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REGULAR ARTICLE

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No additional long-term effect of group vs individual family intervention in the treatment of childhood obesity—A randomised trial

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Abstract

Aim: Long-term evaluations of childhood obesity treatments are needed. We examined changes in weight and cardiometabolic risk 1 year after children completed individual family or group-based weight management interventions.

Methods: In 2009-2010, 6- to 12-year-old children with overweight or obesity from Finnmark and Troms (Norway) were recruited after media coverage and randomised to 24 months of individual family (n = 49) or group intervention (n = 48). Individual family intervention included counselling by a paediatric hospital team and a public health nurse in the local community. Group intervention included meetings with other families and a multidisciplinary hospital team, weekly physical activity sessions and a family camp. The primary outcome body mass index (BMI) and cardiometabolic risk factors were analysed 12 months after intervention.

Results: From baseline to 36 months, children's BMI increased 3.0 kg/m^2 in individual family and 2.1 kg/m^2 in group intervention (between-group −0.9kg/m^2, P = 0.096). Data were available from 62 children (64%). Between-group differences in C peptide (P = 0.01) were detected in favour of group intervention. Pooled data from both treatment groups showed continued decrease in BMI standard deviation score (P < 0.001).

Conclusion: No between-group difference in BMI was observed 12 months after intervention. Both groups combined showed sustained decrease in BMI standard deviation score.

KEYWORDS
child healthcare centre, clinical trial, multidisciplinary treatment, overweight, paediatric obesity

Abbreviations: BMI, body mass index; CI, confidence interval; CRF, cardiorespiratory fitness; HOMA, homeostatic model assessment; PA, physical activity; SDS, standard deviation score.

Trial registration: www.clinicaltrials.gov (NCT00872807)

1 | INTRODUCTION

Childhood obesity is associated with physical and psychological comorbidities including adverse cardiometabolic outcomes such as high blood pressure, dyslipidaemia and insulin resistance.[1] Given these health risks, there is a need for accessible and effective weight management interventions for children with obesity and their families.[2]

Modest improvements in weight and weight-related outcomes have been reported often in response to multicomponent lifestyle interventions for treating childhood obesity.[3,4] A review of lifestyle interventions indicated that immediate post-intervention improvements in children's weight status were not sustained over long term.[3] Additionally, changes in health outcomes other than weight status as cardiometabolic risk factors have been reported infrequently.[3] This shortcomings highlight the need for long-term follow-up data from paediatric weight management interventions, with outcomes that include both weight and weight-related measures.

Group-based treatment of childhood obesity may be more effective than individual approaches. A review suggested an enhanced effect of group interventions at 6-month follow-up, but there was no evidence for longer-term effects.[4] Although health services in the primary care setting are important for detecting and managing obesity and its complications,[2] there is little evidence of effective primary care interventions.[5] The most effective weight management interventions include frequent interactions between multidisciplinary teams and families, which differ from how most primary care visits are conducted. There is a need to extend existing evidence and test novel interventions that are feasible in primary care.

The Finnmark Activity School trial was a collaborative initiative across primary and specialist care settings. The study was designed to treat childhood obesity in a region of Norway with high prevalence of overweight and obesity in children and cardiovascular disease in adults and limited staff resources in the healthcare system.[6,7] The trial tested a more conventional individual family intervention vs a multicomponent group-based intervention. Follow-up at 24 months immediate after intervention showed no between-group difference in body mass index (BMI) but a modest between-group effect in BMI standard deviation score (SDS) and waist circumference in favour of group intervention.[8] When data from both groups were pooled, we found improvements in BMI SDS and psychological outcomes at 24 months.

The objective of the current report was to examine changes in BMI and cardiometabolic risk factors 12 months after completing the 24-month intervention to investigate whether long-term changes in weight and weight-related outcomes were present.

Key notes

- Long-term data are needed to examine whether improvements experienced by children enrolled in weight management interventions are maintained over an extended period.
- An intensive group-based intervention did not outperform an individual family intervention 12 months after completion in terms of weight, small between-group effects in cardiometabolic outcomes were detected.
- Data from both groups combined revealed that body mass index standard deviation score continued to decrease 1 year after intervention

2 | MATERIALS AND METHODS

2.1 | Participants and settings

Our study was described previously.[9] The study was conducted at the Paediatric Department at Hammerfest Hospital, and participants were recruited through media coverage in 2009-2010. Altogether 97 children aged 6-12 years with overweight or obesity[10] from six municipalities in Finnmark county, and the municipality of Tromsø were randomised to individual family intervention or group intervention in a parallel design. The intervention lasted 24 months and was followed by a 12-month observational period that included no active treatment. The trial was designed, conducted and reported in accordance with the Consolidated Standards of Reporting Trials guidelines.

2.2 | Individual family and group intervention

The individual family intervention included counselling by a nurse, consultant physician and nutritionist at the paediatric outpatient clinic and follow-up by a public health nurse in the local community. The group intervention included an initial 3-day inpatient stay at the hospital with other families and a multidisciplinary team, individual and group-based follow-up visits by local public health nurses, weekly physical activity (PA) sessions in their local community and a 4-day family camp. Local coaches with experience in children's sports led the PA sessions. During the 24-month intervention period, children in individual family intervention were offered 11 hours of health-care provider contact while their peers in the group intervention were offered 119 hours of contact, which included 76 hours of PA sessions.[8] Families who requested more support after 24 months were recommended to contact their primary care provider for follow-up care, no additional intervention sessions were offered through the study.

2.3 | Study preparation and implementation

Chief municipal executives in each municipality and the chief executive officer at Finnmark Hospital Trust committed resources prior to the start of the intervention. All intervention-related visits occurred between families and healthcare providers in the local comm-based data collection visits at baseline, 3, 12, 24 and 36 months. Healthcare

providers from both the municipalities and the hospital participated in 4, 1.5 day training courses covering aspects of childhood obesity including genetics and biology, weight bias and counselling skills including Brief Solution Focused Method and Motivational Interviewing.[11-13] Providers from the local communities and multidisciplinary hospital team met quarterly (formally) and *ad hoc* by telephone and video conferences to address challenges, optimise intervention delivery and reinforce learning and networking.

2.4 | Primary and secondary outcomes

Body mass index kg/m^2 (primary outcome), waist circumference and cardiometabolic risk measurements (secondary outcomes) were collected at hospital visits at baseline, 3 (anthropometry only), 12, 24 and 36 months.

As previously described,[9] height, weight, waist circumference and pubertal status were measured by trained nurses. Primary outcome assessors were blinded to group allocation. BMI and BMI SDS were calculated using an online calculator that was based on British reference data.[14]

After a 5-minute seated rest, systolic and diastolic blood pressure were measured three times using Dinamap ProCare100 calibrated sphygmomanometer (Med-Electronics) and appropriate sized cuff while children remained in a supine position. The three values were averaged and classified according to percentile for height, gender and age in three categories: <90th percentile, ≥90th and <95th percentile and ≥95th percentile.[15]

Fasting blood samples were collected by biomedical engineers according to Finnmark Hospital Trust routine guidelines. Analyses of serum triglycerides, total cholesterol, HDL cholesterol and LDL cholesterol were performed by Siemens Advia Chemistry enzymatic methods (Siemens Healthineers) and serum glucose concentration by Siemens Advia Chemistry hexokinase method (Siemens Healthineers). Serum insulin and C peptide were analysed by Cobas 8000 electrochemiluminence (Roche Diagnostics) according to University Hospital of North Norway routine guidelines. Insulin resistance was estimated according to the homeostatic model assessment (HOMA): (insulin [μU/mL] × glucose [mmol/L])/22.5.[16]

Cardiorespiratory fitness (CRF) was measured with the validated Andersen intermittent running test (15 seconds running, 15 seconds resting) using a 20 m lane whereby children aimed to cover as long a distance as possible over 10 minutes.[17]

Z-scores ([observed value—baseline mean value]/baseline standard deviation) were computed for waist circumference, systolic blood pressure, serum triglycerides and HDL cholesterol, HOMA and CRF. High values reflect elevated cardiovascular risk, except for HDL cholesterol and CRF which were subtracted in the continuous cardiometabolic sum score defined as (Z systolic BP + Z triglycerides + Z HOMA − Z HDL cholesterol − Z CRF)/6.[18]

Metabolic syndrome was defined using the MEtSPed definition, which included overweight or obesity status according to Cole[10] plus ≥2 of the following four risk factors (systolic or diastolic BP ≥90th percentile, triglycerides ≥1.7 mmol/L, HDL cholesterol ≤1.03 mmol/L and fasting glucose ≥5.6 mmol/L).[15,19]

2.5 | Statistical analyses

Between-group differences at baseline were assessed by independent samples *t* test and Pearson's chi-square tests. The data were analysed in accordance with the original allocation independent of participation and according to intention-to-treat principles. Linear mixed models were used to compare time trends in the primary outcome BMI and the secondary cardiometabolic outcomes waist circumference, lipids, insulin, CRF and cardiometabolic sum score over five and four time points, respectively. The independent variables were group and time, where time was modelled with three or four indicator variables and cross product terms between each indicator variable of time and group. A significant group-by-time interaction indicated different time trends between the intervention groups. To control for possible dependencies between repeated measures a random intercept was included in the model. In secondary analyses, we adjusted for baseline values of the dependent variable. Between-group differences of time trends in binary variables were tested using mixed effects regression models for binary responses with a random intercept and with the same modelling of the independent variables as in the linear mixed models. Possible dependencies between repeated measures were controlled for by specifying a compound symmetry covariance matrix. Overall changes in binary variables (e.g., metabolic syndrome, BP ≥ 90th percentile) in both groups combined from baseline to 24 and 36 months were tested using Mc Nemar`s test. All analyses were performed using Stata version 14.2 (StataCorp). Two-sided $P < 0.05$ was considered statistically significant.

2.6 | Ethics

The Regional Committee for Medical and Health Research Ethics, Region North approved the study, and it was conducted in accordance with the Helsinki Declaration. The parents provided written informed consent, and all children ≥12 years gave their assent.

3 | RESULTS

Altogether, 62 out of 97 children randomised (64%) attended the end of follow-up visit at 36 months and 51 children (53%) provided blood samples (Figure 1). There were no significant differences in baseline variables between individual family intervention and group intervention except for total cholesterol. There were no overall or within-group differences in baseline BMI between those who attended vs those who did not attend 36-month follow-up (Table 1).

From baseline to 36 months BMI increased by 3.0 kg/m^2 in individual family and 2.1 kg/m^2 in group intervention, no between-group differences were detected (−0.9, 95% CI: −1.9 to 0.2 $P = 0.096$). Over this same period, BMI SDS decreased by −0.13 units in individual

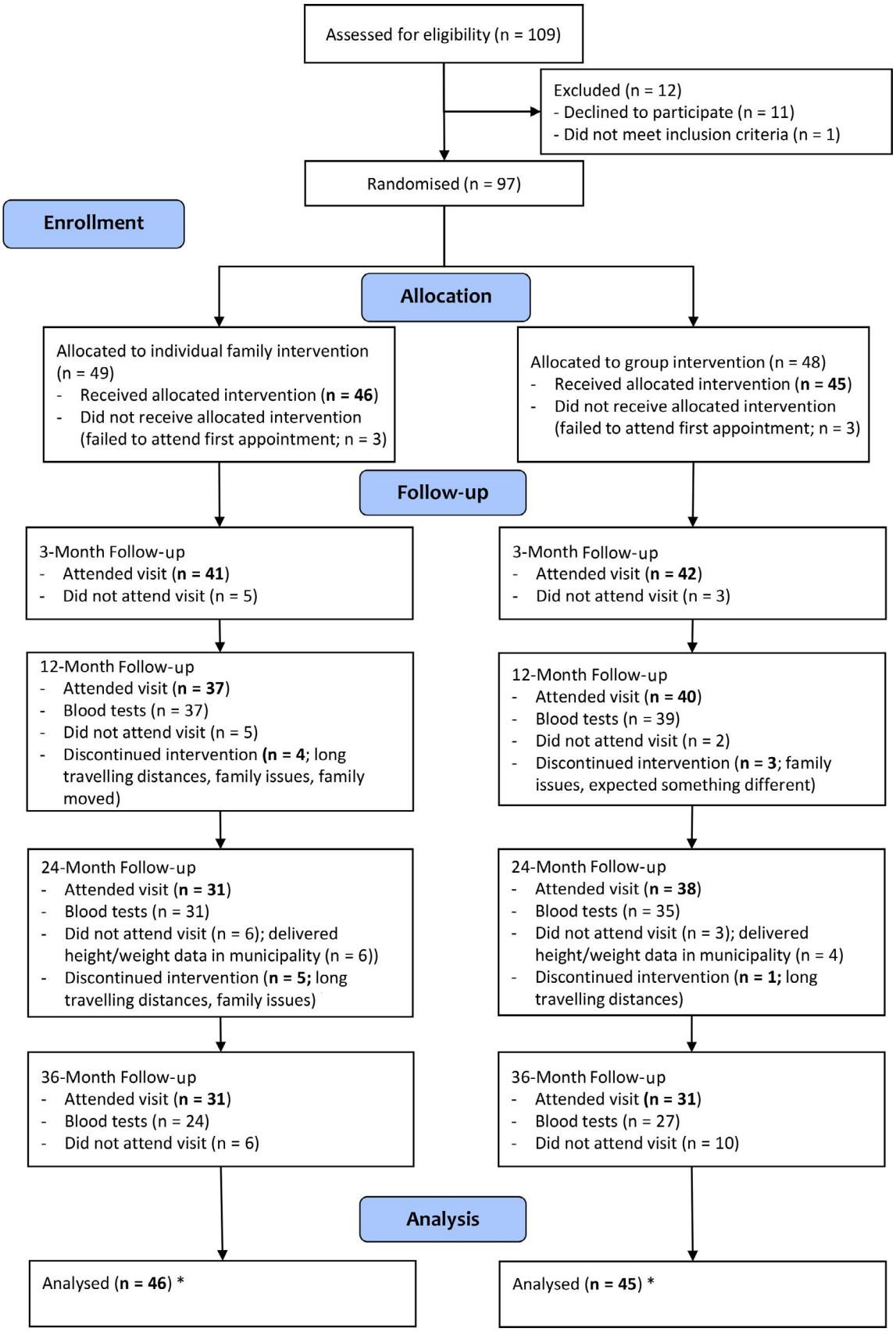

FIGURE 1 Flow of participants in the Finnmark Activity School. * Longitudinal analyses including all available data from participants up to the point of study withdrawal or completion

TABLE 1 Baseline characteristics of children enrolled in the Finnmark Activity School

Baseline characteristics	Individual family intervention n = 46	Group intervention n = 45	*P* value
Age (y)	10.5 ± 1.7	10.1 ± 1.7	0.24
Girls/Boys (n)	22/24	27/18	0.24
Tanner puberty stage ≥2 (n; %)	14 (31.1)	14 (32.6)	0.71
BMI (kg/m^2)	27.6 ± 4.3	26.9 ± 4.2	0.42
BMI SD score[a]	2.81 ± 0.60	2.76 ± 0.58	0.70
Waist circumference (cm)	89.2 ± 11.9	87.9 ± 12.0	0.62
Systolic blood pressure (mm Hg)	111.0 ± 11.8	113.3 ± 13.2	0.39
Blood pressure ≥90th percentile (n; %)	11 (23.9)	15 (33.3)	0.32
Serum (mmol/L)			
Triglycerides	1.12 ± 0.69	1.13 ± 0.68	0.91
Total cholesterol	4.96 ± 0.89	4.62 ± 0.66	0.04
HDL cholesterol	1.38 ± 0.32	1.26 ± 0.37	0.11
LDL cholesterol	3.30 ± 0.94	2.99 ± 0.59	0.06
Insulin (pmol/L)	97.9 ± 58.4	108.1 ± 77.5	0.50
Fasting glucose (mmol/L)	4.9 ± 0.3	5.0 ± 0.4	0.19
C peptide (pmol/L)	800.6 ± 347.0	894.3 ± 435.7	0.30
HOMA[b]	3.08 ± 1.83	3.52 ± 2.66	0.38
Cardiorespiratory fitness (m)[c]	631.9 ± 114.4	628.0 ± 143.4	0.89
Cardiometabolic sum score[d]	−0.02 ± 0.62	0.06 ± 0.71	0.58
Metabolic syndrome (n;%)[e]	3 (7)	11 (24)	0.07
Age among those met vs not met at 36 mo[f]	10.3 vs 10.7	10.1 vs 9.8	0.49, 0.45
Girls/Boys among those met vs not met at 36 mo	12/19 vs 10/5	19/12 vs 8/6	0.08, 0.80
BMI among those met vs not met at 36 mo	27.4 vs 28.0	27.0 vs 26.6	0.70, 0.80

Note: Baseline characteristics presented as mean ± standard deviation for continuous variables unless otherwise specified.
[a]BMI SD score according to British reference (Cole 1990).
[b]HOMA (homeostatic model assessment): Insulin (μU/mL) × glucose (mmol/L)/22.5.
[c]CRF measured using Andersen test, a 20 m run test [17].
[d]Continuous cardiometabolic sum score calculated based on systolic blood pressure, serum triglycerides, serum HDL cholesterol, HOMA and CRF.
[e]MetSPed definition: Overweight or obesity status (Cole 2000) plus ≥2 of 4 remaining risk component criteria (systolic or diastolic blood pressure ≥90th percentile, triglycerides ≥1.7 mmol/L, HDL cholesterol ≤1.03mmol/L and fasting glucose ≥5.6 mmol/L [26].
[f]Age, gender and BMI baseline among participants who met vs those who did not meet at 36-mo follow-up. *P*-values within single family and multiple family intervention, respectively.

family and by −0.24 units in group intervention (−0.11, 95% CI: −0.26 to 0.04, *P* = 0.15, Figure 2).

Between−group differences in change of HDL cholesterol (0.18 mmol/L, 95% CI: 0.05-0.32, *P* = 0.008), insulin (−44.0 pmol/L, 95% CI: −86.6 to −1.51, *P* = 0.04) and C peptide (−244.8 pmol, 95% CI: −441.7 to −48.0, *P* = 0.01) were detected in favour of group intervention at 36 months. Total cholesterol and LDL cholesterol decreased to a greater extent in individual family, between-group difference 0.46 mmol/L (95% CI: 0.16-0.77, *P* = 0.003) and 0.38 mmol/L (95% CI: 0.09-0.68, *P* = 0.012), respectively. Adjusting for baseline values did not change these results (Table 2, Figure 3). No between-group effects in the binary variables blood pressure and metabolic syndrome were detected.

Pooled data from the two groups combined showed a decrease in BMI SDS (−0.19, 95% CI: −0.27 to −0.11, *P* < 0.001), total cholesterol (−0.57 mmol/L, 95% CI: −0.73 to −0.42, *P* < 0.001), LDL cholesterol (−0.52 mmol/L, 95% CI: −0.67 to −0.37, *P* < 0.001) and an increase in CRF (151.6 m, 95% CI: 125.8-177.5, *P* < 0.001) from baseline to 36 months. There were no overall changes in prevalence of metabolic syndrome from baseline to 24 or 36 months. A significant

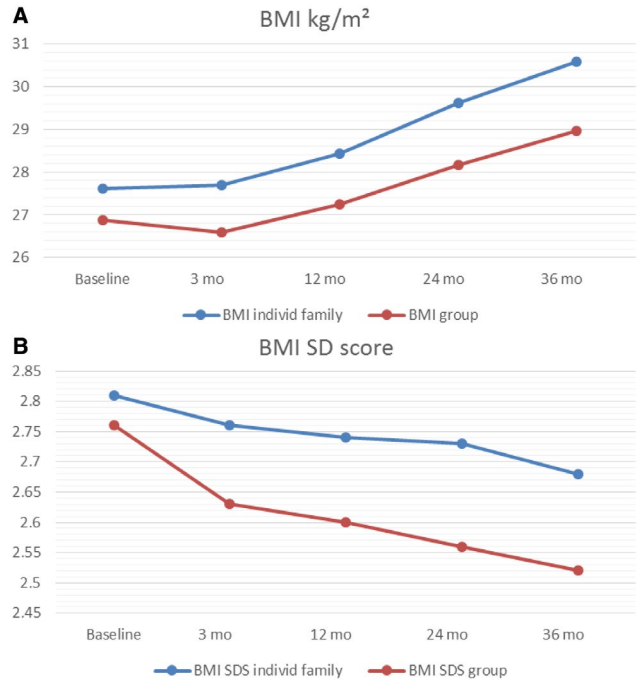

FIGURE 2 Mean BMI kg/m^2 and BMI SD score from baseline to 36-mo follow-up by intervention group, Finnmark Activity School

decrease in prevalence of systolic or diastolic BP ≥ 90th percentile was detected from baseline to 24 months (20/68 to 10/68, $P = 0.03$) and from baseline to 36 months (17/56 to 4/56, $P = 0.001$).

4 | DISCUSSION

We found no between-group differences in changes in BMI or BMI SDS at 36-month follow-up, 12 months after the end of individual family or group interventions. Smaller between-group effects in cardiometabolic risk factors were detected, mostly favouring group intervention. Results from analyses when data from both intervention groups were combined showed a continued decrease in BMI SDS, improvements in total and LDL cholesterol, CRF and prevalence of high blood pressure.

4.1 | Primary outcomes

Our findings implied that group-based, multidisciplinary treatment that included a large intervention dose did not outperform an individual family, low-intensity intervention 12 months after intervention with respect to BMI. These results contrast with previous reports

TABLE 2 Changes in cardiometabolic risk factors through 36 mo in individual family and group interventions; Finnmark Activity School

	Mean change from baseline (95 % confidence intervals)		Between-group difference	*P* value
	Individual family intervention	Group intervention	(95% confidence interval)	Group × Time[a]
Waist circumference (cm)				
3 mo	−0.02 (−1.81 to 1.77)	−1.46 (−3.23 to 0.31)	−1.44(−3.95 to 1.07)	0.26
12 mo	0.95 (−0.89 to 2.79)	−0.99 (−2.78 to 0.81)	−1.94 (−4.51 to 0.63)	0.14
24 mo	2.8 (0.80 to 4.80)	0.18 (−1.66 to 2.03)	−2.62 (−5.34 to 0.10)	0.06
36 mo	4.24 (2.20 to 6.29)	1.99 (−0.05 to 4.02)	−2.26 (−5.14 to 0.63)	0.13
Systolic blood pressure(mm Hg)				
12 mo	−0.6 (−3.9 to 2.7)	−1.4 (−4.6 to 1.8)	−0.8 (−5.4 to 3.7)	0.72
24 mo	0.6 (−2.9 to 4.1)	1.3 (−2.0 to 4.6)	0.7 (−4.1 to 5.5)	0.78
36 mo	3.2 (−0.4 to 6.9)	3.9 (0.4 to 7.6)	0.7 (−4.4 to 5.9)	0.78
Serum (mmol/L)				
Triglycerides				
12 mo	−0.11 (−0.34 to 0.12)	−0.04 (−0.27 to 0.18)	0.07 (−0.25 to 0.39)	0.68
24 mo	−0.07 (−0.32 to 0.17)	−0.06 (−0.30 to 0.17)	0.01 (−0.33 to 0.35)	0.1
36 mo	0.13 (−0.13 to 0.4)	0.08 (−0.18 to 0.34)	−0.05 (−0.42 to 0.32)	0.77
Total cholesterol				
12 mo	−0.31 (−0.50 to − 0.12)	−0.15 (−0.33 to 0.03)	0.16 (−0.10 to 0.42)	0.23
24 mo	−0.59 (−0.79 to − 0.39)	−0.32 (−0.51 to − 0.13)	0.27 (−0.01 to 0.54)	0.06
36 mo	−0.82 (−1.04 to − 0.60)	−0.36 (−0.56 to − 0.15)	0.46 (0.16 to 0.77)	0.003[e]
HDL cholesterol				
12 mo	−0.02 (−0.11 to 0.06)	0.04 (−0.05 to 0.12)	0.06 (−0.06 to 0.17)	0.33
24 mo	−0.03 (−0.12 to 0.05)	0.06 (−0.02 to 0.15)	0.1 (−0.03 to 0.22)	0.12
36 mo	−0.17 (−0.27 to − 0.07)	0.01 (−0.08 to 0.11)	0.18 (0.05 to 0.32)	0.008[e]

(Continues)

TABLE 2 (Continued)

	Mean change from baseline (95 % confidence intervals)		Between-group difference	*P* value
	Individual family intervention	Group intervention	(95% confidence interval)	Group × Time[a]
LDL cholesterol				
12 mo	−0.09 (−0.28 to 0.09)	0.08 (−0.10 to 0.26)	0.18 (−0.08 to 0.43)	0.18
24 mo	−0.38 (−0.58 to − 0.18)	−0.13 (−0.32 to 0.05)	0.24 (−0.03 to 0.51)	0.08
36 mo	−0.72 (−0.94 to − 0.50)	−0.34 (−0.54 to − 0.13)	0.38 (0.09 to 0.68)	0.02[e]
Fasting glucose (mmol/L)				
12 mo	−0.01 (−0.15 to 0.12)	−0.11 (−0.24 to 0.02)	−0.10 (− 0.29 to 0.09)	0.31
24 mo	−0.10 (−0.24 to 0.05)	−0.13 (−0.27 to 0.00)	−0.04 (−0.24 to 0.16)	0.7
36 mo	0.04 (−0.12 to 0.20)	−0.08 (−0.23 to 0.07)	−0.12 (−0.34 to 0.10)	0.28
Insulin (pmol/L)				
12 mo	0.8 (−25,4 to 27.0)	−2.2 (−28.1 to 23.7)	−3.0 (−39.9 to 33.9)	0.88
24 mo	14.7 (−13.1 to 42.5)	2.3 (−23.6 to 28.3)	−12.4 (−50.4 to 25.6)	0.52
36 mo	72.3 (41.3 to 103.2)	28.30 (−0.9 to 57.4)	−44.0 (−86.6 to − 1.5)	0.04[e]
C peptide (pmol/L)				
12 mo	32.9 (−89.1 to 155.0)	−8.3 (−133.5 to 117.0)	−41.2 (−216.1 to 133.7)	0.64
24 mo	130.8 (3.2 to 258.3)	11.8 (−113.7 to 137.2)	−119.0 (−297.9 to 59.9)	0.19
36 mo	362.8 (220.8 to 504.7)	117.9 (−18.4 to 254.3)	−244.9 (−441.7 to − 48.0)	0.01[e]
HOMA score[b]				
12 mo	0.01 (−0.88 to 0.90)	−0.21 (−1.09 to 0.68)	−0.22 (−1.48 to 1.04)	0.73
24 mo	0.42 (−0.53 to 1.37)	−0.05 (−0.93 to 0.84)	−0.47 (−1.77 to 0.83)	0.48
36 mo	2.33 (1.28 to 3.39)	0.89 (−0.11 to 1.88)	−1.44 (−2.90 to 0.01)	0.05
Cardiorespiratory fitness (m)[c]				
12 mo	53.3 (21.5 to 85.10)	83.1 (51.9 to 114.2)	29.8 (−14.7 to 74.3)	0.19
24 mo	94.7 (59.0 to 130.3)	133.6 (101.2 to 165.9)	38.9 (−9.3 to 87.0)	0.11
36 mo	130.9 (94.3 to 167.5)	171.5 (135.2 to 207.9)	40.6 (−10.9 to 99.2)	0.12
Cardiometabolic Sum score[d]				
12 mo	−0.088 (−0.251 to 0.076)	−0.215 (−0.373 to − 0.570)	−0.127 (−0.354 to 0.100)	0.27
24 mo	−0.047 (−0.231 to 0.137)	−0.188 (−0.346 to − 0.030)	−0.141 (−0.383 to 0.102)	0.26
36 mo	0.165 (−0.030 to 0.360)	−0.074 (−0.261 to 0.113)	−0.240 (−0.510 to 0.031)	0.08

Note: Data based on mixed models analysis unadjusted with single-family intervention as reference group.
[a]Effect estimates and *P*-values are from unadjusted linear mixed models.
[b]HOMA (homeostatic model assessment): Insulin (μU/mL) × glucose (mmol/L)/22.5.
[c]CRF measured with Andersen test, 20 m run test.[17]
[d]Continuous cardiometabolic sum score calculated from systolic blood pressure, triglycerides, HOMA, HDL cholesterol and CRF.
[e]Significant group by time effects remaining after adjustment for baseline values.

concerning differences in weight outcomes between moderate-to-high intensity and very low intensity interventions.[20] However, children in the individual family intervention received more than standard care or wait-list control group, which might have reduced our ability to detect between-group differences. Observational and clinical studies have reported increasing BMI SDS in children with overweight and obesity in the absence of treatment.[21,22] Significant reductions in BMI SDS in both individual family and group intervention may indicate that both interventions were beneficial, though being aware the limitations of generalising BMI SDS outcomes.[23] Nevertheless, these findings suggested that a lower intensity individual family intervention yielded sustainable weight changes in children with overweight and obesity.

4.2 | Cardiometabolic outcomes

The improvements in cardiometabolic risk factors were aligned with findings from a meta-analysis of lifestyle interventions that demonstrated favourable effects on systolic blood pressure, HDL cholesterol and triglycerides with decreasing BMI or weight.[24]

Cardiometabolic outcomes are less frequently reported in clinical trials in childhood obesity and lack of a universal definition of the metabolic syndrome in children makes it difficult to make comparisons between studies.[25]

The decrease in BMI SDS observed in our study was smaller in magnitude than the level ≥0.25 suggested by Ford et al according to British reference,[14,26] necessary to achieve significant improvements in cardiometabolic risk factors in children with obesity. Others showed that a smaller BMI SDS reduction (≥0.125) was associated with improved cardiovascular risk factors and suggested that even smaller improvements in weight status can lead to meaningful improvements in cardiometabolic risk factors.[27]

Because insulin resistance is an underlying metabolic abnormality of the metabolic syndrome and exercise increases insulin sensitivity, PA is considered a main therapeutic tool to treat metabolic syndrome in childhood.[28] In our study, insulin and C peptide increased among participants from 24 to 36 months, perhaps due to puberty or decreased PA after the 24-month intervention ended. However, we found an overall increase in CRF among participating children in this current study corresponding to approximately 7.5 mL/min/kg, 1.2 SD,[17] an improvement that may explain, in part, the improvements in cardiometabolic risk factors. The PA sessions offered in the group intervention may have contributed to the between-group differences in serum insulin and C peptide at 36 months; however, the multicomponent design of this intervention limits our ability to link specific interventions elements to outcomes. BMI does not distinguish weight associated with muscle vs fat and can explain why between-group effect in cardiometabolic outcomes may be observed despite absences of between-group differences in BMI, especially in interventions targeting PA.

4.3 | Pooled effects in weight status

Mean BMI SD score decreased in both intervention groups. This finding differs from other reports that failed to find sustainable improvements in weight-related outcomes after the period of active

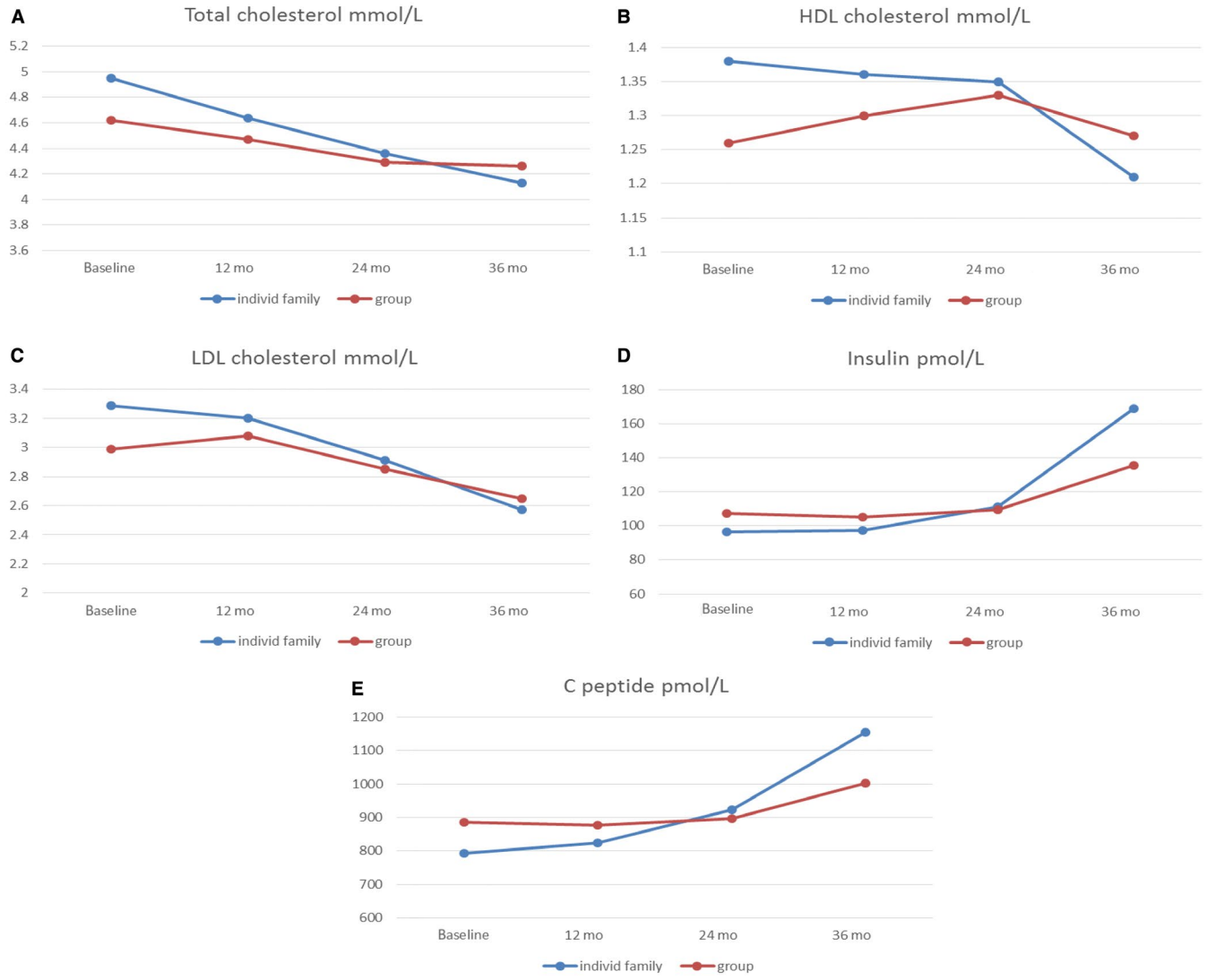

FIGURE 3 A-E, Mean serum total cholesterol, HDL cholesterol, LDL cholesterol, insulin and C peptide from baseline to 36-mo follow-up by intervention group, Finnmark Activity School

intervention.[3,29] We may speculate if the collaborative approach between our hospital and community team members (which were similar in both interventions) may have contributed to the beneficial effects on children's weight and cardiometabolic outcomes beyond the active intervention period. This hypothesis supports integration of clinical and community systems in order to address childhood obesity and its related chronic diseases.[2,30]

4.4 | Limitations

Despite study strengths including the randomised design and long-term follow-up, we acknowledge some study limitations. First, we were unable to retrieve follow-up data on 36% of our original sample. High drop-out is common in paediatric weight management, so we applied linear mixed models that assume missing data at random (MAR) and includes all available data at each time point in the main analyses. Although age, gender and BMI did not significantly differ between those who did vs those who did not contribute their data at 36 months, we cannot rule out the possibility that missing data may have biased our results. The possible effects of performing many statistical tests and multiple comparisons also needs to be taken into account when considering the statistical level and results.

The study was originally planned to compare an intensive multidisciplinary intervention with usual care. However, most families lived in small municipalities with only 1-2 public health nurses available and educational training had to be offered to all staff involved. The training may have contributed to individual family intervention receiving more support than expected in usual care. Finally, we observed larger variability in change in BMI than originally anticipated, leaving the study slightly underpowered to detect group differences, especially in the absence of a conventional control group.

The analysis of the between-group differences in BMI SDS should not be influenced by the reference population chosen. However, the comparison of pooled BMI SDS results across studies applying different reference populations must be interpreted with caution.

This trial was performed in collaborating local municipalities of various sizes from population 1000-70 000, and healthcare providers were participating as a part of their regular work. Although performed in the northernmost region of Norway, these findings should be applicable to other settings.

5 | CONCLUSION

There was no additional effect of an intensive group-based intervention compared with a low-intensive individual family programme in terms of BMI improvements with the chosen study design. However, improvements in cardiometabolic risk factors tended to be better in the group intervention. The extended decrease in BMI SDS in both groups at 36 months from baseline suggests long-term weight maintenance and overall improvements in cardiometabolic outcomes.

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CONFLICT OF INTEREST

The authors have no conflicts of interests to declare.

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