Diets of European children, with focus on BMI, well-being, and families

The IDEFICS/I.Family cohort

Louise Arvidsson

Department of Public Health and Community Medicine
Institute of Medicine
Sahlgrenska Academy at University of Gothenburg

UNIVERSITY OF GOTHENBURG

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louise.arvidsson@gu.se

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“It always seems impossible until it's done”
- Nelson Mandela

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Louise Arvidsson
Department of Public Health and Community Medicine, Institute of Medicine
Sahlgrenska Academy at University of Gothenburg
Göteborg, Sweden

Abstract

The overall aim of this thesis was to investigate children's diet, BMI z-score, and parental feeding practices (PFPs), in relation to mutans streptococci (MS) count, psychosocial well-being and children's BMI trajectory, as well as exploring the effect of the intervention in the IDEFICS (Identification and prevention of dietary- and lifestyle-induced health effects in children and infants) study on diets of families five years after the intervention.

About 16,000 children from eight European countries participated in the IDEFICS study in 2007/2008 (referred to as index children). During 2008 a community intervention was carried out targeting diet, physical activity and stress. In 2009, 68% of the children returned for a follow-up examination. In 2013, I.Family started and 6,055 of the children returned for a third follow-up, and at this time point 7,794 parents and 2,512 siblings also participated. Height and weight were measured, saliva was collected, and information on eating habits, feeding practices, well-being, and socioeconomic factors were reported at all time points.

High salivary MS count was found among 18% of the children in a sub-sample from the Swedish IDEFICS cohort. Higher BMI z-score, more frequent intake of meals and higher propensity for consuming sugar were all independently associated with higher MS count. In contrast, an inverse association was found between hours of sleep and MS count.

Bi-directional associations were identified between a healthy diet, measured by adherence to healthy dietary guidelines, and better self-esteem. Additionally, a healthy diet was associated with fewer emotional and peer problems two years later, with a monotonic trend entailing a consistent increase for all indicators of well-being associated with higher adherence. These associations were moderated by children's sex.

PFPs at IDEFICS baseline explained 22% of the variation in children's BMI z-score at I.Family. More specifically, PFPs involving restriction and considering putting the child on a diet were associated with higher odds of developing overweight independent of baseline BMI z-score and parental BMI. This association was stable across social vulnerability groups.

At I.Family, better diet quality (as measured by lower propensity for consuming fat and sugar, and higher propensity for consuming water, and fruit and vegetables) was reported by families in the intervention communities. However, investigation of the five-year change in fat, sugar, and water propensity ratio among index children failed to identify any differences between the intervention and control groups longitudinally.

This thesis documents the importance of healthy eating habits in reducing the risk of dental caries and maintaining good psychosocial well-being in children. Furthermore, restrictive PFPs are not helpful in promoting a healthy weight development. It is therefore important to identify other, more effective, PFPs and to include parents more directly in future intervention studies aiming at improving children's eating habits.

Keywords: body mass index, child, diet, feeding practices, intervention, mutans streptococci, parents, restriction, well-being

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Populärvetenskaplig sammanfattning

Det övergripande syftet med denna avhandling var att undersöka matvanor och viktstatus hos barn, samt föräldrars måltidspraxis, i relation till karies-orsakande bakterier, psykosocialt välbefinnande och viktutveckling, och därtill även utforska effekten av den intervention som ingick i IDEFICS-studien (Identifiering och förebyggande av kost- och livsstilsorsakade hälsoeffekter hos barn) på familjernas matvanor fem år efter interventionen.

Omkring 16,000 barn från åtta europeiska länder medverkade i IDEFICS-studien 2007/2008. Under 2008 genomfördes en samhällsintervention med fokus på att förbättra kostintaget, den fysiska aktiviteten och minska stress. Vid andra uppföljningen 2009 deltog 68% av barnen. År 2013 startade I.Family och 6,055 av barnen från IDEFICS kohorten kom tillbaka för en tredje uppföljning. Vid denna tidpunkt deltog även 7,794 föräldrar och 2,512 syskon. Längd och vikt mättes, saliv samlades in och information om matvanor, måltidspraxis, välbefinnande och socioekonomiska faktorer rapporterades vid alla undersökningstillfällen.

Högt koloniantal av mutans streptokocker hittades bland 18% av barnen i en subgrupp från den svenska IDEFICS-kohorten. BMI z-score, måltidsfrekvens och benägenhet för att konsumera socker var oberoende av varandra positivt associerade med högt koloniantal av MS. Dessutom var lång sömn negativt associerad med högt koloniantal av MS.

En dubbelriktad association identifierades mellan hälsosamma matvanor (definierat som högre följsamhet till generella kostrekommendationer) och högre självkänsla. Hälsosamma matvanor var dessutom associerat med färre emotionella problem och bättre kompisrelationer två år senare, och ju högre följsamheten till kostrekommendationerna var desto bättre var välbefinnandet två år senare. Associationen mellan hälsosamma matvanor och välbefinnande var dock olika för pojkar och flickor.

Måltidspraxis, som rapporterades vid starten av IDEFICS förklarade 22% av barnens BMI z-score 5-6 år senare. Att begränsa barnets matintag samt att överväga att banta sitt barn var associerat med högre odds för att barnet utvecklade en övervikt, oberoende av barnens tidigare BMI z-score, och föräldrarnas BMI. Denna association var lika stark både i mer- och mindre socialt utsatta grupper.

En tvärsnittsstudie inom I.Family antydde att familjer i interventionsområdena hade bättre matvanor (definierat som benägenhet att konsumera mindre fett och socker, men mer vatten, och frukt och grönsaker). Dock kunde ingen skillnad i förändring identifieras från IDEFICS till I.Family i benägenhet att konsumera fett, socker, och vatten mellan barn från interventions- och kontrollgrupperna.

Avhandlingen dokumenterar vikten av hälsosamma matvanor för att minska risken för karies och upprätthålla ett bra psykosocialt välbefinnande hos barn. Måltidspraxis som begränsar barnens matintag främjar inte en hälsosam viktutveckling. Det är därför viktigt att identifiera andra, mer gynnsamma metoder för föräldrar samt att inkludera föräldrar mer direkt i framtida interventioner som syftar till att förbättra barns matvanor.

List of papers

This thesis is based on the following studies, referred to in the text by their Roman numerals. The original publications are reprinted with the permission of the copyright holders.

I. Arvidsson, L., Birkhed, D., Hunsberger, M., Lanfer, A., Lissner, L., Mehlig, K., Mårild, S., Eiben, G. on behalf of the IDEFICS consortium.
BMI, eating habits and sleep in relation to salivary counts of mutans streptococci in children
– the IDEFICS Sweden study
Public Health Nutrition 2015;19: 1088-92

II. Arvidsson, L., Eiben, G., Hunsberger, M., De Bourdeaudhuij, I., Molnar, D., Jilani, H., Thumann, B., Veidebaum, T., Russo, P., Tornatitis, M., Santaliestra-Pasías, A. M., Pala, V., Lissner, L. on behalf of the IDEFICS consortium.
Bidirectional associations between psychosocial well-being and adherence to healthy dietary guidelines in European children: prospective findings from the IDEFICS study
(Revision submitted to BMC Public Health)

III. Arvidsson, L., Hunsberger, M., Siani, A., Szommer, A., Solea, A., Börnhorst, C., Williams, G., Pigeot, I., Iguacel, I., Bogl, L.-H, Moreno, L., Leu, M., Michels, N., Veidebaum, T., Pala, V., Ahrens, W., Lissner, L. on behalf of the IDEFICS and I.Family consortium.
Parental feeding practices and social vulnerabilities in relation to subsequent BMI trajectories of European children
– the IDEFICS/I.Family cohort
(Submitted)

IV. Arvidsson, L., Bogl, L.-H., Eiben, G., Hebestreit, A., Nagy, P., Tornaritis, M., Moreno, L. A., Siani, A., Veidebaum, T., De Henauw, S., Lissner, L. on behalf of the IDEFICS and I.Family consortia
Fat, sugar and water intakes among families from the IDEFICS intervention and control groups: first observations from I.Family
Obesity Reviews 2015;16 (Suppl. 2):127–137

Content

Abbreviations

BMI	Body mass index
BTG	BMI trajectory group
CFU	Colony-forming units
CI	Confidence interval
DMFT	Decayed, Missing and Filled Teeth
EST	Ecological systems theory
FFQ	Food Frequency Questionnaire
GLMM	Generalized linear mixed model
HDAS	Healthy Dietary Adherence Score
HRQoL	Health related quality of life
IDEFICS	Identification and prevention of Dietary- and lifestyle-induced health EFfects In Children and infantS
I.FAMILY	Determinants of eating behavior in European children, adolescents and their parents
IMP	Intervention mapping protocol
IOTF	International Obesity Task Force
IQR	Interquartile range
ISCED	International Standard Classification of Education
KINDL	Kinder Lebensqualität Fragebogen
MS	Mutans streptococci
OR	Odds ratio

PA	Physical activity
PFP	Parental feeding practices
RCT	Randomized controlled trial
SACINA	Self-Administered Children and Infant Nutrition Assessment
SD	Standard deviation
SDQ	Strengths and Difficulties Questionnaire
SE	Standard error
SEP	Socioeconomic position
WHO	World Health Organization
24-HDR	24-hour diet recall

Brief definitions

BMI	Body mass index (BMI) is calculated as weight (in kg) divided by squared height (in m)
BMI z-score	*BMI z-score is calculated based on a reference population [1] and represents the number of standard deviation units above or below the mean. It is age- and sex adjusted meaning that for any z-score level a specified BMI z-score represents the same difference in adult BMI units*
Normal weight	*Defined by the International Obesity Task Force (IOTF) [1] and corresponds to an adult BMI of 18.5 to 24.9 kg/m*^{2}.
Overweight	*Defined by the IOTF and corresponds to an adult BMI of 25 to 29.9 kg/m*^{2}. *In this thesis, the term 'overweight' includes obesity*
Obesity	*Defined by the IOTF and corresponds to an adult BMI of* $\geq$*30 kg/m*^{2}
Major weight gain	Children who started with a BMI z-score of more than -1 at IDEFICS baseline (T_0 or T_1) and who gained more than +0.1 in BMI z-score per year during the follow-up period
Major weight loss	Children who started off overweight with a BMI z-score of more than 1 at IDEFICS baseline and decreased more than -0.1 in BMI z-score per year during the during the follow-up period
Index child	Child participating in the original IDEFICS study

Parental education	Maximum education level of both parents. Based on the International Standard Classification of Education (ISCED 2011), a reference classification system for education, allowing for international comparisons. ISCED levels range from 0 to 8
Low education	*Defined as ISCED level ≤2 and represents lower secondary education or less*
Medium education	*Defined as ISCED level >2 and ≤4 and represents upper secondary and post-secondary education*
High education	*Defined as ISCED level ≥5 and represents tertiary education (includes short-cycle tertiary, bachelor's, master's and doctoral degree)*
Parental income	Highest level of income of both parents. Country-specific income levels were assigned with reference to the average net equivalence income, considering the median income and poverty line. Possible income levels ranged from 1 to 9.
Lower income	*Defined as income level <6 and represents income below median*
Higher income	*Defined as income level ≥6 and represents income above median*

1 Introduction

Prevalence and consequences of childhood obesity

There is an ongoing debate on whether the rapid increase in childhood obesity observed during the 1980s and 1990s has slowed down. Nonetheless, consensus is that the prevalence of children and adolescents with obesity remains at unacceptably high levels [2-5]. The prevalence of obesity was estimated to be 17% among 2-19-year-olds in the United States in 2011-2014 [6]. In Europe, the combined prevalence of childhood overweight and obesity ranges from 10 to 40%, with higher levels found in the southern European countries and in populations with lower socioeconomic position (SEP) [7-9]. Data from the World Health Organization (WHO) European Childhood Obesity Surveillance Initiative (COSI) showed that the prevalence of overweight (including obesity) in 6-9-year-old children in 2007-2008 was 19-47% in boys and 18-43% in girls, whereof the obesity represented 6-27% in boys and 5-17% in girls [7]. Comparable numbers are reported from 2007-2010: 18-57% among boys (6-31% obese) and 18-50% (5-21% obese) among girls [8]. Somewhat lower prevalence was observed among European children below the age of 10 participating in the Identification and prevention of dietary- and lifestyle-induced health effects in children and infants (IDEFICS) study [9]. However, in contrast to previous findings they reported a higher combined prevalence of overweight in girls (11-44%, of which the obesity prevalence was 3-20%) as compared to boys (8-41%, of which the obesity prevalence was 2-20%) [9].

Compared to healthy-weight peers, children and adolescents with obesity are at higher risk of physical and psychological co-morbidities, such as cardio-metabolic morbidity, type 2 diabetes, depression, emotional and behavioral disorders, as well as of lower health-related quality of life (HRQoL) [10-13]. Obesity is also associated with higher risk of dental disease in children and adolescents [14, 15]. It is essential to reduce childhood obesity in order to lower the disease burden, both currently and in the future, as childhood obesity tends to track into adulthood [16, 17].

Eating behavior

There is great variability in eating habits around the world. However, parallel with the changes in obesity prevalence, many countries have undergone a dietary transition, entailing sweeter and more energy-dense diets in which fiber-rich foods are replaced with processed foods [18]. There has been a shift in how we eat, drink and move [19]. Today, soft drinks, fast food and ready-to-eat food are endlessly available [20, 21]. This easy access in combination with powerful marketing, is referred to as "the snack attack"

[22], affecting not only food choices but also frequency of consumption [23]. The high availability of unhealthy foods and beverages might be one reason why European children do not currently eat healthily and according to dietary guidelines, instead adhering to a diet low in fruit, vegetables and whole meal and high in refined carbohydrates, added sugars, fats and meat [24]. This change in food choice is not limited to high income countries, low- and middle-income countries such as Brazil, China, India, and Mexico are also adapting to a "Western diet" (high in refined carbohydrates, added sugars, fat, and animal-source foods) with an overall decrease in the intake of vegetables, legumes and whole meal foods [19].

Intake of added sugar has long been a global concern [25]. Based on the well-documented relationship between sugar intake and body weight as well as dental caries there is a strong recommendation from the WHO that both children and adults should limit their intake of sugars (added sugars as well as sugars in honey, syrups, and fruit juices) to <10E% per day [26]. In the Unites States, the intake of added sugar from foods has been stable during the last three decades while the intake of added sugar from beverages has increased considerably and contributes to approximately 66% of the total intake of added sugar [27]. The relationship between fat intake and body weight is not clear; however, findings from RCTs (randomized controlled trials) and cohort studies suggest an association between reduced fat intake and small reductions in body weight as well as with BMI and waist circumference in both children and adults [28]. However, it is important to acknowledge that it is the combination of dietary factors (dietary patterns) that contributes to the development of obesity [29]. More specifically, there is a positive association between dietary patterns containing energy-dense foods low in fiber and the development of obesity in children and adolescents [29, 30]. Since obesity, like many of its co-morbidities, is a result of repeated exposure to unhealthy behaviors and both healthy and unhealthy eating habits may persist from childhood into adolescence and on into adulthood, it is crucial to improve children's eating habits early in life.

1.1 Theoretical framework

In both children and adults, excessive weight gain is primarily the result of energy intake exceeding energy expenditure. However, the etiology of overweight and obesity is complex. There is thus more than one reason why someone might end up obese and it is important to understand that individual choices and behaviors are influenced by both modifiable and non-modifiable factors. Several models have been developed in order to capture the multifactorial etiology of obesity and they all focus on the interaction between individual, familial, and societal factors. One well-known ecological framework is the obesity system map developed by the UK Government's Foresight Programme in 2007 [31]. It includes 108 determinants of weight status, grouped in seven thematic clusters all applicable on the individual, familial, and societal levels: physiology, food production, food consumption, individual activity, physical activity (PA), environment, individual psychology, and social psychology. Additionally, the complexity and interdependence of the determinants of obesity are demonstrated by more than 300 interconnecting arrows. Another more comprehensible framework specifically focusing on children's weight status is the Ecological Systems Theory (EST) developed by Davison and Birch in 2001 [32]. According to the EST, childhood obesity is developed within a larger context in which individual, familial, and societal factors interact. Hence, risk factors for childhood obesity are not only moderated by individual characteristics such as age and sex, but also influenced by familial and societal factors. The EST depicts that on the familial level the relationship between children's diet and weight status is evolved and shaped by parental nutritional knowledge and role modeling as well as the parents' feeding practices, and familial level factors are in turn influenced by societal factors such as SEP and food availability [32]. Moreover, school- and childcare settings are important societal factors influencing the relationship between children's diet and weight status [32].

Another theoretical framework that places focus on parents-child interaction is the family consumer socialization framework for childhood obesity developed by Moore *et al.* [33] (Figure 1). In this framework, the family is the surrounding context in which the child develops his/her patterns of behavior through interactions with the parents. In contrast to the obesity system map and the EST, the family consumer socialization framework includes time – both linear and cyclic – to emphasize that childhood obesity is not a static condition. Linear time reflects the fact that both the child and the family continue to develop and change over time, while cyclic time reflects the fact that childhood obesity evolves through repeated behaviors and exposures over time.

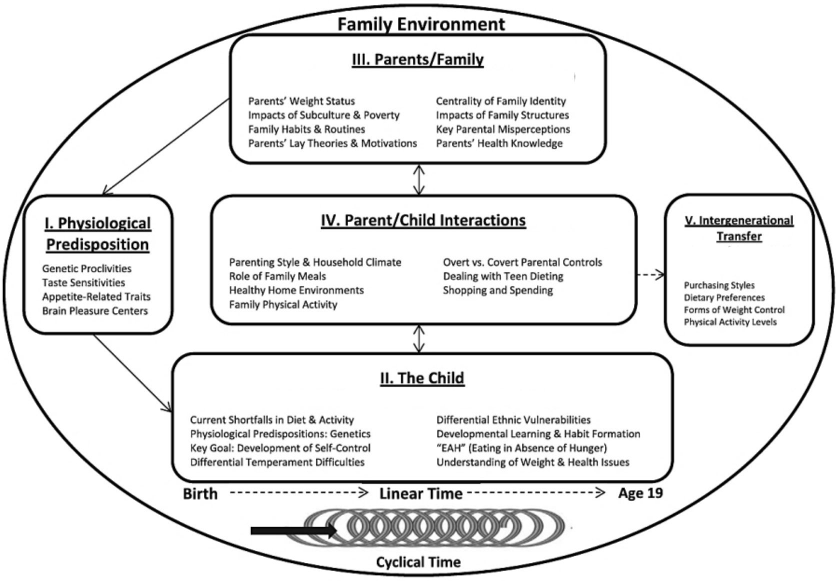

Figure 1. Family consumer socialization framework for childhood obesity (adapted version). Author: Moore et al. Published in: All in the Family? Parental Roles in the Epidemic of Childhood Obesity [33]: page 826.

The obesity system map, the EST and the family consumer socialization framework illustrate the modifiable and non-modifiable factors behind the development of childhood obesity and clearly demonstrate the importance of a multilevel approach in order to improve children's eating behavior, an important determinant of childhood obesity.

1.2 Defining overweight and obesity

Overweight and obesity are defined as an excessive amount of body fat and can be measured by several methods, such as dual-energy X-ray absorptiometry, underwater weighing (densitometry), measurement of skinfold thickness, and bioelectrical impedance analysis. However, BMI, calculated as weight (in kg) divided by squared height (in m), is the most common method currently used to screen for obesity. There is consensus that BMI is not only a non-invasive and cost-effective method, but is also easy to use in both clinical settings and large study populations [34-36].

According to the WHO definition, adults with a BMI of 25-29.9 are classified as overweight, and those with a BMI above 30 as obese. These cut-points are based on the relationship between BMI and metabolic risk factors such as hypertension and type 2 diabetes. Age- and sex-specific BMI references are used for children. In this thesis the International Obesity Task Force (IOTF) reference from 2012 was used. It was developed by Cole *et al*. in 2007 and is based on six large nationally representative cross-sectional growth studies including Brazil, Great Britain, Hong Kong, the Netherlands, Singapore and the United States [37]. The authors created age- and sex-specific overweight and obesity cut-points corresponding to the adult BMI cut-points at age 18 years for children aged 2 to 18 years [37]. In 2012, the IOTF reference was updated with BMI centiles and z-scores with minor adaptations to the cut-points resulting in a small (0.2%) mean change in prevalence rates [1]. Children's weight status can be classified from the BMI z-score where the group mean is zero and overweight corresponds to >1 standard deviation (SD) and obesity to >2SD above the mean BMI z-score of a representative reference population.

Another non-invasive and cost-effective method for measuring body composition is waist circumference. Waist circumference provides information on abdominal fat but the method has limitations, compared to BMI, due to measurement difficulties. Different measurement sites have been suggested (at the navel, at the narrowest part of the waist or between the lowest rib and the iliac crest) and there is no consensus concerning waist circumference cut-points in children. Moreover, breathing and difficulties standing still make waist circumference measurement more difficult in children than measuring height and weight.

1.3 Mutans streptococci and dental caries

From the 1990's there has been a decline in the number of teeth affected by caries in 12-year old children from the WHO European region. However, although dental caries has decreased, with the Decayed, Missing and Filled Teeth (DMFT) index decreasing from 3.0 to 1.8 there is a social gradient both within and between countries with caries prevalences ranging from 20% to 90% in the European region, with highest levels in Eastern Europe [38]. Further, the WHO/Europe oral health target for 2020, DMFT below 1.5 and at least 80% of six-year-olds free from caries [38] has not been achieved yet.

In 1976, Loesche's specific plaque hypothesis suggested that "dental caries was an infection with specific bacteria in the dental plaque of which the most relevant were mutans streptococci (MS) and lactobacilli (LB)" [39]. MS is an acidogenic microorganism that ferments dietary carbohydrates and is known to contribute to dental caries by lowering oral cavity pH, resulting in tooth demineralization [40, 41]. However, the later finding, that only 6-10% of the caries prevalence could be explained by MS counts while the large decrease in caries prevalence occurred without any changes in salivary MS counts [42, 43], fueled the "ecological plaque hypothesis", developed by Marsh in 1994 [44]. Similar to the ecological systems theory, the ecological plaque hypothesis suggests that "the selection of 'pathogenic' bacteria is directly coupled to changes in the environment" [45]. Hence, like childhood obesity, dental caries is a multifactorial disease highly dependent on diet [46] and the oral microflora imbalance caused by changes in the surrounding environment, such as high sugar intake (>10E%) [47], promoting MS colonization.

In children, positive associations between MS counts in saliva and risk of dental caries have been established in both cross-sectional [48-50] and longitudinal studies [51, 52]. However, the evidence regarding the relationship between BMI and dental caries in children is inconclusive. A U-shaped relationship was found by Hooley *et al.* [53], while Hayden *et al.* [54] reported positive associations between dental caries and childhood obesity. However, a recent review by Li *et al.* [55] concluded that evidence from longitudinal studies was conflicting and inconclusive [55]. It has, however, been established that children's weight status and dental health have common denominators, such as SEP and eating habits [40, 56].

1.4 Mental health

Mental health is conceptualized by the WHO as "a state of well-being in which the individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to his or her community" [57]. In children, the focus is mainly on developmental aspects such as self-concept, ability to cope with emotions and thoughts, and social relations [57]. The term 'mental health problems' ranges from daily worries and concerns to clinical disorders such as anxiety and depression. In children, mental health problems are normally classified in one of two dimensions: internal problems, including inward-directed behaviors or feelings (e.g. anxiety and depression), and external problems, including behaviors more directed at others (e.g. attention deficit, hyper-activity, and conduct problems) [58]. Similar to what has been reported on obesity, mental health problems in childhood seriously increase the risk of psychiatric disorders in adolescence [59] and adulthood [60, 61].

The approximate global prevalence of mental health problems in children is around 20% [62, 63], and many more children most likely experience mental health symptoms. From the 1980s to the mid-1990s, the prevalence of clinically identified psychosocial problems increased from 7% to 19% among children and adolescents in the United States [64]. There are differences between boys and girls in the types, prevalence, comorbidities and developmental course of mental health problems [65]. External problems are more common in boys than in girls [66, 67], whereas the opposite is true for internal disorders [66, 67]. However, a recent review on secular trends in mental health problems reported a stabilization in young children, while internal problems are still increasing among adolescent girls and findings among adolescent boys are inconclusive [68].

There is some evidence that childhood obesity is associated with both internal and external mental health problems [69]. Moreover, obesity and mental health problems are comorbidities across the life course [70-73] and the risk of future depression and emotional problems increases if the child is obese [74-76]. Findings in adults suggest that merely identifying oneself as overweight, independent of weight status, can increase the risk of depressive symptoms [77]. Similar to obesity, mental health can be impacted by socioeconomic factors. Children and adolescents with low SEP have an almost threefold risk of developing mental health problems, compared to children in high-SEP families [78]. Generally, the prevalence rates of mental health problems in children ranges from 13% to 33% in low SEP families and from 9% to 16% in high SEP families [78]. Moreover, reporting minimal social networks and living in a non-traditional family were associated with higher odds of mental health problems among children from the IDEFICS cohort [79]. Findings from the same study revealed that accumulation of social vulnerabilities was associated with higher risk of developing mental health problems [79].

Methods for treatment and prevention of mental health problems are important from both an individual and public health perspective. Mental health problems are commonly treated with psychotherapy, cognitive behavioral therapy or medication. However, during the 1990s PA was promoted as a potential preventive measure and treatment for common mental health problems such as anxiety, depression and behavioral problems [80, 81]. PA is important for physical and metabolic health [82], and may increase endorphin levels and reduce cortisol levels [83]. Nonetheless, the effects of PA on mental health seems to be minor and there is an overall lack of prospective studies supporting the general consensus concerning a positive effect of PA on mental health in either children [84-87] or adult populations [88].

Diet is another modifiable behavior increasingly gaining attention as a potential important determinant of mental health. Although the mechanisms behind the association require further investigation in longitudinal studies, there is a growing body of evidence supporting an inverse association between a healthy dietary pattern (high in fruit and vegetables, olive oil, nuts, fish and whole meal foods; low in saturated fat, sugar-rich products and red and processed meat) and the risk of depression in adult populations [89, 90]. Recently, the RCT ‘SMILES’ reported that higher adherence to healthy dietary guidelines improved mental health among adults with clinical depression. The authors concluded that “dietary improvement guided by a clinical dietician could be one treatment strategy for the management of depression” and that the benefits of this diet treatment might improve management of comorbidities such as obesity and cardiovascular disease [91]. These findings are in line with findings from a previous RCT conducted in adults [92].

In children and adolescents, the evidence base concerning the potential link between diet and mental health is still sparse and mainly consists of cross-sectional studies without the possibility to control for important confounders that could influence both dietary intake and mental health, such as SEP, and BMI. In adolescents, higher adherence to a dietary pattern high in red and processed meats, take-away foods, fried foods, saturated fat, sugar-rich products, and soft drinks at age 14 predicted an increase in external problems at age 17 in girls but not in boys [93]. However, no protective effect on either internal or external problems was found for a healthy dietary pattern (high in fruit and vegetables, legumes and whole meal) in either girls or boys [93]. The authors highlight the importance of not only examining heathy and unhealthy dietary patterns but also potential sex differences in future research on diet and mental health [93]. In 2014, a review by O’Neil *et al*. proposed a relationship between unhealthy diets and poor mental health [94]. Although the overall effect sizes were small, a recent review supports the findings of a link between unhealthy diets and poorer mental health, with additional indications that healthy dietary patterns may have an improving effect on children’s and adolescents’ mental health [95]. The authors called for prospective studies including clearly defined diet constructs as well as the potential to control for important confounders [94, 95].

1.5 Parental feeding practices

Birch *et al.* [96] noted, as early as in 2001, the influence of parents and the family food environment in shaping children’s food choices and eating habits; they are hence important determinants for the development of childhood obesity. This idea is supported by recent research, suggesting that parenting style and feeding practices have major impact on family meals and obesity development [97-99], and that healthy eating habits can be encouraged by parental role modeling [100]. A review examining the effect of parental feeding practices on children's intake of fruit and vegetables, sugar-sweetened beverages and snacks found food availability and parental role modeling to be the overall strongest predictors of both desirable and undesirable food consumption [101]. Moreover, a recent study in Norwegian pre-school children found strong positive associations between availability and accessibility of vegetables at home and the amount of vegetables consumed [102].

Parents shape the physical and social food environment, i.e. they decide on the 'when', 'what', and 'where' of the food provided [103-105]. While parenting style refers to the emotional climate of the interaction between parent and child (in which the parental practices are situated), parenting practices means the intentional or unintentional behaviors practiced by parents in order to shape the child's attitudes, behaviors and beliefs [106]. Hence, parenting practices refers to what parents do while parenting style refers to how they do it [106]. Generally, four types of parenting styles are distinguished, described by Maccoby and Martin [107] as: "authoritative (parents who are both demanding and responsive), authoritarian (parents who are demanding but low in responsiveness), indulgent (parents who are responsive but not demanding), and neglectful (parents who are neither responsive nor demanding)".

Studies of parental feeding practices (PFPs) have found that contextual factors such as education, income, ethnicity and acculturation [108-113], as well as parental characteristics such as weight [114, 115] and eating habits [112], determine PFPs as well as the manifestation and influence of PFPs on children's eating habits. Moreover, a bi-directional perspective on PFPs and children's eating habits and weight development has been investigated [116-118]; findings suggest that parents influence children's eating habits as well as responding to their child's individual characteristics [119].

The child's individual characteristics can moderate the effects of the home food environment for example the child's taste preferences and levels of PA. Moreover, both parenting style and parental eating habits can moderate the effects of parental feeding practices [120]; "children do as you do, not as you say", as the old saying puts it. Hence, it has been suggested that rather than restrict, pressure, and reward children the most effective feeding practices in promoting healthy eating habits in children may be those influencing the home food environment, such as availability, access, and parental role modeling [120].

Although parenting practices concerning children’s food intake and eating habits have become a fast-growing topic in child nutrition research [121, 122], there is no consensus around the assessment of PFPs. In 2016, Vaughn *et al.* [123] performed a systematic review on measures related to food parenting practices and assessed the quality of these practices. Based on current research a content map was presented (Figure 2) in order to clarify terminology and definitions in this area. Three constructs in PFPs were identified: coercive control, structure and autonomy support. Coercive control is referred to as “parent-centered” feeding practices and includes restriction, pressure to eat, threats and bribes, and using food to control negative emotions. As opposed to coercive control, structure and autonomy support are considered “child-centered”. Structure is defined as “parents’ organization of children’s environment to facilitate children’s competence” and includes, for example, rules and limits, limited or guided choices, monitoring, creation of meal- and snack-time routines, role modeling, home food availability and accessibility. Structure is applied in order for the child to learn and maintain eating habits, as well as in shaping the home food environment. Autonomy support includes nutrition education, child involvement, encouragement, praise, reasoning, and negotiation. In order to develop the child’s independence, the parents shape a food environment in which the child can be involved in making age-appropriate choices and they explain the reasons for rules and boundaries in the food environment.

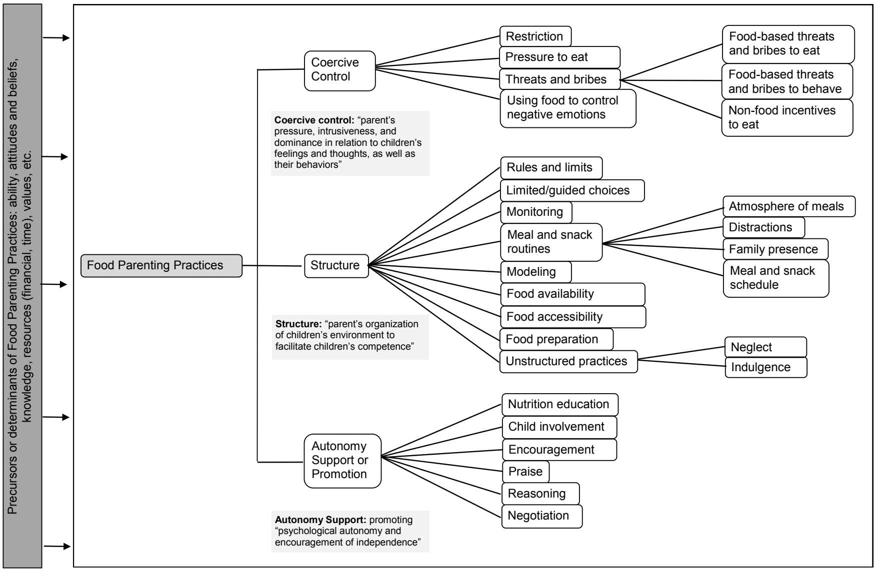

Figure 2. Content map of food parenting practices. Author: Vaughn et al. Published in: Fundamental constructs in food parenting practices: a content map to guide future research [123]: page 100

Ochoa and Berge [113] found that "parents are the policy makers for the home" in that they decide the quality and amount of food eaten as well as the mealtime environment. By being role models, by promoting ideals and attitudes, and by rewarding specific behaviors and discouraging others, parents are major influencers of children's eating habits. However, it is important to remember that feeding practices are formed as well as executed within different contexts meaning that factors of parenting at higher level (family and parent characteristics) can have an impact on lower level factors (parental feeding practices) [123]. Therefore, PFPs might have more or less influence on children's eating habits and weight development depending on the structure in which they occur.

1.6 Obesity interventions

Decreased sedentary behavior, increased PA and improved eating habits such as increased intake of fruit, vegetables, and whole meal foods, in combination with limiting intake of added sugar and saturated fat, are often included in interventions aimed at preventing childhood obesity. Although there is a large body of literature on this matter, the current state of evidence on the effectiveness of intervention studies aiming to prevent childhood obesity is inconclusive. However, in 2009 the proportion of studies showing positive results on children's BMI was approximately 40-50% [124]. Additionally, a 2011 review from the Cochrane Collaboration [125] reported evidence supporting a positive effect of intervention programs on childhood obesity in 6-to-12-year-olds. Recent meta-analyses also report modest but beneficial effects of well-designed intervention studies on children's BMI [126, 127]. On the other hand, some authors are less optimistic regarding the effect of intervention studies in preventing childhood overweight, due to the large variety in both intervention elements and settings, the lack of information on sustainability over time and the modest improvements in diet and PA [128-130]. Williams [131] summarizes the current state of evidence as:

> Many different interventions have been attempted. Quite a few have been tested. Some have demonstrated benefits in terms of preventing obesity or behavior change. Some have not. A minority of interventions have been rigorously evaluated, such that they provide robust evidence for (in)effectiveness in the trial setting. A few interventions have been well-described, such that other teams could attempt to implement a similar intervention again. Very few interventions have been evaluated in the years following their completion. Hardly any have been costed. Almost none have been retested at another time or place. Given this situation, it is not surprising that overall assessments differ.

Interventions centered on the family level emphasize the parent-child interaction and contextual factors defining the daily home environment [132]; the home food environment is important for both children's eating behaviors and obesity development [97-99]. Results from a pilot study empowering low-income parents of pre-school children to directly engage in the intervention design, and implementation, showed significant improvements in children's obesity rates as well as improvements in healthy eating- and activity behaviors [133]. Moreover, after the intervention, parents reported significantly greater self-efficacy to support healthy behaviors in their child [133]. Engaging parents and teachers is generally recommended, as they are important in modelling children's eating- and activity behaviors [134] but home-based obesity prevention studies targeting eating- and activity behaviors need to be accompanied by changes in environmental factors such as availability of healthy foods or access to green areas [135]. In order to prevent the development of obesity in children, the extensive context in which childhood obesity is developed must be acknowledged [32, 136]. Interventions aiming at preventing childhood obesity must thus have a multi-level approach and include support both on an individual, familial and community level [125, 137]. Moreover, the effectiveness and sustainability of interventions needs to be evaluated in order to establish what factors are important for a successful intervention program on childhood obesity.

2 Aim

The overall aim of this thesis was to investigate children's diet, BMI z-score, and parental feeding practices, in relation to mutans streptococci count, psychosocial well-being and children's BMI trajectory, as well as exploring the effect of the intervention in the IDEFICS (Identification and prevention of dietary- and lifestyle-induced health effects in children and infants) study on diets of families five years after the intervention.

Specific aims:

- Explore the cross-sectional association between BMI z-score, eating habits, and sleep and salivary mutans streptococci counts in a sub-sample of children from the Swedish IDEFICS cohort

- Investigate the bi-directional association between adherence to healthy dietary guidelines and psychosocial well-being in European children, and explore the impact of weight status on this association

- Investigate the prospective association between parental feeding practice and children's BMI trajectory, and explore the impact of social vulnerability on this association

- Explore the impact of the embedded community intervention on families' diet quality five years after the IDEFICS intervention

3 Methods

3.1 Sampling

In each of the eight countries participating in the IDEFICS study, publicly available statistical data were used to select intervention and control regions that were comparable with regard to infrastructure as well as to sociodemographic and socioeconomic characteristics. Additional inclusion criteria included reasonable travel distance for both participants and research teams but locations still separate enough to avoid contamination between the regions. In total, 24 communities were selected, whereof a majority were distinct cities or municipalities (except in Italy, where clusters of villages were selected), and within each community a majority of the kindergartens and primary schools were enrolled in the IDEFICS study. Via these settings, parents (or other caregivers) were approached and asked for consent to examine their children. Three municipalities in western Sweden were selected; Partille was the intervention municipality while Alingsås and Mölndal served as controls.

A flow chart of the study cohort is illustrated in Figure 3. Between September 2007 and June 2008, 31,643 children from schools and kindergartens in the selected regions were asked to participate in the IDEFICS baseline examination (T_0). Of those invited, 16,864 consented and participated. However, the number of children eligible for inclusion (parental questionnaire completed and height and weight measured) was 16,228. Hence, the response proportion at IDEFICS baseline was 51%. Of these 16,228 children, 1,809 (11%) originated from the Swedish IDEFICS cohort. More specifically, 902 children participated in the intervention municipality Partille.

Two years later, 68% of the original IDEFICS cohort returned for a second examination (T_1) (in the Swedish cohort the follow-up rate was 84%). Due to the setting-based recruitment, participation was also offered to all classmates of study participants, if the former had not participated in the first baseline survey; hence, 2,555 children were newly recruited at T_1. Children from the original IDEFICS cohort will be referred to as index children from here on. Three years later (in 2013), I.Family started and 7,105 of the index children agreed to participate in a third examination. At this time-point, an additional 2,512 siblings and 7,794 parents were examined. In total 6,167 families, with on average 2 children and 4.1 members, were included in the I.Family study.

In Sweden, 763 of the index children participated, together with 79 siblings and 683 parents (572 families) [138].

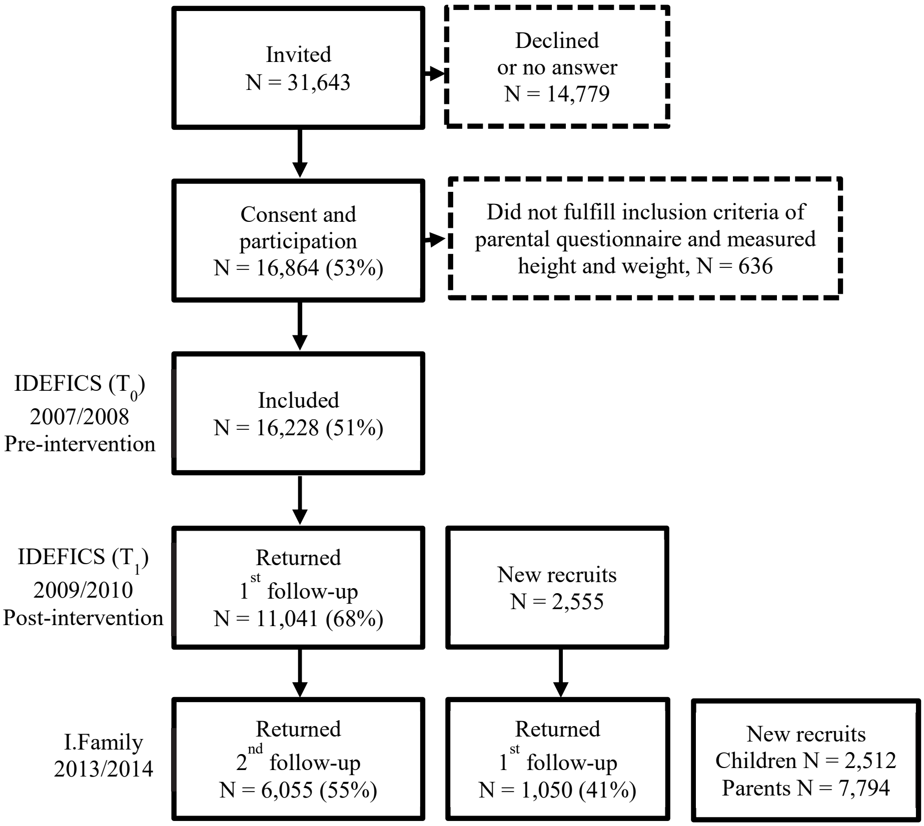

Figure 3. Flow chart of the baseline recruitment and subsequent follow-up examinations of the IDEFICS cohort and its extension with the I.Family study. T_0, baseline survey; T_1, first follow-up examination; I.Family, second follow-up examination. Adapted from Ahrens et al. [138].

The response proportion at T_0 was 51%, resembling that reported in other current epidemiological studies ([139, 140]) (Figure 3). Whether these 16,228 children participating at IDEFICS baseline differed from the non-participants regarding social and demographic factors is unknown. However, potential selection bias was recently assessed in the IDEFICS Sweden cohort [141]. Regber *et al.* used data from Statistics Sweden population registers to compare children participating in the IDEFICS Sweden cohort with referent children living in the same municipality (matched for age and sex). In line with previous findings in epidemiological studies [142, 143], they found higher participation among children in families with married or cohabiting parents, higher parental education and Swedish origin [141]. Moreover, compared to the reference population, the participating children had a lower obesity prevalence at 8 years of age [141]. Based on these findings [141], along with previous knowledge of non-participation bias in epidemiological studies [142], it is likely that the IDEFICS study had a greater participation of families with more advantageous social and demographic factors, which may create bias in prevalence estimates. However, it is important to remember that non-participation is not necessarily associated with the exposure or the outcome. There are several reasons for the declining participation rate, such as an increasing number of requests to participate in studies, difficulties contacting potential participants, and higher demands on study participants [142].

As seen in Figure 3, the participation rate was 68% at T_1 and 55% at I.Family (although among newly recruited children at T_1, the participation proportion was only 41%, possibly because they chose to not participate in the IDEFICS baseline examination). Hense *et al.* [144] investigated determinants of attrition to IDEFICS follow-up examination and reported that the drop-out rate varied from 18% in Sweden to 51.5% in Hungary [144]. Determinants of attrition were missing values and non-response at IDEFICS baseline (mainly information on social, demographic, and psychosocial factors), parents with foreign origin, low education level among parents, low well-being and overweight or obesity among the children [144].

3.2 The IDEFICS intervention

One part of the IDEFICS study was to develop, implement and evaluate evidence-based primary prevention strategies for childhood overweight and obesity. The community-based IDEFICS intervention had a holistic approach in the sense that it was executed on three intertwining levels: community, school, and family. Each of the eight countries included an intervention group and a control group of equal size, generating data on about 16,000 children.

The intervention modules were derived from existing knowledge, targeting diet, PA and stress. The standardized primary prevention activities addressed individuals, families, kindergartens and primary school settings and communities. The intervention mapping protocol (IMP) [145] was used as the theoretical framework for the development of the intervention. Detailed information on how the IMP was used in the IDEFICS project can be found in a previous paper by Verbestel *et al.* [146].

The specific program objectives of the intervention, i.e. the behaviors targeted, were: diet - increase daily consumption of water, fruit and vegetables; PA - increase daily PA levels, reduce daily TV viewing; stress - spend more time together as a family and ensure an adequate amount of sleep (Figure 4). The IDEFICS intervention toolbox [147] includes an overview of the intervention and its general content and structure. Implementation of the key messages regarding diet is described below and also includes local examples from Partille (see above) in Sweden.

Figure 4. Key behaviors chosen and translated into program objectives concerning diet, physical activity and stress (including spending time with family and sleep) Picture source: http://www.ideficsstudy.eu

The intervention components regarding key messages on diet are summarized in Figure 5 (on page 21). On the community level, the objectives were to facilitate community cooperation with the study, thereby preventing objections and resistance to it; inform all stakeholders and participants about the details, schedule and rationale of the study; support recruitment; retain participants; and help implement changes in the environment. In order to promote programs and structural changes encouraging targeted behaviors, there was a long-term media campaign including posters with the key messages on diet. Furthermore, residents of each intervention community were provided with opportunities to drink water at public places. In Partille, some workplaces started offering a basket of fruit to the staff one or several days per week.

On the school level, the objectives were to inspire involvement and commitment among staff members for implementation of the school-based intervention modules. Changes in the school environment were made in order to stimulate consumption of water, while discouraging consumption of sugar-sweetened beverages, as well as to make fruit and vegetables available at least once a week during recesses. Moreover, in order to promote the key dietary messages in the schools and kindergartens, they were integrated into the class curricula. Regarding water intake possible strategies were: to report water and soft drink consumption during the day and compare with the national recommendations, organize tasting activities to provide children with the opportunity to try different kinds of water such as sparkling water and water flavored with fresh fruits or vegetables (repeated exposure was essential), and organize activities to 'prepare' flavored water. Moreover, the IDEFICS project provided teachers with curriculum for classroom activities to increase water consumption during class. Examples of such activities: water cards filled in by the children for every glass of water, tasting games with different kinds of water, tap water available during the day (e.g. personal drinking cups or bottles in the classroom), and scheduled time for drinking water. There were also take- home materials, i.e. children brought their water cards and flavored water (see above) recipes home and were encouraged to show the cards to and try the recipes with their parents. Strategies to increase the intake of fruit and vegetables included a visit to a vegetable garden, making a picture collage or finger painting of fruit and vegetables and weighting and measuring fruit and vegetables in order to be able to identify what a respective standard portion of fruit or vegetables looks like. A curriculum provided to the teachers on classroom activities to increase the consumption of fruit and vegetables during the lectures included; children reporting their daily intake of fruit and vegetables; teachers making time for eating fruit and vegetables in class on fixed days; each child bringing his/her own piece of fruit and/or a vegetable that was peeled, cut, and used to make a fruit or vegetable salad; teachers preparing fruit and vegetables with the children in class; and tasting games.

In Partille the IDEFICS study encouraged the ongoing initiative of not serving sugar-sweetened beverages in schools and kindergartens. Also, tasting of flavored water was done in different places to show how fruit and vegetables can make water more fun and tasty. Water was also promoted as "the best thirst quencher", a line easy to remember in school, at sport practice and at home. In addition to the information on the key message to increase fruit and vegetable consumption some other good ideas regarding lunch were raised. The children were served raw and cooked vegetables first, followed by pasta/potatoes/rice and, last, fish/meat. Committed cooks willingly shared ideas and recipes with interested colleagues. The habit of bringing a piece of fruit to school as snack was presented as a good example. One kindergarten had a "vegetables time" before lunch.

On the family level, the diet objective was to enhance parents' skills in order to increase social support as well as accessibility to and availability of fruit and vegetables at home, and to increase their awareness and self-efficacy. A water newsletter was sent to the parents, containing encouragement to try different kinds of unsweetened flavored water together with their children, information on how to stimulate them to drink more water and less soft drinks and suggestions to purchase water together with their children and buy less soft drinks. The parents also received a newsletter on fruit and vegetables, containing information on the importance of acting as role models for children's consumption of these items, encouragement to increase availability and accessibility at home in order to stimulate their child to eat more fruits and vegetables, and suggestions to prepare a variety of fruit and vegetables at home with their children.

In Partille, folders with the key IDEFICS messages were sent to all parents with children in kindergarten or school. The folders were also available at other places such as child health care centers, antenatal clinics, and dental health clinics. Parents from the intervention areas were invited to seminars about children's diet, sleep and PA. The community webpage contained suggestions and ideas around a healthy lifestyle. Furthermore, representatives from the IDEFICS study visited nursery schools and child health services to share the key messages.

Community level

- Long-term media campaign including posters with the key messages on water, and fruit and vegetables
- Provide opportunities to drink water at public places

Family level

Water
- Newsletter
    - try different waters together with their child
    - stimulate more water and less soft drinks
    - buy water together with the child

Fruit and vegetables
- Newsletter
    - parental role modeling and children's consumption of fruit and vegetables
    - increase access and availability
    - prepare fruit and vegetables at home together with the child

Kindergarten and school level

Water
- stimulate consumption of water
    - report water and soft drink consumption during the day and compare with the national recommendations
    - tasting activities
    - increased water consumption during the lectures
    - take home materials

Fruit and vegetables
- increase consumption of fruit and vegetables
    - visit to a vegetables garden
    - collage with pictures of fruit and vegetables
    - identify what is a portion of fruit or vegetables
    - report intake of fruit and vegetables every day
    - fruit and vegetables are eaten in class on fixed days
    - tasting games in class

Figure 5. Summary of the intervention components regarding key messages on diet

3.3 Body mass index

Standardized anthropometric measurements were performed in children participating in IDEFICS and I.Family and in parents participating in I.Family. Weight was measured to the nearest 0.1 kg with a Tanita BC 420 SMA scale (TANITA, Tokyo, Japan), and height was measured to the nearest 0.1 cm by a SECA 225 Stadiometer (Seca GmbH & Co. KG., Hamburg, Germany). Waist circumference was measured according to a standard protocol [148] with an inelastic tape (Seca 200: precision 0.1 cm, range 0–150 cm), with the subject in the standing position. All measurements were performed in the morning, with the participants fasting and wearing only light clothes. In the case of a few parents (9%) and children (1.6%) in I.Family, height and/or weight were self-reported.

Age- and sex-specific BMI and BMI z-score for children and adolescents were calculated and used to categorize children as normal-weight (including thin) or overweight (including obese) [1]. BMI of the parents was categorized according to the WHO definition, i.e. BMI < 25 was classified as normal-weight (including thin) and BMI ≥ 25 as overweight (including obese). Moreover, the percentage of overweight in the family was determined based on the number of children and parents categorized as overweight (<25%, >25 and ≤ 50%, >50 and ≤ 75%, >75%).

Children's BMI trajectory from IDEFICS (T_0 or T_1) to I.Family was calculated using data on BMI z-scores and the children were categorized in one of four BMI trajectory groups (BTGs):

I. Children who remained normal-weight (BMI z-score between -1 and 1 at IDEFICS baseline and I.Family, and BMI z-score did not change more than ±0.1 per year)
II. Children with major weight gain (started with a BMI z-score of more than -1 at IDEFICS baseline and gained more than +0.1 in BMI z-score per year during the follow-up period)
III. Children who remained overweight or obese (BMI z-score of more than 1 at IDEFICS baseline and I.Family, and BMI z-score did not change more than ±0.1 per year)
IV. Children with major weight loss, i.e. who started off overweight with a BMI z-score of more than 1 at IDEFICS baseline and whose BMI z-score fell more than -0.1 per year during the follow-up period)

Standardization of measurement is of great importance for obtaining valid and reliable data and intra- and inter-observer errors were thus examined in a sub-sample of children during all surveys. Intra- and inter-observer reliability of the anthropometric measurements performed were calculated using Technical Error of Measurement (TEM) and the coefficient of reliability (R), an estimate of the proportion of between-subject variance that is free from measurement error. Reports from T_0 showed that intra-observer TEMs were low for measurements of height (0.2 cm) and weight (0.05kg) and somewhat higher for waist circumference (0.6 cm), and that R ranged from 99 to 100%. Inter-observer TEMs were low for height (0.3 cm), weight (0.06 kg) and waist circumference (0.6 cm) and, again, R ranged from 99 to 100% [149].

3.4 Diet

3.4.1 Parental feeding practices

The questions on parental feeding practices (PFPs) originated from the Preschooler Feeding Questionnaire developed by Baughum *et al.* [150]. The original questionnaire explores PFPs and concerns related to child overweight status and was designed and tested as a self-administered questionnaire for mothers of children aged 2 to 5. The original questionnaire included 35 items derived from focus groups, existing child feeding questionnaires, literature regarding mother-child feeding relationships, and the authors' clinical experience. Baughum *et al.* used factor analysis for item refinement and found eight constructs (factor loadings ranging from 0.49-0.82), including: 1) difficulty in child feeding, 2) concern about the child overeating or being overweight, 3) pushing the child to eat more, 4) using food to calm the child, 5) concern about the child being underweight, 6) child's control of feeding interactions, 7) structure during feeding interactions, and 8) age-inappropriate feeding [150]. In Baughum's analysis of internal consistency, constructs 1-3 had acceptable inter-item reliability (α) (0.87, 0.83, and 0.70, respectively), constructs 4 and 5 had near-acceptable reliability (0.68 and 0.69, respectively), while constructs 6-8 had poor reliability (0.50, 0.37, and 0.18, respectively).

In the IDEFICS study, only items with the highest factor loadings (obtained from the original article by Baughum *et al.* [150]) were chosen in order to limit questionnaire length. Hence, only questions relating to constructs 1-3 and constructs 7-8 were included: 1) difficulty in child feeding (struggle to get child to eat, child has poor appetite), 2) concern about child overeating or being overweight (has to stop child from eating too much, thinks about putting the child on a diet to keep him/her from becoming overweight, worried child is eating too much), 3) pushing the child to eat more (make child eat all the food on the plate, uses food child likes as a way to get child to eat healthy), 7) structure during feeding interaction (child watches TV at meals, sits down with child during meal time), and 8) age-inappropriate feeding (parent feeds child her-/himself if child does not eat enough). Possible answers ranged from 'Never' to 'Always' and the answers were dichotomized into "yes" if the parents answered 'Sometimes', 'Often' or 'Always' and "no" if the parents reported 'Rarely' or 'Never'. When these PFPs were investigated in relation to children's BMI trajectories in Study IV, questions relating to difficulty in child feeding were not included, as they are likely to be related to picky eating and underweight [151-153] rather than overweight, which was the study's focus. The question relating to age-inappropriate feeding was not included either, as it was loaded on the same factor as daytime bottle use [150] and was therefore not considered appropriate in our age group.

3.4.2 Food Frequency Questionnaire

The food frequency part of the Children's Eating Habits Questionnaire is best regarded as a screening instrument to investigate the consumption of foods shown by consistent evidence to be related, either positively or negatively, to overweight and obesity in children. The food frequency questionnaire (FFQ) was not designed to provide a valid measure of total energy intake or total food intake, and neutral foods or foods less likely to be associated with obesity, were not included. The FFQ questions were aimed at distinguishing healthy from unhealthy food types (e.g. white vs. whole meal bread), thereby making it possible to compare consumption of these alternatives. From an initial list of 60 food items for which frequency of intake was investigated, 43 were retained. These items were grouped into 14 food groups: Vegetables; Fruit; Drinks; Breakfast cereals; Milk; Yoghurt; Fish; Eggs and mayonnaise; Meat replacements and soy products; Cheese; Spreadable products; Cereal products; and Snacks. Country-specific food-items were included as examples to enable a correct understanding of the food groups. Before the start of the IDEFICS study, the FFQ was pre-tested in all centers in order to determine whether the food groups covered the foods consumed and the preparation methods used by the parents, and to ensure that the included questions were understandable [154]. Based on the pre-test results, the IDEFICS diet panel suggested food group modifications containing additional country-specific food items as well as rephrasing in order to clarify the FFQ [154].

In I.Family, the original 43-item FFQ used in IDEFICS was expanded with an additional food group (Cooking oil) as well as 5 new food items: coffee (unsweetened and sweetened), tea (unsweetened and sweetened), and alcohol (only included in the version for parents and adolescents). Moreover, some food groups from the original 43-item FFQ used in IDEFICS were divided into several food items in I.Family. For example, the food group “Drinks”, originally containing 4 food items (“water”, “fruit juices”, “sweetened drinks”, “diet coke or soft drinks”) was divided into 8 food items (“water”, “fruit juices”, “carbonated sugar-sweetened drinks”, “diet carbonated drinks”, “sugar-sweetened drinks, not carbonated”, “artificially sweetened drinks, not carbonated”, “coffee, unsweetened”, “coffee, sweetened”, “tea, unsweetened”, “tea, sweetened”). Overall, these changes resulted in an extended 58-item FFQ for I.Family.

In both IDEFICS and I.Family parents (or other caregivers) were asked to report the usual consumption frequency in a typical week during the preceding 4 weeks for all foods consumed, excluding foods served at school, i.e. "In the last month, how many times did your child eat or drink the following food items?" Response options were: never/less than once a week, 1-3 times a week, 4-6 times a week, 1 time per day, 2 times per day, 3 times per day, 4 or more times per day, I have no idea. This scale was adopted from the proxy eating habits questionnaire of the Early Childhood Longitudinal Survey from the United States Department of Agriculture [155]. No portion size estimates were obtained. In I.Family, self-reported data on consumption frequency was collected from parents and children above age 12. Moreover, the response alternative 'I have no idea' was no longer included due to the fact that it would have been treated as missing anyway. Children, adolescents and parents for whom more than 50% of the FFQ items were missing were excluded. In the remaining sample, the missing food items and the answer 'I have no idea' were treated as not consumed when composite scores were created. This is a common practice in nutrition surveys because food items often elicit blank responses if not consumed [156-158].

The relative validity of the FFQ was investigated by Bel-Serrat *et al.* in a sub-sample of 2,508 children from the IDEFICS study [159]. In comparison to repeated 24-hour dietary recall (24-HDR) the FFQ yielded both higher mean numbers of portions and food intakes. Less than 12% of the children were misclassified in the opposite quartile of food group intake by both 24-HDR and the FFQ, while around 30% were correctly classified in the same quartile of food group intakes. This is somewhat lower, compared to the results presented by Saeedi *et al.* [160], according to which 38%-52% of the children were correctly classified into the same food group intake quartile when a 23-item FFQ was validated against a 4-day estimated food diary. This lower relative validity could be due to the fact a different validation method was used and that the children's diets were proxy-reported in IDEFICS but self-reported in the study by Saaedi *et al.* [160]. Additionally, milk consumption frequency from the FFQ was validated against urinary calcium (UCa/Cr) and potassium (UK/Cr) in 10,309 children from the IDEFICS study [161]. There were significantly higher ratios of UCa/Cr and UK/Cr in children who reported ≥ 2 servings of milk per day, compared to those who reported less. Moreover, urinary calcium and potassium increased by increasing tertiles of reported milk consumption frequencies.

A sub-sample (N=258) of IDEFICS study participants filled out the FFQ again more than 4 months after the first FFQ in order to test reproducibility [162], showing that Spearman's correlation coefficients ranged from 0.32 to 0.76 and were higher than 0.5 for about 80% of the food items. This correlation coefficient range was similar to that found in 9-10-year-olds who self-reported their food intake using a 23-item FFQ (0.40-0.82) [160], as well as to the ranges found in 11-12-year-old Italian and Belgian children (0.40-0.83 and 0.40-0.82, respectively) [163]. The reproducibility was additionally investigated using weighted Cohen's kappa. It was found that the kappa coefficients ranged from 0.23 to 0.68, and that the level of reproducibility was similar across children's sex and ages. In general, reproducibility was related to consumption frequency, i.e. food items with the lowest mean consumption frequencies were those with the lowest mean Cohen's kappa and Spearman's correlation coefficients, and vice versa. Reproducibility was considered 'moderate' (between 0.41–0.60) for a majority of the food items according to the Landis and Koch classification [164]. On the other hand, McHugh [165] argues that a kappa coefficient between 0.41 and 0.60 allows for too much disagreement and suggests a different interpretation: kappa coefficients between 0.40-0.59 should be considered 'weak'.

Although there are few studies performed in children investigating FFQ reliability and validity, the overall level of reproducibility varies between 0.3-0.7 for food items [166]. A recent meta-analysis on the use of FFQs in adolescent populations concluded that the FFQ is a robust instrument for assessing food intakes, particularly for ranking adolescents according to intake levels [167]. However, the validity is likely to drop for children below the age of 12 [166], due to proxy reporting, difficulties recalling and high day-to-day variability in the diets of growing children. The FFQ used in I.Family is included in Appendix I.

Propensity ratios

The propensity ratios were calculated from the FFQ and provide a description of diet quality avoiding misclassification of participants as high consumers simply because they consume all types of food more frequently. It also corrects for eating occasions away from home that are not covered by the parental reported FFQ.

Sugar: the sugar propensity ratio was calculated using the formula of 'total intake frequency of sugar-rich food items per week/total intake frequency of all food items per week'. Food items considered rich in sugar were: fresh fruit with added sugar, fruit juices, carbonated sugar-sweetened drinks, sugar-sweetened non-carbonated drinks, sweetened coffee, sweetened tea, sweetened or sugared breakfast cereals, sweetened and/or flavored milk, sweetened and/or flavored yoghurt, sweet spreadables (jam, honey, chocolate or nut-based) and snacks (chocolate-based candies, non-fat candies, cake, pudding, cookies or ice cream).

Fat: the fat propensity ratio was calculated using the formula of 'total intake frequency of fatty food items per week/total intake frequency of all food items per week'. Food items considered rich in fat were: fried potatoes, whole-fat milk, whole-fat yoghurt, fried fish, cold cuts/sausages, fried meat, fried poultry, fried eggs, mayonnaise and mayonnaise-based products, cheese, chocolate-based or nut-based spread, butter/margarine on bread, oil, nuts and seeds, salty snacks, savory pastries, chocolate-based candies, cake, pudding, cookies and ice cream.

Water: the propensity to consume water as opposed to other beverages was calculated using the formula of 'intake frequency of water per week/total intake frequency of all beverages per week'. The denominator included the following 12 beverages: water, fruit juices, carbonated sugar-sweetened drinks, sugar-sweetened non-carbonated drinks, diet carbonated drinks, artificially sweetened non-carbonated drinks, unsweetened and sweetened coffee, unsweetened and sweetened tea and unsweetened and sweetened milk. Participants for whom more than six (50%) of the beverages were missing were excluded from the analysis. When composite scores were created, missing beverages were treated as not consumed.

Fruit and vegetables: the propensity to consume fruit and vegetables as opposed to other foods and beverages was calculated using the formula of 'intake frequency of fruit and vegetables per week/total intake frequency of all food items per week'. It included the following six items: legumes, boiled potatoes, other boiled vegetables, raw vegetables, fresh fruit without added sugar (including juice) and fresh fruits with added sugar (including juice).

The Healthy Dietary Adherence Score

An a priori diet score (i.e. hypothesis-driven, as opposed to data-driven), the Healthy Dietary Adherence Score (HDAS), was calculated from the previously mentioned 43-item FFQ. The HDAS was developed to reflect the guidelines established by Waijers *et al.* [168]. Specifically, it aims at capturing adherence to food-based dietary guidelines common for all eight countries participating in the IDEFICS study. The guidelines included: limit the intake of refined sugars; reduce fat intake, especially of saturated fat; choose whole meal foods when possible; consume 400-500 gram of fruit and vegetables per day and fish 2-3 times per week. Hence, the HDAS contains five sub-components: sugar, fat, whole meal, fruit and vegetables, and fish. The frequency data from the FFQ were converted into the HDAS and grouped into five sub-components reflecting adherence to food-based dietary guidelines on consumption of fruit and vegetables, fish, whole meal foods, fat, and sugar (for a detailed description, see Appendix II).

3.5 Psychosocial well-being

Instruments for measuring quality of life in children should be "age-appropriate and child-centered, preferably take into account self-reporting, be usable independently of the health status and cross-culturally, and should include both positive and negative aspects" [169]. At T_0 and T_1 parents reported on children's psychosocial well-being (referred to as well-being from here on) using the indicators self-esteem and parent relations from the Kinder Lebensqualität Fragebogen (KINDL®) [170], and emotional and peer problems from the Strengths and Difficulties Questionnaire (SDQ) [171].

3.5.1 Kinder Lebensqualität Fragebogen

Self-esteem and parent relations were calculated from responses to the Kinder Lebensqualität Fragebogen (KINDL®) [170]. This questionnaire was developed with the help of focus groups involving children and adolescents. It was designed for measuring HRQoL in children and adolescents with disabilities and later validated for healthy populations [170]. The IDEFICS study included a version of the KINDL® developed for parent response on behalf of children and adolescents between 7 and 17 years of age. The self-esteem questions included: During the last week my child… (1) had fun and laughed a lot, (2) didn't feel much like doing anything, (3) felt alone, and (4) felt scared or unsure of him/herself. The parent relations questions included: During the last week my child… (1) got on well with us as parents, (2) felt fine at home, (3) we quarreled at home, and (4) felt that I was bossing him/her around. The items were scored from 1 (never) to 4 (often or always) with reversals according to the wording of the question, added to generate total scores and transformed to percentage scores ranging from 0 to 100%. The total scores were then dichotomized into 'poor' or 'good' using sex- and age-specific cut-off scores from the KINDL® manual [170]. However, due to skewness of the data (a majority of the children - 98 % at baseline and 97 % at follow-up - reported 'good'), self-esteem was re-categorized into 'lower' and 'better' self-esteem at the group median.

Comparison of similar dimensions of KINDL® and KIDSCREEN-52 [172] (a generic instrument used to assess subjective health and well-being in children and adolescents) revealed that the correlation coefficients (Pearson's r) varied between 0.23 (self-esteem) and to 0.53 (parent relations) [173]. The correlation between self- and parent reported responses to the KINDL® was 0.23 for self-esteem and 0.50 for parent relations [173]. However, the convergent validity was slightly higher for the parent reported version than for the self-reported version (0.44-0.63 vs. 0.33-0.59, respectively) [174]. Two studies reported the internal consistency (Cronbach's alpha) for the sub-scales self-esteem and parent relations to be 0.68-0.70 and 0.72-0.74, respectively for both self- and parent reported responses [173, 174]. Moreover, by comparing answers from healthy children and children with a chronic disease it was concluded that KINDL® could detect significant differences between these groups for all sub-scales with effect sizes above 0.20 (Cohen's d) for both self-esteem and parental relations [173].

3.5.2 Strengths and Difficulties Questionnaire

Emotional and peer problems were calculated from the Strengths and Difficulties Questionnaire (SDQ) [171], created by Goodman *et al.* and based on the Rutter Questionnaires developed in the 1960s [175]. It measures psychosocial problems and strengths in 3 to 16-year old children. As the SDQ is translated into over 60 languages, it has been widely used as a screening and research tool, a treatment-outcome measure, and a part of clinical assessment [176]. Moreover, it has been used in several studies of diet and psychosocial problems [177-182]. The IDEFICS study used the informant-rated version for children aged 4 to 16, which has been found to correlate well with the child-rated version [183, 184]. The emotional problem questions included: To what extent do the following characterizations apply to your child? (1) often complains of headaches, stomach-aches or sickness, (2) many worries or often seems worried, (3) often unhappy, depressed or tearful, (4) nervous in new situations, easily loses confidence, and (5) many fears, easily scared. The peer problem questions included: To what extent do the following characterizations apply to your child? (1) rather solitary, tends to play alone, (2) has at least one good friend, (3) generally liked by other children (4) picked on or bullied by other children, and (5) gets on better with adults than with other children. Items were scored from 0 ('not true') to 2 ('certainly true') and added up to generate total scores ranging from 0 to 10, where a high value indicated more difficulties. In accordance with the scoring procedures available online (http://www.sdqinfo.org), the emotional and peer problem scores were divided into: 'inconspicuous', 'borderline' and 'abnormal'. In "The Strengths and Difficulties Questionnaire: a Research Note" [171], Goodman suggested that the borderline cut-points should be used in studies of high-risk samples where false positives are not a major concern, while the abnormal cut-points should be used in studies of low-risk samples where it is more important to reduce the rate of false positives. Hence, a dichotomized variable was created consisting of poor well-being (including both 'borderline' and 'abnormal' groups) versus the remaining children with no detectable ('inconspicuous') poor well-being.

In children, the parent reported version of the SDQ showed internal consistencies ranging from 0.46 to 0.82 [176, 185]. Re-test reliability

(Pearson's R) of the parent-reported version was 0.72 for the emotional problems score and 0.54 for the peer problem score [185]. The SDQ was tested against psychiatric disorders and it was found that both the total score and the sub-scores could distinguish between low-risk and high-risk groups (i.e. presence or absence of any DSM-IV psychiatric diagnosis) [186]. The emotional problems score was associated with higher odds of anxiety or depression for both parent- and self-reported responses [186]. In 4-6-year-olds, the SDQ scores correlated with those on the Child Behavior Check List (CBCL) [185]. More specifically, the emotional problems score correlated highly with the internal problems scale as measured by the CBCL while external problems such as peer problems were closely related to the CBCL External problems scale [185]. Although a multi-informant approach in both clinical practice and research is suggested [176], studies investigating the psychometric properties of the parent-reported version of the SDQ find strong reliability and validity for both the total SDQ and the sub-scores included [176, 185, 186].

3.6 Meal frequency and sleep duration

Meal frequency and sleep duration were calculated from SACINA (Self-Administered Children and Infant Nutrition Assessment) at T_1. SACINA is a 24-hour diet recall (24-HDR) program based on the previous Young Adolescents Nutrition Assessment on Computer (YANA-C) [187]. Assisted by a dietitian, the parents, or other caregivers, reported what the child had eaten and drunk during the previous 24 hours, as well as the child's wake-up and bed times. At T_0 it was only possible to report six meals in SACINA (breakfast, lunch, dinner and three snack occasions). This was later changed and there were no limits on the number of meals during the day in T_1 and I.Family.

3.7 Sedentary behavior

Information on the duration of the child's TV/DVD/video viewing and computer/game-console use during weekdays and weekends was reported by the parents in all surveys. The response options were: not at all; ≤0.5 h/d; <1 h/d; between 1 and <2 h/d; between 2 and <3 h/d; and ≥3 h/d. Total screen time per day was calculated as (5*weekday + 2*weekend)/7, based on combined computer and TV hours.

3.8 Mutans streptococci counts

Saliva was collected in connection with a dietary sub-study, preceded by strategic sampling to represent both the control (Alingsås and Mölndal) and intervention (Partille) areas in Sweden, including 728 children of whom 40 % provided a saliva sample. Reasons for not providing saliva were lack of time (no time for the research dietitian to collect saliva or parents were in a hurry), child refused or parents did not want their child to chew the paraffin. The saliva was collected during the fasting morning examination and delivered to the Institute of Odontology at University of Gothenburg the same day, where it was processed within 24 hours according to standard protocol at this time-point. Colony-forming units (CFU) of MS were counted and identified by their colony morphology [188] and divided into two groups for categorical analysis: medium–high counts (>105 CFU/ml), also referred to as 'higher', and low counts (≤105 CFU/ml). These thresholds are commonly used in dental research and known to predict high or low risk of caries [189].

3.9 Parental income and education

Data on parental education and income were obtained from a parental questionnaire at T_0, T_1, and I.Family. Parents were asked to indicate their own and their partners' highest level of education. The particular response categories for each country were coded according to the International Standard Classification of Education (ISCED) [190]. ISCED ranges from level 0 to level 8, where levels 0-3 represent upper secondary school and levels 4–8 represent post-secondary education. For the analysis, parental education was included as a continuous variable (except for Paper I). However, for the descriptive data, it was re-categorized into lower education (ISCED levels 0-3) or higher education (ISCED levels 4-8) in all papers except Paper IV, in which education was re-categorized into three categories: low (ISCED levels 0-2), medium (ISCED levels 3-4) and high (ISCED level 5-8).

At all three time-points (T_0 to I.Family), parents were also asked to indicate their own and their partners' highest level of income. Country-specific income levels were assigned with reference to the average net equivalence income, considering the median income and poverty line. Possible income levels ranged from 1 to 9. For the analysis, parental income level was included as a continuous variable and for the descriptive data, it was re-categorized into lower (levels 1-5) and higher income level (levels 6-9).

3.10 Social vulnerability score

The social vulnerability score was developed and described in detail by Iguacel *et al.* [191]. In addition to the classic indicators of SEP, i.e. income and education, the vulnerability score also includes minimal social network, non-traditional family, migrant status, and unemployment (all dichotomized as vulnerable or non-vulnerable), obtainable from the parental questionnaire at T_0. A child was considered as having a minimal social network if the parents answered that they had 0-1 person that they could rely on in case of need (including relatives). Children living in single-parent families, step-parent families, with grandparents or foster parents or in an institution were considered to be living in a non-traditional family. If one or both parents were from a country that differed from the study country, a migrant background was assumed. If one or both parents were unemployed or living on social assistance or welfare, this was considered an indicator of social vulnerability. The total social vulnerability score was calculated by adding the number of vulnerabilities a child was exposed to. The score ranged from 0 (no vulnerability indicators) to 6 (all six vulnerability indicators) and was divided into three categories for the analysis: 0 vulnerability indicators, i.e. non-vulnerable, 1 vulnerability indicator and 2 or more vulnerability indicators.

3.11 Statistics

The structure of the IDEFICS/I.Family studies consists of multiple levels, i.e. countries, communities, schools and families. Due, for instance, to different sociodemographic factors, cultures and norms clusters are likely to be detected in the data. Children in a country may resemble each other more than they resemble children in another country, and children from one school or one family may be more similar to each other than to children in a different school or family. Since regression analysis is based on the assumption that the observations are independent of each other, i.e. no relationship exists between measures in one individual and measures in any other individual, the regression model has to take into account the correlations due to the clustered study design. Each country, community, school and family was assigned a code that made it possible to explore the outcome variability at each level of the hierarchical structure (Figure 6). In Papers II-IV, we found statistically significant variability on different levels implicating the necessity to use a multilevel model. Hence, similar analyses with the Generalized Linear Mixed Model (GLMM) procedure for regression analyses in SPSS were used in Papers II-IV. The procedures were modified to suit analyses with continuous and dichotomous outcomes. The data was analyzed using IBM SPSS Statistics Version 20 and the significance level was set at $\alpha = 0.05$ (two-tailed), not accounting for multiple testing.

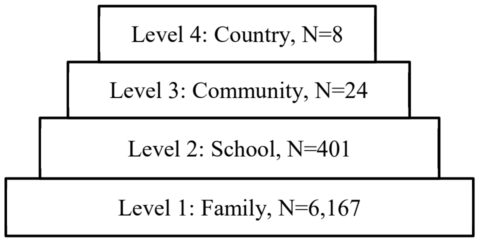

Figure 6. Hierarchical data structure in the IDEFICS-I.Family cohort

In Paper I, age-adjusted univariate logistic regression was used to investigate the association of potential covariates with higher MS count. The final model was obtained by multiple logistic regression with stepwise forward selection among the whole set of covariates. The model was checked for multicollinearity using variance inflation factor (VIF). VIF is calculated as 1-R squared for each variable and is the inverse of tolerance (1/Tolerance), hence a low tolerance value implies a high VIF and vice versa. VIF varied around 1.0, suggesting low inter-correlations between effect estimates of variables (not presented in the paper). Next, the area under the receiver-operating characteristic curve (ROC curve) was used to estimate how well the final model predicted the outcome. To examine the possible respective effect of dietary under-reporting and age, sensitivity analyses were performed by excluding participants with only one meal reported (n=4) in the 24-hour diet recall, and by forcing age into the multivariable model. The statistical software package SAS version 9.3 was used for calculation of the ROC curve.

In Paper II, multilevel logistic regression was used for bidirectional modeling of dichotomous dietary and well-being variables as both exposures and outcomes, since they were measured both at T_0 and T_1. Statistically significant variability in the outcome was found for country; hence random intercepts for country were included to account for the clustered study design. The models were adjusted for age, sex, BMI z-score, highest parental education and income, as well as for baseline diet or well-being respectively. Interaction terms were included in the analyses to test for modification by weight status and significant interactions terms were confirmed (p-value <0.05); hence, stratified analyses were performed to investigate whether the association between diet and well-being differed between children with overweight and children with normal weight. Additionally, stratified analyses were performed in boys and girls separately. Next, sensitivity analyses were performed to further explore the associations between diet and well-being. Firstly, group medians (as opposed to clinically derived cut-points) were used as cut-points for the indicators of well-being (as both exposure and outcome) to estimate standardized effect sizes in both directions. Secondly, the diet score was divided into quartiles to investigate the potential dose-response relationship between diet at T_0 and children's well-being at T_1.

In Paper III, associations between parental feeding practices (PFPs) at baseline and children's BMI trajectory were investigated. Because of statistically significant variability in BMI trajectories due to center and school level multilevel linear regression with random intercepts for study center (community) and school was used. The mutually adjusted model only included PFPs independently associated with BMI z-score at a significance level of 0.05. The prospective analysis by multilevel logistic regression models was restricted to two comparisons, using groups with similar baseline BMI z-scores as outcomes (Remained normal weight vs Experienced excessive weight gain, and Remained overweight vs. Experienced major weight loss). The models were checked for multicollinearity using VIF, and VIF values varied between 1.0-1.2 suggesting low intercorrelations between variables. Initially, our basic models accounted for sex of the child, age at baseline, baseline BMI z-score, parental education, and intervention exposure. Random intercepts for community (study center) and school were included to account for the clustered study design. The full models were additionally adjusted for time spent in sedentary behavior, parental BMI, and parental concern about the child becoming overweight. However, a sensitivity analysis was performed in which children's adherence to healthy dietary guidelines was added to the full model in order to explore the potential impact of diet on the associations. Finally, interactions between the number of social vulnerabilities and PFPs on children's BMI trajectory were tested by including interaction terms in the full model, and we performed stratified analyses by vulnerability group when the interaction was significant at a p-value <0.05.

In Paper IV, differences in diet quality (as measured by propensity for consuming fat, sugar, water and fruit and vegetables) between intervention and control families five years after the IDEFICS intervention were investigated. Multilevel models using the mean diet propensity ratio of the families as the dependent variable were used to assess whether there were significant differences between intervention and control families. Community (study center) was included both as a random intercept and as a random slope for the effect of intervention on the outcome (propensity ratio). The basic model was adjusted for country, age (mean age of the parents and children) and sex (percentage of female participants in the family). The full model was additionally adjusted for overweight (percentage of overweight participants in the family) and household size. Among the subset of families with available parental education data, stratified analyses were performed by lower and higher education strata using the same models as described above. The family data were disaggregated into parents and children to ascertain whether the differences in diet propensity ratios between intervention and control families could be confirmed in parents and children individually. Multilevel models included an additional random effect for family membership. To adjust for potential confounding, the following covariates were considered in the basic model: country, sex and age. The fully adjusted model further included BMI and household size. Finally, we included parental educational level as a covariate. Additionally, stratified analyses were performed among families with available parental education data. The same multilevel models described in the previous text were used to evaluate whether index children from control and intervention groups already differed in propensity ratios at baseline. Additionally, multilevel models with community (study center) included as random effect and additionally adjusted for country, sex, age, BMI, household size and parental education were used to evaluate the whether there was a difference in change from IDEFICS to I.Family for propensity to consume fat, sugar and water between index children from control and intervention groups (not presented in the paper as the harmonization of the FFQs was not agreed on at the time of publication). All analyses for Paper IV were performed with Stata 13.1 (StataCorp, College Station, TX, USA; http://www.stata. com) except the prospective analyses which were performed with IBM SPSS Statistics Version 20.

3.12 Ethical considerations

All applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research. In each country, local research ethics committees approved the IDEFICS (for Sweden: DNR 264-07) and I.Family (for Sweden: DNR 927-12) studies. During the planning and processing of both studies, participants were informed about study objectives, that participation was voluntary and that they could consent to single components of the study while abstaining from others. Participants were also informed that personal data and collected samples would be treated with confidentiality and that all collected data would be used for research purposes only. Before the studies started, written informed consent was given by parents and children older than age 16 (as suggested by the review board), for all examinations and sample collection, data analysis, and storage of personal data and samples. Children under age 16 gave additional oral consent at the time for examinations and sample collection.

Involving children in research is problematic, from an ethical perspective, because their judgement regarding risks and consequences is limited, and because they are easily influenced by others. In the IDEFICS and I.Family studies, field personnel were well trained in the examination process (registered nurses collected all samples requiring invasive procedures) and attentive to the fact that some children agreed to the examination due to pressure from their parents. However, the methods included in this thesis were not invasive and posed no physical risk to the child. Nonetheless, height and weight can be a sensitive matter even in this age group, requiring well-trained and tactful field personnel.

4 Results

4.1 Characteristics of the study population

At IDEFICS baseline (T_0), approximately half of the children (52%) were included in the intervention group (Table 1). The number of children participating in each country ranged from 1,508 in Spain to 2,568 in Hungary. Overall, there was an equal distribution of boys and girls and the mean age was 6.0 years. The mean BMI z-score was 0.3, and at this time-point the percentage of overweight (including obese) children was 20%. Moreover, 47% of the children had parents with high education and 52% had parents with high income at T_0. Finally, in 87% of the cases it was the mother who was the reporting parent.

At I.Family, 56% of the index children in the intervention group participated (Table 1). The number of participating children in each country ranged from 412 in Belgium to 2,505 in Cyprus. At this time-point, the mean age was 11.0 years and there was still an equal distribution of boys and girls. The mean BMI z-score was 0.5 and the percentage of overweight (including obese) children was now 27%. At I.Family, the percentage of parents with high education and income was 42% and 60%, respectively. Moreover, it was still most common that the mother was the reporting parent (82%).

In the Swedish cohort, half of the children were included in the intervention group. Moreover, the distribution of boys and girls and the mean age were similar to those of the total sample at both T_0 and I.Family (T_0: 5.7 years, 49% girls; I.Family: 10.8 years, 49% girls). However, the percentage of children with overweight (including obesity) was lower, compared to the total sample, at both time-points (T_0: 11%; I.Family: 13%), and high parental income (T_0: 75%; I.Family: 85%) and education (T_0: 69%; I.Family: 79%) were more common. Finally, the percentage of mothers reporting was somewhat lower than that in the total sample (T_0: 83%; I.Family: 77%).

Table 1. *Descriptive characteristics of the children participating at IDEFICS baseline and at I.Family*

	IDEFICS baseline (T_0) N = 16,228		I.Family N = 9,617	
	Mean (SD)	Min. Max.	Mean (SD)	Min. Max.
Age	6.0 (1.8)	2.0;9.9	11.0 (2.9)	1.8;20.7
BMI z-score	0.3 (1.2)	-6.4;5.8	0.5 (1.1)	-9.8;4.4
	n	%	n	%
Sex of the child (female)	7,977	49	4,745	49
Overweight (incl. obese)	3,201	20	2,550	27
Number of siblings	3,469	21	6,326	66
Reporting parent(s)				
Mother	13,459	87	4,279	82
Father	1,815	12	847	16
Both/other	231	1	102	2
Participating parent(s)				
Mother	-	-	5,113	64
Father	-	-	2,821	36
Parental education				
High	7,375	47	2,422	42
Medium	7,068	46	3,010	52
Low	1,087	7	370	6
Parental income				
High	7,243	52	3,235	60
Low	6,674	48	2,174	40
Country				
Italy	2,250	14	1,523	16
Estonia	1,719	11	1,338	14
Cyprus	2,383	15	2,505	26
Belgium	1,925	12	412	4
Sweden	1,809	11	842	9
Germany	2,066	13	1,299	14
Hungary	2,568	16	1,142	12
Spain	1,508	9	585	6
Intervention group	8,482	52	4,070	56

4.2 Mutans streptococci and BMI

The cross-sectional association between salivary MS counts (see Introduction) and children's weight status was explored in a sub-sample of 271 children (46% females) from the Swedish IDEFICS cohort. A higher MS count ($>10^5$ CFU/ml) was found in 18% of the children. As presented in Table 2, compared to children with low MS counts, children with high counts were older, reported a higher propensity for consuming sugar and higher meal frequency. Moreover, they reported shorter sleep duration, had larger waist circumference and marginally higher BMI z-score. Investigation of the relationship between these covariates and salivary MS counts with multivariable logistic regression revealed positive associations between higher MS count and number of meals, sugar propensity ratio, BMI z-score and female sex, while a negative association was found between higher MS count and sleep duration (Table 3). However, when age was forced into the model, the effect of sex was attenuated while the other estimates remained unchanged (data not shown).

Table 2. *Distribution of variables by bacterial status (low vs. medium-high counts of mutans streptococci). N=271*

	Low counts[1] ($\leq 10^5$ CFU/ml) n = 223		Medium-high counts ($>10^5$ CFU/ml) n = 48		
Variable	Mean	SD	Mean	SD	P-value†
Age (year)	8.2	1.9	8.9	1.6	0.01
Sugar propensity ratio (%)	21.9	3.5	23.5	4.6	0.02
Sleep duration (hour)	10.1	0.7	9.7	0.6	0.00
Number of meals	5.5	1.2	5.9	1.2	0.03
BMI z-score	0.1	1.3	0.5	1.2	0.05
Waist circumference (cm)	57.7	6.8	60.5	8.4	0.03

[1]Subjects below detection limit of MS (200 CFU/ml) = 183 (67.5 %)
Counts of MS among subjects with MS above detection limit = 1.3 (0.46-13.8) x 10^5 (described as median and Inter Quartile Range)
†t-test for continuous variables

Table 3. *Odds ratio for medium-high counts of mutans streptococci (MS). Included in both analyses N=233*

Variable	OR	95 % CI	P-value
Univariate regression			
Age (year)	1.3	1.0,1.6	0.02
Age-adjusted logistic regression			
Sugar propensity ratio (%)	1.1	1.0,1.2	0.03
Sleep duration (hour)	0.5	0.3,0.9	0.02
Number of meals	1.3	1.0,1.8	0.08
BMI z-score	1.4	1.0,2.0	0.03
Waist (cm)	1.0	1.0,1.1	0.10
Sex (female)	1.6	0.8,3.4	0.20
Education level (high)	0.8	0.3,2.1	0.61
Intervention area	1.0	0.5,1.8	0.89
Final model (multivariable logistic regression with forward stepwise selection of variables)†			
Sugar propensity ratio (%)	1.1	1.0,1.3	0.03
Sleep duration (hour)	0.5	0.3,1.0	0.04
Number of meals	1.5	1.1,2.2	0.01
BMI z-score	1.6	1.1,2.3	0.01
Sex (female)	2.4	1.1,5.4	0.03

†Area under the receiver-operating characteristic (ROC) curve = 0.8 (95% CI=0.7,0.9)

4.3 Diet and psychosocial well-being

The relationship between adherence to healthy dietary guidelines and psychosocial well-being was investigated in a sample of 7,675 children. At T_0, a majority reported high mean scores for self-esteem and parental relations (89.4 and 84.8, respectively) and low mean scores for emotional and peer problems (1.6 and 1.3, respectively) (Table 4). Regarding adherence to a healthy diet, the group HDAS mean was 22 at baseline. On the group level, all indicators of well-being and adherence to a healthy diet, as indicated by the HDAS, were stable two years later.

Table 4. *Descriptive characteristics at IDEFICS baseline and at the two year follow-up (N=7,675)*

	Baseline T_0		Follow-up T_1	
	mean ± SD	IQR	mean ± SD	IQR
Self-esteem score (KINDL®)	89.4 (10.4)	81.3;100.0	87.0 (10.7)	81.3;93.8
Parent relations score (KINDL®)	84.8 (10.2)	75.0;93.8	83.9 (10.5)	75.0;93.8
Emotional problems score (SDQ)	1.6 (1.7)	0.0;2.0	1.6 (1.7)	0.0;2.0
Peer problems score (SDQ)	1.3 (1.5)	0.0;2.0	1.2 (1.5)	0.0;2.0
HDAS	22.0 (9.0)	16.0;28.0	23.0 (9.0)	17.0;28.0
	n	%	n	%
Indicators of well-being				
Better self-esteem (KINDL®)$^{\Delta}$	4,086	53	4,702	61
Good parent relations (KINDL®)$^{\Delta}$	3,914	51	3,824	50
No detectable emotional problems (SDQ)$^{\Lambda}$	6,625	86	6,620	86
No detectable peer problems (SDQ)$^{\Lambda}$	6,194	81	6,349	83
Healthy Dietary Adherence Score				
Total HDAS (higher adherence)†	4,017	52	3,970	52
HDAS components (higher adherence)				
fruit and vegetables	4,983	65	4,993	65
fish	5,383	71	5,315	70
whole meal	4,011	52	4,217	56
sugar	1,642	21	1,868	24
fat	4,846	63	4,196	55

ΔCut-off values for Better self-esteem ≥ 87.50 (both sexes) and Good parent relations > 83.58(m)/84.40(f)
ΛCut-off values for No detectable emotional problems ≤ 3 and No detectable peer problems ≤ 2
†Cut-off value for Higher adherence to the HDAS ≥21 at baseline and ≥22 at follow-up
IQR, Interquartile Range; SD, standard deviation; SDQ, Strengths and Difficulties Questionnaire; HDAS, Healthy Dietary Adherence Score

Baseline diet as a predictor of well-being at follow-up

In the total sample, higher adherence to a healthy diet at T_0 (HDAS ≥21) was associated with better self-esteem (OR=1.2; 95% confidence interval (CI)=1.0,1.4) and fewer emotional and peer problems (OR=1.2; 95% CI=1.1,1.3 and OR=1.3; 95% CI=1.2,1.4, respectively) two years later (Table 5). Further analyses of the separate components included in the HDAS identified positive associations between baseline consumption of fruit and vegetables, fish, whole meal and fat, in accordance with dietary guidelines, and indicators of well-being two years later.

Table 5. *Prospective associations between baseline adherence to the HDAS and indicators of psychosocial well-being two years later (N=7,196)*

	KINDL®				SDQ			
	Better self-esteem		Good parent relations		No detectable emotional problems		No detectable peer problems	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
HDAS (higher adherence)	1.2**	1.1,1.3	1.1	1.0,1.3	1.2**	1.1,1.3	1.3***	1.1,1.4

*p<0.05, **p<0.01, ***p<0.001
P-value obtained using a multilevel model correcting for cluster design (country)
Models adjusted for: age, sex, BMI z-score, well-being at baseline, and highest parental education and income
HDAS, Healthy Dietary Adherence Score; OR, Odds ratio; SDQ, Strengths and Difficulties Questionnaire

By dividing the HDAS into quartiles of healthy diet adherence, we identified a monotonic trend in the odds of experiencing better well-being for all well-being indicators (Table 6). In comparison to children with the lowest adherence to the HDAS-defined healthy diet, those with the highest adherence had a 1.2 times higher odds ratio of reporting better self-esteem, 1.4 times higher odds ratio of reporting fewer emotional problems and 1.3 times higher odds ratio of reporting good peer relations.

Table 6. *Analysis of the dose-response relationship between baseline adherence to the HDAS (divided into quartiles) and indicators of psychosocial well-being two years later (N=7,196)*

	KINDL®				SDQ			
	Better self-esteem		Good parent relations		No detectable emotional problems		No detectable peer problems	
HDAS[†]	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Q4 (>28)	1.2	1.0,1.5	1.2	1.0,1.5	1.4	1.2,1.6	1.3	1.1,1.6
Q3 (21-27)	1.2	1.0,1.4	1.1	0.9,1.3	1.3	1.1,1.5	1.3	1.1,1.4
Q2 (16-20)	0.9	0.8,1.1	1.1	0.9,1.3	1.2	1.1,1.4	1.0	0.9,1.2
Q1 (<15)	reference		reference		reference		reference	

OR and CI obtained using a multilevel model correcting for cluster design (country)
Model adjusted for: age, sex, BMI z-score, well-being at baseline, highest parental education and income
[†]HDAS ranges from 0-49
HDAS, Healthy Dietary Adherence Score; OR, Odds ratio; SDQ, Strengths and Difficulties Questionnaire

Among children with normal weight (n=5,597), higher adherence to the HDAS-defined healthy diet at T_0 was associated with fewer emotional and peer problems two years later (OR=1.2; 95% CI=1.1,1.4 and OR=1.3; 95% CI=1.1,1.4, respectively). When it came to the overweight group (n=1,599), higher adherence to the healthy diet was associated with fewer peer problems (OR=1.4; 95% CI=1.1,1.7). Furthermore, in boys, significant associations were found between higher adherence to the HDAS-defined healthy diet and fewer emotional (OR=1.2; 95% CI=1.1,1.4; p=0.007) and peer problems (OR=1.2; 95% CI=1.0,1.4; p=0.009) while higher adherence to the healthy diet in girls was significantly associated with better self-esteem (OR=1.3; 95% CI=1.0,1.5; p=0.027) and fewer peer problems (OR=1.3; 95% CI=1.2,1.6; p<0.001) (not reported in Paper II).

Baseline well-being as a predictor of diet at follow-up

Conversely, better self-esteem at T_0 was significantly associated with higher adherence to the healthy diet, according to the HDAS, two years later (OR=1.1; 95% CI=1.0,1.3) (Table 7). Additional analysis of the components included in the HDAS revealed positive associations between baseline indicators of well-being and consumption of fruit and vegetables, sugar and fat, in accordance with dietary guidelines, two years later.

Table 7. *Prospective associations between baseline indicators of well-being and adherence to the HDAS two years later (N=7,196).*

		HDAS (higher adherence)	
		OR	95% CI
KINDL®	Better self-esteem	1.1*	1.0,1.3
	Good parent relations	1.0	0.9,1.1
SDQ	No detectable emotional problems	1.1	1.0,1.3
	No detectable peer problems	1.0	0.9,1.1

*p<0.05
P-value obtained using a multilevel model correcting for cluster design (country)
Model adjusted for: age, sex, BMI z-score, diet at baseline, highest parental education and income
HDAS, Healthy Dietary Adherence Score; OR, Odds ratio; SDQ, Strengths and Difficulties Questionnaire

Stratification by weight group failed to reveal significant associations between baseline diet and well-being at follow-up, either in the normal-weight or overweight group. Moreover, among boys, no significant associations between well-being at T_0 and adherence to the HDAS-defined healthy diet at T_1 could be found. However, among girls, a positive association was identified between fewer emotional problems at T_0 and higher adherence to the healthy diet two years later (OR=1.3; 95% CI=1.0,1.6; p=0.026) (not reported in Paper II).

4.4 Parental feeding practices and children's BMI trajectories

Parental feeding practices at IDEFICS baseline

The BMI trajectories from IDEFICS baseline (T_0/T_1) to I.Family could be calculated for 5,542 children. In this sample, half of the parents reported, at T_0/T_1, concern about their child becoming overweight. Specifically, 76% of the parents of children who already were overweight reported this concern, while 37% of the parents of children who were normal- weight reported this same concern. Regarding PFPs at IDEFICS baseline, a majority of the parents reported sitting with the child during meal time (Table 8). However, approximately half of the children watched TV while eating, with the highest proportions reported in Italy (76%) and Cyprus (73%). Seventeen percent of the parents reported concern that their child was eating too much, to the highest extent in Italy (30%), and an equal proportion (16%) reported stopping their child from eating too much, ranging from 8% in Sweden to 23% in Italy and Cyprus. Furthermore, 12% of the parents had considered putting their child on a diet (most frequently reported by parents in Italy and Cyprus). Overall, 39% of the parents reported that they made their child eat all the food on his/her plate, with the highest proportions reported in Belgium (73%) and Spain (70%). Finally, around 40% of the parents reported using favorite foods to get their child to eat disliked healthy foods; this was most common in Italy (70%) and Belgium (52%) and least common in Hungary (16%) and Germany (23%). (Country-specific results are not presented in Paper III).

Table 8. *Prevalence proportions of parental feeding practices at IDEFICS baseline*

	n	%
Never or rarely sits down with child during meal time	274	5
Child often watches TV while eating	2,757	53
Parents stop child from eating too much	827	16
Parents consider to put child on a diet to avoid overweight	627	12
Parents make child eat all the food on his/her plate	2,060	39
Parents worry that child eats too much	900	17
Parents use favorite food to get child to eat disliked healthy foods	2,186	42

Social vulnerability groups and parental feeding practices

Information on social vulnerability indicators was collected from a sub-sample of 3,809 children. In this group, 47% of the parents reported no vulnerability indicators, 27% reported one vulnerability indicator and 26% reported 2 or more vulnerability indicators. PFPs were compared between the three social vulnerability groups, demonstrating that sitting with the child during mealtime was more common in the non-vulnerable group. However, compared to the groups reporting 1 or more social vulnerability indicator, reports that ‘child often watches TV while eating’, ‘parents make the child eat all the food on the plate’ and ‘parents use favorite food to get child to eat disliked healthy foods’ were also more common in the non-vulnerable group.

BMI trajectories from IDEFICS baseline to I.Family

Approximately 40% of the children remained normal-weight, while 31.5% underwent a major weight gain (Table 9). Almost 19% remained overweight and only 9.5% underwent major weight loss. During the follow-up period, similar percentages of girls and boys were categorized as having undergone major weight gain (30%) and major weight loss (10%). Moreover, in children with one or more reported social vulnerabilities, 30-33% underwent major weight gain while the corresponding number in children with no social vulnerabilities was 26%.

Table 9. *Characteristics of children belonging to the four BMI trajectory groups at I.Family*

	Remained normal weight[1]	Major weight gain[2]	Remained overweigh[3]	Major weight loss[4]
Subjects, n	2,210	1,746	1,041	545
BMI z-score (SD) at IDEFICS (T_0/T_1)	0.0 (0.5)	0.2 (0.7)	1.8 (0.6)	2.0 (0.9)
BMI z-score (SD) at I.Family	0.0 (0.6)	1.3 (0.8)	1.7 (0.6)	1.1 (1.0)
Age (SD)	11.7 (1.9)	11.1 (1.9)	12.2 (1.7)	12.1 (1.9)
Boys (%)	39	33	18	9
Girls (%)	41	30	19	10
Social vulnerability group[Δ]				
2 or more vulnerabilities (%)	31	33	24	12
1 vulnerability (%)	41	30	19	10
Non-vulnerable (%)	51	26	15	8

[1]BMI z-score between -1 and 1 at IDEFICS baseline and I.Family, and did not change more than ±0.1 in BMI z-score per year
[2]BMI z-score of more than -1 at IDEFICS baseline and gained more than +0.1 in BMI z-score per year
[3]BMI z-score of more than 1 at IDEFICS baseline and I.Family, and did not change more than ±0.1 in BMI z-score per year
[4]BMI z-score of more than 1 at IDEFICS baseline and reduced more than -0.1 in BMI z-score per year during the time of follow-up
[Δ]A total vulnerability score was calculated by adding up the scores (1 vs 0) of the six vulnerability indicators (low social network, single-parent family, migrant background, unemployed, low-income and low-education). Total vulnerability score ranges from 0 (the child has none of the six vulnerability indicators) to 6 (the child has all six vulnerability indicators)
BMI: body mass index, SD: standard deviation

Parental feeding practices and the development of childhood overweight

Twenty-two percent of the variation in children's BMI z-score at I.Family could be explained by the combined PFPs reported by the parents at IDEFICS baseline. The PFPs that contributed most to the variation in children's BMI z-score were: 'stop child from eating too much', 'consider putting the child on a diet to avoid overweight', and 'worry that child eats too much'.

The relationship between baseline PFPs and children's BMI trajectory was further investigated using multilevel regression analyses. It was found that, among children who were normal-weight at IDEFICS baseline, the PFPs previously shown to contribute most to the variation in BMI z-score were in fact associated with higher odds of major weight gain during the duration of follow-up: 'stop child from eating too much' (OR=2.1; 95% CI=1.5,2.9), 'consider putting child on a diet to avoid overweight' (OR=2.5; 95% CI=1.5,4.1), and 'worry that child eats too much' (OR=1.5; 95% CI=1.1,2.1) (Table 10).

Table 10. *Parental feeding practices at IDEFICS baseline and the odds ratio for major weight gain (ref. remained normal weight[2]) at I.Family*

Parental feeding practices[1]	n	Major weight gain[3]	
		OR	95% CI
Never or rarely sits down with child during meal time	2,917	1.1	(0.7,1.7)
Child often watches TV while eating	2,919	1.1	(0.9,1.3)
Parents stop child from eating too much	2,920	2.1***	(1.5,2.9)
Parents consider to put child on a diet to avoid overweight	2,917	2.5***	(1.5,4.1)
Parents make child eat all the food on his/her plate	2,915	0.9	(0.8,1.1)
Parents worry that child eats too much	2,910	1.5**	(1.1,2.1)
Parents use favorite food to get child to eat disliked healthy foods	2,912	1.0	(0.8,1.2)

**p<0.01, ***p<0.001 P-value obtained using a multilevel model correcting for cluster design (study center and school).
OR: odds ratio; CI: confidence interval
[1]Dichotomized into yes vs. no, with no as the reference
[2]BMI z-score between -1 and 1 at IDEFICS baseline and I.Family, and did not change more than ±0.1 in BMI z-score per year
[3]BMI z-score of more than -1 at IDEFICS baseline and gained more than +0.1 in BMI z-score per year
Models adjusted for: sex of the child, age, baseline BMI z-score, parental education, intervention/control region, time spent in sedentary behavior (h/day), parental BMI and parental concern about child becoming overweight (yes/no)

After exclusion of children referred to as “normal-weight at baseline” but with a BMI z-score above 1.0 (291 children, 7%), the association between ‘parents worry that child eats too much’ and higher odds of major weight gain was attenuated and no longer significant (OR=1.1; 95% CI=0.8,1.6). It should also be mentioned that after exclusion of children with a BMI z-score above 1.0 at baseline, the interquartile range (IQR) for baseline BMI z-score was -0.5 to 0.4 in the group undergoing major weight gain.

Next, stratified analyses by social vulnerability group identified significant associations between the PFP ‘stop child from eating too much’ and higher odds of major weight gain in all three vulnerability groups (Table 11). Moreover, compared to the non-vulnerable group, the effect estimates were somewhat higher among children with 1 or more vulnerability indicator. However, overlap between CIs indicated that the difference was not significant.

Table 11. *Parental feeding practices at IDEFICS baseline and the odds ratio for major weight gain (ref. remained normal weight[3]) at I.Family, stratified by social vulnerability groups*

Vulnerability group[1]	Parental feeding practices[2]	n	Major weight gain[4]	
			OR	95% CI
2 or more vulnerabilities	Parents stop child from eating too much	496	2.7*	(1.1,6.7)
	Parents consider to put child on a diet to avoid overweight	497	3.2	(0.4,26.3)
	Parents worry that child eats too much	497	1.5	(0.7,3.2)
	Parents use favorite food to get child to eat disliked healthy foods	496	1.0	(0.7,1.5)
1 vulnerability	Parents stop child from eating too much	535	2.6*	(1.1,6.2)
	Parents consider to put child on a diet to avoid overweight	536	1.3	(0.5,3.2)
	Parents worry that child eats too much	536	1.5	(0.7,3.3)
	Parents use favorite food to get child to eat disliked healthy foods	534	0.9	(0.6,1.3)
Non-vulnerable	Parents stop child from eating too much	972	2.1*	(1.1,3.9)
	Parents consider to put child on a diet to avoid overweight	972	1.5	(0.5,4.7)
	Parents worry that child eats too much	972	1.2	(0.7,2.2)
	Parents use favorite food to get child to eat disliked healthy foods	972	1.1	(0.8,1.5)

*p<0.05 P-value obtained using a multilevel model correcting for cluster design (study center and school)
OR: odds ratio; CI: confidence interval
[1]A total vulnerability score was calculated by adding up the scores (1 vs 0) of the six vulnerability indicators (low social network, single-parent family, migrant background, unemployed, low-income and low-education). Total vulnerability score ranges from 0 (the child has none of the six vulnerability indicators) to six (the child has all six vulnerability indicators).
[2]Dichotomized into yes vs. no, with no as the reference
[3]BMI z-score between -1 and 1 at IDEFICS baseline and I.Family, and did not change more than ±0.1 in BMI z-score per year
[4]BMI z-score of more than -1 at IDEFICS baseline and gained more than +0.1 in BMI z-score per year
Models adjusted for: sex of the child, age, baseline BMI z-score, time spent in sedentary behavior (h/day), parental BMI, intervention/control region and parental concern about child becoming overweight

Among children who were overweight at baseline, only weak associations were observed between higher odds of remaining overweight for the PFPs ‘parents never or rarely sit down with child during meal time’ (OR=1.3; 95% CI=0.8,2.2), ‘child watches TV while eating’ (OR=1.2; 95% CI=0.9,1.6), and ‘parents use favorite food to get child to eat disliked healthy food’ (OR=1.2; 95% CI=0.9,1.6). Moreover, stratification by social vulnerability failed to reveal any significant associations between PFPs and the odds of remaining overweight in any of the social vulnerability groups (results not shown).

4.5 Evaluation of the diet intervention in IDEFICS

In order to evaluate the sustainability of the diet intervention in the IDEFICS study we investigated differences in diet quality (as measured by propensity for consuming fat, sugar, water and fruit and vegetables) between intervention and control families in I.Family. In total, 4,691 families (i.e. at least one parent and one child), consisting of 7,739 index children and siblings and 6,631 parents, participated in I.Family. Of these, 54% had at least one child exposed to the intervention. The number of participating families in each country ranged from 946 in Italy to 131 in Belgium. On the country level, significant differences in diet propensity ratios between the intervention and control groups were found in Cyprus (all propensity ratios) and Germany (sugar and water propensity ratios). Moreover, there were no significant differences in the opposite direction (i.e. poorer diet quality in the intervention group) in any country.

On the family level, the reported propensity for consuming sugar was found to be lower in the intervention families than in the control families, and the former reported higher propensity to consume water over other beverages (Table 12). Although the difference was not significant, the propensity for consuming fat was marginally lower in the intervention families, while the propensity for consuming fruit and vegetables was similar in the control and intervention families (not shown elsewhere). Moreover, when the propensity ratios in parents and children were analyzed separately, the dietary quality differences related to the intervention remained in both groups (Table 12).
Using parental education as a proxy for SEP, the impact of SEP on the sustainability of the diet intervention was explored in stratified analyses. The differences in diet quality between intervention and control families were similar in families with high and low education, although it was only among the highly educated families that these differences were significant (Table 13).

Table 12. *Fat, sugar and water propensity ratios in intervention and control families, and parents and children separately*

	Families				
Propensity ratio (%)	Intervention group n=2,424		Control group n=2,034		
	Mean ± SE	95% CI	Mean ± SE	95% CI	P-value
Fat	25.1 ± 0.1	24.8, 25.4	25.5 ± 0.2	25.2, 25.8	0.073
Sugar	19.8 ± 0.2	19.3, 20.3	20.7 ± 0.2	20.3, 21.2	0.004
Water	47.3 ± 0.5	46.4, 48.2	46.0 ± 0.5	45.1, 46.9	0.048
	Parents				
	Intervention group n=3,501		Control group n=2,832		
	Mean ± SE	95% CI	Mean ± SE	95% CI	P-value
Fat	22.7 ± 0.2	22.3, 23.0	23.1 ± 0.2	22.7, 23.5	0.110
Sugar	17.2 ± 0.3	16.7, 17.7	18.2 ± 0.3	17.6, 18.7	0.013
Water	43.2 ± 0.4	42.5, 43.9	41.9 ± 0.4	41.1, 42.6	0.014
	Children				
	Intervention group n=3,870		Control group n=3,325		
	Mean ± SE	95% CI	Mean ± SE	95% CI	P-value
Fat	27.4 ± 0.2	27.0, 27.7	27.8 ± 0.2	27.4, 28.2	0.072
Sugar	22.2 ± 0.3	21.7, 22.7	23.3 ± 0.3	22.7, 23.8	0.006
Water	51.8 ± 0.6	50.7, 52.9	49.9 ± 0.8	48.4, 51.5	0.045

Families with at least 1 parent and 1 child are included
P-value obtained using a multilevel model correcting for cluster design (center)
Models adjusted for: country, mean age of the parents and mean age of the children, percent females in the family, percent overweight and obese subjects in the family, highest parental education and household size

Table 13. *Fat, sugar and water propensity ratios in intervention and control families, and parents and children separately*

	Highly educated families				
	Intervention group n=1,278		Control group n=1,052		
Propensity ratio (%)	Mean ± SE	95% CI	Mean ± SE	95% CI	P-value
Fat	25.0 ± 0.2	24.6, 25.3	25.1 ± 0.2	24.7, 25.5	0.680
Sugar	18.4 ± 0.2	18.1, 18.8	19.4 ± 0.2	18.9, 19.8	0.003
Water	47.9 ± 0.4	47.1, 48.7	46.4 ± 0.5	45.5, 47.2	0.013
	Lower educated families				
	Intervention group n=916		Control group n=860		
	Mean ± SE	95% CI	Mean ± SE	95% CI	P-value
Fat	25.5 ± 0.2	25.0, 26.0	26.1 ± 0.3	25.6, 26.6	0.070
Sugar	21.5 ± 0.4	20.8, 22.2	22.4 ± 0.4	21.6, 23.1	0.090
Water	46.8 ± 0.6	45.6, 48.1	45.4 ± 0.8	43.9, 47.0	0.170

Families with at least 1 parent and 1 child are included
P-value obtained using a multilevel model correcting for cluster design (center)
Models adjusted for: country, mean age of the parents and mean age of the children, percent females in the family, percent overweight and obese subjects in the family, and household size

An attempt was made later to harmonize the FFQs, in order to allow longitudinal analysis of diet quality. Changes in propensity for consuming sugar, fat and water were investigated in the 4,914 index children that participated in I.Family, using multilevel linear regression and controlling for country, age, sex, overweight at baseline, highest parental education, household size and the respective baseline propensity ratio for sugar/fat/water. The mean propensity for consuming fat was shown to be increased in both the intervention (4.3±0.4 SE; 95% CI=3.8,4.8)) and control group (4.5±0.4 SE; 95% CI=4.0,5.0) while the mean propensity for consuming sugar decreased in both groups (-2.9±0.5 SE; 95% CI=-3.5,-2.3 and -2.7±0.5 SE; 95% CI=-3.3,-2.1, respectively). The mean propensity for consuming water increased in both the intervention (0.9±1.0; 95% CI=-1.0,2.9) and control group (0.5±1.0; 95% CI=-1.4,2.3). However, no significant differences between the intervention and control groups regarding change from IDEFICS baseline to I.Family baseline could be detected for any of the propensity ratios and we hence failed to confirm dietary quality differences by intervention exposure longitudinally in index children.

5 Discussion

5.1 Mutans streptococci and BMI

In a sub-sample of children from the Swedish IDEFICS cohort we identified a positive association between BMI z-score and salivary MS counts that, to our knowledge, has only been observed in adult populations previously [192-195]. High salivary MS counts is a risk factor for dental caries [48-52]; however, the actual development of dental caries is also dependent on environmental factors such as frequent and high intake of fermentable carbohydrates, sugars in particular [47, 196], according to the ecological plaque hypothesis [44]. Both higher meal frequency and intake of sugar-rich foods are related to increased colonization with cariogenic microorganisms [40, 41, 46, 197] and are likely to contribute to higher energy intake and subsequent weight gain [198-202]. Although we found positive associations between both meal frequency and propensity for consuming sugar and counts of MS in saliva, this could not explain the positive association between BMI z-score and MS counts.

To our knowledge the association between sleep duration and salivary MS counts was for the first time studied in this thesis, while the association between shorter sleep duration and higher risk of developing obesity has been demonstrated. Among children less than 10.5 hours of sleep daily is associated with higher odds of being overweight or obese, one-hour decrease in sleep duration is a risk factor for developing overweight and obesity [203]. It has been found that the impact of sleep deprivation on obesity development is greater in children as compared to adults [204] and that several potential pathways may be involved, such as hormonal imbalance and subclinical inflammation [204]. Findings from experimental studies indicate that sleep deprivation result in decreased levels of the appetite stimulating hormone leptin, which could stimulate food intake [205]. A recent review on sleep and eating habits in adults reports associations between insufficient sleep (<6 hours) and more frequent snacking specifically snacks high in energy but low in nutrients [206]. Hence, it could be speculated that adequate sleep duration could be protective against both overweight and colonization with MS.

However, the positive association observed between children's BMI z-score and their salivary MS counts could not be explained either by meal frequency, sugar propensity or sleep duration. This could be due to difficulties in capturing and sufficiently adjusting for eating and sleep habits, but it is also possible that the association between BMI z-score and MS counts in saliva is driven by other factors not accounted for in this study.

Given the shared risk factors for overweight and dental caries, it is possible that limiting the intake of sugar to <10E%, limiting the intake of snack foods that are high in energy but low in nutrients, and adhering to the general sleep recommendation of 9-12 hours [207], could promote a healthy BMI trajectory as well as preventing colonization with MS and future development of dental caries in children. In order to achieve this it would be of great importance if the general dentistry and child health services had the opportunity to collaborate as both these institutions are to some extent working toward the same goal – improving public health by encouraging healthy eating habits.

5.2 Diet and psychosocial well-being

Previous studies in children and adolescents have indicated a relationship between unhealthy diets and poor mental health in children [94, 95], with additional suggestions that healthy diets improve children's mental health [95]. In this study, bi-directional associations were identified between higher adherence to a healthy diet, according to the Healthy Dietary Adherence Score (HDAS), and better self-esteem (an indicator of HRQoL), suggesting a positive reinforcement of a healthier diet on better self-esteem and vice versa. It might be hypothesized that, just as PA improves self-perception and self-esteem in children [208], consuming a healthy diet (socially desirable) could improve self-esteem which might in turn increase the chance of engaging in healthy behaviors.

One general observation in this study was that baseline diet more frequently predicted psychosocial well-being at follow-up than the reverse. In addition to the bi-directional association between diet and self-esteem, we found that higher adherence to the HDAS-defined healthy diet predicted fewer emotional and peer problems (indicators of psychosocial problems) two years later, independently of baseline well-being, weight status, and parental income and education. Moreover, although the association between higher adherence to the healthy diet and fewer peer problems was found in both normal-weight and overweight children, the effect estimates were somewhat higher in children with overweight. Unhealthy eating habits have been associated with weight stigma [209] and children are aware already at young ages (as early as age 3) that a healthy diet is important if you want to lose weight [210]. Hence, this finding might reflect that consuming a healthy diet is less stigmatizing than consuming an unhealthy one, and that this is even more important for good peer relations in children with overweight than for children with normal weight.

Overall, our findings add to the growing body of evidence supporting the inverse association between a healthy diet and mental health problems previously found in adult [89, 90] and adolescent populations [211-213]. Additionally, stratification by sex revealed significant associations between higher adherence to the HDAS-defined healthy diet at baseline and both fewer emotional problems in boys and better self-esteem in girls. Conversely, fewer emotional problems at baseline was associated with higher adherence to the HDAS-defined healthy diet two years later in girls. This contradicts the results from a recent study, in which no protective effect of higher adherence to a healthy dietary pattern on the development of external or internal problems was observed in either boys or girls [93]. Possible explanations for these inconsistent findings could be that the other authors investigated this association in an adolescent sample, in which other factors than diet might be more important for external and internal problems. Moreover, data on indicators of external and internal problems were self-reported and included a dietary pattern that was data-driven rather than an *a priori* dietary pattern.

A recent cross-sectional study of dietary behaviors and indicators of mental well-being in a large sample of adolescents from New Zealand [214] found that depressive symptoms were reported by a third of those with the least healthy diet (lowest quartile), but only by 6% of those with the healthiest diet (highest quartile) [214]. By using quartiles of adherence to the HDAS-defined healthy diet as the exposure, we also identified a monotonic trend entailing a consistent increase for all indicators of well-being associated with higher adherence. Recent evidence suggests an improved effect of healthy dietary patterns on children's mental health [95]. Moreover, although no intervention studies on overall eating habits and mental health have as yet been performed in children to our knowledge, several intervention studies in adult populations have reported a reduction in clinical depression symptoms and anxiety after diet interventions, including improved adherence to nutritional guidelines [91, 92, 215]. However, Kaseva *et al.* found no effect of a repeated dietary and lifestyle intervention on psychological well-being in young adults [216]. The effect of improved diet quality on psychosocial well-being thus requires further investigation.

The potential mechanisms behind the association between diet and mental health are complex and not yet fully known [217, 218]. In a review from 2004, Bellisle *et al.* [217] suggested that improved nutrient composition could have a beneficial effect on mental health among children and adolescents with poor nutritional status. In a later review, Bamber *et al.* [218]

discuss the potential mechanisms in more detail. Concurring with Bellisle *et al.* [217], they suggest that improving a nutrient-poor diet might have positive effects on mental health [218]. Moreover, they discuss n-3 fatty acids, folic acid, and B12, as important key nutrients for mental health due to their importance for central nervous system function, neurotransmitter function, and homocysteine concentrations [218]. While the importance of nutrients for brain development and functioning has been established [219, 220], it must be kept in mind that people do not consume individual nutrients, but rather a variety of foods and beverages. In this study, a higher HDAS score represents a diverse healthy diet rich in fruit and vegetables, whole m, and fish, which might also be a good source of the key nutrients n-3 fatty acids, folic acid, and B12. This might then be one potential explanation for the associations, found in our study, between higher adherence to the HDAS-defined healthy diet and both better self-esteem and fewer emotional and peer problems. Bamber *et al.* [218] further suggests that meal regularity and social interactions during mealtime are potential mechanisms explaining the relationship between diet and mental health, and that these factors could be considered indicators of overall good family functioning (defined by Bamber *et al.* as: the home environment is loving, supportive and promotes the development of family members and ensure that their basic needs are fulfilled) [218]. Recent studies have shown that good family functioning is associated with 'healthy behaviors' such as healthy eating habits [221-223], eating together as a family [223], higher levels of PA [223] and lower BMI [224] in children and adolescents. Hence, our findings of associations between higher adherence to a healthy diet (according to the HDAS), better self-esteem, and fewer emotional and peer problems could also reflect generally better functioning families, which might also promote better psychosocial well-being.

5.3 Parental feeding practices and children's BMI trajectories

Taking the EST [32] into consideration, the family might be considered to be the bridge between the community (the structural environment) and the child, since children's physical and social food environment is mainly shaped by the parents [103, 104]. Our finding that 22% of the variation in children's BMI z-score could be explained by PFPs reported by the parents at baseline aligns with the previous finding that parental pressure to eat and food restriction account for 15% of the variation in BMI z-score in 8-12-year-olds [225]. In that study the authors also reported positive associations between food restriction and children's BMI z-scores and moreover, although not significant, there was an inverse association between food restriction and higher diet quality scores (measured by higher adherence to American dietary guidelines) [225].

Regarding the parent-child interaction described in the family consumer socialization framework for childhood obesity [33], a unidirectional pathway between PFPs and children's BMI trajectory is highly unlikely. Rather, PFPs are a reaction to children's eating habits and perceived weight status [97, 116, 119] also referred to as a 'child-responsive model' [226]. This theory is further supported by studies reporting stronger influence of children's weight and eating habits on PFPs rather than the reverse, regardless of baseline BMI [227, 228]. Moreover, social factors (such as income, education, ethnicity, and culture) impact both parents' perceptions of, and possibility to act on, their child's weight and eating habits [229].

In this study, we investigated the association between PFPs and children's BMI trajectories separately in normal-weight and overweight groups at baseline in order to adjust for the potential effect of children's weight on the PFPs. We found that, among children with normal weight at baseline, the PFP's 'stop child from eating too much', and 'consider putting the child on a diet to avoid overweight' were associated with higher odds of developing overweight. It should be mentioned that 75% of the children in the normal-weight group had a BMI z-score below 0.4. These associations remained significant after adjusting for baseline BMI z-score, sedentary time, parental education and BMI, and parental concern about the child becoming overweight. However, in the overweight group, no significant associations were observed between any of the PFPs reported at baseline and the odds of remaining overweight. Our findings add to the existing literature indicating that restrictive PFPs predict higher BMI and greater increase in BMI in children [116, 119, 226-228]. Moreover, we found that the association between 'stop child from eating too much' and higher odds of the child developing overweight was significant across all social vulnerability groups suggesting that this association is not dependent on social factors. However, it is possible that the influence of PFPs on children's BMI trajectory differ depending on parenting style [107]. For example, restrictive PFPs in combination with an authoritative parenting style (meaning that parents emphasize the reason for the restriction and enforce the restriction in a responsive and respectful way) could in theory have a positive impact on children's BMI trajectory. In contrast, restrictive PFPs in combination with an indulgent or neglectful parenting style (meaning that parents are more permissive or less respectful against their child) could potentially have a negative effect on children's weight development. Unfortunately information on different parenting style was not obtained at IDEFICS baseline and could therefore not be investigated as a potential moderator in this study.

Previous studies have reported an underestimation of weight status in adults with overweight and obesity [230], as well as parents having difficulties perceiving overweight and obesity in their children [230-232]. Indeed, according to a recent meta-analysis, around 50% of parents cannot identify overweight or obesity in their children [232]. Hence, it was interesting to find that a relatively high number of parents of normal-weight children (37%) reported concern about their child becoming overweight. We cannot know as to whether this reported concern is due to the adverse health effects or to the stigma of being overweight [233, 234]. However, parents' concern about their child's current or future overweight has been found to be a mediator for the association between restrictive PFPs and children's weight status [226, 235-237], regardless of the child's actual BMI [235, 236]. Webber *et al.* conclude that "restriction appears to be a consequence of mothers' concern about their child becoming overweight rather than a cause of children's weight gain" [237]. Children's food approach (i.e. food responsiveness/satiety, emotional overeating, and enjoyment of food and beverages) seems to have an indirect effect on food restriction via parents' concern about their child's weight status [235]. Surprisingly considering the child-responsive model mentioned earlier [97, 116, 119, 226], adjusting for parental concern only slightly attenuated the associations between the PFPs 'stop child from eating too much', 'consider putting the child on a diet to avoid overweight' and higher odds of the child developing overweight. Additionally, adding adherence to healthy dietary guidelines to the fully adjusted model did not affect the associations found between these PFPs and children's BMI trajectory (results not further shown). However, it is likely that children's diet moderates the effect of parental feeding practices and children's BMI trajectory, and future studies should therefore include indicators of unhealthy eating habits in order to investigate this further.

Although we cannot determine all the underlying reasons for applying the PFPs 'stop child from eating too much' and 'consider putting the child on a diet to avoid overweight', or how these PFPs were expressed by the parents and experienced by the children, our results align with previous findings that restrictive PFPs are counterproductive weight control behaviors. Moreover, based on our findings among children with overweight at baseline, they are not associated with weight reduction either. It has been suggested that this ineffectiveness is due to increased preference for and excess intake of the restricted foods and beverages [238-241] and that controlling feeding practices override internal satiety cues and children's ability to self-regulate their food intake [238, 239, 241].

Paradoxically, at the same time as parental perception of children's overweight and obesity must be improved, many parents, independent of their children's weight status, are concerned about them becoming overweight. Based on our findings, we can conclude that restrictive PFPs (also referred to as coercive control or 'parent-centered' [123]) are not helpful in preventing the development of overweight in children. Future research should focus on identifying which other PFPs that are influential in improving children's eating habits and preventing future overweight, and whether the influence of PFPs on children's BMI trajectory differs depending on parenting style.

5.4 Evaluation of the diet intervention in IDEFICS

Previous findings from the IDEFICS study have suggested that the intervention was unsuccessful in that it neither reversed the increase in childhood overweight [242] nor had any effect on dietary intake or activity behaviors [243]. For example, at the 2-year follow-up no intervention effect on eating habits (intake frequency of water, soft drinks, fruit juice, fruit and vegetables) was found on the country level or after analyzing boys and girls separately [243]. One of the goals of the IDEFICS study was to implement the intervention so that it would be sustained in the community, schools and families without support from the local intervention team. It was thus important to investigate possible effects of the intervention on diet quality in children and their families five years after the IDEFICS intervention.

We explored diet quality in the families using propensity ratios for consuming fat, sugar, water, and fruit and vegetables. Cross-sectional analyses of 4,691 families at I.Family identified an approximately 1% lower propensity for consuming sugar as well as a 1% higher propensity for consuming water over other beverages in the intervention group, both on the family level and when parents and children were analyzed separately. No significant difference between the intervention and control families could be found concerning propensity for consuming foods rich in fat or propensity for consuming fruit and vegetables rather than other foods and beverages. Additionally, stratification by parental education revealed that the lower propensity for consuming sugar and higher propensity for consuming water among intervention families were significant only among the highly educated families. However, also in the lower educated families the intervention group had a somewhat lower propensity for consuming sugar and higher propensity for consuming water as compared to the control group. It is possible that the small number of families in the lower educated group as compared to the highly educated group resulted in a loss of power to detect a difference between the intervention and control families. Nevertheless, it is well-known that families with low SEP generally make less healthy food choices than high-SEP families due to several reasons, such as lack of experience in preparing healthy foods, the higher cost of healthy foods, and/or low availability of and access to healthy foods [244]. It is important that interventions aiming at improving eating habits do not increase this gap further. However, it seems that interventions focusing on behavioral change on an individual level (such as dietary counseling and health education) are more likely to increase socioeconomic inequalities in healthy eating while interventions focusing on availability and access to healthy foods are more likely to have the reverse effect [245].

The somewhat lower propensity for consuming sugar and higher propensity for consuming water observed cross-sectionally in intervention families may suggest that the diet quality of parents and siblings were influenced by the intervention. Similar findings have been reported from the Shape Up Summerville (SUS) study [246] and the Health In Adolescents (HEIA) study [247]. In the SUS study they found decreases in BMI among parents in the intervention group and suggested that the effect of the school-centered community intervention spilled over to the parents [246]. The HEIA study investigated the effect of a school-based intervention among 11-to-13-year-old children and their parents and reported higher intake of fruit among mothers in the intervention group (albeit only borderline significant) and higher intake of vegetables among highly educated fathers in the intervention group [247]. However, contrary to our findings, both the SUS study and the HEIA study reported prospective beneficial effects of the intervention on children's intake of fruit [247] and sugar-sweetened beverages [247, 248].

After an attempt to harmonize the FFQ used in I.Family with the original FFQ used in IDEFICS, longitudinal analyses among index children participating in the five year follow-up showed no differences between the intervention and control group regarding changes over time for any of the propensity ratios. We hence failed to confirm an effect of the intervention on diet quality in index-children. There might have been a spill-over to parents and siblings, but considering the previous findings of no intervention effect on either BMI, eating habits or activity behaviors [242, 243] and the minor differences found between the intervention and control families regarding propensity for consuming sugar and water in our cross-sectional analysis we could not demonstrate that the key messages regarding diet were sustained.

Similar to our findings of no effect of the IDEFICS intervention on diet quality the Copenhagen School Child Intervention Study (CoSCIS) failed to detect any differences in dietary intake change for either protein, fat, carbohydrate, sugar or fiber intake between the intervention and control group 3 years after the intervention [249]. In CoSCIS, the school-based intervention targeting diet included health education added to the curriculum, additional hours of physical education focusing on diet, establishment of school canteens selling healthy meals and snacks, and parental involvement consisting of newsletters from the school regarding healthy diet [249]. In contrast, the previously mentioned SUS community intervention reduced the consumption of sugar sweetened beverages by 3.5 dl per week in the intervention group, although no impact was found for the intake of fruit and vegetables [248]. Similar to the IDEFICS intervention, the SUS community intervention included an extensive school intervention as well as parental education involving, for instance, newsletters, nutrition forums, and family events [248]. Moreover, the SUS was more extensive than the IDEFICS intervention in that it involved, for example, restaurants in offering healthier food options and local physicians were trained in approaching and counseling parents regarding childhood overweight and obesity [248].

The IDEFICS intervention provided information about healthy food options (emphasizing the importance of water consumption, and the health benefits of an increased intake of fruit and vegetables) and aimed to increase access to these items. The key messages were structured and a study manual was provided in order to standardize not only methods used but also the implementation and sustainability of the intervention. However, as already mentioned by Baranowski *et al.* [250], due to cultural or societal differences country-specific adaptation was allowed in the implementation of the key messages and not all adaptations were documented. Moreover, we have limited information on how the intervention communities have continued their work after the ending of the study. Although, no country specific effects could be found for the key messages on water consumption, and intake of fruit and vegetables in index children at the 2 year follow-up [243] we observed significant differences between the intervention and control families for all indicators of diet quality (lower fat and sugar propensity while higher propensity for consuming water) in Cyprus and higher propensity for consuming water among the intervention families in Germany. This was however not reflected by any beneficial effect on indicators of body composition neither in the total sample nor on a country level [242].

The IDEFICS study included several components known to be important in the development of childhood obesity and the study did take into account the extensive context in which childhood obesity is developed [32, 136]. It targeted both environmental and individual factors via family, schools and community [251]. However, when the parents were asked how often they; heard about the IDEFICS study; noticed efforts related to the key messages;

or received material related to these, it was found that there was a lower than intended exposure to the intervention among a majority of the parents (90%) [252]. Moreover, parents reported that they received the intervention messages through the schools rather than the community [252]. A recently published review concluded that interventions in the home environment including parental role modeling can increase water intake and reduced intake of sugar sweetened beverages in children [253]. Parental components such as increased experience, skills and awareness of healthy eating and activity habits as well as role modeling and parent-child interactions regarding these behaviors, seem to be effective in managing children's weight [254]. However, the evidence is inconsistent regarding the additional effect of these parental components in school-based interventions [254] perhaps due to the difficulties in engaging parents [255, 256]. Nevertheless, in a meta-analysis of RCT studies, Sobol-Goldberg *et al.* [257] found long-term interventions (1-4 years) including a combination of diet and activity components as well as parental support and involvement to be important factors for successful school-based obesity prevention programs [257]. Moreover, focus group research shows that parents in Europe want to take an active part in school-based interventions and to be involved in energy balance-related activities at school or at home together with their children [255]. They do, however, prefer the activities to focus solely on the child and not be expensive, educating or theoretical [255]. Similar findings are reported from focus groups in the IDEFICS study [256]. However, both these studies have an underrepresentation of fathers and parents with low SEP [255, 256]. It is important to acknowledge that these findings are based on reports from parents that already are involved in a study and represents a group of parents that want/have the ability to participate.

The intervention exposure in IDEFICS was low [252] and the parents, the important bridge between the community and the child, were not directly involved but rather informed via newsletters and homework, not shown to be successful in previous studies [254]. These might be reasons for the small intervention effect on diet quality found cross-sectionally in the families and the failure to find any effect in the exposed index-children longitudinally. Since the ecological environment of a child includes the family and the school which are in turn situated in the community interactions within and among these social contexts results in changes within, and influence the development of, the individual child [96]. Hence, without sufficient changes in environmental factors to support the parents in improving children's eating behaviors the effects are likely to be minor and only temporary [134, 135, 137, 258, 259]. The home, school and community are all important settings that must be involved in supporting healthy choices and interventions aimed at preventing overweight via healthy eating- and activity behaviors so that children can benefit from a multi-level approach [125, 260].

5.5 Conceptual model

Figure 7 includes a conceptual model of the findings presented in this thesis and how they are related. Children's diet was investigated both as an outcome and an exposure. Although cross-sectional findings suggested better diet quality among intervention families (moderated by parental education) no effect of the IDEFICS intervention on diet quality could be confirmed in index-children longitudinally. Cross-sectional findings were also observed between higher MS count in children's saliva and higher BMI z-score, higher propensity for consuming sugar, and more frequent consumption, while sleep duration was inversely related to higher MS count.

A healthy diet, as measured by adherence to the HDAS, was associated with better self-esteem, and fewer emotional and peer problems two years later. Conversely, better self-esteem was associated with a healthier diet two years later. The associations between diet and well-being were moderated by children's sex and weight status. Moreover, restrictive parental feeding practices ('stop child from eating too much' and 'consider putting child on a diet') were associated with major weight gain in children independent of social vulnerability. However, there might be a potential moderating effect of children's diet on these associations that we failed to capture.

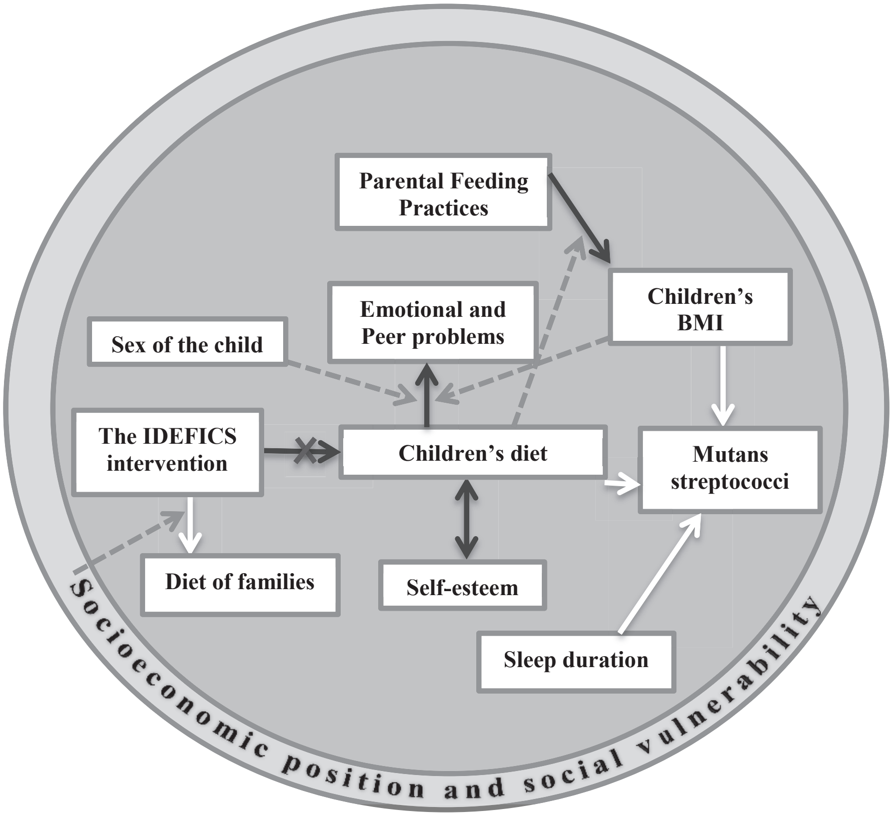

Figure 7. Conceptual model including a summary of the findings in this thesis. The black arrows represent prospective associations while the white arrows represent cross-sectional associations. The crossed arrow means that no association was found. Dashed arrows suggest potential moderating factors.

5.6 Methodological considerations

Study population

The findings in this thesis are derived from a large cohort of children from eight European countries. However, generalizability of these findings to the general population might be limited due to the fact that the sampling was not population based and similar to other epidemiological studies [139, 140] the participation rate was around 50% with a decreasing attrition rate of 68% at T_1 and 55% at I.Family. Moreover, it should be noted that this cohort of children were more likely to have normal weight, good well-being and report more advantageous social and demographic background factors as compared to non-participants and children who did not participate in the follow-up [141, 144] which could affect prevalence rates and introduce bias in the observed associations.

Dietary assessment method

In IDEFICS and I.Family dietary data were obtained both via FFQ and 24 hour diet recall (24-HDR). Both these methods capture dietary intake retrospectively, hence they do not influence eating habits but rely on memory [261]. The 24-HDR is relatively accurate (also for children) if trained personnel perform the interview(s), but a single 24-HDR is not representative of habitual diet. In contrast, FFQ cannot capture all foods consumed and usually does not estimate quantity [261]. However, FFQ measures long-term dietary patterns and has the possibility to rank individuals as higher or lower consumers. Moreover, FFQ has a low burden on the participants and is relatively time- and cost effective as compared to the 24-HDR, making it the most frequently used assessment tool in large epidemiological studies [261]. However, in children below the age of 9 parental reports are needed for both 24-HDR and FFQ [261].

In this thesis information on dietary intake was mainly obtained from the 43-item FFQ used in IDEFICS and the modified 59-item FFQ used in I.Family. The reason for using the FFQ rather than 24-HDR was due to concerns regarding sample size. Dietary data from the FFQ could be obtained from approximately 90% of the children at all three time-points, and from 90% of the parents at I.Family [262]. In contrast, at T_0 70% of the children reported one or more 24-HDR and at T_1 only 40% reported one or more 24-HDR (corresponding numbers for ≥2 24-HDR was 20% and 9.5%). In I.Family one or more 24-HDR was obtained from 53% of the children and 40% of the parents (corresponding numbers for ≥2 24-HDR was 40% and 30%) [262]. By using dietary data obtained from the FFQ we did not have to reduce our sample size, and although we are aware that the information from FFQ could not assess detailed information on intake of nutrients it functioned as a marker for diet quality in this thesis.

Paper I

The data included in paper I is cross-sectional therefore no conclusion can be drawn on causality or direction of the association between dietary habits, sleep, BMI z-score and salivary counts of MS. Moreover, child's meal frequency and sleep duration are based on a single 24-HDR reported by the parental proxy. As mentioned above, a single 24-HDR is not representative of habitual diet and it is therefore unlikely that usual meal frequency was captured. Additionally, the reported sleep duration could be affected by the fact that the child participated in the early morning IDEFICS examination and therefore might not reflect the child's usual wake-up time. Repeated 24-HDR and assessment of sleep via e.g. accelerometer over several days might have given a more valid measurement of these covariates and decreased the uncertainties regarding the confounding effect of dietary intake and sleep on the association between BMI z-score and counts of MS.

Paper II

Children's diet and well-being were reported by the parents. Although the KINDL® and the SDQ were developed for parental reports, it must be acknowledged that it may be difficult for parents to accurately determine psychosocial well-being in their children. Parents may not know about, or even understand, problem behaviors in their children. Moreover, since there is stigma around mental health problems it is possible that parental reports of children's well-being were affected by an unwillingness to report poor well-being. However, previous validation studies of the KINDL® and the SDQ found that the proxy-versions of these instruments could distinguish between groups of healthy and chronically ill children [173] and groups with low- and high-risk of mental health problems [186]. For the FFQ used in IDEFICS, validity and reproducibility could be considered modest but sufficient enough to rank children regarding their intake of healthy and unhealthy foods [159, 161, 162]. However, regarding reports of eating habits one has to consider that it is parentally reported and could be affected both by the fact that parents do not always know what the child consumes outside of the home, and by social desirability, willingness to report a healthier diet. Moreover, under-reporting is a common problem, particularly among children with overweight and obesity, both for parental-reported and self-reported dietary intake [263]. Under-reporting of unhealthy foods could potentially bias the effects size of the associations reported in Paper II.

Since we were interested in adherence to healthy dietary guidelines an *a priori* method was used in order to develop the HDAS used in Paper II, rather than using a posteriori diet patterns previously derived from the IDEFICS cohort [264, 265]. However, due to the lack of information on children's consumption away from home, assumptions had to be made regarding intake of fruit, vegetables and fish since the recommendations included specific quantities that could not be captured by the FFQ. Although the HDAS is similar to both the dietary guidelines recommended by the WHO European Region [266] and the dietary recommendations for prevention of depression [267] its validity needs to be further evaluated in future studies, preferably using objective measures of dietary intake such as biomarkers.

Finally, we acknowledge that both the HDAS and the indicators of well-being could have been treated as continuous variables. However, to estimate risk factors and distinguish children with clinically poor psychosocial well-being we decided to use the pre-defined cut-points for these instruments. Due to the bi-directional comparison, it was therefore important to also transform the HDAS into categories for the analyses in order to compare the outcomes.

Paper III

The reported parental feeding practices originated from a questionnaire designed for children aged 2-5 years which does not fully cover the age range of the children participating at IDEFICS baseline. However, the mean age of the sample included in Paper III was 6 years hence the three constructs included from the original questionnaire were considered relevant also in our sample where the children were still controlled by and highly dependent on their parents. However, we cannot know how these PFPs were conveyed to and experienced by the children. It is important to understand how and why parents use these PFPs and how the PFPs are expressed hence there is a need for qualitative studies within this area. Another potential source of error is children's eating habits since these could affect not only PFPs but also weight development. Therefore, children's adherence to healthy dietary guidelines was added to the model in a separate analysis, however this did not notably affect the associations between the PFPs 'stop child from eating too much', 'consider putting child on a diet to avoid overweight' and major weight gain. However, lower adherence to healthy dietary guidelines does not capture the quantity of unhealthy foods consumed which could be an important moderator for the association found between PFPs and children's weight development.

Paper IV

In I.Family data on diet quality (as measured by propensity for consuming fat, sugar, water and fruit and vegetables) was available only from one time-point for parents and siblings; hence no conclusions can be drawn on causality of the association between exposure to the IDEFICS intervention and differences in diet quality between intervention and control families at I.Family. Moreover, the mean difference in reported sugar and water propensity between intervention and control families was around 1% and we acknowledge that a difference of this size may have little practical significance on a population level. Furthermore, we were not able to confirm any effect of the IDEFICS intervention longitudinally on changes in fat, sugar and water propensity between index children in the control and intervention groups. The better diet quality found in intervention families cross-sectionally could be due to report bias since social desirability perhaps is stronger in the intervention families as opposed to the control families. Moreover, it should be noted that diet quality was self-reported by parents and adolescents while parental-reported for children below the age of 12 years.

6 Conclusions

In investigating diets of European children, with focus on BMI, Mutans streptococci, well-being, and the family, the following separate conclusions were made:

About one fifth of the children in a sub-sample from the Swedish IDEFICS cohort had high salivary counts of MS (>10^5 CFU/ml). Higher BMI z-score, more frequent intake of meals and higher propensity for consuming sugar were all independently associated with high MS count. In contrast, an inverse association was found between high MS count and duration of sleep.

Bi-directional associations were found between adherence to a healthy diet, according to the Healthy Dietary Adherence Score (HDAS), and better self-esteem. Additionally, prospective associations were observed between a healthy diet and fewer emotional and peer problems. The association between a healthy diet and fewer emotional problems could only be confirmed in children with normal weight, and sex differences were found where higher adherence to the HDAS was associated with fewer emotional problems in boys and with better self-esteem in girls.

Parental feeding practices reported during IDEFICS explained 22% of the variation in children's BMI z-score at I.Family. PFPs regarding restriction and putting the child on a diet were associated with higher odds of children developing overweight independent of baseline BMI z-score, parental BMI and parental concern about their child becoming overweight. Moreover, this association was stable across social vulnerability groups.

Five years after the IDEFICS intervention families in the intervention communities had a lower propensity for consuming sugar rich-products and higher propensity for consuming water rather than other beverages cross-sectionally. However, the change from IDEFICS baseline to I.Family in propensity for consuming fat, sugar, and water did not differ between index children from the intervention and control communities; hence we could not confirm an intervention effect on diet quality longitudinally.

Overall, this thesis has identified diet as an important determinant for psychosocial well-being and dental health in European children. Moreover, children's diet and weight development has been found to be an outcome difficult to improve in community interventions as well as influence using parental feeding practices.

7 Future perspectives

More studies are needed to investigate the mechanism behind the association between BMI z-score and MS count. However, improving children's eating habits by reducing intake of sugar rich foods and beverages, and limiting the intake frequency (specifically of unhealthy snacks) may provide multiple benefits in preventing both dental caries and the development of childhood obesity. A close collaboration between the general dentistry and child health services could bring on better opportunities to early prevention of the development of both caries and childhood overweight.

Eating habits are important for healthy weight development and there seems to be evidence to suggest that healthy food choices including fruit and vegetables, whole meal foods, fish, and limited intake of refined sugars and saturated fat also could be one pathway to good psychosocial well-being. However, the mechanism behind this association is not yet established. Considering the early life onset and the relatively high prevalence of mental health problems in children and adolescents the importance of healthy food choices should not be overlooked in future studies aiming at preventing mental health problems and improving the overall health of children.

Despite difficulties for parents to identify overweight in their own children they may still be concerned about their child developing overweight. PFPs related to structure and support (such as role modeling, access and availability of healthy foods) rather than coercive control (restriction) might be more helpful in supporting a healthy weight development, although this might differ by parenting styles. In the future, qualitative studies should explore why parents use specific PFPs and how these PFPs are expressed, as well as experienced, by the child. Moreover, future interventions on preventing childhood obesity should focus not only on healthy lifestyle choices but also on empowering parents in creating healthy food environments that support healthy eating behaviors.

The key messages of the IDEFICS intervention were, for the most part, delivered by the schools, and the intervention exposure received by the parents was much lower than expected. Future studies should investigate how parents could be reached and involved more directly in intervention studies aiming at preventing childhood obesity as they have the main influence on children's eating habits. Since there are many potential reasons for unhealthy eating habits (e.g. lack of experience in preparing healthy foods, and cost, availability and access to healthy foods) it is important that community interventions identify which barriers to healthy eating habits exists so that these can be targeted, most likely these barriers will vary depending on community. Moreover, future studies need to find out how to increase participation and engagement among families with lower SEP in intervention studies aimed at preventing childhood obesity.

Finally, it would be valuable to continue monitoring the IDEFICS/I.Family cohort. The youngest index children are approaching adolescence, while the oldest index children are at an age where they are entering college and moving away from home. This is an important time of transition to independence for these children. Following this cohort in to the future could increase our knowledge base on how factors such as parental feeding practices, eating habits, sleep, and well-being in childhood and adolescence influence future eating behaviors and the risk of developing overweight among young adults.

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Louise Arvidsson

References

1. Cole, T.J. and T. Lobstein, *Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity.* Pediatric Obesity, 2012. **7**(4): p. 284-94.
2. Olds, T., et al., *Evidence that the prevalence of childhood overweight is plateauing: data from nine countries.* International Journal of Pediatric Obesity, 2011. **6**(5-6): p. 342-360.
3. Ogden, C.L., et al., *Prevalence of Obesity and Trends in Body Mass Index Among US Children and Adolescents, 1999-2010.* JAMA 2012. **307**(5): p. 483-490.
4. Townsend, N., H. Rutter, and C. Foster, *Evaluating the evidence that the prevalence of childhood overweight is plateauing.* Pediatric Obesity, 2012. **7**(5): p. 343-346.
5. Ahluwalia, N., et al., *Trends in overweight prevalence among 11-, 13-and 15-year-olds in 25 countries in Europe, Canada and USA from 2002 to 2010.* European Journal of Public Health, 2015. **25**: p. 28-32.
6. Ogden, C.L., et al., *Trends in Obesity Prevalence Among Children and Adolescents in the United States, 1988-1994 Through 2013-2014.* JAMA, 2016. **315**(21): p. 2292-2299.
7. Wijnhoven, T.M.A., et al., *WHO European Childhood Obesity Surveillance Initiative 2008: weight, height and body mass index in 6-9-year-old children.* Pediatric Obesity, 2013. **8**(2): p. 79-97.
8. Wijnhoven, T.M.A., et al., *WHO European Childhood Obesity Surveillance Initiative: body mass index and level of overweight among 6-9-year-old children from school year 2007/2008 to school year 2009/2010.* BMC Public Health, 2014. **14**.
9. Ahrens, W., et al., *Prevalence of overweight and obesity in European children below the age of 10.* International Journal of Obesity, 2014. **38**: p. 99-107.
10. Sanders, R.H., et al., *Childhood obesity and its physical and psychological co-morbidities: a systematic review of Australian children and adolescents.* European Journal of Pediatrics, 2015. **174**(6): p. 715-746.
11. Park, M.H., et al., *The impact of childhood obesity on morbidity and mortality in adulthood: a systematic review.* Obesity Reviews, 2012. **13**(11): p. 985-1000.
12. Lloyd, L.J., S.C. Langley-Evans, and S. McMullen, *Childhood obesity and risk of the adult metabolic syndrome: a systematic review.* International Journal of Obesity, 2012. **36**(1): p. 1-11.
13. Gibson, L.Y., et al., *Clustering of psychosocial symptoms in overweight children.* Australian and New Zealand Journal of Psychiatry, 2008. **42**(2): p. 118-125.
14. Hayden, C., et al., *Obesity and dental caries in children: a systematic review and meta-analysis.* Community Dent Oral Epidemiol, 2013. **41**: p. 289-308.

15. Alm, A., *On dental caries and caries-related factors in children and teenagers.* Swed Dent J Suppl, 2008(195): p. 7-63, 1p preceding table of contents.

16. Freedman, D.S., et al., *The relation of childhood BMI to adult adiposity: The Bogalusa Heart Study.* Pediatrics, 2005. **115**(1): p. 22-27.

17. Singh, A.S., et al., *Tracking of childhood overweight into adulthood: a systematic review of the literature.* Obesity Reviews, 2008. **9**(5): p. 474-488.

18. Popkin, B.M., *Global nutrition dynamics: the world is shifting rapidly toward a diet linked with noncommunicable diseases.* American Journal of Clinical Nutrition, 2006. **84**(2): p. 289-298.

19. Popkin, B.M., L.S. Adair, and S.W. Ng, *Global nutrition transition and the pandemic of obesity in developing countries.* Nutrition Reviews, 2012. **70**(1): p. 3-21.

20. Monteiro, C.A., *Nutrition and health. The issue is not food, nor nutrients, so much as processing.* Public Health Nutr, 2009. **12**(5): p. 729-31.

21. Adair, L.S. and B.M. Popkin, *Are child eating patterns being transformed globally?* Obesity Research, 2005. **13**(7): p. 1281-1299.

22. Monteiro, C.A., F.S. Gomes, and G. Cannon, *The snack attack.* Am J Public Health, 2010. **100**(6): p. 975-81.

23. Bellisle, F., *Meals and snacking, diet quality and energy balance.* Physiol Behav, 2014. **134**: p. 38-43.

24. Moreno, L.A., et al., *Nutrition and lifestyle in european adolescents: the HELENA (Healthy Lifestyle in Europe by Nutrition in Adolescence) study.* Adv Nutr, 2014. **5**(5): p. 615-623.

25. Popkin, B.M. and S.J. Nielsen, *The sweetening of the world's diet.* Obesity Research, 2003. **11**(11): p. 1325-1332.

26. WHO, *Guideline: Sugars intake for adults and children*. 2015, World Health Organization: Geneva.

27. Duffey, K.J. and B.M. Popkin, *High-fructose corn syrup: is this what's for dinner?* American Journal of Clinical Nutrition, 2008. **88**(6): p. 1722-1732.

28. Hooper, L., et al., *Effect of reducing total fat intake on body weight: systematic review and meta-analysis of randomised controlled trials and cohort studies.* BMJ, 2012. **345**.

29. Ambrosini, G.L., *Conference on 'Childhood Nutrition and Obesity: Current Status and Future Challenges' Symposium 3: Effects of early nutrition on later health Childhood dietary patterns and later obesity: a review of the evidence.* Proceedings of the Nutrition Society, 2014. **73**(1): p. 137-146.

30. Ambrosini, G.L., et al., *Free Sugars and Total Fat Are Important Characteristics of a Dietary Pattern Associated with Adiposity across Childhood and Adolescence.* Journal of Nutrition, 2016. **146**(4): p. 778-784.

31. Vandenbroeck IP, Goossens J, and C. M., *Foresight Tackling Obesities: future Choices - Obesity System Atlas*. 2007, Government Office for Science, UK Government's Foresight Programme: Reducing obesity: obesity system map.

32. Davison, K.K. and L.L. Birch, *Childhood overweight: a contextual model and recommendations for future research.* Obes Rev, 2001. **2**(3): p. 159-71.

33. Moore, E.S., W.L. Wilkie, and D.M. Desrochers, *All in the Family? Parental Roles in the Epidemic of Childhood Obesity.* Journal of Consumer Research, 2017. **43**(5): p. 824-859.
34. WHO, *Obesity: Preventing and managing the global epidemic. Report of a WHO Consultation*, in *WHO technical report series*. 2000: Geneva, Switzerland.
35. Mei, Z., et al., *Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents.* Am J Clin Nutr, 2002. **75**(6): p. 978-85.
36. Must, A. and S.E. Anderson, *Body mass index in children and adolescents: considerations for population-based applications.* International Journal of Obesity, 2006. **30**(4): p. 590-594.
37. Cole, T.J., et al., *Establishing a standard definition for child overweight and obesity worldwide: international survey.* BMJ, 2000: p. 1240-3.
38. WHO/Europe, *Oral Health - data and statistics*. 2017, World health Organization Regional Office For Europe.
39. Loesche, W.J., *Chemotherapy of dental plaque infections.* Oral Sci Rev, 1976. **9**: p. 65-107.
40. Kawashita, Y., M. Kitamura, and T. Saito, *Early childhood caries.* Int J Dent, 2011.
41. Law, V., W.K. Seow, and G. Townsend, *Factors influencing oral colonization of mutans streptococci in young children.* Aust Dent J, 2007. **52**: p. 93-100.
42. Granath, L., et al., *Prevalence of dental caries in 4- to 5-year-old children partly explained by presence of salivary mutans streptococci.* J Clin Microbiol, 1993. **31**(1): p. 66-70.
43. Sullivan, A., L. Granath, and J. Widenheim, *Correlation between Child Caries Incidence and S-Mutans Lactobacilli in Saliva after Correction for Confounding Factors.* Community Dentistry and Oral Epidemiology, 1989. **17**(5): p. 240-244.
44. Marsh, P.D., *Microbial ecology of dental plaque and its significance in health and disease.* Adv Dent Res, 1994. **8**(2): p. 263-71.
45. Marsh, P.D., *Are dental diseases examples of ecological catastrophes?* Microbiology, 2003. **149**(Pt 2): p. 279-94.
46. Touger-Decker, R. and C. van Loveren, *Sugars and dental caries.* Am J Clin Nutr, 2003. **78**: p. 881-892.
47. Karjalainen, S., et al., *High Sucrose Intake at 3 Years of Age Is Associated with Increased Salivary Counts of Mutans Streptococci and Lactobacilli, and with Increased Caries Rate from 3 to 16 Years of Age.* Caries Research, 2015. **49**(2): p. 125-132.
48. Parisotto, T.M., et al., *Early Childhood Caries and Mutans Streptococci: A Systematic Review.* Oral Health & Preventive Dentistry, 2010. **8**(1): p. 59-70.
49. Nanda, J., et al., *Correlation between dental caries experience and mutans streptococci counts using saliva and plaque as microbial risk indicators in 3-8 year old children. A cross Sectional study.* J Clin Exp Dent, 2015. **7**(1): p. 114-8.

50. Damle, S.G., et al., *Correlation between dental caries experience and mutans streptococci counts by microbial and molecular (polymerase chain reaction) assay using saliva as microbial risk indicator.* Dent Res J (Isfahan), 2016. **13**(6): p. 552-559.
51. Edelstein, B.L., S.D. Ureles, and A. Smaldone, *Very High Salivary Streptococcus Mutans Predicts Caries Progression in Young Children.* Pediatric Dentistry, 2016. **38**(4): p. 325-330.
52. Thenisch, N.L., et al., *Are mutans streptococci detected in preschool children a reliable predictive factor for dental caries risk? A systematic review.* Caries Research, 2006. **40**(5): p. 366-374.
53. Hooley, M., et al., *Body mass index and dental caries in children and adolescents: a systematic review of literature published 2004 to 2011.* Syst Rev, 2012. **1**: p. 57.
54. Hayden, C., et al., *Obesity and dental caries in children: a systematic review and meta-analysis.* Community Dent Oral Epidemiol, 2013. **41**(4): p. 289-308.
55. Li, L.W., et al., *Anthropometric Measurements and Dental Caries in Children: A Systematic Review of Longitudinal Studies.* Advances in Nutrition, 2015. **6**(1): p. 52-63.
56. Marshall, T.A., et al., *Dental caries and childhood obesity: roles of diet and socioeconomic status.* Community Dent Oral Epidemiol, 2007. **35**(6): p. 449-58.
57. WHO, *Comprehensive Mental Health Action Plan 2013–2020* 2013, World Health Organization: Geneva.
58. Reynolds, W.M., *Internalizing Disorders in Children and Adolescents*. 1 ed. 1992: John Wiley & Sons.
59. Costello, E.J., et al., *Prevalence and development of psychiatric disorders in childhood and adolescence.* Arch Gen Psychiatry, 2003. **60**(8): p. 837-44.
60. Kessler, R.C., et al., *Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication.* Arch Gen Psychiatry, 2005. **62**(6): p. 593-602.
61. Costello, E.J., H. Egger, and A. Angold, *10-year research update review: the epidemiology of child and adolescent psychiatric disorders: I. Methods and public health burden.* J Am Acad Child Adolesc Psychiatry, 2005. **44**(10): p. 972-86.
62. Belfer, M.L., *Child and adolescent mental disorders: the magnitude of the problem across the globe.* J Child Psychol Psychiatry, 2008. **49**(3): p. 226-36.
63. Kieling, C., et al., *Child and adolescent mental health worldwide: evidence for action.* Lancet, 2011. **378**(9801): p. 1515-25.
64. Kelleher, K.J., et al., *Increasing identification of psychosocial problems: 1979-1996.* Pediatrics, 2000. **105**(6): p. 1313-21.
65. Zahn-Waxler, C., E.A. Shirtcliff, and K. Marceau, *Disorders of childhood and adolescence: gender and psychopathology.* Annu Rev Clin Psychol, 2008. **4**: p. 275-303.
66. Copeland, W., et al., *Cumulative prevalence of psychiatric disorders by young adulthood: a prospective cohort analysis from the Great Smoky*

Mountains Study. J Am Acad Child Adolesc Psychiatry, 2011. **50**(3): p. 252-61.

67. Merikangas, K.R., et al., *Prevalence and Treatment of Mental Disorders Among US Children in the 2001-2004 NHANES.* Pediatrics, 2010. **125**(1): p. 75-81.
68. Bor, W., et al., *Are child and adolescent mental health problems increasing in the 21st century? A systematic review.* Aust N Z J Psychiatry, 2014. **48**(7): p. 606-16.
69. Nigg, J.T., *Attention-deficit/hyperactivity disorder and adverse health outcomes.* Clin Psychol Rev, 2013. **33**(2): p. 215-28.
70. Halfon, N., K. Larson, and W. Slusser, *Associations Between Obesity and Comorbid Mental Health, Developmental, and Physical Health Conditions in a Nationally Representative Sample of US Children Aged 10 to 17.* Academic Pediatrics, 2013. **13**(1): p. 6-13.
71. Incledon, E., M. Wake, and M. Hay, *Psychological predictors of adiposity: Systematic review of longitudinal studies.* International Journal of Pediatric Obesity, 2011. **6**(2-2): p. 1-11.
72. Luppino, F.S., et al., *Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies.* Arch Gen Psychiatry, 2010. **67**(3): p. 220-9.
73. Zhao, G.X., et al., *Depression and Anxiety among US Adults: Associations with Body Mass Index.* Obesity, 2008. **16**: p. 175-176.
74. Goldfield, G.S., et al., *Body Dissatisfaction, Dietary Restraint, Depression, and Weight Status in Adolescents.* Journal of School Health, 2010. **80**(4): p. 186-192.
75. Cornette, R., *The emotional impact of obesity on children.* Worldviews on Evidence-Based Nursing, 2008. **5**(3): p. 136-141.
76. Pine, D.S., et al., *The association between childhood depression and adulthood body mass index.* Pediatrics, 2001. **107**(5): p. 1049-1056.
77. Daly, M., E. Robinson, and A.R. Sutin, *Does Knowing Hurt? Perceiving Oneself as Overweight Predicts Future Physical Health and Well-Being.* Psychological Science, 2017.
78. Reiss, F., *Socioeconomic inequalities and mental health problems in children and adolescents: a systematic review.* Soc Sci Med, 2013. **90**: p. 24-31.
79. Iguacel, I., et al., *Associations between social vulnerabilities and psychosocial problems in European children. Results from the IDEFICS study.* Eur Child Adolesc Psychiatry, 2017.
80. Biddle, S., *Children, Exercise and Mental-Health.* International Journal of Sport Psychology, 1993. **24**(2): p. 200-216.
81. Calfas, K.J. and W.C. Taylor, *Effects of Physical-Activity on Psychological Variables in Adolescents.* Pediatric Exercise Science, 1994. **6**(4): p. 406-423.
82. Baker, P.R., et al., *Community wide interventions for increasing physical activity.* Cochrane Database Syst Rev, 2015. **1**.

83. Chen, M., *The neurobiology of depression and physical exercise*, in *Handbook of Physical Activity and Mental Health*. 2013, Routledge: London. p. 169–184.
84. Hinkley, T., et al., *Early childhood physical activity, sedentary behaviors and psychosocial well-being: a systematic review.* Prev Med, 2014. **62**: p. 182-92.
85. Larun, L., et al., *Exercise in prevention and treatment of anxiety and depression among children and young people.* Cochrane Database of Systematic Reviews, 2006(3).
86. Biddle, S.J. and M. Asare, *Physical activity and mental health in children and adolescents: a review of reviews.* Br J Sports Med, 2011. **45**(11): p. 886-95.
87. Ahn, S. and A.L. Fedewa, *A Meta-analysis of the Relationship Between Children's Physical Activity and Mental Health.* Journal of Pediatric Psychology, 2011. **36**(4): p. 385-397.
88. Cooney, G.M., et al., *Exercise for depression.* Cochrane Database of Systematic Reviews, 2013(9).
89. Martinez-Gonazlez, M.A. and A. Sanchez-Villegas, *Food patterns and the prevention of depression.* Proceedings of the Nutrition Society, 2016. **75**(2): p. 139-146.
90. Vermeulen, E., et al., *The association between dietary patterns derived by reduced rank regression and depressive symptoms over time: the Invecchiare in Chianti (InCHIANTI) study.* British Journal of Nutrition, 2016. **115**(12): p. 2145-2153.
91. Jacka, F.N., et al., *A randomised controlled trial of dietary improvement for adults with major depression (the 'SMILES' trial).* BMC Med, 2017. **15**(1): p. 23.
92. Forsyth, A., F.P. Deane, and P. Williams, *A lifestyle intervention for primary care patients with depression and anxiety: A randomised controlled trial.* Psychiatry Research, 2015. **230**(2): p. 537-544.
93. Trapp, G.S.A., et al., *A prospective investigation of dietary patterns and internalizing and externalizing mental health problems in adolescents.* Food Science & Nutrition, 2016. **4**(6): p. 888-896.
94. O'Neil, A., et al., *Relationship Between Diet and Mental Health in Children and Adolescents: A Systematic Review.* American Journal of Public Health, 2014. **104**(10): p. 31-42.
95. Khalid, S., C.M. Williams, and S.A. Reynolds, *Is there an association between diet and depression in children and adolescents? A systematic review.* British Journal of Nutrition, 2016. **116**(12): p. 2097-2108.
96. Birch, L.L. and K.K. Davison, *Family environmental factors influencing the developing behavioral controls of food intake and childhood overweight.* Pediatr Clin North Am, 2001. **48**(4): p. 893-907.
97. Ventura, A.K. and L.L. Birch, *Does parenting affect children's eating and weight status?* Int J Behav Nutr Phys Act, 2008. **5**: p. 15.
98. Berge, J.M., et al., *Parenting style and family meals: cross-sectional and 5-year longitudinal associations.* J Am Diet Assoc, 2010. **110**(7): p. 1036-42.

99. Berge, J.M., et al., *Parenting style as a predictor of adolescent weight and weight-related behaviors.* J Adolesc Health, 2010. **46**(4): p. 331-8.
100. Gibson, E.L., et al., *A narrative review of psychological and educational strategies applied to young children's eating behaviours aimed at reducing obesity risk.* Obes Rev, 2012. **13 Suppl 1**: p. 85-95.
101. Yee, A.Z., M.O. Lwin, and S.S. Ho, *The influence of parental practices on child promotive and preventive food consumption behaviors: a systematic review and meta-analysis.* Int J Behav Nutr Phys Act, 2017. **14**(1): p. 47.
102. Kristiansen, A.L., et al., *Associations between physical home environmental factors and vegetable consumption among Norwegian 3-5-year-olds: the BRA-study.* Public Health Nutrition, 2017. **20**(7): p. 1173-1183.
103. Birch, L.L. and J.O. Fisher, *Development of eating behaviors among children and adolescents.* Pediatrics, 1998. **101**(3): p. 539-549.
104. Pinard, C.A., et al., *Measures of the home environment related to childhood obesity: a systematic review.* Public Health Nutrition, 2012. **15**(1): p. 97-109.
105. Reicks, M., et al., *Influence of Parenting Practices on Eating Behaviors of Early Adolescents during Independent Eating Occasions: Implications for Obesity Prevention.* Nutrients, 2015. **7**(10): p. 8783-801.
106. Jansen E, Daniels LA, and Nicholson JM, *The dynamics of parenting and early feeding-constructs and controversies: a viewpoint.* Early Child Dev Care, 2012. **182**(8): p. 967-981.
107. Maccoby EE and Martin JA, *Socialization in the context of the family: Parent-child interaction*, in *Handbook of child psychology*, E.M. Hetherington, Editor. 1983, Wiley: Chichester, New York. p. 1-101.
108. Vereecken, C.A., E. Keukelier, and L. Maes, *Influence of mother's educational level on food parenting practices and food habits of young children.* Appetite, 2004. **43**(1): p. 93-103.
109. Cardel, M., et al., *Parental feeding practices and socioeconomic status are associated with child adiposity in a multi-ethnic sample of children.* Appetite, 2012. **58**(1): p. 347-53.
110. Worobey, J., et al., *Feeding Practices of Mothers from Varied Income and Racial/Ethnic Groups.* Early Child Dev Care, 2013. **183**(11): p. 1661-1668.
111. Loth, K.A., et al., *Eat this, not that! Parental demographic correlates of food-related parenting practices.* Appetite, 2013. **60**(1): p. 140-7.
112. McPhie, S., et al., *Maternal correlates of maternal child feeding practices: a systematic review.* Matern Child Nutr, 2014. **10**(1): p. 18-43.
113. Ochoa, A. and J.M. Berge, *Home Environmental Influences on Childhood Obesity in the Latino Population: A Decade Review of Literature.* J Immigr Minor Health, 2017. **19**(2): p. 430-447.
114. Berge, J.M., et al., *Parent/Adolescent Weight Status Concordance and Parent Feeding Practices.* Pediatrics, 2015. **136**(3): p. 591-598.
115. Sylvetsky-Meni A. C., et al., *The Impact of Parents' Categorization of Their Own Weight and Their Child's Weight on Healthy Lifestyle Promoting Beliefs and Practices.* Journal of Obesity, 2015. **2015**: p. 7.
116. Webber, L., et al., *Child adiposity and maternal feeding practices: a longitudinal analysis.* Am J Clin Nutr, 2010. **92**(6): p. 1423-8.

117. Jansen, P.W., et al., *Feeding practices and child weight: is the association bidirectional in preschool children?* Am J Clin Nutr, 2014. **100**(5): p. 1329-36.
118. Skouteris H, et al., *Parent-child interactions and obesity prevention: a systematic review of the literature.* Early Child Dev Care, 2012. **182**(2): p. 153-174.
119. Shloim, N., et al., *Parenting Styles, Feeding Styles, Feeding Practices, and Weight Status in 4-12 Year-Old Children: A Systematic Review of the Literature.* Front Psychol, 2015. **6**: p. 1849.
120. Larsen, J.K., et al., *How parental dietary behavior and food parenting practices affect children's dietary behavior. Interacting sources of influence?* Appetite, 2015. **89**: p. 246-57.
121. Pinard, C.A., et al., *Measures of the home environment related to childhood obesity: a systematic review.* Public Health Nutr, 2012. **15**(1): p. 97-109.
122. Vaughn, A.E., et al., *Measuring parent food practices: a systematic review of existing measures and examination of instruments.* Int J Behav Nutr Phys Act, 2013. **10**: p. 61.
123. Vaughn, A.E., et al., *Fundamental constructs in food parenting practices: a content map to guide future research.* Nutr Rev, 2016. **74**(2): p. 98-117.
124. Doak, C., et al., *Prevention of childhood obesity - what type of evidence should we consider relevant?* Obesity Reviews, 2009. **10**(3): p. 350-356.
125. Waters, E., et al., *Interventions for preventing obesity in children.* Cochrane Database of Systematic Reviews, 2011(12).
126. da Silveira, J.A.C., et al., *The effect of participation in school-based nutrition education interventions on body mass index: A meta-analysis of randomized controlled community trials.* Preventive Medicine, 2013. **56**(3-4): p. 237-243.
127. Lavelle, H.V., D.F. Mackay, and J.P. Pell, *Systematic review and meta-analysis of school-based interventions to reduce body mass index.* Journal of Public Health, 2012. **34**(3): p. 360-369.
128. Bleich, S.N., et al., *Systematic review of community-based childhood obesity prevention studies.* Pediatrics, 2013. **132**(1): p. 201-10.
129. Bourke, M., P.J. Whittaker, and A. Verma, *Are dietary interventions effective at increasing fruit and vegetable consumption among overweight children? A systematic review.* Journal of Epidemiology and Community Health, 2014. **68**(5): p. 485-490.
130. Metcalf, B., W. Henley, and T. Wilkin, *Effectiveness of intervention on physical activity of children: systematic review and meta-analysis of controlled trials with objectively measured outcomes* British Journal of Sports Medicine, 2013. **47**(4): p. 226-226.
131. Williams, G., *The IDEFICS intervention: what can we learn for public policy?* Obesity Reviews, 2015. **16**: p. 151-161.
132. Davison, K.K., H.A. Lawson, and J.D. Coatsworth, *The Family-centered Action Model of Intervention Layout and Implementation (FAMILI): the example of childhood obesity.* Health Promot Pract, 2012. **13**(4): p. 454-61.

133. Davison, K.K., et al., *A childhood obesity intervention developed by families for families: results from a pilot study.* International Journal of Behavioral Nutrition and Physical Activity, 2013. **10**.
134. Summerbell, C.D., et al., *Evidence-based recommendations for the development of obesity prevention programs targeted at preschool children.* Obesity Reviews, 2012. **13**: p. 129-132.
135. Showell, N.N., et al., *A Systematic Review of Home-Based Childhood Obesity Prevention Studies.* Pediatrics, 2013. **132**(1): p. 193-200.
136. Swinburn, B.A., et al., *Obesity 1 The global obesity pandemic: shaped by global drivers and local environments.* Lancet, 2011. **378**(9793): p. 804-814.
137. Foltz, J.L., et al., *Population-Level Intervention Strategies and Examples for Obesity Prevention in Children.* Annual Review of Nutrition, Vol 32, 2012. **32**: p. 391-415.
138. Ahrens, W., et al., *Cohort Profile: The transition from childhood to adolescence in European children-how I.Family extends the IDEFICS cohort.* Int J Epidemiol, 2016.
139. Manios, Y., et al., *Designing and implementing a kindergarten-based, family-involved intervention to prevent obesity in early childhood: the ToyBox-study.* Obes Rev, 2014. **15 Suppl 3**: p. 5-13.
140. Silva Junior, S.H., et al., *Assessment of participation bias in cohort studies: systematic review and meta-regression analysis.* Cad Saude Publica, 2015. **31**(11): p. 2259-74.
141. Regber, S., et al., *Assessment of selection bias in a health survey of children and families - the IDEFICS Sweden-study.* BMC Public Health, 2013. **13**.
142. Galea, S. and M. Tracy, *Participation rates in epidemiologic studies.* Ann Epidemiol, 2007. **17**(9): p. 643-53.
143. Wolke, D., et al., *Selective drop-out in longitudinal studies and non-biased prediction of behaviour disorders.* Br J Psychiatry, 2009. **195**(3): p. 249-56.
144. Hense, S., et al., *Determinants of Attrition to Follow-Up in a Multicentre Cohort Study in Children-Results from the IDEFICS Study.* Epidemiology Research International, 2013. **2013**: p. 9.
145. Bartholomew, L.K., et al., *Planning Health Promotion Programs. An Intervention Mapping Approach.* 3 ed. 2011: Jossey-Bass. 768.
146. Verbestel, V., et al., *Using the intervention mapping protocol to develop a community-based intervention for the prevention of childhood obesity in a multi-centre European project: the IDEFICS intervention.* International Journal of Behavioral Nutrition and Physical Activity, 2011. **8**.
147. Verbestel, V., et al., *The IDEFICS Intervention Toolbox - A Guide to Successful Obesity Prevention at Community Level*. Public Health - Social and Behavioral Health, ed. P.J. Maddock. 2012: InTech.
148. Marfell-Jones M, et al., *International Standards for Anthropometric Assessment*. 2006, Potchefstroom, South Africa: International Society for the Advancement of Kinanthropometry.
149. Stomfai, S., et al., *Intra- and inter-observer reliability in anthropometric measurements in children.* International Journal of Obesity, 2011. **35**: p. 45-51.

150. Baughcum, A.E., et al., *Maternal feeding practices and beliefs and their relationships to overweight in early childhood.* J Dev Behav Pediatr, 2001. **22**(6): p. 391-408.
151. Berger, P.K., et al., *Girls' picky eating in childhood is associated with normal weight status from ages 5 to 15 y.* American Journal of Clinical Nutrition, 2016. **104**(6): p. 1577-1582.
152. Antoniou, E.E., et al., *Picky eating and child weight status development: alongitudinal study.* Journal of Human Nutrition and Dietetics, 2016. **29**(3): p. 298-307.
153. Dubois, L., et al., *Problem eating behaviors related to social factors and body weight in preschool children: A longitudinal study.* International Journal of Behavioral Nutrition and Physical Activity, 2007. **4**.
154. Suling, M., et al., *Design and results of the pretest of the IDEFICS study.* Int J Obes, 2011. **35 Suppl 1**: p. 30-44.
155. *Developing Effective Wording and Format Options for a Children's Nutrition Behavior Questionnaire for Mothers of Children in Kindergarten*. 2005, USDA/ORC Macro: Washington.
156. Caan, B., R.A. Hiatt, and A.M. Owen, *Mailed dietary surveys: response rates, error rates, and the effect of omitted food items on nutrient values.* Epidemiology, 1991. **2**(6): p. 430-6.
157. Michels, K.B. and W.C. Willett, *Self-Administered Semiquantitative Food Frequency Questionnaires Patterns, Predictors, and Interpretation of Omitted Items.* Epidemiology, 2009. **20**(2): p. 295-301.
158. Rimm, E.B., et al., *Reproducibility and Validity of an Expanded Self-Administered Semiquantitative Food Frequency Questionnaire among Male Health-Professionals.* American Journal of Epidemiology, 1992. **135**(10): p. 1114-1126.
159. Bel-Serrat, S., et al., *Relative validity of the Children's Eating Habits Questionnaire-food frequency section among young European children: the IDEFICS Study.* Public Health Nutr, 2013. **17**(2): p. 1-11.
160. Saeedi, P., et al., *Reproducibility and Relative Validity of a Short Food Frequency Questionnaire in 9-10 Year-Old Children.* Nutrients, 2016. **8**(5).
161. Huybrechts, I., et al., *Evaluation of the Children's Eating Habits Questionnaire used in the IDEFICS study by relating urinary calcium and potassium to milk consumption frequencies among European children.* Int J Obes, 2011. **35 Suppl 1**: p. 69-78.
162. Lanfer, A., et al., *Reproducibility of food consumption frequencies derived from the Children's Eating Habits Questionnaire used in the IDEFICS study.* Int J Obes, 2011. **35**: p. 61-8.
163. Vereecken, C.A., et al., *Comparison of a short food-frequency questionnaire and derived indices with a seven-day diet record in Belgian and Italian children.* Int J Public Health, 2008. **53**(6): p. 297-305.
164. Landis, J.R. and G.G. Koch, *The measurement of observer agreement for categorical data.* Biometrics, 1977. **33**(1): p. 159-74.
165. McHugh, M.L., *Interrater reliability: the kappa statistic.* Biochem Med (Zagreb), 2012. **22**(3): p. 276-82.

166. Willett, W., *Reproducibility and Validity of Food Frequency Questionnaires*, in *Nutritional epidemiology*. 2013, Oxford University Press.
167. Tabacchi, G., et al., *A meta-analysis of the validity of FFQ targeted to adolescents.* Public Health Nutr, 2016. **19**(7): p. 1168-83.
168. Waijers, P.M., E.J. Feskens, and M.C. Ocke, *A critical review of predefined diet quality scores.* Br J Nutr, 2007. **97**(2): p. 219-31.
169. WHO, *Measurement of Quality of Life in Children*. 1994, Geneva, Switzerland: World Health Organization - Division of Mental Health.
170. Ravens-Sieberer, U. and M. Bullinger. *Questionnaire for measuring health-related quality of life in children and adolescents - manual - revised version*. 2000; Available from: http://kindl.org/cms/wp-content/uploads/2009/11/ManEnglish.pdf.
171. Goodman, R., *The Strengths and Difficulties Questionnaire: a research note.* J Child Psychol Psychiatry, 1997. **38**(5): p. 581-6.
172. Ravens-Sieberer, U., et al., *The KIDSCREEN-52 quality of life measure for children and adolescents: Psychometric results from a cross-cultural survey in 13 European countries.* Value in Health, 2008. **11**(4): p. 645-658.
173. Bullinger, M., et al., *Psychometric properties of the KINDL-R questionnaire: results of the BELLA study.* Eur Child Adolesc Psychiatry, 2008. **17 Suppl 1**: p. 125-32.
174. Erhart, M., et al., *Measuring adolescents' HRQoL via self reports and parent proxy reports: an evaluation of the psychometric properties of both versions of the KINDL-R instrument.* Health Qual Life Outcomes, 2009. **7**: p. 77.
175. Rutter, M., *A children's behaviour questionnaire for completion by teachers: preliminary findings.* J Child Psychol Psychiatry, 1967. **8**(1): p. 1-11.
176. Stone, L.L., et al., *Psychometric Properties of the Parent and Teacher Versions of the Strengths and Difficulties Questionnaire for 4-to 12-Year-Olds: A Review.* Clinical Child and Family Psychology Review, 2010. **13**(3): p. 254-274.
177. Jacka, F.N., et al., *Diet quality and mental health problems in adolescents from East London: a prospective study.* Soc Psychiatry Psychiatr Epidemiol, 2013. **48**(8): p. 1297-306.
178. Michels, N., et al., *Stress, emotional eating behaviour and dietary patterns in children.* Appetite, 2012. **59**(3): p. 762-9.
179. Wiles, N.J., et al., *'Junk food' diet and childhood behavioural problems: results from the ALSPAC cohort.* European Journal of Clinical Nutrition, 2009. **63**(4): p. 491-498.
180. Kohlboeck, G., et al., *Food Intake, Diet Quality and Behavioral Problems in Children: Results from the GINI-plus/LISA-plus Studies.* Annals of Nutrition and Metabolism, 2012. **60**(4): p. 247-256.
181. Peacock, P.J., et al., *Childhood diet and behavioural problems: results from the ALSPAC cohort.* European Journal of Clinical Nutrition, 2011. **65**(6): p. 720-726.
182. Renzaho, A.M.N., S. Kumanyika, and K.L. Tucker, *Family functioning, parental psychological distress, child behavioural problems, socio-*

economic disadvantage and fruit and vegetable consumption among 4-12 year-old Victorians, Australia. Health Promotion International, 2011. **26**(3): p. 263-275.

183. Goodman, R., H. Meltzer, and V. Bailey, *The Strengths and Difficulties Questionnaire: a pilot study on the validity of the self-report version.* Eur Child Adolesc Psychiatry, 1998. **7**(3): p. 125-30.
184. Goodman, R., *Psychometric properties of the strengths and difficulties questionnaire.* J Am Acad Child Adolesc Psychiatry, 2001. **40**(11): p. 1337-45.
185. Stone, L.L., et al., *The Strengths and Difficulties Questionnaire: psychometric properties of the parent and teacher version in children aged 4-7.* BMC Psychology, 2015. **3**(1): p. 4.
186. Goodman, R., *Psychometric properties of the strengths and difficulties questionnaire.* Journal of the American Academy of Child and Adolescent Psychiatry, 2001. **40**(11): p. 1337-1345.
187. Vereecken, C.A., et al., *Development and evaluation of a self-administered computerized 24-h dietary recall method for adolescents in Europe.* Int J Obes, 2008. **32 Suppl 5**: p. 26-34.
188. Emilson, C.G., *Prevalence of Streptococcus mutans with different colonial morphologies in human plaque and saliva. Scand J Dent Res*, 1983. **91**: p. 26-32.
189. Klock, B. and B. Krasse, *A comparison between different methods for prediction of caries activity. Scand J Dent Res*, 1979. **87**: p. 129-39.
190. UNESCO *International Standard Classification of Education ISCED 2011*. 2012.
191. Iguacel, I., et al., *Associations between social vulnerabilities and dietary patterns in European children: the Identification and prevention of Dietary- and lifestyle-induced health EFfects In Children and infantS (IDEFICS) study.* British Journal of Nutrition, 2016. **116**(7): p. 1288-1297.
192. Barkeling, B., et al., *Intake of sweet foods and counts of cariogenic microorganisms in relation to body mass index and psychometric variables in women.* Int J Obes Relat Metab Disord, 2002. **26**: p. 1239-44.
193. Barkeling, B., et al., *Intake of sweet foods and counts of cariogenic microorganisms in obese and normal-weight women.* Eur J Clin Nutr, 2001. **55**: p. 850-5.
194. Vågstrand, K., et al., *Associations between salivary bacteria and reported sugar intake and their relationship with body mass index in women and their adolescent children.* Public Health Nutr, 2007. **11**: p. 341-8.
195. Andreen, I. and B. Kohler, *Effects of Weight Watchers' diet on salivary secretion rate, buffer effect and numbers of mutans streptococci and lactobacilli.* Scand J Dent Res, 1992. **100**(2): p. 93-7.
196. Moynihan, P.J., *The role of diet and nutrition in the etiology and prevention of oral diseases.* Bull World Health Organ, 2005. **83**(9): p. 694-9.
197. Vågstrand, K.E. and D. Birkhed, *Cariogenic bacteria as biomarkers for sugar intake. Nutr Rev*, 2007. **65**: p. 111-21.

198. Howarth, N.C., et al., *Eating patterns and dietary composition in relation to BMI in younger and older adults.* International Journal of Obesity, 2007. **31**(4): p. 675-684.
199. Mattes, R., *Energy intake and obesity: Ingestive frequency outweighs portion size.* Physiology & Behavior, 2014. **134**: p. 110-118.
200. Schoenfeld, B.J., A.A. Aragon, and J.W. Krieger, *Effects of meal frequency on weight loss and body composition: a meta-analysis.* Nutr Rev, 2015. **73**(2): p. 69-82.
201. Malik, V.S., et al., *Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis.* Am J Clin Nutr, 2013. **98**(4): p. 1084-102.
202. Te Morenga, L., S. Mallard, and J. Mann, *Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies.* BMJ, 2013. **346**.
203. Seegers, V., et al., *Short sleep duration and body mass index: a prospective longitudinal study in preadolescence.* Am J Epidemiol, 2011. **173**(6): p. 621-9.
204. Sharma, S. and M. Kavuru, *Sleep and metabolism: an overview.* Int J Endocrinol, 2010.
205. Van Cauter, E. and K.L. Knutson, *Sleep and the epidemic of obesity in children and adults.* Eur J Endocrinol, 2008. **159 Suppl 1**: p. S59-66.
206. Dashti, H.S., et al., *Short sleep duration and dietary intake: epidemiologic evidence, mechanisms, and health implications.* Adv Nutr, 2015. **6**(6): p. 648-59.
207. Paruthi, S., et al., *Recommended Amount of Sleep for Pediatric Populations: A Consensus Statement of the American Academy of Sleep Medicine.* Journal of Clinical Sleep Medicine, 2016. **12**(6): p. 785-786.
208. Lubans, D., et al., *Physical Activity for Cognitive and Mental Health in Youth: A Systematic Review of Mechanisms.* Pediatrics, 2016. **138**(3).
209. Vartanian, L.R. and A.M. Porter, *Weight stigma and eating behavior: A review of the literature.* Appetite, 2016. **102**: p. 3-14.
210. Rodgers, R.F., et al., *"Stop eating lollies and do lots of sports": a prospective qualitative study of the development of children's awareness of dietary restraint and exercise to lose weight.* International Journal of Behavioral Nutrition and Physical Activity, 2015. **12**.
211. Jacka, F.N., et al., *A prospective study of diet quality and mental health in adolescents.* PLoS One, 2011. **6**(9).
212. Kulkarni, A.A., B.A. Swinburn, and J. Utter, *Associations between diet quality and mental health in socially disadvantaged New Zealand adolescents.* European Journal of Clinical Nutrition, 2015. **69**(1): p. 79-83.
213. Oellingrath, I.M., M.V. Svendsen, and I. Hestetun, *Eating patterns and mental health problems in early adolescence - a cross-sectional study of 12-13-year-old Norwegian schoolchildren.* Public Health Nutrition, 2014. **17**(11): p. 2554-2562.
214. Puloka, et al., *Dietary behaviours and the mental well-being of New Zealand adolescents.* J Paediatr Child Health, 2017. **53**(7): p. 657-662.

215. Opie, R.S., et al., *The impact of whole-of-diet interventions on depression and anxiety: a systematic review of randomised controlled trials.* Public Health Nutrition, 2015. **18**(11): p. 2074-2093.
216. Kaseva, K., et al., *Psychological wellbeing in 20-year-old adults receiving repeated lifestyle counselling since infancy.* Acta Paediatrica, 2015. **104**(8): p. 815-822.
217. Bellisle, F., *Effects of diet on behaviour and cognition in children.* Br J Nutr, 2004. **92 Suppl 2**: p. 227-32.
218. Bamber, D.J., C.S. Stokes, and A.M. Stephen, *The role of diet in the prevention and management of adolescent depression.* Nutrition Bulletin, 2007. **32**: p. 90-99.
219. Isaacs, E. and J. Oates, *Nutrition and cognition: assessing cognitive abilities in children and young people.* European Journal of Nutrition, 2008. **47**: p. 4-24.
220. Schmitt, J.A.J., D. Benton, and K.W. Kallus, *General methodological considerations for the assessment of nutritional influences on human cognitive functions.* European Journal of Nutrition, 2005. **44**(8): p. 459-464.
221. Berge, J.M., et al., *Family functioning: associations with weight status, eating behaviors, and physical activity in adolescents.* J Adolesc Health, 2013. **52**(3): p. 351-7.
222. Neumark-Sztainer, D., et al., *What's for dinner? Types of food served at family dinner differ across parent and family characteristics.* Public Health Nutrition, 2014. **17**(1): p. 145-155.
223. Speirs, K.E., et al., *Is family sense of coherence a protective factor against the obesogenic environment?* Appetite, 2016. **99**: p. 268-276.
224. Berge, J.M., et al., *Structural and Interpersonal Characteristics of Family Meals: Associations with Adolescent Body Mass Index and Dietary Patterns.* Journal of the Academy of Nutrition and Dietetics, 2013. **113**(6): p. 816-822.
225. Loth, K.A., et al., *Directive and non-directive food-related parenting practices: Associations between an expanded conceptualization of food-related parenting practices and child dietary intake and weight outcomes.* Appetite, 2016. **107**: p. 188-195.
226. Melbye, E.L. and H. Hansen, *Child weight and parental feeding practices: a child-responsive model.* Nutrition & Food Science, 2015. **45**(1): p. 174-188.
227. Afonso, L., et al., *Bidirectional association between parental child-feeding practices and body mass index at 4 and 7 y of age.* American Journal of Clinical Nutrition, 2016. **103**(3): p. 861-867.
228. Jansen, P.W., et al., *Feeding practices and child weight: is the association bidirectional in preschool children?* American Journal of Clinical Nutrition, 2014. **100**(5): p. 1329-1336.
229. Sigman-Grant, M., et al., *Family Resiliency: A Neglected Perspective in Addressing Obesity in Young Children.* Childhood Obesity, 2015. **11**(6): p. 664-673.
230. Robinson, E., *Overweight but unseen: a review of the underestimation of weight status and a visual normalization theory.* Obes Rev, 2017.

231. Regber, S., et al., *Parental perceptions of and concerns about child's body weight in eight European countries--the IDEFICS study.* Pediatr Obes, 2013. **8**(2): p. 118-29.
232. Lundahl, A., K.M. Kidwell, and T.D. Nelson, *Parental Underestimates of Child Weight: A Meta-analysis.* Pediatrics, 2014. **133**(3): p. E689-E703.
233. Puhl, R.M. and C.A. Heuer, *The Stigma of Obesity: A Review and Update.* Obesity, 2009. **17**(5): p. 941-964.
234. Puhl, R.M. and C.A. Heuer, *Obesity Stigma: Important Considerations for Public Health.* American Journal of Public Health, 2010. **100**(6): p. 1019-1028.
235. Ek, A., et al., *Associations between Parental Concerns about Preschoolers' Weight and Eating and Parental Feeding Practices: Results from Analyses of the Child Eating Behavior Questionnaire, the Child Feeding Questionnaire, and the Lifestyle Behavior Checklist.* Plos One, 2016. **11**(1).
236. Gregory, J.E., S.J. Paxton, and A.M. Brozovic, *Pressure to eat and restriction are associated with child eating behaviours and maternal concern about child weight, but not child body mass index, in 2-to 4-year-old children.* Appetite, 2010. **54**(3): p. 550-556.
237. Webber, L., et al., *Associations between child weight and maternal feeding styles are mediated by maternal perceptions and concerns.* European Journal of Clinical Nutrition, 2010. **64**(3): p. 259-265.
238. Fisher, J.O. and L.L. Birch, *Restricting access to palatable foods affects children's behavioral response, food selection, and intake.* Am J Clin Nutr, 1999. **69**(6): p. 1264-72.
239. Galloway, A.T., et al., *'Finish your soup': counterproductive effects of pressuring children to eat on intake and affect.* Appetite, 2006. **46**(3): p. 318-23.
240. Patrick, H., et al., *Parenting Styles and Practices in Children's Obesogenic Behaviors: Scientific Gaps and Future Research Directions.* Childhood Obesity, 2013. **9**: p. 73-86.
241. Frankel, L.A., et al., *Parental Influences on Children's Self-Regulation of Energy Intake: Insights from Developmental Literature on Emotion Regulation.* J Obes, 2012. **2012**.
242. De Henauw, S., et al., *Effects of a community-oriented obesity prevention programme on indicators of body fatness in preschool and primary school children. Main results from the IDEFICS study.* Obes Rev, 2015. **16 Suppl 2**: p. 16-29.
243. De Bourdeaudhuij, I., et al., *Behavioural effects of a community-oriented setting-based intervention for prevention of childhood obesity in eight European countries. Main results from the IDEFICS study.* Obes Rev, 2015. **16 Suppl 2**: p. 30-40.
244. Whitehead, M., *A typology of actions to tackle social inequalities in health.* Journal of Epidemiology and Community Health, 2007. **61**(6): p. 473-478.
245. McGill, R., et al., *Are interventions to promote healthy eating equally effective for all? Systematic review of socioeconomic inequalities in impact (vol 15, 457, 2015).* BMC Public Health, 2015. **15**.

246. Coffield, E., et al., *Shape Up Somerville: Change in Parent Body Mass Indexes During a Child-Targeted, Community-Based Environmental Change Intervention.* American Journal of Public Health, 2015. **105**(2): p. 83-89.
247. Bjelland, M., et al., *Changes in adolescents' and parents' intakes of sugar-sweetened beverages, fruit and vegetables after 20 months: results from the HEIA study - a comprehensive, multi-component school-based randomized trial.* Food & Nutrition Research, 2015. **59**.
248. Folta, S.C., et al., *Changes in diet and physical activity resulting from the Shape Up Somerville community intervention.* Bmc Pediatrics, 2013. **13**.
249. Jensen, B.W., et al., *Intervention effects on dietary intake among children by maternal education level: results of the Copenhagen School Child Intervention Study (CoSCIS).* British Journal of Nutrition, 2015. **113**(6): p. 963-974.
250. Baranowski, T. and L. Lytle, *Should the IDEFICS outcomes have been expected?* Obesity Reviews, 2015. **16**: p. 162-172.
251. De Henauw, S., et al., *The IDEFICS community-oriented intervention programme: a new model for childhood obesity prevention in Europe?* International journal of obesity, 2011. **35**: p. 16-23.
252. De Bourdeaudhuij, I., et al., *Implementation of the IDEFICS intervention across European countries: perceptions of parents and relationship with BMI.* Obesity Reviews, 2015. **16**: p. 78-88.
253. Vargas-Garcia, E.J., et al., *Interventions to reduce consumption of sugar-sweetened beverages or increase water intake: evidence from a systematic review and meta-analysis.* Obes Rev, 2017.
254. Van Lippevelde, W., et al., *Does parental involvement make a difference in school-based nutrition and physical activity interventions? A systematic review of randomized controlled trials.* International Journal of Public Health, 2012. **57**(4): p. 673-678.
255. Van Lippevelde, W., et al., *What do parents think about parental participation in school-based interventions on energy balance-related behaviours? a qualitative study in 4 countries.* BMC Public Health, 2011. **11**.
256. Haerens, L., et al., *Developing the IDEFICS community-based intervention program to enhance eating behaviors in 2-to 8-year-old children: findings from focus groups with children and parents.* Health Education Research, 2009. **24**(3): p. 381-393.
257. Sobol-Goldberg, S., J. Rabinowitz, and R. Gross, *School-based obesity prevention programs: a meta-analysis of randomized controlled trials.* Obesity 2013. **21**(12): p. 2422-8.
258. Walls, H.L., et al., *Public health campaigns and obesity - a critique.* Bmc Public Health, 2011. **11**.
259. Gielen, A.C. and L.W. Green, *The Impact of Policy, Environmental, and Educational Interventions: A Synthesis of the Evidence From Two Public Health Success Stories.* Health Education & Behavior, 2015. **42**: p. 20-34.
260. Bleich, S.N., et al., *Systematic Review of Community-Based Childhood Obesity Prevention Studies.* Pediatrics, 2013. **132**(1): p. 201-210.

261. Hu, F.B., *Obesity epidemiology*. 2008, Oxford, New York: Oxford University press.

262. Ahrens, W., et al., *The IDEFICS cohort: design, characteristics and participation in the baseline survey. International journal of obesity*, 2011. **35** p. 3-15.

263. Burrows, T.L., R.J. Martin, and C.E. Collins, *A systematic review of the validity of dietary assessment methods in children when compared with the method of doubly labeled water.* J Am Diet Assoc, 2010. **110**(10): p. 1501-10.

264. Pala, V., et al., *Dietary patterns and longitudinal change in body mass in European children: a follow-up study on the IDEFICS multicenter cohort.* Eur J Clin Nutr, 2013. **67**(10): p. 1042-9.

265. Fernandez-Alvira, J.M., et al., *Prospective associations between socio-economic status and dietary patterns in European children: the Identification and Prevention of Dietary- and Lifestyle-induced Health Effects in Children and Infants (IDEFICS) Study.* British Journal of Nutrition, 2015. **113**(3): p. 517-525.

266. WHO, *Food based dietary guidelines in the WHO European Region* 2003, World Health Organization: Copenhagen, Denmark.

267. Opie, R.S., et al., *Dietary recommendations for the prevention of depression.* Nutr Neurosci, 2015.

Appendix I. The Swedish version of the Food Frequency Questionnaire used in I.Family

<u>Under den senaste månaden</u>, hur många gånger åt eller drack du följande livsmedel?
Vänligen markera ett svar för varje rad.

Under den senaste månaden…	Aldrig/ mindre än en gång i veckan	1-3 gånger per vecka	4-6 gånger per vecka	1 gång per dag	2 gånger per dag	3 gånger per dag	4 eller fler gånger per dag
Grönsaker							
Baljväxter (t ex bönor, ärtor, linser och kikärtor)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Potatis (kokt, inte stekt)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Stekt potatis, pommes frites, potatiskroketter	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kokta grönsaker (t ex kokta morötter, kokt broccoli)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Råa grönsaker (t ex blandad sallad, morot, paprika, gurka, tomat)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Färsk frukt							
Färsk frukt (även som färsk-pressad juice) **utan** tillsatt socker	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Färsk frukt (även som färsk-pressad juice) **med** tillsatt socker	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Drycker							
Vatten (kranvatten, vatten med eller utan kolsyra)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Fruktjuice (100% frukt), färdig-förpackad (t ex apelsinjuice, äppeljuice)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kolsyrade, sötade drycker (t ex Coca Cola, Fanta, cider utan alkohol)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kolsyrade drycker med sötningsmedel (t ex Coca Cola light, Pepsi Max)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Sötade drycker utan kolsyra (t ex saft, iste, Festis, fruktjuice med mindre än 100% frukt, sport-/energi-drycker, alkoholfritt vin)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7

Under den senaste månaden...	Aldrig/ mindre än en gång i veckan	1-3 gånger per vecka	4-6 gånger per vecka	1 gång per dag	2 gånger per dag	3 gånger per dag	4 eller fler gånger per dag
Drycker							
Drycker med sötningsmedel, utan kolsyra (t ex iste och sport-/energidrycker med sötningsmedel)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kaffe och liknande:							
a) Osötat	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
b) Sötat (t ex med socker eller honung)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Te, örtte och liknande:							
a) Osötat	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
b) Sötat (t ex med socker eller honung)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Alkoholhaltiga drycker	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Frukostflingor							
Sötade frukostflingor eller müsli med tillsatt socker (t ex frosties, start, socker el sylt på flingor/ gröt)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Gröt, havregryn, välling, osötade flingor, osötad müsli	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Mjölk							
Vanlig osötad mjölk (glöm inte mjölken i kaffet och mjölken du serverar ihop med flingorna)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Sötad och/eller smaksatt mjölk (t ex med choklad-pulver, socker och honung)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7

Vilken sorts mjölk brukar du dricka?
Vänligen markera endast ett svar.

a) Standardmjölk .... ○ 1
b) Mellanmjölk / lättmjölk .... ○ 2
c) All slags mjölk .... ○ 3
d) Jag dricker inte någon mjölk ...... ○ 4

	e) Jag vet inte ○ 7

Under den senaste månaden...	Aldrig/ mindre än en gång i veckan	1-3 gånger per vecka	4-6 gånger per vecka	1 gång per dag	2 gånger per dag	3 gånger per dag	4 eller fler gånger per dag
Yoghurt							
Vanlig osötad yoghurt, fil eller Kefir	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Sötad och smaksatt yoghurt, fil samt jästa mjölkdrinkar (t ex Actimel®)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Vilken sorts yoghurt och fil brukar du äta? *Vänligen markera endast ett svar.*	a) Med 3% fett ○ 1 b) Med 1,5 eller 0,5% fett ○ 2 c) All slags yoghurt ○ 3 d) Jag äter inte någon yoghurt ○ 4 e) Jag vet inte ○ 7						

Under den senaste månaden...	Aldrig/ mindre än en gång i veckan	1-3 gånger per vecka	4-6 gånger per vecka	1 gång per dag	2 gånger per dag	3 gånger per dag	4 eller fler gånger per dag
Fisk							
Fiskkonserver (t ex tonfisk och makrill i tomatsås)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Fisk - kokad, grillad ugnsbakad, rå, inte stekt eller panerad	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Fisk, stekt och/eller panerad (t ex fiskpinnar, fiskbiffar, panerad fiskfilé)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kött och köttprodukter							
Pålägg, kallskuret och köttprodukter som är förberedda för tillagning (t ex salami, falukorv)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kött, kokt, grillat, ugns-bakat, inte stekt, utan pa-nering (t ex nötkött, fläsk)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Stekt kött (t ex nötkött, fläsk)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7

Fågel, kokt, grillad, ugnsbakad, inte stekt, ingen panering (t ex kyckling, kalkon)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Stekt fågel (t ex kyckling, kalkon)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7

Under den senaste månaden…	Aldrig/ mindre än en gång i veckan	1-3 gånger per vecka	4-6 gånger per vecka	1 gång per dag	2 gånger per dag	3 gånger per dag	4 eller fler gånger per dag
Ägg och majonnäs							
Stekt ägg eller äggröra	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kokade eller pocherade ägg	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Majonnäs eller majonnäsbaserade produkter	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Sojaprodukter och liknande som ersätter kött							
Tofu, tempeh, quorn, sojakött, sojakorv, sojamjölk	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Ost							
Skivbar ost (t ex Herrgård, Prästost, Grevé)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Bredbar ost (t ex keso, mjukost)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Riven ost	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Äter du vanligtvis ost med låg fetthalt? *Vänligen markera endast ett svar.*	a) Ja .... ○ 1 b) Nej .... ○ 2 c) Jag äter inte någon slags ost .... ○ 3 d) Jag vet inte .... ○ 7						

Under den senaste månaden…	Aldrig/ mindre än en gång i veckan	1-3 gånger per vecka	4-6 gånger per vecka	1 gång per dag	2 gånger per dag	3 gånger per dag	4 eller fler gånger per dag
Bredbara produkter							
Sylt, marmelad, honung	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7

Choklad- eller nötbaserat Pålägg (t ex jordnötssmör, Nutella)	1	2	3	4	5	6	7
Smör och margarin att ha på smörgåsen (fetthalt mer än 60%, t ex Bregott, smör)	1	2	3	4	5	6	7
Margarin med låg fetthalt att ha på smörgåsen (fetthalt 60% eller mindre t ex Lätta, Becel)	1	2	3	4	5	6	7
Ketchup (även till pommes frites)	1	2	3	4	5	6	7

Under den senaste månaden…	Aldrig/ mindre än en gång/ vecka	1-3 gånger /vecka	4-6 gånger /vecka	1 gång /dag	2 gånger /dag	3 gånger /dag	4 gånger eller mer/dag
Matolja för matlagning och salladsdressing							
Olivolja	1	2	3	4	5	6	7
Spannmålsprodukter							
Vitt bröd, ljust knäckebröd	1	2	3	4	5	6	7
Fullkornsbröd, grovt knäckebröd	1	2	3	4	5	6	7
Vit pasta, nudlar, ris	1	2	3	4	5	6	7
Fullkornspasta, råris	1	2	3	4	5	6	7
Maträtt av malda spannmål (t ex mannagrynsgröt, polenta, couscous, bulgur)	1	2	3	4	5	6	7
Pizza som lunch eller middag	1	2	3	4	5	6	7
Hamburgare (som inte är hemmagjorda), korv med bröd, kebab, wrap, falafel, smörgåsar, fyllda baguetter	1	2	3	4	5	6	7
Mellanmål/Snacks							
Nötter och frön (t ex valnötter, solrosfrön)	1	2	3	4	5	6	7
Torkad frukt (t ex russin, torkade bär)	1	2	3	4	5	6	7
Salta snacks (t ex chips, ostbågar, salta pinnar, popcorn)	1	2	3	4	5	6	7
Ostpaj, korvpaj, pizzabitar, pirog och liknande	1	2	3	4	5	6	7
Choklad (t ex Mars, Kexchoklad, Geisha)	1	2	3	4	5	6	7

Övrigt godis (t ex lösgodis, bilar, geléhallon)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Kakor, bakelser, pannkakor, våfflor eller mousse (t ex sockerkaka, mariekex, chokladpudding, kanelbullar, chokladbollar)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7
Glass (mjölk- eller fruktbaserad)	○ 1	○ 2	○ 3	○ 4	○ 5	○ 6	○ 7

Appendix II. The Healthy Dietary Adherence Score

The Healthy Dietary Adherence Score (HDAS) was developed using total weekly frequency of specific foods and/or beverages divided by the child's total reported consumption frequency of all foods and/or beverages. This provides standardization on number of foods and beverages reported and consumption frequency, avoiding misclassification of children into low or high adherence because they consume all types of food frequently. Moreover, this corrects for variation in eating occasions away from home that were not captured by the Food Frequency Questionnaire (FFQ). Based on answers to the question "How often does your child usually eat at home or at other people's home?" reports of less than 3 meals per week were excluded. The HDAS aimed to capture adherence to food-based dietary guidelines including: limiting the intake of refined sugars, reducing fat intake, especially of saturated fat, choosing whole meal when possible, consuming 400-500 gram of fruit and vegetables per day and fish 2-3 times per week. Hence, it contains five components: sugar, fat, whole meal, fruit and vegetables, and fish. Each component has a minimum score of 0 and a maximum score of 10. To obtain the total HDAS, scores for all five food groups were summed up to a maximum score of 50, where the highest score indicates the highest possible adherence to the dietary guidelines.

Sugar component: To calculate the intake of sugar we used the sugar propensity score developed earlier in IDEFICS. The intake frequency of all food items containing sugar (sweetened breakfast cereals, sweetened drinks, fruit juices, sweetened milk, sweet yoghurt and fermented milk beverages, fruit with added sugar, jam and honey, chocolate or nut based chocolate, candy bars, loose candies, marshmallows, biscuits, cakes, pastries, puddings, ice cream, milk or fruit based bars) have been summed up and divided by the total intake frequency of all food items reported. At the time of the development of the HDAS, the World Health Organization (WHO) stated that the intake should be less than 10E% from refined sugars. It was decided to set the highest score of 10 if the intake frequency of sugar containing food items was 10% of the total intake frequency or less. The quartiles of the sugar propensity score were counted. The upper limit of the lowest quartile (15.7%) was used to establish the score of 0. The scores between 10% and 15.7% were equally distributed in steps by 0.6%. Children with a sugar propensity score above the first quartile (15.7%) received the score of 0 (Table 1).

Table 1. Scoring of the sugar component

Proportion of sugar containing food items	Score
<= 10%	10
>= 10% to <10.6%	9
>= 10.6% to <11.3%	8
>= 11.3% to <11.9%	7
>= 11.9% to <12.6%	6
>= 12.6% to <13.2%	5
>= 13.2% to <13.8%	4
>= 13.8% to <14.5%	3
>= 14.5% to <15.1%	2
>= 15.1% to <15.7%	1
>= 15.7%	0

Fat component: The principle is that healthier low fat versions of food items are set in relation to less healthier versions. The intake frequencies of food items generally low in fat (cooked vegetables, eggs, fish, meat and low fat dairy products and spread) was summed up. This sum was set in relation to the total intake frequency of both cooked and fried vegetables, eggs, fish, meat and low and high fat dairy products and spread. The proportion of the total intake frequency of low fat food items/week of the total intake frequency of food items containing fat per week was calculated according to the formula 'total intake frequency of healthy food items a week / total intake frequency of all food items a week x 100'. For 100% low fat food items the score 10 was given and for less than 10% the score 0 was given. The scores between 0 and 10 were distributed over equidistant categories of the healthy food item score (Table 2).

Whole meal component: The proportion of the total intake frequency of "whole meal bread, dark roll, dark crispbread" of the total intake frequency of bread ("white bread, white roll, white crispbread" + "whole meal bread, dark roll, dark crispbread") was counted. The scoring was done in the same way as for fat, i.e. for 100% whole meal food items the score 10 was given and 0 for less than 10%. Similar to the fat score, the whole meal scores between 0 and 10 were distributed over equidistant categories of the healthy food item score (Table 2).

Table 2. Scoring of the fat and whole meal components

Proportion of healthy food items	Score
= 100%	10
>= 90% to < 100%	9
>= 80% to < 90%	8
>= 70% to < 80%	7
>= 60% to < 70%	6
>= 50% to < 60%	5
>= 40% to < 50%	4
>= 30% to < 40%	3
>= 20% to < 30%	2
>= 10% to < 20%	1
< 10%	0

Fruit and vegetables component: The total fruit and vegetable intake frequency was calculated as the intake frequency of fresh fruit without added sugar + half of the intake frequency of fresh fruit with added sugar + cooked vegetables, potatoes and beans + legumes + raw vegetables. Because parents had no control over meals eaten at pre-schools and schools estimations were made for these missing meals. According to the question "How often does your child usually eat at pre-school or school meals?" the amount of meals eaten at pre-school or school was calculated. The assumption was that children eat breakfast, lunch, dinner and at least one snack during the day, which adds up to 28 meals a week. The intake frequency per week reported in the FFQ was set in relation to the missing meals eaten in pre-school or school and converted into the amount the child would have eaten if school meals were included. The rationale behind the scoring for fruit and vegetables was that the recommendation for fruit and vegetables are five portions a day, i.e. 35 portions a week. The number of 35 portions or more was considered as optimal and received the highest score of 10. Less than two portions per week was scored as 0. The scores between 0 and 10 were distributed over equidistant categories of the healthy food item score (Table 3).

Table 3. Scoring of the fruit and vegetables component

Intake frequency of fruit and vegetables per week	Score
>= 35	10
>= 31.4 to < 35	9
>= 27.6 to < 31.4	8
>= 24 to < 27.6	7
>= 20.4 to < 24	6
>= 16.8 to < 20.4	5
>= 13 to < 16.8	4
>= 9.4 to < 13	3
>= 5.8 to < 9.4	2
>= 2.0 to < 5.8	1
< 2.0	0

Fish component: The total intake frequency for fish was calculated in a similar way as for fruit and vegetables. The parents reported the intake of cooked and fried fish which was added and extrapolated for the missing meals. Fish intake was scored as the highest at an intake of 2.5 times/week; a value which corresponds to the recommendation of 2-3 portions a week. No fish intake was scored as 0. The remaining interval between 0 and 2.5 times/week was subdivided into nine intervals of size 0.3 times/week (Table 4).

Table 4. Scoring of the fish component

Intake frequency of fish/week	Score
>= 2.5	10
>= 2.25 to < 2.5	9
>= 2 to < 2.25	8
>= 1.75 to < 2	7
>= 1.5 to < 1.75	6
>= 1.25 to < 1.5	5
>= 1 to < 1.25	4
>= 0.75 to < 1	3
>= 0.5 to < 0.75	2
>= 0.25 to < 0.5	1
< 0.25	0