

This is an Accepted Manuscript version of the following article, accepted for publication in Journal of Sports Sciences:

Skrede, T., Aadland, E., Anderssen, S. A., Resaland, G. K., & Ekelund, U. (2021). Bi-directional prospective associations between sedentary time, physical activity and adiposity in 10-year old Norwegian children. *Journal of Sports Sciences*, 39(15), 1772-1779.
<https://doi.org/10.1080/02640414.2021.1898114>

1 Title: Bi-directional prospective associations between sedentary time, physical activity and
2 adiposity in 10-year old Norwegian children

3 Running title: Adiposity and activity – which direction?

4 Authors: Turid Skrede^{1,3}, Eivind Aadland¹, Sigmund Alfred Anderssen^{3,1}, Geir Kåre
5 Resaland² and Ulf Ekelund³

6 ¹Faculty of Education, Arts and Sports, Western Norway University of Applied Sciences,
7 Sogndal, Norway

8 ²Center for Physically Active Learning, Western Norway University of Applied Sciences,
9 Sogndal, Norway

10 ³Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway

11 Corresponding author: Turid Skrede (ORCID: 0000-0002-5418-6053), Faculty of Education,
12 Arts and Sports, Western Norway University of Applied Sciences, Sogndal, Norway.

13 E-mail: turid.skrede@hvl.no

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15 Funding information: The study was supported by grants from the Research Council of
16 Norway (grant number 221047/F40 and 249932/F20), the Gjensidige Foundation (grant
17 number 1042294), and the Western Norway University of Applied Sciences (formerly
18 credited as the Sogn og Fjordane University College).

19 Trial registration: Clinical Trial Registry: www.clinicaltrials.gov ID nr: NCT02132494

20 Keywords: accelerometer, MVPA, intensity, longitudinal, overweight

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22 Word count: 3,094

23 Background: There is an adverse cross-sectional association between sedentary time, physical
24 activity (PA) and adiposity, but weak and inconsistent estimates raise question to the direction
25 of associations. The present study aims to examine whether the prospective association
26 between sedentary time, different PA intensities and indicators of adiposity are bi-directional.

27 Methods: The Active Smarter Kids Study obtained data from 869 ten-year-old Norwegian
28 children with valid measurements for sedentary time, PA, and adiposity at baseline (Autumn
29 2014) and follow-up (Spring 2015). Time spent sedentary and PA was measured by
30 accelerometry, adiposity was assessed by three different measures: body mass index (BMI),
31 waist circumference (WC) and sum of four skinfolds (S4SF). Analyses are performed by
32 linear mixed models adjusted for sex, pubertal stage, birth weight, parental weight, parental
33 education, and baseline value of the outcome.

34 Results: Neither overall PA nor time spent sedentary predicted lower BMI or WC at follow-
35 up, but time spent in moderate-and-vigorous PA (MVPA) and vigorous PA (VPA) predicted
36 lower S4SF at follow-up among boys (MVPA β -0.066 [95% CI -0.105, -0.027] $p = 0.001$).
37 Baseline BMI and WC predicted less overall PA, MVPA and VPA in boys. All adiposity
38 measures predicted more time spent sedentary at follow-up in boys.

39 Conclusions: The results suggest that overall PA and sedentary time do not predict future
40 adiposity. Baseline adiposity may rather predict more sedentary time and less higher intensity
41 activity.

42 Introduction

43 Overweight and obesity among children is an unfavorable condition that adversely affects
44 almost every organ system (Daniels, 2009) and increases the risk of early onset cardiovascular
45 disease and type 2 diabetes (Maffeis & Tato, 2001). Reasons for overweight and obesity in
46 children include a complex mix of social, cultural, genetic, and behavioral factors (Ebbeling,
47 Pawlak, & Ludwig, 2002). At a fundamental level, however, weight gain occurs when energy
48 intake exceeds energy expenditure over time (Lee, Djousse, Sesso, Wang, & Buring, 2010).
49 Physical activity (PA) increases energy expenditure and is acknowledged as an important
50 modifiable factor for preventing weight gain (Han, Lawlor, & Kimm, 2010). Cross-sectional
51 studies report that increasing time spent in moderate-to-vigorous PA (MVPA) not only
52 reduces risk of obesity (Katzmarzyk et al., 2015; Mitchell et al., 2017), but also is inversely
53 associated with cardiometabolic risk factors (Renninger et al., 2020). On the other hand, time
54 spent in sedentary behaviors is strongly associated with weight gain (Ekelund et al., 2006;
55 Mitchell, Pate, Beets, & Nader, 2013), suggesting that sedentary time is a risk factor for
56 children's health (Saunders, Chaput, & Tremblay, 2014).

57 However, the cross-sectional associations between sedentary time, PA, and adiposity are not
58 straightforward due to lack of temporality between exposure and outcome. A recent
59 systematic review of prospective studies using objective PA measurement methods found no
60 association between baseline sedentary time and adiposity, and inconsistent association
61 between MVPA and adiposity at follow-up (Skrede, Steene-Johannessen, Anderssen,
62 Resaland, & Ekelund, 2019). Correspondingly, a few prospective studies have observed that
63 high body fat or body mass index (BMI) predicts a decrease in children's PA levels (Hjorth et
64 al., 2014; Kwon, Janz, Burns, & Levy, 2011; Metcalf et al., 2011; Tanaka et al., 2018) and
65 abdominal adiposity predicts more time spent in sedentary behaviors (Ekelund et al., 2012).
66 Thus, it is hypothesized that adiposity could be a determinant for PA, and physical inactivity

67 the result of adiposity rather than its cause (Ekelund, Brage, Besson, Sharp, & Wareham,
68 2008; Ekelund et al., 2017). Therefore, the aim of this study was to examine whether
69 sedentary time or higher intensity PA predict indicators of adiposity or vice versa using a
70 prospective study design in a sample of healthy 10-year-old Norwegian children.

71 Materials and methods

72 The study comprises data from the Active Smarter Kids study (ASK), a seven-month cluster-
73 randomized controlled school trial conducted during the school year of 2014–2015 in Western
74 Norway (Resaland et al., 2016). As PA levels did not differ from baseline to follow-up
75 between intervention and control schools (Resaland et al., 2016), data were pooled and
76 analyzed as a prospective observational cohort in the present study. The methods and
77 procedures relevant for the present study are presented in the following, and a detailed
78 description of the ASK study and sample size calculation have been previously published
79 (Resaland et al., 2015). All measurements were assessed at school sites from April–Oct 2014
80 and from April–June 2015. Children who provided valid measurements for all exposures and
81 outcomes (PA and adiposity) at both time points were included. Figure 1 shows the flow of
82 schools and children throughout the study. Our procedures and methods conformed to the
83 ethical guidelines defined by the World Medical Association’s Declaration of Helsinki and its
84 subsequent revisions. The Western Norway Regional Committee for Medical Research Ethics
85 committee in Norway approved the study protocol (Resaland et al., 2015). We obtained
86 written informed consent from each child’s parents or legal guardians and from the
87 responsible school authorities prior to all testing.

88 Physical activity and sedentary time were measured using GT3X/GT3X+ accelerometers
89 (ActiGraph, LLC, Pensacola, Florida, USA). All children were fitted with accelerometers at
90 the school sites and instructed to wear the accelerometers on their right hips for seven
91 consecutive days, except during water-based activities and while sleeping. Valid monitor
92 wear-time was defined as achieving ≥ 480 minutes day accumulated between 06:00 a.m. and
93 00:00 p.m. Continuous bouts of ≥ 20 minutes of zero counts were defined as non-wear time
94 (Esliger, Copeland, Barnes, & Tremblay, 2005). Children recording valid monitor wear time
95 for \geq four of seven days were included in the analyses. Sedentary time (< 100 counts per

96 minute [cpm]), MVPA ($\geq 2,296$ cpm), and vigorous PA (VPA; $\geq 4,012$ cpm) were defined
97 according to previously established and validated cut points (Evenson, Catellier, Gill, Ondrak,
98 & McMurray, 2008; Trost, Loprinzi, Moore, & Pfeiffer, 2011). All accelerometer data were
99 analyzed in 10-second epochs using the Kinesoft analytical software (KineSoft version 3.3.80,
100 Loughborough, UK).

101 Body weight was measured to the nearest 0.1 kg using an electronic scale (SECA 899, SECA
102 GmbH, Hamburg, Germany). Height was measured to the nearest 0.1 cm using a portable
103 altimeter (SECA 217, SECA GmbH, Hamburg, Germany). Body mass index (kg/m^2) was
104 calculated and children were categorized as being of normal weight, overweight, or obese
105 according to age-adjusted thresholds (Cole, Bellizzi, Flegal, & Dietz, 2000). Waist
106 circumference was measured with an ergonomic circumference measuring tape (SECA 201,
107 SECA GmbH, Hamburg, Germany). Two measurements were taken between the lowest rib
108 and the iliac crest with the abdomen relaxed at the end of a gentle expiration. If the two
109 results differed by > 1 cm, new measurements were taken until the two results were ≤ 1 cm
110 apart. The mean of the two nearest measurements was recorded and used in analyses.

111 Body fat was assessed by the sum of four skinfolds (S4SF), which is a reliable and valid
112 method for use in population-based studies in children (Silva et al., 2013). We collected
113 skinfold thickness from four sites (biceps, triceps, subscapular, and suprailiac) on the non-
114 dominant side of the body using a Harpenden skinfold caliper (Bull; British Indicators Ltd.,
115 West Sussex, England). We collected two measurements at each site in sequence. If the
116 difference between measures was > 2 mm, a third measurement was conducted. The mean of
117 the two nearest measurements was recorded and the S4SF in mm was used in analyses.

118 Trained test personnel performed all anthropometric measurements, but as skinfold
119 measurements are prone to high measurement error, we recruited seven persons specifically
120 trained to perform skinfold measurements.

121 Children self-assessed pubertal stage according to the Tanner method using a scale of color
122 images (stage 1–5) in a private room (Carel & Leger, 2008). Stage 2 marks the onset of
123 pubertal development. Parents self-reported their education levels, body weights, and their
124 respective children’s birth weights. Parental education level was used as an indicator of socio-
125 economic status (SES) and was categorized into three groups: low (< two years of high
126 school), middle (< four years of college/university), or high (\geq four years of
127 college/university).

128 Statistical analysis

129 Descriptive characteristics are presented as mean and standard deviation (SD), median and
130 interquartile range, or frequencies (%). All adiposity and PA variables except sedentary time
131 were log-transformed (ln) to improve the normality of the distributions. All adiposity and PA
132 variables were standardized to z scores for ease of interpretation; thus, all regression
133 coefficients are presented in SD units with confidence intervals (CI). First, we modelled the
134 prospective association between baseline MVPA, VPA, and sedentary time (exposure) and
135 the three different adiposity measures (outcome) in separate linear mixed models. The models
136 were adjusted for sex, accelerometer wear time, pubertal stage, parental SES and weight,
137 children’s birth weight, group allocation, and baseline value of the outcome. Second, we
138 modelled the prospective association between baseline adiposity (BMI, waist circumference
139 [WC], and skinfolds; exposure) and MVPA, VPA, and sedentary time (outcome), adjusted for
140 the same covariates as in the previous model and the baseline value of the outcome. We also
141 tested for interactions by sex (baseline exposure \times sex) and group allocation (group allocation
142 \times baseline PA). If a significant interaction ($P < 0.05$) was observed, the analyses were
143 additionally stratified. Lastly, BMI was split into the categories of normal weight versus
144 overweight/obese according to Cole et al. (2000), and MVPA was split into two groups based
145 on whether the current recommendations for PA for children – a mean of ≥ 60 min

146 MVPA/day – were achieved or not. Thereafter, we examined if BMI (normal weight vs.
147 overweight/obese) and MVPA (≥ 60 min MVPA/day) categories at baseline differed
148 regarding PA and adiposity outcomes respectively at follow-up. In all models, sedentary time
149 and PA variables were analyzed one by one to avoid multi-collinearity, and schools were
150 included as random intercepts to account for clustering within data.

151 Analyses were performed using the SPSS software, version 26 (IBM SPSS Statistics for
152 Windows, Armonk, NY: IBM Corp., USA). A p-value of < 0.05 indicated statistical
153 significance.

154 Results

155 Participants' characteristics at baseline are presented in Table 1. Of the 1,129 participants who
156 agreed to participate, only $n = 8$ children dropped out/withdrew during the study, and $n = 869$
157 children provided measurements for PA and adiposity at baseline and follow-up. Excluded
158 children (total $n = 253$) did not differ in any of the adiposity measures at baseline ($p \geq 0.280$),
159 but they had lower overall PA (cpm) at baseline ($p = 0.030$). At baseline, 77.4% were normal
160 weight, while 18.6% were categorized as overweight and 3.9% were obese. Mean
161 accelerometer wear time was 6.3 days (± 0.9) at baseline and 6.4 days (± 0.9) at follow-up.

162 Time spent sedentary, overall PA, or VPA did not predict lower adiposity at follow-up ($p \geq$
163 0.080) (Table 2), but there was an interaction for sex and MVPA ($p = 0.017$) for S4SF.

164 Moderate-to-vigorous PA predicted lower S4SF at follow-up among boys (MVPA β -0.066
165 [95% CI -0.105, -0.027] $p = 0.001$), but not among girls ($p = 0.889$).

166 All adiposity measures predicted a decrease in overall PA, MVPA, and VPA and a higher
167 amount of sedentary time at follow-up ($p < 0.043$) (Table 3). We observed significant
168 interactions by sex between all baseline adiposity measures and PA outcomes ($p < 0.048$), but
169 not for sedentary time ($p \geq 0.477$). Baseline BMI and WC predicted a decrease in overall PA,

170 MVPA, and VPA in boys ($p < 0.001$) but not in girls ($p \geq 0.112$). When S4SF was modelled
171 as the exposure, time spent in VPA was lower at follow-up in both girls ($\beta -0.098$ [95% CI -
172 0.194, -0.002] $p = 0.045$) and boys ($\beta -0.276$ [95% CI -0.372, -0.180] $p < 0.001$).

173 Lastly, we examined the associations according to weight status categories and whether
174 MVPA recommendations were met or not (Table 4). There was no difference in adiposity at
175 follow up between children categorized as sufficiently active or insufficiently active at
176 baseline ($p > 0.235$). Children who were overweight or obese according to cut points had
177 lower overall PA ($\beta -0.235$ [95% CI -0.405, -0.065] $p = 0.007$), MVPA ($\beta -0.199$ [95% CI -
178 0.347, -0.052] $p = 0.008$), and VPA at follow-up ($\beta -0.266$ [95% CI -0.423, -0.110] $p = 0.001$)
179 than normal-weight children. There was no difference between groups (normal-weight and
180 overweight/obese) in relation to sedentary time at follow-up.

181 Discussion

182 To our knowledge, this is the first study based on a larger sample and a longer follow-up time
183 than previous studies. The aim of this study was to examine the bi-directional prospective
184 associations between sedentary time, PA, and adiposity in children. At seven months follow-
185 up, both time spent sedentary and higher intensity PA predicted a lower BMI or WC. Baseline
186 MVPA predicted lower S4SF only in boys. All adiposity measures at baseline (BMI, WC, and
187 S4SF) predicted a decrease in overall PA, MVPA, and VPA in boys. All adiposity measures
188 at baseline predicted more time spent sedentary at follow-up in both girls and boys.

189 Our findings corroborate with the few studies examining bi-directional associations between
190 PA, sedentary time, and adiposity. In a study of 10-year-old Danish children, a higher fat
191 mass index at baseline was associated with less PA and more sedentary time at a six-month
192 follow-up (Hjorth et al., 2014). A meta-analysis from the International Children's
193 Accelerometer Database found that neither children's MVPA nor sedentary time were
194 associated with WC at follow-up, but baseline WC did predict more sedentary time two years
195 later (Ekelund et al., 2012). A study using the Mendelian Randomization approach to infer
196 putative causal relations between modifiable risk factors and disease suggested that adiposity
197 is a causal risk factor for lower overall PA and MVPA and increased sedentary time, but it
198 was not able to elucidate whether low PA may lead to increases in adiposity (Richmond et al.,
199 2014). Recently, data from the Gateshead Millennium Study examined a bi-directional
200 hypothesis and found that high body fat at age seven predicted less MVPA and VPA two
201 years later (Tanaka et al., 2018). It should be noted that the prospective associations were
202 based on modelling change versus change, which may be considered a "masked" cross-
203 sectional study. Nonetheless, these studies imply that PA and sedentary time do not predict
204 adiposity and instead support the hypothesis that the association could be in the opposite
205 direction.

206 The present study observed interaction in associations by sex. One possible explanation is that
207 boys with excess weight are less active than their peers (Page et al., 2005). Moreover, PA is
208 progressively lower across the weight spectrum in boys but consistently lower across all
209 weight categories in girls, indicating a continuous negative association between PA and
210 weight in boys (Purslow, Hill, Saxton, Corder, & Wardle, 2008). Indeed, the amount of
211 MVPA declines and sedentary time increases between age six and 11, but the decline in
212 MVPA is larger over time for those who are overweight and obese compared to ‘healthier’
213 children (Jago et al., 2020). In addition, boys were generally more active at baseline than girls
214 in the present sample (data not shown), which is well-known from other studies (Cooper et
215 al., 2015). Thus, a possible effect of the regression-to-the-mean phenomenon could be
216 present, meaning that those with high levels of PA at baseline potentially experience a larger
217 decrease in PA levels compared with those starting with lower PA levels (Collings et al.,
218 2015), and so the decrease in PA is likely to be greater in boys than in girls (Nader, Bradley,
219 Houts, McRitchie, & O'Brien, 2008). With time, these differences between boys and girls
220 might be attenuated, as recently published data from the Norwegian PA Surveillance System
221 show that the proportion of boys at age nine who meet PA recommendations decreased
222 between 2005 and 2018, but not the proportion of girls (Steene-Johannessen et al., 2019).

223 It is a common belief that the longitudinal reduction observed in PA levels, with its
224 subsequent increases in sedentary time, is responsible for weight gain. Our present findings
225 do not support this assumption. We found no differences in either BMI, WC, or S4SF at
226 follow-up when children who achieved the PA recommendations were compared to those that
227 did not achieve the PA recommendations at baseline. This does not mean that PA is an
228 ineffective strategy in tackling childhood overweight and obesity (Hallal et al., 2012). Current
229 recommendations for the minimum dose of PA are not specifically tailored to prevent weight
230 gain, but rather aimed at several health outcomes beyond adiposity, including

231 cardiorespiratory fitness, cardiometabolic risk factors, and bone and mental health (Janssen &
232 Leblanc, 2010; Renninger et al., 2020). Prevention is the best approach to counter the short
233 and long-term health problems of excess weight, and increased MVPA is suggested as a main
234 component in obesity prevention (Mitchell et al., 2017). However, the most effective
235 intervention is unknown (Han et al., 2010). Actions to solely increase PA level do not solve
236 the complex issue of overweight and obesity in children. Reasons for excess weight is a
237 complex figure including i.e. parental weight, pubertal status, SES, and sleep duration, but
238 unequivocally a result of long-term energy imbalances mainly resulting from dietary factors
239 (Lean, Astrup, & Roberts, 2018; Swinburn, Sacks, & Ravussin, 2009), with PA as an
240 important moderator of weight gain and maintenance (Swinburn et al., 2009).

241 Accelerometers are considered a criterion method for measuring PA, but are limited by
242 misclassification and underestimation of PA intensity (Brond & Arvidsson, 2016). Repeated
243 measurements of PA in the same individuals have substantial intra-individual variability with
244 an intraclass correlation of about 0.5, suggesting that PA levels are highly variable over time
245 (Aadland et al., 2017). Physical activity and sedentary time are measured less precisely than
246 BMI, WC, and skinfolds, and therefore, it is not surprising that baseline BMI and WC predict
247 PA at follow-up whereas, because of measurement error, the reverse may not be detectable
248 (Ekelund et al., 2008). Examining the direction of associations between exposure and
249 outcome measures with different degrees of measurement error is therefore problematic.
250 When a more imprecise variable is modelled as the outcome (i.e., PA), the magnitude of
251 effect is estimated accurately, but with wider CIs. In contrast, when a more imprecise variable
252 is modelled as the exposure, it tends to attenuate the regression coefficient (Hutcheon,
253 Chiolero, & Hanley, 2010). Increasing sample size, however, does not solve the issue and
254 may only result in a more precisely erroneous estimate of the effect size (Hutcheon et al.,
255 2010).

256 The main strengths of the current study are the use of objective measurements of PA, the use
257 of clinically relevant adiposity measures, and the use of prospective analyses with baseline
258 adjustments of the outcome. The short time frame between baseline and follow-up, however,
259 could raise questions as to whether the present observations are meaningful and have lasting
260 effects in either direction. Moreover, the temporal association between PA and adiposity
261 *before* our baseline measurements is also unknown. The traditional association between
262 baseline sedentary time, PA, and adiposity at follow-up could therefore be evident in studies
263 with longer durations (Hallal et al., 2012). Seven months is a fair amount of time in a child's
264 life, but the "short-term" observations could be stronger in magnitude if the study had a
265 longer follow-up. Repeated measurements of PA and more precise measures of adiposity (i.e.,
266 DEXA) over a longer period would allow additional modelling of the complex longitudinal
267 relationships between sedentary time, PA, and adiposity. Experimental and observational
268 studies using the Mendelian Randomization approach are needed to examine the causal
269 associations between sedentary time, PA, and adiposity in children. However, physical
270 activity interventions are unfortunately difficult to conduct given the long time it takes to
271 develop excess weight or achieve weight loss in children, combined with issues of cost and
272 compliance. Lastly, we cannot exclude the possibility that our observations are explained by
273 residual confounding, and the study was carried out in a rural county with most children being
274 Caucasian. Thus, the generalization of our results to other populations is at best limited. It
275 should be noted that we did not adjust for multiple testing, which may inflate type 1 error.
276 However, correction for multiple comparisons is debated, as it may increase the risk of type 2
277 error (Perneger, 1998).

278 Our results suggest that overall PA and sedentary time do not predict future adiposity in this
279 sample of 10-year-old children. Baseline adiposity rather predicts more sedentary time and

280 less high-intensity PA. Our findings were more pronounced among boys. Adiposity level
281 may therefore be a determinant of lower levels of physical activity.

282

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