva?_____
24. Har barnet fått påvist nedsatt hørsel? ☐ Nei ☐ Ja; Årsak?_____
Hvis ja; bruker barnet høreapparat? ☐ Nei ☐ Ja
25. Har, eller har barnet hatt dren i ørene?
☐ Nei ☐ Ja; tidligere ☐ Ja, fortsatt
26. Har barnet fått fjernet falsk mandel (polypp, adenoid) ☐ Nei ☐ Ja
27. Har barnet fått fjernet mandlene? ☐ Nei ☐ Ja
28. Har/har barnet hatt hull i tennene? ☐ Nei ☐ Ja
29. Har barnet en medfødt funksjonshemning (f.eks. Downs syndrom, cerebral parese e.l.)? ☐ Nei ☐ Ja, Hvis ja, beskriv:

30. Har, eller har barnet hatt, andre kroniske sykdommer som ikke er nevnt ovenfor (f.eks. cøliaki, diabetes? ☐ Nei ☐ Ja; tidligere ☐ Ja, fortsatt
Beskriv i så fall:_____

Spørsmål om ernæring

31. Har barnet vegetarisk kosthold? ☐ Nei ☐ Ja
Hvis ja; spiser hun/han melkeprodukter eller egg? ☐ Nei ☐ Ja

32. Hvordan vil du beskrive hvor flink barnet er til å spise?
(Fyll ut alle aktuelle rubrikker)
☐ Normalt flink til å spise
☐ Spiser lite, vanskelig å få til å spise (småspist)
☐ Vansker med å spise/svelge klumper og fast mat
☐ Liker bare enkelte ting; i så fall: Hva vil han/hun ikke spise? _____
☐ Har for god matlyst, må forsøke å begrense inntak
☐ Vet ikke/ har ingen bestemt mening om dette

33. Hvor ofte pleier barnet å spise følgende måltider i løpet av en uke?

	Aldri/ sjelden	1 gang i uken	2 ganger i uken	3 ganger i uken	4 ganger i uken	5 ganger i uken	6 ganger i uken	7 ganger i uken
Frokost	☐	☐	☐	☐	☐	☐	☐	☐
Formiddags-mat/lunsj	☐	☐	☐	☐	☐	☐	☐	☐
Middag	☐	☐	☐	☐	☐	☐	☐	☐
Kveldsmat	☐	☐	☐	☐	☐	☐	☐	☐

34. Hvor mange ganger i uken spiser eller drikker barnet ditt noe av dette?

	Aldri	Sjeldnere enn 1 gang pr. uke	En gang pr. uke	2-4 dager i uken	5-6 dager i uken	En gang hver dag	Flere ganger hver dag
a) frukt	☐	☐	☐	☐	☐	☐	☐
b) grønnsaker	☐	☐	☐	☐	☐	☐	☐
c) godterier med sukker (drops, sjokolade, smågodt o.l)	☐	☐	☐	☐	☐	☐	☐
d) Cola, brus eller andre leskedrikker med sukker	☐	☐	☐	☐	☐	☐	☐
e) fast food (hamburger, pølser og lignende)	☐	☐	☐	☐	☐	☐	☐

35. Ble barnet ammet som spedbarn/småbarn?
☐ Ja ☐ Nei ☐ Vet ikke

I tilfelle ja, hvor lenge fikk barnet **bare** morsmelk uten annet tillegg enn tran/vitaminer?
☐ uker *eller* ☐ måneder ☐ vet ikke

Hvis du ga morsmelk, hvor gammelt var barnet da du *helt sluttet* å amme som tillegg til annen mat?
☐ uker *eller* ☐ måneder ☐ vet ikke

36. Hva synes du om barnets kropp?
☐ Altfor tynn
☐ Litt for tynn
☐ Omtrent passe
☐ Litt for tykk
☐ Altfor tykk

Spørsmål om aktivitet, ferdigheter og utvikling

37. Hvor utholdende er barnet i lek og aktivitet?
☐ Holder følge med jevnaldrende barn i lek og aktivitet
☐ Litt mindre utholdende enn jevnaldrende barn
☐ Mye mindre utholdende enn jevnaldrende barn

38. Hvordan oppfatter du barnets fysiske ferdigheter (grovmotorikk)?
(F.eks. løpe, hoppe, sparke ball, sykle o.s.v.?)
☐ Mer "klønete" eller umoden i sine ferdigheter
☐ Lik jevnaldrende
☐ Flinkere enn de fleste jevnaldrende

39. Hvor aktiv synes du barnet er i lek, sport o.l?
☐ Meget aktiv, mer enn gjennomsnitt for andre av samme kjønn og alder
☐ Vanlig aktiv, omtrent som gjennomsnitt for andre av samme kjønn og alder
☐ Lite aktiv, mindre enn gjennomsnitt for andre av samme kjønn og alder

40. Hvor ofte driver barnet med sport eller leker så aktivt at han/hun blir andpusten og /eller svett?
☐ aldri ☐ mindre enn en gang i måneden ☐ 1-3 ganger i måneden
☐ en gang i uka ☐ 2-3 ganger i uken ☐ 4-6 ganger i uka ☐ hver dag

41. Hvor mange timer i uka driver barnet med sport eller leker så mye at han/hun blir andpusten og/eller svett?
☐ ingen ☐ omtrent ½ time ☐ omtrent 1 time
☐ omtrent 2-3 timer ☐ omtrent 4-6 timer ☐ 7 timer eller mer

42. Hvor mange timer gjennomsnittlig sitter barnet daglig foran TV (TV, DVD, video, TV-spill) og/eller foran PC'en?
☐ ikke i det hele tatt ☐ mindre enn en ½ time om dagen
☐ 1/2-1 time ☐ 2-3 timer ☐ 3-4 timer ☐ mer enn 4 timer

43. Har barnet TV inne på soverommet?
☐ ja ☐ nei

44. Hvordan oppfatter du barnets ferdigheter med hendene?
(Eks. tegne, klippe, bygge med Lego o.s.v.?)
☐ Mer "kløkete" eller umoden i sine ferdigheter enn jevnaldrene
☐ Lik jevnaldrende
☐ Flinkere enn de fleste jevnaldrende

45. Hvordan vil du beskrive barnets språk i dag?
(Velg det alternativet du synes passer best).
☐ Barnet snakker bedre enn vanlig for jevnaldrende barn
☐ Barnet snakker like godt som andre jevnaldrende barn
☐ Barnet har samme ordforråd som andre, men dårligere uttale
☐ Barnet har mindre ordforråd, men god uttale
☐ Barnet har både mindre ordforråd og dårligere uttale
☐ Barnet har ikke, eller svært lite, språk

46. Har barnet etter 2 års alder hatt behov for spesielle hjelpetiltak som kontakt med:
(Kryss av for alle aktuelle faggrupper).

Fysioterapeut	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag
Logoped	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag
Ekstra støttetiltak i barnehagen	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag
PPT (Pedagogisk psykologisk tjeneste)	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag
Psykolog/psykiater	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag
Barne- og ungdoms-Psykiatrisk (BUP)	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag
Fylkets habiliteringstjeneste	☐ Nei	☐ ja, tidligere	☐ ja, fortsatt i dag

47. Har barnet gått i barnehage siden 2 års alder?
☐ Nei
☐ I barnehage tidligere, men ikke nå; Hvor lenge i barnehagen? ☐☐ år og ☐☐ mndr
☐ Går fortsatt i barnehage, og har gått hvor lenge? ☐☐ år og ☐☐ mndr

48. Hvordan fungerer barnet sammen med andre barn, for eksempel i barnehagen?
☐ Barnet skiller seg ikke fra andre jevnaldrende barn
☐ Barnet har samspillvansker med andre barn
Hvis samspillvansker, angi hvordan *(flere rubrikker kan krysses av):*
☐ Barnet plages av andre barn, føler seg utenfor, sky og isolert
☐ Barnet er aggressivt, urolig og plager andre barn
☐ Barnet mistrives, føler seg utenfor og isolert, uten å plage andre
☐ Andre vansker i samspill med andre; vennligst beskriv disse:_____

Spørsmål om søvn

49. Har barnet noen ganger hatt søvnvansker (flere rubrikker kan fylles ut)?
☐ Aldri hatt søvnvansker av betydning
☐ Søvnvansker før 2 års alder
☐ Søvnvansker etter 2 år, men ikke siste 1 år
☐ Fortsatt søvnvansker

50. Hvis barnet har hatt søvnvansker etter 2 års alder eller fortsatt har søvnvansker, hvordan vil du beskrive disse *(flere rubrikker kan merkes):*
☐ Vansker med å legge seg til å sove om kvelden
☐ Våkner i løpet av natten
☐ Våkner uvanlig tidlig
☐ Våkner uvanlig sent
☐ Andre søvnvansker; beskriv: _____

Avføring og vannlatning

51. Tisser barnet på seg om dagen?
☐ Nei ☐ Sjeldnere enn 1 g/uke ☐ Ca 1-3 ggr/uke ☐ Oftere

52. Tisser barnet på seg om natten?
☐ Nei ☐ Sjeldnere enn 1 g/uke ☐ Ca 1-3 ggr/uke ☐ Oftere

53. Får barnet avføring i bukse/bleie om dagen?
☐ Nei ☐ Sjeldnere enn 1 g/uke ☐ Ca 1-3 ggr/uke ☐ Oftere

54. Får barnet avføring i bukse/bleie om natten?
☐ Nei ☐ Sjeldnere enn 1 g/uke ☐ Ca 1-3 ggr/uke ☐ Oftere

55. Hvor ofte har barnet avføring?
☐ Mer enn 1-2 ggr/dag ☐ 1-2 ggr/dag ☐ Ca 1-2 ggr/uke ☐ Sjeldnere

56. Hvordan er avføringen?
☐ Normalt formet ☐ Løs ☐ Hard

Litt om familien

57. Hvor mange søsken eller halvsøsken har barnet?
☐☐ søsken/halvsøsken

For **helsøsken:** Oppgi alder, kjønn, høyde og vekt:

Søsken nr. 1: ☐☐ år og ☐☐ mndr: ☐ gutt ☐ jente
Høyde ☐☐☐ cm Vekt ☐☐☐ kg

Søsken nr. 2: ☐☐ år og ☐☐ mndr: ☐ gutt ☐ jente
Høyde ☐☐☐ cm Vekt ☐☐☐ kg

Søsken nr. 3: ☐☐ år og ☐☐ mndr: ☐ gutt ☐ jente
Høyde ☐☐☐ cm Vekt ☐☐☐ kg

Søsken nr. 4: ☐☐ år og ☐☐ mndr: ☐ gutt ☐ jente
Høyde ☐☐☐ cm Vekt ☐☐☐ kg

58. Hva er foreldrenes høyde og vekt:
Mors høyde ☐☐☐ cm Mors vekt ☐☐☐ kg
Fars høyde ☐☐☐ cm Fars vekt ☐☐☐ kg

59. Hvem bor barnet sammen med til daglig?
☐ Mor og far
☐ Bare mor
☐ Bare far
☐ Både mor og far, men hver for seg (for eksempel en uke hos hver)
☐ Mor og ny partner (stefar)
☐ Far og ny partner (stemor)
☐ Fosterforeldre
☐ Andre, hvem:_____

60. Spørsmål om spesielle sykdommer i familien
Har, eller har noen hatt, astma?
☐ Ingen ☐ Ja, mor ☐ Ja, far ☐ Ja, søsken
Har, eller har noen hatt, høysnue?
☐ Ingen ☐ Ja, mor ☐ Ja, far ☐ Ja, søsken
Har, eller har noen hatt, atopisk eksem?
☐ Ingen ☐ Ja, mor ☐ Ja, far ☐ Ja, søsken
Har, eller har noen hatt atferdsvansker vansker med konsentrasjon, lærevansker (ADHD o.l)
☐ Ingen ☐ Ja, mor ☐ Ja, far ☐ Ja, søsken

61. Røyker foreldre eller omsorgspersoner *(fyll ut alle aktuelle)?*
☐ Nei, verken mor eller far eller andre omsorgspersoner
☐ Ja, mor
☐ Ja, far
☐ Ja, samboer av mor eller far
☐ Ja, andre som bor i huset

62. Røykes det inne i huset?
☐ Nei ☐ Ja

63. Hva er høyeste fullførte utdanning for mor og far?
(Hvis dere for tiden holder på med en utdannelse, så kryss av denne)

MOR:
- ☐ 9-årig skole (ungdomsskole)
- ☐ 9-årig skole + 1-2 års videregående skole
- ☐ 9-årig skole + 3 år videregående skole (inkl. gymnas)
- ☐ Høyere utdanning, for eksempel distriktshøgskole, sykepleierhøgskole, lærerhøgskole
- ☐ Høyere utdanning på universitetsnivå

Mors etniske bakgrunn: ☐ Opprinnelig norsk
☐ Utenlandsk, hvilken nasjon?_____

FAR:
- ☐ 9-årig skole (ungdomsskole)
- ☐ 9-årig skole + 1-2 års videregående skole
- ☐ 9-årig skole + 3 år videregående skole (inkl. gymnas)
- ☐ Høyere utdanning, for eksempel distriktshøgskole, sykepleierhøgskole, lærerhøgskole
- ☐ Høyere utdanning på universitetsnivå

Fars etniske bakgrunn: ☐ Opprinnelig norsk
☐ Utenlandsk, hvilken nasjon?_____

64. Hva er mors og fars yrkesmessige situasjon?

MOR:
- ☐ Fulltidsarbeidende (minst 30 t/u)
- ☐ Deltidsarbeidende (under 30 t/u)
- ☐ Arbeidsledig/på tiltak/arbeidssøkende
- ☐ Student/elev
- ☐ Hjemmearbeidende
- ☐ Trygdet/under attføring
- ☐ Annet

Mors yrke: _____

FAR:
- ☐ Fulltidsarbeidende (minst 30 t/u)
- ☐ Deltidsarbeidende (under 30 t/u)
- ☐ Arbeidsledig/på tiltak/arbeidssøkende
- ☐ Student/elev
- ☐ Hjemmearbeidende
- ☐ Trygdet/under attføring
- ☐ Annet

Fars yrke: _____

FYLLES UT AV HELSESØSTER

Vennligst før på barnets mål

Dato for undersøkelsen: ☐☐ ☐☐ ☐☐

Høyde : ☐☐,☐ cm

Vekt : ☐☐,☐ kg (veid med truse og eventuelt T-skjorte)

Hodeomkr : ☐☐,☐ cm

Helsestasjon: _____

Helsesøster: _____

Vekst og helse blant barn i Oppland

STERKE OG SVAKE SIDER (SDQ-Nor)

Vennligts kryss av for hvert utsagn: Stemmer ikke, Stemmer delvis eller Stemmer helt.
Prøv å svare på alt selv om du ikke er helt sikker eller synes utsagnet virker rart.
For denne siden, svar på grunnlag av barnets oppførsel de siste 6 månedene.

Barnets navn: _____

Fødselsnummer: | | | | | | | | | | | |

	Stemmer ikke	Stemmer delvis	Stemmer helt
Omtenksom, tar hensyn til andre menneskers følelser	☐	☐	☐
Rastløs, overaktiv, kan ikke være lenge i ro	☐	☐	☐
Klager ofte over hodepine, vondt i magen eller kvalme	☐	☐	☐
Deler gjerne med andre barnt (godter, leker, andre ting)	☐	☐	☐
Har ofte raserianfall eller dårlig humør	☐	☐	☐
Ganske ensom, leker ofte alene	☐	☐	☐
Som regel lydig, gjør vanligvis det voksne ber om	☐	☐	☐
Mange bekymringer, virker ofte bekymret	☐	☐	☐
Hjelpsom hvis noen er såret, lei seg eller føler seg dårlig	☐	☐	☐
Stadig urolig eller i bevegelse	☐	☐	☐
Har minst en god venn	☐	☐	☐
Slåss ofte med andre barn eller mobber dem	☐	☐	☐
Ofte lei seg, nedfor eller på gråten	☐	☐	☐
Vanligvis likt av andre barn	☐	☐	☐
Lett avledet, mister lett konsentrasjonen	☐	☐	☐
Nervøs eller klengete i nye situasjoner, lett utrygg	☐	☐	☐
Snill mot yngre barn	☐	☐	☐
Lyver eller jukser ofte	☐	☐	☐
Plaget eller mobbet av andre barn	☐	☐	☐
Tilbyr seg ofte å hjelpe andre (foreldre, lærer, andre barn)	☐	☐	☐
Tenker seg om før hun/han handler (gjør noe)	☐	☐	☐
Stjeler hjemme, i barnehagen eller andre steder	☐	☐	☐
Kommer bedre overens med voksne enn med barn	☐	☐	☐
Redd for mye, lett skremt	☐	☐	☐
Fullfører oppgaver, god konsentrasjon	☐	☐	☐

Har du andre kommentarer eller bekymringer? (skriv her):

Vær så snill å snu arket – det er noen få spørsmål til på den andre siden

Samlet, synes du at dette barnet har vansker på ett eller flere av følgende områder:
Med følelser, konsentrasjon, oppførsel eller med å komme overens med andre mennesker?

Nei	Ja - små vansker	Ja - tydelige vansker	Ja - alvorlige vansker
☐	☐	☐	☐

Hvis du har svart "Ja", vennligst svar på følgende spørsmål:

• Hvor lenge har disse vanskene vært tilstede ?

Mindre enn én måned	1-5 måneder	6-12 måneder	Mer enn ett år
☐	☐	☐	☐

• Blir barnet selv forstyrret eller plaget av vanskene?

Ikke i det hele tatt	Bare litt	En god del	Mye
☐	☐	☐	☐

• Påvirker vanskene barnets dagligliv på noen av de følgende områdene ?

	Ikke i det hele tatt	Bare litt	En god del	Mye
Hjemme/ i familien	☐	☐	☐	☐
Forhold til venner	☐	☐	☐	☐
Læring i barnehagen	☐	☐	☐	☐
Fritidsaktiviteter	☐	☐	☐	☐

• Er vanskene en belastning for deg eller familien som helhet?

Ikke i det hele tatt	Bare litt	En god del	Mye
☐	☐	☐	☐

Jeg/vi som har fylt ut dette skjemaet er: ☐ Mor ☐ Far ☐ Annen omsorgsperson

---

Underskrift (er)

(dato)

Til foreldre

Forespørsel om å delta i spørreundersøkelse om 5-6-åringers vekst og helse

Hvorfor en spørreundersøkelse?

I et samarbeid mellom helsestasjonene i Oppland og barnepoliklinikkene på Lillehammer og Gjøvik ønsker vi å kartlegge en del forhold knyttet til vekst og helse hos alle barn på 5-6 år i Oppland.

Bakgrunnen er at vi vil starte et tverrfaglig program for å hindre at barn som er litt tunge på denne alderen, utvikler alvorlig overvekt. Vekst og vekt er i stor grad arvelig betinget og kan også ha sammenheng med forhold i svangerskapet og med barnets ernæring, fysiske aktivitet, fysisk og psykisk helse og sosiale forhold. For å kunne forstå hvordan slike forhold henger sammen og utvikle et effektivt opplegg, trenger vi å vite hva som er vanlig for barn på denne alderen med hensyn til vekt og høyde, ernæring, fysisk aktivitet og flere faktorer som har sammenheng med fysisk og psykisk helse. Vi trenger også å vite mors og fars høyde og vekt og litt om svangerskapet og familien.

Vi vil også spørre dere om samtykke til å få innhente opplysninger om svangerskapet fra sykehuset, opplysninger om barnets høyde og vekt fra tidligere helsestasjonskontakter og fra målingene som vil skje på skolen senere. På den måten vil vi finne ut om svangerskapsforhold (f.eks. svangerskapsforgiftning) kan påvirke vekst og om vi kan forutsi senere høyde og vekt ut fra målingene i tidlig barnealder.

Hvordan skjer spørreundersøkelsen?

Dere har fått tilsendt denne forespørselen og et spørreskjema sammen med innkallelsen til skolestartundersøkelsen ved helsestasjonen. Dere kan fylle ut skjemaet hjemme eller ved frammøte. Det er frivillig å delta, og dere må gjerne la være å fylle ut spørsmål dere ikke ønsker å svare på.

Hva skjer med spørreskjemaene?

Skjemaene vil bli samlet inn av helsesøster. Opplysningene lagres på et sikret datasystem ved Sykehuset-Innlandet HF. Opplysningene vil lagres på en slik måte at bare prosjektledelsen, som har taushetsplikt, har tilgang til filen og mulighet for å vite hvem opplysningene gjelder. All bearbeidelse av dataene vil skje på avidentifiserte datafiler, og det vil ikke være mulig å gjenkjenne enkeltpersoner i rapporter og publikasjoner.

Bearbeidelse av innsamlete data vil være avsluttet senest i 2015. Koden som knytter opplysningene til person, vil da makuleres. Dersom barnet fyller 16 år før 2015 ber vi om tillatelse til å kunne skrive til barnet og minne om at det er registrert i studien. Barnet vil da gis mulighet for å trekke seg fra studien. Dersom forskningen skulle vise at det er ønskelig å følge barna videre, vil vi spørre om ny tillatelse til å beholde koden. Dere har hele tiden rett til å vite hva som er registrert om dere og barnet og til å kreve eventuelle feil rettet opp.

Dere kan trekke dere fra prosjektet når som helst uten å behøve å oppgi noen grunn. Dersom dere trekker dere underveis, vil tidligere innsamlete opplysninger om barnet beholdes, men på en slik måte at ingen, heller ikke prosjektledelsen, kan knytte opplysningene opp mot et bestemt barn eller familie.

Finansiering og godkjenning av prosjektet

Undersøkelsen er finansiert av Sosial- og helsedirektoratet og Sykehuset innlandet HF. Den er vurdert av Regional komité for medisinsk forskningsetikk som ikke har innvendinger, og godkjent av Personvernombudet for forskning.

Vennligst fortsett på baksiden

Spørsmål

Dersom dere har spørsmål, kan disse rettes til helsesøster eller til en av oss i prosjektledelsen.

Anne Berit Sundby	Turid Skundberg	Jørgen Hurum	Trond Markestad
Prosjektsykepleier	Prosjektsykepleier	Ansvarlig overlege	Lege, prosjektleder
Barneavd. Lillehammer	Barnepol. Gjøvik	Barneavd. Lillehammer	Barneavd. SIHF
Tlf 61272231/97661916	Tlf 61157091/99587364	Tlf 06200/90766962	Tlf 97114143
anne.berit.sundby@	turid.skundberg@	Jorgen.hurum@	trond_markestad@
sykehuset-innlandet.no	sykehuset-innlandet.no	sykehuset-innlandet.no	hotmail.com

Samtykke

Det er nødvendig med skriftlig samtykke fra en eller begge foreldre for å kunne delta. For å innhente opplysninger fra svangerskapet, må vi ha skriftlig samtykke fra mor (se nedenfor).

Samtykkeerklæring:

Minst én av foreldrene må skrive under

Jeg/Vi har lest informasjonsskrivet og fått anledning til å spørre om eventuelle uklarheter.

☐ Jeg/Vi ønsker å delta i undersøkelsen nå da barnet er 5-6 år.

☐ Jeg/Vi gir tillatelse til at det innhentes opplysninger om vekt og høyde hos barnet fra helsestasjons- og skolehelseundersøkelsene.

☐ Jeg, som mor, gir tillatelse til at det innhentes opplysninger fra svangerskapet

..... (mor)

Deltagelse i prosjektet er frivillig, og dere kan når som helst trekke barnet fra prosjektet uten at dere behøver å oppgi noen grunn.

Dersom en trekker seg, vil tidligere innsamlete opplysninger om barnet beholdes, men på en slik måte at ingen, heller ikke prosjektledelsen, kan knytte opplysningene opp mot et bestemt barn eller familie. All personidentifikasjon vil altså fjernes.

Dato: .....

Navn (foresatte):..... .....

Navn (foresatte): .....

BMJ Open

Social and somatic determinants of underweight, overweight and obesity at 5 years of age: a Norwegian regional cohort study

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ABSTRACT

Objective To identify associations between the weight groups underweight (UW), overweight (OW) and obesity (OB) at 5 years of age and exposures related to pregnancy, anthropometric measures at birth, sociodemographic factors, and family health, anthropometric measures and habits.

Design Regional cohort study.

Setting Oppland County, Norway.

Methods Pregnancy data were obtained from a prospective perinatal register for children born in the county, and weight and height were measured by midwives at birth and by public health nurses at 5 years. Other information was obtained from questionnaires completed by parents.

Participants Of 1895 eligible children, current weight and height were obtained for all, weight and length at birth and information from parents for 1119 (59%) and pregnancy register data for 749 (40%) of the children. The significance of potential explanatory variables from descriptive statistics was tested in multinomial logistic regression analysis.

Results The prevalence of UW, OW and OB among participants was 7.8%, 10.6% and 3.5%, respectively. UW was associated with anthropometric measures at birth and those of parents, but not with sociodemographic or behavioural characteristics. OW and OB were associated with anthropometric measures of parents and siblings and with a variety of unfavourable social characteristics, lack of prolonged breast feeding, sedentary behaviour and dental caries, but not with current dietary habits. After adjustments, OW and OB were marginally related to birth parameters and diet and unrelated to physical activity, but significantly related to parental body mass index, low parental education and maternal smoking.

Conclusion The strong associations between sociodemographic and behavioural factors and OW and OB, but not with UW, may suggest that environmental factors are major contributing causes of OW and particularly OB at 5 years. These results may be helpful in targeting preventive measures against OW and OB.

Strengths and limitations of this study

- ► Height and weight were measured by health professionals and current measurements were obtained for all the eligible children.
- ► Information on the families' social characteristics, health, behaviour and habits was extensive.
- ► Overweight and obesity, but not underweight, were associated with sociodemographic and behavioural factors, suggesting that early childhood intervention aimed at environmental factors may be effective.
- ► Selection bias cannot be excluded since information other than current weight and height was missing for 41% of the children.

INTRODUCTION

The prevalence of overweight and obesity (OWOB) has increased throughout the world during the last decades and represents a major health problem.[1 2] Obesity in the paediatric population is particularly worrisome since obese children are at high risk of remaining obese as adults[3] and face the somatic, mental and social consequences of the condition.[4] It is currently estimated that 41 million children under 5 years of age are overweight (OW) or obese (OB) globally[5] and reported prevalence of OWOB in children aged 6–9 years in Europe are 18%–57% for boys and 18%–50% for girls.[6] In Norway, reported prevalence of OWOB in children aged 2–19 years is in the range 15%–17%.[7 8]

Both genetic and environmental factors are considered risk factors for OWOB.[9 10] Twin studies have shown a strong genetic component, but also effects of environmental factors, and adoption studies and other studies have supported the role of family environment. However, to our knowledge, there is no consensus regarding their relative significance. In Norway, as in other countries, children of divorced parents,[11] of mothers with low education[7] and children living in rural areas[12] were more often OW or OB, implying that environmental factors are

essential and that behaviour related to such sociodemographic factors needs to be addressed in the attempts to avoid persistent OWOB. For unknown reasons, the rising prevalence of OWOB in young children may have been somewhat curtailed in Norway[8] and other countries,[13] suggesting that preventive measures, such as increased attention to diet and physical activity may be effective.[8] However, strategies need to be targeted due to the large scale of the OWOB epidemic.

Due to the apparent multifactorial causes of OWOB, a broad variety of potentially explanatory variables needs to be included in studies in order to disentangle its complexity and thereby to understand how prevention and early treatment may be addressed more efficiently. Furthermore, the understanding of predictors of childhood OWOB may improve by addressing the significance of potential exposures in societies with different habits and prevalence of OWOB. Underweight (UW) in childhood has rarely been addressed in otherwise healthy children, and studies in Norway indicate that the prevalence of UW during early childhood has not changed in the same manner as OWOB during the last decades.[14 15] We therefore suggest that UW during early childhood is more related to genetic predisposition than OWOB and that including UW in studies of OWOB may add to the understanding of the OWOB epidemic. The aim of this population-based Norwegian study was to identify social and somatic determinants of UW, OW and OB at 5–6 years of age from comprehensive data obtained during pregnancy and early childhood.

METHODS

Participants

Parents of all children entering school in Oppland County, Norway, in 2007, were invited to participate in the study when bringing their children to the routine school entry health check-up at 5–6 years of age. Information on demographic and social factors, somatic health, behaviour and family habits was collected by means of a parental questionnaire completed at home, and current height and weight, and crown–heel length and weight registered at birth were reported by the public health nurses. For children of families who declined to participate, anonymous information on current age, sex, weight and height was obtained and reported by the public health nurse. For children born in Oppland County, parents were asked for permission to link the data to the Oppland perinatal database. In this database, data on mothers' prepregnancy height and weight, weight at the end of pregnancy, health and habits before and during pregnancy, birth and infant characteristics were recorded prospectively.

In Norway, virtually all families attend the standard follow-up programme for children from birth to school entry in the public child health clinics. In 2007, the population of Oppland County, which is 1 of 20 counties in Norway, was approximately 183 000. The county has 26 municipalities; two of them are cities with 25 000–30 000 citizens in each, while the rest are rural municipalities with towns of variable sizes.

The study was approved by the Regional Committee on Medical Research Ethics (REK 1.2006.3491) and the Norwegian Data Protection Official for Research (02–2006 SI). A signed consent was obtained from one of the parents.

Details about measurements and variables used for describing characteristics and evaluated as potential exposures of UW, OW and OB are presented in online supplementary table 1.

Measurements

Birth weight (BW) and crown–heel length were measured by midwives at the time of birth and reported to the public healthcare clinics. Height and weight at 5 years were measured by the public health nurses. Body mass index (BMI) was calculated as weight/height^2 (kg/m^2). Prepregnancy height and weight were self-reported, and weight at the end of pregnancy was measured in the hospital prior to delivery. Pregnancy weight gain was calculated and classified as appropriate or excessive according to US guidelines from 2009.[16]

Definitions of the weight groups UW, normal weight (NW), OW and OB were based on the sex-specific and age-specific BMI criteria (iso-BMI) of the International Obesity Task Force[17 18] for the children and according to the WHO classification for their parents.[19] SD scores (SDS) for the children were based on the current Norwegian growth references.[20] Infants were defined as being born small for gestational age (SGA), appropriate for gestational age or large for gestational age according to sex-specific Norwegian percentiles.[21]

Variables

The main outcomes were UW, OW and OB at 5 years of age.[17] Exposure variables were anthropometric measurements at birth and a wide set of information provided by the parents in the questionnaire and data from the pregnancy database. We dichotomised several ordinal variables in order to create variables that allowed for meaningful comparisons between weight groups and avoid excessive skewness.

Frequencies of different meals and consumption of various food and drink items were recorded, and among these we chose to use frequency of consumption of fruits and vegetables as an index of healthy nutrition and of sugar-sweetened beverages and sweets/snacks as an index of unhealthy nutrition. Physical activity was rated by parents in three dimensions, endurance, gross motor skills and level of activity in sports and play, and a combined activity score was computed and dichotomised as below or at/above a score of 6 (high score reflecting low activity). Additionally, physical activity per week was reported as frequency and as hours of being active enough to experience heavy breathing or sweating. Screen time (TV, videogames and so on) was recorded as hours per day. Binary exposure variables in addition to sex were

(yes/no) premature birth, kindergarten since 2 years of age, antibiotics more than three times, asthma medication, dental caries, exclusive breast feeding for more than 4 months, television in the child's bedroom, living with one caretaker, having siblings, maternal and paternal education beyond high school, mother smoking, parents working full-time or part-time, parents of western origin (Europe and North America) and parents with UW, OW or OB. Place of residence was categorised as urban and rural.

From the Oppland perinatal database, we obtained prepregnancy height and weight, pregnancy weight gain, BW and the following binary variables (yes/no): mother working before pregnancy and smoking in pregnancy.

Statistics

Descriptive statistics are presented as counts and per cents (%) for categorical variables and means with SD for continuous variables. Participants and children whose parents declined were compared using Student's t-test and Pearson's χ^2 test. Among participants, differences in characteristics between the four weight groups (BMI category: UW, NW, OW and OB) were explored using one-way analysis of variance (ANOVA) for continuous and χ^2 test for categorical variables. When ANOVA or χ^2 test across all weight groups were significant, we performed post hoc pairwise testing, comparing groups to NW children, by Dunnett's t-test (continuous variables) or χ^2 test (categorical variables).

The significance of potential explanatory variables on outcome in terms of the current weight groups UW, OW and OB relative to NW was first analysed in separate bivariate analyses with the ordinal weight group variable (increasing from UW through NW, OW and OB) at 5 years of age, and thereafter assessed in a multinomial logistic regression analysis with variable-specific reference categories for categorical variables or per unit increase for continuous variables. In developing the regression model, all explanatory variables that in bivariate analyses showed significant relation to BMI category in ANOVA or χ^2 test were explored for inclusion and significance in a multinomial regression model. The explanatory variables included in an a priori model were based on earlier literature and strength of association in the post hoc bivariate analyses following the ANOVA and χ^2 tests, that is, BMI of the mother and father, crown–heel length SDS and weight SDS at birth, maternal education, single parent and maternal and paternal smoking. To avoid excessive loss of statistical power due to a high number of variables in a limited study sample, we then sequentially tested inclusion of potential explanatory variables *one at a time* into the model. All variables found significant during this sequential testing were assessed in combination using an all-in backward stepwise selection procedure. In the final model, we tested 38 possibly relevant interactions between the remaining variables. They were selected on the basis of literature and theoretical considerations by the authors. Important variables were tested for different interactions and multicollinearity. Finally, the effects of parental education and parental BMI group (stratified) on child outcome in terms of UW and OWOB versus NW were assessed in two separate logistic regression models based on the variable selection in our final multinomial model. SPSS Statistics for Windows V.21.0 was used for all analyses. p Values ≤0.05 was considered statistically significant. The BMI SDS were calculated in R V.2.6.0 (The R Foundation for Statistical Computing, Vienna, Austria) using the Norwegian growth references.[20]

RESULTS

The mean age (SD) of all eligible children (n=1895) was 5.70 (0.49) years, and 923 (48.7%) were boys. The mean BMI (SD) was 15.93 (1.97) for the girls and 15.92 (1.71) for the boys (p=0.90). There were no significant sex differences in prevalence of UW, OW or OB (p=0.28), that is, the respective prevalence for boys and girls were 9.1% versus 8.5% for UW, 10.4% versus 13.1% for OW and 3.7% versus 4.2% for OB. For 2.7% of the children, one or both parents were of non-Western, mostly Asian, ethnicity.

Of the 1119 families who answered the questionnaires (59% of eligible families), 31 were excluded from further analyses because data on height, weight or age at 5 years were missing. The participation rate varied from 20% to 85% for the various municipalities in the county. The mean BMI did not differ between the participants and those who declined, but the prevalence of OW and OB were slightly lower for the participants (table 1). Pregnancy data were available for 749 of the children. This subgroup did not differ from those without pregnancy data on any of the exposure or outcome variables (data not shown).

In separate bivariate analyses, the ordinal weight group variable (increasing from UW through NW, OW and OB) at 5 years of age was positively associated with *exposures during foetal and early life*, that is weight, BMI and their respective SDS at birth, prepregnancy weight, excessive pregnancy weight gain and smoking during pregnancy and exclusive breast feeding less than 4 months (table 2a), with unfavourable *current child health and habits*, that is, increasing rate of dental caries, a combined physical activity score lower than peers and TV in the bedroom (table 2b) and *sociodemographic factors and health of the family*, that is, increasing prevalence of low parental education, father out of work, parents of non-Western ethnicity, single parenthood, smoking mothers, being an only child and increasing BMI and prevalence of obesity among parents and siblings (table 2c). Increasing weight group was not associated with being a twin, having a chronic disease (prevalence 2.1%, eg, diabetes, heart disease and coeliac disease), hospital admissions, being breast fed or not (overall prevalence 89.3%, range 81.6–90.6 between the groups), frequency or extent of physical activity, screen time, teenage mother, pre-eclampsia,

Table 1 Characteristics of the children who participated and declined

Characteristic	Participated (n=1088)	Declined (n=776)	p Value*
Age (years), mean (SD)	5.71 (0.44)	5.68 (0.57)	0.185
Height (cm), mean (SD)	116.4 (5.8)	116.1 (5.7)	0.307
Weight (kg), mean (SD)	21.62 (3.55)	21.73 (4.01)	0.536
Body mass index (kg/m^2), mean (SD)	15.87 (1.71)	16.01 (2.02)	0.107
Boys, n (%)	526 (47.0)	397 (51.2)	0.075
Weight groups, n (%)			0.018
Underweight†	85 (7.8)	78 (10.1)	
Normal weight†	850 (78.1)	553 (71.7)	
Overweight†	115 (10.6)	104 (13.5)	
Obese†	38 (3.5)	36 (4.7)	
Overweight or obese	153 (14.1)	140 (18.2)	0.017

*Students' t- and χ^2 tests.
†Based on body mass index.[17]

gestational diabetes, gestational age at birth or mode of delivery (online supplementary table 2).

When compared with NW children in separate bivariate analyses, UW at 5 years was significantly associated with lower weight, BMI and being SGA at birth, and lower prepregnancy and current maternal, paternal and sibling BMI, but not with indices of habits related to nutrition, physical activity or sociodemographic factors (table 2a-c). OW and OB, however, were not significantly associated with weight, length or BMI at birth, or with being born with BW above 4000 g, but with higher prepregnancy BMI and current high maternal, paternal and sibling BMI, pregnancy weight gain above recommended, maternal smoking during pregnancy and current smoking status, limited breast feeding and low parental education. Parents of children with OW and OB reported that their children had a poorer combined physical activity score than their peers, that is, lower endurance, motor skills and physical activity, and the OW and OB children were more often living with one caretaker and with parents of non-western ethnicity. OB children more often had TV in the bedroom and dental caries, but according to the parents, they ate sweets as frequently as their NW peers (table 2a-c).

In the multinomial logistic regression model, the only significant interaction was between maternal BMI and BW SDS, and this interaction was included in the final model (table 3). Test for possible multicollinearity revealed a moderately reduced tolerance for BW SDS and birth length SDS (both 0.33), but minimal multicollinearity among other covariates. Overall, deviations from NW (UW, OW and OB) at 5 years were significantly associated with maternal age, education and smoking habits, and maternal and paternal BMI (equally strong), having no siblings, eating fruit 5 days or more often per week and weight and length SDS at birth (table 3). UW was associated with low paternal BMI, low BW SDS and high birth length SDS, OW with low maternal education and smoking, high parental BMI, no siblings, eating fruits 5 days or more often per week and high BW SDS, and OB with low maternal age, high parental BMI, low maternal education and maternal smoking (table 3). The model explained 13% of the variance (McFadden's pseudo-R-squared).

Since there was a significant interaction between the associations of maternal BMI and offspring BW SDS and the risk of childhood OWOB, we performed additional multinomial regression analyses with stratification by the interaction variables. In this model, increasing BW was only significantly associated with increasing risk of childhood OW when mothers had NW or were overweight, but not when the mothers were obese, and BW was not associated with risk of childhood obesity. However, increasing maternal BMI was only associated with a risk of OW and OB for children with medium BW SDS, and not with low or high BW SDS (online supplementary table 3).

In the multivariable logistic regression analyses, OWOB was strongly associated with parental OWOB and low education, especially on part of the mother (table 4a). These effects were still strong after adjusting for the somatic and environmental factors in the multinomial model in table 3. For UW children, there were no such associations (table 4b).

DISCUSSION

The prevalence of OW and OB was lower than reported for this age group in many other countries[6 22] but similar to a previous report from another part of Norway.[7] The broad picture from the unadjusted analyses was that high BMI at 5 years of age was related to a wide range of unfavourable sociodemographic and behavioural factors in addition to high parental BMI and marginally to anthropometric measures at birth. However, in the adjusted analyses, OW and OB and OWOB combined, were still closely related to high parental BMI and sociodemographic and

Table 2 Association between weight groups of the children at 5 years of age and early life predictors (2a), early childhood and current health and habits of the children (2b) and current sociodemographic and health characteristics of parents and siblings (2c). For variables with significant associations, pairwise bivariate analyses with normal weight as reference was performed

Characteristics (mean (SD) or %)	Valid n	All (n=1088)	Underweight (n=85)	Normal weight (n=850)	Overweight (n=115)	Obese (n=38)	p¶
2a. Early life predictors							
Girls	1088	52.5	51.8	51.4	59.1	57.9	0.41
Birth weight (kg)	1069	3.56 (0.61)	3.29 (0.65)*	3.58 (0.59)	3.57 (0.66)	3.72 (0.61)	<0.01
Birth weight SDS	1060	−0.27 (1.35)	−0.87 (1.49)*	−0.23 (1.31)	−0.25 (1.44)	0.07 (1.32)	<0.01
Crown–heel length at birth (cm)	963	50.54 (2.41)	49.90 (2.67)	50.62 (2.41)	50.37 (2.29)	50.73 (2.10)	0.09
Crown–heel length SDS at birth	954	−0.02 (1.17)	−0.33 (1.31)	0.01 (1.16)	−0.10 (1.10)	0.10 (0.97)	0.10
BMI SDS birth	940	−0.15 (0.99)	−0.64 (1.08)*	−0.13 (0.97)	−0.02 (0.97)	−0.01 (0.94)	<0.01
Birth weight <2500 g	1069	4.8	9.8	4.2	6.1	2.8	
>4000 g		22.3	12.2	22.7	22.8	33.3	0.06
Birth weight <10 percentile	746	4.3	10.7	3.6	4.1	7.4	0.07
Birth weight >90 percentile	744	11.8	5.4	12.3	12.2	14.8	0.46
Premature birth	1088	6.8	10.6	6.6	7.0	2.6	0.39
Exclusive breast feeding >4 months	1003	72.8	80.9	73.9	65.6	51.7*	<0.01
Mothers' age at birth (years)	1088	29.90 (4.78)	29.16 (4.46)	30.03 (4.73)	30.26 (5.27)	27.52 (4.57)*	<0.01
Mothers' height (cm)	1025	167.80 (5.82)	167.78 (6.48)	168.01 (5.73)	166.56 (5.67)*	165.69 (6.23)	0.01
Prepregnancy BMI (kg/m^2)	673	24.15 (4.20)	22.76 (3.22)	23.91 (3.96)	26.18 (4.85)*	27.43 (5.43)*	<0.01
Gestational weight gain, kg	678	15.15 (5.56)	14.82 (5.64)	15.22 (5.53)	15.10 (5.68)	14.14 (6.30)	0.81
Gestational weight gain above recommended	661	54.4	34.0	41.7	59.4*	50.0	0.03
Smoking at week 18 in pregnancy	738	15.9	7.0	13.3	34.2*	39.3*	<0.01
2b. Children's early childhood and current health and habits							
Current child height SDS	1025	0.18 (1.03)	0.10 (1.08)	0.11 (1.00)	0.55 (1.13)*	0.77 (0.89)*	<0.01
Current child BMI SDS	1085	0.01 (1.03)	−1.91 (0.55)*	−0.10 (0.65)	1.41 (0.27)*	2.36 (0.44)*	<0.01
Asthma medication†	1058	11.6	9.8	11.0	14.7	21.1	0.18
Antibiotics ≥3 times‡	1088	23.8	20.0	23.6	24.3	34.2	0.14
Dental caries	1079	20.0	18.8	18.7	25.9	34.2*	0.04
Combined physical activity score poorer than peers§	1073	5.1	5.9	3.8	8.0*	24.3*	<0.01
Kindergarten since 2 years old	1033	92.1	91.5	92.7	90.6	82.4	0.16
TV in the child's bedroom	1084	14.1	12.9	13.0	15.7	38.9*	<0.01
Vegetables <5 times/week	1071	50.6	56.5	50.6	44.2	56.8	0.31
Fruits <5 times/week	1078	19.5	21.2	20.2	11.3	24.3	0.12

Continued

Table 2 Continued

Characteristics (mean (SD) or %)	Valid n	All (n=1088)	Underweight (n=85)	Normal weight (n=850)	Overweight (n=115)	Obese (n=38)	p¶
Sugar-sweetened beverages >1 time/week	1077	31.2	29.4	31.6	29.8	29.7	0.95
Sweets/snacks >1 time/week	1079	42.7	34.5	43.4	45.1	37.8	0.38
2c. Sociodemographic factors and health characteristics of parents and siblings							
Living in a municipality with >20 000 inhabitants	1087	32.4	38.8	32.6	30.4	18.4	0.16
Maternal education above high school	1068	55.1	55.0	58.3	43.9*	18.9*	<0.01
Paternal education above high school	1037	39.3	48.1	41.1	27.8*	14.3*	<0.01
Both parents education above high school	1077	32.5	39.7	34.4	21.3*	5.9*	<0.01
Mother working (full-time/part-time, student)	1069	91.8	91.5	92.5	90.3	81.1	0.09
Father working (full-time/part-time, student)	1046	95.2	95.1	96.0	92.9	84.8*	0.02
One or both parents from non-Western countries	1088	2.7	3.5	2.1	3.5	10.5*	0.01
Living with one caretaker	1073	13.0	9.8	12.0	17.5	29.7*	<0.01
No siblings	1065	9.1	3.6	9.0	16.8*	2.9	<0.01
Mother smoking	1088	19.0	14.1	16.6	32.2*	44.7*	<0.01
Current BMI mother (kg/m^2)	981	24.32 (3.85)	23.06 (3.31)	24.07 (3.66)	26.03 (4.15)*	27.52 (5.12)*	<0.01
Current BMI father (kg/m^2)	924	26.38 (3.30)	25.13 (2.72)*	26.25 (3.16)	27.57 (3.75)*	28.89 (4.33)*	<0.01
BMI SDS average for siblings	630	–0.21 (1.23)	–0.76 (1.26)*	–0.24 (1.20)	0.26 (1.06)*	0.55 (1.45)*	<0.01
Mother currently obese	980	9.1	6.5	7.2	16.8*	34.3*	<0.01
Father currently obese	924	13.0	3.9*	11.2	26.1*	37.5*	<0.01
Both parents currently obese	891	2.7	1.4	2.0	3.4	19.4*	<0.01

*Post hoc pairwise testing by Dunnett's t-test (continuous variables) or χ^2 (categorical) showed a significant difference ($p \leq 0.05$) compared with children of normal weight at 5 years of age; only when significant ANOVA or χ^2 across groups.
†Steroids or other maintenance medications or medication for asthma attacks after 2 years of age.
‡Since birth irrespective of cause.
§Computed from the three dimensions of physical activity: frequency, intensity and endurance, each scored from 1 to 3 (better than peers, like peers and worse than peers), giving a total score of 3–9, where 9 meant poorer than peers in all three dimensions. The figure is the prevalence of children with a score of 6 or higher.
¶Analyses across weight groups, ANOVA for continuous variables, χ^2 for categorical.
ANOVA, analysis of variance; BMI, body mass index; SDS, SD score.

Table 3 Significant* predictors of underweight (UW), overweight (OW) or obesity (OB) at 5 years of age from multinomial logistic regression analysis with normal weight (NW) children as reference†

Variables *Reference category*	Valid n	Weight group (BMI)†	Unadjusted‡		Adjusted model§ (n=709)		Final model¶** (n=759)	
			OR (95% CI)††	P	OR (95% CI)††	p	OR (95% CI)††	p
Age of mother at birth	1088	UW	0.96 (0.92 to 1.01)	0.005	0.95 (0.89 to 1.01)	0.043	0.95 (0.89 to 1.01)	0.059
		OW	1.01 (0.97 to 1.05)		1.02 (0.97 to 1.08)		1.02 (0.96 to 1.07)	
		OB	0.89 (0.83 to 0.96)		0.90 (0.82 to 1.00)		0.91 (0.83 to 1.00)	
Current BMI mother	981	UW	0.91 (0.84 to 0.98)	<0.001	0.95 (0.87 to 1.04)	0.002	0.96 (0.87 to 1.05)	0.002
		OW	1.13 (1.07 to 1.18)		1.10 (1.03 to 1.17)		1.10 (1.04 to 1.17)	
		OB	1.19 (1.12 to 1.28)		1.14 (1.03 to 1.25)		1.13 (1.03 to 1.25)	
Current BMI father	924	UW	0.88 (0.81 to 0.96)	<0.001	0.86 (0.77 to 0.96)	0.001	0.86 (0.78 to 0.95)	0.001
		OW	1.12 (1.05 to 1.19)		1.05 (0.98 to 1.14)		1.08 (1.01 to 1.17)	
		OB	1.22 (1.12 to 1.33)		1.14 (1.02 to 1.28)		1.08 (0.97 to 1.20)	
Birth weight SDS	1060	UW	0.73 (0.63 to 0.85)	<0.001	0.54 (0.35 to 0.82)	0.003	0.34 (0.06 to 1.89)	0.005
		OW	0.99 (0.85 to 1.15)		1.49 (1.01 to 2.18)		6.38 (1.50 to 27.20)	
		OB	1.21 (0.92 to 1.59)		0.87 (0.45 to 1.68)		0.16 (0.02 to 1.24)	
Birth length SDS	954	UW	0.80 (0.67 to 0.97)	0.121	1.51 (0.99 to 2.32)	0.071	1.57 (1.04 to 2.37)	0.020
		OW	0.92 (0.77 to 1.01)		0.74 (0.50 to 1.09)		0.68 (0.45 to 1.02)	
		OB	1.07 (0.78 to 1.47)		1.27 (0.62 to 2.60)		1.32 (0.73 to 3.24)	
Birth weight SDS*current BMI mother‡‡	961	UW					1.02 (0.95 to 1.09)	0.025
		OW					0.95 (0.90 to 1.00)	
		OB					1.07 (0.99 to 1.14)	
Sex	1088			0.406		0.540		0.602
Boys		UW	0.99 (0.63 to 1.54)		0.93 (0.53 to 1.65)		0.93 (0.54 to 1.60)	
		OW	0.73 (0.49 to 1.09)		0.72 (0.42 to 1.24)		0.78 (0.46 to 1.33)	
		OB	0.77 (0.40 to 1.49)		0.63 (0.25 to 1.62)		0.61 (0.25 to 1.51)	
Girls (reference)		UW	1		1		1	
		OW	1		1		1	
		OB	1		1		1	
Siblings	1065			0.09		0.008		0.002
No siblings		UW	0.38 (0.12 to 1.22)		0.32 (0.07 to 1.55)		0.23 (0.05 to 1.08)	
		OW	1.96 (1.12 to 3.43)		3.13 (1.40 to 6.99)		3.13 (1.45 to 6.74)	
		OB	0.31 (0.04 to 2.28)		0.36 (0.04 to 3.31)		0.34 (0.04 to 3.11)	

Continued

Table 3 Continued

Variables *Reference category*	Valid n	Weight group (BMI)†	Unadjusted‡ OR (95% CI)††	P	Adjusted model§ (n=709) OR (95% CI)††	p	Final model¶** (n=759) OR (95% CI)††	p
One or more siblings (reference)		UW	1		1		1	
		OW	1		1		1	
		OB	1		1		1	
Maternal education	1068			<0.001		0.068		0.022
Maximum high school		UW	1.14 (0.72 to 1.82)		1.27 (0.67 to 2.40)		1.26 (0.64 to 2.30)	
		OW	1.79 (1.21 to 2.66)		1.60 (0.92 to 2.80)		1.75 (1.01 to 3.01)	
		OB	5.99 (2.6 to 13.80)		3.13 (1.05 to 9.32)		3.54 (1.20 to 10.42)	
Above high school (reference)		UW	1		1		1	
		OW	1		1		1	
		OB	1		1		1	
Maternal smoking	1088			<0.001		0.106		0.018
Mother smoking		UW	0.83 (0.44 to 1.56)		0.76 (0.31 to 1.89)		0.66 (0.28 to 1.57)	
		OW	2.39 (1.55 to 3.67)		2.05 (1.09 to 3.84)		2.08 (1.14 to 3.79)	
		OB	4.07 (2.09 to 7.91)		1.74 (0.65 to 4.65)		2.57 (1.03 to 6.42)	
Mother not smoking (reference)		UW	1		1		1	
		OW	1		1		1	
		OB	1		1		1	
Fruit	1078			0.088		0.012		0.022
≥5 days/week		UW	0.94 (0.55 to 1.63)		0.97 (0.45 to 2.08)		0.98 (0.48 to 2.02)	
		OW	1.99 (1.09 to 3.63)		3.35 (1.36 to 8.28)		3.41 (1.39 to 8.38)	
		OB	0.79 (0.37 to 1.70)		2.80 (0.71 to 9.86)		1.64 (0.54 to 4.97)	
<5 days/week (reference)		UW	1		1		1	
		OW	1		1		1	
		OB	1		1		1	
Exclusive breast feeding	1003			0.004		0.186		
<4 months		UW	0.73 (0.43 to 1.25)		0.85 (0.43 to 1.67)			
		OW	1.49 (0.98 to 2.25)		1.14 (0.65 to 2.00)			
		OB	2.69 (1.35 to 5.33)		2.72 (1.06 to 7.00)			
≥4 months (reference)		UW	1		1			
		OW	1		1			
		OB	1		1			

Continued

Table 3 Continued

Variables *Reference category*	Valid n	Weight group (BMI)†	Unadjusted‡ OR (95% CI)††	P	Adjusted model§ (n=709) OR (95% CI)††	p	Final model¶** (n=759) OR (95% CI)††	p
Caretakers	1073			0.014		0.392		
Living with one caretaker		UW	0.79 (0.37 to 1.69)		0.69 (0.19 to 2.55)			
		OW	1.56 (0.92 to 2.63)		1.22 (0.50 to 2.97)			
		OB	3.10 (1.48 to 6.46)		2.99 (0.83 to 10.78)			
Living with two caretakers (reference)		UW	1		1			
		OW	1		1			
		OB	1		1			

*Exposure variables (table 2a-c) that did not remain significant in this model are not included.
†Definitions of weight class according to International Obesity Task Force age-specific and sex-specific cut-off values.
‡Bivariate multinomial logistic regression.
§Adjusted model created through an all-in backward stepwise selection procedure.
¶The final model was created from the adjusted model by including the significant interaction between birth weight SDS and maternal BMI, and excluding two variables from the adjusted model: breastfeeding due to high number of missing (and p>0.1 in the adjusted model) and living with two caretakers due to p>0.1.
**Explained variance of the final model=0.13 (McFadden's pseudo-R-squared).
††Odds of being in the respective BMI category group compared with reference category (NW) for categorical variables, and per unit increase in continuous variables.
‡‡Interaction between birth weight SDS and current BMI mother.
BMI, body mass index; OB, obese; OW, overweight; SDS, SD score; UW, underweight.

Table 4 Relative risk of overweight or obesity (OWOB, table 4a) and underweight (UW, table 4b) at 5 years of age expressed as ORs and 95% CIs according to parental level of education and presence or absence of OWOB or UW. Higher education was defined as one or both parents having education beyond high school

	Valid n (total n=963)	OR (95% CI) Unadjusted	OR (95% CI) Adjusted*
4a. Relative risk of OWOB			
Normal weight parents with higher education	196	1	1
Normal weight parents with lower education	87	3.6 (1.5 to 8.4)	3.3 (1.3 to 8.6)
OWOB parents with higher education	416	2.4 (1.2 to 5.0)	2.1 (1.0 to 4.5)
OWOB parents with lower education	264	5.1 (2.5 to 10.3)	4.6 (2.1 to 9.9)
	Valid n (total n=953)	**OR (95% CI) Unadjusted**	**OR (95% CI) Adjusted***
Normal weight mother with higher education	375	1	1
Normal weight mother with lower education	242	2.1 (1.3 to 3.4)	3.0 (1.6 to 5.8)
OWOB mother with higher education	162	2.3 (1.3 to 3.9)	3.4 (1.7 to 6.8)
OWOB mother with lower education	174	5.0 (3.0 to 8.1)	6.2 (3.3 to 11.7)
	Valid n (total n=901)	**OR (95% CI) Unadjusted**	**OR (95% CI) Adjusted***
Normal weight father with higher education	144	1	1
Normal weight father with lower education	185	2.6 (1.5 to 4.5)	2.6 (1.0 to 6.8)
OWOB father with higher education	223	2.8 (1.5 to 5.1)	1.9 (0.7 to 5.1)
OWOB father with lower education	349	6.2 (3.6 to 10.5)	4.2 (1.7 to 10.2)
4b. Relative risk of underweight (UW). Since no fathers were UW, only the mothers' data are presented.			
	Valid n (total n=634)	**OR (95% CI) Unadjusted**	**OR (95% CI) Adjusted***
Normal weight mother with higher education	375	1	1
Normal weight mother with lower education	242	1.4 (0.8 to 2.3)	1.3 (0.6 to 2.6)
UW mother with higher education	9	3.0 (0.6 to 14.8)	2.4 (0.4 to 13.8)
UW mother with lower education	8	n/a	n/a

*Adjusted for the variables in the final model in table 3 (sex, maternal smoking, siblings, fruit >5 days/week, maternal age, birth weight SDS, birth length SDS) stratified by combinations of parental body mass index and education.

behavioural factors, while OW, but not OB, was positively related to BW. Furthermore, increasing BW was only associated with OW for offspring of mothers who were not obese, while high maternal BMI was only associated with OW and OB for children with normal BW. UW was mainly related to paternal BMI and measurements at birth, but not to sociodemographic or behavioural factors. Thus, as the risks of UW as well as OW and OB were related to parental BMI, genetic factors probably contributed in all groups, but from the overall pattern, we suggest that sociodemographic and behavioural factors were the most important risk factors of OW and OB at 5 years and that genetic factors were a relatively more important risk factor of OW than OB. This speculation may be plausible since BMI of OW in early childhood to a significant part may reflect high lean body mass. UW was unrelated to environmental factors, but low parental and child BMI may reflect both genetic heredity and shared environmental factors, and we cannot exclude that rigorous behaviour towards presumed healthy living may have contributed to the risk of UW since our exposure variables were rather crude and based on information on behaviour provided by the parents. Furthermore, the study possibly lacked statistical power to detect effects of environmental factors due to the relatively low number of UW children.

High parental BMI may reflect genetic vulnerability towards OWOB,[23] as well as unfavourable in utero environmental influences[16 24] and postnatal maternal and family dietary and other shared habits.[25 26] Such relationships may affect the offspring through foetal programming or developmental plasticity, and this relationship may be signalled, or possibly mediated, by weight and body composition at birth.[27 28] Heavier mothers tend to give birth to heavier babies[29] and babies with a higher relative fat mass,[30] and studies have shown a positive association between higher BW and OB in later childhood and adulthood.[31 32] Our finding that anthropometric measures at birth were associated with OW, but not with OB, in the adjusted analyses may indicate that shared environmental factors were relatively more important for the

development of excess fat mass than lean body mass since OB is usually characterised by higher body fat percentage than OW.[33 34]

Offspring of smoking mothers tend to have lower BW, but they are reported to have a higher risk of later adiposity.[35] In our study, maternal smoking remained a significant risk factor for OW and OB after adjusting for other factors, but whether smoking just reflected other unfavourable family habits or had a direct effect remains speculative. Intrauterine growth restriction has also been associated with risk of adiposity,[36 37] but no such tendency was observed at the age of 5 years in the present study.

We found a cumulative effect of parental BMI and education for the outcome OWOB but not for UW (table 4). The effects on OWOB were present for both parents but were considerably stronger for the mothers than the fathers. This may suggest a more profound effect of maternal factors, possibly through genetic factors or fetal programming, or merely reflect the mothers' dominating influence on family habits, at least when the children are young.

Our findings that the prevalence of OWOB did not differ between urban and rural living or between single caretaker and two-parent families in adjusted analyses are in agreement with some,[7 38 39] but not with other studies.[11 12 40 41] The lack of effect of urbanisation may be due to the small size of the cities and their proximity to rural areas or that relevant differences may not occur or take effect until later[12] when differences in everyday transportation to school and leisure time activities may differ. The non-significant association between living with a single caretaker and risk of OB may be due to lack of statistical power since 87% of the children lived with two caretakers. It is remarkable that dietary habits were not or only marginally associated with BMI, OW or OB, as positive associations have been found in other Norwegian studies.[42–44] It is possible that unhealthy diets were under-reported, as suggested for OW children in another study,[45] but another possibility was reversed causality, that is, that the OWOB children were put on more healthy or energy restricted diets because they were overweight. Reported physical activity did not differ much between the groups. The reason may be that differences in physical activity are small at this young age when spontaneous play is predominant and more organised activity is largely limited to joint activities in kindergarten, which was attended by more than 90% of the children. Alternatively, the questions related to physical activity may have been too weak and subjective to disclose differences. In the present study, there were also tendencies towards positive relationships between additional socioeconomic and environmental exposures and OWOB, also in the adjusted analyses, like limited breast feeding, use of asthma medications and antibiotics, presence of caries and TV in the child's bedroom. These findings may be of interest as such associations have been reported by others,[46–50] and the current study may have been too small to detect significant associations. Whether associations with asthma and antibiotics are directly related to the medications, indirectly to the effects of disease or just were markers of harmful family habits or environments, such as household smoking, need to be explored.

The strengths of this study were both the knowledge of weight and height for all eligible children in the county and the extensive information on those who participated. We cannot exclude some selection bias since there were slightly more children with UW, OW and OB in the families who declined participation. However, the difference in growth parameters were small, and limited participation were probably mainly due to variation in willingness of public health nurses to recruit families rather than variation in willingness on part of the families, as suggested by the wide variation in participation rates in different municipalities. Recall bias may have led to underestimation of true effects of certain exposures, and the relatively small numbers of OW and UW, and particularly OB children, may have resulted in a lack of statistical power to detect additional true risk factors for these categories. Parental weight, height and habits, for example, related to nutrition and physical activity, were based on self-report and may be biased, although it has been shown that self-reported weights and heights are closely related to actual measurements in adults.[51] In general, it is important to keep in mind that cross-sectional associations in observational studies may not be causal or may result from reverse causality.

CONCLUSION

Our results suggest that the predictive value of BW and other measures of size at birth may be relatively high for UW, but limited for OW and, in particular, for OB, at 5 years of age and that high parental BMI, particularly when combined with unfavourable family sociodemographic factors, are the dominant inducting risk factors for early childhood OWOB. Prevention is essential in order to curtail the OWOB epidemic, and in our opinion, this study shows that families with these risk factors should be targeted for close intervention early, preferably before or during pregnancy.

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Supplementary Table 1. Study variables: definitions and data sources.

Data sources for the extracted variables:

- OHGS: Oppland health and growth study
- OPD: Oppland perinatal database

Covariate	Categories / definitions
Early life predictors	
Body mass index (BMI) category groups	Based on sex and age specific BMI criteria of the International Obesity Task Force (Iso-BMI) [1 ,2] Iso-BMI are sex specific BMI values equivalent to specific adult values (e.g. adult BMI 25) at different ages Underweight: Iso-BMI < 18.5 Normal weight: Iso-BMI 18,5-25 Overweight: Iso-BMI 25-30 Obesity: Iso-BMI >30
Birth weight	Continuous measure, kg (two decimals) Recorded by midwives immediately after birth. OHGS
Birth weight standard deviation score (SDS)	Based on current Norwegian growth references [3] OHGS
Crown-heel length at birth (cm)	Continuous measure, cm Recorded by midwives immediately after birth. OHGS
Crown-heel length SDS at birth	Based on current Norwegian growth references [3] OHGS
BMI SDS birth	Based on current Norwegian growth references [3] OHGS
Small for gestational age (SGA)	Birth weight < 10^{th} percentile for gestational age Based on recent Norwegian birth percentiles [4] OPD
Large for gestational age (LGA)	Birth weight > 90^{th} percentile for gestational age Based on recent Norwegian birth percentiles [4] OPD
Gestational age (at birth) (days)	Relative to estimated date of delivery (EDD). Based on ultrasound assessment at 17-19 weeks' gestation or last menstrual period if ultrasound assessment was unavailable. OPD
Premature birth	Birth at < 37 weeks of gestation OHGS

Caesarean section	Elective and acute. Yes/no. OPD
Twins	Yes/no. OHGS
Breastfed	Yes/no. OHGS
Exclusive breastfeeding	Months of exclusive breastfeeding dichotomized: 0-4 vs > 4 months OHGS
Mothers' age at birth (years)	Age in years (two decimals) at time of delivery. OHGS
Mother < 20 years at birth	Yes/no. OHGS
Mother's height (cm)	Height in full centimeters (no decimals). OHGS
Pre-pregnancy BMI (kg/m^2)	Pre-pregnancy BMI based on recall at 15 weeks gestation or earlier (usually registered in pregnancy records at first pregnancy care visit) and entered in the perinatal database at the time of ultrasound screening at approximately 17 weeks´ gestation OPD
Gestational weight gain (kg)	Weight gain: weight recorded at delivery minus pre-pregnancy weight (see above). OPD
Gestational weight gain above recommended	Defined according to the guidelines from the Institute of Medicine and National Research Council (USA) from 2009, depend on pre-pregnancy BMI category[5] 1 = Appropriate or insufficient weight gain 2 = Excessive weight gain OPD
Smoking at week 18 in pregnancy	Recorded at approximately 17 weeks' gestation No/yes (occasional or daily). OPD
Preeclampsia during pregnancy	Yes/no. Diastolic blood pressure > 90 mmHg and proteinuria. OPD
Diabetes during pregnancy	Including both gestational diabetes and type I and II diabetes, plus unspecified. Current definition in Norway for gestational diabetes during study period: fasting blood glucose > 6.1 mmol/l, or 2-hour-glucose-level > 7.8 mmol/l on an oral glucose tolerance test. OPD

Children's early childhood and current health and habits.	
Current child height SDS	Height was measured by public health nurses at pre-school health examinations, and recorded to the nearest 0.1 cm SDS scores were based on current Norwegian growth references [3] OHGS
Current child weight SDS	Weight was measured by public health nurses at pre-school health examinations, while wearing light clothes, and recorded to the nearest 0.1 kg SDS scores were based on current Norwegian growth references [3] OHGS
Current child BMI SDS	BMI was calculated from current height and weight (weight/height^2) SDS scores were based on current Norwegian growth references [3] OHGS
Chronic malformation or disease	Diagnosis reported by the parents, manually checked by the authors. The major diagnoses were diabetes, heart disease and celiac disease. Asthma or allergy was not included. OHGS
Asthma medication	Medication for asthma after 2 years of age: Corticosteroids, medication for asthma attacks or other maintenance medications, yes/no OHGS
Antibiotics ≥ 3 times	Number of times the child had been treated with antibiotics, irrespective of course Dichotomized: 1: 0-2 times (n = 852) 2: ≥ 3 times (n = 267) OHGS
Hospital admissions	Yes/no. Of any cause, from birth until preschool examination OHGS
Dental caries	Yes/no. OHGS
Combined physical activity score poorer than peers	Physical activity was rated by parents in three dimensions: Endurance 1 = like peers 2 = slightly less than peers

	3 = markedly less than peers Gross motor skills: 1 = better than peers 2 = like peers 3 = poorer than peers Level of activity in sports and play: 1 = more active than peers 2 = like peers 3 = less active than peers A combined activity score was computed from the three dimensions (minimum 3-maximum 9), increasing score reflected lower activity Dichotomized: 1 = score < 6 (n = 1048) 2 = score ≥ 6 (n = 56) OHGS
Physical activity (frequency)	Reported as frequency of being active enough to experience heavy breathing or sweating 1 = never 2 = <1 time/month 3 = 1-3 times/month 4 = 1 time/week 5 = 2-3 times/week 6 = 4-6 times/week 7 = daily Dichotomized as: <2 times/week (n=170) or ≥2 times/week (n=879) OHGS
Physical activity per week	Reported as number of hours being active enough to experience heavy breathing or sweating per week 1 = none 2 = ½ hour 3 = 1 hour 4 = 2-3 hours 5 = 4-6 hours 6 = ≥ 7 hours OHGS
Screen time per day (hours)	TV, videogames etc. 1 = None 2 = < ½ hour 3 = ½ - 1 hour 4 = 2-3 hours 5 = 3-4 hours

	6 = > 4 hours Dichotomized as: < 2 hours (n=752) or ≥ 2 hours (n=344) OHGS
Kindergarten since 2 years of age	Yes/no OHGS
TV in the child's bedroom	Yes/no OHGS
Frequency of breakfast and consumption of different food/drink items (with cut-offs)	Breakfast was recorded as never or number of days/week Food items were recorded as frequency of consumption per day/week: 1 = never 2 = < 1 time/week 3 = 1 time/week 4 = 2-4 times/week 5 = 5-6 times/week 6 = daily 7 = several times a day -Breakfast < 4 (n=30) or ≥ 4 per week (n = 1086) Food/drink item groups (per week): -Vegetables, <5 (n=554) or ≥ 5 (n=546) -Fruit, <5 (n=213) or ≥ 5 (n=894) -Sugar sweetened beverages, ≤ 1 (n=764) or > 1 (n=343) -Sweets/snacks, ≤ 1 (n=635) or > 1(n=474) -Fast food, ≤ 1(n=1015) or > 1 (n=93) OHGS
Sociodemographic factors and health characteristics of parents and siblings	
Living in a municipality with >20 000 inhabitants	According to data obtained from Statistics Norway and postal codes Yes/no OHGS
Maternal education above high school	≤ 12 years of school vs. > 12 years of school OHGS
Paternal education above high school	≤ 12 years of school vs. > 12 years of school OHGS
Mother working	Not working vs. full/part-time working or student OHGS
Father working	Not working vs. full/part-time working or student OHGS

Parents from non-Western countries	Parents of Western origin (Europe, North America) vs. one or both parent of non-Western origin OHGS
Living with one caretaker	Living with two caretakers (mother and father, mother/father with new partner or two foster parents) vs. living with one caretaker OHGS
Siblings	Number of siblings No siblings vs. ≥ 1 siblings OHGS
Mother smoking	Yes/no OHGS
Current BMI mother (kg/m^2)	Calculated from weight and height values reported in the questionnaire OHGS
Current BMI father (kg/m^2)	Calculated from weight and height values reported in the questionnaire OHGS
BMI SDS average for siblings	Based on current Norwegian growth references [3] Calculated from weight and height values reported by the parents in the questionnaire OHGS
BMI category of mother	Calculated from weight and height values reported by the parents in the questionnaire BMI categories according to WHO definitions: Underweight(UW): < 18.50 Normal weight (NW): 18.50-24.99 Overweight (OW): 25.00-29.99 Obesity (OB): > 30.00 OHGS
BMI category of father	Calculated from weight and height values reported by the parents in the questionnaire BMI categories according to WHO definitions: Underweight(UW): < 18.50 Normal weight (NW): 18.50-24.99 Overweight (OW): 25.00-29.99 Obesity (OB): > 30.00 OHGS

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Supplementary table 2. Association between weight groups of the children at five years of age and early life predictors, early childhood and current health and habits of the children, and current sociodemographic and health characteristics of parents. These predictors are not significantly associated with the weight groups, and therefore not included in table 2a-c.

Characteristics	Valid n	All (n = 1088) %	Underweight (n = 85) %	Normal weight (n = 850) %	Overweight (n = 115) %	Obese (n = 38) %	p [a)]
Twins, %	1088	3.1	3.5	3.1	4.3	0.0	0.60
Chronic malformation or disease[b)]	1088	2.1	1.2	2.2	0.9	5.3	0.38
Hospital admissions	1088	33.5	35.3	32.5	39.3	34.2	0.53
Breastfed (yes/no)	1084	90.1	89.3	91.0	86.1	83.8	0.20
Physical activity that makes the child sweat <2 times/week	1049	16.2	15.3	17.0	10.1	18.9	0.31
Screen time > 2 hours daily	1065	31.6	31.8	31.2	35.7	29.7	0.80
Mother < 20 years at birth	1088	1.7	1.2	1.8	1.7	2.6	0.95
Preeclampsia during pregnancy	749	5.7	5.3	5.9	5.3	3.6	0.95
Diabetes during pregnancy[c)]	749	1.3	0.0	1.2	4.0	0.0	0.15
Gestational age (days)	746	278.9 (13.59) [d)]	276.2 (16.8) [d)]	279.1 (13.2) [d)]	277.7 (13.9) [d)]	282.7 (11.9) [d)]	0.16
Caesarian section	749	16.0	14.0	15.6	24.0	7.1	0.14

a) Analyses across weight groups, ANOVA for continuous variables, chi square for categorical; b) Major malformations or diseases, e.g. diabetes, heart disease, celiac disease; c) Both gestational diabetes and type I and II diabetes; d) mean(standard deviation)

Supplementary table 3. Effects of 1 SD increases in BW SDS and maternal BMI on childhood overweight and obesity in strata by maternal BMI and BW SDS, respectively, estimated from the multiple multinomial regression model [1]

Stratifying variable	Stratum	BW SDS (continuous) Overweight (n = 73) OR	Obesity (n = 25) OR	95%CI	P
Maternal BMI[2]	Normal weight (n = 480)	**3.31**	1.10	**(1.82, 6.04)** (0.47, 2.71)	**< 0.01** 0.84
	Overweight (n = 195)	**3.28**	2.09	**(1.41, 7.63)** (0.70, 6.21)	**< 0.01** 0.19
	Obese (n = 69)	1.21	2.16	(0.38, 3.84) (0.67, 7.02)	0.74 0.20
Maternal BMI (continuous)					
Birth weight SDS[3]	Low (n = 168)	1.18	1.03	(0.98, 1.41) (0.75, 1.42)	0.08 0.89
	Medium (n = 485)	**1.26**	**1.23**	**(1.09, 1.45)** **(1.04, 1.45)**	**< 0.01** **0.02**
	High (n = 106)	0.97	1.28	(0.74, 1.28) (0.93, 1.76)	0.85 0.12

Abbreviations: BW SDS: birth weight standardization score; BMI: body mass index; OR: odds ratio; CI: confidence interval

[1]Adjusted for the other variables in Table 3

[2]Body mass index (kg/m^2): Normal weight: BMI 18.5-24.9, Overweight: BMI 25-29.9, Obese: BMI ≥ 30.

[3]Standard deviation score: Low: <1 SDS, Medium: -1 to +1 SDS, High: > 1 SDS.

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REGULAR ARTICLE

ACTA PÆDIATRICA NURTURING THE CHILD WILEY

A family-oriented intervention programme to curtail obesity from five years of age had no effect over no intervention

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Abstract

Aim: To examine the effect of a family-oriented multidisciplinary intervention programme to curtail weight increase in young children with obesity.

Methods: Children who weighed more than one kilogram above the 97th percentile for height at the preschool assessment in Oppland County, Norway, were identified. Parents residing in one part of the county were invited to participate in a group-based three-year intervention programme while the rest had no interventions. Body mass index (BMI) and family characteristics at entry and measurements at birth were explanatory variables, and change in BMI standard deviation score (SDS) the outcome measure. For the intervention group, outcome was also related to skinfold thicknesses, waist-to-height ratio and physical ability.

Results: The programme was completed by 31 families in the intervention and 33 in the control group. At entry, the respective median (interquartile) age was 5.83 (0.36) and 5.74 (0.66) years, and the BMI SDS 2.35 (1.06) and 1.95 (0.49), P = .012. The median decrease in BMI SDS was 0.19 in both groups. The decline increased with increasing BMI SDS at entry, but irrespective of group. Social or behavioural factor or other anthropometric measures were not associated with outcome.

Conclusion: The intervention programme had no effect on BMI SDS.

KEYWORDS

body mass index, child, intervention, obesity, standard deviation score

1 | INTRODUCTION

The prevalence of overweight and obesity among children has increased throughout the world, and the World Health Organization (WHO) estimates that 41 million children under five years of age are overweight or obese.[1] In Norway,[2] as in some other European countries,[3] the prevalence of overweight and obesity among children may have stabilized over the last 10-20 years. Despite this development, 16% of eight-year-old children,[2] and 13%-17% of children aged 2-19 years[4] in Norway were overweight or obese in studies published during the last decade.

Children with obesity, and in particular adolescents, are at extremely high risk of being affected by obesity as adults,[5] and intervention studies to treat overweight and obesity in childhood have generally had limited or no success.[6-9] Furthermore, studies with some success have usually been evaluated after

Abbreviations: Δ BMI, delta BMI; BMI, body mass index; IQR, interquartile range; SDS, standard deviation score; WHO, World Health Organization.

short follow-up periods and may therefore have limited clinical significance since the risk of relapse may be high.[6-9] However, the majority of studies have addressed children in mid or late childhood, and the chance of success may decrease with increasing age.[10]

Our hypothesis was that intervention to curtail obesity is more effective when addressing young children when parents may have a greater impact on their child's behaviour. In a meta-analysis of children younger than 11 years with obesity, the mean age at entry was less than seven years in only six of the studies, and the interventions tended to be limited in terms of approach and involved personnel.[6] Furthermore, the intervention lasted between 3 and 6 months in 18 of 20 studies, and effects were assessed shortly thereafter. Therefore, our aim was to compare the effect of a three-year, group-based multidisciplinary intervention programme with no intervention in children aged five to six years. The programme only involved the parents, and the purpose was to alter the lifestyle of the family and child. The parents' perceived challenges as the intervention progressed were important in adjusting the programme to their specific needs. Our secondary aim was to identify potential success factors within the intervention group.

Key Notes

- Intervention programmes to treat obesity during childhood and adolescence have had limited success, but few studies have involved families of young children.
- This multidisciplinary and group-based programme which addressed parents of five-year-old children with severe overweight or obesity and lasted 2-3 years had no effect over no intervention on the median BMI standard deviation score.
- Anthropometric measures at entry, social or behavioural factors or attendance were not associated with outcome.

2 | METHODS

2.1 | Study population

We asked the public health nurses in Oppland County, Norway, to invite the parents of all the children who met for the school entry

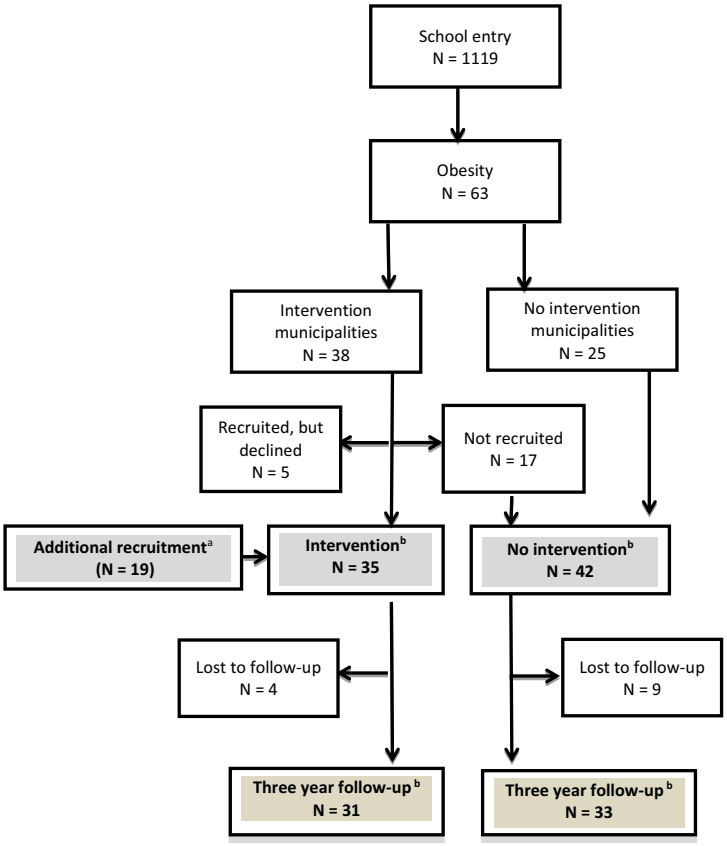

FIGURE 1 Recruitment of families of 5- to 6-year-old children with severe overweight or obesity for intervention or no intervention. [a]Included at the request of parents, [b]measurements at public healthcare clinics

health assessment in 2007, to participate in a longitudinal cohort study on health and growth (Figure 1). Virtually all children attend this examination together with at least one of the parents. Of 1895 children who met for the assessment, the parents of 1119 gave written consent to participate. The parents completed questionnaires on health and habits for the children, and on demographic, socioeconomic, health and lifestyle characteristics of the family.[11] The public health nurses measured the child's weight and height and reported these measures together with the recorded weight and length at birth. For children of families who declined to participate, the public health nurse anonymously reported sex, age, height and weight at the time of recruitment, and we have previously reported that the participants were probably representative of the population.[11]

From the cohort, children who weighed at least one kilogram above the 97th percentile for height were identified as eligible for the study. Body mass index (BMI) charts were not available at the public health clinics, but this measure was close to the definition of obesity according to the International Obesity Task Force definition of obesity, although some of the children had a BMI slightly below this limit.[12]

Oppland is one of 20 counties in Norway. It covers 25 192 km^2, has 26 municipalities and had a population of approximately 183 000 in 2007. On behalf of the research group, the public health nurses in the six municipalities that were geographically closest to the two hospitals in the county were asked to invite the families of eligible children to participate in the intervention programme. These municipalities had approximately 60% of the population in the county. They are mainly rural, but contain the only two cities in the county, each with 25 000-30 000 inhabitants. The other municipalities are rural with towns of variable sizes. The families from the other municipalities and families who were not referred from the intervention municipalities served as controls.

Some families of children with obesity in the intervention municipalities, who were not in the originally recruited group, became aware of the project and asked to be allowed to participate. These children were close in age and were included since a larger group allowed for a more accurate estimate of potential effects of the programme.

2.2 | Intervention and control programme

The intervention programme was organised in cooperation with the Learning and Mastery Service at the hospitals. This service is established as part of the specialist health services in Norway, and the purpose is to promote health through group-based patient education programmes aimed at promoting self-management for people living with chronic health challenges.[13] The programme is led by nurses who are trained in providing guidance. Other relevant personnel participate according to specific needs. In this project, only the parents attended the group sessions. In addition to nurses, one or more of the following professions contributed at each session: paediatricians, nutritionists, physiotherapists and a psychologist. The various professionals participated according to a predetermined schedule early in the programme, but some variation evolved as needs was identified by the groups. The professionals gave practical advice regarding diet and physical activity, but in particular, they encouraged and participated in discussions on experienced challenges in changing lifestyles and on how to deal with them in terms of changing behaviour.

Each group consisted of 5-7 pairs of parents, and each session was scheduled to last 2.5 hours after working hours. The children were occupied in play under the supervision of a preschool teacher while the parents participated in the sessions. The intervention programme was planned to last three years. At the Learning and Mastery Service, the groups were scheduled to meet four times during the first year, twice during the second and one time during the third year. Between each of these sessions, each family (parents and the child) was invited once for discussions and assessments by the study nurses.

The no-intervention control children and their families received no information about the intervention programme and had no scheduled appointments with healthcare services during the three years of the study.

2.3 | Measurements

The weight and length at birth were measured by midwives and reported to the public healthcare clinics. Comparisons between the intervention and control group were based on routine measurements of height and weight around school entry and in third grade. Public health nurses performed these measurements according to national guidelines. The children were wearing light underclothes. Height was measured to the nearest millimetre and weight to the nearest 100 g.[14]

In the intervention programme, the children were assessed by two specifically trained study nurses at entry and after each of the three years. The measurements included height, weight, triceps and subscapular skinfold thicknesses, abdominal circumference and maximum walking distance on a 6-minute walk test.[15] Waist circumference was measured to the nearest millimetre, and waist-to-height ratio was calculated as the waist circumference divided by the simultaneously measured height. The skinfolds were measured with a Holtain Tanner/Whitehouse skinfold calliper (Crosswell, Pembrokeshire, UK) and in a way that was identical to how Norwegian references were established.[16,17] On the 6-minute walk test, the nurses recorded the length in metres as the children were able to walk during 6 minutes on a 50 metre lane.

BMI was calculated as weight/height^2 (kg/m^2). The standard deviation score (SDS) for the BMI, waist circumference, waist-to-height ratio and skinfolds of the children were based on current Norwegian growth references.[17-19] The parents' heights and weights were self-reported.

2.4 | Explanatory and outcome measures

The BMI SDS at inclusion was the primary explanatory variable and the change in BMI SDS from entry to the end of the programme the primary outcome measure. In adjusted analyses, we included birth weight, child and family health, lifestyles and other characteristics reported at the study entry as possible confounders. Several of the descriptive ordinal variables were dichotomized in order to do meaningful comparisons.[11] Hospital admissions were admissions for any cause from birth until the preschool assessment. Physical activity per week was reported as frequency of being active enough to experience heavy breathing or sweating.[11] Place of residence was categorised as urban if they lived in one of the two cities. Asthma medication included medication for asthma attacks, inhaled corticosteroids and other maintenance medications.

2.5 | Statistical methods

Descriptive statistics are presented as percentages for categorical variables and as medians with interquartile range (IQR) for continuous variables. Correlations are reported as the Pearson's correlation coefficient (*r*). We compared the intervention and control group with Mann-Whitney's *U* and Chi-square tests and performed a multiple linear regression analysis across both groups to test whether being in the intervention or the control group had a significant impact on the change in BMI SDS when adjusting for the registered exposures.

The power estimate was based on a Scandinavian study of 10- to 11-year-old children with obesity where children in a family treatment group achieved a mean BMI benefit of 1.7 kg/m^2 after one year compared with a control group with no intervention.[20] The eligible children in our study had a mean (standard deviation) BMI of 20.40 (1.92) kg/m^2. Although our children were younger and therefore had lower BMIs, an effect of 1.7 kg/m^2 was considered possible since our study was designed to last for three years. With this premise, we calculated that 22 children had to be included in each group to detect such a difference with the statistical significance level of 5% and a power of 80%. However, since the study did not have a true randomised design and the intervention and controls groups varied on some variables, a larger number was desirable. We used intention to treat, in that all who had attended at least one of the sessions were included in the analyses.

Within the intervention group, we used the related-samples Wilcoxon signed-rank test to compare measures at entry and at the end. In order to assess which factors were associated with success, we performed simple and multiple linear regression analyses with change in BMI SDS as the outcome measure. In this model, we included skinfold thickness as the mean of the sum of the triceps SDS and subscapular SDS measurements, and the waist-to-height ratio SDS. Our hypothesis was that relatively high values for a given BMI may suggest a higher fat deposit and therefore true obesity, while relatively low values may suggest a relatively high lean body mass. We used the number of attendances as proxy for motivation to change lifestyle. The regression analysis was performed in an all-in backward model, and the potential explanatory variables were selected from earlier literature and the strength of association in the post-hoc bivariate analyses. Results are reported as estimated regression coefficients (b), *P* values and determination coefficient (R^2).

P values ≤ .05 were considered statistically significant. The SPSS Statistics for Windows, Version 23.0 (IBM Corp., NY, USA) was used for all analyses. The BMI SDS, skinfolds SDS, waist circumference and waist-to-height ratio were calculated in R.2.6.0 (The R Foundation for Statistical Computing, Vienna, Austria) using Norwegian growth references.[16-19]

2.6 | Ethical considerations

The study was approved by the Regional Committee on Medical Research Ethics (REK 1.2006.3491) and the Norwegian Data Protection Official for Research (02-2006 SI). One of the patents gave written consent. The study was registered at ClinicalTrials.gov (NCT00458224) before recruitment.

3 | RESULTS

3.1 | Comparing the intervention and control group

Figure 1 describes the recruitment of subjects. Of 63 originally eligible children, 38 lived in the municipalities of recruitment for intervention. The 25 families from the other municipalities and 17 families from the recruitment area who were not referred for intervention served as controls. An additional 19 children of similar age from the intervention municipalities were included at the request of the parents. Data from both the entry and the end of the programme were available for 31 children in the intervention and 33 in the control group. In the intervention group, three children were born in 1999, 11 in 2000, 12 in 2001 and five in 2002. In the control group, one was born in 2000 and 32 in 2001. The measurements at entry of the children who were lost to follow-up (Figure 1) did not differ from those who completed the comparison study (data not shown).

The children in the intervention group had a higher median weight, BMI and BMI SDS at entry and a higher BMI SDS at the end of the intervention than the control group, but the median increase in BMI (Δ BMI 2.02 vs 1.95 kg/m^2) and decline in BMI SDS (Δ BMI SDS 0.19 in both groups) did not differ (*P* = .731, Table 1). The fathers in the intervention group had a somewhat higher median BMI, and a higher proportion of the parents were of the opinion that their child looked overweight. Due to the study design, a higher proportion of the families in the intervention group lived in the two cities. The median time interval between the measurements tended to be shorter for the intervention than the no-intervention group (2.05, IQR 1.23 vs 2.59, IQR 1.65) years, *P* = .119. There were no other significant differences between the groups (Table 1). The Δ BMI and Δ BMI SDS did not differ between the controls recruited from the

TABLE 1 Characteristics of the children with severe overweight or obesity and their families, and change in anthropometric measurements following intervention or no intervention in Oppland county, Norway

Child characteristics	Intervention (n = 31)	No intervention (n = 33)	
Continuous variables	**Median (IQR)[a]**	**Median (IQR)[a]**	***P*-value[b]**
Birth weight, kg	3.62 (0.80)	3.72 (0.78)	.466
BMI SDS at birth[c]	−0.11 (1.43)	0.07 (1.35)	.287
Age at entry, years	5.83 (0.36)	5.74 (0.66)	.979
Age at the end, years	7.84 (1.20)	8.42 (1.65)	.066
Height at entry, cm	119.00 (6.00)	118.00 (8.00)	.261
Height at the end, cm	133.20 (10.00)	135.00 (16.00)	.476
Weight at entry, kg	30.00 (7.40)	27.60 (3.00)	.012
Weight at the end, kg	40.30 (14.90)	39.00 (14.90)	.481
BMI at entry[d], $kg/m^{2,}$	20.83 (3.21)	19.32 (1.26)	.005
BMI at the end, kg/m^2	22.94 (5.24)	21.89 (4.37)	.078
Δ BMI[e], $kg/m^{2,}$	2.02 (3.32)	1.95 (3.76)	.825
BMI SDS at entry	2.35(1.06)	1.95 (0.49)	.012
BMI SDS at the end	2.25 (0.90)	1.86 (0.64)	.018
Δ BMI SDS	−0.19 (0.73)	−0.19 (0.76)	.731
Binary variables	**Prevalence (%)**	**Prevalence (%)**	***P*-value[f]**
Sex, girls	54.8	57.6	.825
Hospital admissions	50.0	18.2	.007
Screen time > 2 hours per day	33.3	46.9	.277
Physical activity > 2 times per week	70.0	93.5	.017
TV in the child's bedroom	36.7	25.0	.319
Asthma medication after 2 years age	13.3	28.1	.153
Kindergarten since 2 years of age	92.3	81.3	.225
Prematurity,	3.3	3.0	.945
Sleep problems after 2 years of age	10.0	6.1	.563
Breastfeeding > 4 months	61.1	63.2	.898
Dental caries	23.3	27.3	.720
Familycharacteristics	**Intervention (n = 31)**	**No intervention (n = 33)**	
Continuous variables	**Median (IQR)**	**Median (IQR)**	***P*-value[b]**
BMI mother, kg/m^2	27.39 (6.64)	26.07 (8.01)	.317
BMI father, kg/m^2	30.09 (6.30)	27.66 (5.80)	.039
Number of siblings	1.00 (2.00)	1.00 (1.00)	.513
Binary variables	**Prevalence (%)**	**Prevalence (%)**	***P*-value[f]**
Maternal education above high school	37.9	30.3	.527
Urban living[g]	45.2	18.2	.020
Smoking by family member	56.7	51.5	.682
Parents think child looks overweight	89.3	33.3	<.0005
Living with single parent	36.7	24.2	.283

[a]Interquartile range.
[b]Mann-Whitney *U* test.
[c]Body mass index, standard deviation score.
[d]Body mass index.
[e]Body mass index at the end minus at the entry of the study.
[f]Chi-square test.
[g]Living in one of the two cities.

TABLE 2 Development of anthropometric measurements and 6-minute walk test in the intervention group during the three-year multidisciplinary intervention programme

Variables (medians and IQR)[a]	Entry (n = 29)	3 years (n = 29)	P[b]
Age, years	6.58 (1.49)	9.61 (1.83)	<.005
Height, cm	125.00 (11.35)	141.20 (8.25)	<.005
Weight, kg	35.50 (8.40)	50.70 (15.80)	<.005
BMI, kg/m^2[,c]	21.76 (4.06)	24.54 (6.89)	<.005
BMI SDS[d]	2.36 (0.93)	2.06 (1.06)	.008
Subscapular skinfold SDS	2.20 (1.10)	1.87 (0.67)	.016
Triceps skinfold SDS	2.23(1.28)	2.20 (0.82)	.272
Median mean skinfold SDS	2.08 (1.22)	2.11 (0.77)	.010
Waist circumference SDS	2.51 (1.08)	2.57 (0.55)	.079
Waist-to-height ratio SDS	2.30 (0.94)	2.64 (0.87)	.498
6-minute walking test, metres[1]	540.00 (109.00)	705.00 (129.00)	.001

[a]Interquartile range.
[b]Related-Samples Wilcoxon signed-rank test.
[c]Body mass index.
[d]Standard deviation score.

intervention municipalities and the no-intervention municipalities (data not shown).

In the multiple linear regression analysis of the whole cohort, we included the BMI SDS at birth and at entry to the study, the BMI of parents, relevant measures of health and lifestyles, the demographic variables in Table 1, and the categories intervention vs no-intervention group as exposures, and Δ BMI SDS as outcome. A higher BMI SDS at entry was associated with a larger decrease in BMI SDS (b = −0.376, P = .002, R^2 = 0.154), but independent of being in the intervention or no-intervention group. Other variables were of no significance.

3.2 | The intervention group

All the measurements at entry and at the end of the programme were available for 29 of the children in the intervention group. The median number of attendances was eight (range 1 to 16); three group attendances at the Learning and Mastery Service (range 0 to 7) and five (range 1 to 9) meetings for individual nurse guidance and measurements. Eleven of the 29 families attended all the planned sessions at the Learning and Mastery Service and the nurse guidance meetings.

The median BMI SDS, but not the median of the mean of the skinfold thickness SDS or waist-to-height ratio SDS, was significantly lower after three years of intervention than at entry (Table 2). The BMI SDS was closely related to the median of the mean skinfold SDS (r = 0.864 at entry and r = 0.825 at the end, P < .005 for both) and to the waist-to-height ratio (r = 0.833 and r = 0.785, P < .005 for both). The results for the 11 children of families who attended all the sessions did not differ from those of the rest of the group (data not shown). In the multiple linear regression analysis, a higher BMI SDS at entry was associated with a larger reduction in BMI SDS, but none of the other exposures were associated with a change in BMI SDS (Table 3).

The parents' weight at entry and the end of the programme was known for 21 mothers and 15 fathers. Their median weight did not change (median difference 0.00 kg).

4 | DISCUSSION

In this study of children aged 5-6 years with severe overweight or obesity, a multidisciplinary educational intervention programme with the intention to change family and child lifestyles had no effect over no intervention on the development of BMI. Both the intervention and the no-intervention group experienced the same moderate reduction in BMI SDS, and potential confounders had no significant effects on outcome, neither when comparing the two groups nor within the intervention group. In particular, adherence to the intervention programme and skinfold thickness at entry were not associated with change in BMI SDS, suggesting that motivation on part of the parents and relative fat mass did not affect outcome.

Cochrane reviews of randomized controlled trials suggest that there were no convincing evidence of significant and persistent weight-reducing effects from published studies involving interventions on diet, physical activity or other behaviour in children with a mean age 10 years[7] or adolescents [8] with obesity. In the study of Mead et al, the overall benefit in favour of interventions over usual care was only a BMI SDS score of 0.06, (95% CI 0.10 to 0.02) units at 6-36 months of follow-up. Furthermore, in 13 of the 27 included studies, the SDS score declined as much in the control as in the intervention group.[7] In another Cochrane review, parent-only interventions were as effective as parent-and-child interventions in 5- to 11-year-old children, but minimally more

TABLE 3 Results from linear regression analyses of differences in body mass index standard deviation score (BMI SDS difference) from entry to the end of the intervention programme after three years in 29 children with obesity aged 5-6 years at entry

	Unadjusted		Adjusted (R^2 = 0.298)[b]	
Exposure	Beta[c]	*P*	Beta[c]	*P*
Birth weight	0.199	.351		
Age at entry	0.183	.116	0.227	.041
BMI SDS at entry	−0.451	**.011**		
Mean skinfold SDS at entry	−0.310	.069	−0.340	.031
Waist-to-height ratio SDS at entry	−1.572	.392		
Walking test	0.001	.225		
Number of attendances	0.018	.467		
Adherence in the intervention[d]	0.312	.179		
Sex (girls)	0.502	**.029**		
Hospital admissions	0.159	.502		
Screen time >2 h daily	0.508	**.035**		
Physical activity >2 times/week	−0.294	.273		
TV in the child's bedroom	−0.121	.631		
Asthma medication (>2 y age)	−0.510	.172		
Kindergarten since 2 years old	−0,009	.986		
Prematurity (<37 weeks GA)	−0.387	.536		
Sleep problems >2 years of age	−0.530	.155		
Breastfeeding >4 months	−0.018	.918		
Dental caries	−0.443	.141		
BMI mother	0.017	.484		
BMI father	−0.015	.651		
Number of siblings	0.024	.843		
Maternal education above high school	0.142	.528		
Urban area	−0.109	.636		
Smoking by family member	−0.124	.595		
Parents think child look fat	0.376	.339		
Living with single parent	−0.080	.750		

[a]Bold indicates the significant *P*-values in the unadjusted analysis.

[b]After backward stepwise exclusion of variables.

[c]A positive beta means increased risk of a positive BMI SDS, that is an increase in BMI SDS from start to 3 years.

[d]Attended all the sessions (n = 11) vrs. less (n=18).

effective than for waiting list controls.[6] In a meta-analysis of few studies on preschool children, an intervention on diet, physical activity and behaviour had a significant, but slight, beneficial effect at 12-18 months of follow-up.[21] However, the quality of evidence was estimated as low or very low, particularly for the youngest groups. The median decline in BMI SDS in our intervention group of 0.19 was similar to the mean decline of 0.20 in the systematic review by O'Connor et al on weight-reducing trials among children and adolescents.[8] Our results were also similar to the mean decline of 0.22 in a Swedish study on children aged 8-12 years with obesity who attended different treatment programmes.[22] The Swedish study had no controls without interventions, and it is remarkable that our non-intervention control group had the same decline, suggesting that the intervention had no effect over the general public attention on childhood obesity in Norway.

Within our intervention group, the children with the highest BMI SDS score had the largest decline in the SDS score. However, this was equally true for our non-intervention group and was probably not a specific effect of the intervention programme. It is noticeable that presumed risk factors were not associated with failure to decrease the BMI SDS. For instance, limited attendance to the programme, which may imply lack of motivation, dental caries, which may be suggestive of unhealthy dietary and other behaviour, lower parental education, and single parenthood were not associated with outcome. One explanation why extent of physical activity was not associated with outcome may be that physical activity at this young age is mainly related to play. It is likely that the children had similar activity in play since almost all of them were in day care from at least two years of age and thereafter in school where play is an important activity during the first years.

As in most studies, we used changes in BMI SDS as the primary outcome. By using standard deviation scores, the effect of minor differences in age and time interval between measurements was limited. However, it has recently been argued that a decrease in BMI SDS with age in children may not necessarily mean a decrease in the degree of obesity because the SDS of BMI may not accurately correct for age, sex and degree of obesity.[23] Therefore, a reduction in BMI SDS of around 0.20, as obtained in the present and most other studies on children, may not necessarily mean a decrease in degree of obesity.[23]

The referred Cochrane studies conclude that studies to prevent or treat obesity in children are generally of low quality. Likewise, our study has several limitations. The number of participants was limited. However, it is unlikely that a larger study would have shown an effect since the decline in both the median and variation in BMI SDS were almost identical in the intervention and the no-intervention group. The two groups differed somewhat in several aspects, partly due to the recruitment process and partly due to different routines between municipalities related to time of measurements around school entry and in third grade. Unpredictable effects of these differences were reduced by adhering to standard deviations scores in the analyses. It may be argued that the higher median BMI at entry of the intervention group may have masked an

effect. However, the significance of BMI at entry was the same in the two groups. Furthermore, the families who participated in the intervention programme were probably more motivated for treatment than many families in the no-intervention group since most of them, as opposed to the no-intervention group, expressed that their children look overweight. In particular, the families who participated on request expressed a concern for their children's health. It is therefore likely that potential effects of confounding would be in favour of the intervention group. Randomized controlled trials are considered the gold standard when studying effects of interventions, but we chose to include families on basis of geographical closeness to the hospitals for two reasons: the county is large, and the distance to the other municipalities would make it difficult for families to attend. Furthermore, a true randomization could possibly have introduced an unrecognized intervention effect from spill-over within the municipalities. Such concern has been raised in randomized intervention studies where one arm is generally accepted as preferable.[24,25] Despite this concern, we chose to include eligible children in the intervention municipalities who were not referred as controls to account for all eligible children in the county. In these municipalities, a spill-over effect was also unlikely since the intervention programme was conducted outside the municipalities and no information about the programme was publicised during study period. This assumption was strengthened by the finding that their development in measurements did not differ from the rest of the no-intervention controls. The analyses of associations between outcome and factors that were considered potential predictors of success or failure of the programme must be interpreted with caution due to lack of power.

5 | CONCLUSION

Our multidisciplinary and relatively long-term approach adds to studies that have failed to significantly decrease severe overweight and obesity in children. The similar reduction in BMI SDS in our intervention and no-intervention groups may suggest that a high national focus on overweight and obesity in children, including societal facilitations to encourage protective lifestyles, is the most important approach to curtail the obesity epidemic among children.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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