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Compensatory Changes in Physical Activity and Sedentary Time in Children and Adolescents with Cystic Fibrosis

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1 **Compensatory Changes in Physical Activity and Sedentary Time in**
2 **Children and Adolescents with Cystic Fibrosis**

3

4 Physical activity (PA) is a key element in Cystic Fibrosis (CF) treatment strategies,
5 yet little is known as to whether activity compensation occurs. This study examined
6 whether PA and/or sedentary time on one day were temporally associated with time
7 spent in these intensities the following day in youth with CF. Time spent sedentary
8 and in different PA intensities were objectively-measured for seven consecutive days
9 in 50 youth (22 boys; 12.0±2.7 years); 25 with mild-to-moderate CF and 25 age- and
10 sex-matched controls. Multilevel analyses (day and child) were conducted using
11 generalised linear latent and mixed models. On any given day, every additional 10
12 minutes spent in sedentary time or moderate-to-vigorous physical activity (MVPA)
13 were associated with 1.9 (95%CI: -3.6 to -1.2) and 12.4 (95%CI: -22.1 to -2.9)
14 minutes less sedentary time the following day, respectively. These temporal
15 associations were also observed when split by group (3.1 vs. 1.9 minutes for healthy
16 and CF, respectively). These findings indicate that youth do not compensate their PA,
17 irrespective of disease status, between days, but may compensate their sedentary time
18 between days. Experimental studies are warranted to fully elucidate whether
19 compensatory responses to PA and sedentary time occur, which is fundamental for
20 informing PA promotion strategies.

21

22 **Keywords:** accelerometry; chronic disease; respiratory health; youth; paediatric

23

24 **Introduction**

25 Cystic fibrosis (CF), currently affecting over 10,000 people in the UK (Cystic
26 Fibrosis Trust, 2017), is the most prevalent inherited genetic disorder in the
27 Caucasian population (Quinton, 1990). CF is a multi-system disease, which
28 primarily affects the lungs and digestive system through mutations in the Cystic
29 Fibrosis Transmembrane Conductance Regulator (CFTR) gene that lead to
30 malfunctioning or absent CFTR proteins and impaired mucosal clearance
31 mechanisms (Davies, Alton, & Bush, 2007; National Institute for Health and Care
32 Excellence, 2017; Ratjen, 2009). Despite therapeutic advances and an increased life
33 expectancy to 47 years for those born in 2017 (Cystic Fibrosis Trust, 2017), CF
34 remains incurable, highlighting the need to enhance patient well-being.

35 Regular participation in physical activity (PA) is an important component in
36 the therapeutic management of individuals with CF (Williams & Stevens, 2013) and
37 is recommended in internationally-recognised guidelines, irrespective of age or
38 disease severity (Radtke, Nevitt, Hebestreit, & Kriemler, 2017; Smyth et al., 2014).
39 In addition to the extensive benefits of leading an active lifestyle in healthy
40 populations (Janssen & Leblanc, 2010), being active elicits further health benefits for
41 those with CF (Hulzebos, Dadema, & Takken, 2013; National Institute for Health
42 and Care Excellence, 2017; Radtke et al., 2017). Specifically, high levels of PA may
43 lead to improved airway clearance by increasing transepithelial fluid transport and
44 reduce, or even prevent, lung function decline (Schneiderman et al., 2014).
45 Furthermore, PA and exercise improve aerobic capacity (Selvadurai et al., 2002),
46 bone mineral density (Buntain et al., 2004), quality of life (Selvadurai, Blimkie,
47 Cooper, Mellis, & Van Asperen, 2004), and ion channel function (Hebestreit,

48 Kersting, Basler, Jeschke, & Hebestreit, 2001), which not only improves mucus
49 hydration and clearance, but reduces hospital admissions (Wilkes et al., 2007).

50 Despite the benefits of PA for youth with CF, few engage in moderate-to-
51 vigorous physical activity (MVPA) for 60 minutes every day (Aznar et al., 2014;
52 Mackintosh, Ridgers, Evans, & McNarry, 2018), highlighting the need to develop
53 and implement population-specific PA intervention strategies. However, a significant
54 challenge for CF multi-disciplinary teams is how to promote and encourage MVPA
55 (Cox, Alison, & Holland, 2013) and how to ensure sustained behavior change is
56 achieved. It has been hypothesised that even if interventions are successful at
57 increasing children's PA levels on one day, they may decrease their PA levels on
58 subsequent days to compensate (i.e., the "activitystat" hypothesis; Dale, Corbin, &
59 Dale, 2000; Rowland, 1998; Wilkin, Mallam, Metcalf, Jeffery, & Voss, 2006). Such
60 compensation is thought to occur due to the central nervous system's homeostatic
61 regulation of total PA but the existence and nature of an activitystat in healthy
62 populations has been debated (Gomersall, Rowlands, English, Maher, & Olds, 2013)
63 and little is known about the presence of such behaviours in clinical populations.
64 Specifically, as the activitystat may be considered a homeostatic mechanism, and
65 given the treatment burden and elevated energetic costs in those with CF, one may
66 postulate that they are more likely to display compensatory behaviours than their
67 healthy counterparts (Ridgers, Barnett, et al., 2018). Indeed, even the set-point itself
68 may differ to healthy counterparts, therefore, identifying compensatory behaviours
69 could provide critical information for treatment strategies and on-going care.

70 The aim of the current study was therefore to examine whether PA and
71 sedentary time on one day are temporally associated with their activity levels on the

72 following day in children and adolescents with mild-to-moderate CF compared to
73 age- and sex-matched healthy controls.

74

75 **Materials and Methods**

76 *Participants*

77 Twenty-five children and adolescents aged 7 to 17 years with mild-to-moderate CF,
78 confirmed by a sweat chloride $>60 \text{ mmol}\cdot\text{l}^{-1}$ and genotyping (11 Homozygote; 14
79 Heterozygote; 3 CF-related liver disease; 1 CF-related diabetes), were recruited from
80 a UK CF outpatient clinic. Children with CF were eligible to participate if they had
81 no increase in symptoms or weight loss two weeks prior to testing and had a stable
82 lung function (defined as within 10% of their best in the previous six months).
83 Unstable non-pulmonary comorbidities or acute infections warranted exclusion. All
84 participants with CF were instructed to continue routine prescribed medications.
85 Twenty-five age- and sex-matched apparently healthy counterparts were recruited
86 from local schools. Ethical approval was granted by the Bromley NHS research
87 ethics committee (REC reference: 13/LO/1907). Written informed consent and
88 assent were obtained from parents/guardians and participants, respectively.

89

90 *Measures*

91 *Anthropometry:*

92 Body mass (Seca 220; Hamburg, Germany) and standing and seated stature (Seca
93 220; Hamburg, Germany) were measured to the nearest 0.01 kg and 0.01 m,
94 respectively. Maturity offset was subsequently estimated as years from peak height
95 velocity using the equations developed by Mirwald et al. (2002). For those with CF,
96 all data was collected during a routine visit to the clinic; healthy age- and sex-

97 matched counterparts were asked to attend one laboratory session. All measures were
98 taken by trained staff using standardised procedures.

99

100 *Lung Function:*

101 Forced vital capacity (FVC) and forced expiratory volume in 1s (FEV₁) were
102 assessed using flow-volume loop spirometry (Vitalograph, UK), with the best of
103 three consistent exhalations (<5% variability) used in further analyses. All
104 participants were thoroughly familiarised with the manoeuvre and undertook practice
105 attempts prior to those considered for inclusion. All lung function measurements
106 were expressed as percentage predicted normal according to appropriate reference
107 data (Stanojevic et al., 2009).

108

109 *Physical Activity and Sedentary Time:*

110 Physical activity and sedentary time were measured at 100 Hz using a hip-mounted
111 ActiGraph GT3X+ accelerometer (ActiGraph LLC, Pensacola, FL). Participants
112 were instructed to wear the monitor for seven consecutive days and advised to
113 remove it for water-based activities (e.g., bathing, swimming) or contact sports. Data
114 were downloaded using ActiLife software (v6.10.4; ActiGraph LLC), processed into
115 15-second epochs, and reduced using a customised Excel macro. Non-wear time was
116 defined as intervals with at least 20 minutes of consecutive zero's, which is
117 commonly used in youth studies examining compensation (Ridgers, Lamb,
118 Timperio, Brown, & Salmon, 2018; Ridgers, Timperio, Cerin, & Salmon, 2015).
119 Sedentary time was defined as 100 counts·min⁻¹ (Ridgers et al., 2012), with time
120 spent in moderate- (MPA; 4-5.99 METs) and vigorous-intensity (VPA; ≥6 METs)
121 physical activity determined using age-adjusted cut-points (Freedson, Pober, & Janz,

122 2005). MPA and VPA were summed to obtain MVPA. Time spent in light-intensity
123 physical activity (LPA) was defined as >100 counts·min⁻¹ to the MPA cut-point. A
124 valid day was defined as at least 9 hours of wear time. Participants with at least 3
125 valid days of data, irrespective of week or weekend day, were included in the
126 analyses (Mattocks et al., 2008).

127

128 *Statistical Analyses*

129 All statistical analyses were conducted using Stata SE v15 (StataCorp, Texas, USA).
130 Independent t-tests were conducted to examine differences between participants who
131 were included and excluded from the analyses and between the CF and healthy
132 groups for all descriptive variables (mean \pm SD).

133 Multilevel analyses were performed using generalised linear latent and mixed
134 models (GLLAMM). This approach accounted for the nested nature of the data
135 arising from multiple days of accelerometer measurements within the same
136 participant (Twisk, 2006). A two-level model was used in all analyses, namely day
137 (level 1) and participant (level 2). GLLAMM was used to estimate associations
138 between temporally adjacent values (i.e., pairs of days) for the outcome variables
139 whilst adjusting for person-level (overall daily mean) sedentary time and/or PA, as
140 appropriate. As GLLAMM can separate participant-level from day-level effects (i.e.,
141 within-person changes), these analyses are a more appropriate measure of
142 compensatory changes (Ridgers, Timperio, Cerin, & Salmon, 2014). The analyses
143 examined whether participants activity on one day (day d in the model) was
144 associated with their activity from the previous day (day $d-1$ in the model). As data
145 were collected over seven consecutive days, each participant provided a maximum of
146 six data points for analysis. In all models, the random structure considered random

147 intercepts and slopes at the participant level. GLLAMMs were initially performed
148 for the whole sample and adjusted for sex, age, day of measurement, group (CF and
149 healthy), monitor wear time on a given day, and person-level PA and/or sedentary
150 time, as appropriate. To examine whether these associations differed between
151 groups, the analyses were subsequently performed for the CF and healthy groups
152 separately. A two-tailed probability level of 0.05 was used for all analyses.

153

154 **Results**

155 Forty-three participants (24 boys, 19 girls; 21 CF, 22 healthy controls) met wear time
156 criteria and were included in the analyses. There were no significant differences in
157 the descriptive data of the included or excluded participants, and both CF and
158 healthy participants engaged in similar levels of sedentary time and PA (Table 1). On
159 average, participants provided 6.1 ± 0.9 valid days for analysis.

160

161 ****Table 1 near here****

162

163 The associations between temporally adjacent values for PA and sedentary
164 time for the whole sample are shown in Table 2. On any given day, every additional
165 10 minutes spent sedentary was associated with 2.1 minutes less sedentary time the
166 following day (95% CI: -3.6 to -1.2). In contrast, on any given day, each additional
167 10 minutes spent in MVPA was associated with 12.4 minutes less sedentary time the
168 following day (95% CI: -22.1 to -2.9).

169

170 ****Table 2 near here****

171

172 The associations between temporally adjacent values for PA and/or sedentary
173 time for the healthy and CF groups are shown in Tables 3 and 4, respectively. Two
174 statistically significant associations were observed for the healthy group, and one
175 observed for the CF group. Specifically, on any given day, each additional 10
176 minutes spent sedentary was associated with 3.1 (95% CI: -5.3 to -1.0) and 1.9 (95%
177 CI: -3.6 to -0.3) minutes less sedentary time in the healthy and CF groups,
178 respectively. Lastly, on any given day, each additional 10 minutes that participants in
179 the healthy group engaged in MVPA was associated with 18.1 minutes less
180 sedentary time the following day (95% CI: -33.8 to -2.4).

181

182 ****Tables 3 and 4 near here****

183

184 **Discussion**

185 This study examined whether increased PA levels or sedentary time on any given
186 day were temporally associated with these behaviours the following day in children
187 and adolescents with CF and age- and sex-matched healthy controls. Youth,
188 regardless of condition, do not appear to compensate for increased PA, of any
189 intensity, but partially compensated for time spent being sedentary. Furthermore,
190 findings suggest that increased MVPA on any given day was associated with
191 decreased sedentary time the following day. However, this effect was not observed
192 for those with CF when independent group models were utilised.

193 No associations were found between time spent in PA on any given day and
194 PA on a subsequent day, irrespective of intensity or condition. This suggests that
195 youths do not compensate for increased PA levels on one day by decreasing their PA
196 levels the following day. Whilst this is not consistent with the “activitystat”

197 hypothesis (Rowland, 1998) and contrasts previous research that used the same
198 analytical approach in healthy children (Ridgers, Barnett, et al., 2018; Ridgers et al.,
199 2014), the present findings are consistent with others who reported that healthy
200 youths do not compensate between days (Baggett et al., 2010; Goodman, Mackett, &
201 Paskins, 2011). Such discrepancies may, at least in part, be attributed to
202 methodological differences in PA measurement analyses (e.g., wear time criteria),
203 thereby limiting inter-study comparability. Nonetheless, a possible explanation may
204 be that populations, or indeed individuals, compensate total PA levels in different
205 ways, though there is a lack of consensus regarding the time frame over which this
206 occurs. Specifically, some research suggests that youths can tolerate discrete bouts of
207 activity (Ridgers, Lamb, et al., 2018), whilst others have demonstrated *within-day*
208 compensation (Ridgers et al., 2015). Gomersall et al. (2013) contend that it is
209 unlikely to be demonstrated on a day-to-day basis, citing previous interventions that
210 have reported compensatory responses which ranged from approximately 1 to 3
211 months, due to the period of the homeostatic regulatory response (Baggett et al.,
212 2010; Goodman et al., 2011). Indeed, it may be that compensatory effects take
213 longer to emerge in clinical populations such as those with CF and that even the
214 specific innate ‘set-point’ may vary between individuals depending on biological,
215 psychosocial and physical environment-related factors (Eisenmann & Wickel, 2009;
216 Rowland, 1998). It is also important to highlight that the intensity of physical
217 activity may be a key determinant in whether compensatory responses are observed,
218 although this remains to be elucidated, along with the potential interaction between
219 an activitystat and health and environmental-related parameters.

220 In this study, temporal associations were found between time spent in MVPA
221 on any given day and time spent being sedentary the subsequent day. Specifically,

222 for every additional ten minutes spent in MVPA, children and adolescents engaged
223 in 12.1 minutes less sedentary time the following day, although this was only
224 statistically significant in the healthy youth. Whilst this may be a spurious finding as
225 suggested in a previous study (Ridgers et al., 2014), if replicable, the current results
226 refute the notion of a compensatory PA response, irrespective of clinical condition.
227 In contrast, the present study suggests that youth may demonstrate a regulatory
228 homeostatic mechanism for sitting time (i.e., a “sedostat”; Olds, Maher, Ridley, &
229 Kittel, 2010). Specifically, for every ten additional minutes spent being sedentary on
230 any given day, youth spent 2.1 minutes less time being sedentary the following day.
231 Caution is required when interpreting this potential compensatory response given the
232 small sample size and magnitude of the response. Furthermore, the day-to-day
233 variability in sedentary time largely remains to be determined, although the limited
234 evidence available appears to suggest that sedentary behaviours are fairly stable
235 (Basterfield, Adamson, Pearce, & Reilly, 2011). However, it is nonetheless
236 interesting to note that the magnitude of compensation was greater in healthy
237 participants compared to those with CF. Given that previous research has questioned
238 whether such homeostatic mechanisms are physiological or predominantly reflect
239 environmental and sociological elements (Epstein, Paluch, Kilanowski, & Raynor,
240 2004), the partial compensation in those with CF could be due to their high treatment
241 burden, inhibiting time available for a greater compensatory response. It is therefore
242 possible that the exhibited compensation may not be evidence of an activitystat, or
243 even a sedostat, *per se*. Nonetheless, given that daily treatment duration remains
244 relatively consistent, excluding treatment associated with exacerbations, it could be
245 argued that activity opportunities for those with CF also remain constant. It could
246 therefore be postulated that healthy populations are less tolerant to increased

247 sedentary time, or those with CF have a smaller relative amount of time to
248 compensate. Future research is required to verify the apparent disease-related
249 differences observed in the present study; future studies may wish to consider
250 moderator analyses to help further elucidate these differences.

251 Additionally, or perhaps alternatively, discrepancies between health
252 conditions may be related to the cut-points used to define activity intensities. Whilst
253 the cut-points utilised have demonstrated acceptable classification accuracy in youth
254 (Troost, Loprinzi, Moore, & Pfeiffer, 2011), the lack of cut-points developed and
255 validated for CF populations may have resulted in misclassifications of time spent in
256 relative intensities (Mackintosh et al., 2018). However, it should be noted that there
257 were no significant differences in PA levels between those with CF and their age-
258 and sex-matched healthy controls. Indeed, 47.6% of our CF population met
259 recommended guidelines, which is a considerably higher proportion than previously
260 reported (Aznar et al., 2014). It could be speculated that greater compensatory
261 responses would have been apparent in less active individuals, who may have a
262 lower innate activity level and thus an increase of 10 minutes MVPA would be a
263 greater percentage of their overall daily activity levels. Alternatively, more active
264 youth may have a greater tolerance around the ‘set-point’. However, Ridgers et al.
265 (2018) found few variables which moderated such compensatory effects. Although
266 research suggests that youths may be able to tolerate isolated increases in PA
267 (Ridgers, Lamb, et al., 2018), the threshold required to trigger a compensatory
268 response is presently unknown. It could be postulated that the reduced exercise
269 capacity characteristic of the CF population may lower such a threshold, though
270 further experimental work is required to identify the smallest meaningful

271 compensatory response. Such research should account for factors such as fitness and
272 lung function (FEV₁), which may moderate any compensatory responses.

273 No other studies have investigated the activitystat hypothesis in those with
274 CF, thereby precluding specific population comparisons. In healthy populations, the
275 literature is dominated by observational studies, which have reported mixed findings;
276 few experimental studies have been conducted. This lack of consensus regarding
277 compensatory changes may be explained, at least in part, by inter-study
278 methodological differences, including, but not limited to, previous studies failing to
279 account for person-level variation in responses. Indeed, Selvadurai et al. (2004)
280 reported significant influences of maturity on PA levels in those with CF, and a
281 failure to account for this, as well as disease severity, may limit interpretation.
282 Nonetheless, the concept of a biological control of PA and the activitystat for those
283 with CF has significant implications; given the numerous additional health benefits
284 for those with CF (National Institute for Health and Care Excellence, 2017;
285 Schneiderman et al., 2014), over and above those identified in healthy children
286 (Janssen & Leblanc, 2010), the present study highlights that it may be feasible to
287 increase total physical activity without compensatory responses to discrete bouts of
288 PA.

289 The present findings have potential implications for the development and
290 delivery of PA interventions for youth with CF. Whilst specific PA and exercise
291 recommendations remain in their infancy, HIIT has been specifically identified as a
292 potential strategy to enhance PA levels in those with CF (Cox & Holland, 2017).
293 However, Kriemler et al. (1999) suggest that high-intensity exercise may be
294 associated with compensation when other intensities are not, questioning the
295 potential applicability of this exercise modality to increasing overall physical activity

296 levels. Future studies should therefore specifically examine whether compensation
297 occurs as a result of participating in prescribed PA or exercise interventions and its
298 interaction with exercise intensity. Moreover, such research should seek to measure
299 total energy expenditure, as well as total PA levels, as, whilst PA is likely to be the
300 predominant contributory factor to total energy expenditure, it could be argued that
301 disease severity may have significant implications on energy expenditure. Indeed,
302 Gomersall et al. (2013) identified that researchers should clearly distinguish between
303 an ‘activitystat’ and ‘energystat’.

304 This is the first study to examine compensatory changes in objectively-
305 measured PA levels and sedentary time between days in children and adolescents
306 with CF. Nonetheless, several limitations should be acknowledged and the present
307 results should be interpreted with caution. Specifically, this study was observational
308 in nature; future experimental research should aim to increase or decrease PA during
309 specific periods of inactivity or activity, respectively, to ascertain *if*, and indeed
310 *when*, compensatory effects occur (Ridgers, Lamb, et al., 2018). The sample size
311 also restricted the number of person-level variables that could be investigated, which
312 may potentially moderate the compensatory effect (Ridgers, Barnett, et al., 2018).
313 Furthermore, given that no CF-specific cut-points are currently available, those
314 developed for healthy children were applied to those with CF. This may have
315 resulted in the misclassification of sedentary time and activity intensities which
316 limits the interpretation of the present results. It is also pertinent to note that further
317 research is required to identify the duration required to classify as clinically
318 meaningful compensatory behaviour.

319

320 **Conclusions**

321 In conclusion, these findings are inconsistent with the activitystat hypothesis,
322 regardless of population. However, youth may compensate for sedentary time, albeit
323 to a lesser extent in those with CF. These findings highlight that it may be possible to
324 enhance PA levels in youth CF populations, without concomitant increases in
325 deleterious sedentary time.

326

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334

335

336 **Declaration of Interest**

337 The authors have no competing interests to declare.

338

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466

467

468 **Table 1: Participant characteristics and activity levels (mean \pm SD)**

	Whole Sample	Cystic Fibrosis	Healthy Controls
	<i>n = 43</i>	<i>n = 21</i>	<i>n = 22</i>
<i>Participant</i>			
<i>Characteristics</i>			
Age (years)	11.9 \pm 2.6	12.1 \pm 2.6	11.7 \pm 2.7
Stature (cm)	147.4 \pm 13.7	146.0 \pm 13.5	148.8 \pm 14.1
Body mass (kg)	43.0 \pm 12.6	41.9 \pm 11.5	44.1 \pm 13.7
BMI (kg·m⁻²)	19.3 \pm 3.2	19.3 \pm 2.8	19.4 \pm 3.6
Maturity Offset (yrs)	-1.04 \pm 2.3	-1.04 \pm 2.4	-1.04 \pm 2.3
FVC (% predicted)	85 \pm 13	82 \pm 12	88 \pm 14
FEV₁ (% predicted)	86 \pm 13	80 \pm 10	92 \pm 14
<i>ActiGraph Data</i>			
Sedentary time	566.4 \pm 65.3	555.8 \pm 59.9	576.5 \pm 69.9
(min·day⁻¹)			
LPA (min·day⁻¹)	222.9 \pm 50.8	225.5 \pm 50.4	220.6 \pm 52.3
MVPA (min·day⁻¹)	60.7 \pm 33.4	58.9 \pm 36.9	62.2 \pm 30.3
Wear time (min·day⁻¹)	848.6 \pm 44.0	840.3 \pm 48.9	856.7 \pm 38.2

469 FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 second.

470

471 **Table 2: Associations between time (min) spent in different physical activity**
 472 **intensities and sedentary time variables between pairs of days (all participants;**
 473 **n=43)**

Between-days model^a		
	b (95% CI)	p value
SED_{D1} → SED_{D2}	-0.24 (-0.36 to -0.12)	<0.001
LPA_{D1} → LPA_{D2}	-0.05 (-0.20 to 0.11)	0.57
MVPA_{D1} → MVPA_{D2}	-0.01 (-0.49 to 0.47)	0.96
SED_{D1} → LPA_{D2}	0.01 (-0.09 to 0.09)	0.97
SED_{D1} → MVPA_{D2}	0.02 (-0.04 to 0.08)	0.61
LPA_{D1} → SED_{D2}	-0.20 (-0.42 to 0.03)	0.08
LPA_{D1} → MVPA_{D2}	0.03 (-0.09 to 0.14)	0.68
MVPA_{D1} → SED_{D2}	-1.24 (-2.21 to -0.29)	0.01
MVPA_{D1} → LPA_{D2}	-0.12 (-0.76 to 0.53)	0.72

474 ^aAnalyses adjusted for: sex, decimal age, condition, measurement day, wear time,
 475 person-level physical activity and/or sedentary time

476

477 **Table 3: Associations between time (min) spent in different physical activity**
 478 **intensities and sedentary time variables between pairs of days (healthy**
 479 **matched controls)**

Between-days model^a		
	b (95% CI)	p value
SED_{D1} → SED_{D2}	-0.31 (-0.53 to -0.10)	<0.01
LPA_{D1} → LPA_{D2}	0.11 (-0.15 to 0.37)	0.40
MVPA_{D1} → MVPA_{D2}	-0.23 (-1.02 to 0.57)	0.58
SED_{D1} → LPA_{D2}	0.11 (-0.04 to 0.25)	0.15
SED_{D1} → MVPA_{D2}	-0.01 (-0.13 to 0.09)	0.81
LPA_{D1} → SED_{D2}	-0.31 (-0.70 to 0.09)	0.13
LPA_{D1} → MVPA_{D2}	-0.03 (-0.23 to 0.17)	0.77
MVPA_{D1} → SED_{D2}	-1.81 (-3.38 to -0.24)	0.02
MVPA_{D1} → LPA_{D2}	0.56 (-0.48 to 1.59)	0.29

480 ^aAnalyses adjusted for: sex, age, measurement day, wear time, person-level physical
 481 activity and/or sedentary time

482

483 **Table 4: Associations between time (min) spent in different physical activity**
 484 **intensities and sedentary time variables between pairs of days (CF patients)**

Between-days model^a		
	b (95% CI)	p value
SED_{D1} → SED_{D2}	-0.19 (-0.36 to -0.03)	0.02
LPAD_{D1} → LPAD_{D2}	-0.10 (-0.30 to 0.10)	0.33
MVPA_{D1} → MVPA_{D2}	0.25 (-0.37 to 0.86)	0.43
SED_{D1} → LPAD_{D2}	-0.43 (-0.16 to 0.07)	0.46
SED_{D1} → MVPA_{D2}	0.04 (-0.04 to 0.12)	0.38
LPAD_{D1} → SED_{D2}	-0.14 (-0.42 to 0.15)	0.36
LPAD_{D1} → MVPA_{D2}	0.06 (-0.08 to 0.21)	0.41
MVPA_{D1} → SED_{D2}	-1.01 (-2.27 to 0.24)	0.11
MVPA_{D1} → LPAD_{D2}	-0.38 (-1.23 to 0.46)	0.38

485 ^aAnalyses adjusted for: sex, age, measurement day, wear time, person-level physical
 486 activity and/or sedentary time

487