Family-based behavioral treatment of children with severe obesity

Effectiveness and perceived barriers

Hanna Flækøy Skjåkødegård

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

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This thesis is based on data derived from the family-based behavioral treatment of childhood obesity (FABO) study. The FABO study was funded by the Western Norway Regional Health Authority (Helse Vest) and conducted at the Obesity Outpatient Clinic, Haukeland University Hospital. This PhD project was funded by the University of Bergen.

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Abstract

Background: Few studies have evaluated the effect of family-based behavioral treatment (FBT) in real-world health-care settings.

Aims: To evaluate the effectiveness of family-based behavioral social facilitation treatment (FBSFT), an enhanced FBT program, for pediatric obesity, compared with treatment as usual (TAU); to assess for perceived barriers to treatment participation in families, as well as differences in sleep behaviors among children with severe obesity, compared to peers with normal weight; and to examine the relationship between sleep and other behavioral factors known to cause obesity in children.

Methods: This was a randomized controlled trial using a wait-list control design. A total of 114 children (mean age 12.6 years) with severe obesity were recruited from the Obesity Outpatient Clinic, Haukeland University Hospital. A matched group of children with normal weight (n = 85) were also recruited for case-control comparison of sleep behaviors. Measurements included body mass index (BMI)-related metrics, objective sleep/physical activity measures, and relevant questionnaires (the Dutch Eating Behavior Questionnaire and the Barriers to Treatment Participation Scale).

Results: A significantly greater decrease in BMI standard deviation scores was obtained from pre- to posttreatment with FBSFT, compared to TAU, with a between-group difference of 0.19 units (p <0.001). Noncompleters of FBSFT reported significantly more barriers to participation related to stressors and obstacles (p = 0.010) and perceived relevance of treatment (p <0.001), compared to completers. Children with severe obesity had significant later sleep timing, compared to normal-weight peers (p <0.001). Later sleep timing was also associated with more screen time (p = 0.030) and less time in moderate-to-vigorous physical activity (p = 0.015).

Conclusion: Significantly greater improvement in BMI-related outcomes was obtained with FBSFT, compared to TAU. Families were more likely to terminate FBSFT prematurely when facing stress-related barriers or when treatment was not meeting their expectations/needs. Sleep timing could represent an independent risk factor for pediatric obesity. The study results here support a more widespread implementation of FBSFT, and emphasize the importance of investigating barriers to participation to enhance retention rates.

Abstrakt

Bakgrunn: Få studier har prøvd ut familiebasert kognitiv atferdsterapi (FBT) innenfor den offentlige helsetjenesten.

Mål: Sammenligne effekten av ett FBT program med forsterket fokus på sosialt miljø (FBSFT) og standard behandlingen (TAU) gitt ved Poliklinikk for overvekt, Haukeland Universitetssykehus (PFO), og å undersøke barrierer for deltakelse i FBSFT. Videre å undersøke forskjeller i søvnatferd hos barn med alvorlig fedme og normalvekt, og om søvn er relatert til annen ugunstig livsstils-atferd.

Metode: Randomisert kontrollert studie med venteliste-kontroller som inkluderte 114 barn (gjennomsnittsalder 12,6 år) med alvorlig fedme rekruttert ved PFO. Videre ble en gruppe barn med normalvekt (n = 85) rekruttert for en kasus-kontroll-studie av søvnatferd. Målemetoder benyttet inkluderte vektrelaterte mål, objektive søvn/aktivitetsmål og spørreskjemaer («Nederlandsk spørsmålsliste om spiseatferd» og «Barrierer for behandlingsdeltakelse skalaen»).

Resultater: FBSFT reduserte KMI standardavvik scoren til deltakerne signifikant mer fra før- til etter behandling enn TAU (p <0.001), forskjellen i endring mellom gruppene var på 0.19 standardavvik. Familier som avsluttet FBSFT prematurt rapporterte signifikant flere barrierer relatert til stress/hindringer (p = 0.010) og opplevd relevans av behandlingen (p <0.001) sammenlignet med familiene som fullførte behandlingen. Barn med alvorlig fedme hadde signifikant senere tidspunkt for søvn sammenlignet med normalvektige barn (p < 0.001), og i barnepopulasjonen totalt sett var senere tidspunkt for søvn relatert til mer skjermtid (p = 0.030) og mindre tid i fysisk aktivitet av moderat til høy intensitet (p = 0.015).

Konklusjon: FBSFT gav bedre effekt på vektrelaterte mål sammenlignet med TAU, et funn som støtter videre implementering av FBSFT i den offentlige helsetjenesten. Videre er det sannsynlig at familier avslutter FBSFT prematurt dersom de opplever mer stressrelaterte barrierer og at behandlingen ikke møter familiens forventinger og behov, dette funnet tydeliggjør at det er viktig å kartlegge hindringer for deltakelse hos pasientene. Senere tidspunkt for søvn i løpet av døgnet utgjør muligens en risikofaktor for fedme hos barn, og denne sammenhengen må undersøkes videre.

Contents

List of publications

Paper I

Skjåkødegård HF, Danielsen YS, Frisk B, Hystad SW, Roelants M, Pallesen S, Conlon RPK, Wilfley DE, Juliusson PB. Beyond sleep duration: Sleep timing as a risk factor for childhood obesity. *Pediatr Obes*. 2021;16:e12698.

Paper II

Skjåkødegård HF, Conlon RPK, Hystad SW, Roelants M, Olsson SJG, Frisk B, Wilfley DE, Danielsen YS, Juliusson PB. Family-based treatment of children with severe obesity in a public healthcare setting: Results from a randomized controlled trial. *Clin Obes*. 2022;12(3):e12513.

Paper III

Skjåkødegård HF, Hystad S, Bruserud I, Conlon RPK, Wilfley D, Frisk B, Roelants M, Juliusson PB, Danielsen YS. Perceived barriers in family-based behavioural treatment of paediatric obesity—Results from the FABO study. *Pediatr Obes*. 2022:e12992.

Related publications

Skjåkødegård HF, Danielsen YS, Morken M, Linde SRF, Kolko RP, Balantekin KN, Wilfley DE, Juliusson PB. Study protocol: A randomized controlled trial evaluating the effect of family-based behavioral treatment of childhood and adolescent obesity—The FABO-study. *BMC Public Health*. 2016;**16**(1):1106.

Danielsen YS[*], **Skjåkødegård HF**[*], Mongstad M, Hystad SW, Olsson SJG, Kleppe M, Juliusson PB, Bente Frisk. Objectively measured physical activity among treatment seeking children and adolescents with severe obesity and normal weight peers. *Obes Sci Prac*. 2022:1–10. doi: 10.1002/osp4.624.
([*] Danielsen YS and Skjåkødegård HF have shared first authorship.)

Danielsen YS, **Skjåkødegård HF**, Bjorvatn B, Juliusson PB, Pallesen S. Polysomnographic comparison of sleep in children with obesity and normal weight without suspected sleep-related breathing disorder. *Clin Obes*. 2022;**12**(1):e12493.

Løkling HL, Roelants M, Kommedal KG, **Skjåkødegård H**, Apalset EM, Benestad B, Morken MH, Hjelmesaeth, Juliusson PB. Monitoring children and adolescents with severe obesity: body mass index (BMI), BMI *z*-score or percentage above the International Obesity Task Force overweight cut-off? *Acta Paediatr*. 2019;**108**(12):2261–6.

Skodvin VA, Lekhal S, Kommedal KG, Benestad B, **Skjåkødegård HF**, Danielsen YS, Linde SRF, Roelants M, Hertel JK, Hjelmesæth J, Juliusson PB. Lifestyle intervention for children and adolescents with severe obesity—results after one year. *Tidsskr Nor Laegeforen*. 2020;**140**(9). doi: 10.4045/tidsskr.19.0682.

Abbreviations

BMI	body mass index
BMI SDS	body mass index standard deviation score
BTPS	Barriers to Treatment Participation Scale
CBT	cognitive behavioral therapy
CDC	Centers for Disease Control and Prevention
CI	confidence interval
COMPASS	Comprehensive Maintenance Program to Achieve Sustained Success (study)
CONSORT	Consolidated Standards of Reporting Trials
Covid-19	coronavirus disease 2019
DEBQ	Dutch Eating Behavior Questionnaire
DEXA	dual-energy X-ray absorptiometry
EASO	European Association for the Study of Obesity
FABO	family-based behavioral treatment of childhood obesity
FBSFT	family-based behavioral social facilitation treatment
FBT	family-based behavioral treatment
IOTF	International Obesity Task Force
MET	metabolic equivalent of the task
MVPA	moderate-to-vigorous physical activity
NCD	noncommunicable disease
NFFF	Norwegian Association for the Study of Obesity
NSF	National Sleep Foundation
OSAS	obstructive sleep apnea syndrome
RCT	randomized controlled trial
SD	standard deviation
SDS	standard deviation score
SEM	social ecological model
TAU	treatment as usual
WHO	World Health Organization
YEDE-Q	Youth Disorder Examination Questionnaire

1. Introduction

1.1 Background for the FABO study

In 2010, the Norwegian Directorate of Health released national guidelines for the prevention and treatment of overweight and obesity in children.[1] The guidelines recommended the use of more structured, family-based behavioral treatments for children with severe obesity.[1] However, both the availability of such programs and training opportunities for health-care providers in treatment delivery were limited. In this context, the family-based behavioral treatment of childhood obesity (FABO) study[2] was set up, with the purpose to implement, evaluate, and make available this type of family-based behavioral programs for health-care services in Norway. The work presented in this doctoral thesis includes data from the FABO study, which involved delivery of family-based behavioral social facilitation treatment (FBSFT) to children and adolescents aged 6–18 years referred to the Obesity Outpatient Clinic at Haukeland University Hospital in Bergen, Norway between 2014 and 2018.

1.2 Pediatric obesity

1.2.1 Obesity: A historical perspective

Historically, there has been limited understanding from the public and health-care providers in general of the adverse medical, psychological, social, and societal consequences of obesity, as well as little recognition of the condition as a chronic, relapsing disease. Ten years ago, the American Medical Association declared obesity a disease requiring treatment and prevention efforts, which was seen as controversial at the time.[3,4] In 2015, the European Association for the Study of Obesity (EASO) issued a position statement on the importance of classifying obesity as a chronic disease in children and adolescents,[5] a move in the classification of pediatric obesity that has been followed over the following years by many European countries.[5,6] In 2020, the European Commission officially recognized obesity as a chronic disease, with a commitment to prioritize focus on obesity as a major noncommunicable disease (NCD).[7] In Norway, the Norwegian Association for the Study of Obesity

(NFFF) has been advocating for political recognition of obesity as a chronic and complex disease.[8] Recognition of obesity as a chronic disease has strong implications in terms of prevention, diagnostics, treatment, and research.[7]

1.2.2 Definitions

The World Health Organization (WHO) defines overweight and obesity as "abnormal or excessive fat accumulation that may impair health."[9]

Overweight and obesity are commonly classified using body mass index (BMI), measured in kg/m^2.[9] The BMI provides an easy-to-measure indirect estimate of body fat content that has been shown to correlate well with future health risks and excess adiposity in children with obesity.[10–12] By contrast, methods for direct measurement of the amount and distribution of adipose tissue, such as dual-energy X-ray absorptiometry (DEXA), are costly and not readily available for routine use.[11,12]

Because of increasing height and weight with growth, the norms for BMI in children and adolescents are age- and sex-dependent, and values must be compared with age- and sex-adjusted population growth references.[13,14] There is currently no international consensus on a specific growth reference/cutoff point to use for defining overweight and obesity in children, with several country-specific, as well as international, growth references using different cutoffs available.[15] The three most commonly used cutoffs internationally are those from the International Obesity Task Force (IOTF),[16,17] WHO,[18] and US Centers for Disease Control and Prevention (CDC).[19] These three references are based on different data sources, and will each provide different prevalence rates of overweight and obesity when applied to the same group of children.[13] The IOTF reference for ages 2–18 years defines overweight as IOTF-25, obesity as IOTF-30, and severe obesity as IOTF-35 (equivalent to a BMI of 25, 30, and 35 kg/m^2, respectively, at 18 years).[17] The WHO reference for ages 5–19 years uses age- and sex-adjusted standard deviation scores (SDS) to define overweight and obesity; overweight is defined as >1 standard deviation (SD) above the WHO growth reference median, and obesity as >2 SD above the median.[18] The CDC reference, on the other hand, defines overweight and obesity in children aged >2 years on percentiles on the CDC growth chart; a BMI above the 85th percentile is defined as overweight, and above the 95th percentile as obesity.[19] Many countries use

their own national growth references for tracking changes in BMI during the course of treatment, but often use one of these three international cutoffs for defining overweight and obesity. In Norway, the IOTF cutoffs are incorporated into the national BMI charts.[20]

The BMI SDS (i.e., the number of SDs from the mean) is often used to follow a child's growth over time.[21] However, BMI SDS may be weakly associated with other measures of adiposity in children with the most severe forms of obesity, since a wide range of high BMI values corresponds to a relative narrow range of BMI SDS in an age-dependent manner.[22,23] Therefore, alternative methods, such as determining the percentage above the IOTF definition of overweight (%IOTF-25), have been suggested as more appropriate measures of adiposity changes in youth with severe obesity.[21,22]

1.2.3 Prevalence

According to the *WHO European Regional Obesity Report 2022*, the overall prevalence of overweight and obesity has been consistently rising in the WHO European Region.[7] None of the 53 member states, including Norway, are on track to meet the target of halting the rise of obesity by 2025.[7] In 2019, the World Obesity Federation published an atlas of childhood obesity that provides the following prevalence predictions in 2030: 254 million children and youth aged 5–19 years living with obesity worldwide, with 133 089 children with obesity in the same age group in Norway.[24]

In 1975, the global prevalence of obesity in 5- to 19-year olds was <1%, in contrast to an increase to 5.6% in girls and 7.8% in boys in 2016.[25] Norwegian prevalence studies have reported that 15–21% of children aged 8–15 years either are overweight or live with obesity.[26–29] The latest study (UngKan)[27] reported that among 9-year olds, 18% presented with overweight and 3% with obesity, while the prevalence among 15- to 16-year olds were 14% and 3%, respectively. From the 2000s, the overall prevalence of childhood obesity has plateaued in many high-income countries, including Norway. However, the prevalence of severe obesity in children has continued to increase.[14,30–32] The onset of the coronavirus disease 2019 (Covid-19) pandemic in 2020 and the ensuing strategies to contain the outbreak,

including lockdown implementations, together with virtual schooling and isolation measures, have markedly changed day-to-day living for children and adolescents, which has further exacerbated the already high prevalence of childhood obesity.[33–35] A recent study of 432 302 children in the United States found that the rate of BMI increase almost doubled during the Covid-19 pandemic, compared to the prepandemic period, particularly in children with overweight or obesity.[33]

1.2.4 Complexity

Obesity results from a chronic imbalanced energy state, whereby energy intake exceeds energy expenditure.[9,36] This basic fact is likely the main reason for the oversimplified understanding of obesity that is often present among the general public and that, in turn, contributes to the stigmatizing advice often given: "It's simply eating less and exercising more." In reality, the nature of obesity as a disease is heterogeneous and multifactorial.[14,37,38] The imbalanced energy state is driven by a complex interplay of multiple factors, including genetic predisposition and physiological, environmental, and psychological factors.[31,39]

Given the influence of various factors across multiple levels on the development and maintenance of obesity in children, the social ecological model (SEM)[40] has been shown to be a useful theoretical framework for understanding the interactions among these factors, and their effects, in pediatric obesity.[14,41–43] Using the SEM, these determinants can be categorized into different levels (individual, interpersonal, institutional, community, and policies)[43] that, together, describe the multifactorial interactions associated with pediatric obesity.[44] Figure 1 shows a recent SEM that aims to decipher the dynamic interrelationships among various factors to better understand the complex public health challenges of the pediatric obesity epidemic.[14]

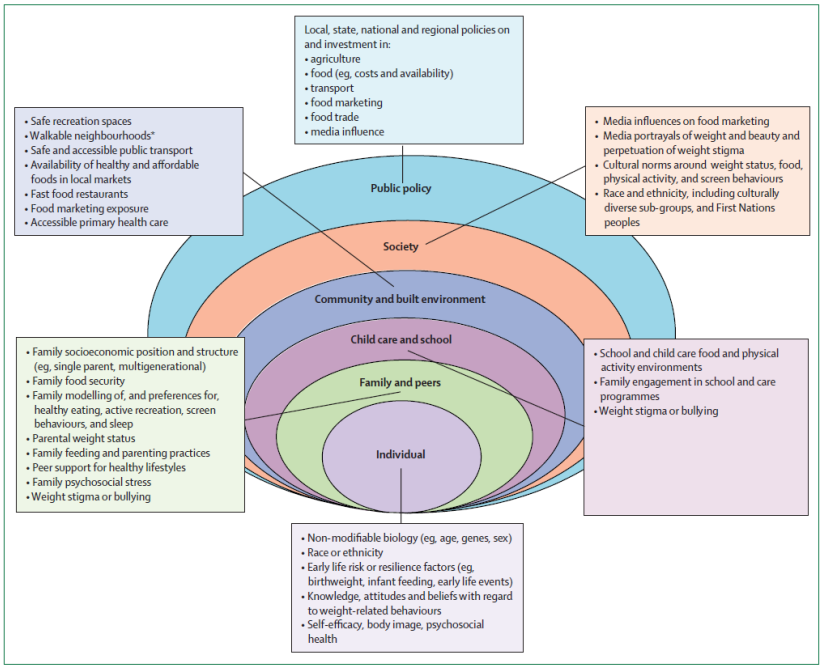

Figure 1. A socioecological model for understanding the dynamic interrelationships between various personal and environmental factors influencing child and adolescent obesity. * Defined as being traversable on foot, compact, physically enticing, and safe. The model was adapted from Centers for Disease Control and Prevention social-ecological framework for prevention.[45] Reprinted from *Lancet Diabetes Endocrinol*, 10/5, Jebeile H, Kelly AS, O'Malley G, Baur LA., Obesity in children and adolescents: epidemiology, causes, assessment, and management, 351-65. Copyright 2022, with permission from Elsevier.

1.2.5 Consequences

There is a vast amount of literature on studies focusing on the consequences of pediatric obesity.[14,36,46–48] As depicted in Figure 2, pediatric obesity is associated with multiple comorbidities and health issues,[42] resulting in a heavy burden on the individual child, as well as on society.[14,36,46]

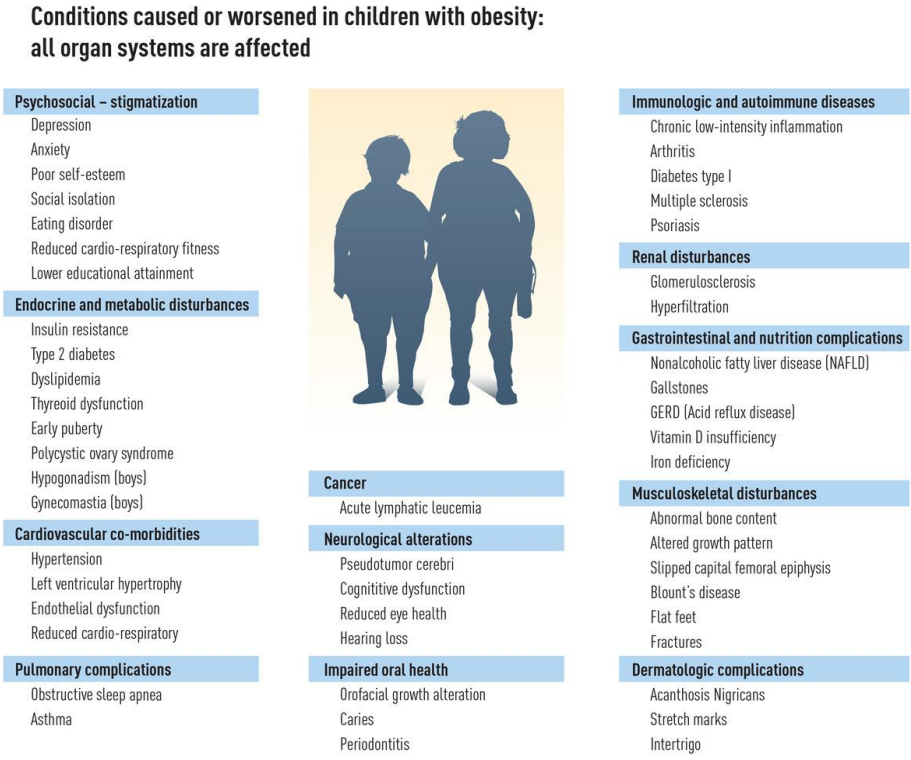

Figure 2. Diseases and conditions caused or aggravated by obesity during childhood and adolescence. Reprinted from *J Intern Med*, 292/6, Marcus C, Danielsson P, Hagman E. Pediatric obesity—Long-term consequences and effect of weight loss, 870-91, Copyright 2022, with permission https://creativecommons.org/licenses/by-nc/4.0/.

Complications linked to pediatric obesity may present immediately or later in life.[14,46] With its rising prevalence, somatic conditions that previously were more typically found in adulthood are becoming more common in youth, including prediabetes and type 2 diabetes.[48–50] Children's weight trajectories track into adulthood,[51,52] and the development and persistence of obesity predict the onset of other major noncommunicable diseases.[48] Pediatric obesity affects almost all organ systems, with long-term negative health consequences, as a result of the interactions of many different contributing factors. For example, obesity affects the immune system, both directly due to immunological effects in the adipose tissue and indirectly

through endocrinological alterations. The altered immune system leads to long-term comorbidities such as metabolic diseases, cardiovascular disease, cancer, and autoimmune diseases.[46]

The link between pediatric obesity and all-cause mortality from middle age onwards has been established for some time,[53] but now pediatric obesity is also known to be associated with premature death in young adulthood.[49] Risk of death due to both somatic diseases and suicide is higher among young adults with pediatric obesity.[54]

It is worth noting that in childhood, the psychosocial consequences of obesity are most pronounced.[14] Weight stigma, a societal devaluation of people living with larger bodies, is highly prevalent in our society,[14,31,55,56] and comes from multiple sources, including peers, teachers, family members, the media and social media, entertainment, and health-care providers.[55,56] Children and adolescents with obesity are, to a great extent, victims of weight stigma through discrimination and social rejection via teasing and bullying.[55,56] Weight-based mistreatment leads to impaired psychological health, and increasingly longitudinal research is emphasizing weight stigma, not weight per se, as the cause of psychological distress.[55] Regarding the established link between pediatric obesity and mental health problems, such as depression and anxiety,[46,57,58] additional studies are warranted to fully understand the role of weight stigma and weight-based teasing and bullying as an underlying mechanism in this relationship.[55] The same mechanisms (i.e., weight-based stigma, not weight per se) may be operating in relation to academic difficulties[59] and mental and physical health consequences (including maladaptive eating behaviors, weight gain, lower physical activity, and substance abuse).[55]

1.3 Treatment approaches for pediatric obesity

Due to its complex pathophysiology, pediatric obesity requires multidisciplinary and specialist interventions.[60]

Relevant literature, and thereby knowledge, on treatment of pediatric obesity is rapidly expanding, resulting in increased therapeutic options available to patients.[50,61]

A broad search in PubMed database (performed on November 8, 2022) using the key terms “obesity” and “treatment,” and applying filters related to age (<18 years), generated 37 651 results, of which 32 584 were published between 2000 and 2022. Narrowing the search using the additional filters “clinical trial” and “randomized controlled trial” generated 5 164 results. Current treatment options include lifestyle interventions, pharmacological therapy, and metabolic and bariatric surgery.[14,46,61] Among these, lifestyle interventions targeting health behaviors are most widely used,[61] whereas pharmacological therapy and bariatric surgery to date have been contextualized as components of an integrated continuum of care for adolescents.[62] A recent study has reported promising results on a glucagon-like peptide-1 analog that induces weight loss by decreasing appetite in adolescents with obesity.[63] However, use of anti-obesity pharmacotherapy in adolescents is still understudied.[64,65] Current recommendation is to combine such treatment with a behavioral change component,[39,63] whereas bariatric surgery is considered as a last resort in cases of obesity among youth with adverse health effects.[39,66]

1.3.1 Lifestyle interventions

A great variety of different treatment approaches for pediatric obesity are classified as “lifestyle interventions,”[61] meaning that the term tells us which area (i.e., the child’s lifestyle) is being targeted, but not the method used.

Lifestyle interventions usually comprise diet, physical activity, and/or behavior change techniques as either a single or a multicomponent behavioral intervention program.[61] The two latest Cochrane reviews on diet and physical and behavioral interventions for pediatric obesity reported a change in BMI SDS of −0.06 units in children aged 6–11 years[67] and of −0.13 units in 12- to 17-year-old adolescents,[68] in favor of the intervention. In recent years, different digital solutions (e.g., mobile apps, online monitoring tools, video coaching sessions) to support in-person interventions have shown promising results.[69–71] Treatment programs targeting multiple lifestyle behaviors (e.g., diet, physical activity, sedentary behavior, sleep) and applying behavioral techniques in a family-based context are considered the first-line treatment for pediatric obesity and recommended by most expert committees and systematic reviews on pediatric obesity.[39,66–68,72]

1.3.2 Family-based behavioral treatment

Pediatric obesity is a familial challenge, as studies have shown there is a strong relationship between obesity among child and their parents.[73] In the 1970s, family-based behavioral treatment (FBT) programs were developed to modify the shared family environment, provide role models, and support child behavioral changes to promote weight management.[74,75] Over the last 40 years, Drs. Epstein and Wilfley, and colleagues have rigorously developed and tested FBT programs,[74–82] and to date, FBT is the most well-documented and effective treatment approach in relation to pediatric obesity.[66,72]

FBT is a standardized behavioral intervention[79] that targets both children and caregivers, and teaches them a set of principles and techniques for modifying obesogenic (behaviors thought to correlate with excess weight gain) lifestyle behaviors in relation to eating and physical/sedentary activity.[66] FBT incorporates theoretical elements from cognitive behavioral therapy (CBT).[68,83,84] The central theory in CBT is that cognitions affect behaviors and emotions, meaning that altering the cognition can result in behavioral and emotional change. Unfavorable behavior and thought patterns (e.g., dysfunctional and negative thoughts) are associated with pediatric obesity, thereby supporting the use of CBT techniques to address these health behavior patterns.[84] Commonly used behavioral strategies in FBT include self-monitoring, positive reinforcement, modeling, stimulus control, preplanning and problem-solving, cognitive restructuring, social facilitation, motivational techniques (e.g., goal setting), and relapse prevention.[72,82] Traditionally, FBT is delivered in a combination of group and individual sessions (child–parent dyads) to children aged between 7 and 12 years.[79] Further, the use of diet and activity plans based on a Traffic Light System is implemented as part of the treatment program, with green representing a favorable choice.[82]

1.3.3 Development of the intervention applied in the FABO study

The efficacy of FBT has been established through numerous studies,[72,75,79] with a pooled BMI SDS change of −1.20 units after 6 months of treatment reported from eight studies by Epstein's group.[75] However, few studies have been conducted in real-world health-care settings.[82] Therefore, evaluation of the generalizability of FBT

when conducted in a routine clinical practice setting with a less selective group of participants was needed.[82,85] Against this backdrop, an international collaboration was initiated by our team that led to the setting up of the FABO study.[86] Treatment delivered in the FABO study is based on a treatment manual developed by Dr. Wilfley's research group at Washington University in St. Louis, Missouri in the United States. The manual was developed as part of the Comprehensive Maintenance Program to Achieve Sustained Success (COMPASS) study,[80,81] and based on a social–ecological approach, taking into consideration the multifactorial interactions associated with pediatric obesity.[2,80] The treatment was named family-based behavioral social facilitation treatment (FBSFT) to reflect the incorporation of all the features of FBT, in addition to a broader focus on facilitation of healthy behaviors outside the family/home context (i.e., peer, community, and cross-contextual).[2]

1.4 Eating behavior and pediatric obesity treatment

Pediatric obesity is the most prevalent food-based disorder among youth worldwide, and parental eating behaviors is a key determinant of the condition.[87] Diet quality and quantity are key focus areas in pediatric obesity treatment. FBT and FBSFT focus on improvement in diet quality, in combination with a reduced, but healthy, calorie range for weight loss. A flexible eating style as part of a generally healthy diet, in which no foods are "forbidden," is the end goal.[88]

However, weight outcomes are determined by much more than simply the nutritional value of a meal. Evidence-based pediatric obesity treatments, including FBT/FBSFT, address eating behaviors in addition to diet quality and quantity. For example, important considerations include where, when, and how children eat, as well as addressing any possible underlying disordered eating behaviors.[89,90]

Unfavorable individual/family eating patterns are linked to obesity. Skipping breakfast seems to influence energy balance and dietary intake negatively, which has been associated with obesity in several studies,[87,91] as is eating in front of a screen.[92] By contrast, regular family meals and frequent meals throughout the day appear to positively influence body weight.[87]

There is a higher prevalence of disordered eating attitudes and behaviors among children with obesity than among their peers with normal weight.[88,93] Emotional, external, and restrained eating patterns are among the most frequently reported disordered eating patterns in youth with overweight/obesity.[89,94] Emotional eating occurs when food is used to regulate or avoid negative emotions; external eating is eating because food is available rather than because one is hungry (often related to availability of snacks and food with high sugar and fat content), whereas restrained eating is eating less than desired to lose or maintain body weight.[95] Disordered eating attitudes and behaviors can develop into full eating disorder syndromes such as binge eating disorder and bulimia nervosa.[88,96] A few studies have reported on the prevalence of clinical eating disorders in treatment-seeking children with obesity.[97] One study reported that among 160 adolescents presenting for treatment, 6.3% had binge eating disorder, while 24% reported binge eating with loss of control within the previous 3 months.[97,98] Another study reported that out of 41 adolescents with obesity who were enrolled in lifestyle treatment, 17 (41%) screened positive for a pathological eating disorder.[99]

The co-occurrence of obesity and disordered eating behaviors in the pediatric population[96] is likely driven by the shared underlying risk factors for the two conditions.[97,100,101] Figure 3 illustrates the individual and environmental correlates of obesity and disordered eating.[88] It is likely that cultural values and societal views related to body weight, size, and shape influence the risk of developing disordered eating behaviors. Weight bias, stigma, and discrimination are pervasive, negative influences to which individuals with obesity, including young children and adolescents, are subject, thus exacerbating the development and maintenance of disordered eating behaviors in efforts to achieve weight management.[88]

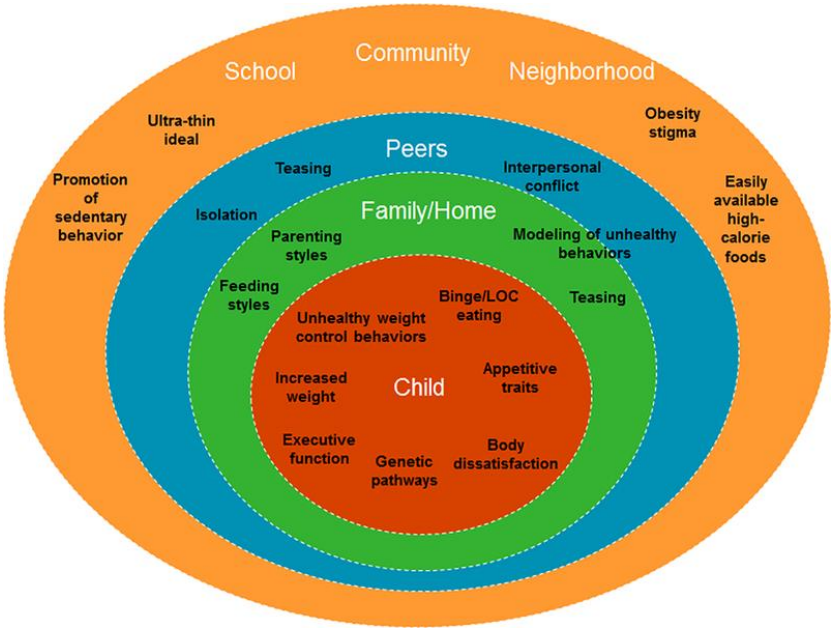

Figure 3. Individual and environmental correlates of disordered eating and obesity. Reprinted from *Curr Obes Rep*, 7/3, Hayes JF, Fitzsimmons-Craft EE, Karam AM, Jakubiak J, Brown ML, Wilfley DE. Disordered Eating Attitudes and Behaviors in Youth with Overweight and Obesity: Implications for Treatment, Copyright 2022, with permission from Springer Nature.

FBT has shown promising results in relation to reducing disordered eating attitudes and behaviors in children with obesity.[88,102,103] Hayes *et al*.[88] outlined different treatment components of FBT/FBSFT and how they may influence disordered eating in addition to excess weight. Of these, goal setting and preplanning allow for planning around disordered eating behavior triggers, while maintenance of consistent, healthy routines counteracts dysregulation.[88]

Given that much of the research has focused on disordered eating attitudes, behaviors, and symptoms, rather than on diagnosed clinical eating disorders, it is recommended that individuals with threshold eating disorders enter treatment designed for the eating disorder itself, rather than undergoing FBT for obesity or other weight management intervention.[97]

1.5 Movement behaviors

Physical activity of varying intensities, sedentary behavior, and sleep are mutually exclusive time-use components that together make up an entire 24-hour day.[104] Movement behaviors, both isolated and in combination,[104] to various extent have been investigated in relation to pediatric obesity.

1.5.1 Physical activity and pediatric obesity treatment

Modification of physical activity behavior is a key element in multicomponent obesity interventions,[61] but justification for its inclusion is shifting. Physical activity has the ability to influence our energy expenditure,[105] and traditionally it has been included in treatment to achieve weight reduction. Throughout the last decades, intervention studies have reported that increased activity levels alone are not sufficient to produce beneficial weight changes in children with obesity.[61] This has raised a debate concerning the role of physical activity in treatment of pediatric obesity and whether it is necessary to include a physical activity component at all.

Active play, movement, and physical activity are essential for healthy growth and development in children.[106] Therefore, the high rates of inactivity among children and adolescents are associated with an increased risk of health problems.[107,108] Children with obesity are particularly vulnerable, and research shows that they are less physically active than their peers with normal weight,[107,109,110] with emerging differences in moderate-to-vigorous physical activity from the age of 6.[111] Independent of weight reduction, the wide spectrum of benefits associated with increased levels of physical activity justifies its inclusion in treatment.[107,108] Effects are seen in terms of psychological, as well as physical, health profile, appetite and energy intake control, social function, and intake of healthy food.[108,112] Another important consideration is that activity behavior (physical and sedentary) tracks across the life span, with a corresponding impact on health.[108,113] Notably, desiring a higher fitness level or to be in better shape has been recognized as one of the greatest motivators for weight loss among adolescents with obesity.[114]

1.5.2 Sedentary behavior

Sedentary behaviors are characterized by low energy expenditure and typically comprise activities performed in a sitting, reclining, or lying posture.[115] Physical inactivity is not the equivalent, as this term refers to an activity level below the given physical activity recommendation.[108] Youth can engage in both high levels of physical activity and sedentary behaviors throughout the day.[108] Screen time is the most common sedentary behavior in children,[96] and likely the most worrisome subset of sedentary behavior in youth, compared to nonscreen behaviors (e.g., reading a book).[116] Recreational screen time seems to affect body weight[117] and is associated with adverse health outcomes, independent of physical activity level.[108,116] Research suggests that the relationship between screen time and obesity can be explained through the following mechanisms: insufficient/delayed sleep (screen replacing sleep and negative effect of blue light from the screen on the ability to fall asleep) and increased food intake and negative impact on food choices.[108,116,118]

Screens are omnipresent in our society.[116] Since the FABO study was initiated in 2012, the evolution of screens has been massive over the last years, as well as challenging for both treatment and research. Rapid advances in technology can, for example, quickly outdate tools for measuring screen time.[116] Two decades ago, the focus was on avoiding television in children's bedrooms—now smartphones and tablets have become an integrated part of youths' day-to-day living.

1.5.3 Sleep and pediatric obesity treatment

Only a few studies applying a family-based treatment approach for pediatric obesity have included a sleep component.[119,120] Emerging evidence, however, implicates that sleep behavior should be assessed and addressed as part of pediatric obesity treatment.

The first aspect of sleep behavior that was investigated in the context of pediatric obesity was sleep duration, followed by other sleep dimensions, including sleep timing (sleep onset, sleep offset, midpoint of sleep) and social jetlag (variability between sleep times on weekdays and weekends) in recent years.[121]

Cross-sectional and longitudinal studies, using subjective and objective sleep measures, have demonstrated that short sleep duration is a risk factor for pediatric

obesity.[121–123] Sufficient sleep duration, on the other hand, appears to protect against weight gain in children—one meta-analysis found that each additional hour of sleep was associated with a 21% decrease in the risk of overweight/obesity.[124]

Few studies have investigated the link between other sleep behaviors and pediatric obesity,[121,125] which is an important step for a more comprehensive understanding of how sleep contributes to the development and maintenance of obesity in children. A recent systematic review identified 17 cross-sectional, and three longitudinal, studies reporting on the association between sleep timing (including social jetlag) and adiposity measures in children aged 4–18 years. Only 11 of these studies used objective sleep measures.[125] The review concluded that findings were inconsistent, with very-low quality of evidence.[125]

The mechanisms driving the sleep–obesity relationship are poorly understood,[126] with no unifying patterns of findings that fully explain how sleep impacts weight regulation in youth.[127] Proposed mechanisms include sleep-related changes in food intake, eating behavior, sedentary behavior, and physical activity, changes in hormonal responses to hunger/satiety, and activation of inflammatory pathways.[108,127,128] Evidence are emerging within the field, but to date, the following findings appear to be the most consistent: experimental (but not cross-sectional) evidence linking restricted time in bed with increased caloric intake; cross-sectional results linking longer sleep duration with higher dietary quality; and cross-sectional findings suggesting a link between shorter sleep duration and later sleep timing and increased sedentary activity, screen time, and breakfast skipping.[127]

Further, children with obesity have an increased risk of developing sleep-related breathing problems, including obstructive sleep apnea syndrome (OSAS).[129] Untreated OSAS is associated with a wide range of negative health and behavioral consequences in the pediatric population.[129] Co-occurrence of obesity and sleep-related breathing problems can exacerbate the cardiometabolic consequences of both conditions.[130,131] However, sleep-related breathing problems often escape the attention of parents.[132] Therefore, objective assessment of sleep, as part of pediatric weight management, is needed to prevent sleep-related breathing problems from remaining unnoticed.

1.5.4 Lifestyle behavior interactions in relation to pediatric obesity

Obesogenic behaviors related to eating and movement (physical/sedentary/sleep) are shaped in different contexts, as outlined in the Social Ecological Model (Figure 1). Another interesting aspect is the interaction between the different lifestyle behaviors and how combinations of these behaviors are associated with pediatric obesity.[104,133] Currently, a few studies have explored these interactions; therefore, the association between lifestyle behavior interactions and pediatric obesity is largely unknown.[133] A study published in 2022 on 28 040 children aged 6–17 years found that adhering to age-appropriate recommendations on dietary intake, physical activity, sedentary activity, and sleep overall resulted in the lowest probability of having overweight/obesity, compared to meeting none, one, or a combination of two or three of these recommendations.[133]

In the context of treatment, it is plausible to think that for treatment-seeking children with obesity, lifestyle behaviors interrelate and form a negative circle of obesogenic behaviors. Therefore, a holistic view on behavioral patterns during the 24-hour day may be beneficial.[108] For movement behaviors, if time is devoted to one behavioral change, there must be an equal and opposite change in other behaviors.[134] From which activity time is taken might impact both health and weight—for example, an increase in physical activity is unlikely to be beneficial if time is taken from sleep as opposed to reducing screen time. In recent years, Canada, Australia, New Zealand, Finland, and Croatia have implemented 24-hour activity guidelines and recommendations.[134]

Recently, alignment of behavioral and social routines with underlying circadian rhythms has been put forward as having potentially particular importance in children's weight management.[135] It has been shown that misalignment of sleep timing and eating behaviors with circadian rhythms can cause alterations in hormones, thus affecting metabolic health.[135] The circadian perspective needs to be considered together with the already well-known components of pediatric obesity treatment to enhance weight management.[135]

1.6 Objectives of treatment and availability

Untreated, pediatric obesity is likely to progress with predictable morbidity and mortality.[50] When providing treatment, a critical question arises: what should be considered a successful outcome of pediatric obesity treatment for the individual child? There is no clear answer, given that pediatric obesity is a chronic disease and although much has been learned about the physiopathology, the condition remains difficult to treat.[7] One factor contributing to this paradox is that treatment often is short-term, with no clear concept of aftercare, when it is likely that structured chronic management strategies are needed.[136]

A favorable change in weight status, commonly assessed by BMI SDS, is often the primary outcome of pediatric obesity treatment. In specialist obesity clinics, treatment goals differ, depending on age and the degree of obesity. For a child who is still growing in height, weight stabilization might be sufficient, whereas for adolescents who have reached their final height, weight reduction may be the goal.[137] Still another question is: what represents a clinically relevant change in weight status? There is no consensus on thresholds that indicate clinically meaningful reductions in BMS SDS among youth.[138] Suggested reductions in BMI SDS required for improvements in cardiometabolic health range from 0.1 to 0.5,[139-140] and in Norwegian children aged 7–17 years even a stable/modest reduction in BMI SDS of 0.00–0.10 have been associated with improvement in several cardiovascular risk factors.[141] It may be reasonable to set a reduction of ≥0.25 BMI SDS as a cutoff point for a clinically relevant change.[69,139,142–145] However, it is important to bear in mind that even smaller BMI SDS reductions can result in improvement in health status, especially in cases of severe obesity or in the presence of obesity-related comorbidities.[141]

Besides body mass changes, outcome measures (e.g., body composition, waist circumference, reduction in obesity-related comorbidities) are less routinely measured, both in obesity clinics and research studies.[67,68,142] As a consequence, beneficial changes that occur independent of changes in body mass might be missed. A critical next step to address this issue is to incorporate examination of patient-

centered outcomes related to pediatric obesity management. One pediatric obesity study in school-aged children identified body image, bullying, and physical ability as outcomes considered relevant to the youths themselves.[146]

Further, it is important to acknowledge that worldwide, access to quality treatment for pediatric obesity remains limited. In 2019, the WHO Regional Office for Europe reviewed pediatric obesity management in 19 Member States (including Norway), and concluded that in general, health system responses to pediatric obesity were lacking.[136] Fragmentation of care, shortage of trained health-care personnel, inadequate funding, insufficient collaboration among relevant sectors, and lack of parental support and education are the most commonly cited barriers.[7] Therefore, political decisions are needed to improve treatment availability and access, and a system thinking incorporating individual, environmental, and policy changes are necessary to curb the prevalence of pediatric obesity.

1.7 Barriers and facilitators influencing patient participation in pediatric obesity treatment

Participation in a treatment program is influenced by multiple factors, which can act as either barriers or facilitators that would determine the likelihood of continued attendance. These interacting factors include both pretreatment participant and family characteristics, as well as factors that arise within the treatment program. Knowledge of such barriers and facilitators can strengthen treatment delivery and prevent attrition in treatment programs, a known challenge in pediatric obesity interventions that leads to poor disease control and compromises treatment effectiveness.[147–150]

Suggested pretreatment predictors of treatment attrition relate to demographic variables (i.e., age, sex, and initial body weight), socioeconomic status, previous dieting attempts, psychopathology, and body image.[147,151] However, findings have been inconsistent between studies, with varying definitions of attrition and heterogeneous samples and interventions.[152,153] A step away from solely investigating pretreatment predictors is to focus on the dynamic process of treatment program delivery and attendance, and on which factors need to be taken into consideration when guiding a family to treatment completion. Research in this area is scarce,

however. Grootens-Wiegers *et al*.[154] have developed an interesting theoretical model of barriers and facilitators that, at different treatment stages, can influence adherence to, and completion of, a pediatric obesity lifestyle intervention (Figure 4). The model is based on a narrative literature review, and the research group has further investigated the factors identified through semi-structured interviews and focus group discussions with children and parents, as well as therapists, participating in a lifestyle intervention.[154]

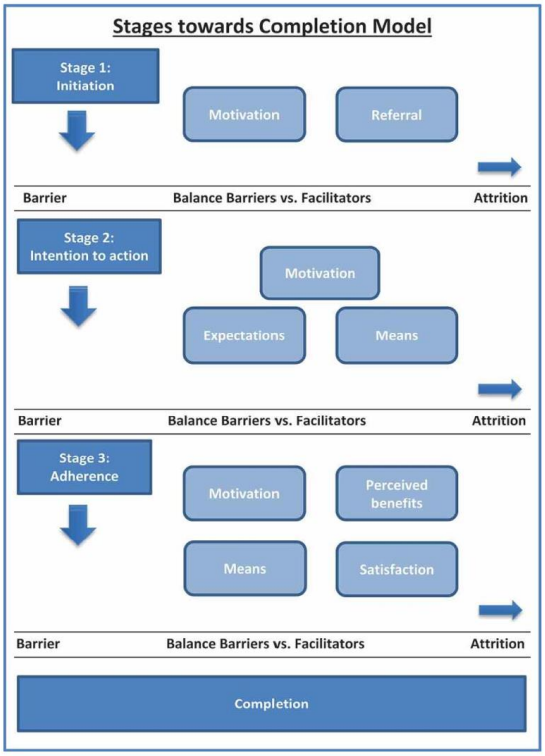

Figure 4. Main factors that influence adherence to, and completion of, pediatric obesity lifestyle treatment. Reprinted from *Int J Qual Stud Health Well-being*, 15/1, Grootens-Wiegers P, van den Eynde E, Halberstadt J, Seidell JC, Dedding C. The "Stages towards Completion Model:" what helps and hinders children with overweight or obesity and their parents to be guided towards, adhere to and complete a group lifestyle intervention, 1735093, Copyright 2020, with permission https://creativecommons.org/licenses/by-nc/4.0/.

This model provides an overview that could represent a useful tool when delivering FBT/FBSFT to families. Stages 1 and 2 comprise factors that impact the child/family's decision to start an intervention. These two stages are influenced by motivation, such as whether the child's weight is seen as a concern or not, the approach and attitude of the referrer, expectations concerning the contents and outcomes of the intervention, and the participant's means/resources to enter the intervention. In Stage 3, the child and family have started the intervention. Motivation continues to play a role and may fluctuate, based on other factors such as satisfaction with the intervention and perceived benefits. Satisfaction is based on the treatment content, the relationship with the treatment provider, and whether expectations are met. Perceived benefits are often related to treatment response (e.g., degree of weight loss). Means/resources include time, logistics, family income, and degree of support from social network. Notably, reduced means and/or lack of resources seem to be one of the most consistent barriers to treatment adherence.[154] Families attending interventions for pediatric obesity often live under complex circumstances, including single-parent households with multiple children, parental chronic illnesses, limited finances, and demanding jobs with irregular hours.[147,148,153–155]

This model highlights the need for treatment providers to build personal relationships with participants, to help the latter identify barriers and/or facilitators among the above-mentioned factors and to provide support towards practical solutions to overcome any recognized barriers.[154] The model builds on knowledge gained from lifestyle interventions for pediatric obesity in general, whereas little is known about the specific factors related to participation in an FBT/FBSFT program. Increased knowledge about treatment-specific factors is therefore much needed so that factors can be addressed that may influence children's and families' engagement and retention in treatment programs. This will help enhance the effectiveness of treatment interventions, thereby supporting greater treatment outcomes and ultimately addressing pediatric obesity more effectively.

1.8 Summary

Treatment interventions for pediatric obesity always include lifestyle changes. FBT represents a standardized behavioral treatment with documented efficacy. Investigating the effectiveness of enhanced FBT in a real-world health-care setting can potentially lead to better treatment options for children and adolescents living with obesity. A known high risk of attrition from pediatric obesity treatment, in combination with a limited understanding of within-treatment barriers that hamper treatment completion, means that it is crucial to explore perceived barriers when performing research on new treatment options. Further, it is of interest to investigate changes in key behavioral elements following a behavioral intervention. To date, little is known about how sleep is linked to obesity and other behavioral factors known to cause obesity in children, and whether unfavorable sleep behaviors can be changed with an FBT approach.

(Literature review completed in November, 2022)

2. Aims and objectives

The overall aims of the work presented in this thesis were: (1) to evaluate the effectiveness of an FBT program for pediatric obesity compared to treatment as usual (TAU) in a real-world health-care setting; (2) to gain further knowledge about families' perceived barriers to treatment participation; and (3) to investigate differences in sleep behavior among treatment-seeking children and adolescents with severe obesity, in comparison with normal-weight peers, and the relationship between various sleep dimensions and other behavioral factors that would increase the risk of school-aged children and adolescents developing obesity.

2.1 Specific objectives and hypotheses of the three publications presented in this thesis

Paper I: To compare sleep behaviors between children and adolescent with severe obesity and their peers with normal weight; to investigate how sleep duration, sleep timing (including social jetlag), and sleep problems are linked to behavioral factors known to cause obesity in children.
Hypothesis: Children and adolescents with severe obesity have less favorable sleep behaviors than their peers with normal weight. In addition to sleep duration, delayed sleep timing, social jetlag, and sleep problems are independently related to behavioral factors that place children and adolescents at a greater risk of developing obesity.

Paper II: To investigate the effectiveness of FBSFT, compared to TAU, on BMI-related metrics, sleep measures, physical activity, and eating behavior in pediatric obesity treatment delivered in a real-world health-care setting.
Hypothesis: FBSFT would yield greater improvements in BMI-related metrics and sleep and eating behavior, compared to TAU, with similar improvements in physical activity due to comparable focus on this component in the two treatment programs.

Paper III: To investigate perceived barriers to treatment participation in FBSFT among families who did or did not complete the intervention.

Hypothesis: There would be a higher level of perceived barriers among families who did not complete FBSFT.

3. Materials and methods

3.1 Study design and participants

The FABO study[2] was a nonblinded, randomized controlled trial (RCT) that used a wait-list control design. Via referral from their general practitioner, children with severe obesity (aged 6-18 years) and their families were invited to participate in the study after initial assessments at the Obesity Outpatient Clinic, Haukeland University Hospital. Where informed consent was obtained, the children and their families were randomized to either FBSFT (Arm A) or TAU (Arm B) followed by FBSFT 1 year later. The study design not only allowed for an RCT comparison of the two treatments (i.e., FBSFT versus TAU), but also provided an opportunity to investigate data from participants who received FBSFT after 1 year of TAU. Further, a group of children with normal weight was recruited from randomly selected schools in the Bergen municipality for a case-control comparison of key behaviors related to pediatric obesity. Figure 5 depicts the study design of the FABO study but only includes measurements used in Papers I, II, and III presented in this thesis. A full overview of measurements and evaluations included in the FABO study is given in the study protocol previously published.[2]

3.2 Child and adolescent sample, inclusion and exclusion criteria

3.2.1 Paper I

In total, cross-sectional data from 170 children (median age 12.4 years, range 5.8–17.1 years) were collected between February 2014 and March 2018: 85 children (50 girls) with severe obesity and 85 children with normal weight, matched by age, sex, and season of accelerometer measurement (April to September versus October to March). Participants with severe obesity were the first 85 children enrolled in the FABO study from the Obesity Outpatient Clinic, with baseline measurements used in Paper I. The criteria for admission into the FABO study were age between 6 and 18 years and severe obesity or obesity with weight-related comorbidity, defined by IOTF

BMI cutoff points (BMI ≥ IOTF-35) and (BMI ≥ IOTF 30) respectively.[16,17] Exclusion criteria included children/caregivers with somatic or psychiatric illnesses affecting participants' weight or adherence to the treatment program and children's current participation in another pediatric obesity treatment program.

Children with normal weight (BMI ≤ IOTF-25) were recruited from randomly selected schools in the catchment area for the FABO study, and letters of invitation were sent based on student class lists. Stratified random sampling ensured that the groups were matched in a ratio of 1:1 for age, sex, and season of accelerometer measurement. Seasonal matching was conducted to prevent bias in the comparison of physical activity and sleep data, as Norway has large seasonal differences in weather and daylight hours that may influence youths' activity and sleep patterns. No exclusion criteria were applied to the normal weight comparison group. If assessments showed that a child belonged to a weight category other than normal weight, measurements were carried out as normal without addressing the issue.

3.2.2 Paper II

Paper II reported on the RCT component of the FABO study. The study sample consisted of 114 children and adolescents with severe obesity (mean age 12.6 years; minimum to maximum 5.9–17.7 years), with 59 participants in Arm A and 55 in Arm B (Figure 5), enrolled in the FABO study between February 2014 and October 2018. All children with a BMI above the IOTF cutoff for severe obesity (≥35) or obesity (≥30) with weight-related comorbidity, who were referred to the Obesity Outpatient Clinic by their general practitioner within the given time frame, were invited to participate. FBSFT required participation from both the child and their caregiver, which meant at least one of the child's caregivers had to consent to active participation in the treatment. Exclusion criteria were as previously mentioned: severe somatic or psychiatric parental or child illness that affected weight or adherence to the treatment program or current participation in other pediatric treatment programs.

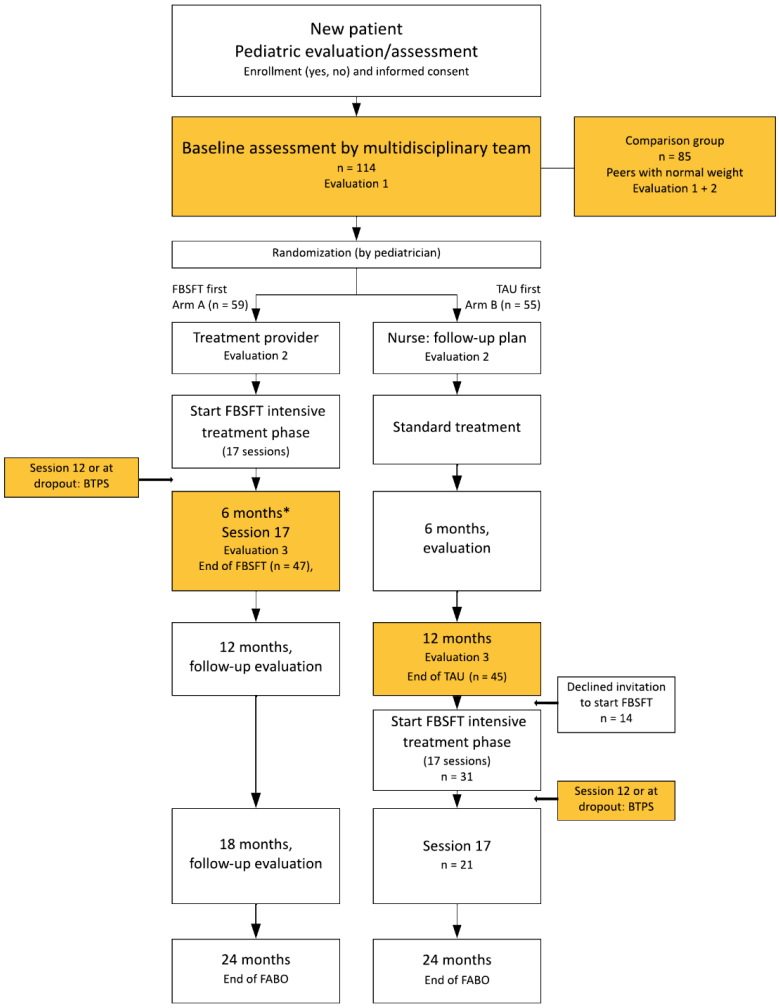

Figure 5. Flow chart showing the FABO study design. Colored boxes represent study measurement points included in the three publications presented in this thesis. Evaluation 1: anthropometry, actigraphy (sleep/physical activity), and demographic questionnaire. Evaluation 2: DEBQ. Evaluation 3: anthropometry, actigraphy (sleep/physical activity), and DEBQ. Abbreviations: BTPS, Barriers to Treatment Participation Scale; DEBQ, Dutch Eating Behavior Questionnaire. *Average treatment period was approximately 6 months. Modified from the FABO study protocol,[2] Copyright 2016, with permission https://creativecommons.org/licenses/by/4.0.

3.2.3 Paper III

In Paper III, observational data from families (n = 90) with children and adolescents with severe obesity (aged 6–18 years) included in the FBSFT part of the FABO study were analyzed. In Arm A, all participants took part in FBSFT (n = 59), whereas in Arm B, 31 of 55 participants chose to start FBSFT after 1 year of TAU.

3.2.4 Use of the term severe obesity

Participants recruited in the FABO study from the Obesity Outpatient Clinic included children and adolescents with obesity (IOTF BMI ≥30) with weight-related comorbidity. However, the term severe obesity is used when referring to our study population. All children included in the FABO study fulfilled the criteria for admission to a tertiary care obesity clinic within the public health-care service in Norway due to the severity of their obesity. Mean baseline BMI SDS among participants included in Papers I, II, and III was between 2.9 and 3.0, while a BMI SDS of ≥2.33 is often used to define severe obesity in children.[156–158] Therefore, we believe that the term severe obesity best describes the study population here.

3.3 Ethics

Written informed consent was obtained prior to inclusion. The consent was obtained from all participating adolescents older than 16 years, or otherwise from their caregiver(s) in addition to informed consent when the child was ≥12 years of age. The FABO study was approved by the Regional Committee for Medical and Health Research Ethics in Western Norway (number 2013/1300) and was registered at http://clinicaltrials.gov (NCT02687516).

3.4 Intervention

3.4.1 FBSFT

FBSFT is based on the fundamental principle of implementation of sustainable healthy lifestyle routines. Both the child and the caregiver(s) are targeted, and treatment consists of seventeen 60-minute sessions, addressing topics related to diet,

physical activity, sedentary activity (including sleep), and social function, as well as how to establish healthy behaviors across key settings of daily life—within the family/home environment, among peers, and in the community. The content of each of the 17 sessions is outlined in the treatment manual.[86] Table 1 gives an overview of the FBSFT phases and session-specific topics. Families were followed up by the same health-care worker from the multidisciplinary team at the Obesity Outpatient Clinic for all sessions, and average treatment duration was approximately 6 months. The team involved in treatment delivery consisted of a pediatrician, nutritionists, physiotherapists, nurses, and a psychologist. All team members were trained in FBSFT prior to treatment delivery. Parental participation was considered important for children of all ages, but age-appropriate adjustments were made, allowing adolescents greater responsibility for making healthy changes, compared to younger children.

Table 1. Overview of FBSFT phases and session-specific topics

Phase	Session	FBSFT Topic
1. Individual and Home Contex	1-2	Introduction to the treatment; plan for the Traffic Light Diet; personalized treatment plan
	3	Healthy and regular eating, communicating with the family about lifestyle changes
	4	Sedentary activity; sleep routines
	5	Physical activity; lifestyle activity
	6	Creating a healthy family and home environment; problem solving skills
	7	Healthy self-instructions; emotions/stress and eating behavior
2. Peer Contex	8	Peers as a support for healthy behaviors (arranging healthy activities with others); assessment of social network
	9	High risk situations (parties, holidays and vacations); prompts for eating and physical activity
	10	A healthy peer environment; communicating with peers about new and healthy habits
	11	Taking on Teasing
3. Community Contex	12	Physical activity and the assessment of RED food in the environment/neighborhood
	13	To be active in your neighborhood; join groups or teams; to elicit support for healthy habits in your neighborhood/environment
	14	To fight weight stigmatization; influences from the media; to build a positive self-image and body image
	15	High risk situations (restaurants and fast food); to focus on healthy habits at school and work
4. Cross-contextual	16	To plan for healthy habits; relapse prevention and consolidating skills across different contexts
	17	Reviewing goals and skills; ending well; planning ahead

Modified from the FABO study protocol.[2] Copyright 2016, with permission https://creativecommons.org/licenses/by/4.0/.

3.4.1.1 Diet component

Diet quality and quantity are addressed throughout FBSFT. The focus is on a flexible eating style within a generally healthy diet, coupled with a regular eating pattern.[88]

Although no foods are fully restricted/"forbidden," the Traffic Light Eating Plan[82] is used to categorize food as "day-to-day food" (i.e., green food) and "sometimes food" (i.e., yellow and red foods), based on the nutritional values of foodstuffs. Red food include nutrient-poor, high-calorie food and drinks such as cookies, pastries, and sugar-sweetened beverages, whereas green food comprise nutrient-dense, low-calorie options such as fruits and vegetables.[159] FBSFT includes elements of interpersonal therapy for eating disorders, with emphasis on addressing emotions and interpersonal conflicts that affect eating habits.[138]

3.4.1.2 Physical/sedentary activity and sleep component

The Traffic Light Activity Plan is a tool used to categorize physical activities according to intensity level, to enable participants making healthy activity changes.[82] This plan applies the same traffic light system used in the Traffic Light Eating Plan; it color-codes activities, such as activities of daily living, leisure or sedentary activities, and sports, based on their intensity level, while providing information on energy expenditure with the activities, given as metabolic equivalents of the task (METs).[82] The MET is an objective measure of the ratio of the rate at which a person expends energy, relative to the mass of that person, while performing some specific physical activity compared to a reference. One MET is defined as the amount of oxygen consumed while sitting still.[160] Sleep habits were introduced in Session 4, and throughout FBSFT, families worked to establish healthy sleep habits, with emphasis on age-appropriate sleep duration and sleep hygiene. Pretreatment objective measures of physical activity and sleep formed the basis for the planning of healthy changes in these areas.

3.4.1.3 Behavioral and cognitive techniques

In addition to increasing knowledge about healthy eating, activity and sleep patterns, families were taught behavioral and cognitive techniques to enable them to make positive behavioral changes and deal with mechanisms that maintain unhealthy lifestyles. Key techniques used included: (1) *self-monitoring*. Both children and their parent(s) monitored their lifestyle behaviors and weight on a week-to-week basis; (2) *goal setting, planning, and positive reinforcements*. Weekly, the therapist, together

with the child/family, outlined a plan for healthy behaviors, with tailored goals. Children and caregivers earned points throughout treatment for completing goals related to achieving a healthier lifestyle. The list of reinforcements was decided upon at treatment onset and typically consisted of activities and privileges such as sleepovers, cinema visits, and family activities. Food was not used as a reinforcer. The goal related to weight status was, in general, weight stabilization for children aged under 10, although individual considerations were made. For older children, a body weight reduction of 250 g from session to session was used as a reference point; (3) *problem-solving and cognitive restructuring*. Families were taught how to identify problems related to making healthy changes and situations/behaviors related to these problems and devise possible solutions. Negative thoughts and obstacles to accomplishing healthy changes were identified, tested, and replaced by more useful ones; (4) *stimulus control*. Because external cues might trigger unhealthy behaviors, cues for undesirable, as well as desirable, habits were identified, with the balance of these cues altered—for instance, ensuring availability of healthy food, planning dinner in relation to grocery shopping, and avoiding screen time after bedtime; and (5) *emotional regulation strategies*. Through self-monitoring, participants learned to identify situations in which they had food in response to emotions and/or craving rather than out of hunger, and together with the therapist, they rehearsed more functional behaviors in response to emotional cues. Other central aspects of FBSFT include techniques of how to cope with teasing, parental strategies for modeling of healthy behaviors, as well for parenting itself, how to achieve good communication and interpersonal skills, and how to develop healthy modes of self-evaluation and self-assertion.

3.4.2 Treatment as usual

The pediatric unit of the Obesity Outpatient Clinic was established in February 2012 and offers treatment to children aged under 18 years. The treatment is standardized, and described in the treatment manual *Towards Normal Weight*. Figure 6 outlines the sessions included in TAU (Arm B, first 12 months) and the FABO study in general. In TAU, initial assessments were carried out by a multidisciplinary team (comprising a pediatrician, a nutritionist, a physiotherapist, and a psychologist). A plan was then

created, focusing on specific lifestyle behavioral changes related to diet, physical/sedentary activity, and sleep, which was thereafter communicated by the clinic nurse to the child's local health-care nurse. Children were then invited by the local health-care nurse to attend monthly sessions, with particular focus on implementation of the healthy lifestyle changes. In some cases, the local physiotherapist was also included in treatment delivery to help those children with barriers to physical activity. Quarterly, the children (generally accompanied by their caregiver) attended follow-up sessions at the Obesity Outpatient Clinic. Most often, the children met with the clinic nurse at these sessions, although other members of the multidisciplinary team could be consulted if needed. The focus of these 30- to 60-minute sessions was on assessment, evaluation of progress, and revision of goals where necessary.

3.5 Measurements

The FABO study included several measures, as outlined in the study protocol.[2] The measures discussed in this thesis include: (1) anthropometric measures; (2) measures of sleep, physical activity, eating behavior, and screen use; and (3) measures of barriers to treatment participation (Figure 5).

Measurement points applied in the published studies presented in this thesis were baseline (in comparison with normal-weight peers), and immediately after completion of 17 sessions of FBFST for Arm A and after completion of 12 months of TAU for Arm B. Further, treatment barriers were measured at Session 12 for completers of FBSFT or at treatment end for noncompleters.

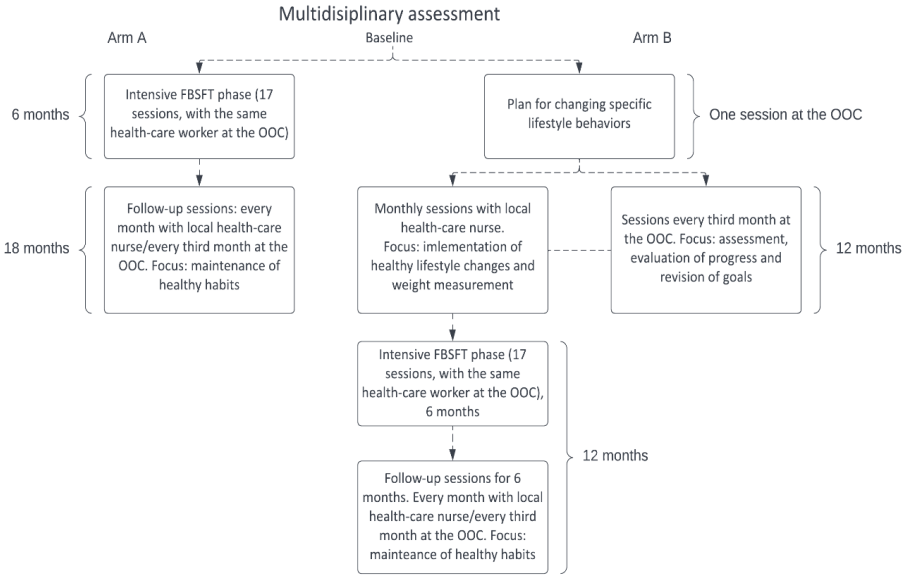

Figure 6. Outline of treatment offered within the FABO study. Abbreviations: OOC, Obesity Outpatient Clinic; FBSFT, family-based behavioral social facilitation treatment.

3.5.1 Anthropometric measures

For the group of children with severe obesity, measures of height and weight were taken at the Obesity Outpatient Clinic by trained assessors who were informed about study participation, but not about treatment assignment. Height was measured with a wall-mounted digital electronic stadiometer (Seca 264, Seca, Hamburg, Germany), and recorded to the nearest 0.1 cm. Weight was measured to the nearest 0.1 kg using a digital scale (InBody 720, Biospace, Seoul, Korea).

For the group of children with normal weight, measurements were collected by the study coordinator during regular school hours in their school nurse's office. Standing height was measured with a Harpenden portable stadiometer (Holtain Ltd, Crosswell, UK) to the nearest 0.1 cm, whereas weight was measured with a calibrated Seca personal digital scale (Hamburg, Germany). Participants in both groups were measured wearing light indoor clothing (excluding shoes and socks).

Weight status was assessed using two metrics converted from the BMI (in kg/m^2): BMI SDS derived from the Norwegian growth references[161] (applied in Papers I, II, and III) and %IOTF-25 from the IOTF reference (applied in Paper II).[22] The BMI metric %IOTF-25 represents the percentage above the IOTF threshold for overweight, based on a child's age and sex, calculated as [100 × (BMI/IOTF-25)].[22] In addition, Paper II includes a cutoff point of ≥ -0.25 BMI SDS to define a clinically relevant change from pre- to posttreatment, as previously published.[139,140]

3.5.2 Objective sleep measures

Free-living sleep was objectively assessed using an Actiwatch 2 device (Philips Respironics, Bend, OR). Actiwatch 2 is a wrist-worn accelerometer with a light sensor and an event marker, which records all uniaxial movement over 0.05 G.[162] Data were collected using 30-second epochs, each scored as either "wake" or "sleep" based on a medium sensitivity threshold.[162,163] Medium sensitivity thresholds have been shown to yield the least biased estimates of wakefulness, total sleep time, and wake after sleep onset in school-aged children.[162] At measurement points, the device was worn on the wrist of the nondominant arm for 7 consecutive days. Participants were instructed to press the event marker when switching off the light at night and when waking up in the morning. Actiwatch 2 has been validated, both in clinical sleep laboratories and in natural home environments, and is commonly used for sleep research in children aged 3–18 years.[163–165]

Sleep statistics were calculated using Respironics Actiware software, version 6.0.9. The rest interval (time in bed) associated with the main sleep period in the 24-hour day was manually set according to a standardized scoring protocol.[166] Furthermore, sleep time within this interval was automatically detected by a standard default algorithm in the proprietary software.

3.5.2.1 Sleep duration

In the studies presented in Papers I and II, data analysis used mean sleep duration over 7 consecutive days. For inclusion in the analysis, participants collected full recordings of at least 5 out of 7 days, including a minimum of three school nights and two weekend nights. In addition, in Paper I, analyses included average sleep duration

on school nights (Sunday through to Thursday nights) and weekend nights (Friday and Saturday nights) separately. Further, in Paper I, sleep duration for 7-day average, and school and weekend nights were also categorized, based on sleep recommendations from the National Sleep Foundation (NSF).[167]

3.5.2.2 Sleep timing

Data on sleep timing (i.e., when sleep occurs), operationalized as the midpoint between sleep onset and wake-up time and calculated as [(sleep onset time + sleep offset time)/2], were analyzed in Papers I and II. In Paper I, the 7-day mean was measured, as well as school and weekend nights separately; in Paper II, only the 7-day mean was reported. For participants who completed six or five nights of recordings, the average of these nights was used. To be included in weekend night's analyses, two nights of recordings were needed. Sleep onset and final wake-up time were reported for additional information about sleep hygiene. Neither Papers I nor II included assessment of sleep during daytime.

3.5.2.3 Social jetlag

Social jetlag quantifies the discrepancy between circadian time and social time.[121] In Paper I, social jetlag was operationalized as the difference between the mean mid-sleep time point on school and weekend nights.

3.5.3 Physical activity measures

Free-living physical activity level was objectively determined using data from the same device (Actiwatch 2) during daytime (8 a.m. to 9 p.m.). Similar time frames for recording of physical activity have been used by other studies in treatment-seeking children with obesity,[168] and in children across the weight spectrum.[169,170] Data were downloaded using Respironics Actiware software, version 6.0.9, and exported into Microsoft Excel 2016 for further processing using a tailor-made algorithm. The collected activity data were categorized into different intensity levels, based on previously used and validated cutoff values for children using a similar Actiwatch model:[169] light intensity (160–523 counts/30-s epochs); moderate intensity (524–811 counts/30-s epochs); and vigorous intensity (≥812/30-s epochs).[171] To be included in

the analysis, participants needed ≥10 hours of wear time each day and ≥4 days of recorded data.[169] Sleep time during this period was automatically coded as either nonwear time or sedentary behavior (movement while sleeping) by the tailor-made algorithm. Physical activity was operationalized as the percentage of time spent on moderate-to-vigorous physical activity (MVPA) (data included in Papers I and II).

3.5.4 Questionnaire

3.5.4.1 Demographic questionnaire

Papers I and III include data on demographic variables collected through a parental questionnaire developed at the Obesity Outpatient Clinic. Paper III reports on family structure, parental education levels, and parental employment; Paper I describes additional variables—child sleep problems and daily screen time, also included in the questionnaire assessment. The question regarding sleep problems was formulated as follows: "Has the child in any period experienced sleep problems?," with the following response categories: "never," "before starting elementary school, but not now," "after starting elementary school, but not now," and "current sleep problems." Participants were grouped according to whether they reported current sleep problems or not. Habitual screen time was rated on a scale ranging from 0 (no screen time) to 5 (>4 hours of screen time). For children with severe obesity, the questionnaires were completed by their parents at the Obesity Outpatient Clinic, whereas for children with normal weight, the questionnaires were addressed to the child with parent(s) and sent by mail for completion by their parents.

3.5.4.2 The Dutch Eating Behavior Questionnaire (DEBQ)

The DEBQ is a self-reported questionnaire developed to measure three different aspects of eating behavior that can cause an overeating response in children and adolescents: emotional eating (overeating in response to emotions); externally induced eating (eating in response to food-related stimuli, regardless of hunger and satiety status); and restrained eating (attempts to refrain from eating).[172–174] Two versions of the questionnaire were administered: an age-adapted 20-item version for children aged younger than 10 years; and a full 33-item questionnaire for children aged ≥10 years.[95,173,174] All 33 items in the full version were rated on a 5-point Likert

scale, ranging from “never” (1) to “very often” (5). The age-adapted 20-item version included a reduced 3-point response scale: “no” (1), “sometimes” (2), and “yes” (3).[95,173,174] Mean scores were calculated for each of the subscales (emotional, external, and restrained eating). The two versions of the questionnaire were merged for analysis by converting responses on the 20-item version as follows: 1 = 1; 2 = 3; and 3 = 5. A high score indicated a high degree of the eating behavior in question.[175] In Paper I, only data for the subscale of emotional eating were analyzed, while Paper II included data for all subscales. Both versions of DEBQ have been increasingly used in pediatric obesity research, and shown to have adequate internal consistency, test–retest reliability, factorial validity, and dimensional stability for measuring disordered eating behavior in children aged 7–17 years.[95,174,176] All children self-reported; children with severe obesity completed the questionnaire at the Obesity Outpatient Clinic, while children with normal weight did so at the school nurse office, with on-site help available from the research coordinator if a question was unclear. In Paper I, the Cronbach’s α coefficient for the emotional eating subscale was 0.96, suggesting a high internal consistency of the scale in the current sample. In Paper II, the Cronbach’s α coefficient for the 33-item version was 0.76 for restrained eating, 0.87 for external eating, and 0.96 for emotional eating at baseline, and for the 20-item version, 0.79 for restrained eating, 0.73 for external eating, and 0.85 for emotional eating at baseline—indicating acceptable to excellent internal consistency for the three subscales in the current sample.

3.5.4.3 Barriers to Treatment Participation Scale (BTPS)

In Paper III, the BTPS was used to measure perceived barriers to treatment. The BTPS[177] was developed and validated to address dropout from treatment with outpatient psychological treatment of children and adolescents. The main section of the questionnaire consists of 44 statements evaluated on a 5-point Likert scale ranging from 1 (“never a problem”) to 5 (“very often a problem”). Scores are distributed across four subscales: (1) “stressors and obstacles that compete with treatment;” (2) “treatment demands and issues;” (3) “perceived relevance of treatment;” and (4) “relationship with the therapist.” In addition, the questionnaire

includes 14 questions about specific critical life events that are answered in a "yes" or "no" format. The scale has been found to yield high levels of internal consistency and to be predictive of treatment dropout, appointment cancellations, and weeks spent in treatment.[177,178] The Cronbach's α coefficient for family- and therapist-rated versions of the questionnaire was as follows: 0.83/0.87 for "stressors and obstacles that compete with treatment;" 0.61/0.72 for "treatment demands and issues;" 0.64/0.71 for "perceived relevance of treatment;" and 0.77/0.84 for "relationship with the therapist"—indicating acceptable consistency for the four subscales in the current sample.

3.5.5 Statistical analyses

Data were analyzed using IBM SPSS version 25–27 (IBM Corp., Armonk, NY), Stata version 17 (StataCorp LLC, 2021, College Station, TX), and R 3.4. Descriptive statistics were expressed as the mean and SD for continuous variables, and as the frequency and percentage for categorical variables.

Papers I, II, and III include comparison of groups. Independent sample *t*-tests and chi-square tests of independence were used to compare groups: in Paper I, children with obesity and children with normal weight; in Paper II, participants in FBSFT and those in TAU; and in Paper III, families completing and those not completing treatment. In addition, the Hotelling's T^2 test was applied to compare the multivariate data (i.e., three of the BTPS subscales) between groups in Paper III. The subscale of "relationship with the therapist" was highly skewed, with limited variance; therefore, a Wilcoxon rank-sum test was used to test the hypothesis that the comparison groups were from populations with the same distribution, as well as to compute the probability that a random case from one group had a higher score on the subscale than a random case from the other group. A Wilcoxon rank-sum test was also used to compare critical life events between groups in Paper III.

Both data from Papers I and II were based on regression analyses; hierarchical multiple regression and logistic regression analyses were performed in the study presented in Paper I, and linear mixed models used in the study in Paper II. In Paper I, hierarchical multiple regression was applied to analyze the effect of mid-sleep time,

sleep duration, social jetlag, and sleep problems on screen time and MVPA. The analyses were adjusted for group (normal weight or severe obesity), age, sex, living situation, and parental education level. In addition, logistic regression was used to examine the association between the sleep variables and a dichotomized emotional eating variable, adjusted for group, age, and sex. The study in Paper II applied linear mixed models to compare changes from pre- to posttreatment under the two treatment conditions (i.e., FBSFT versus TAU).

3.5.5.1 Sample size calculations

The required sample sizes were calculated with G*Power, version 3.1.3, prior to commencement of the FABO study. For the comparison between children with severe obesity and those with normal weight, a minimum of 51 individuals in both groups was required to detect a medium effect size (Cohen's $d = 0.50$), with a two-tailed significance level α of 0.05 and power $(1 - \beta)$ of 80%. Eighty-five children and adolescents were recruited to each group, as this was the number of participants with severe obesity recruited to the FABO study at the time when the matched comparison group was collected. The calculations for the RCT part of the study were based on two treatment groups (FBSFT and TAU) and three measurement points (pretreatment, posttreatment, and 12-month follow-up). For an α level of 0.05, a power of 0.80, and a correlation of 0.5 between measurement points, a sample size of 28–164 subjects would allow detection of moderate (Cohen's $f = 0.25$) to small (Cohen's $f = 0.10$) effects of treatment on the primary outcome over time. Based on the number of referrals to the Obesity Outpatient Clinic, a total of 120 participants were estimated as a realistic sample size to recruit during the study period, and large enough to detect small to moderate differences in the primary outcome between groups.

4. Summary of results

This section gives a summary of the main results from Papers I, II, and III presented in this thesis. The original articles can be found in the Appendix of the thesis.

4.1 Paper I

4.1.1 Beyond sleep duration: Sleep timing as a risk factor for childhood obesity

Paper I presents a comparison of sleep behavior between children and adolescents with severe obesity and peers with normal weight, as well as examines the association of sleep duration, problems, and timing (including social jetlag) with obesity and obesogenic behaviors in children and adolescents. The findings demonstrated significant differences in sleep timing (operationalized as mid-sleep time) and sleep problems between children and adolescents with severe obesity and normal-weight peers. The group of children and adolescents with severe obesity had significantly later mean mid-sleep time, compared to children and adolescents with normal weight: overall (36 minutes later; p <0.001); on school nights (36 minutes later; p <0.001); and on weekend nights (39 minutes later; p = 0.002). Children and adolescents with severe obesity also presented with more sleep problems than their normal-weight peers (p = 0.030), whereas no differences emerged in sleep duration or social jetlag between the two groups. Examination of the association between sleep behavior and other obesogenic behaviors (screen time, time in MVPA, and emotional eating) in the total sample of participating children and adolescents showed that later sleep timing was related to longer screen time (p = 0.03). In addition, sleep timing and duration were inversely related to time in MVPA (p ≤0.041). There were no other significant associations between the sleep variables and obesogenic behaviors.

4.2 Paper II

4.2.1 Family-based treatment of children with severe obesity in a public healthcare setting: Results from a randomized controlled trial

Paper II presents the results from the RCT part of the FABO study, which showed a significantly greater improvement in BMI-related outcomes with FBSFT, compared to TAU, from pre- to posttreatment. The statistically significant difference in change in BMI SDS was 0.19 units (95% confidence interval [CI] 0.10–0.28; p <0 .001) and 5.48 %IOTF-25 (95% CI 2.74–8.22; p <0.001) between groups. Further, FBSFT participants achieved significant reductions in mean BMI SDS (0.16 units, 95% CI −0.22 to −0.10; p <0.001) and %IOTF-25 (6.53%, 95% CI −8.45 to −4.60; p <0.001), compared to the TAU group with nonsignificant changes in BMI SDS (0.03 units, 95% CI −0.03 to 0.09; p = 0.30) and %IOTF-25 (−1.04%, 95% CI −2.99 to 0.90; p = 0.29). Assessment of individual treatment response revealed that significantly more FBSFT participants had clinically meaningful BMI SDS reductions of ≥0.25 from pre- to posttreatment, compared to the TAU group (31.5% versus 13.0%, respectively; p = 0.021). The beneficial changes in weight-related outcomes observed in the FBSFT group, compared to the TAU group, were likely not explained by differences in the secondary outcomes (sleep, physical activity, eating behavior), with only changes in sleep timing being significantly different between treatment groups.

4.3 Paper III

4.3.1 Perceived barriers in family-based behavioral treatment of pediatric obesity - Results from the FABO study

In Paper III, perceived barriers to participation in FBSFT among families who did and did not complete treatment are presented. Families who did not complete treatment scored significantly higher on two of the four subscales of the BTPS, compared to those who completed treatment. Families who did not complete FBSFT scored significantly higher on “stressors and obstacles that compete with treatment” (mean = 2.03, SD = 0.53) than those who completed treatment (mean = 1.70, SD = 0.42; p = 0.010). Further, families who did not complete FBSFT scored

significantly higher on barriers related to “perceived relevance of treatment” (mean = 2.27, SD = 0.48) than families who completed treatment (mean = 1.80, SD = 0.50; p <0.001). No significant differences between the two groups were observed for the subscales “treatment demands and issues” and “relationship with the therapist,” or for critical life events during treatment.

5. Discussion

Results presented in this thesis showed that children and adolescents with severe obesity had significantly later sleep timing and presented with more sleep problems, compared to peers with normal weight, with no significant difference in sleep duration and social jetlag between the two groups. Later sleep timing was also found to be associated with more screen time and less time in MVPA in the whole study population. The results further showed that significantly greater improvement in weight-related outcomes was obtained with FBSFT, compared with TAU, when delivered in a real-world health-care setting. Examination of individual treatment responses showed that significantly more FBSFT participants had clinically meaningful BMI SDS reductions from pre- to posttreatment, compared to TAU participants. Finally, the results demonstrated that families who did not complete FBSFT reported more barriers related to "stressors and obstacles that compete with treatment" and to "perceived relevance of treatment," compared to families who completed treatment. For barriers related to "treatment demands and issues" and "relationship with the therapist," no significant differences were observed between families who did and those who did not complete treatment.

5.1 Methodological considerations

Quoting Krauss in his recent publication on assessing the validity of RCTs, "[t]*he ultimate purpose of evaluating the effectiveness of trials is to improve the lives of people in the real world. This is why researchers and practitioners need a broad understanding of how the range of various biases facing trials can arise.*"[179]

5.1.1 Study participants and recruitment

5.1.1.1 Children and adolescents with severe obesity

All children and adolescents fulfilling the inclusion criteria who were referred to the Obesity Outpatient Clinic between February 2014 and October 2018 were invited to participate in the FABO study, thereby minimizing selection bias. Of note, the number of children/families who declined study participation and their reason for

declining were not reported. Based on yearly referrals to the Obesity Outpatient Clinic, approximately 270 children and adolescents were referred during the study period. The lack of knowledge concerning characteristics of those who declined study participation versus those enrolled in the study is a limitation that makes it difficult to contextualize individuals who did and those who did not enroll and impacts the generalizability of the findings. Given that the Obesity Outpatient Clinic serves a large geographical area (which requires, for some patients, up to 3 hours of travel by car to the clinic), the general impression from the research and clinic teams who were in contact with individuals/families regarding opportunities for participation was that living far from the clinic was the main reason for declined participation. However, additional factors could have influenced individuals' or families' decisions to enroll; for example, those who enrolled might have been more motivated to engage in behavioral changes than those who declined. Nevertheless, based on the low number of exclusion criteria and the fact that children who were invited for study participation had already been referred the Obesity Outpatient Clinic for treatment, it is likely that the recruited study sample is representative of treatment-seeking children and adolescents with severe obesity in Norway.

5.1.1.2 Peers with normal weight

Peers with normal weight were recruited from randomly selected schools in the catchment area for the FABO study. Schools from both inner city and more rural areas were represented. Letters of invitation were sent based on class lists provided by school administrators, and only four children/families declined to participate in the comparison group. Stratified random sampling was used to ensure that children in the comparison group were matched on age, sex, and season of accelerometer measures, and had similar characteristics to those of children with severe obesity, except for weight category. Although the study aimed to increase the generalizability of the comparison group through the recruitment methods used, it should be noted that it is possible the comparison group may not be representative of all Norwegian children and adolescents with normal weight. For instance, although the comparison group was not presented with treatment opportunities, it is possible that children, adolescents, and/or their parents who were interested, and/or willing to enroll, in

research related to health behaviors differed from those who were not interested in participating or those who were not recruited to participate in the study.

5.1.1.3 Sample size

The sample size needed for the RCT part of the FABO study was calculated prior to study initiation and was based on two factors: (1) to obtain a realistic sample size based on the number of referrals to the Obesity Outpatient Clinic; and (2) to estimate the study power so a large enough sample could be recruited that would detect significant group differences in the primary outcome measures when small to moderate effect sizes were expected. It is worth noting that as per the protocol, this power analysis was based on a MANOVA approach, and not on a linear mixed model.[2] Accordingly, the recruitment goal was a total of 120 participants/families, with 60 in each arm. Recruitment was completed with a final sample of 114 participants/families, with 59 in Arm A (FBSFT administered first) and 55 in Arm B (standard treatment administered first from the Obesity Outpatient Clinic, followed by FBSFT). Although the sample size was slightly smaller than that calculated in the a priori power analysis for this study, this is highly unlikely to have influenced the conclusions of the study presented in Paper II.

5.1.2 Design and measurement points

The FABO study included an RCT, which is considered as the most reliable method for assessing the effects of treatment interventions.[180] The Consolidated Standards of Reporting Trials (CONSORT) guidelines for reporting of clinical RCTs were followed,[181] and a CONSORT statement checklist completed as part of the submission process of Paper II. One key advantage of randomization is that known and unknown prognostic factors in treatment assignment can be balanced and it allows to determine whether differences in outcomes between two treatment groups might be due to chance by applying the probability theory.[181] A limitation of the FABO study design is that the point at posttreatment represented the last possible measurement point for the RCT part of the study. At posttreatment, the group in Arm A had completed FBSFT and subsequently underwent follow-up; by contrast, the control group in Arm B was offered FBSFT at posttreatment (i.e., after having

received standard treatment from the Obesity Outpatient Clinic), such that there was a lack of control condition (i.e., a group that did not receive FBSFT) beyond this time for this group. The ethical justification for this choice is elaborated in Section 5.1.6 on ethical considerations. Further, the duration of active treatment differed between Arm A and Arm B, resulting in different end points—on average, approximately 6 months for the FBSFT group and 12 months for the TAU group, such that the maturation factor also differed between the two groups. As a result, this signifies a potential impact on internal validity, as participant maturation can have both positive and negative effects on outcomes.[182] A plausible hypothesis regarding pediatric obesity treatment is that longer treatment durations produce better results,[183] and therefore an even larger difference between treatment groups in favor of FBSFT would be expected with similar treatment duration. However, no adjustments for the difference in treatment time were applied in the analyses, as the aim was to compare the two treatment programs, despite their known differences in treatment duration, as delivered in routine clinical practice. Of note, follow-up analyses of anthropometric data from the TAU group after 6 months of treatment showed the same BMI SDS increase (0.03 units) as after 12 months of treatment, and thus did not affect the study conclusions here.

Although FBSFT had a more intensive treatment phase than TAU, both treatment programs had a similar total number of sessions. This represents a study strength, as research has shown that the proportion of participants responding to treatment increases with the number of sessions received.[71,72,182] Another strength is that dropout rates were comparable, and relatively low (19.3%), in both FBSFT and TAU groups, compared to dropout rates of 27–73% reported in previous studies.[148]

In the FABO study, masking of both participants and treatment providers to intervention conditions was not possible due to the nature of the intervention. Masking of outcome assessors was initially planned but became difficult to implement, as both participant follow-up and data plotting and analyses were overseen by the study coordinator. Lack of masking can lead to performance and detection bias, and thus inadvertently influence the results. Interestingly, a recent meta-epidemiological study found no evidence that lack of masking of health-care

providers, participants, or outcome assessors had an impact on effect estimates in RCTs, suggesting that masking might be less important than widely assumed in such research.[184]

5.1.2.1 Choice of control group

A crucial question in treatment research is whether a new treatment has better effects than the existing alternative. Thus, using TAU as the control condition would inform the conclusions that can be drawn about the effectiveness of a new treatment approach, as well as its potential to improve health-care services, compared to treatments that are already available.[185]

The design of the FABO study offered an opportunity to perform an observational study among participants while taking part in the FBSFT component of the study (Paper III). All 59 participants in Arm A participated in FBSFT. Among the 55 participants in Arm B, 10 individuals did not complete their 12 months of TAU. Of the remaining 45 completers in Arm B, 31 individuals/families (68.9%) chose to start FBSFT after 1 year of TAU. The other 14 individuals either left the treatment program entirely or extended their TAU treatment beyond the first 12 months. Reasons given by participants for declining the invitation to enroll in FBSFT included, for some, the feeling that TAU already had produced the necessary effects on their weight status and, for others, treatment fatigue. One interpretation of these observations is that for some participants, undergoing 1 year of TAU (or a treatment duration of >1 year) in itself may constitute a barrier to participating in FBSFT. The measurement point in respect of barriers to FBSFT participation was the same for both Arms A and B (Paper III). However, individuals in Arm B had already participated in 1 year of obesity treatment when entering FBSFT, which might have resulted in different perceptions of treatment, compared to those in Arm A who had no immediate prior treatment experiences from the clinic when entering FBSFT. Whereas previous research on barriers to participation in lifestyle treatment for pediatric obesity has mainly focused on noncompleters of treatment,[147,154] here barriers to participation were investigated among all families receiving FBSFT. A comparison of perceived barriers in treatment completers and noncompleters would

give valuable information on similarities and differences in reported barriers between the two groups.[147]

At baseline, participants in the FABO study were compared to a comparison group of peers with normal weight (Paper I). This offered an opportunity to gain insight into behavioral factors that represent risk factors for developing obesity in school-aged children and adolescents. However, since the comparison had a cross-sectional design, it was not possible to draw conclusions about causality.

5.1.3 Training of health-care workers and treatment delivery (treatment fidelity)

Training of health-care workers in intervention delivery and delivery of treatment itself are both considered domains of treatment fidelity. There is no single agreed-upon definition of treatment fidelity. However, it often refers to processes that ensure that an intervention is implemented as outlined in the protocol, such that the results of a trial are directly attributable to the intervention, thereby increasing internal validity.[186] At the same time, the reproducibility of the study is also increased through enhanced external validity, which is the extent to which results can be generalized to clinical settings.[186]

5.1.3.1 Training of health-care workers

In the study, before treatment was administered to families, measures were taken to ensure that all health-care workers involved in treatment delivery were adequately trained to deliver FBSFT. Those who were involved in the FABO study prior to patient enrollment attended 2 days of FBSFT workshops conducted by the research group from St. Louis, MO, in the United States, and also performed a "trial run" of FBSFT with one pilot family prior to providing actual treatment to families enrolled in the study. Some of these sessions were recorded for subsequent reviewing of competency and for supervision purposes, but no formal fidelity coding was carried out. As FBSFT was delivered through an outpatient clinic, new health-care workers were recruited to serve as intervention providers throughout the study period. Due to limited resources, these health-care workers did not conduct "trial runs" of FBSFT

with pilot families. However, they were offered specific training through workshops prior to delivering actual treatment to families enrolled in the study.

5.1.3.2 Treatment delivery

In treatment research, it is important to minimize variability in treatment delivery to participants. If a reported effect is due to factors not related to the treatment protocol (extra treatment factors), this can result in implementation of an ineffective intervention in clinical practice.[186] To monitor and enhance delivery of FBSFT, weekly meetings for on-site supervision of treatment delivery and monthly virtual supervision with the research team in St. Louis, and later in Pittsburgh, PA, both in the United States, were conducted. Some sessions were audio-recorded and evaluated at the weekly meetings, although this was not systematically done. Audio/video recordings (or direct observation) of sessions throughout the intervention would have helped minimize drift and perform additional training of health-care personnel if considered necessary.[186] Further, adherence coding of audiotapes would have strengthened the internal validity of the study by assessing and confirming the extent to which therapists were adhering to the treatment manual/principles. A strength of the study, however, is that FBSFT is a manualized intervention program, with detailed outlines of session contents, such that it was more likely that all participants received the same treatment and all health-care workers delivered the intervention in the same way. Importantly, the FBSFT manual and supervision included discussion of adaptations and offered ways to standardize intervention delivery, while also providing flexibility in tailoring the intervention to families. For instance, age-adjusted content was provided in terms of different caloric intake and sleep-related goals for children and adolescents; such flexibility within treatment fidelity is an important feature of intervention delivery to promote optimal outcomes.[187] In addition, FBSFT sessions were rescheduled, if needed, to ensure that each family received the same content/dose of treatment. Regarding TAU, no weekly/monthly meetings were conducted by the FABO research team. However, weekly meetings for discussion of patients were run by the pediatric team at the Obesity Outpatient Clinic. Further, TAU content was standardized through a treatment manual. Monthly sessions with local health-care nurses, as part of the TAU program, were not formally

evaluated, which could have resulted in marked variance in the content offered in these sessions. The FABO research team invited all local health-care nurses working with FABO patients to monthly workshops at the Obesity Outpatient Clinic. These meetings had high attendance rates, but the focus was mainly on follow-up of FABO patients after completion of FBSFT, and not on treatment content during TAU.

In total, 10 health-care workers delivered FBSFT throughout the study, all of whom were also involved in delivery of TAU. Thus, it is possible that elements from the new treatment (FBSFT) were incorporated into the existing treatment (TAU). Again, this could cause a higher degree of similarity between the two treatments, likely resulting in smaller differences between the groups. In addition, involvement of 10 different health-care workers in treatment delivery possibly reduced the therapist effect on outcomes, while strengthening the generalizability of the study.[188]

5.1.4 Measurements

The studies presented in this thesis included a mix of objectively measured variables and variables measured by self- and/or parental reporting. Measurement-related considerations, threats to internal validity, and ways in which the risk of potential errors was mitigated are discussed in the following sections.

5.1.4.1 Anthropometric measurements

Weight status and comparisons of the effect of intervention on weight status were based on measurements recorded by health-care workers. As different health-care workers performed the measurements, measurement error was possible. However, all health-care workers were trained in measuring height and weight through attending a workshop at the Obesity Outpatient Clinic prior to the study. Errors were expected to be random and not systematic, and therefore not to be a cause of significant bias.

All children with severe obesity were measured using the same equipment. For the normal weight control group, adjustments had to be made since measurements were taken at the children's respective schools. However, to minimize the risk of measurement errors or using different equipment, all anthropometric measures obtained from this group were carried out by the study coordinator using the same portable equipment.

Assessment of caregivers' height and weight was conducted as part of the treatment, but not systematically recorded for research purposes. Therefore, the relationship between child and parent weight outcomes could not be further investigated within the FABO study. Previous research showed that these outcomes were strongly associated, both immediately posttreatment[189,190] and at 5-year follow-up.[191]

BMI SDS calculations were based on national growth references from the Bergen Growth Study 1.[20] Variation in BMI distribution across populations[192,193] is an argument for the use of national references in such calculations. However, one can also argue for the use of international references (IOTF or WHO), as these enable comparison among countries.[192,193] In the FABO study, however, the IOTF cutoff for defining overweight, obesity, and severe obesity[17] was used as an inclusion criterion.[2] BMI SDS have been found inadequate in correcting for age, sex, and the degree of obesity in the group of children with severe obesity.[22,23] The percentage above the IOTF cutoff for overweight (i.e., %IOTF-25) has been suggested as a better alternative[21] and was included into the work described in this thesis.

5.1.4.2 Measurement of dietary intake in the FABO study

For the first 2 years of the study, parents were instructed to help their child keep a food diary for 5 days, including two weekend days at baseline and posttreatment, with an electronic kitchen scale used to weigh the food. This method was chosen based on validation against other measures of food intake, and also given that food diaries in general have been found to have similar, if not better, accuracy, compared to other self-reporting assessments.[194] However, due to poor compliance, it was decided to stop collecting food diaries. It is possible that another method for measuring dietary intake would have been better suited for the study population such as the 24-hour recall method.[168] Data on dietary intake at baseline and postintervention would have provided a valuable addition to the lifestyle behaviors studied here and thus would represent a key area for future study.

5.1.4.3 Objective measures of sleep and physical activity

Actiwatch 2 was used to measure both sleep and physical activity. The fact these movement behaviors were measured objectively is considered a major strength of the work presented in Papers I and II, as the majority of previous studies had resorted to either self- or parent-reported data on these measures.[67,68] A limitation, however, is that the algorithm used for analyzing physical activity is a poor discriminator between sleep and sedentary behavior and nonwear time, resulting in sleep being coded either as nonwear time or as sedentary behavior (movement while sleeping). Nevertheless, this does not affect the estimates of physical activity of light, moderate, or vigorous intensity. Therefore, to ensure validity of the measurement, physical activity was measured only during daytime (from 8 p.m. to 9 p.m.) and further sedentary activity was not used as a measure in the work described in Papers I and II. MVPA was chosen as the outcome variable for physical activity to avoid multicollinearity, as MVPA and light-intensity activity were highly correlated. Given that MVPA represents a combination of the two independent variables moderate- and vigorous-intensity physical activity, additional inclusion of these variables would result in singularity in the regression analysis. One can argue that the 10 hours of wake time between 8 a.m. and 9 p.m. required for inclusion in the analyses of time in MVPA is a potential limitation as some children may engage in physical activity outside this time frame. However, this requirement is in accordance with previously used scoring protocols for objectively measured physical activity.[169,170] The sleep data were based solely on the longest sleep interval in the 24-hour day, to ensure validity, due to greater difficulty in classifying shorter sleep intervals in some cases. Although measurements of all movement throughout the 24-hour day would have strengthened the study, these were not considered possible with the equipment used. In addition, it would have been advantageous to measure sleep and physical activity over a longer period, rather than over 7 consecutive days. However, due to limited availability and high cost of accelerometers, this was not possible. Physical activity and sleep are both influenced by many contextual factors such as special events and weather, and multiple weeks of measurements would have enabled a greater understanding of these complex and dynamic behaviors.[195] Further, a limitation with the sleep measurements

is the lack of sleep diaries to support the scoring of actigraphy recordings. Therefore, 30% of sleep recordings (reported in Paper I) were scored by two independent observers to ensure interrater reliability, and a standardized scoring protocol was applied.[162]

5.1.4.4 Questionnaires

Eating behavior, sleep problems, and screen time were self/parent-reported in Paper I, and eating behavior in Paper II, while Paper III relied on self-reported data (parent and therapist reporting) on barriers to treatment participation. Data collection through self-reporting can be considered a threat to internal validity, potentially from response biases (e.g., social desirability bias) and extreme response bias (only responding "1" or "5" on a 5-point Likert scale).[182]

5.1.4.4.1 The Dutch Eating Behavior Questionnaire (Papers I and II)

It is worth noting that the age-adapted 20-item version for children aged <10 was used in the work presented in Papers I and II, but only described in Paper II.

In the study described in Paper I, only the subscale "emotional eating" was used in the analyses, with clinical cutoff values applied (≥2.22 for boys and ≥2.36 for girls), and participants grouped accordingly.[174] This particular subscale was selected, based on the likelihood of short sleep duration/unfavorable sleep behaviors affecting mood, thereby increasing the risk of emotional eating. In retrospect, it would have been potentially informative to also include the subscale "external eating" in the analyses, as short sleep duration was shown to be associated with increased caloric intake and poorer diet quality.[127] In the work presented in Paper II, all three subscales of the DEBQ were analyzed, with no clinical cutoff used, the rationale being that emotional, external, and restrained eating behaviors are actively addressed in FBSFT. Therefore, the aim was to examine potential changes in all three areas from pre- to posttreatment.

The DEBQ was developed to measure three different aspects of eating behavior that can contribute to overeating.[173] Children, especially those in the youngest age groups, may find it difficult to understand and conceptualize associations between food and emotions,[97] so it is worth considering whether they

fully comprehend the items on the different subscales, which could affect construct validity. One can argue that it is a limitation of the work in Paper II that only one measure of disordered eating behaviors was included, without any additional measure of eating disorder psychopathology (e.g., attitudes and cognitions), as this would have allowed a broader understanding of eating disorder psychopathology in the study population. Use of the Youth Disorder Examination Questionnaire (YEDE-Q), a self-reported measure of eating disorder psychopathology in youth, was included in the FABO study.[2] However, the questionnaire was only used among those participants aged >10 years, which meant that inclusion of the YEDE-Q in the work reported in Paper II would have resulted in a lower *N* in the analyses.

5.1.4.4.2 The Barriers to Treatment Participation Scale (Paper III)

Due to limited resources, the BTSP was administered in a questionnaire format. The interview format used in previous studies[177,196,197] would have allowed for more elaboration if a question formulation came across as unclear, possibly resulting in more precise answers. On the other hand, the interview format could increase the risk of social desirability bias. Inclusion of a self-report version for participating adolescents would have been a valuable extension to gain a better understanding of the perceived barriers to participation in FBSFT from a family perspective.

Research studies applying the BTPS often report sum scores.[177,196,197] Here mean scores were reported, based on the fact that three questions were excluded in the analyses due to lack of relevance to the study population. Reporting sum scores could be misleading, as they would not be comparable to scores from studies that include all questions, whereas mean scores are easier to compare between studies that include different numbers of questions in the subscales. The BTPS was translated into Norwegian for this study, with no psychometric evaluation available in the Norwegian version, which could be considered a limitation.

5.1.4.4.3 Demographic questionnaire: screen time and sleep problems (Paper I)

Data on screen time and sleep problems were parent-reported using a demographic questionnaire developed by the pediatric team from the Obesity Outpatient Clinic. Although this questionnaire is routinely used for assessment in the clinic, it can be

considered a limitation as it has not been validated. Further, a discrepancy between parent and child in perception of sleep problems and actual screen time was likely, possibly reducing the accuracy of the measurements. Another threat to the accuracy of the measurements of screen time and sleep problem is the lack of differentiation between the types of sleep disturbances, and no separation between school and leisure time screen use.

5.1.5 Statistical considerations

In Paper II, the analysis used for the primary outcomes differs from that presented in the protocol paper.[2] In the startup phase of the FABO study, the plan was to perform MANOVA analyses (two-way and one-way within subjects). However, following subsequent discussion of the analytic plan among the research group, it became apparent that linear mixed model analysis was more appropriate and more straightforward to interpret. Importantly, this decision was made prior to the actual analysis.

In Paper III, the Hotelling T^2 test was used as this method allows for multivariate data analysis, rather than using individual *t*-tests for each of the subscales, thus minimizing the risk of type 1 error. Due to the sample size in the study presented in Paper III, it was decided not to differentiate between timings of dropout (e.g., early or late) in the analyses. It is possible that different timings of dropout result in different perceived barriers to treatment.

5.1.5.1 Handling of missing data

The study in Paper II included all participants in the analyses, following the principle of intention to treat, whereas Papers I and III used only observations that were available. Multiple imputation of missing data[198] is a method that could have been applied for data analysis in Papers I and III. However, the method used was more straightforward and deemed appropriate, as the rate of missing data in Papers I and III in general was low.

5.1.6 Ethical considerations

As it was hypothesized that FBSFT as additional treatment would produce better treatment effects than standard treatment, there was always the ethical debate of withholding a potentially better treatment for half of the group and how long this would be considered acceptable. A study design of this type is, however, necessary to answer the question of whether the new treatment (FBSFT) is more effective than the one currently used. Further, both treatments are in line with the Norwegian Directorate of Health's guidelines[1] for treatment of childhood obesity and all families included in the study were given the opportunity to join the FBSFT, albeit at different time points.

Regarding the normal weight comparison group, a letter of invitation to participation was addressed to the children and their parents. No classification of body weight was done before a child entered the study. On reviewing the data collected, data from children with a BMI classified as either "overweight" or "obesity" were excluded (in total four children) from the analysis, and a new child was recruited. Sending out invitation letters based on class lists involved the risk of inviting children with overweight or obesity to participate in the normal weight group. This was unfortunate, but it was optional to respond to the letter and no children with overweight or obesity were singled out in the data collection process.

All participants, and their parents, were informed about their right to withdraw from the study at any given time.

Other ethical considerations related to the FABO study included the following: (1) completing measures and discussing weight, eating, body size and shape, and disordered eating behaviors could cause discomfort or distress, although health-care workers involved in treatment delivery were trained in how to provide interventions for children and families in a sensitive and supportive manner; (2) it has been posited that weight management programs in children may place them at greater risk of eating disorders,[199] although meta-analytic data suggest that the risk of disordered eating behaviors/pathology is reduced among participants in evidence-based weight management programs, including the family-based programs upon which FBSFT is built.[88,200] Given that FBSFT addresses body image (including influences from the

media and peers), negative affect, communication within the home setting, and advocacy for health behaviors at all levels in children's lives, it is likely that participants who took part in FBSFT might have received further benefits from the information, validation, and support provided by their health-care workers. In addition, FBSFT incorporates the involvement of parents/caregivers within the treatment process, thus informing the children's families and support networks about health behaviors and ways to support the children's health behaviors.

5.2 Discussion of results

5.2.1 Obesogenic behaviors and pediatric obesity

The main objective of the work presented in Paper I was to compare sleep behaviors in treatment-seeking children and adolescents with severe obesity and peers with normal weight. Further aims were to investigate the relationship between sleep duration and timing, social jetlag, and sleep problems and other behavioral factors known to cause obesity in children. The study in Paper II examined for any changes in children's sleep, physical activity, and eating behaviors following two different obesity treatment programs, namely FBSFT and TAU. While many of the findings were in accordance with those from previous research and/or clinical impression, the study also yielded some results that provided new insights.

Paper I compared sleep duration between children and adolescents with severe obesity and peers with normal weight. Children and adolescents with severe obesity were expected to have significantly shorter sleep duration than those with normal weight, given previous reports by others.[121–123] However, this was not the case in our study, with children from both groups sleeping almost 8 hours, on average, per night. Interestingly, a difference in sleep timing was noted between the two groups. Children and adolescents with severe obesity had significantly later sleep timing, both overall and when considering school nights and weekend nights separately, compared to peers with normal weight. To our knowledge, the study in Paper I is the first to compare sleep timing between treatment-seeking children with obesity and peers with normal weight using objective sleep measures. Current research identified at the time

of writing Paper I described similar associations between sleep timing and weight status in children across the weight spectrum.[201–205] By contrast, others' research reported since publication of Paper I provided both contradictory and similar findings. Taylor *et al*.[123] used objective measures (actigraphy) to measure sleep behavior in 823 children aged 6–10 years and found a consistent beneficial effect of a longer sleep duration on BMI *z*-scores, but with no association between sleep timing and BMI. These findings were supported by a US study from 2020 based on self-reporting of 1254 adolescents (aged 12–17 years) on sleep duration and timing.[206] An inverse relationship between duration and obesity was found, but no association between sleep timing (and social jetlag) and obesity, in the overall population. However, later weekend sleep timing was associated with higher odds of developing obesity in girls.[206] Further, a study of treatment-seeking children with obesity aged 8–12 years[168] found that each hour of weekday bedtime delay was associated with an additional 6.17% overweight.[168] These inconsistent findings highlight the need for further studies to elucidate the relationship between sleep timing and weight status.[125,206]

Of importance is to have a clear understanding of the mechanisms driving the sleep–obesity relationship.[108,126] Therefore, we investigated how different sleep behaviors were linked to other behavioral factors known to cause obesity in children. In our sample of children and adolescents with severe obesity and those with normal weight, later sleep timing was associated with more screen time and less time in MVPA. These results align with previous cross-sectional findings, suggesting a link between later sleep and increased sedentary activity and screen time.[127] In contrast to previous research, no association between sleep duration and screen time was found in our sample,[127,128] and surprisingly, we found an inverse association between sleep duration and time in MVPA. Our findings highlight the need for longitudinal studies to fully understand how sleep impacts weight regulation in youth.

In the study described in Paper I, significantly more sleep problems were observed in children and adolescents with severe obesity, compared to peers with normal weight. Of note, while the term "sleep problems" can include a wide range of different problems and conditions related to sleep, the study in Paper I only addressed

the question of whether children experienced “current sleep problems” or not; the lack of elaboration of the subject is a clear limitation. It is possible that the observed group difference relates to the later sleep timing observed in children with severe obesity as parents might have interpreted going to bed later and waking up later as sleep problems. The findings could also potentially indicate the presence of other kinds of sleep problems such as obstructive sleep apnea or insomnia, as many studies have reported a higher prevalence of obstructive sleep apnea in children with obesity.[132,207–209] Interestingly, another recently published study from our research group, not included in this thesis, compared sleep between children with obesity and those with normal weight using polysomnography.[132] The results showed that symptoms of sleep-related breathing disorders in children with obesity often escaped the attention of parents, who also did not recognize the potential significance of snoring.[132]

The study presented in Paper II assessed for changes in sleep duration and timing, physical activity, and eating behavior following FBSFT and TAU. Of these, sleep timing was the only variable that showed a significant difference between treatment groups from pre- to posttreatment. However, as within-treatment group changes from pre- to posttreatment were not statistically significant for either group, it was considered unlikely that the observed difference in changes was clinically meaningful. Further, an overall significant reduction in time spent on MVPA was observed for all participants, regardless of treatment group. This is disappointing, albeit not unexpected, given the lack of evidence of a substantial increase in MVPA in both intervention and control groups in the literature.[4,9] Altogether, these results indicated that the observed differences in weight-related outcomes between FBSFT and TAU reported in Paper II are unlikely to be explained by differences in the lifestyle behaviors measured.

In the FABO study, changes in eating behavior were assessed using the DEBQ, and a relevant point of discussion is whether this assessment was sufficient. A central component of FBSFT is establishing healthy eating patterns within the family, which includes regular family meals, frequent meals (breakfast, lunch, dinner, and supper), and avoiding eating in front of a screen. While favorable changes in

these areas were not measured, they might have positively influenced the participants' weight-related outcomes, as reported by others.[87,91] Further, features of the FBSFT program, such as focus on weight loss and dietary restriction, may be conceptualized as part of eating disorder pathology,[210,211] while at the same time representing desired outcomes in pediatric obesity treatment.[212] This is a topic of long-standing interest and considerable debate.[211] Mean scores for restrained eating (measured with the DEBQ) at baseline were 2.65 and 2.74 for the FBSFT group and TAU group, respectively, with scores defined within the normal eating spectrum.[174] No significant differences in between- or within-group changes were observed from pre- to posttreatment. However, measurement of eating disorder symptoms in patients undergoing obesity treatment is complicated. Dietary restraint, for instance, may be considered an indicator of adherence to the intervention that might be necessary for treatment success rather than a symptom of disordered eating.[211,212] The phenomenon of restrained eating would therefore be context-dependent, and could bear different meanings and implications in a general population sample/normal weight sample, compared to a sample of individuals undergoing obesity treatment. In pediatric obesity treatment, dietary changes, including dietary restrictions, are undertaken under professional supervision; the plan is designed to maintain nutritional adequacy and regularity of meals, while focusing on reinforcing behaviors (e.g., realistic goal setting), which allow for long-term behavioral change and reduce the risk for disordered eating pathology.[212]

5.2.2 Improved weight-related outcomes following FBSFT

The findings reported in Paper II demonstrated that significantly greater improvements in weight-related outcomes were obtained with FBSFT than with TAU. The between-group difference in change in BMI SDS of 0.19 units was consistent with findings from the two latest Cochrane reviews on diet, physical activity, and behavioral interventions in pediatric obesity.[67,68] Due to various differences across pediatric obesity intervention studies, including different referral processes, recruitment methods, clinical settings, training of clinical staff, demographics, and treatment delivery and duration, as well as timing, it is difficult to perform meaningful comparisons between studies on treatment of pediatric obesity.[60]

Therefore, in Paper II, our review of extant research focused on studies that evaluated standardized FBT. The efficacy studies reported by Epstein *et al*. showed an impressive pooled BMI SDS change of −1.20 units at 6 months,[75] compared to our BMI SDS change of −0.16 units, for the FBSFT group. As previous implementation studies both in children and adults have shown reduced treatment effectiveness, compared to results from efficacy studies, lower treatment effectiveness was therefore expected,[82] in line with previous effectiveness studies utilizing FBT.[213–217] To date, the best results when implementing FBT in a clinical health-care setting have been generated from two Icelandic studies, one an RCT[213] (n = 16) and the other a study on a clinical sample of children with obesity (n = 84).[217] These two studies reported a mean BMI SDS reduction of −0.32 and −0.40, respectively.[213,217] However, a limitation of these studies is that the effect was restricted to those who completed treatment, which likely produced better outcomes than if all participants were included or an intention-to-treat analysis was performed. Other effectiveness studies on FBT have reported a mean reduction of BMI SDS in the range of −0.10 to −0.15.[214–216]

Plausible explanations for the more modest BMI SDS reduction in our sample, when compared to the original studies from Epstein's group[75] discussed in Paper II, include the fact that our study was performed in a regular outpatient clinic, in contrast to tightly controlled settings in a research clinic,[75] the experience level of staff involved in treatment delivery, treatment modification from a mixed (i.e., group and individual) format to an individual family format,[82] and high mean BMI scores of participants at baseline. Hayes and colleagues recently[218] reported greater changes in BMI SDS in children with nonsevere obesity, compared to those with severe obesity, following FBT. However, it is worth nothing that higher initial BMI SDS in participating children in other studies have been associated with greater reductions in BMI SDS.[72,217,219] Further, the broad age range of the participating children in our study (6–18 years), compared to an age range of 7–12 years often applied in FBT studies,[83] might have resulted in more modest treatment results in terms of BMI SDS changes, as younger children seem to achieve larger reductions in BMI SDS when following pediatric obesity treatment.[62,72,220,221] This finding, however, might reflect

the limitation imposed by the use of BMI SDS in children and adolescents with severe obesity, with a wide range of high BMI values corresponding to a relative narrow range of BMI SDS, and more so among adolescents than in preschool-aged children.[22] In a clinical sample from two specialist outpatient clinics in Norway, younger age (<12 years) was associated with a larger reduction in BMI SDS, when compared with adolescence, although this age-related difference was not observed for %IOTF-25.[220] The number of sessions in our study, 17 in total, which was designed to fit with treatment delivery in the health-care setting, was also lower than the current recommendation of at least 26 hours of comprehensive, intensive behavioral intervention to improve weight status in children and adolescents with obesity.[183]

5.2.2.1 Why is FBSFT more effective than TAU?

A highly interesting question is which features of FBSFT make the treatment more effective in improving weight-related outcomes, compared to TAU. Our study was not a dismantling study and thus was not designed to scientifically examine the effectiveness of different treatment components. However, the effectiveness in producing change in different targeted lifestyle behaviors was examined. As previously discussed, the observed difference in BMI-related measures is likely not explained by changes in the measured lifestyle behaviors (sleep, physical activity, and eating behavior). It is plausible that the observed difference could be partly explained by a more beneficial reduction in caloric intake in FBSFT. Both FBSFT and TAU focus on portion size/quantity of food, but FBSFT has a more structured approach with self-monitoring of food intake on a week-to-week basis, and also introduces families to the easy-to-use Traffic Light Eating Plan.[82] Overall, FBSFT represents a more structured approach with shorter between-session intervals that might be beneficial, in addition to also having a more family-oriented approach compared to TAU. A family-oriented approach when treating pediatric obesity is advantageous, although the optimal extent of family involvement has not been established.[72] Parental weight changes have been shown to be a strong predictor of child weight change in FBT.[189,222] However, actual interdependence—that is, where the outcomes (child/parent) are dependent on, and influences, one another—have been underexplored.[223] As FBT catalyzes changes in the shared family environment,

it would have been interesting to assess for changes also in nontarget siblings/family members, thus representing a broader family-based effect of the treatment and potential support for the participating children. Previous research has shown beneficial weight losses for nontargeted siblings.[224]

5.2.2.2 Individual treatment responses

Children and adolescents with obesity are a heterogeneous group,[220] as reflected by their treatment responses. Therefore, it is important to report on individual treatment responses, along with mean data. In the FABO study, examination of individual treatment responses showed that significantly more participants in FBSFT achieved a clinically meaningful reduction of ≥0.25 BMI SDS, compared to those in TAU (31.5% versus 13%, respectively). One can question whether a change of ≥0.25 BMI SDS is an appropriate cutoff value, as there is no consensus on a threshold that indicates a clinically meaningful reduction in BMI SDS. A reduction of ≥0.25 BMI SDS has been reported to produce beneficial metabolic changes and is a cutoff value applied by others.[69,144,145] However, as even stable/modest reductions in BMI SDS (≥0.00–0.10) have been associated with improvement in several cardiovascular risk factors in Norwegian children aged 7–17 years,[141] it could be justified to use a lower cutoff value. In fact, some previous studies have defined successful treatment as no increase in BMI SDS,[141,225] which may be reasonable considering that untreated pediatric obesity is likely to progress with predictable morbidity and mortality.[50]

Inclusion of data on individual treatment responses revealed that a subgroup of participants responded rather well to TAU, an observation that would have been left unnoticed if only the mean group change of 0.03 BMI SDS was reported. To date, data on heterogeneity in weight loss and BMI-related responses in pediatric obesity treatment are sparse.[38] A US study from 2019 reported great variability in BMI changes following lifestyle, pharmacotherapy, and surgical treatment of severe obesity in adolescents; the group also reported that changes in cardiometabolic risk factors demonstrated similarly high variability.[38] Identifying the factors driving variability in response to treatment would help in advancing precision medicine approaches to treatment of pediatric obesity.[38,62] Younger age, rapid treatment

response,[226,227] higher initial body weight,[228] parental weight loss, greater social support, and lower degree of parental psychopathology have been identified as predictors of treatment effect.[72] Further, tailoring of FBT to address various comorbid concerns has been suggested by some groups.[217] However, factors that could be predictors of treatment response in youth need to be further investigated.[62,229] Prospective studies examining potential biomarkers and biopsychosocial factors (observable traits resulting from genetic and environmental influence) are warranted, and can help in better tailoring of treatment and targeting key factors to optimize health behaviors and weight management.[62]

5.2.3 Reported barriers to treatment participation among families

The study presented in Paper III investigated families' perceived barriers to treatment participation. Of the four subscales of barriers examined in relation to completion of FBSFT, families who did not complete FBSFT reported significantly more barriers related to the subscales "stressors and obstacles that compete with treatment" and "perceived relevance of treatment," compared to those who completed treatment. No group difference was observed for the subscales "treatment demands and issues" and "relationship with the therapist."

These findings contribute to improving our understanding of barriers hindering treatment completion, and add to previous research reporting a high degree of family stressors as a challenge in terms of adherence to pediatric obesity treatment.[154,155] Stressors can represent a wide variety of issues (e.g., psychological, logistical) that need to be addressed to enhance the likelihood of treatment completion. The group difference observed for the subscale "perceived relevance of treatment" suggests that FBSFT was perceived as less suited to meet expectations and needs by families who did not complete treatment. This finding underscores the importance of helping caregivers understand the importance of participating in treatment when a child is diagnosed with obesity,[154,230] and of health-care providers to recognize and consider each family's expectations and needs before and during treatment.

The lack of difference between completers and noncompleters for the subscales "treatment demands and issues" and "relationship with the therapist" contrasts with previous findings reporting barriers related to treatment demands[217]

and dissatisfaction with treatment providers as reasons for participants leaving treatment prematurely.[147] In our study sample, the whole group of participating families had a low mean score for the subscale "relationship with the therapist," indicating that these families experienced a supportive, strong therapeutic alliance with their therapist. This is an encouraging result in respect of FBSFT and for the FABO study, and contrasts with previous research reporting that patients with obesity may experience stigmatization and a lack of trust and connection with health-care providers within the health-care system.[56,151,231,232]

Moreover, the study described in Paper III also examined the ten barriers with the highest mean rating for families and therapists, with a view to include two different perspectives on perceived barriers and to gain insight into alignments and differences between caregiver(s) and receiver(s). Both parents and therapists perceived stress in the parents' life during treatment as the main barrier, whereas the rest of the top ten list had different ranking of barriers between families and therapists. The largest discrepancy was reported for the barrier "treatment did not seem necessary," with parents perceiving their child as less in need of treatment, compared to therapists' perception. Interestingly, a discrepancy was observed in relation to how demanding the treatment was perceived for the families: the therapists perceived that the treatment added another stressor to (the parents') life to a larger degree than did the parents (third highest barrier versus eighth highest barrier, respectively). This is an important observation. Lack of agreement between caregivers' and receivers' view on what is perceived to be challenging while on treatment can represent misalignment in understanding of barriers and potentially can become a barrier on its own. Therefore, inclusion of participants' perspectives and perceptions and related discussions between participants and therapists when designing and implementing clinical interventions should be emphasized.[233]

In addition to the four subscales of barriers, assessment also included 14 questions about specific critical events that could result in treatment termination, including specific, discrete events that might be more prevalent among noncompleters, but not seen as barriers that account for the high attrition rates that are characteristic in child and adolescent therapy.[177] Of note, these were reported critical

events that occurred during the course of treatment, and not before. In our sample, no significant differences between noncompleters and completers of treatment were observed for the critical event scale, although a trend towards a higher number of reported critical events among treatment completers was noted. Future work on understanding how barriers to participation affect treatment retention and on the development of strategies to address these barriers, as well as information on the timing and frequency of such discussions, could enhance treatment engagement and retention among families.

6. Conclusions

Based on the specific study aims and hypotheses, the conclusions can be summarized as follows:

- Children and adolescents with severe obesity had significantly later sleep timing, compared to peers with normal weight. Children and adolescents with severe obesity also presented with significantly more sleep problems. In disagreement with the study hypothesis, sleep duration and social jetlag were not significantly different between children and adolescents with severe obesity and those with normal weight.
- Later sleep timing was related to other obesogenic behaviors—that is, longer screen time and reduced MVPA—in children and adolescents. Sleep duration was inversely associated with MVPA. No other significant associations were observed between the sleep variables and obesogenic behaviors investigated.
- Delivered from the Obesity Outpatient Clinic, significantly greater improvements in BMI-related outcomes were obtained with FBSFT, than with TAU among children with severe obesity (aged 6–18 years). Investigation of individual treatment responses showed that significantly more children receiving FBSFT achieved a clinically meaningful BMI SDS reduction of ≥0.25, compared to children receiving TAU (31.5% versus 13%, respectively).
- There was a significant difference in changes in sleep timing from pre- to posttreatment between FBSFT and TAU groups, although this was deemed not to be clinically meaningful. There were, however, no significant differences in change in sleep duration, physical activity, and eating behavior between treatment groups. This outcome was against the study hypothesis, and it was therefore concluded that it was unlikely the beneficial changes in weight-related outcomes exhibited with FBSFT, compared to TAU, could be explained by differences in sleep duration, physical activity, or eating behavior.

- Families were more likely to withdraw from FBSFT prematurely when experiencing a high burden of life stressors or when treatment was not meeting their expectations and addressing the perceived needs of the family.

7. Perspectives

7.1 Implications for research and clinical practice

The study results presented in this thesis support a more widespread implementation of FBSFT, given the positive findings on weight-related outcomes and the possibility to implement FBSFT and engage families in the intervention in a real-world health-care setting. However, the different elements of FBSFT should be investigated further to determine which components would require consolidating and which could be potentially redundant. Studies measuring caloric intake and diet quality from pre- to posttreatment are certainly warranted. Further, the long-term effects of FBSFT also need to be established.

The wide variability in individual treatment responses observed in relation to the two different treatment interventions for pediatric obesity is not sufficiently understood. Hence, factors that can predict treatment outcomes need to be investigated in prospective studies.

Results here showed that sleep timing might represent a risk factor for pediatric obesity, independent of sleep duration. However, inconsistent findings across studies highlight the need for more research in this area, including research on the mechanisms driving the sleep–obesity relationship. Results from Paper I and the overall literature on the subject emphasize the role of sleep as an important element of pediatric obesity treatment in the future.

Study findings here showed that physical activity was addressed similarly in both FBSFT and TAU, with a significant decrease in time spent on MVPA observed for all participants combined. These results highlight the need to include other strategies when addressing movement behavior in children and adolescents with obesity. For example, treatment could include additional focus on barriers that deter children with severe obesity from engaging in physical activity, as well as supervised sessions exploring different activities, possibly for the whole family.

The nonsignificant findings on eating behavior, both in relation to sleep behaviors (Paper I) and treatment (Paper II), has led us to reflect on the tools that are

available for assessing disordered eating among children and adolescents with severe obesity. We see a need to develop tools that are validated specifically for youth with obesity, and that are meaningful in terms of their health behavior change and health outcomes, including weight, as well as other health domains (e.g., physical, mental, social).

Increased knowledge in experienced barriers during treatment could inform the development of strategies aimed at enhancing retention rates and minimizing or preventing treatment dropout. The results showing a higher degree of perceived barriers in noncompleters highlight the need to investigate potential barriers that arise when families enter treatment, and the need for therapists to take these barriers into consideration when planning treatment sessions and follow-up.

7.2 Pediatric obesity treatment in the future

Pediatric obesity treatment in the future is likely to include a more personalized approach, with potential sequences, or combinations, of interventions targeting patients' unique pathophysiology.[234] Recent research on the use of anti-obesity pharmacotherapy, in combination with lifestyle intervention, in adolescents has yielded promising results.[63] Regarding innovations in lifestyle treatment, different digital solutions, such as use of mobile apps and online monitoring tools, are likely to play an important role in the future.[69,79] Thus, multiple promising avenues exist that would help enhance personalization, improve the effects, and promote accessibility of pediatric obesity treatment, all of which involve lifestyle intervention as a core approach. In this way, we can optimize youth's weight-related behaviors and health trajectories.

References

1. Helsedirektoratet. *Nasjonale faglige retningslinjer for primærhelsetjenesten. Forebygging og behandling av overvekt og fedme hos barn og unge*. Oslo, Helsedirektoratet; 2010.
2. Skjåkødegård HF, Danielsen YS, Morken M, *et al*. Study protocol: a randomized controlled trial evaluating the effect of family-based behavioral treatment of childhood and adolescent obesity—The FABO-study. *BMC Public Health*. 2016;**16**(1):1106.
3. Rosen H. Is obesity a disease or a behavior abnormality? Did the AMA get it right? *Mo Med*. 2014;**111**(2):104–8.
4. Katz DL. Perspective: obesity is not a disease. *Nature*. 2014;**508**(7496):S57.
5. Farpour-Lambert NJ, Baker JL, Hassapidou M, *et al*. Childhood obesity is a chronic disease demanding specific health care—a position statement from the Childhood Obesity Task Force (COTF) of the European Association for the Study of Obesity (EASO). *Obesity Facts*. 2015;**8**(5):342–9.
6. Fonvig CE, Kloppenborg JT, Nielsen TRH, *et al*. [Obesity in children and adolescents is a chronic disease]. *Ugeskr Laeger*. 2021;**183**(31):V01210082.
7. World Health Organization. *WHO European Regional Obesity Report 2022.* Copenhagen, WHO Regional Office for Europe; 2022.
8. Cammermeyer I. *Derfor må vi anerkjenne fedme som en sykdom*. 2022. Available from: https://www.altomdinhelse.no/verdens-fedmedag/derfor-ma-vi-anerkjenne-fedme-som-en-sykdom/. Accessed March 4, 2022.
9. World Health Organization. *Obesity and overweight*. [Fact sheet]. Geneva: World Health Organization; 2021.
10. Freedman DS, Sherry B. The validity of BMI as an indicator of body fatness and risk among children. *Pediatrics*. 2009;**124** Suppl 1:S23–34.
11. Adab P, Pallan M, Whincup PH. Is BMI the best measure of obesity? *BMJ*. 2018;**360**:k1274.
12. Kansra AR, Lakkunarajah S, Jay MS. Childhood and adolescent obesity: a review. *Front Pediatr*. 2020;**8**:581461.
13. Cole TJ, Lobstein T. An improved algorithm to harmonize child overweight and obesity prevalence rates. *Pediatr Obes*. 2022:e12970.
14. Jebeile H, Kelly AS, O'Malley G, Baur LA. Obesity in children and adolescents: epidemiology, causes, assessment, and management. *Lancet Diabetes Endocrinol*. 2022;**10**:351–65.
15. Torbahn G, Brauchmann J, Axon E, *et al*. Surgery for the treatment of obesity in children and adolescents. *Cochrane Database Syst Rev*. 2022;**9**(9):CD011740.
16. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*. 2000;**320**(7244):1240–3.
17. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;**7**(4):284–94.

18. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. *Bull World Health Organ*. 2007;**85**(9):660–7.
19. Barlow SE, Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics*. 2007;**120** Suppl 4:164–92.
20. Juliusson PB, Roelants M, Nordal E, *et al*. Growth references for 0–19 year-old Norwegian children for length/height, weight, body mass index and head circumference. *Ann Hum Biol*. 2013;**40**(3):220–7.
21. Løkling HL, Roelants M, Kommedal KG, *et al*. Monitoring children and adolescents with severe obesity: body mass index (BMI), BMI z-score or percentage above the International Obesity Task Force overweight cut-off? *Acta Paediatr*. 2019;**108**(12):2261–6.
22. Júlíusson PB, Roelants M, Benestad B, *et al*. Severe obesity is a limitation for the use of body mass index standard deviation scores in children and adolescents. *Acta Paediatr*. 2018;**107**(2):307–14.
23. Woo JG. Using body mass index Z-score among severely obese adolescents: a cautionary note. *Int J Pediatr Obes*. 2009;**4**(4):405–10.
24. World Obesity Federation. *Atlas of Childhood Obesity.* London, World Obesity Federation; 2019.
25. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet*. 2017;**390**(10113):2627–42.
26. Folkehelseinstituttet. *[Norgeshelsa statistikkbank] Overvekt og fedme, 8-åringer*. Oslo, Folkehelseinsituttet; 2017. Available from: http://norgeshelsa.no/norgeshelsa/index.jsp?
27. Folkehelseinstituttet. *Overvekt og fedme blant ungdom (indikator 13) [nettdokument].* Oslo, Folkehelseinstituttet; 2021 (last updated November 24, 2021). Available from: https://www.fhi.no/nettpub/ncd/overvekt/ungdom/?term=&h=1
28. Júlíusson PB, Eide GE, Roelants M, Waaler PE, Hauspie R, Bjerknes R. Overweight and obesity in Norwegian children: prevalence and socio-demographic risk factors. *Acta Paediatr*. 2010;**99**(6):900–5.
29. Øvrebø B, Bergh IH, Stea TH, *et al*. Overweight, obesity, and thinness among a nationally representative sample of Norwegian adolescents and changes from childhood: associations with sex, region, and population density. *PLoS One*. 2021;**16**(8):e0255699.
30. Afshin A, Forouzanfar MH, Reitsma MB, *et al*. Health effects of overweight and obesity in 195 countries over 25 years. *N Engl J Med*. 2017;**377**(1):13–27.
31. Kumar S, Kelly AS. Review of childhood obesity: from epidemiology, etiology, and comorbidities to clinical assessment and treatment. *Mayo Clin Proc*. 2017;**92**(2):251–65.
32. Meyer HE, m.fl. *[Nettpublikasjon] Overvekt og fedme i Noreg*. Oslo, Folkehelseinstituttet; 2017 (last updated August 31, 2022).Available from: https://www.fhi.no/nettpub/hin/ikke-smittsomme/overvekt-og-

fedme/#:~:text=definert%20som%20bukfedme.-,Overvekt%20og%20fedme%20hos%20barn%20og%20ungdom,2021%3B%20FHI%2C%202021a).

33. Lange SJ, Kompaniyets L, Freedman DS, *et al*. Longitudinal trends in body mass index before and during the COVID-19 pandemic among persons aged 2–19 years: United States, 2018–2020. *MMWR Morb Mortal Wkly Rep*. 2021;**70**(37):1278–83.
34. Jarnig G, Jaunig J, Kerbl R, Strenger V, Haeusler G, van Poppel MNM. Acceleration in BMI gain following COVID-19 restrictions. A longitudinal study with 7- to 10-year-old primary school children. *Pediatr Obes*. 2022;**17**(6):e12890.
35. Vogel M, Geserick M, Gausche R, *et al*. Age- and weight group-specific weight gain patterns in children and adolescents during the 15 years before and during the COVID-19 pandemic. *Int J Obes (Lond)*. 2022;**46**(1):144–52.
36. Smith JD, Fu E, Kobayashi MA. Prevention and management of childhood obesity and its psychological and health comorbidities. *Annu Rev Clin Psychol*. 2020;**16**:351–78.
37. Sulc J, Winkler TW, Heid IM, Kutalik Z. Heterogeneity in obesity: genetic basis and metabolic consequences. *Curr Diab Rep*. 2020;**20**(1):1.
38. Ryder JR, Kaizer AM, Jenkins TM, Kelly AS, Inge TH, Shaibi GQ. Heterogeneity in response to treatment of adolescents with severe obesity: the need for precision obesity medicine. *Obesity (Silver Spring)*. 2019;**27**(2):288–94.
39. Cardel MI, Atkinson MA, Taveras EM, Holm JC, Kelly AS. Obesity Treatment among adolescents: a review of current evidence and future directions. *JAMA Pediatr*. 2020;**174**(6):609–17.
40. Bronfenbrenner U, Ceci SJ. Nature–nurture reconceptualized in developmental perspective: a bioecological model. *Psychol Rev*. 1994;**101**(4):568–86.
41. Davison KK, Birch LL. Childhood overweight: a contextual model and recommendations for future research. *Obes Rev*. 2001;**2**(3):159–71.
42. Ohri-Vachaspati P, DeLia D, DeWeese RS, Crespo NC, Todd M, Yedidia MJ. The relative contribution of layers of the Social Ecological Model to childhood obesity. *Public Health Nutr*. 2015;**18**(11):2055–66.
43. Pereira M, Padez CMP, Nogueira H. Describing studies on childhood obesity determinants by Socio-Ecological Model level: a scoping review to identify gaps and provide guidance for future research. *Int J Obes (Lond)*. 2019;**43**(10):1883–90.
44. Browne NT, Snethen JA, Greenberg CS, *et al*. When pandemics collide: the impact of COVID-19 on childhood obesity. *J Pediatr Nurs*. 2021;**56**:90–8.
45. Centers for Disease Control and Prevention. *The social-ecological model: a framework for prevention*. 2007. Available from: https://www.cdc.gov/violenceprevention/about/social-ecologicalmodel.html. Accessed December 9, 2022.
46. Marcus C, Danielsson P, Hagman E. Pediatric obesity: long-term consequences and effect of weight loss. *J Intern Med*. 2022;**292**(6):870–91.

47. Morales Camacho WJ, Molina Díaz JM, Plata Ortiz S, Plata Ortiz JE, Morales Camacho MA, Calderón BP. Childhood obesity: aetiology, comorbidities, and treatment. *Diabetes Metab Res Rev*. 2019;**35**(8):e3203.
48. Caprio S, Santoro N, Weiss R. Childhood obesity and the associated rise in cardiometabolic complications. *Nat Metab*. 2020;**2**(3):223–32.
49. Horesh A, Tsur AM, Bardugo A, Twig G. Adolescent and childhood obesity and excess morbidity and mortality in young adulthood: a systematic review. *Curr Obes Rep*. 2021;**10**(3):301–10.
50. Cuda S, Censani M. Progress in pediatric obesity: new and advanced therapies. *Curr Opin Pediatr*. 2022;**34**(4):407–13.
51. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. *Obes Rev*. 2016;**17**(2):95–107.
52. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. *Pediatrics*. 2005;**115**(1):22–7.
53. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes (Lond)*. 2011;**35**(7):891–8.
54. Lindberg L, Danielsson P, Persson M, Marcus C, Hagman E. Association of childhood obesity with risk of early all-cause and cause-specific mortality: a Swedish prospective cohort study. *PLoS Med*. 2020;**17**(3):e1003078.
55. Puhl RM, Lessard LM. Weight stigma in youth: prevalence, consequences, and considerations for clinical practice. *Curr Obes Rep*. 2020;**9**(4):402–11.
56. Haqq AM, Kebbe M, Tan Q, Manco M, Salas XR. Complexity and stigma of pediatric obesity. *Child Obes*. 2021;**17**(4):229–40.
57. Lindberg L, Hagman E, Danielsson P, Marcus C, Persson M. Anxiety and depression in children and adolescents with obesity: a nationwide study in Sweden. *BMC Med*. 2020;**18**(1):30.
58. Quek YH, Tam WWS, Zhang MWB, Ho RCM. Exploring the association between childhood and adolescent obesity and depression: a meta-analysis. *Obes Rev*. 2017;**18**(7):742–54.
59. Martin A, Booth JN, McGeown S, *et al*. Longitudinal associations between childhood obesity and academic achievement: systematic review with focus group data. *Curr Obes Rep*. 2017;**6**(3):297–313.
60. Wyse C, Case L, Walsh Ó, *et al*. Evaluating 12 years of implementing a multidisciplinary specialist child and adolescent obesity treatment service: patient-level outcomes. *Front Nutr*. 2022;**9**:895091.
61. Ells LJ, Rees K, Brown T, *et al*. Interventions for treating children and adolescents with overweight and obesity: an overview of Cochrane reviews. *Int J Obes (Lond)*. 2018;**42**(11):1823–33.
62. Kelly AS, Marcus MD, Yanovski JA, Yanovski SZ, Osganian SK. Working toward precision medicine approaches to treat severe obesity in adolescents: report of an NIH workshop. *Int J Obes (Lond)*. 2018;**42**(11):1834–44.

63. Weghuber D, Barrett T, Barrientos-Pérez M, *et al*. Once-weekly semaglutide in adolescents with obesity. *N Engl J Med*. 2022 Nov 2. doi: 10.1056/NEJMoa2208601.
64. Srivastava G, Fox CK, Kelly AS, *et al*. Clinical considerations regarding the use of obesity pharmacotherapy in adolescents with obesity. *Obesity (Silver Spring)*. 2019;**27**(2):190–204.
65. Woodard K, Louque L, Hsia DS. Medications for the treatment of obesity in adolescents. *Ther Adv Endocrinol Metab*. 2020;**11**:2042018820918789.
66. Wilfley DE, Hayes JF, Balantekin KN, Van Buren DJ, Epstein LH. Behavioral interventions for obesity in children and adults: evidence base, novel approaches, and translation into practice. *Am Psychol*. 2018;**73**(8):981–93.
67. Mead E, Brown T, Rees K, *et al*. Diet, physical activity and behavioural interventions for the treatment of overweight or obese children from the age of 6 to 11 years. *Cochrane Database Syst Rev*. 2017;**6**(6):CD012651.
68. Al-Khudairy L, Loveman E, Colquitt JL, *et al*. Diet, physical activity and behavioural interventions for the treatment of overweight or obese adolescents aged 12 to 17 years. *Cochrane Database Syst Rev*. 2017;**6**(6):CD012691.
69. Hagman E, Johansson L, Kollin C, *et al*. Effect of an interactive mobile health support system and daily weight measurements for pediatric obesity treatment, a 1-year pragmatical clinical trial. *Int J Obes (Lond)*. 2022;**46**(8):1527–33.
70. Fowler LA, Grammer AC, Staiano AE, *et al*. Harnessing technological solutions for childhood obesity prevention and treatment: a systematic review and meta-analysis of current applications. *Int J Obes (Lond)*. 2021;**45**(5):957–81.
71. Boff RM, Liboni RPA, Batista IPA, de Souza LH, Oliveira MDS. Weight loss interventions for overweight and obese adolescents: a systematic review. *Eat Weight Disord*. 2017;**22**(2):211–29.
72. Altman M, Wilfley DE. Evidence update on the treatment of overweight and obesity in children and adolescents. *J Clin Child Adolesc Psychol*. 2015;**44**(4):521–37.
73. Lee JS, Jin MH, Lee HJ. Global relationship between parent and child obesity: a systematic review and meta-analysis. *Clin Exp Pediatr*. 2022;**65**(1):35–46.
74. Epstein LH, Wing RR, Steranchak L, Dickson B, Michelson J. Comparison of family-based behavior modification and nutrition education for childhood obesity. *J Pediatr Psychol*. 1980;**5**(1):25–36.
75. Epstein LH, Paluch RA, Roemmich JN, Beecher MD. Family-based obesity treatment, then and now: twenty-five years of pediatric obesity treatment. *Health Psychol*. 2007;**26**(4):381–91.
76. Epstein LH, Wing RR, Koeske R, Andrasik F, Ossip DJ. Child and parent weight loss in family-based behavior modification programs. *J Consult Clin Psychol*. 1981;**49**(5):674–85.
77. Epstein LH, Valoski A, Wing RR, McCurley J. 10-year follow-up of behavioral, family-based treatment for obese children. *JAMA*. 1990;**264**(19):2519–23.

78. Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychol*. 1994;**13**(5):373–83.
79. Fowler LA, Hampl SE, Dreyer Gillette ML, *et al*. Translating family-based behavioral treatment for childhood obesity into a user-friendly digital package for delivery to low-income families through primary care partnerships: the MO-CORD Study. *Child Obes*. 2021;**17**(S1):S30–8.
80. Wilfley DE, Stein RI, Saelens BE, *et al*. Efficacy of maintenance treatment approaches for childhood overweight: a randomized controlled trial. *JAMA*. 2007;**298**(14):1661–73.
81. Wilfley DE, Saelens BE, Stein RI, *et al*. Dose, content, and mediators of family-based treatment for childhood obesity: a multisite randomized clinical trial. *JAMA Pediatr*. 2017;**171**(12):1151–9.
82. Epstein LH, Schechtman KB, Kilanowski C, *et al*. Implementing family-based behavioral treatment in the pediatric primary care setting: design of the PLAN study. *Contemp Clin Trials*. 2021;**109**:106497.
83. Wilfley DE, Tibbs TL, Van Buren DJ, Reach KP, Walker MS, Epstein LH. Lifestyle interventions in the treatment of childhood overweight: a meta-analytic review of randomized controlled trials. *Health Psychol*. 2007;**26**(5):521–32.
84. Wilfley DE, Kolko RP, Kass AE. Cognitive-behavioral therapy for weight management and eating disorders in children and adolescents. *Child Adolesc Psychiatr Clin N Am*. 2011;**20**(2):271–85.
85. Epstein LH, Wrotniak BH. Future directions for pediatric obesity treatment. *Obesity*. 2010;**18**:S8–12.
86. Skjåkødegård HF, Danielsen YS, Morken M, *et al*. Study protocol: a randomized controlled trial evaluating the effect of family-based behavioral treatment of childhood and adolescent obesity: the FABO-study. *BMC Public Health*. 2016;**16**(1):1–9.
87. Verduci E, Bronsky J, Embleton N, *et al*. Role of dietary factors, food habits, and lifestyle in childhood obesity development: a position paper from the European Society for Paediatric Gastroenterology, Hepatology and Nutrition Committee on Nutrition. *J Pediatr Gastroenterol Nutr*. 2021;**72**(5):769–83.
88. Hayes JF, Fitzsimmons-Craft EE, Karam AM, Jakubiak J, Brown ML, Wilfley DE. Disordered eating attitudes and behaviors in youth with overweight and obesity: implications for treatment. *Curr Obes Rep*. 2018;**7**(3):235–46.
89. Braet C, O'Malley G, Weghuber D, *et al*. The assessment of eating behaviour in children who are obese: a psychological approach. A position paper from the European Childhood Obesity Group. *Obes Facts*. 2014;**7**(3):153–64.
90. Grammer AC, Best JR, Fowler LA, *et al*. General and eating disorder psychopathology in relation to short- and long-term weight change in treatment-seeking children: a latent profile analysis. *Ann Behav Med*. 2021;**55**(7):698–704.
91. Monzani A, Ricotti R, Caputo M, *et al*. A systematic review of the association of skipping breakfast with weight and cardiometabolic risk factors in children

and adolescents. What should we better investigate in the future? *Nutrients*. 2019;**11**(2):387.
92. Robinson TN, Banda JA, Hale L, *et al*. Screen media exposure and obesity in children and adolescents. *Pediatrics*. 2017;**140**(Suppl 2):S97–101.
93. Goldschmidt AB, Aspen VP, Sinton MM, Tanofsky-Kraff M, Wilfley DE. Disordered eating attitudes and behaviors in overweight youth. *Obesity (Silver Spring)*. 2008;**16**(2):257–64.
94. Welling MS, Abawi O, van den Eynde E, *et al*. Impact of the COVID-19 pandemic and related lockdown measures on lifestyle behaviors and well-being in children and adolescents with severe obesity. *Obes Facts*. 2022;**15**(2):186–96.
95. van Strien T, Oosterveld P. The children's DEBQ for assessment of restrained, emotional, and external eating in 7- to 12-year-old children. *Int J Eat Disord*. 2008;**41**(1):72–81.
96. Stabouli S, Erdine S, Suurorg L, Jankauskienė A, Lurbe E. Obesity and eating disorders in children and adolescents: the bidirectional link. *Nutrients*. 2021;**13**(12):4321.
97. Jebeile H, Lister NB, Baur LA, Garnett SP, Paxton SJ. Eating disorder risk in adolescents with obesity. *Obes Rev*. 2021;**22**(5):e13173.
98. Glasofer DR, Tanofsky-Kraff M, Eddy KT, *et al*. Binge eating in overweight treatment-seeking adolescents. *J Pediatr Psychol*. 2007;**32**(1):95–105.
99. Giel KE, Zipfel S, Schweizer R, *et al*. Eating disorder pathology in adolescents participating in a lifestyle intervention for obesity: associations with weight change, general psychopathology and health-related quality of life. *Obes Facts*. 2013;**6**(4):307–16.
100. Haines J, Kleinman KP, Rifas-Shiman SL, Field AE, Austin SB. Examination of shared risk and protective factors for overweight and disordered eating among adolescents. *Arch Pediatr Adolesc Med*. 2010;**164**(4):336–43.
101. Haines J, Neumark-Sztainer D. Prevention of obesity and eating disorders: a consideration of shared risk factors. *Health Educ Res*. 2006;**21**(6):770–82.
102. Bishop-Gilyard CT, Berkowitz RI, Wadden TA, Gehrman CA, Cronquist JL, Moore RH. Weight reduction in obese adolescents with and without binge eating. *Obesity (Silver Spring)*. 2011;**19**(5):982–7.
103. Grammer AC, Best JR, Fowler LA, *et al*. Change in parent and child psychopathology following obesity treatment and maintenance: a secondary data analysis. *Pediatr Obes*. 2022:e12971.
104. Talarico R, Janssen I. Compositional associations of time spent in sleep, sedentary behavior and physical activity with obesity measures in children. *Int J Obes (Lond)*. 2018;**42**(8):1508–14.
105. Westerterp KR, Plasqui G. Physical activity and human energy expenditure. *Curr Opin Clin Nutr Metab Care*. 2004;**7**(6):607–13.
106. O'Malley G, Ring-Dimitriou S, Nowicka P, *et al*. Physical activity and physical fitness in pediatric obesity: what are the first steps for clinicians? Expert conclusion from the 2016 ECOG Workshop. *Int J Exerc Sci*. 2017;**10**(4):487–96.

107. Wyszyńska J, Ring-Dimitriou S, Thivel D, *et al*. Physical activity in the prevention of childhood obesity: the position of the European Childhood Obesity Group and the European Academy of Pediatrics. *Front Pediatr*. 2020;**8**:535705.
108. Julian V, Haschke F, Fearnbach N, *et al*. Effects of movement behaviors on overall health and appetite control: current evidence and perspectives in children and adolescents. *Curr Obes Rep*. 2022;**11**(1):10–22.
109. Kolle E., Stokke JS, Hansen BH, Anderssen S. *Fysisk aktivitet blant 6-, 9- og 15-åringer i Norge. Resultater fra en kartlegging i 2011*. Oslo, Helsedirektoratet; 2012.
110. Bassett DR, John D, Conger SA, Fitzhugh EC, Coe DP. Trends in physical activity and sedentary behaviors of United States youth. *J Phys Act Health*. 2015;**12**(8):1102.
111. Jago R, Salway R, Emm-Collison L, Sebire SJ, Thompson JL, Lawlor DA. Association of BMI category with change in children's physical activity between ages 6 and 11 years: a longitudinal study. *Int J Obes*. 2019;**44**(2020):104–13.
112. Manz K, Mensink GBM, Finger JD, *et al*. Associations between physical activity and food intake among children and adolescents: results of KiGGS Wave 2. *Nutrients*. 2019;**11**(5):1060.
113. Thivel D, Chaput JP, Duclos M. Integrating sedentary behavior in the theoretical model linking childhood to adulthood activity and health? An updated framework. *Physiol Behav*. 2018;**196**:33–5.
114. Halford JCG, Bereket A, Bin-Abbas B, *et al*. Misalignment among adolescents living with obesity, caregivers, and healthcare professionals: ACTION Teens global survey study. *Pediatr Obes*. 2022:e12957.
115. Tremblay MS, Aubert S, Barnes JD, *et al*. Sedentary Behavior Research Network (SBRN): Terminology Consensus Project process and outcome. *Int J Behav Nutr Phys Act*. 2017;**14**(1):75.
116. LeBlanc AG, Gunnell KE, Prince SA, Saunders TJ, Barnes JD, Chaput J-P. The ubiquity of the screen: an overview of the risks and benefits of screen time in our modern world. *Translational Journal of the American College of Sports Medicine*. 2017;**2**(17):104–13.
117. Haghjoo P, Siri G, Soleimani E, Farhangi MA, Alesaeidi S. Screen time increases overweight and obesity risk among adolescents: a systematic review and dose-response meta-analysis. *BMC Prim Care*. 2022;**23**(1):161.
118. Crowe M, Sampasa-Kanyinga H, Saunders TJ, Hamilton HA, Benchimol EI, Chaput JP. Combinations of physical activity and screen time recommendations and their association with overweight/obesity in adolescents. *Can J Public Health*. 2020;**111**(4):515–22.
119. Agaronov A, Ash T, Sepulveda M, Taveras EM, Davison KK. Inclusion of sleep promotion in family-based interventions to prevent childhood obesity. *Child Obes*. 2018;**14**(8):485–500.
120. Yoong SL, Chai LK, Williams CM, Wiggers J, Finch M, Wolfenden L. Systematic review and meta-analysis of interventions targeting sleep and their

impact on child body mass index, diet, and physical activity. *Obesity (Silver Spring)*. 2016;**24**(5):1140–7.
121. Morrissey B, Taveras E, Allender S, Strugnell C. Sleep and obesity among children: a systematic review of multiple sleep dimensions. *Pediatr Obes*. 2020;**15**(4):e12619.
122. Glasgow TE, Adams EL, Ksinan A, *et al*. Sleep onset, duration, or regularity: which matters most for child adiposity outcomes? *Int J Obes (Lond)*. 2022;**46**(8):1502–9.
123. Taylor RW, Williams SM, Galland BC, *et al*. Quantity versus quality of objectively measured sleep in relation to body mass index in children: cross-sectional and longitudinal analyses. *Int J Obes (Lond)*. 2020;**44**(4):803–11.
124. Ruan H, Xun P, Cai W, He K, Tang Q. Habitual sleep duration and risk of childhood obesity: systematic review and dose–response meta-analysis of prospective cohort studies. *Sci Rep*. 2015;**5**:16160.
125. Dutil C, Podinic I, Sadler CM, *et al*. Sleep timing and health indicators in children and adolescents: a systematic review. *Health Promot Chronic Dis Prev Can*. 2022;**42**(4):150–69.
126. LeMay-Russell S, Schvey NA, Kelly NR, *et al*. Longitudinal associations between facets of sleep and adiposity in youth. *Obesity (Silver Spring)*. 2021;**29**(11):1760–9.
127. Krietsch KN, Chardon ML, Beebe DW, Janicke DM. Sleep and weight-related factors in youth: a systematic review of recent studies. *Sleep Med Rev*. 2019;**46**:87–96.
128. St-Onge MP. Sleep-obesity relation: underlying mechanisms and consequences for treatment. *Obes Rev*. 2017;**18** Suppl 1:34–9.
129. Blechner MMD, Williamson AAMA. Consequences of obstructive sleep apnea in children. *Curr Probl Pediatr Adolesc Health Care*. 2016;**46**(1):19–26.
130. Koren D, Gozal D, Philby MF, Bhattacharjee R, Kheirandish-Gozal L. Impact of obstructive sleep apnoea on insulin resistance in nonobese and obese children. *Eur Respir J*. 2016;**47**(4):1152–61.
131. Koren D, Dumin M, Gozal D. Role of sleep quality in the metabolic syndrome. *Diabetes Metab Syndr Obes*. 2016;**9**:281–310.
132. Danielsen YS, Skjåkødegård HF, Bjorvatn B, Juliusson PB, Pallesen S. Polysomnographic comparison of sleep in children with obesity and normal weight without suspected sleep-related breathing disorder. *Clin Obes*. 2022;**12**(1):e12493.
133. Ding C, Fan J, Yuan F, *et al*. Association between physical activity, sedentary behaviors, sleep, diet, and adiposity among children and adolescents in China. *Obes Facts*. 2022;**15**(1):26–35.
134. Matricciani L, Dumuid D, Paquet C, *et al*. Sleep and cardiometabolic health in children and adults: examining sleep as a component of the 24-h day. *Sleep Med*. 2021;**78**:63–74.
135. Hart CN, Jelalian E, Raynor HA. Behavioral and social routines and biological rhythms in prevention and treatment of pediatric obesity. *Am Psychol*. 2020;**75**(2):152–62.

136. World Health Organization. *Mapping the health system response to childhood obesity in the WHO European Region: an overview and country perspectives.* Copenhagen, WHO Regional Office for Europe; 2019.
137. Goldschmidt AB, Wilfley DE, Paluch RA, Roemmich JN, Epstein LH. Indicated prevention of adult obesity: how much weight change is necessary for normalization of weight status in children? *JAMA Pediatr*. 2013;**167**(1):21–6.
138. Skjåkødegård HF, Conlon RPK, Hystad SW, *et al*. Family-based treatment of children with severe obesity in a public healthcare setting: results from a randomized controlled trial. *Clin Obes*. 2022:e12513.
139. Ford AL, Hunt LP, Cooper A, Shield JP. What reduction in BMI SDS is required in obese adolescents to improve body composition and cardiometabolic health? *Arch Dis Child*. 2010;**95**(4):256–61.
140. Reinehr T, Lass N, Toschke C, Rothermel J, Lanzinger S, Holl RW. Which amount of BMI-SDS reduction is necessary to improve cardiovascular risk factors in overweight children? *J Clin Endocrinol Metab*. 2016;**101**(8):3171–9.
141. Kolsgaard ML, Joner G, Brunborg C, Anderssen SA, Tonstad S, Andersen LF. Reduction in BMI z-score and improvement in cardiometabolic risk factors in obese children and adolescents. The Oslo Adiposity Intervention Study: a hospital/public health nurse combined treatment. *BMC Pediatr*. 2011;**11**:47.
142. Dubnov-Raz G, Berry EM. What paediatric obesity treatment programmes work, and how can we measure their success? *Acta Paediatr*. 2017;**106**(11):1724–6.
143. Dalla Valle M, Laatikainen T, Lehikoinen M, Nykänen P, Jääskeläinen J. Paediatric obesity treatment had better outcomes when children were younger, well motivated and did not have acanthosis nigricans. *Acta Paediatr*. 2017;**106**(11):1842–50.
144. Hagman E, Danielsson P, Lindberg L, Marcus C. Paediatric obesity treatment during 14 years in Sweden: Lessons from the Swedish Childhood Obesity Treatment Register—BORIS. *Pediatr Obes*. 2020;**15**(7):e12626.
145. Danielsson P, Kowalski J, Ekblom Ö, Marcus C. Response of severely obese children and adolescents to behavioral treatment. *Arch Pediatr Adolesc Med*. 2012;**166**(12):1103–8.
146. Sharifi M, Marshall G, Goldman RE, Cunningham C, Marshall R, Taveras EM. Engaging children in the development of obesity interventions: exploring outcomes that matter most among obesity positive outliers. *Patient Educ Couns*. 2015;**98**(11):1393–401.
147. Miller BML, Brennan L. Measuring and reporting attrition from obesity treatment programs: a call to action! *Obes Res Clin Pract*. 2015;**9**(3):187–202.
148. Skelton JA, Beech BM. Attrition in paediatric weight management: a review of the literature and new directions. *Obes Rev*. 2011;**12**(5):e273–81.
149. Skelton JA, Martin S, Irby MB. Satisfaction and attrition in paediatric weight management. *Clin Obes*. 2016;**6**(2):143–53.
150. Kelleher E, Davoren MP, Harrington JM, Shiely F, Perry IJ, McHugh SM. Barriers and facilitators to initial and continued attendance at community-

based lifestyle programmes among families of overweight and obese children: a systematic review. *Obes Rev*. 2017;**18**(2):183–94.
151. Nobles J, Griffiths C, Pringle A, Gately P. Design programmes to maximise participant engagement: a predictive study of programme and participant characteristics associated with engagement in paediatric weight management. *Int J Behav Nutr Phys Act*. 2016;**13**(1):76.
152. Danieles PK, Ybarra M, Van Hulst A, *et al*. Determinants of attrition in a pediatric healthy lifestyle intervention: the CIRCUIT program experience. *Obes Res Clin Pract*. 2021;**15**(2):157–62.
153. Dhaliwal J, Nosworthy NM, Holt NL, *et al*. Attrition and the management of pediatric obesity: an integrative review. *Child Obes*. 2014;**10**(6):461–73.
154. Grootens-Wiegers P, van den Eynde E, Halberstadt J, Seidell JC, Dedding C. The "Stages Towards Completion Model": what helps and hinders children with overweight or obesity and their parents to be guided towards, adhere to and complete a group lifestyle intervention. *Int J Qual Stud Health Well-being*. 2020;**15**(1):1735093.
155. Silver MP, Cronin SM. Health care providers' perspectives on family compliance and behavior change in a childhood obesity program. *Health Educ Behav*. 2019;**46**(4):582–91.
156. Chung AE, Skinner AC, Steiner MJ, Perrin EM. Physical activity and BMI in a nationally representative sample of children and adolescents. *Clin Pediatr (Phila)*. 2012;**51**(2):122–9.
157. Avnieli Velfer Y, Phillip M, Shalitin S. Increased prevalence of severe obesity and related comorbidities among patients referred to a pediatric obesity clinic during the last decade. *Horm Res Paediatr*. 2019;**92**(3):169–78.
158. Flegal KM, Wei R, Ogden CL, Freedman DS, Johnson CL, Curtin LR. Characterizing extreme values of body mass index-for-age by using the 2000 Centers for Disease Control and Prevention growth charts. *Am J Clin Nutr*. 2009;**90**(5):1314–20.
159. Rotman SA, Fowler LA, Ray MK, *et al*. Family encouragement of healthy eating predicts child dietary intake and weight loss in family-based behavioral weight-loss treatment. *Child Obes*. 2020;**16**(3):218–25.
160. Jetté M, Sidney K, Blümchen G. Metabolic equivalents (METS) in exercise testing, exercise prescription, and evaluation of functional capacity. *Clin Cardiol*. 1990;**13**(8):555–65.
161. Júlíusson PB, Roelants M, Nordal E, *et al*. Growth references for 0–19 year-old Norwegian children for length/height, weight, body mass index and head circumference. *Ann Hum Biol*. 2013;**40**(3):220–7.
162. Chow CM, Wong SN, Shin M, *et al*. Defining the rest interval associated with the main sleep period in actigraph scoring. *Nat Sci Sleep*. 2016;**8**:321–8.
163. Meltzer LJ, Walsh CM, Traylor J, Westin AML. Direct comparison of two new actigraphs and polysomnography in children and adolescents. *Sleep*. 2012;**35**(1):159–66.
164. Meltzer LJ, Montgomery-Downs HE, Insana SP, Walsh CM. Use of actigraphy for assessment in pediatric sleep research. *Sleep Med Rev*. 2012;**16**(5):463–75.

165. Meltzer LJ, Wong P, Biggs SN, *et al*. Validation of actigraphy in middle childhood. *Sleep*. 2016;**39**(6):1219–24.
166. Chow CM, Wong SN, Shin M, *et al*. Defining the rest interval associated with the main sleep period in actigraph scoring. *Nat Sci Sleep*. 2016;**8**:321.
167. Hirshkowitz M, Whiton K, Albert SM, *et al*. National Sleep Foundation's updated sleep duration recommendations: final report. *Sleep Health*. 2015;**1**(4):233–43.
168. Zhou M, Lalani C, Banda JA, Robinson TN. Sleep duration, timing, variability and measures of adiposity among 8- to 12-year-old children with obesity. *Obes Sci Pract*. 2018;**4**(6):535–44.
169. Ekblom O, Nyberg G, Bak EE, Ekelund U, Marcus C. Validity and comparability of a wrist-worn accelerometer in children. *J Phys Act Health*. 2012;**9**(3):389–93.
170. Ekstedt M, Nyberg G, Ingre M, Ekblom O, Marcus C. Sleep, physical activity and BMI in six to ten-year-old children measured by accelerometry: a cross-sectional study. *Int J Behav Nutr Phys Act*. 2013;**10**:82.
171. Ekblom O, Nyberg G, Bak EE, Ekelund U, Marcus C, Ekblom O. Validity and comparability of a wrist-worn accelerometer in children. *J Phys Act Health*. 2012;**9**(3):389–93.
172. Braet C, Van Strien T. Assessment of emotional, externally induced and restrained eating behaviour in nine to twelve-year-old obese and non-obese children. *Behav Res Ther*. 1997;**35**(9):863–73.
173. Vanstrien T, Frijters JER, Bergers GPA, Defares PB. The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *Int J Eating Disord*. 1986;**5**(2):295–315.
174. Van Strien T. *Dutch Eating Behaviour Questionnaire (DEBQ): Manual*. London, Harcourt Assessment; 2002.
175. Van Strien T, Schippers GM, Cox WM. On the relationship between emotional and external eating behavior. *Addict Behav.* 1995;**20**(5):585–94.
176. Halberstadt J, van Strien T, de Vet E, Eekhout I, Braet C, Seidell JC. The association of eating styles with weight change after an intensive combined lifestyle intervention for children and adolescents with severe obesity. *Appetite*. 2016;**99**:82–90.
177. Kazdin AE, Holland L, Crowley M, Breton S. Barriers to treatment participation scale: evaluation and validation in the context of child outpatient treatment. *J Child Psychol Psychiatry*. 1997;**38**(8):1051–62.
178. Colonna-Pydyn C, Gjesfjeld CD, Greeno CG. The factor structure of the barriers to treatment participation scale (BTPS): implications for future barriers scale development. *Administration and Policy in Mental Health and Mental Health Services Research*. 2007;**34**(6):563–9.
179. Krauss A. Assessing the overall validity of randomised controlled trials. *International Studies in the Philosophy of Science*. 2021;**34**(3):159–82.
180. Berlin JA, Golub RM. Meta-analysis as evidence: building a better pyramid. *JAMA*. 2014;**312**(6):603–5.

181. Moher D, Hopewell S, Schulz KF, *et al*. CONSORT 2010 explanation and elaboration: updated guidelines for reporting parallel group randomised trials. *BMJ*. 2010;**340**:c869.
182. Öst L-G. *Att utvärdera psykoterapi och kritiskt granska psykoterapistudier.* Stockholm, Natur & Kultur; 2016.
183. O'Connor EA, Evans CV, Burda BU, Walsh ES, Eder M, Lozano P. Screening for obesity and intervention for weight management in children and adolescents: evidence report and systematic review for the US Preventive Services Task Force. *JAMA*. 2017;**317**(23):2427–44.
184. Moustgaard H, Clayton GL, Jones HE, *et al*. Impact of blinding on estimated treatment effects in randomised clinical trials: meta-epidemiological study. *BMJ*. 2020;**368**:l6802.
185. Kazdin AE. *Research Design in Clinical Psychology*, 4th ed. Boston, MA, Allyn & Bacon; 2003.
186. O'Shea O, McCormick R, Bradley JM, O'Neill B. Fidelity review: a scoping review of the methods used to evaluate treatment fidelity in behavioural change interventions. *Phys Ther Rev*. 2016;**21**(3–6):207–14.
187. Kendall PC, Frank HE. Implementing evidence-based treatment protocols: flexibility within fidelity. *Clin Psychol (New York)*. 2018;**25**(4):e12271.
188. Johns RG, Barkham M, Kellett S, Saxon D. A systematic review of therapist effects: a critical narrative update and refinement to Baldwin and Imel's (2013) review. *Clin Psychol Rev*. 2019;**67**:78–93.
189. Boutelle KN, Cafri G, Crow SJ. Parent predictors of child weight change in family based behavioral obesity treatment. *Obesity (Silver Spring)*. 2012;**20**(7):1539–43.
190. Wrotniak BH, Epstein LH, Paluch RA, Roemmich JN. Parent weight change as a predictor of child weight change in family-based behavioral obesity treatment. *Arch Pediatr Adolesc Med*. 2004;**158**(4):342–7.
191. Epstein LH, Valoski AM, Kalarchian MA, McCurley J. Do children lose and maintain weight easier than adults: a comparison of child and parent weight changes from six months to ten years. *Obes Res*. 1995;**3**(5):411–17.
192. Reilly JJ. Assessment of childhood obesity: national reference data or international approach? *Obes Res*. 2002;**10**(8):838–40.
193. Reilly JJ, Kelly J, Wilson DC. Accuracy of simple clinical and epidemiological definitions of childhood obesity: systematic review and evidence appraisal. *Obes Rev*. 2010;**11**(9):645–55.
194. Trabulsi J, Schoeller DA. Evaluation of dietary assessment instruments against doubly labeled water, a biomarker of habitual energy intake. *Am J Physiol Endocrinol Metab*. 2001;**281**(5):891–9.
195. Beets MW, Brazendale K, Weaver RG, Armstrong B. Rethinking behavioral approaches to compliment biological advances to understand the etiology, prevention, and treatment of childhood obesity. *Child Obes*. 2019;**15**(6):353–8.
196. Kazdin AE, Holland L, Crowley M. Family experience of barriers to treatment and premature termination from child therapy. *J Consult Clin Psychol*. 1997;**65**(3):453–63.

197. Kazdin AE, Wassell G. Barriers to treatment participation and therapeutic change among children referred for conduct disorder. *J Clin Child Psychol*. 1999;**28**(2):160–72.
198. Lydersen S. Multiple imputation of missing data. *Tidsskr Nor Laegeforen*. 2022;**142**(2). doi: 10.4045/tidsskr.21.0772.
199. Lister NB, Baur LA, Paxton SJ, Jebeile H. Contextualising eating disorder concerns for paediatric obesity treatment. *Curr Obes Rep*. 2021;**10**(3):322–31.
200. Jebeile H, Gow ML, Baur LA, Garnett SP, Paxton SJ, Lister NB. Treatment of obesity, with a dietary component, and eating disorder risk in children and adolescents: a systematic review with meta-analysis. *Obes Rev*. 2019;**20**(9):1287–98.
201. Jarrin DC, McGrath JJ, Drake CL. Beyond sleep duration: distinct sleep dimensions are associated with obesity in children and adolescents. *Int J Obes (Lond)*. 2013;**37**(4):552–8.
202. Olds TS, Maher CA, Matricciani L, Olds TS. Sleep duration or bedtime? Exploring the relationship between sleep habits and weight status and activity patterns. *Sleep*. 2011;**34**(10):1299–307.
203. Thivel D, Isacco L, Aucouturier J, *et al*. Bedtime and sleep timing but not sleep duration are associated with eating habits in primary school children. *J Dev Behav Pediatr*. 2015;**36**(3):158–65.
204. Martoni M, Carissimi A, Fabbri M, Filardi M, Tonetti L, Natale V. 24-h actigraphic monitoring of motor activity, sleeping and eating behaviors in underweight, normal weight, overweight and obese children. *Eat Weight Disord*. 2016;**21**(4):669–77.
205. Golley RK, Maher CA, Matricciani L, Olds TS. Sleep duration or bedtime? Exploring the association between sleep timing behaviour, diet and BMI in children and adolescents. *Int J Obes (Lond)*. 2013;**37**(4):546–51.
206. Schneider AC, Zhang D, Xiao Q. Adolescent sleep characteristics and body-mass index in the Family Life, Activity, Sun, Health, and Eating (FLASHE) Study. *Sci Rep*. 2020;**10**(1):13277.
207. Beebe DW, Lewin D, Zeller M, *et al*. Sleep in overweight adolescents: shorter sleep, poorer sleep quality, sleepiness, and sleep-disordered breathing. *J Pediatr Psychol*. 2007;**32**(1):69–79.
208. Su M-S, Zhang H-L, Cai X-H, *et al*. Obesity in children with different risk factors for obstructive sleep apnea: a community-based study. *Eur J Pediatr*. 2016;**175**(2):211–20.
209. Chou CH, Kang KT, Weng WC, Lee PL, Hsu WC. Central sleep apnea in obese children with sleep-disordered breathing. *Int J Obes (Lond)*. 2014;**38**(1):27–31.
210. Stice E, Marti CN, Durant S. Risk factors for onset of eating disorders: evidence of multiple risk pathways from an 8-year prospective study. *Behav Res Ther*. 2011;**49**(10):622–7.
211. Schaumberg K, Anderson DA, Anderson LM, Reilly EE, Gorrell S. Dietary restraint: what's the harm? A review of the relationship between dietary restraint, weight trajectory and the development of eating pathology. *Clin Obes*. 2016;**6**(2):89–100.

212. House ET, Gow ML, Lister NB, *et al*. Pediatric weight management, dietary restraint, dieting, and eating disorder risk: a systematic review. *Nutr Rev*. 2021;**79**(10):1114–33.
213. Gunnarsdottir T, Sigurdardottir ZG, Njardvik U, Olafsdottir AS, Bjarnason R. A randomized-controlled pilot study of Epstein's family-based behavioural treatment for childhood obesity in a clinical setting in Iceland. *Nordic Psychology*. 2011;**63**(1):6–19.
214. Croker H, Viner RM, Nicholls D, *et al*. Family-based behavioural treatment of childhood obesity in a UK National Health Service setting: randomized controlled trial. *Int J Obes (Lond)*. 2012;**36**(1):16–26.
215. Edwards C, Nicholls D, Croker H, Van Zyl S, Viner R, Wardle J. Family-based behavioural treatment of obesity: acceptability and effectiveness in the UK. *Eur J Clin Nutr*. 2006;**60**(5):587–92.
216. Riggs KR, Lozano P, Mohelnitzky A, Rudnick S, Richards J. An adaptation of family-based behavioral pediatric obesity treatment for a primary care setting: group health family wellness program pilot. *Perm J*. 2014;**18**(3):4–10.
217. Gunnarsdottir T, Njardvik U, Olafsdottir AS, Craighead L, Bjarnason R. Childhood obesity and co-morbid problems: effects of Epstein's family-based behavioural treatment in an Icelandic sample. *J Eval Clin Pract*. 2012;**18**(2):465–72.
218. Hayes JF, Fowler LA, Balantekin KN, *et al*. Children with severe obesity in family-based obesity treatment compared with other participants: conclusions depend on metrics. *Obesity (Silver Spring)*. 2021;**29**(2):393–401.
219. Reinehr T, Temmesfeld M, Kersting M, de Sousa G, Toschke AM. Four-year follow-up of children and adolescents participating in an obesity intervention program. *Int J Obes (Lond)*. 2007;**31**(7):1074–7.
220. Skodvin VA, Lekhal S, Kommedal KG, *et al*. Lifestyle intervention for children and adolescents with severe obesity: results after one year. *Tidsskr Nor Laegeforen*. 2020;**140**(9). doi: 10.4045/tidsskr.19.0682.
221. Knop C, Singer V, Uysal Y, Schaefer A, Wolters B, Reinehr T. Extremely obese children respond better than extremely obese adolescents to lifestyle interventions. *Pediatr Obes*. 2015;**10**(1):7–14.
222. Boutelle KN, Kang Sim DE, Rhee KE, Manzano M, Strong DR. Family-based treatment program contributors to child weight loss. *Int J Obes (Lond)*. 2021;**45**(1):77–83.
223. Fowler LA, Grammer AC, Ray MK, *et al*. Examining the interdependence of parent–child dyads: effects on weight loss and maintenance. *Pediatr Obes*. 2021;**16**(1):e12697.
224. Epstein LH, Paluch RA, Raynor HA. Sex differences in obese children and siblings in family-based obesity treatment. *Obes Res*. 2001;**9**(12):746–53.
225. Nowicka P, Pietrobelli A, Flodmark CE. Low-intensity family therapy intervention is useful in a clinical setting to treat obese and extremely obese children. *Int J Pediatr Obes*. 2007;**2**(4):211–17.
226. Eichen DM, Mestre ZL, Strong DR, Rhee KE, Boutelle KN. Defining and identifying predictors of rapid response to pediatric obesity treatment. *Pediatr Obes*. 2020;**15**(6):e12621.

227. Gross AC, Kaizer AM, Kelly AS, *et al*. Long and short of it: early response predicts longer-term outcomes in pediatric weight management. *Obesity (Silver Spring)*. 2019;**27**(2):272–9.
228. Braet C. Patient characteristics as predictors of weight loss after an obesity treatment for children. *Obesity (Silver Spring)*. 2006;**14**(1):148–55.
229. Kuhlemeier A, Jaki T, Jimenez EY, *et al*. Individual differences in the effects of the ACTION-PAC intervention: an application of personalized medicine in the prevention and treatment of obesity. *J Behav Med*. 2022;**45**(2):211–26.
230. Perez A, Holt N, Gokiert R, *et al*. Why don't families initiate treatment? A qualitative multicentre study investigating parents' reasons for declining paediatric weight management. *Paediatr Child Health*. 2015;**20**(4):179–84.
231. Tomiyama AJ, Carr D, Granberg EM, *et al*. How and why weight stigma drives the obesity 'epidemic' and harms health. *BMC Med*. 2018;**16**(1):123.
232. Phelan SM, Burgess DJ, Yeazel MW, Hellerstedt WL, Griffin JM, van Ryn M. Impact of weight bias and stigma on quality of care and outcomes for patients with obesity. *Obes Rev*. 2015;**16**(4):319–26.
233. Natland S, Tveiten S, Knutsen IR. Why should patients participate in research? *Tidsskr Nor Laegeforen*. 2017;**137**(3):210–12.
234. Epstein LH, Quattrin T. Ideas for next-generation treatments of adolescent obesity. *JAMA Pediatr*. 2020;**174**(6):527–8.

Appendix

Paper 1

Paper 2

Paper 3

I

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ORIGINAL RESEARCH

Pediatric OBESITY WILEY

Beyond sleep duration: Sleep timing as a risk factor for childhood obesity

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Summary

Background: Ample evidence attests to the relationship between short sleep duration, sleep problems and childhood obesity. However, few studies have examined the association between sleep timing and obesity in children.

Objectives: To investigate how sleep duration, problems and timing relate to obesity and obesogenic behaviours in children.

Methods: Eighty-five children (58.8% girls) with severe obesity and mean (SD) age of 12.1 (2.9) years, were matched by age and sex with peers with normal weight (n = 85,12.0 [2.8] years). Sleep and moderate-to-vigorous physical activity (MVPA) were measured via accelerometer for seven consecutive days. Children self-reported emotional eating on the Dutch eating behavior questionnaire. Parents reported children's screen time and sleep problems.

Results: Children with severe obesity had significantly later mean mid-sleep time, overall (36 minutes later, $P < .001$), on school nights (36 minutes later, $P < .001$) and weekend nights (39 minutes later, $P = .002$) compared to children with normal weight. Children with obesity had more sleep problems ($P = .030$), but no differences emerged in sleep duration or social jetlag. After adjusting for demographic factors, mid-sleep time was positively related to screen time ($P = .030$). Mid-sleep time and sleep duration were inversely related to time in MVPA ($Ps \leq .041$). There were no other significant associations between the sleep variables and the obesogenic behaviours.

Conclusions: Later sleep timing was related to obesogenic behaviours in children and may represent an obesity risk factor.

KEYWORDS

childhood obesity, sleep, sleep timing, social jetlag

Abbreviations: BMI, body mass index; DEBQ-child, the Dutch eating behavior questionnaire child version; IOTF, International Obesity Task Force; MVPA, moderate-to-vigorous physical activity; NSF, National Sleep Foundation; SD, standard deviation; SDS, standard deviation score.

1 | INTRODUCTION

Childhood obesity is a complex, multicausal health issue with major consequences for both the individual and society.[1] The detection of risk factors associated with weight gain is fundamental to offer adequate prevention and treatment. Short sleep, as well as sleep problems, have for some time been recognised as risk factors for obesity.[2] The majority of research on sleep and childhood obesity has focused on sleep duration.[3-5] Accordingly, an increasing number of studies have reported cross-sectional and longitudinal associations between short sleep duration and childhood obesity, suggesting sleep duration to be an independent and modifiable risk factor for the condition.[3-5] However, recent studies indicate that, in addition to sleep duration, other aspects of sleep needs to be taken into consideration to provide a more comprehensive understanding of how sleep contributes to the development and maintenance of obesity in children.[2,6,7]

Late sleep timing (i.e., when sleep occurs) and social jetlag (usually defined as the difference in mid-sleep time between weekdays and weekends[8]) have recently been suggested as unique contributors to obesity risk in school-aged children and adolescents, independent of sleep duration.[9-13] Late sleep timing has specifically been associated with increased weight, unhealthy eating habits, decreased physical activity levels and more screen time.[9-11,13-17] A small cross-sectional study in adolescents with obesity (n = 26) found that later sleep timing was associated with a higher caloric intake and more screen time independent of total sleep duration.[15] A recent study on treatment seeking adolescents with overweight and obesity found that later weekend bedtimes and greater social jetlag were significantly associated with severity of overweight.[6] Further, social jetlag has been associated with metabolic changes and obesogenic behaviours such as more screen time, less physical activity and emotional eating.[6,10,12,18-21]

Both biological and behavioural causes of weight gain seem to be associated with delayed and shifted sleep timing, but there is dearth of knowledge regarding our understanding of these associations.[16,22,23] It is possible that mistiming of sleep promotes circadian misalignment and, eventually, increases risk of developing obesity.[23,24] Therefore, a focus on alignment of sleep timing with underlying circadian rhythms could enhance paediatric obesity prevention and treatment.[23] Only a handful of previous studies have so far used objective sleep measures to investigate the sleep-obesity relationship.[14,17,20,25] Subjective sleep measures are associated with various biases,[26-28] therefore, more studies using objective sleep measures in a natural home environment, such as accelerometers, are needed. The present cross-sectional study adds to current research by using accelerometers (instead of self- or parent-reported measures) to assess sleep timing and social jetlag in school-aged children and adolescents.

The aims of this study were to investigate how children's sleep duration, sleep timing (including social jetlag) and sleep problems were linked to obesity and behavioural factors known to cause obesity in children. We hypothesised that in addition to sleep duration, delayed sleep timing and social jetlag were also independently related to behavioural factors that place school-aged children at a greater risk for developing obesity.

2 | METHODS

2.1 | Participants

In total, cross-sectional data from 170 children (median age 12.4 years, range 5.8-17.1 years) were collected between February 2014 and March 2018; 85 children (50 girls) with severe obesity and 85 children with normal weight, matched by age, sex and season of accelerometer measurement (April-September vs October-March). Participants with severe obesity were recruited from the Obesity Outpatient Clinic, Haukeland University Hospital, Bergen, Norway via referral from general practitioners. The criterion for clinic admission was a body mass index (BMI) above the International Obesity Task Force (IOTF) cut-off for severe obesity (≥ IOTF 35),[29] or above the cut-off for obesity (≥ IOTF 30) in the presence of weight-related comorbidity (e.g., psychosocial problems or emergence of cardio-metabolic risk factors). The group with normal weight (BMI ≤ IOTF 25) was recruited from randomly selected schools in the Bergen municipality. Stratified random sampling ensured that the comparison group were matched for age and sex.

Written informed consent was obtained from the parent(s) and from participating children above 12 years of age. The study was approved by the Regional Committee for Medical and Health Research Ethics in Western Norway (number 2013/1300) and was registered at http://clinicaltrials.gov (NCT02687516).

2.2 | Anthropometric measures

Weight status was assessed by the BMI (kg/m^2) calculated from measured height and weight and converted to BMI z-scores using the Norwegian growth references.[30] For the group of children with severe obesity, height and weight were measured by trained assessors at the Obesity Outpatient Clinic. Height was measured to the nearest 0.1 cm with a wall-mounted electronic stadiometer (Seca 264, Seca, Hamburg, Germany), and weight was measured to the nearest 0.1 kg using a digital scale (InBody 720, Biospace, Seoul, Korea). For the group of children with normal weight, measurements were collected by a trained assessor during regular school hours in their school nurse's office. Standing height was measured with a Harpenden portable stadiometer (Crosswell, UK) to the nearest 0.1 cm. Weight was measured on a calibrated Seca personal digital scale (Hamburg, Germany) to the nearest 0.1 kg. Participants in both groups were measured wearing light indoor clothing (excluding shoes and socks).

2.3 | Sleep measures

Sleep was assessed using the Actiwatch 2 (Philips Respironics, BEND, OR). Actiwatch 2 devices are wrist-worn accelerometers with a light sensor and an event marker and record all uni-axial movement over 0.05G.[31] Data was collected using 30-second epochs, each scored as either "wake" or "sleep" based on a medium sensitivity threshold. Medium sensitivity threshold has shown to yield the least biassed

estimates of wakefulness, total sleep time and wake after sleep onset in school-aged children.[31,32] The Actiwatch 2 was worn on the wrist of the non-dominant arm for seven consecutive days. Participants were instructed to press the event marker when switching off the light at night and when waking up in the morning. Actiwatch 2 is validated, both in clinical sleep laboratories and in natural home environments, and is commonly used for sleep research in children aged 3 to 18 years. [32-34]

Respironics Actiware software version 6.0.9 was used to calculate sleep statistics. The rest interval (time in bed) associated with the main sleep period in the 24-hours day was manually set according to a standardises scoring protocol.[31] To ensure inter-rater reliability, 30% of the actigraphy recordings were scored twice, by two independent observers, and compared in terms of total time in bed and total sleep time. The percentage of agreement among observers were 99.6% for total time in bed and 99.9% for total sleep time. After the rest interval was manually defined, the proprietary software automatically produced sleep statistics within the interval. The variables sleep onset time and wake up time (sleep offset) were used in our analyses.

2.4 | Sleep duration

When the rest interval was defined, the software automatically detected time spent asleep within the rest period. Average sleep duration for 7 days, average sleep duration on school nights (Sunday through Thursday nights) and weekend nights (Friday and Saturday nights) were used in the analyses. To be included, the participant had to have completed recordings of at least 5 days (out of 7 days) including at least three school nights and two weekend nights. We also categorised sleep duration for 7-day average, and school and weekend nights separately, based on recommendations from the National Sleep Foundation (NSF).[35] The NSF recommends 9 to 11 hours of sleep for children aged 6 to 13 and 8 to 10 hours of sleep for adolescents aged 14 to 17, respectively, while <7 hours is "not recommended" for either age group.[35]

2.5 | Mid-sleep time

Sleep timing was operationalised as mid-sleep time according to the formula: (sleep onset time + sleep offset time)/2. The mid-sleep time point of each individual child was calculated as a 7 day average as well as for school nights and weekend nights separately. For participants with six or five nights of recordings, the average of these nights was used. To be included in weekend night's analyses, two nights of recordings were needed. Further, sleep onset time and final wake up time are reported to provide additional information about sleep timing. Sleep during daytime was not assessed in the study.

2.6 | Social jetlag

Social jetlag quantifies the discrepancy between circadian time and social time[19] and was operationalised as the difference between the mean mid-sleep time point on school nights and the mean mid-sleep time point on weekend nights.

2.7 | Physical activity measures

Physical activity level was objectively assessed using data from the same device (Actiwatch 2) during daytime (8 AM-9 PM). Data were downloaded using Respironics Actiware software version 6.0.9 and transferred to Microsoft Excel 2016 for further processing with a tailored-made algorithm to divide the collected activity data into different activity levels based on previously used and validated cut-off values.[36] The cut-off values were: light intensity (160-523 counts/30 second-epochs), moderate intensity (524-811 counts/30 seconds-epochs) and vigorous intensity (>812 counts/30 second-epochs).[36] Physical activity level was operationalised as the percentage of time spent in moderate-to-vigorous physical activity (MVPA). Participants had to have at least 10 hours of wear time between 8 AM to 9 PM and at least 4 days of recorded data to be included in the physical activity analysis.[36] Sleep during this period was automatically coded as either non-wear time or sedentary behaviour (movement while sleeping) by the tailor-made algorithm.

2.8 | Emotional eating

2.8.1 | The Dutch eating behavior questionnaire child version

Emotional eating was assessed with the Dutch eating behavior questionnaire child version (DEBQ-child).[37] The DEBQ-child is a 33-item self-report questionnaire and consists of three sub-scales: emotional eating, external eating and restrained eating. All items are rated on a five-point scale ranging from never (1) to very often (5). For each subscale a mean score is calculated, with the following clinical cut-off values for emotional eating: >2.22 (boys) and >2.36 (girls). Participants were grouped according to whether they were below or above the clinical cut-off value. The DEBQ-child is increasingly used for research on children with obesity and has adequate internal consistency, test-retest reliability, factorial validity and dimensional stability for measuring disordered eating behaviours in children aged 7 to 17 years. [37,38] In the current study, the Cronbach alpha coefficient for the emotional eating subscale was .96, suggesting a high internal consistency of the scale in the current sample. The questionnaire was completed at the Obesity Outpatient Clinic by children with obesity and at the school nurse office by children with normal weight.

2.8.2 | Demographic information

Family structure, parental education levels, parental employment, child sleep problems and child daily screen time were measured with a parental questionnaire. Family structure was evaluated with the

following questions: "Are both parents living together" and "Do the child live together with siblings". Parental education level was categorised as either low (≤3 years of high school), intermediate (≤4 years of college/university) or high >4 years of college/university). Sleep problems were identified with the following question: "Has the child in any period experienced sleep problems", with the following response categories: "never", "before starting elementary school but not now", "after starting elementary school but not now», and "current sleep problems". Participants were grouped according to whether they reported current sleep problems or not. Habitual screen time was rated on a scale from 0 (no screen time) to 5 (>4 hours of screen time). The questionnaires were completed at the Obesity Outpatient Clinic by the parents of children with obesity and sent by mail to the parents of children with normal weight.

2.9 | Statistical analyses

Data were analysed with IBM SPSS version 25 (IBM Corp., Armonk, NY). Descriptive statistics of continuous variables are given by the mean and SD, and of categorical variables by the frequency and percentage. Demographic variables in the normal weight and obesity groups were compared with independent sample *t* tests and chi-square tests of independence. Sleep parameters were compared between groups with independent sample *t* tests. In addition to group mean differences on the measurement scale, we also calculated the effect sizes (Cohen's *d*). An effect size of 0.2 is considered small, 0.5 medium and 0.8 large,[39] respectively. We used hierarchical multiple regression analyses to regress screen time and MVPA on mid-sleep time, sleep duration, social jetlag and sleep problems adjusted for group (normal weight or severe obesity), age, sex, living situation (operationalised as parents living together or not) and parental education level. Group, age, sex, living situation and parental education level were entered in Step 1 of the analysis, while the focal predictors were entered in Step 2. Parental education was entered as two dummy variables with the low education group as reference category. Finally, we included interaction terms between the group variable and the four focal predictors in a final, third step. The continuous predictors (mid-sleep time, sleep duration and social jetlag) were all mean centred prior to computing the interaction terms.

A logistic regression analysis was used to examine the association between sleep duration, mid-sleep time, social jetlag and sleep problems with emotional eating, adjusted for group, age and sex.

2.10 | Power estimates

The required sample size was determined with G*Power, version 3.1.3.[40] An α of .05 (two-tailed) and power (1–β) of .80 was used to determine statistical significance. For the group comparison, a minimum of 51 individuals in both groups of children (with obesity/normal weight) is required to detect a medium effect size (Cohen's d = 0.50) with a significance level of .05, a power of 80%. The present sample size of 85 children per group allows to detect effect sizes of 0.4 onwards.

2.11 | Missing data

Because of some missing data, the number of children with useable data observations ranged from 124-170 in the different analyses.

Of the 170 participants, 168 (98.8%) provided valid accelerometer data on sleep and were included in the analyses. Of these, 154 provided valid recordings for seven consecutive days and, 14 for 6 or 5 days. Of the 168 eligible participants, two did not have sufficient actigraphy data for weekend nights, reducing the sample to 166 (97.6%) for these analyses. Further, 16 (9.4%) participants in total, 12 from the group of children with obesity and four of the normal weight peers, were excluded from the analyses involving MVPA because they had less than 10 hours of wear time between 8 AM to 9 PM and/or less than 4 days of recording. All parents of children with obesity and 65 out of 85 (76.5%) of parents of children with normal weight completed the questionnaire on demographic data, sleep problems and screen time. Seventy-seven out of 85 (90.6%) participants in the group of children with obesity and all participants in the group of peers with normal weight completed the DEBQ-child questionnaires on emotional eating.

3 | RESULTS

3.1 | Demographical and clinical characteristics of the sample

The groups of children with obesity and their peers with normal weight were balanced in terms of age, sex and ethnicity (Table 1). However, children with normal weight more often lived with both parents and with siblings, and their parents were more often employed and higher educated. Overall, 92.3% of the children did not meet the NSF age-appropriate sleep recommendation, while 13.1% had an average sleep duration classified as not recommended (<7 hours). For school nights the percentage not meeting the recommendations where 91.7%, with 22.6% sleeping less than 7 hours. For weekend nights the percent not meeting the recommendations where 69.9%, with 9.0% sleeping less than 7 hours.

3.2 | Sleep behaviour: Comparison of children with severe obesity and normal weight

Children with severe obesity had a significantly later mid-sleep time, both overall (on average 36 minutes later, P < .001) as well as on school nights (36 minutes later, P < .001) and weekend nights (39 minutes later, P = .002) separately (Table 2). In addition,

TABLE 1 Characteristics of the study population according to weight group

	Obesity	Normal weight	*P* value**
Total (N)	85	85	
Age (mean, SD)	12.1 (2.9)	12.0 (2.8)	.86
Range	5.9-17.1	5.8-16.4	
Sex: girls (%)	50 (58.8%)	50 (58.8%)	
BMI *z*-score mean (SD)	2.91 (0.45)	−0.24 (0.24)	**<.001**
Parent reported data			
Number with survey data[a]	85	65	
Mother born in Norway (%)	89.4%	97.0%	.07
Father born in Norway (%)	84.5%	93.9%	.07
Parents living together (%)	56.5%	89.4%	**<.001**
Living with siblings (%)	69.4%	95.5%	**<.001**
Father, full time work (%)	67.8%	90.9%	**.02**
Father, part time work (%)	1.2%	0.0%	**.02**
Mother, full time work (%)	47.6%	78.7%	**.02**
Mother, part time work (%)	19.0%	9.1%	**.02**
Father, completed education (%)			
Elementary school	20.5%	0.0%	
High school	41.0%	45.4%	
College/University ≤4 years	19.2%	29.7%	
College University >4 years	11.5%	23.4%	**.001**
Mother, completed education (%)			
Elementary school	11.9%	3.0%	
High school	46.4%	28.8%	
College/University ≤4 years	20.2%	34.8%	
College University >4 years	20.2%	33.3%	**.001**

Abbreviations: BMI, body mass index; SD, standard deviation.
[a]The percentages reported below this line are based on the number of returned questionnaires.
***P* values from a chi-square test for categorical data, and a *t* test for continuous data; Statistically significant p values (*P* < .05) are marked in bold.

children with obesity had more often sleep problems (28.0% vs 20.9% in normal weight children, *P* = .03). However, sleep duration and social jetlag did not significantly differ between groups (Table 2).

3.3 | Association between sleep behaviour and obesogenic behaviours

In the total sample, mid-sleep time point and sleep duration significantly correlated with screen time and MVPA (*P*s ≤ .01). Social jetlag was significantly correlated with screen time (*P* = .02), but not with MVPA (*P* = .08). Sleep problems were not significantly correlated with either screen time (*P* = .07) or MVPA (*P* = .30). The results are summarised in Table 3.

In the hierarchical model, age, sex, group, living situation and parental education level entered at Step 1 combined explained approximately 21% of the total variability in screen time use (R^2 = .212, F[8, 127] = 4.27, *P* < .001). Of our focal predictors entered in Step 2, only mid-sleep time was significantly related to screen time use (β = .26, *P* = .03). Combined, adding mid-sleep time, sleep duration, social jetlag and sleep problems in Step 2 resulted in a statistically non-significant increase in explained variability of about 4% (ΔR^2 = .036, F[4, 123] = 1.48, *P* = .21). Adding the interaction terms in Step 3 also resulted in a non-significant increase in explained variability (ΔR^2 = .042, F[4, 119] = 1.73, *P* = .15). Further, none of the interaction terms reached statistical significance (all *P*s > .05). The results are summarized in Table 4.

Age, sex, group, living situation and parental education level combined explained about 54% of the total variability in MVPA (R^2 = .542, F[8, 115] = 17.04, *P* < .001). Both mid-sleep time (β = −.23, *P* = .015) and sleep duration (β = −.19, *P* = .041) were statistically significant predictors when entered at Step 2. Combined, the four focal predictors explained an additional 4% of the variability in MVPA (ΔR^2 = .037, F[4, 111] = 2.39, *P* = .05). Adding the interaction terms in Step 3 revealed a statistically significant interaction between group and social jetlag (β = −.28, *P* = .012) (Table 4). This interaction shows that the effect of social jetlag is

TABLE 2 Comparison of children with severe obesity and normal weight on sleep outcomes

Sleep outcome	Children with obesity Mean (SD)	Children with normal weight Mean (SD)	Group difference Mean	*P* value	Effect size (d)
7 day mean					
Mid-sleep time	3:37:50 (1:14:07)	3:02:08 (0:47:41)	0:35:42	**<.001**	0.57
Sleep onset time	23:23:17 (1:33:10)	22:44:22 (1:04:29)	0:38:55	**.002**	0.49
Wake-up time	7:53:39 (1:03:41)	7:20:28 (0:38:39)	0:33:10	**<.001**	0.63
School days/nights					
Mid-sleep time	3:22:21 (1:10:22)	2:46:46 (0:45:30)	0:35:35	**<.001**	0.60
Sleep onset time	23:03:27 (1:29:20)	22:25:04 (1:04:12)	0:38:22	**.002**	0.49
Wake-up time	7:40:50 (0:58:00)	7:10:00 (0:39:19)	0:30:50	**<.001**	0.62
Weekends					
Mid-sleep time	4:18:07 (1:33:40)	3:39:12 (0:59:20)	0:38:54	**.002**	0.50
Sleep onset time	00:12:14 (1:55:58)	23:31:37 (1:18:04)	0:40:36	.009	0.41
Wake up time	8:27:36 (1:31:21)	7:47:03 (0:53:43)	0:40:33	**.001**	0.54
Sleep duration, 7 day mean	7:48:31 (0:46:42)	7:52:02 (0:41:50)	−0:03:30	.608	−0.08
Sleep duration, school nights	7:39:19 (0:58:10)	7:43:01 (0:51:52)	−0:03:41	.664	−0.07
Sleep duration, weekend nights	8:09:25 (1:00:25)	8:13:36 (0:50:40)	−0:04:11	.628	−0.07
Social jetlag	00:54:32 (00:52:48)	00:52:12 (00:37:12)	0:02:24	.720	0.04

Note: All sleep outcomes are reported as hours: minutes: seconds. The *t* tests were evaluated using Bonferroni adjusted alpha levels of 0.004 per test (.05/13). Statistically significant *P* values in bold.
Abbreviations: d, Cohen's d; SD, standard deviation.

TABLE 3 Correlations between sleep behaviour and screen time and MVPA

	Screen time	MVPA	Mid-sleep time	Sleep duration	Social jetlag
Mid-sleep time	0.458***	−0.536***			
Sleep duration	−0.267**	0.293***	−0.529***		
Social jetlag	0.170*	−0.113	0.292***	−0.114	
Sleep problems[a]	0.121	−0.051	0.246**	−0.181*	−0.009

Abbreviation: MVPA, moderate-to-vigorous physical activity.
[a]Correlations involving the dichotomous sleep-problems variable are point-biserial correlations, otherwise the table shows the Pearson product-moment correlations.
*$P < .05$.
**$P < .01$.
***$P < .001$.

opposite for the two groups. For the normal weight group, the effect of social jetlag on MVPA is positive, while for the obese group the effect seems to be negative. Follow-up analyses of these two simple slopes showed that effect was statistically significant for the normal weight group ($b = .28$, $P = .039$) but not for the obese group ($b = -.15$, $P = .151$).

Logistic regression was used to predict participants' odds of scoring above the clinical cut-off on emotional eating. The complete model containing all predictors was not statistically significant, χ^2 (5, $N = 170$) = 8.242, $P = .31$, indicating that the model as a whole was not able to distinguish well between respondents scoring below and above the clinical cut-off. None of the individual independent variables contributed significantly to the predictive ability of the model.

4 | DISCUSSION

The current study found that children with obesity had significantly later sleep timing, both overall and on school nights and weekend nights separately, compared to peers with normal weight. However, sleep duration and social jetlag were not significantly different between the two groups. To our knowledge, the present study is the first to compare sleep timing in a group of obesity treatment-seeking children and adolescents with normal weight peers using objective sleep measures.

Although the amount of sleep occurring throughout the night was similar among children with obesity and children with normal weight, we found differences in the timing of when sleep occurs. There is

limited research examining the association between sleep timing and BMI in school-aged children and adolescents,[9-11,13-15,17,25] but our findings are still in accordance with the results from the majority of previous studies.[9-11,13,17] A large cross-sectional study from Australia with 2200 participants aged 9 to 16 years[11] found that the odds of having obesity were 1.5 times higher for adolescents with late bed/rise time than for adolescents with early bed/rise time, independent of sleep duration.

Similarly, another cross-sectional study[10] in children and adolescents aged 8 to 17 years found that later sleep and wake times were associated with greater adiposity, regardless of sleep duration. The fact that the present and previous studies[9-11] report an association

TABLE 4 Hierarchical multiple regression predicting screen time and MVPA

	Screen time (n = 136)			MVPA (n = 124)		
	B	R^2	ΔR^2	B	R^2	ΔR^2
Step 1		.212***			.542***	
Group	.16			−.39***		
Age	.42***			−.58***		
Sex	−.03			−.19**		
Mothers' education						
Intermediate	.06			−.00		
Higher	.17			.03		
Fathers' education						
Intermediate	.01			−.02		
Higher	−.21*			.13		
Parents live together	.02			.09		
Step 2		.248***	.036		.579***	.037
Group	.10			−.34***		
Age	.29*			−.57***		
Sex	−.02			−.19**		
Mothers' education						
Intermediate	.06			.01		
Higher	.15			.05		
Fathers' education						
Intermediate	.02			−.04		
Higher	−.18			.11		
Parents live together	.02			.09		
Mid-sleep time[a]	.26*			−.23*		
Sleep duration[a]	.08			−.19*		
Social jetlag	.05			.03		
Sleep problems	−.03			.04		
Step 3		.290***	.042		.617***	.038*
Group	.03			−.38***		
Age	.18			−.57***		
Sex	−.02			−.18**		
Mothers' education						
Intermediate	.06			.02		
Higher	.17			.06		
Fathers' education						
Intermediate	.01			−.03		
Higher	−.19			.11		
Parents live together	.03			.07		
Mid-sleep time	.32			−.32*		

(Continues)

TABLE 4 (Continued)

	Screen time (n = 136)			MVPA (n = 124)		
	B	R^2	ΔR^2	B	R^2	ΔR^2
Sleep duration	−.12			−.30*		
Social jetlag	−.16			.23*		
Sleep problems	−.22			−.04		
Group X mid-sleep time	.03			.17		
Group X sleep duration	.18			20		
Group X social jetlag	.26			−.28*		
Group X sleep problems	.26			.11		

Note: Group is coded normal weight = 0 and obese = 1. Sex is coded 0 = male and 1 = female. Parents living together is coded 0 = no and 1 = yes. Lower education is the base category.

Abbreviation: MVPA, moderate-to-vigorous physical activity.

[a]Seven day averages are used for sleep duration and mid-sleep time.

*P < .05.

**P < .01.

***P < .001.

between sleep timing and increased BMI independent of sleep duration is interesting. Sleep onset time in the present study was in total 36 minutes later for the group of children with obesity. A logical assumption, based on the growing body of evidence demonstrating an association between short sleep duration and increased BMI in children[3,4] and the fact that the school day starts early (between 08.00 and 08.30 AM), is that the group of children with obesity, due to having later sleep onset time, also would have shorter sleep duration. However, in this study we observed a compensatory delay in wake time on school days for the group of children with obesity, resulting in a sleep duration approximately the same in the two groups. The late wake-up time in the group of children with obesity makes it difficult for many in this group to reach school timely in the morning. This finding is supported by a recent meta-analysis[41] that found that the odds of being absent from school was 54% higher among children with obesity compared to normal weight peers. In the present study it is observed that children with obesity more rarely lived with both parents. It is known that treatment-seeking children with obesity have a high degree of psychological comorbidity and often unstable family situations with increased psychological and psychosocial stress,[42] which may influence their wake-up time. Further, it is probable that a late wake-up time might result in omitting breakfast, which is associated with weight gain in children.[43]

The later sleep and wake up time in the group of children with obesity compared to normal weight peers were consistent throughout the week. Further, both groups have approximately one-hour later mid-sleep time on weekends compared to weekdays, leading to no difference in social jetlag between the groups. This finding is inconsistent with previous research on social jetlag and BMI.[6,12,19,20] One study reported that social jetlag was associated with higher BMI z-scores and waist-to-height ratios in adolescents aged 14 to 17 years.[20] Similarly, another study in children aged 8 to 10 years[12] found that social jetlag was independently associated with body fat, fat mass, fat mass index, waist-to-height ratios and BMI. Further, a study from treatment-seeking adolescents with overweight and obesity found that greater bedtime shift from weekdays to weekends were significantly associated with severity of overweight.[6] Interestingly, a large epidemiological study with approximately 65 000 participants aged >10 years found that social jetlag did not explain significant proportions of the variance in weight in the normal BMI group, but that it was positively associated with weight increase in the overweight group.[19] The lack of group difference in social jetlag in the present study could be explained by previous research showing that social jetlag is prevalent in adolescents across the whole weight spectrum since as many as 88% of adolescents report going to bed later on weekend nights than school nights, and 44% of high school students report a two or more hour difference in bedtimes on free nights and school nights.[44]

Additionally, it is a concern that on school nights 22.6% of the children fall below the scientific consensus-based cut off <7 hours of sleep,[35] in terms of the many well documented adverse physical and mental health outcomes associated with insufficient sleep in children and adolescents aged 5 to 17 years.[45] The trend of sleeping less than recommended during childhood years is apparent in several countries.[46,47] However, only one recent study has provided data on the prevalence and stability of objectively measured insufficient sleep (<7 hours) throughout childhood.[48] That study[48] found that at age 12 years 14% of the children slept less than 7 hours on average, in accordance, the present study found that 13.1% of the children slept less than 7 hours on average.

Further, an aim of the present study was to examine whether sleep duration and sleep timing could explain variation in obesogenic behaviours among children and adolescents across the weight spectrum. One interesting study in this context is a recent cross-sectional investigation of the association between sleep timing with diet and physical activity in children between 9 to 11 years, using objectively measured sleep and physical activity where no significant difference in BMI or BMI z-score between children with late and early sleep

timing was found.[14] However, the researchers found that children with early sleep timing had healthier eating patterns, and spent more time in MVPA than children with late sleep timing, suggesting that sleep timing has less to do with BMI and more to do with behaviours that subsequently and over time may impact BMI.[14] It is also possible that having a late sleep timing results in being awake when the internal circadian timing system favours sleep. A discrepancy between actual sleep timing and circadian rhythms may result in alterations in metabolic processes, that in the long run may have a negative effect on weight status.[23,24]

In the current study, screen time was associated with higher BMI SDS and age, as well as later sleep timing. Sleep duration did not explain a significant proportion of the variance in screen time. Additionally, MVPA was found to be inversely associated with BMI z-scores, age, sleep duration and sleep timing. Previous research relating sleep duration and timing with time in MVPA reports mixed results. Cross-sectional studies have contradicted each other with positive,[11,14] negative [25] and no significant findings.[2] The majority of studies on this topic find that both shorter sleep duration and later sleep timing are associated with more screen time.[2,49] However, the present results mirror those of a previous study by Olds et al[11] who found that children with later bedtime/later wake-time engaged in less MVPA and in more screen time compared to a group of early bedtime/early wake-time children, despite having similar sleep duration.[11] It is of interest that for the normal weight group in the current study, we found that more social jetlag was associated with increased MVPA, however, for the obese group this relationship was negative.

Finally, no associations between emotional eating and the sleep measures were found in the present study. Previous research links both sleep duration and sleep timing to poorer diets (higher energy intake and poor eating habits) in children.[9,13-16,50,51] The few studies that have investigated sleep timing in relation to diet in children have consistently reported that later sleep timing is associated with poorer diets independently of sleep duration.[9,13-16] Further, it is interesting that later bedtime seems to be associated with delayed time of the first meal of the day (which implies skipping breakfast) independent of sleep duration.[52] To our knowledge, the present study is the first that specifically has investigated emotional eating in relationship to sleep timing.

The present study has several strengths and limitations that should be noted. Assessing sleep with objective measure using seven consecutive 24-hours recordings was a major asset, given that previous studies mainly have resorted to self-reported or parent-reported bed/wake-time, or sleep timing preferences (sleep chronotype) as opposed to observed sleep timing behaviours. A limitation of the present study is the lack of sleep diaries as a support to the scoring of the actigraphy recordings. However, 30% of the actigraphy recordings were scored by two independent observers to ensure inter-rater reliability, and a standardised scoring protocol was used. The low percentage of missing data, for self- and objectively measured, strengthen the findings from the present study. However, the cross-sectional nature puts restrictions on inferences about directionality and causality, and there is always a possibility for residual confounding in observational studies. Further, wrist-worn accelerometers have a lower accuracy in estimating physical activity when compared to hip worn accelerometers, which is a possible limitation of the present study. Nevertheless, both locations have been found acceptable for use in children and adolescents, but placement on the wrist has shown to have better compliance.[53] The 10 hours of wake time between 8 AM to 9 PM required for inclusion in the analyses of time in MVPA could be a possible limitation as some children may engage in physical activity outside this time frame, but is still in accordance with previously used scoring protocols for objectively measured physical activity.[36] Inclusion of the 16 participants with late wake-up time who were excluded due to this requirement may have provided an even stronger association between time in MVPA and sleep timing. In addition, it is possible that emotional eating is an insufficient measure related to sleep timing as diet quality, calorie intake and eating patterns may be more relevant in this context. Finally, the parent-reported data on sleep problems and screen time are a limitation of the present study because of potential parent - child discrepancy in perception of sleep problems and actual screen time. Further, the question about sleep problems does not differentiate between types of sleep disturbances and there is no separation of school (mostly used for educational purposes) and leisure time (mostly used for entertainment purposes) screen use.

In conclusion, later sleep timing was related to obesogenic behaviours in children and adolescents and may represent a risk factor for obesity independent of sleep duration. These findings highlight the importance of including other aspect of sleep, in addition to duration, when conducting research and clinical work related to childhood obesity. Future longitudinal and intervention studies, with objective measurers of sleep, are warranted to better understand the association between sleep timing and childhood obesity and more studies should be devoted to understanding the underlying mechanisms of the sleep-obesity link.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHORS' CONTRIBUTION

H. F. Skjåkødegård, Y. S. Danielsen, M. Roelants, S. Pallesen, R. P. K. Conlon, D. E. Wilfley and P. B. Juliusson conceived and designed the study. H. F. Skjåkødegård, Y. S. Danielsen and P. B. Juliusson collected the data. H. F. Skjåkødegård, Y. S. Danielsen, B. Frisk and S. W. Hystad performed the statistical analyses. H. F. Skjåkødegård wrote the paper in consultation with Y. S. Danielsen, B. Frisk, S. W. Hystad, S. Pallesen, R. P. K. Conlon, P. B. Juliusson. All authors discussed the results and contributed to the final manuscript.

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REFERENCES

1. Kumar S, Kelly AS. Review of childhood obesity: from epidemiology, etiology, and comorbidities to clinical assessment and treatment. *Mayo Clin Proc*. 2017;92(2):251-265.
2. Krietsch KN, Chardon ML, Beebe DW, Janicke DM. Sleep and weight-related factors in youth: a systematic review of recent studies. *Sleep Med Rev*. 2019;46:87-96.
3. Miller MA, Kruisbrink M, Wallace J, Ji C, Cappuccio FP. Sleep duration and incidence of obesity in infants, children, and adolescents: a systematic review and meta-analysis of prospective studies. *Sleep*. 2018; 41(4):zsy018. https://doi.org/10.1093/sleep/zsy018.
4. Magee L, Hale L. Longitudinal associations between sleep duration and subsequent weight gain: a systematic review. *Sleep Med Rev*. 2012;16(3):231-241.
5. Sluggett L, Wagner SL, Harris RL. Sleep duration and obesity in children and adolescents. *Can J Diabetes*. 2019;43(2):146-152.
6. Hayes JF, Balantekin KN, Altman M, Wilfley DE, Taylor CB, Williams J. Sleep patterns and quality are associated with severity of obesity and weight-related behaviors in adolescents with overweight and obesity. *Child Obes*. 2018;14(1):11-17.
7. Morrissey B, Taveras E, Allender S, Strugnell C. Sleep and obesity among children: a systematic review of multiple sleep dimensions. *Pediatr Obes*. 2020;15(4):e12619.
8. Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. *Chronobiol Int*. 2006;23(1–2): 497-509.
9. Golley RK, Maher CA, Matricciani L, Olds TS. Sleep duration or bedtime? Exploring the association between sleep timing behaviour, diet and BMI in children and adolescents. *Int J Obes (Lond)*. 2013;37(4): 546-551.
10. Jarrin DC, McGrath JJ, Drake CL. Beyond sleep duration: distinct sleep dimensions are associated with obesity in children and adolescents. *Int J Obes (Lond)*. 2013;37(4):552-558.
11. Olds TS, Maher CA, Matricciani L. Sleep duration or bedtime? Exploring the relationship between sleep habits and weight status and activity patterns. *Sleep*. 2011;34(10):1299-1307.
12. Stoner L, Castro N, Signal L, et al. Sleep and adiposity in preadolescent children: the importance of social jetlag. *Child Obes*. 2018;14(3): 158-164.
13. Thivel D, Isacco L, Aucouturier J, et al. Bedtime and sleep timing but not sleep duration are associated with eating habits in primary school children. *J Dev Behav Pediatr*. 2015;36(3):158-165.
14. Harrex HAL, Skeaff SA, Black KE, et al. Sleep timing is associated with diet and physical activity levels in 9-11-year-old children from Dunedin, New Zealand: the PEDALS study. *J Sleep Res*. 2018;27(4):e12634.
15. Adamo KBWS, Belanger K, Chaput JP. Later bedtime is associated with greater daily energy intake and screen time in obese adolescents independent of sleep duration. *J Sleep Disord Ther*. 2013;2:126.
16. Thellman KE, Dmitrieva J, Miller A, Harsh JR, LeBourgeois MK. Sleep timing is associated with self-reported dietary patterns in 9- to 15-year-olds. *Sleep Health*. 2017;3(4):269-275.
17. Martoni M, Carissimi A, Fabbri M, Filardi M, Tonetti L, Natale V. 24-h actigraphic monitoring of motor activity, sleeping and eating behaviors in underweight, normal weight, overweight and obese children. *Eat Weight Disord*. 2016;21(4):669-677.
18. Kjeldsen JS, Hjorth MF, Andersen R, et al. Short sleep duration and large variability in sleep duration are independently associated with dietary risk factors for obesity in Danish school children. *Int J Obes (Lond)*. 2014;38(1):32-39.
19. Roenneberg T, Allebrandt KV, Merrow M, Vetter C. Social jetlag and obesity. *Curr Biol*. 2012;22(10):939-943.
20. Malone SK, Zemel B, Compher C, et al. Social jet lag, chronotype and body mass index in 14-17-year-old adolescents. *Chronobiol Int*. 2016; 33:1255-1266.
21. Chung K-F, Kan KK-K, Yeung W-F. Sleep duration, sleep–wake schedule regularity, and body weight in Hong Kong Chinese adolescents. *Bio Rhythm Res*. 2013;44(2):169-179.
22. Miller AL, Lumeng JC, LeBourgeois MK. Sleep patterns and obesity in childhood. *Curr Opin Endocrinol Diabetes Obes*. 2015;22(1):41-47.
23. Hart CN, Jelalian E, Raynor HA. Behavioral and social routines and biological rhythms in prevention and treatment of pediatric obesity. *Am Psychol*. 2020;75(2):152-162.
24. McHill AW, Wright KP Jr. Role of sleep and circadian disruption on energy expenditure and in metabolic predisposition to human obesity and metabolic disease. *Obes Rev*. 2017;18(Suppl 1):15-24.
25. McNeil J, Tremblay MS, Leduc G, et al. Objectively-measured sleep and its association with adiposity and physical activity in a sample of Canadian children. *J Sleep Res*. 2015;24(2):131-139.
26. Iwasaki M, Iwata S, Iemura A, et al. Utility of subjective sleep assessment tools for healthy preschool children: a comparative study between sleep logs, questionnaires, and actigraphy. *J Epidemiol*. 2010; 20(2):143-149.
27. Kushnir J, Sadeh A. Correspondence between reported and actigraphic sleep measures in preschool children: the role of a clinical context. *J Clin Sleep Med*. 2013;9(11):1147-1151.
28. Girschik J, Fritschi L, Heyworth J, Waters F. Validation of self-reported sleep against actigraphy. *J Epidemiol*. 2012;22(5):462-468.
29. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;7 (4):284-294.
30. Juliusson PB, Roelants M, Nordal E, et al. Growth references for 0-19 year-old Norwegian children for length/height, weight, body mass index and head circumference. *Ann Hum Biol*. 2013;40(3):220-227.
31. Chow CM, Wong SN, Shin M, et al. Defining the rest interval associated with the main sleep period in actigraph scoring. *Nat Sci Sleep*. 2016;8:321-328.
32. Meltzer LJ, Walsh CM, Traylor J, Westin AML. Direct comparison of two new actigraphs and polysomnography in children and adolescents. *Sleep*. 2012;35(1):159-166.
33. Meltzer LJ, Montgomery-Downs HE, Insana SP, Walsh CM. Use of actigraphy for assessment in pediatric sleep research. *Sleep Med Rev*. 2012;16(5):463-475.
34. Meltzer LJ, Wong P, Biggs SN, et al. Validation of Actigraphy in middle childhood. *Sleep*. 2016;39(6):1219-1224.
35. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's updated sleep duration recommendations: final report. *Sleep Health*. 2015;1(4):233-243.
36. Ekblom O, Nyberg G, Bak EE, Ekelund U, Marcus C. Validity and comparability of a wrist-worn accelerometer in children. *J Phys Act Health*. 2012;9(3):389-393.
37. Braet C, Beyers W, Goossens L, Verbeken S, Moens E. Subtyping children and adolescents who are overweight based on eating pathology and psychopathology. *Eur Eat Disord Rev*. 2012;20(4):279-286.
38. Halberstadt J, van Strien T, de Vet E, Eekhout I, Braet C, Seidell JC. The association of eating styles with weight change after an intensive combined lifestyle intervention for children and adolescents with severe obesity. *Appetite*. 2016;99:82-90.
39. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. 2nd ed. Hillsdale: NJ: Lawrence Erlbaum; 1988.
40. Faul F, Erdfelder E, Buchner A, Lang AG. Statistical power analyses using G*power 3.1: tests for correlation and regression analyses. *Behav Res Methods*. 2009;41(4):1149-1160.
41. An R, Yan H, Shi X, Yang Y. Childhood obesity and school absenteeism: a systematic review and meta-analysis. *Obes Rev*. 2017;18(12): 1412-1424.
42. Rankin J, Matthews L, Cobley S, et al. Psychological consequences of childhood obesity: psychiatric comorbidity and prevention. *Adolesc Health Med Ther*. 2016;7:125-146.

43. Blondin SA, Anzman-Frasca S, Djang HC, Economos CD. Breakfast consumption and adiposity among children and adolescents: an updated review of the literature. *Pediatr Obes*. 2016;11(5):333-348.
44. National Sleep Foundation Sleep in America Poll. https://www.sleepfoundation.org/2006
45. Chaput JP, Gray CE, Poitras VJ, et al. Systematic review of the relationships between sleep duration and health indicators in school-aged children and youth. *Appl Physiol Nutr Metab*. 2016;41(6 Suppl 3):S266-S282.
46. Buxton OM, Chang AM, Spilsbury JC, Bos T, Emsellem H, Knutson KL. Sleep in the modern family: protective family routines for child and adolescent sleep. *Sleep Health*. 2015;1(1):15-27.
47. Hysing M, Pallesen S, Stormark KM, Lundervold AJ, Sivertsen B. Sleep patterns and insomnia among adolescents: a population-based study. *J Sleep Res*. 2013;22(5):549-556.
48. Ranum BM, Wichstrom L, Pallesen S, Steinsbekk S. Prevalence and stability of insufficient sleep measured by actigraphy: a prospective community study. *Pediatr Res*. 2020;88:110–116.
49. Ogunleye AA, Voss C, Sandercock GR. Delayed bedtime due to screen time in schoolchildren: importance of area deprivation. *Pediatr Int*. 2015;57(1):137-142.
50. Hjorth MF, Quist JS, Andersen R, et al. Change in sleep duration and proposed dietary risk factors for obesity in Danish school children. *Pediatr Obes*. 2014;9(6):e156-e159.
51. Saidi O, Rochette E, Bovet M, Merlin E, Duché P. Acute intense exercise improves sleep and decreases next morning consumption of energy-dense food in adolescent girls with obesity and evening chronotype. *Pediatr Obes*. 2020;15(6):e12613.
52. Spaeth AM, Hawley NL, Raynor HA, et al. Sleep, energy balance, and meal timing in school-aged children. *Sleep Med*. 2019;60:139-144.
53. Fairclough SJ, Noonan R, Rowlands AV, Van Hees V, Knowles Z, Boddy LM. Wear compliance and activity in children wearing wrist- and hip-mounted accelerometers. *Med Sci Sports Exerc*. 2016;48(2):245-253.

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ORIGINAL RESEARCH ARTICLE

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Family-based treatment of children with severe obesity in a public healthcare setting: Results from a randomized controlled trial

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Summary

To compare the effectiveness of family-based behavioural social facilitation treatment (FBSFT) versus treatment as usual (TAU) in children with severe obesity. Parallel-design, nonblinded, randomized controlled trial conducted at a Norwegian obesity outpatient clinic. Children aged 6–18 years referred to the clinic between 2014 and 2018 were invited to participate. Participants were randomly allocated using sequentially numbered, opaqued, sealed envelopes. FBSFT ($n = 59$) entailed 17 sessions of structured cognitive behavioural treatment, TAU ($n = 55$) entailed standard lifestyle counselling sessions every third month for 1 year. Primary outcomes included changes in body mass index standard deviation score (BMI SDS) and percentage above the International Obesity Task Force cut-off for overweight (%IOTF-25). Secondary outcomes included changes in sleep, physical activity, and eating behaviour. From pre- to posttreatment there was a statistically significant difference in change in both BMI SDS (0.19 units, 95% confidence interval [CI]: 0.10–0.28, $p < .001$) and %IOTF-25 (5.48%, 95%CI: 2.74–8.22, $p < .001$) between FBSFT and TAU groups. FBSFT participants achieved significant reductions in mean BMI SDS (0.16 units, (95%CI: −0.22 to −0.10, $p < .001$) and %IOTF-25 (6.53%, 95% CI: −8.45 to −4.60, $p < .001$), whereas in TAU nonsignificant changes were observed in BMI SDS (0.03 units, 95% CI: −0.03 to 0.09, $p = .30$) and %IOTF-25 (−1.04%, 95% CI: −2.99 to −0.90, $p = .29$). More FBSFT participants (31.5%) had clinically meaningful BMI SDS reductions of ≥0.25 from pre- to posttreatment than in TAU (13.0%, $p = .021$). Regarding secondary outcomes, only changes in sleep timing differed significantly between groups. FBSFT improved weight-related outcomes compared to TAU.

Yngvild S. Danielsen and Petur B. Juliusson are joint senior authors.

KEYWORDS
adolescent, behavioural treatment, children, family-based treatment, paediatric obesity, randomized controlled trial

What is already known about this subject

- Family-based behavioural treatment is recommended as an evidence-based treatment for childhood obesity.
- Family-based behavioural treatment delivered in research clinics, has been shown to yield clinically significant weight loss in children with obesity.

What this study adds

- Delivered at an obesity outpatient clinic, family-based behavioural social facilitation treatment (FBSFT) improved weight-related outcomes significantly more than treatment as usual (TAU) among children (ages 6–18 years) with severe obesity.
- Investigation of individual treatment response showed that significantly more children receiving FBSFT achieved a clinically meaningful body mass index standard deviation score reduction of ≥0.25 compared to children receiving TAU.
- The beneficial changes in weight outcomes exhibited in FBSFT compared to TAU were not explained by differences in sleep, physical activity, or eating behaviour.

1 | INTRODUCTION

Paediatric obesity is one of the major global health challenges of the 21st century. Effective treatment options are urgently needed, given the increasing prevalence of paediatric obesity, the risk of adverse health consequences, and the fact that obesity-related risk factors track into adulthood.[1,2]

To date, various treatment options have been tested, including interventions focusing on lifestyle modification, as well as pharmacological therapy and bariatric surgery,[1,3,4] which are summarized in numerous reviews and meta-analyses.[3–8] Despite this rapidly growing body of treatment research, studies have yielded similar findings over the last decades,[1,9] and lifestyle modification has remained the preferred treatment strategy for children and adolescents.[1,10,11]

Treatment programmes targeting multiple lifestyle behaviours whilst applying behavioural techniques in a family-based approach have shown the most promise and are considered best practise in obesity treatment for children aged 6–17 years.[10,12,13] However, evidence is mostly derived from efficacy trials in research clinics, with strict control of internal validity and participant selection.[14,15] An important next step is to conduct effectiveness trials focusing on treatment delivery in public healthcare settings with less stringent participant selection criteria.[9,11] Evidence for the effectiveness of such treatment programmes that can be extrapolated to different healthcare settings is sparse,[16,17] and sought by national health authorities.[18] The family-based behavioural treatment of childhood obesity (FABO) study aimed to address this need. The study enrolled children who met the criteria for admission to a tertiary care obesity clinic within the public healthcare service in Norway,[19] and compared family-based behavioural social facilitation treatment (FBSFT) with treatment as usual (TAU) (comprising lifestyle intervention, including diet and physical activity). This study design provided the opportunity to investigate the FBSFT approach in a growing, but often overlooked, patient population of children with the most severe form of obesity[20] (International Obesity Task Force [IOTF] body mass index [BMI] ≥35 or ≥30[21] with comorbidity).

Moreover, emerging data suggest that obesity risk is influenced by sleep patterns. Several aspects of sleep, including duration and timing, have been identified as contributors to the development and maintenance of childhood obesity.[22] However, family-based lifestyle interventions usually target diet and physical activity, and less commonly sleep.[13,23] A recent review found that only 20% of 119 family-based intervention studies included a sleep component, usually in children aged 2–5 years,[23] with most studies assessing sleep using parent-reported data. As sleep problems may impact the effectiveness of treatment interventions, evaluating sleep patterns, along with changes in other lifestyle behaviours (e.g., eating behaviour, physical activity), will inform our understanding of key treatment components that are critical to target in family-based obesity interventions.

The aim of the present study was to compare the effectiveness of FBSFT to TAU in severe childhood obesity treatment delivered in a public healthcare setting. Outcome measures included BMI-related metrics, sleep measures, physical activity, and eating behaviour. We hypothesized that FBSFT would yield greater improvements in BMI-related metrics as well as sleep and eating behaviour, compared to TAU, with similar improvements in physical activity due to a comparable focus on this component in both treatment programmes.

2 | METHODS

2.1 | Study design

The FABO study is a parallel-design, nonblinded, randomized controlled trial (RCT), conducted at the Obesity Outpatient Clinic at

Haukeland University Hospital, Bergen, Norway. All children (aged 6–18 years) referred by their general practitioner to the clinic between February 2014 and October 2018 were invited to participate. Written informed consent was obtained after an initial clinic assessment. Participating families were randomized to either FBSFT (Arm A) or TAU (Arm B). Randomization was in 1:1 ratio, and sequentially numbered, opaque sealed envelopes were used to conceal the randomization sequence. Figure 1 depicts the study design, including the primary measurement time points at baseline and after FBSFT (Arm A) or 1 year of TAU (Arm B).

The study was approved by the Regional Committee for Medical and Health Research Ethics, Western Norway (number 2013/1300) and was registered on ClinicalTrials.gov (NCT02687516).

2.2 | Participants

One hundred fourteen children and adolescents (mean age 12.6 years; minimum–maximum: 5.9–17.7 years) participated, with 59 participants in Arm A and 55 in Arm B. Inclusion criteria were BMI above the IOTF cut-off for severe obesity (≥35 kg/m^2) or for obesity (≥30 kg/m^2)[21] in the presence of weight-related comorbidities. The family-based approach to this intervention required that both the child and at least one parent agreed to actively participate. Parental weight status was not assessed prior to inclusion. Families were excluded if either the child or one or both parents experienced severe somatic or psychiatric illness affecting weight or adherence to the treatment programme, or if the child was participating in other obesity treatment programmes.[19]

2.3 | Description of treatments

FBSFT focuses on promoting healthy lifestyle behaviours and attitudes using a combination of behavioural and cognitive techniques. It builds on family-based behavioural treatment (FBT) for paediatric obesity, which is the most documented approach in childhood obesity treatment[10] and has been shown to yield clinically significant weight loss in children with obesity.[17] FBSFT not only incorporates all of the features of FBT, which focuses on the individual, as well as on the family/home environment,[10] but also extends the focus across socio-ecological contexts, thus supporting and sustaining changes in health behaviours.[19] This extension includes evaluation and engagement of supports across the peer network and community levels such as school settings. FBSFT also includes elements of interpersonal therapy for eating disorders aimed at tackling emotions and interpersonal conflicts that affect eating habits. Health behaviours in terms of diet, physical activity, sedentary behaviour, and sleep are targeted in both children and their parents applying the Traffic Light Eating Plan and activity programme.[15] Pretreatment measures of the mentioned lifestyle behaviours were used to form the basis for the planning of healthy changes. Progress was monitored from session-to-session using specific weight goals and relevant lifestyle behaviours adjusted thereafter. The goal for children aged ≤10 years was stable weight maintenance throughout the programme, whereas for children aged ≥10 years session-to-session weight reduction of 250 g was used as a reference point.

Traditionally, FBT is implemented in a mixed (group + individual) format,[15] whilst FBSFT was delivered in 17 fortnightly individual family sessions (mean treatment duration 178 ± 47 days). Families had to attend 15 of 17 sessions to be considered completers. The majority of children were accompanied by one parent to each session. Parental participation was considered important for children of all ages, but individual adjustments related to age were implemented, providing adolescents with greater responsibility for healthy changes compared to younger participants. Families met with the same healthcare worker from the multidisciplinary team at the obesity clinic for all sessions. The team consisted of a paediatrician, nutritionist, physiotherapist, nurse and psychologist, and all team members were trained in FBSFT prior to treatment delivery. The intensive treatment phase, including session-specific topics and application of behavioural and cognitive techniques, was delivered as previously described in the study protocol.[19]

Families assigned to TAU (Arm B), a lifestyle intervention targeting the child, were provided with a personalized plan for changing specific lifestyle behaviours and were advised to participate in monthly counselling sessions with their local healthcare nurse. TAU was delivered over the course of 12 months[19] (mean treatment duration 374 ± 41 days) and included quarterly assessments, progress evaluation, and goal revision in clinic. Of the participants, 87% attended all the appointed assessments at the obesity clinic.

2.4 | Anthropometric measures

Height and weight were measured by trained assessors in clinic. The assessors were informed about study participation, but not treatment assignment. Height was measured to the nearest 0.1 cm with an electronic wall-mounted seca 264 stadiometer (Seca), and weight was measured to the nearest 0.1 kg using a digital InBody720 scale (Biospace). Measurements were taken with participants wearing light indoor clothing only (without socks and shoes).[19] Weight status was assessed using two metrics converted from the BMI (kg/m^2): BMI standard deviation score (SDS) and percentage above the IOTF cut-off for overweight[21] (%IOTF-25). The BMI SDS was calculated using the Norwegian growth reference,[24] whereas %IOTF-25 is the percentage above the IOTF threshold for overweight based on a child's age and sex, calculated as 100 × (BMI/IOTF-25).[25] A cut-off point of ≤−0.25 BMI SDS was used to define a clinically relevant change from pre- to posttreatment in participants from each group.[26,27]

2.5 | Sleep measures

Sleep was objectively measured using an Actiwatch 2 (Philips Respironics). Actiwatch 2 devices are wrist-worn accelerometers with a light sensor and an event marker, which record all uniaxial movement

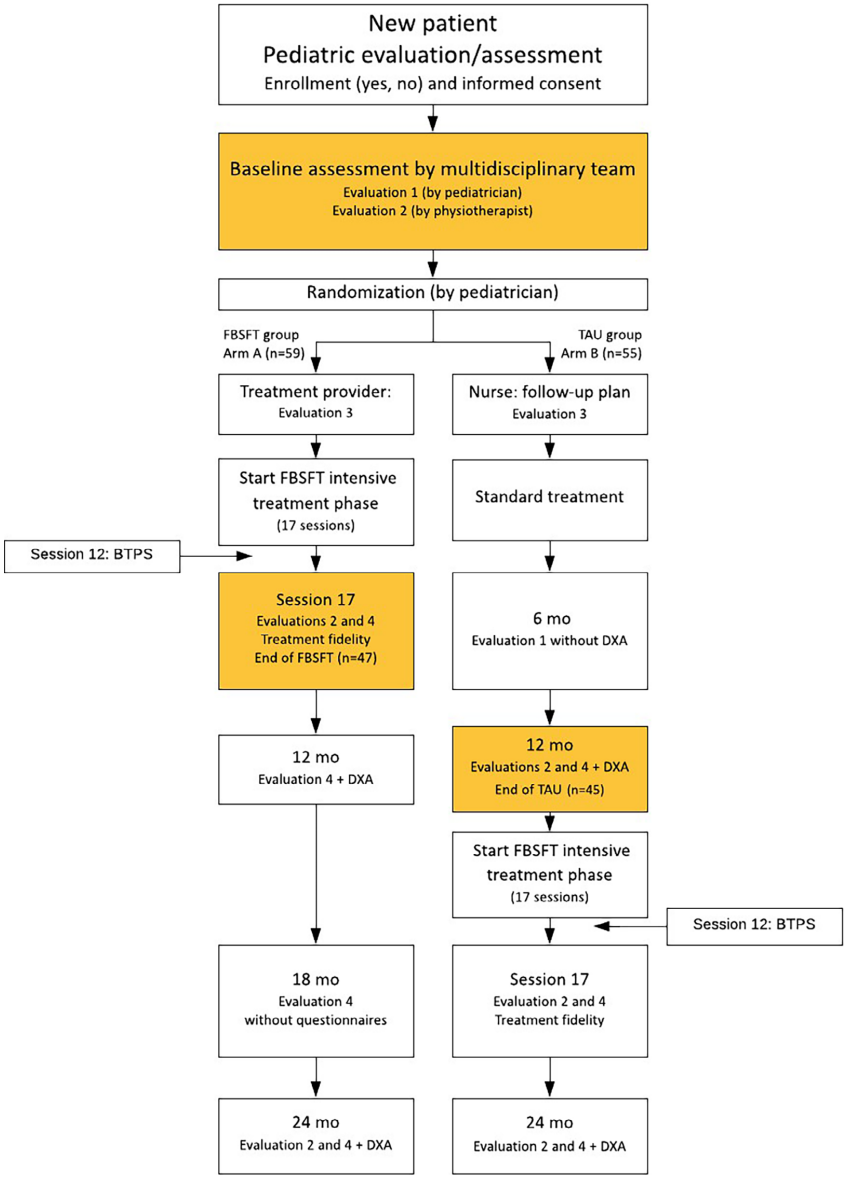

FIGURE 1 Flow chart showing the FABO study design (modified from study protocol previously described).[19] Coloured boxes represent study measurement time points. Evaluation 1: DXA, BIA, BP, height, weight, waistC. Evaluation 2: actigraphy, sleep and physical activity. Evaluation 3: questionnaire assessment (DEBQ, YEDE-Q, YSR, CBCL, CDI, SPPC). Evaluation 4: BIA, BP, height, weight, waistC, questionnaire assessment (as for Evaluation 3). BTPS: applied after 12 FBSFT sessions and in dropout population. Abbreviations: BIA, bioelectrical impedance analysis; BP, blood pressure; BTPS, Barriers to Treatment Participation Scale; CBCL, Child Behaviour Checklist; CDI, Children's Depression Inventory; DEBQ, Dutch Eating Behaviour Questionnaire; DXA, dual-energy X-ray absorptiometry; FBSFT, family-based behavioural social facilitation treatment; mo, months; SPPC, Self-Perception Profile for Children; TAU, treatment as usual; waistC, waist circumference; YEDE-Q, Youth Eating Disorder Examination Questionnaire; YSR, The Youth Self-Report

over 0.05G.[28] Data were collected using 30-s epochs, and a medium sensitivity threshold was used to score the epochs as either "wake" or "sleep." Medium sensitivity thresholds have been shown to yield the least biased estimates of wakefulness, total sleep time, and wake after sleep onset in school-aged children.[28] The device was worn on the wrist of the nondominant arm for 7 consecutive days pre- and posttreatment in both groups. Participants were instructed to press the event marker when switching off the light at night and on waking up in the morning. Actiwatch 2 has been validated, both in clinical sleep laboratories and in the natural home environment and is commonly used in sleep research in children aged 3–18 years.[29,30]

Sleep statistics were calculated using Respironics Actiware software, version 6.0.9. The rest interval (time in bed) associated with the main sleep period in the 24-h day was manually set, according to a standardized scoring protocol.[28] Furthermore, sleep time within this interval was automatically detected by a standard default algorithm in the proprietary software.

2.5.1 | Sleep duration

The mean sleep duration over 7 consecutive days was used in the analyses. For inclusion, participants completed recordings of at least 5 (out of 7) days, including at least three school nights and two weekend nights. At baseline 105 of 114 participant presented with valid sleep recordings, 96 with 7 nights, 7 with 6 nights and 2 with 5 nights. Posttreatment the numbers where 79 of 114 participants in total, 66 with 7 nights, 11 with 6 nights and 2 with 5 nights.

2.5.2 | Sleep timing

Sleep timing (i.e., when sleep occurs) was calculated as a 7-day mean of the mid-sleep time, i.e., the midpoint between sleep onset time and wake-up time: (sleep onset time + sleep offset time)/2. For participants with only five or six nights of recordings, the mean of these nights was used. Furthermore, sleep onset and final wake-up times were reported for additional information about sleep timing. Seen together, these parameters give valuable information about sleep hygiene. Sleep during daytime was not assessed in the study.

2.6 | Physical activity measures

Daytime physical activity (between 8:00 AM and 9:00 PM) was objectively assessed using data from the same device (Actiwatch 2). Wrist-worn accelerometers have been validated for measuring physical activity in children,[31] and their use shown to maximize compliance.[32]

Data were downloaded using Respironics Actiware software, version 6.0.9, and exported into Microsoft Excel 2016 for further processing using a tailor-made software. The collected activity data were categorized into different intensity levels based on previously used and validated cut-off values: light (160–523 counts/30-s epochs), moderate (524–811 counts/30-s epochs), and vigorous intensity (>812/30-s epochs).[33] To be included in the analysis, participants needed ≥10 h of wear time each day and ≥4 days of recorded data.[33] Physical activity level was calculated as the percentage of time spent on moderate-to-vigorous physical activity (MVPA).

2.7 | Eating behaviour

2.7.1 | Dutch Eating Behaviour Questionnaire

The Dutch Eating Behaviour Questionnaire (DEBQ) is a measure of disturbed eating patterns in children and adolescents.[34] Two versions of the questionnaire were used. An age-adapted 20-item version[35] for children aged <10 years and a full 33-item questionnaire for children aged ≥10 years.[34] Both versions comprised three subscales: restrained, external, and emotional eating.[34,35] All 33 items on the full version were rated on a 5-point Likert scale, ranging from "never" (1) to "very often" (5), and the age-adapted 20-item version included a reduced 3-point response scale: "no" (1), "sometimes" (3) and "yes" (5). Mean scores were calculated for each subscale. Both versions were merged for analysis by converting responses on the 20-item version as follows: $1 = 1$, $2 = 3$ and $3 = 5$. The questionnaires were self-reported and completed pre- and posttreatment in both groups. Both the full 33-item and the reduced 20-item DEBQ versions have been used increasingly in paediatric obesity research and shown to have adequate internal consistency, test–retest reliability, factorial validity, and dimensional stability for measuring disordered eating behaviour in children aged 7–17 years.[20,34,35] In the current study, the Cronbach's α coefficient for the 33-item version was .76 for restrained, .87 for external and .96 for emotional eating at baseline. For the 20-item version, the Cronbach's α coefficient was .79 for restrained, .73 for external and .85 for emotional eating at baseline. These indicate acceptable (>0.7) to excellent (>0.9) internal consistency for the three subscales in the current sample.

2.8 | Statistical analyses

Data were analysed with IBM SPSS version 26 (IBM Corp.) and Stata version 17 (StataCorp LLC., 2021). Descriptive statistics are expressed as the mean and standard deviation (*SD*) for continuous variables, and as frequency and percentage for categorical variables. Baseline differences between FBSFT and TAU participants, and between children who completed the intervention and those who did not, were tested using *t*-tests and χ^2 tests of independence, with the significance level set as 0.05.

Linear mixed models were used to estimate and compare changes from pre- to posttreatment under the two treatment conditions. The mixed models included the treatment condition (FBSFT vs. TAU), time (baseline and posttreatment), and a treatment-by-time interaction, and were fitted with an unstructured residual covariance structure for all primary and secondary outcomes. Models were checked for

heteroscedasticity and normality of residuals. Differences in means and 95% confidence intervals were computed from the fixed effect model parameter estimates. This analytical approach deviates from the original protocol,[19] but our main goal was to determine whether the change in scores over time differs according to the treatment condition, and this is more straightforward to test and interpret with a treatment-by-time interaction within a mixed models framework.[36] This decision was taken prior to the analysis.

Following the principle of intention to treat, all participants were included in the analyses, irrespective of missing data at any measurement points. Mixed models are not based on balanced data assumption and use all available data on each participant, thus accounting for missing data on a response variable. Under the 'missing at random' (MAR) assumption, these models provide unbiased estimates.[37] Intervention (within-group) effect sizes were estimated on complete data using Glass's Δ, with pretreatment *SD* as denominator. An effect size is commonly interpreted as small (0.2), moderate (0.5) and large (0.8).[38]

2.8.1 | Sample size and statistical power

Power analysis was performed prior to the FABO trial based on two treatment groups (FBSFT and TAU) and three measurement points (pretreatment, and 6- and 12-month posttreatment). For an α level of .05, a power of 80%, and a correlation of .5 between measurement points, a sample size of 28–164 subjects would allow to detect moderate (Cohen's $f = 0.25$) to small (Cohen's $f = 0.10$)[38]effects of treatment on the primary outcome over time. Based on number of referrals to the obesity clinic, a total of 120 participants were estimated as a realistic sample size to recruit during the study period, and large enough to detect small to moderate differences in the primary

TABLE 1 Anthropometric characteristics at baseline by treatment group

	FBSFT group			TAU group			
Variables	***N***	**Mean ± *SD***	**Min–Max**	***N***	**Mean ± *SD***	**Min–Max**	***p* Value***
Age (years)	59	12.6 ± 3.3	5.9–17.7	55	12.6 ± 2.8	6.9–17.4	.975
Weight (kg)	59	80.8 ± 28.9	29.4–165.7	55	82.3 ± 22.4	40.6–114.7	.758
Height (cm)	59	157.1 ± 16.7	112.9–186.4	55	159.3 ± 14.3	130.4–183.7	.457
Height SDS[a]	59	0.6 ± 1.2	−2.5 to 4.2	55	0.7 ± 0.8	−1.3-4.5	.757
BMI (kg/m^2)	59	31.9 ± 5.4	22.2–50.0	55	31.7 ± 4.3	23.4–38.9	.826
BMI SDS[a]	59	3.0 ± 0.5	2.2–4.9	55	2.9 ± 0.4	2.1–3.8	.761
%IOTF-25[b]	59	146.2 ± 14.1	124.1–204.3	55	144.9 ± 11.3	121.6–171.6	.598

Abbreviations: BMI, body mass index; FBSFT, family-based behavioural social facilitation treatment; Max, maximum; Min, minimum; *SD*, standard deviation; SDS, standard deviation score; TAU, treatment as usual; %IOTF-25, percentage above the International Obesity Task Force cut-off for overweight.
[a]Calculated using the Norwegian growth reference.
[b]Calculated using the International Obesity Task Force criterion for overweight.
*p Value obtained by independent t-test.

TABLE 2 Behavioural characteristics at baseline by treatment group

	FBSFT group	TAU group	Group difference	
Behavioural outcome	Mean ± *SD*	Mean ± *SD*	Mean	*p* Value
Sleep duration, 7 days' mean	7:39 (0:57)	7:42 (0:45)	−0: 03	.802
Mid-sleep time, 7 days' mean	3:32 (1:06)	3:49 (1:23)	−0: 17	.255
Sleep onset time, 7 days' mean	23:23 (1:31)	23:36 (1:36)	−0: 13	.480
Wake-up time, 7 days' mean	7:43 (0:56)	8:02 (1:17)	−0: 19	.142
Percentage time in MVPA	9.87 (5.62)	8.61 (5.20)	1.26	.245
DEBQ scores				
Emotional eating	1.74 (0.90)	1.78 (0.89)	−0.03	.828
External eating	3.15 (0.88)	2.94 (0.92)	0.21	.243
Restrained eating	2.65 (0.86)	2.74 (0.67)	−0.09	.544

Note: All sleep outcomes are reported as hours:minutes. Mid-sleep time is the midpoint between time of sleep onset and wake-up time. DEBQ scores are rated on a 5-point scale, ranging from 1 to 5.
Abbreviations: DEBQ, Dutch Eating Behaviour Questionnaire; FBSFT, family-based behavioural social facilitation treatment; MVPA, moderate-to-vigorous physical activity; *SD*, standard deviation; TAU, treatment as usual.

outcome between groups. The sample sizes were calculated with G*Power, version 3.1.3.[39]

2.8.2 | Missing data

Anthropometric data at baseline were available from all participants, including dropouts. Anthropometric data posttreatment were available for all completers. Approximately 90% of participants provided accelerometer and questionnaire data at baseline, whilst the percent decreased to 68% posttreatment.

3 | RESULTS

Participants' baseline characteristics in the FBSFT and TAU groups are presented in Tables 1 and 2. There were no significant group differences at baseline in weight, height, BMI-related metrics, sleep behaviour, physical activity and eating behaviour ($p > .05$). The percentage of females in FBSFT and TAU groups was 61.0% and 56.4%, respectively ($p = .618$). Twenty-two participants (19.3%) dropped out of the study: 12 (20.3%) from the FBSFT group and 10 (18.2%) from the TAU group ($p = .771$). There were no significant differences in age, sex, and BMI SDS between 'treatment completers' and 'dropouts' ($p > .05$), although the latter group had higher %IOTF-25 at baseline (150.5% vs. 144.4%, $p = .045$) (Table S1).

3.1 | Primary outcome (change in BMI-related metrics)

The treatment-by-time interaction indicated statistically significant differences in changes from baseline to posttreatment in both BMI SDS (0.19 units, $p < .001$) and %IOTF-25 (5.48%, $p < .001$) between FBSFT and TAU (Table 3). Furthermore, BMI SDS and %IOTF-25 decreased significantly in the FBSFT group from baseline to posttreatment (0.16 units, $p < .001$ and 6.53%, $p < .001$), whilst changes in the TAU group were not statistically significant for BMI SDS or %IOTF-25 (0.03 units, $p = .30$ and −1.04%, $p = .29$) (Table 3).

Individual changes in BMI SDS of all participants in both groups are shown in Figure 2. A clinically meaningful reduction in BMI SDS of ≥0.25 was observed in 31.5% ($n = 17$) of the participants in FBSFT and 13% ($n = 7$) in TAU, a significant difference ($\chi^2(1) = 5.357$, $p = .021$). Intervention effects are presented in Table 4.

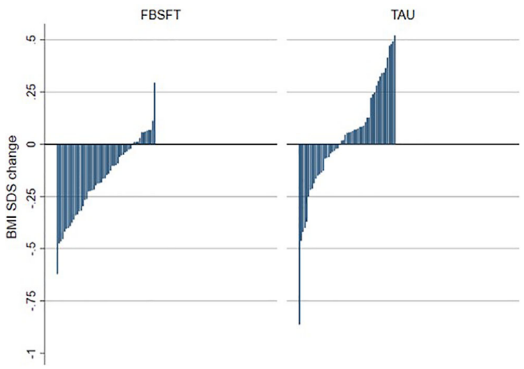

FIGURE 2 Individual variation in BMI SDS change from pretreatment to posttreatment for family-based behavioural social facilitation treatment (FBSFT) and treatment as usual (TAU) groups. Each bar represents the change in a single patient

TABLE 3 Changes in outcome variables by treatment group and difference in outcome among the treatment groups from baseline to posttreatment

Outcome	Treatment group Mean change from baseline to posttreatment (95% CI)			Time	Mean difference between groups (95% CI)	Group × time
	FBSFT	TAU	All participants	p		p
BMI SDS	−0.16 (−0.22; −0.10)	0.03 (−0.03; 0.09)	−0.06 (−0.11; −0.02)	.004	0.19 (0.10; 0.28)	<.001
%IOTF-25	−6.53 (−8.45; −4.60)	−1.04 (−2.99; 0.90)	−3.79 (−5.16; −2.42)	<.001	5.48 (2.74; 8.22)	<.001
Sleep duration	1.53 (−12.2; 15.26)	−17.95 (−33.73; −2.17)	−8.21 (−18.67; 2.25)	.124	−19.48 (−40.40; 1.44)	.068
Mid-sleep time	15.43 (−0.55; 31.415)	−10.90 (−29.73; 7.93)	2.27 (−10.08; 14.62)	.719	−26.33 (−51.03; −1.63)	.037
%MVPA	−1.32 (−2.43; −0.21)	−0.98 (−2.28; 0.32)	−1.15 (−2.01; −0.30)	.008	0.34 (−1.37; 2.05)	.696
Restrained eating	0.21 (−0.07; 0.50)	−0.12 (−0.44; 0.20)	0.05 (−0.17; 0.26)	.660	−0.33 (−0.76; 0.10)	.131
External eating	−0.13 (−0.33; 0.07)	−0.09 (−0.31; 0.14)	−0.11 (−0.26; 0.04)	.158	0.04 (−0.26; 0.34)	.782
Emotional eating	0.01 (−0.24; 0.26)	−0.04 (−0.32; 0.24)	−0.01 (−0.20; 0.17)	.876	−0.05 (−0.43; 0.32)	.776

Note: All sleep outcomes are reported in minutes. Mid-sleep time is the midpoint between time of sleep onset and wake-up time. Restrained eating, external eating, emotional eating are the three subscales of the Dutch Eating Behaviour Questionnaire.
Abbreviations: BMI SDS, BMI standard deviation score; FBSFT, family-based behavioural social facilitation treatment; TAU, treatment as usual; 95% CI, 95% confidence interval; %IOTF-25, percentage above the IOTF cut-off for overweight; %MVPA, percentage of time spent on moderate-to-vigorous physical activity.

TABLE 4 Intervention (within-group) effect sizes

	Within-group effect size[a]
Variables	Glass Δ
BMI SDS[b]	
FBSFT ($n = 59$)	0.30
TAU ($n = 55$)	0.03
%IOTF-25[c]	
FBSFT ($n = 59$)	0.46
TAU ($n = 55$)	0.16
Sleep duration, 7 days mean	
FBSFT ($n = 59$)	0.04
TAU ($n = 55$)	0.32
Mid-sleep time, 7 days mean	
FBSFT ($n = 59$)	0.24
TAU ($n = 55$)	0.09
Emotional eating	
FBSFT ($n = 59$)	0.05
TAU ($n = 55$)	0.10
External eating	
FBSFT ($n = 59$)	0.15
TAU ($n = 55$)	0.05
Restrained eating	
FBSFT ($n = 59$)	0.16
TAU ($n = 55$)	0.25
Percent time in MVPA	
FBSFT ($n = 59$)	0.25
TAU ($n = 55$)	0.14

Note: Mid-sleep time is the midpoint between time for sleep onset and wake-up time.
Abbreviations: BMI, body mass index; FBSFT, family-based behavioural social facilitation treatment; MVPA, moderate-to-vigorous physical activity; SDS, standard deviation score; TAU, treatment as usual; %IOTF-25, percentage above the International Obesity Task Force cut-off for overweight.
[a]Glass's Δ was calculated by dividing the mean of the difference scores by the pretreatment standard deviation.
[b]Calculated using the Norwegian growth reference.
[c]Calculated using the International Obesity Task Force criterion for overweight.

3.2 | Secondary outcomes

There was a significant difference in changes in sleep timing (operationalized as mid-sleep time) from pre- to posttreatment (−26.3 min, $p = .037$) between the FBSFT and TAU groups (Table 3). The mid-sleep time increased for FBSFT and decreased for TAU from pre- to posttreatment, but neither was statistically significant by itself (FBSFT: $p = .058$, TAU: $p = .257$). For the percentage of time spent in MVPA we observed an overall significant reduction from pre- to posttreatment ($p = .008$), but no differences between treatment groups.

There were no significant differences between or within groups for any of the other secondary outcome measures.

4 | DISCUSSION

This RCT demonstrated that in a public healthcare setting, children with severe obesity receiving FBSFT reduced their BMI SDS and % IOTF-25 significantly more during the treatment period than children enrolled in TAU. In addition, a larger proportion of FBSFT participants showed a significant reduction in BMI SDS of ≥0.25 from pre- to post-treatment. Changes in eating, sleep and physical activity behaviour were minimal, with only changes in sleep timing showing a significant difference between the two groups.

The between group difference in change in BMI SDS (0.19 units) is of similar magnitude as the findings from two recent Cochrane reviews on diet, physical activity and behavioural interventions.[5,7] These reviews reported a significant pooled treatment effect in favour of the interventions compared to control conditions of −0.06 BMI SDS units in 6–11-year-old children[7] and of −0.13 units in 12–17-year-old adolescents.[5] Interestingly, our study produces better results than majority of studies on behaviour-based interventions with similar contact hours[8] and follow-up period.[5]

Narrowing the comparison to studies on standardized FBT, the pooled result of eight pioneer studies from Epstein et al. shows a BMI SDS change in FBT of −1.20 units at 6 months,[17] which is considerably larger than in our study. However, all eight studies were efficacy studies conducted in research clinics.[17] In contrast to these tightly controlled settings our study aimed to assess the response to FBSFT in a regular outpatient clinic where lower treatment effectiveness was expected. The few RCTs on FBT carried out in effectiveness studies up to date have not been able to reproduce the effects reported by Epstein's group.[40,41] Furthermore, the high mean BMI SDS score at baseline may have influenced the effectiveness. A recent study on FBT compared the BMI SDS change after 4 months of treatment for children with severe obesity and nonsevere obesity.[42] Children with severe obesity had a mean reduction of −0.20 units, a result aligning with ours. For children with less severe obesity the reduction were of −0.37 units.[42] Other reasons for the more modest BMI SDS reduction observed in our sample can be related to the experience level of the treatment staff and the modification from mixed (group + individual) format to an individual family format.[15]

In the present study, significantly more participants in the FBSFT group achieved a BMI SDS reduction of ≥0.25 (31.5% in FBSFT group compared to 13% in TAU group). Individual treatment response is an important outcome measure, in addition to mean changes.[43,44] A previous study found that half of children improved their anthropometric status, despite no mean group change in BMI SDS.[44] Currently, there is no consensus on thresholds that indicate clinically meaningful changes in BMI SDS among children and adolescents. Suggested reductions in BMI SDS required to improve metabolic health range from 0.1 to 0.5.[26,27,45] In general, it appears that a reduction in BMI SDS of ≥0.25 is required for clinical effectiveness,[26,43,44] and larger

benefits can be expected with reductions of ≥0.50.[44] However, another Norwegian study found that even small or modest BMI SDS reductions from ≥0.00 to <0.10 were associated with an improvement in several cardiovascular risk factors.[45] Therefore, it is likely that any BMI SDS reduction among children with obesity is clinically beneficial,[26,44] especially in those with severe obesity or obesity-related comorbidities. Furthermore, it is plausible that children with obesity not receiving treatment will increase in percentage of overweight.[16]

In this study, we presented BMI outcomes in terms of %IOTF-25, in addition to BMI SDS. Changes in adiposity in children with severe obesity might be difficult to detect using BMI SDS, because large BMI differences corresponds to only small BMI SDS changes.[25] Therefore, BMI expressed as a percentage of the limits of obesity has been proposed as an alternative measure to BMI SDS, more specifically the % IOTF-25.[25] Since the use of %IOTF-25 as an alternative measure to BMI SDS has recently been suggested,[25] no directly comparable studies are available. However, a recent US study including 7–11-year old children,[42] reported that when using a similar parameter, percent of the 95th percentile of the Centers of Disease Control and Prevention BMI-reference, the degree of change was found similar irrespective of weight status being overweight/obesity/severe obesity, whereas the reduction in BMI SDS was found lower in the group with severe obesity.[42]

The overall positive results in BMI outcomes with FBSFT versus TAU might be related to treatment content. In contrast to TAU, FBSFT is a structured cognitive behavioural approach that targets both children and parents.[19] In this study, FBSFT was delivered across an intensive treatment phase with 17 fortnightly sessions, whilst TAU consisted of monthly counselling sessions with a local healthcare nurse and quarterly sessions at the obesity clinic for 1 year. It is possible that a shorter between-session interval proved advantageous to FBSFT participants, but less so with longer intervals in TAU, suggesting a more concentrated intervention delivery schedule may be beneficial. Notably, the differences exhibited in BMI-related outcomes between the two groups are most likely driven by their distinct treatment content and targets, indicating the importance of FBSFT and its family-based approach. Family involvement is a key to treatment success, although its optimal extent remains unclear.[46] However, parents changing their own behaviours to help their child has been reported as crucial to treatment success,[46] and this is an important component of FBSFT.

No significant differences for changes in sleep duration, physical activity, or eating behaviour during treatment were observed between the two groups. Analyses of sleep behaviour showed that changes in sleep timing were significantly different between the groups, with a small increase in mid-sleep time from baseline to posttreatment in the FBSFT group, compared to a small decrease in the TAU group. However, the changes in mid-sleep time from pre- to posttreatment were not statistically significant for either group separately. Therefore, it is unlikely that the observed changes between groups are clinically meaningful. To our knowledge, this study is the first to objectively measure sleep behaviour in school-aged children receiving FBT for obesity.[23] Our study was not able to detect meaningful differences in change between groups. However, since obesity and insufficient sleep are bidirectionally associated in children,[22] we recommend further investigation of sleep as a part of obesity treatment.

Physical activity is addressed similarly in FBSFT and TAU, therefore the nonsignificant between group difference in change was somewhat expected and in line with results from previous studies.[5,7] However, it is surprising that a significant mean reduction in time spent on MVPA was observed in both groups combined. A wider focus on the barriers that deter children with obesity from engaging in physical activity could possibly strengthen the physical activity component of intervention programmes.[47]

The effects of multidisciplinary treatment for childhood obesity on eating behaviour in children with obesity has until recently been largely unknown.[48] A systematic review from 2019 concluded that multidisciplinary treatment with a cognitive behavioural component had a positive impact on external and emotional eating, whilst findings for efficacy on dietary restraint were mixed.[48] Another recent systematic review[49] reported on five studies using DEBQ emotional eating subscale as a pre-postmeasure in obesity treatment trials including a dietary component. Two of the studies reported a significant reduction of emotional eating and another study found this change among boys but not among girls. The last two studies reported no change,[49] as in our study data. In our study, participants in both groups presented with few symptoms of emotional eating at baseline. Therefore, marked favourable changes were not expected. Interestingly, however, healthcare workers involved with the FBSFT group often observed symptoms of emotional eating among participating adolescents, although these behaviours were not reported in questionnaire assessments. One possible explanation for our finding is that awareness and understanding of emotional eating among children and adolescents at baseline might be limited, and self-report measures may not capture the extent of symptoms of emotional eating experienced. Another explanation is that emotional eating might be considered shameful to report.

Altogether, the results from secondary outcomes (sleep, physical activity and eating behaviour) indicate that observed differences in change in weight outcomes cannot be explained by differences in the lifestyle behaviours measured.

This study has several strengths and limitations. We used an RCT design which is given a high level of evidence for evaluation of treatment options. Because the average BMI SDS at baseline was relatively high, compared to similar RCTs,[1] with few exclusion criteria, we had the opportunity to investigate the FBSFT approach in a growing patient group of children with the most severe form of obesity.[20] Dropout rates were comparable in both treatment groups and relatively low (19.3%), compared to rates of 27%–73% previously reported.[50] Finally, we used objective measures of sleep and physical activity, in contrast to most previous studies that relied on self- or parent-reported data.[5,7,23]

Our study also has some limitations. First, there are no data on energy intake. In the first year of the study, participants were asked to complete a 4-day food record at baseline and posttreatment, but

this was abandoned due to low compliance. It is plausible that the beneficial BMI outcomes in the FBSFT group are due to a reduced calorie intake.[51] Another limitation is the difference in mean treatment duration between the two groups. Participants in both groups were evaluated as planned at the end of their respective programmes which included a comparable number of sessions, but with the FBSFT group having shorter between-session intervals than the TAU group. Consequently, the treatment period lasted on average approximately 6 months for the FBSFT group and 12 months for the TAU group. If longer treatment duration is hypothesized to produce better results, we would expect an even larger difference between groups in favour of FBSFT with similar duration of treatment. However, analyses of anthropometric data from the TAU group after 6 months showed the same BMI SDS increase (0.03 units) as after 12 months, and thus did not affect our study conclusions. Finally, we cannot comment on the sustainability of the demonstrated results, and further work on examining long-term follow-up data are warranted.

In conclusion, this study demonstrates that among children and adolescents with severe obesity, FBSFT delivered in a public healthcare setting has overall better treatment effects on BMI-related outcomes, compared to 1 year of TAU. However, changes in the measured lifestyle behaviours were minimal, thus indicating that observed differences in weight outcomes cannot be explained by differences in the included lifestyle behaviours. Considering these findings, expanding access to FBSFT for children and adolescents with severe obesity is an important next step in the treatment of childhood obesity. Alternatively, it may be beneficial to include FBSFT in a stepped approach offered to individuals who do not respond to standard lifestyle treatment.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Hanna F. Skjåkødegård, Yngvild S. Danielsen, Mathieu Roelants, Rachel P. K. Conlon, Denise E. Wilfley and Petur B. Juliusson conceived and designed the study. Hanna F. Skjåkødegård, Yngvild S. Danielsen and Petur B. Juliusson collected the data. Sven J. G. Olsson developed the tailor-made software for processing physical activity data. Hanna F. Skjåkødegård, Yngvild S. Danielsen, Bente Frisk and Sigurd W. Hystad performed statistical analyses. Hanna F. Skjåkødegård wrote the paper in consultation with Yngvild S. Danielsen, Mathieu Roelants, Bente Frisk, Sigurd W. Hystad, Sven J. G. Olsson, Rachel P. K. Conlon, Denise E. Wilfley and Petur B. Juliusson. All authors discussed the results and contributed to the final manuscript.

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REFERENCES

1. Ells LJ, Rees K, Brown T, et al. Interventions for treating children and adolescents with overweight and obesity: an overview of Cochrane reviews. *Int J Obes*. 2018;42(11):1823-1833.
2. Afshin A, Forouzanfar MH, Reitsma MB, et al. Health effects of overweight and obesity in 195 countries over 25 years. *N Engl J Med*. 2017;377(1):13-27.
3. Selvendran SS, Penney NC, Aggarwal N, Darzi AW, Purkayastha S. Treatment of obesity in young people-a systematic review and meta-analysis. *Obes Surg*. 2018;28(8):2537-2549.
4. Mead E, Atkinson G, Richter B, et al. Drug interventions for the treatment of obesity in children and adolescents. *Cochrane Database Syst Rev*. 2016;11(11):Cd012436.
5. Al-Khudairy L, Loveman E, Colquitt JL, et al. Diet, physical activity and behavioural interventions for the treatment of overweight or obese adolescents aged 12 to 17 years. *Cochrane Database Syst Rev*. 2017;6(6):Cd012691.
6. Ells LJ, Mead E, Atkinson G, et al. Surgery for the treatment of obesity in children and adolescents. *Cochrane Database Syst Rev*. 2015;6(6):Cd011740.
7. Mead E, Brown T, Rees K, et al. Diet, physical activity and behavioural interventions for the treatment of overweight or obese children from the age of 6 to 11 years. *Cochrane Database Syst Rev*. 2017;6(6):Cd012651.
8. O'Connor EA, Evans CV, Burda BU, Walsh ES, Eder M, Lozano P. Screening for obesity and intervention for weight Management in Children and Adolescents: evidence report and systematic review for the US preventive services task force. *JAMA*. 2017;317(23):2427-2444.
9. Tran BX, Dang KA, Le HT, et al. Global evolution of obesity research in children and youths: setting priorities for interventions and policies. *Obes Facts*. 2019;12(2):137-149.
10. Wilfley DE, Hayes JF, Balantekin KN, Van Buren DJ, Epstein LH. Behavioral interventions for obesity in children and adults: evidence base, novel approaches, and translation into practice. *Am Psychol*. 2018;73(8):981-993.
11. Cardel MI, Atkinson MA, Taveras EM, Holm JC, Kelly AS. Obesity treatment among adolescents: a review of current evidence and future directions. *JAMA Pediatr*. 2020;174(6):609-617.
12. Hamid MSA, Sazlina SG. Interventions for obesity among schoolchildren: a systematic review and meta-analyses. *PLoS One*. 2019;14(1):e0209746.
13. Alman KL, Lister NB, Garnett SP, Gow ML, Aldwell K, Jebeile H. Dietetic management of obesity and severe obesity in children and adolescents: a scoping review of guidelines. *Obes Rev*. 2021;22(1):e13132.
14. Epstein LH, Wrotniak BH. Future directions for pediatric obesity treatment. *Obesity*. 2010;18:S8-S12.
15. Epstein LH, Schechtman KB, Kilanowski C, et al. Implementing family-based behavioral treatment in the pediatric primary care setting: design of the PLAN study. *Contemp Clin Trials*. 2021;109:106497.
16. Wilfley DE, Tibbs TL, Van Buren DJ, Reach KP, Walker MS, Epstein LH. Lifestyle interventions in the treatment of childhood overweight: a meta-analytic review of randomized controlled trials. *Health Psychol*. 2007;26(5):521-532.
17. Epstein LH, Paluch RA, Roemmich JN, Beecher MD. Family-based obesity treatment, then and now: twenty-five years of pediatric obesity treatment. *Health Psychol*. 2007;26(4):381-391.

18. Helsedirektoratet. Nasjonale faglige retningslinjer for primærhelsetjenesten. Forebygging og behandling av overvekt og fedme hos barn og unge. 2010.
19. Skjakodegard HF, Danielsen YS, Morken M, et al. Study protocol: a randomized controlled trial evaluating the effect of family-based behavioral treatment of childhood and adolescent obesity-the FABO-study. *BMC Public Health*. 2016;16(1):1106.
20. Halberstadt J, van Strien T, de Vet E, Eekhout I, Braet C, Seidell JC. The association of eating styles with weight change after an intensive combined lifestyle intervention for children and adolescents with severe obesity. *Appetite*. 2016;99:82-90.
21. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;7(4):284-294.
22. Morrissey B, Taveras E, Allender S, Strugnell C. Sleep and obesity among children: a systematic review of multiple sleep dimensions. *Pediatr Obes*. 2020;15(4):e12619.
23. Agaronov A, Ash T, Sepulveda M, Taveras EM, Davison KK. Inclusion of sleep promotion in family-based interventions to prevent childhood obesity. *Child Obes*. 2018;14(8):485-500.
24. Juliusson PB, Roelants M, Nordal E, et al. Growth references for 0-19 year-old Norwegian children for length/height, weight, body mass index and head circumference. *Ann Hum Biol*. 2013;40(3):220-227.
25. Júlíusson PB, Roelants M, Benestad B, et al. Severe obesity is a limitation for the use of body mass index standard deviation scores in children and adolescents. *Acta Paediatr*. 2018;107(2):307-314.
26. Ford AL, Hunt LP, Cooper A, Shield JP. What reduction in BMI SDS is required in obese adolescents to improve body composition and cardiometabolic health? *Arch Dis Child*. 2010;95(4):256-261.
27. Reinehr T, Lass N, Toschke C, Rothermel J, Lanzinger S, Holl RW. Which amount of BMI-SDS reduction is necessary to improve cardiovascular risk factors in overweight children? *J Clin Endocrinol Metab*. 2016;101(8):3171-3179.
28. Chow CM, Wong SN, Shin M, et al. Defining the rest interval associated with the main sleep period in actigraph scoring. *Nat Sci Sleep*. 2016;8:321-328.
29. Meltzer LJ, Montgomery-Downs HE, Insana SP, Walsh CM. Use of actigraphy for assessment in pediatric sleep research. *Sleep Med Rev*. 2012;16(5):463-475.
30. Meltzer LJ, Wong P, Biggs SN, et al. Validation of actigraphy in middle childhood. *Sleep*. 2016;39(6):1219-1224.
31. Bonomi AG, Westerterp KR. Advances in physical activity monitoring and lifestyle interventions in obesity: a review. *Int J Obes*. 2012;36(2):167-177.
32. Fairclough JS, Noonan VR, Rowlands MA, Van Hees MV, Knowles MZ, Boddy ML. Wear compliance and activity in children wearing wrist- and hip-mounted accelerometers. *Med Sci Sports Exerc*. 2016;48(2):245-253.
33. Ekblom O, Nyberg G, Bak EE, Ekelund U, Marcus C. Validity and comparability of a wrist-worn accelerometer in children. *J Phys Act Health*. 2012;9(3):389-393.
34. van Strien T. *Dutch Eating Behaviour Questionnaire: Manual*. Thames Valley Test Company; 2002.
35. van Strien T, Oosterveld P. The children's DEBQ for assessment of restrained, emotional, and external eating in 7- to 12-year-old children. *Int J Eat Disord*. 2008;41(1):72-81.
36. Leppink J, O'Sullivan P, Winston K. Are differences between groups different at different occasions? *Perspect Med Educ*. 2017;6(6):413-417.
37. Schafer JL, Graham JW. Missing data: our view of the state of the art. *Psychol Methods*. 2002;7(2):147-177.
38. Lakens D. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for *t*-tests and ANOVAs. *Front Psychol*. 2013;4:863.
39. Faul F, Erdfelder E, Buchner A, Lang A-G. Statistical power analyses using G* Power 3.1: tests for correlation and regression analyses. *Behav Res Methods*. 2009;41(4):1149-1160.
40. Croker H, Viner RM, Nicholls D, et al. Family-based behavioural treatment of childhood obesity in a UK National Health Service setting: randomized controlled trial. *Int J Obes*. 2012;36(1):16-26.
41. Gunnarsdottir T, Sigurdardottir ZG, Njardvik U, Olafsdottir AS, Bjarnason R. A randomized-controlled pilot study of Epstein's family-based behavioural treatment for childhood obesity in a clinical setting in Iceland. *Nordic Psychology*. 2011;63(1):6-19.
42. Hayes JF, Fowler LA, Balantekin KN, et al. Children with severe obesity in family-based obesity treatment compared with other participants: conclusions depend on metrics. *Obesity*. 2021;29(2):393-401.
43. Dubnov-Raz G, Berry EM. What paediatric obesity treatment programmes work, and how can we measure their success? *Acta Paediatr*. 2017;106(11):1724-1726.
44. Dalla Valle M, Laatikainen T, Lehikoinen M, Nykänen P, Jääskeläinen J. Paediatric obesity treatment had better outcomes when children were younger, well motivated and did not have acanthosis nigricans. *Acta Paediatr*. 2017;106(11):1842-1850.
45. Kolsgaard ML, Joner G, Brunborg C, Anderssen SA, Tonstad S, Andersen LF. Reduction in BMI *z*-score and improvement in cardiometabolic risk factors in obese children and adolescents. The Oslo Adiposity Intervention Study—a hospital/public health nurse combined treatment. *BMC Pediatr*. 2011;11:47.
46. Bean MK, Caccavale LJ, Adams EL, et al. Parent involvement in adolescent obesity treatment: a systematic review. *Pediatrics*. 2020;146(3):E20193315.
47. Stankov I, Olds T, Cargo M. Overweight and obese adolescents: what turns them off physical activity? *Int J Behav Nutr Phys Act*. 2012;9:53.
48. De Giuseppe R, Di Napoli I, Porri D, Cena H. Pediatric obesity and eating disorders symptoms: the role of the multidisciplinary treatment. A systematic review. *Front Pediatr*. 2019;7:123.
49. Jebeile H, Gow ML, Baur LA, Garnett SP, Paxton SJ, Lister NB. Treatment of obesity, with a dietary component, and eating disorder risk in children and adolescents: a systematic review with meta-analysis. *Obes Rev*. 2019;20(9):1287-1298.
50. Skelton JA, Beech BM. Attrition in paediatric weight management: a review of the literature and new directions. *Obes Rev*. 2011;12(5):e273-e281.
51. Duncanson K, Shrewsbury V, Burrows T, et al. Impact of weight management nutrition interventions on dietary outcomes in children and adolescents with overweight or obesity: a systematic review with meta-analysis. *J Hum Nutr Diet*. 2021;34(1):147-177.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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Family-based treatment of children with severe obesity in a public healthcare setting: Results from a randomized controlled trial

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TABLE S1 Comparison of baseline characteristics between participants who completed the study and dropouts

	Completed		Dropouts		
Variables	**N**	**Mean ± SD or %**	**N**	**Mean ± SD or %**	***P*-value**
Age (years)	92	12.5 ± 3.1	22	12.9 ± 2.9	0.632*
Gender (% girls)	92	60.9	22	50.0	0.352**
BMI SDS[a]	92	2.9 ± 0.5	22	3.0 ± 0.5	0.621*
%IOTF-25[b]	92	144.4 ± 11.4	22	150.5 ± 16.8	0.045*

Abbreviations: BMI, body mass index; SD, standard deviation; SDS, standard deviation score; %IOTF-25, percentage above the International Obesity Task Force cut-off for overweight.

[a]Calculated using the Norwegian growth reference.

[b]Calculated using the International Obesity Task Force criterion for overweight.

**P*-value obtained by independent *t*-test indicating differences between treatment groups.

***P*-value obtained by Pearson's chi-square test.

III

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ORIGINAL RESEARCH

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Perceived barriers in family-based behavioural treatment of paediatric obesity – Results from the FABO study

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Summary

Background: To date, few studies have investigated perceived barriers among those who participate in and drop out of family-based behavioural treatment (FBT) for paediatric obesity. Examining experienced barriers during treatment, and their role in participation and completion of treatment has important implications for clinical practice.

Objectives: To compare perceived barriers to participating in a family-based behavioural social facilitation treatment (FBSFT) for obesity among families who completed and did not complete treatment.

Methods: Data were analysed from 90 families of children and adolescents (mean (*M*) age = 12.8 years, standard deviation (*SD*) = 3.05) with severe obesity enrolled in a 17-session FBSFT program. After completing 12 sessions or at the time of dropout, parents and therapists completed the *Barriers to Treatment Participation Scale* (BTPS), a 5-point Likert scale (1 = never a problem, 5 = very often a problem) which includes four subscales: 1. *Stressors and obstacles that compete with treatment*, 2. *Treatment demands and issues*, 3. *Perceived relevance of treatment*, 4. *Relationship with the therapist*.

Abbreviations: BTPS, barriers to treatment participation scale; FBT, family-based behavioural treatment; FABO, family-based behavioural treatment of childhood obesity study; RCT, randomized controlled study; FBSFT, family-based behavioural social facilitation treatment; IOTF, international obesity task force; BMI, body mass index; SDS, standard deviation score; SD, standard deviation.

Results: Families who did not complete treatment scored significantly higher on the BTPS subscales *stressors and obstacles that compete with treatment* ($M = 2.03$, $SD = 0.53$ vs. $M = 1.70$, $SD = 0.42$), $p = 0.010$ and *perceived relevance of treatment* ($M = 2.27$, $SD = 0.48$ vs. $M = 1.80$, $SD = 0.50$), $p < 0.001$ than families who completed treatment. No other significant differences between groups were observed.

Conclusion: Families are more likely to drop out of FBSFT when experiencing a high burden from life stressors or when treatment is not meeting the expectations and perceived needs of the family.

KEYWORDS
adolescent, attrition, barriers to treatment, children, dropout, family-based treatment, paediatric obesity

1 | INTRODUCTION

Paediatric obesity, recognized as a global health challenge for decades, is now further exacerbated in the Covid-19 pandemic.[1] In this context, efforts to develop effective interventions for children with obesity are critically important, especially addressing the high risk of attrition from intervention programming that impairs disease control and decreases treatment effectiveness.[2–6] Examining the barriers families experience during treatment, and the role these barriers play in participation and completion of treatment, offers an opportunity to improve delivery methods, identify families at risk for dropping out, and tailoring the treatment to improve compliance and impact.

The majority of studies on attrition from paediatric obesity treatment have focused on pre-treatment predictors,[2,7] commonly comprised of demographic variables, for example, age, sex, initial body weight and socioeconomic status.[7] Previous dieting attempts, psychopathology and body image have also been investigated, all with mixed findings regarding their ability to predict attrition.[2] The lack of consistent findings can result from differences in the target populations, treatment approaches and definitions of attrition between studies.[2,7] Nevertheless, the inconsistent findings indicate that factors other than pre-treatment predictors may play an important role for treatment retention.[2] Efforts to identify these factors, and thereby make it possible to develop strategies to enhance retention rates and prevent dropout are highly needed.[5] To address this, the *Barriers to Treatment Participation Scale* (BTPS)[8,9] has been proposed as a suitable measure to identify factors perceived as barriers for participation in paediatric obesity treatment.[7]

To date, few studies have investigated perceived barriers for treatment participation in lifestyle interventions for paediatric obesity.[2,6,10] The existing studies, are mainly qualitative, and report that a high burden from life stressors (e.g., single parent household with multiple children, parental chronic illnesses, limited means and logistical challenges) forms a complex interplay of barriers interfering with treatment participation.[2,6,11–13] Interestingly, logistical challenges have been put forward as more related to treatment attrition than program satisfaction.[4,12,14,15] It seems like busy work schedules for parents, lack of transportation and insurance coverage may contribute to attrition despite low degree of dissatisfaction with the programs.[4,14,16] Furthermore, it is worth noting, that previous research on barriers for participating in lifestyle treatment for paediatric obesity has mainly focused on those who did not complete treatment, without comparison of experienced barriers among those who completed treatment,[2,6] resulting in a lack of knowledge related to similarities and differences in reported barriers between the two groups.[2]

Family-based behavioural treatment (FBT) is an evidence-based intervention for paediatric obesity, shown to yield clinically significant weight loss.[17–19] Investigating barriers for participation in this kind of treatment and the associations to attrition or retention is an important addition to research on pre- to post-treatment change in weight and behavioural outcomes.[14,19] Studies have indicated that, in addition to family stressors, different aspects of the treatment (demands and relevance) and alliance with the therapist are likely to influence treatment attendance and outcome in psychological treatment of children and families.[8,20] These kinds of within-treatment barriers have rarely been examined in relation to FBT for paediatric obesity. The family-based behavioural treatment of childhood obesity (FABO) study,[21] offers an opportunity to investigate barriers evident during and in relation to participation in an enhanced FBT for paediatric obesity.

Thus, the aim of the present study was to compare perceived barriers to treatment participation in family-based behavioural social facilitation treatment (FBSFT) for paediatric obesity among families who did or did not complete the intervention. We hypothesized that there would be a higher level of perceived barriers among families who did not complete the treatment.

2 | METHODS

2.1 | Study design

This research is part of the FABO study,[21] a randomized controlled trial (RCT) evaluating the effect of FBSFT compared to the standard

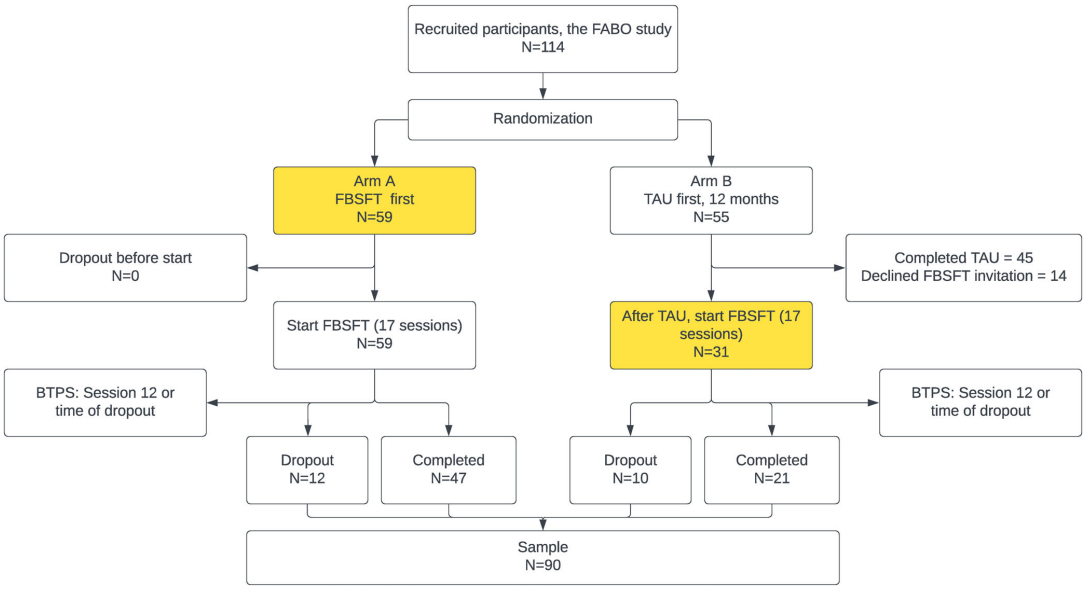

FIGURE 1 Flow chart showing the participant flow for the FBSFT-part of the FABO study. Coloured boxes represent the baseline time points for participants included in the current study. BTPS, barriers to treatment participation scale; FBSFT, family-based behavioural social facilitation treatment; TAU, treatment as usual

treatment given to children with severe obesity at the Obesity Outpatient Clinic, Haukeland University Hospital, Bergen, Norway.[19,21] Participants were recruited from February 2014 to October 2018. The FABO study involved a waitlist control design in which all participants eventually were offered FBSFT, and the current analysis includes data from families while participating in the FBSFT portion of the trial. Figure 1 describes the study design and participant flow. Written informed consent was obtained prior to inclusion. The consent was obtained from all participating adolescents older than 16 years, or otherwise from their parents, complemented with an informed consent when the child was 12 years of age or older.

The study was approved by the Regional Committee for Medical and Health Research Ethics, Western Norway (number 2013/1300) and was registered on ClinicalTrials.gov (NCT02687516).

2.2 | Participants

A total of 90 families with children and adolescents (aged 6–18 years) with severe obesity are included in this analysis. Criteria for admission to the study was an International Obesity Task Force (IOTF)[22] body mass index (BMI) ≥35 or BMI ≥30 with obesity related co-morbidity. The child participated in the treatment together with her/his family, such that both the child and at least one of the parents agreed to actively participate. Families were excluded if either the child or parent(s) experienced severe somatic or psychiatric illness that could interfere with the treatment program, or current participation in other obesity treatment programs.

2.3 | Description of treatment

FBSFT builds on FBT,[19,21] and consisted of 17 individual family sessions. The intention was to deliver the sessions weekly,[21] but due to logistical challenges when delivering the treatment in a real-world health care clinic, the treatment ended up being delivered in an unstructured combination of weekly and fortnightly sessions. Mean treatment delivery was approximately 6 months. In the sessions each family worked on changing lifestyle behaviours using a structured cognitive behavioural approach.[21]

The treatment targets healthy lifestyle changes in both children and parents in the domains of diet, physical activity, sedentary activity, sleep, and social function. Through the treatment sessions, the families are taught a set of behavioural and cognitive techniques for promoting healthy behaviour change and dealing with factors that maintain unhealthy lifestyle behaviours. There are session-specific components and goals, and from session-to-session the families are encouraged to self-monitor their behaviours and support for health behaviours in their home, peer, and community environments. Further description of the treatment is provided in the published study protocol.[21]

2.3.1 | Completion of treatment

Completion of treatment is defined as attending >75%[23] (i.e., ≥13) of the 17 sessions. Families who attended <13 sessions were considered to have dropped out (i.e., did not complete treatment).

2.4 | Anthropometric measures

Height and weight were measured by trained personnel at the Obesity Outpatient Clinic. Height was measured with a digital wall-mounted stadiometer (Seca 264, Seca, Hamburg, Germany) and recorded to the nearest 0.1 cm. The participant was wearing underwear (without socks and shoes). Body weight was measured with a digital scale (InBody720, Biospace, Seoul, Korea) and recorded to the nearest 0.1 kg. BMI was calculated by dividing the persons weight in kilograms by the square of height in meters (kg/m^2), and further converted to BMI standard deviation score (SDS) derived from the Norwegian growth reference.[24]

2.5 | Demographic information

Family structure, parental education levels and parental employment were measured with a parental questionnaire at baseline.[19] The questionnaire was part of the baseline assessment at the Obesity Outpatient Clinic.

2.6 | Barriers for treatment measure

Barriers for treatment were investigated with the Barriers to Treatment Participation Scale (BTPS).[8,9] The BTPS was developed and validated to address dropout from treatment with outpatient psychological treatment of children and adolescents.[8,9] The main section of the questionnaire consists of 44 statements evaluated on a 5-point Likert scale (1 = never a problem, 5 = very often a problem). Scores were distributed across four subscales: (1) *Stressors and obstacles that compete with treatment*, (2) *Treatment demands and issues*, (3) *Perceived relevance of treatment*, (4) *Relationship with the therapist*. Statements 9 and 10, related to treatment costs, and statement 37 *the therapist did not call often enough* were not applicable for our study, and were therefore excluded when calculating scores. Subscale scores are calculated using the average of the items. In addition to the four subscales, BTPS includes 14 questions about specific critical life events that are answered in a yes or no format. The purpose of these 14 questions is to distinguish perceived barriers associated with treatment participation from specific life-changing events.[25] The question *my medical insurance did not cover this treatment* was not applicable since the treatment was free of charge and excluded when calculating critical event score. Parent and therapist versions of BTPS were used and completed by both families and therapists either at program dropout or after completion of 12 out of the 17 FBSFT sessions. On average, session 12 was delivered in week 18 of the FBSFT program. The BTPS[8] is outlined in Table 1 with permission from the authors. The BTPS has been found to yield high levels of internal consistency and to be predictive of treatment drop-out, cancellation of appointments and weeks spent in treatment.[8]

2.7 | Statistical analyses

Data were analysed with IBM SPSS version 27 (IBM Corp., Armonk, NY). Descriptive statistics of continuous variables are given by the mean and SD, and of categorical variables by the frequency and percentage. Demographic variables for the groups of families completing and not completing treatment were compared with *t*- and chi-square tests for continuous and categorical variables.

To compare perceived barriers to treatment between families who did and did not complete treatment, we first calculated the four different BTPS subscales. Higher scores indicate greater presence of problems and barriers to treatment. A Hotelling's T^2 test was then used to compare the multivariate data (i.e., the BTPS subscales) between groups. A Box M test was used to test the assumption of homogeneity, that is, that both populations have a common variance–covariance matrix. Statistically significant T^2 values were followed-up with post-hoc comparisons of individual subscales, using independent-samples *t*-tests with a Bonferroni correction.

The subscale *Relationship with the therapist* was not included in the above multivariate analysis. As more than 50% of the sample had a mean score equal to 1 on this subscale, the variable was highly skewed with limited variance, and a comparison of groups was therefore not feasible or meaningful. Instead, we performed a Wilcoxon rank-sum test to test the hypothesis that the comparison groups are from populations with the same distribution and computed the probability that a random case from one group has a higher score on *Relationship with the therapist* than a random case from the other group.

To compare critical life events between families who did and did not complete treatment, we first summed the life events questions into a composite score, and then performed a Wilcoxon rank-sum test as explained above.

3 | RESULTS

Baseline characteristics of the study population are presented in Table 2, both in total and for families who completed and did not complete treatment separately. No significant differences between groups were observed. Of the 90 participants (mean age 12.8 years; minimum – maximum: 5–9 to 17.7 years) who participated in the FBSFT-part of the FABO study, 68 (75.5%) families completed treatment, while 22 (24.5%) families did not complete treatment. Mean dropout session was session 6, with session 12 representing latest dropout point.

81 of 90 families (90%) participating in FBSFT filled out the BTPS. The therapist questionnaire was filled out for 86 of 90 families (95.5%). For three families both parent- and therapist questionnaire was missing, for six families only the parent questionnaire was missing, and for one family only the therapist questionnaire was missing.

Means, standard deviations and Pearson correlations between the BTPS subscales are presented in Table 3, whereas Cronbach's alphas and correlations between family- and therapist ratings are presented in Table 4. Internal consistency of the subscales was

TABLE 1 Subscales and Items of the Barriers to Treatment Participation Scale

I . Stressors and obstacles that compete with treatment (20 items, Scored 1–5)
2. Transportation (getting a ride, driving, taking a bus) to the clinic for a session
3. My child was in other activities (sport, music lessons) that made it hard to come to a session
4. Scheduling of appointment times for treatment
6. Treatment was in conflict with another of my activities (classes, job, friends)
14. During the course of treatment I experienced a lot of stress in my life
16. I was sick on the day when treatment was scheduled
17. My child was sick on the day when treatment was scheduled
18. Crises at home made it hard for me to get to a session
20. Treatment added another stressor to my life
31. There was bad weather and this made coming to treatment a problem
34. I did not have time for the assigned work
35. My child was never home to do the assigned homework
36. There was always someone sick in my home
38. Getting a baby-sitter so I could come to the sessions
39. Finding a place to park at the clinic
40. I had a disagreement with my husband, boyfriend, or partner about whether we should come to treatment at all
41. I was too tired after work to come to a session
42. My job got in the way of coming to a session
43. Treatment took time away from spending time with my children
44. I had trouble with other children at home which made it hard to come to treatment
II. Treatment demands and issues (10 items, Scored 1–5)
1. My child refused to come to the session
5. Treatment lasted to long (too many weeks)
9. I felt that treatment cost too much
10. I was billed for the wrong amount
12. Information in the session and handouts seemed confusing
13. My child had trouble understanding treatment
22. I felt this treatment was more work than expected
23. The atmosphere at the clinic makes it uncomfortable for appointments
24. I did not feel that I had enough to say about what goes on in treatment
33. The assigned work for me to do as part of this treatment was much too difficult
III. Perceived relevance of treatment (8 items, Scored 1–5)
7. Treatment did not seem necessary
11. Treatment was not what I expected
15. I lost interest in coming to sessions
21. I felt treatment did not seem as important as the sessions continued
25. I feel treatment did not focus on my life and problems
28. My child now has new or different problems
29. My child's behaviour seems to have improved, therefore, treatment no longer seems necessary
30. Treatment did not seem to be working
IV. Relationship with the therapist (6 items, Scored 1–5)
8. I did not like the therapist
19. I felt I had to give too much personal information to the therapist
26. The therapist did not seem confident that treatment would work for my child
27. The therapist did not seem confident in my ability to carry out programs
32. I do not feel the therapist supported me or my efforts
37. The therapist did not call often enough

(Continues)

TABLE 1 (Continued)

V. Critical events (14 items, Scored yes, no)
45. I moved to another house or apartment during the time my child was in treatment
46. My medical insurance did not cover this treatment
47. I moved to far way from clinic to come to treatments sessions (out of the area)
48. My family changed in size (another baby or someone moved in or out of the home)
49. I lost my job or had a change in income
50. I got a job or changed jobs
51. There was an alcohol or drug problem in my family
52. There was physical or sexual abuse in my family
53. A close friend or relative got very sick or died during treatment
54. My child moved out of the home
55. My child was put into an in-patient program or residential program
57. My child changed schools during treatment
56. I had legal problems (arrest, driving violations, etc.)
58. I got separated or divorced

Note: Reproduced from Kazdin et al. *J Child Psychol Psychiatry*. 1997,[8] with permission. The items constitute the parent version of the scales, the items are the same for the therapist version, with adjusted wording to convey that parent and child are to be evaluated.

TABLE 2 Characteristics of the study population at baseline, in total and by groups of families who did and did not complete treatment

		Completed	Not completed	*p* value
Total (*N*)	90	68	22	
Age (mean, SD)	12.79 (3.05)	12.7 (3.1)	13.2(2.9)	0.490
Range	5.9–17.7	5.9–17.4	10.7–17.7	
Sex: girls (%)	53 (58.9%)	42 (61.8%)	11 (50%)	0.468
BMI (mean, SD)	32.18 (4.88)	31.6(4.59)	33.9(5.46)	0.056
BMI z-score mean (SD)	2.99 (0.49)	2.93(0.48)	3.16(0.49)	0.062
Parent reported data				
Mother born in Norway (%)	87.6%	85.3%	95.2%	0.406
Father born in Norway (%)	86.4%	85.1%	90.5%	0.791
Biological parents living together (%)	60.2%	64.2%	47.6%	0.272
Living with siblings (%)	72.2%	75.0%	63.6%	0.447
Father, full time work (%)	71.9%	71.7%	72.6%	0.908
Father, part time work (%)	1.1%	0.0%	1,5%	0.549
Mother, full time work (%)	52.2%	53.0%	45.0%	0.777
Mother, part time work (%)	18.2%	20.6%	10.0%	0.505
Father, completed education (%)				
≤High school	62.6%	54.9%	85%	0.066
College/University <4 years	20.0%	23.3%	10%	
College/University >4 years	11.3%	13.3%	5%	0.088
Mother, completed education (%)				
≤High school	58.5%	56.7%	65.0%	0.685
College/University <4 years	23.0%	25.0%	15.0%	
College/University >4 years	18.4%	17.9%	20.0%	0.386

Note: *p* values from a chi-square test for categorical data, and independent samples t-test for continuous data. The categories < or >4 years of College/University were merged for the group comparisons.
Abbreviations: BMI, body mass index; SD, standard deviation.

TABLE 3 Means, standard deviations and pearson's correlations r between subscales of the barriers to treatment participation scale for family and therapist reports ($N = 81$)

		1	2	3	4	Mean	SD
1.	Competing stressors and obstacles	—	0.53	0.43	0.54	1.77	0.50
2.	Treatment demands	0.53	—	0.70	0.57	1.67	0.51
3.	Relevance of treatment	0.37	0.59	—	0.52	1.87	0.56
4.	Relationship with therapist	0.44	0.59	0.55	—	1.44	0.49
Mean		1.77	1.57	1.89	1.23	—	—
SD		0.46	0.44	0.53	0.39	—	—

Note: Family ratings are presented below the diagonal and therapist ratings are presented above the diagonal. All correlation *r*s are statistically significant at $p < 0.001$.
Abbreviation: SD, standard deviation.

TABLE 4 Cronbach's alphas and correlations between family-rated and therapist-rated barriers to treatment

Variables		R	α (family/therapist)
1.	Competing stressors and obstacles	0.53***	0.83/0.87
2.	Treatment demands	0.43***	0.61/0.72
3.	Relevance of treatment	0.37***	0.64/0.71
4.	Relationship with therapist	0.16	0.77/0.84

***$p < 0.001$.

TABLE 5 Differences in parent-reported barriers to treatment between families who did and did not complete treatment

	Completed			Not completed					
Subscale	M	SD	*n*	M	SD	*n*	T	*p*[a]	D
Competing stressors and obstacles	1.70	0.42	65	2.03	0.53	16	2.625	**0.010**	0.73
Treatment demands	1.53	0.43	65	1.73	0.46	16	1.586	0.117	0.44
Relevance of treatment	1.80	0.50	65	2.27	0.48	16	3.458	**<0.001**	0.97

Note: Hotelling $T^2 = 16.645$, with Mahalanobis $D^2 = 0.42$. Higher scores indicate greater presence of barriers to treatment.
Abbreviations: M, mean; SD, standard deviation.
[a]*p*-values in bold indicates statistically significant values after applying a Bonferroni correction ($\alpha/m = 0.05/3 = 0.016$).

acceptable in general. There was a high correlation between family and therapist scores for three subscales, but not for the scale *Relationship with the therapist*.

3.1 | Parent version of BTPS, families who completed versus did not complete FBSFT

The Hotelling's T^2 test indicated differences between those who did ($n = 65$) and did not ($n = 16$) complete FBSFT on the BTPS subscales, $T^2 = 16.645$, $df = 3,77$, $p = 0.002$. The Box M test was not statistically significant, $F\ (6,4308.9) = 1.04$, $p = 0.39$, indicating that the covariance matrices were not different, and that the assumption of homogeneity is not violated.

The post-hoc comparison of mean scores on the different subscales (Table 5) showed that families who did versus did not complete treatment differed on the subscales *Stressors and obstacles that compete with treatment* and *Perceived relevance of treatment*. Families who did not complete FBSFT scored significantly higher on *stressors and obstacles* ($M = 2.03$, $SD = 0.53$) than those who completed treatment ($M = 1.70$, $SD = 0.42$), $T = 2.625$, $p = 0.010$. Furthermore, families who did not completed FBSFT scored significantly higher on *relevance of treatment* ($M = 2.27$, $SD = 0.48$) than those who completed treatment ($M = 1.80$, $SD = 0.50$), $T = 3.458$, $p < 0.001$. The mean differences in *stressors and obstacles* (Cohen's $D = 0.73$) and *treatment relevance* (Cohen's $D = 0.97$) represent medium-to-large and large effect sizes, respectively.

The Wilcoxon rank-sum test that compared the groups on *Relationship with the therapist* showed that the two distributions were not statistically different at a 0.05 significance level, $Z = 1.462$, $p = 0.144$. The probability of a random case from the group that did not complete FBSFT having a higher score on *Relationship with the therapist* was not much higher than chance ($p = 0.61$).

The Wilcoxon rank-sum test that compared families who completed versus did not complete on the number of reported critical events also showed that the two distributions were not statistically different at a 0.05 significance level, $Z = 1.237$, $p = 0.216$. Among families who completed treatment ($N = 65$), 66.2% reported no critical events, while 18.5% reported one, 7.7% two, 3.1% three and 4.5% four critical events. Among families who did not complete treatment ($N = 16$), 81.3% reported no critical events, while 12.5% reported one and 6.2% reported two critical events.

TABLE 6 The ten barriers with highest mean ratings for families and therapists

			Family		Therapist	
#	Subscale	Item content	M	Rank	M	Rank
4	CS	Scheduling of appointment times for treatment	1.77	17	1.98	**10**
6	CS	Treatment was in conflict with other activities (classes, job, friends)	2.72	**2**	2.31	**5**
7	TR	Treatment did not seem necessary	2.20	**7**	1.73	18
11	TR	Treatment was not what expected	2.30	**4**	2.07	**8**
14	CS	During the course of treatment parent experienced a lot of stress in life	2.99	**1**	3.03	**1**
20	CS	Treatment added another stressor to life	2.04	**8**	2.57	**3**
22	TD	Treatment was more work than expected	1.96	11	2.20	**6**
29	TR	Child's behaviour seems to have improved, therefore, treatment no longer seems necessary	2.58	**3**	2.93	**2**
30	TR	Treatment did not seem to be working	2.03	**9**	2.19	**7**
34	CS	Did not have time for the assigned work	2.28	**5**	2.47	**4**
39	CS	Finding a place to park at the clinic	2.26	**6**	1.79	16
42	CS	Job got in the way of coming to a session	1.99	**10**	1.99	**9**

Note: The items are the same for both versions, with different wording. # = item number on the questionnaire. #29, score 1 = improved, higher scores indicate greater presence of barriers to treatment. Bold value rank within top ten list.
Abbreviations: CS, competing stressors and obstacles; TD, treatment demands; TR, treatment relevance.

3.2 | Mean ratings for family and therapist versions of the BTPS

The 10 barriers with highest mean rating for families and therapists are reported in Table 6. For both groups, the barrier *during the course of treatment I (the parent) experienced a lot of stress in my life* was the barrier with highest mean rating. Thereafter, the rank of barriers differs between families and therapists.

4 | DISCUSSION

This study demonstrated that families who did not complete FBSFT reported significantly more barriers related to the subscales *stressors and obstacles that compete with treatment* and *perceived relevance of treatment* than families who completed treatment. No group differences were observed for the *treatment demands and issues* and *relationship with the therapist* subscales. The barrier *during the course of treatment I (the parent) experienced a lot of stress in my life* was highest ranked both by parents and therapists. To our knowledge, this is one of the first studies comparing perceived barriers for treatment participation in families who did versus did not complete an enhanced FBT for paediatric obesity.

4.1 | Stressors and obstacles that competed with treatment

Families who did not complete FBSFT reported more perceived stressors and obstacles compared to those who completed treatment. This subscale consists of a wide range of barriers related to events interfering with the ability to attend sessions and treatment serving as, and adding to, other stressors experienced in the family.[26] Our finding is in line with previous research, reporting high degree of family stressors as a challenge for treatment adherence.[6,11] Across all participating families in our study, the barrier *during the course of treatment I (the parent) experienced a lot of stress in my life* was the most prevalent, followed by *treatment conflicting with other activities*. Out of the 10 barriers with highest mean ratings for participating families, six were from the stressors and obstacles subscale. This finding, describing a patient group experiencing a high burden of life stressors, aligns with previous literature on families seeking paediatric obesity treatment.[27,28] The associations between family stress (including both parental perceived stress and stress across the entire home environment) and paediatric obesity are complex, and need to be further investigated to enhance our understanding of their impact on treatment engagement.[29,30] In addition, the experience of stress warrants further investigation, as families experience stress in different ways and parents' response to stress varies.[29] The present study show that the families with the highest degree of competing stressors and obstacles were more likely to leave treatment prematurely. Stressors can be both psychological (e.g., health issues, conflicts, crisis) and logistical, and some of the logistical challenges might be easy to work around if the therapist/clinic is aware of them. In our study, the barrier *finding a place to park at the clinic* had the sixth highest mean rating among families, while for therapists it was ranked as number sixteenth. Increasing therapists' awareness of these issues can increase the likelihood of addressing them. For example, if the therapists had been more aware of this barrier, they could have helped families finding a suitable parking arrangement.

4.2 | Perceived relevance of treatment

This subscale, which reflects the extent to which treatment was seen as relevant to the child's problem, was viewed as important, and met with the families' expectations and needs.[26] Significantly less burden was reported among families who completed FBSFT. These data suggest that the intervention was perceived as less able to meet the expectations and needs of families who did not complete treatment. Previous studies on paediatric obesity also report treatment not meeting expectations as a barrier for participation,[3,6] and mainly it seems like this barrier is related to not achieving the desired weight loss effect.[6] Such outcomes may reflect participants' desires for weight loss that often are accompanied by unrealistic expectations going into the intervention.[6,31] FBSFT has a modest weight loss goal with focus on long-term healthy lifestyle changes,[19] possibly in conflict with the expectations of some of the enrolled families, and thereby potentially increasing risk for dropout.[31] Another issue related to perceived relevance of treatment is parents' divergent views about paediatric obesity,[11] with some parents considering the condition as not in need of treatment. Not viewing obesity as a problem is a known barrier during admission to treatment.[6,32] In our study, the participating families actively agreed to take a more intensive treatment approach,[21] but ambivalence concerning whether the treatment is necessary was still present in the study population: The barrier *treatment did not seem necessary* was the seventh most frequently reported barrier among families. However, the barrier *my child's behaviour seems to have improved, therefore, treatment no longer seems necessary* (score of 1 = improved) is ranked as number three. Nevertheless, the observed differences between non-completers and completers on this subscale highlights the importance of supporting families in identifying, discussing, and managing their expectations and collaboratively establishing realistic treatment goals.[31]

4.3 | Treatment demands and issues and relationship with the therapist

No differences between those who did and did not complete FBSFT were observed for the *treatment demands and issues* and *relationship with therapist* subscales. These findings contrast with previous studies that reported barriers related to treatment demands, especially regarding collection of research data, and dissatisfaction with treatment providers as a reason for ending treatment prematurely.[2]

The *treatment demands and issues* subscale reflects the families' concerns and complaints related to treatment participation and the extent to which the treatment was considered confusing, too long, difficult or demanding.[26] In addition to no differences between those who did and did not complete, none of the barriers on this subscale were on the top 10 list for the total sample of participating families. This is of course encouraging, but also a bit surprising. From a clinical perspective, FBSFT is perceived as requiring a lot of work from the families (e.g., frequent sessions, monitoring behaviours, homework), which may explain why therapists rated the barrier *treatment was more work than expected* higher than families (rank 6 versus 11).

Furthermore, the *relationship with the therapist* subscale investigated alliance, bonding, liking of, perceived support from and disclosure with the therapist.[26] Within psychotherapeutic approaches, the therapeutic alliance is a known predictor for patient outcomes.[33,34] Our study was not able to detect any group difference related to treatment completion, as the whole group of participating families had a low mean score on this subscale. However, it is very positive for the FABO study and the FBSFT intervention that participating families experienced a supportive, strong therapeutic alliance with their therapist. Stigmatization and unequal treatment within the healthcare system have previously been reported for both children and adults with obesity,[35–37] and a lack of trust and connection with healthcare providers represent barriers for adherence in paediatric obesity treatment.[7]

4.4 | Strengths and limitations

There are multiple strengths of the present study. The main strength of this study is the inclusion of all families that received FBSFT, both those who did and did not complete treatment. In addition, 90% of participating families filled out the BTPS. All families were informed that their therapist was not given access to their scores, reducing the risk for social-desirability bias. Furthermore, the use of both parent and therapist versions of the BTPS is novel and made it possible to compare scores and compare perceived barriers among recipients and providers of FBSFT. The study has limitations. The BTPS is often administered by means of an interview,[8,9,26] which may provide more precise answers than the questionnaire format. Furthermore, the BTPS was filled out by the parents. Inclusion of a self-report version for the participating adolescent would have provided valuable insight into their own experienced barriers. Inclusion of qualitative interviews in addition to the use of the BTPS could also have broadened the understanding of the phenomenon. Lastly, due to the sample size, we could not differentiate between the timing of dropout (e.g., early or late), but previous research has shown that there may be meaningful difference based on the timing of dropout.[2]

4.5 | Implications for practice

The results from this study indicate that barriers for participation should be investigated ahead of, during, and when leaving treatment. Examination at multiple time points will enable discussions of barriers and identifications of modifiable components that can be addressed as a part of treatment, and may optimize families' experience during, participation and completion of treatment for paediatric obesity.

Our finding that families with a high degree of stressors and obstacles were more likely to dropout is important to note. Offering families practical support with day-to-day tasks as a part of treatment may prevent dropout and improve treatment impacts for families. At Norwegian obesity clinics, these kinds of support have been offered to some families in collaboration with the child welfare service/ medical social workers. Furthermore, implementing methods of

service delivery that are better suited to the logistical challenges experienced by the families are of great importance. In this study, sessions were delivered during daytime clinic hours, and a potential modification would be to also facilitate evening sessions for families.

A pre-treatment phase to discuss and manage families' expectations and collaboratively establish realistic goals may serve as a valuable addition to FBSFT and paediatric obesity treatment delivery.[31] Unrealistic expectations related to weight effect is a major barrier to participation, while having a positive and realistic expectations is an facilitator for completion.[6,38] Other facilitators for treatment completion and overcoming perceived barriers should also be further investigated. Previous research has demonstrated that the main reason for adherence was a personalized approach by the treatment provider, and the providers effort to establish a personal connection.[6]

4.6 | Conclusion

The results from this study indicate that families participating in family-based behavioural social facilitation treatment for paediatric obesity are more likely to dropout, when experiencing a high burden from life stressors or when treatment is not meeting expectations and perceived needs of the family. Identifying and addressing families' treatment expectations and how they fit with intervention as well as the degree of burden from life stressors that families are experiencing may increase their participation in and completion of family-based treatment for paediatric obesity.

AUTHOR CONTRIBUTIONS

Hanna F. Skjåkødegård, Rachel P. K. Conlon, Denise Wilfley, Petur B. Juliusson and Yngvild S. Danielsen conceived and designed the study. Hanna F. Skjåkødegård, Ingvild Bruserud, Petur B. Juliusson and Yngvild S. Danielsen collected and scored the data. Hanna F. Skjåkødegård, Sigurd Hystad and Yngvild S. Danielsen performed statistical analyses. Hanna F. Skjåkødegård wrote the paper in consultation with Sigurd Hystad, Ingvild Bruserud, Rachel P. K. Conlon, Denise Wilfley, Bente Frisk, Mathieu Roelants, Petur B. Juliusson and Yngvild S. Danielsen. All authors discussed the results and contributed to the final manuscript.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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REFERENCES

1. Jebeile H, Kelly AS, O'Malley G, Baur LA. Obesity in children and adolescents: epidemiology, causes, assessment, and management. *Lancet Diabetes Endocrinol*. 2022;10:351-365.
2. Miller BML, Brennan L. Measuring and reporting attrition from obesity treatment programs: a call to action! *Obes Res Clin Pract*. 2015;9(3):187-202.
3. Skelton JA, Beech BM. Attrition in paediatric weight management: a review of the literature and new directions. *Obes Rev*. 2011;12(5):e273-e281.
4. Skelton JA, Martin S, Irby MB. Satisfaction and attrition in paediatric weight management. *Clin Obes*. 2016;6(2):143-153.
5. Kelleher E, Davoren MP, Harrington JM, Shiely F, Perry IJ, McHugh SM. Barriers and facilitators to initial and continued attendance at community-based lifestyle programmes among families of overweight and obese children: a systematic review. *Obes Rev*. 2017;18(2):183-194.
6. Grootens-Wiegers P, van den Eynde E, Halberstadt J, Seidell JC, Dedding C. The 'stages towards completion model': what helps and hinders children with overweight or obesity and their parents to be guided towards, adhere to and complete a group lifestyle intervention. *Int J Qual Stud Health Well-Being*. 2020;15(1):1735093.
7. Nobles J, Griffiths C, Pringle A, Gately P. Design programmes to maximise participant engagement: a predictive study of programme and participant characteristics associated with engagement in paediatric weight management. *Int J Behav Nutr Phys Act*. 2016;13(1):76.
8. Kazdin AE, Holland L, Crowley M, Breton S. Barriers to treatment participation scale: evaluation and validation in the context of child outpatient treatment. *J Child Psychol Psychiatry*. 1997;38(8):1051-1062.
9. Kazdin AE, Holland L, Crowley M. Family experience of barriers to treatment and premature termination from child therapy. *J Consult Clin Psychol*. 1997;65(3):453-463.
10. Staniford LJ, Breckon JD, Copeland RJ, Hutchison A. Key stakeholders' perspectives towards childhood obesity treatment: a qualitative study. *J Child Health Care*. 2011;15(3):230-244.
11. Silver MP, Cronin SM. Health care Providers' perspectives on family compliance and behavior change in a childhood obesity program. *Health Educ Behav*. 2019;46(4):582-591.
12. Dhaliwal J, Nosworthy NM, Holt NL, et al. Attrition and the management of pediatric obesity: an integrative review. *Child Obes*. 2014;10(6):461-473.
13. Watson PM, Dugdill L, Pickering K, et al. Distinguishing factors that influence attendance and behaviour change in family-based treatment of childhood obesity: a qualitative study. *Br J Health Psychol*. 2021;26(1):67-89.
14. Staiano AE, Marker AM, Comeaux J, Frelier JM, Hsia DS, Broyles ST. Family-based behavioral treatment for childhood obesity: caretaker-reported barriers and facilitators. *Ochsner J*. 2017;17(1):83-92.
15. Kwitowski M, Bean MK, Mazzeo SE. An exploration of factors influencing attrition from a pediatric weight management intervention. *Obes Res Clin Pract*. 2017;11(2):233-240.
16. Berry DC, Rhodes ET, Hampl S, et al. Stay in treatment: predicting dropout from pediatric weight management study protocol. *Contemp Clin Trials Commun*. 2021;22:100799.
17. Epstein LH, Paluch RA, Roemmich JN, Beecher MD. Family-based obesity treatment, then and now: twenty-five years of pediatric obesity treatment. *Health Psychol*. 2007;26(4):381-391.
18. Epstein LH, Schechtman KB, Kilanowski C, et al. Implementing family-based behavioral treatment in the pediatric primary care setting: design of the PLAN study. *Contemp Clin Trials*. 2021;109:106497.

19. Skjåkødegård HF, Conlon RPK, Hystad SW, et al. Family-based treatment of children with severe obesity in a public healthcare setting: results from a randomized controlled trial. *Clin Obes*. 2022;12(3):e12513.
20. Acri MC, Bornheimer L, Hamovitch E, Lambert K. An examination of the relationship between maternal depression and barriers to child mental health services. *Child Youth Serv Rev*. 2018;93:270-275.
21. Skjakodegard HF, Danielsen YS, Morken M, et al. Study protocol: a randomized controlled trial evaluating the effect of family-based behavioral treatment of childhood and adolescent obesity-the FABO-study. *BMC Public Health*. 2016;16(1):1106.
22. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;7(4):284-294.
23. Riggs KR, Lozano P, Mohelnitzky A, Rudnick S, Richards J. An adaptation of family-based behavioral pediatric obesity treatment for a primary care setting: group health family wellness program pilot. *Perm J*. 2014;18(3):4-10.
24. Juliusson PB, Roelants M, Nordal E, et al. Growth references for 0-19 year-old Norwegian children for length/height, weight, body mass index and head circumference. *Ann Hum Biol*. 2013;40(3):220-227.
25. Gresl BL, Fox RA, Besasie LA. Development of a barriers scale to predict early treatment success for young children in poverty with behavior problems. *Clinical Pract Pediatr Psychol*. 2016;4(3):249-262.
26. Kazdin AE, Wassell G. Barriers to treatment participation and therapeutic change among children referred for conduct disorder. *J Clin Child Psychol*. 1999;28(2):160-172.
27. Baskind MJ, Taveras EM, Gerber MW, Fiechtner L, Horan C, Sharifi M. Parent-perceived stress and its association with Children's weight and obesity-related behaviors. *Prev Chronic Dis*. 2019;16:E39.
28. Wilson SM, Sato AF. Stress and paediatric obesity: what we know and where to go. *Stress Health*. 2014;30(2):91-102.
29. Jang M, Owen B, Lauver DR. Different types of parental stress and childhood obesity: a systematic review of observational studies. *Obes Rev*. 2019;20(12):1740-1758.
30. Gundersen C, Mahatmya D, Garasky S, Lohman B. Linking psychosocial stressors and childhood obesity. *Obes Rev*. 2011;12(5):e54-e63.
31. Staniford LJ, Copeland RJ, Breckon JD. 'What's the point when you only lose a pound?' Reasons for attrition from a multi-component childhood obesity treatment intervention: a qualitative inquiry. *Qual Res Sport Exercise Health*. 2019;11(3):382-397.
32. Perez A, Holt N, Gokiert R, et al. Why don't families initiate treatment? A qualitative multicentre study investigating parents' reasons for declining paediatric weight management. *Paediatr Child Health*. 2015;20(4):179-184.
33. Sturgiss EA, O'Brien K, Elmitt N, et al. Obesity management in primary care: systematic review exploring the influence of therapeutic alliance. *Fam Pract*. 2021;38(5):644-653.
34. Sturgiss EA, Sargent GM, Haesler E, Rieger E, Douglas K. Therapeutic Alliance and obesity management in primary care - a cross-sectional pilot using the working Alliance inventory. *Clin Obes*. 2016;6(6):376-379.
35. Haqq AM, Kebbe M, Tan Q, Manco M, Salas XR. Complexity and stigma of pediatric obesity. *Child Obes*. 2021;17(4):229-240.
36. Tomiyama AJ, Carr D, Granberg EM, et al. How and why weight stigma drives the obesity 'epidemic' and harms health. *BMC Med*. 2018;16(1):123.
37. Phelan SM, Burgess DJ, Yeazel MW, Hellerstedt WL, Griffin JM, van Ryn M. Impact of weight bias and stigma on quality of care and outcomes for patients with obesity. *Obes Rev*. 2015;16(4):319-326.
38. Farnesi BC, Perez A, Holt NL, et al. Continued attendance for paediatric weight management: a multicentre, qualitative study of parents' reasons and facilitators. *Clin Obes*. 2019;9(3):e12304.

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