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1 **Alpine Skiing With total knee ArthroPlasty (ASWAP): Muscular adaptations.**

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10 **Running head:** Muscular adaptations after alpine skiing

11

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22 **Abstract**

23 This study investigated the effectiveness of recreational skiing as intervention to improve
24 quadriceps muscle architecture, strength and antagonistic co-activation in patients with unilateral
25 total knee arthroplasty (TKA). Hence, TKA patients were assigned to either an intervention group
26 (IG) or control group (CG). The IG completed a 12 week guided skiing program whereas the CG
27 was instructed not to change their daily routines for the same period and was not allowed to ski.
28 Before, after the intervention / after an eight week retention period m. rectus femoris (RF) cross
29 sectional area (CSA), m. vastus lateralis (VL) muscle thickness, fascicle length and pennation
30 angle were measured with ultrasonography, while isometric (90° knee angle) knee extension,
31 flexion torque and m. biceps femoris (BF) co-activation were assessed on an isokinetic
32 dynamometer in 26 patients. There were significant and stable increases in RF CSA for the
33 operated (10%; $P < 0.05$) and non-operated leg (12%; $P < 0.01$) after the training period in the IG
34 whereas no changes were observed for the CG (all $P > 0.05$). There were no significant effects
35 for other parameters (all $P > 0.05$). Overall, the skiing intervention was successful in increasing
36 muscle mass in TKA older patients.

37

38 **Keywords:** strength, muscle weakness, ageing, sarcopenia

39

40

41 **Introduction**

42 For end-stage osteoarthritis, total knee arthroplasty (TKA) is a commonly used procedure which
43 successfully reduces pain and activity limitations (Kösters et al., 2015). Although quadriceps
44 strength is remarkably reduced (~60%) in the first month postoperatively, there is a subsequent
45 increase in the following weeks until preoperative values are reached, six months post-TKA
46 (Mizner et al., 2005; Stevens-Lapsley et al., 2010). However, results from recent publications
47 indicate that muscle weakness persists after this period, leading to a 19-29% deficit in knee
48 extensor strength of the operated (OP) leg compared to the non-operated (NOP) one (Lorentzen
49 et al., 1999; Maffiuletti et al., 2010). At first sight, this imbalance seems to disappear after a year
50 (Petterson et al., 2011), but using the NOP leg as a control may underestimate the magnitude of
51 impairment since disuse stemming from osteoarthritis or inactivity could have weakened this leg
52 (Farquhar & Snyder-Mackler, 2010). In fact, comparisons with healthy controls indicate a
53 substantial strength deficit (20-35%) even three years after surgery (Walsh et al., 1998).

54 The persistent muscle weakness is often ascribed to the loss of muscle mass and/or neural
55 impairments. Accordingly, muscle cross sectional area (CSA) and activation capacity follow a
56 similar time course of recovery for quadriceps strength (Mizner et al., 2005; Petterson et al.,
57 2011). However, activation capacity of both legs becomes comparable after 12 weeks post-
58 surgery, while quadriceps CSA of the OP leg is still significantly smaller (7%) at week 52
59 (Petterson et al., 2011). Hence, it seems the loss of muscle CSA contributes more to the persistent
60 muscle weakness than the deficit in muscle activation (Petterson et al., 2011).

61 Another less considered mechanism leading to muscle weakness is an excessive co-activation of
62 antagonists during knee extension. In healthy conditions, co-activation of hamstrings provides

63 stability to the knee joint (Baratta et al., 1988). However, excessive co-activation of these
64 muscles effectively reduces the knee extension moment. Such a higher co-activation of
65 hamstrings was found during maximum knee extensions (Stevens-Lapsley et al., 2010) and gait
66 analyses (Benedetti et al., 2003) one and 24 month respectively after TKA. This higher co-
67 activation, despite the loss of muscle mass, may also influence knee extension torque and
68 movement quality (Benedetti et al., 2003).

69 Functional impairment in daily activities of these patients is a direct consequence of muscle
70 weakness (Walsh et al., 1998; Mizner & Snyder-Mackler, 2005) and can even worsen with
71 ageing (Narici & Maffulli, 2010). Sarcopenia is characterized by the loss of muscle mass and
72 strength in old age, associated with other factors such as reduction of agonist activation (Roos et
73 al., 1997; Häkkinen et al., 1998) and excessive co-activation (Macaluso et al., 2002).

74 Additionally, the effects of muscle atrophy seem to worsen by concomitant changes in
75 architectural arrangement of muscle fascicles (Narici et al., 2003). While numerous publications
76 demonstrate the effectiveness of training to mitigate deficits related to ageing (Häkkinen et al.,
77 1998; Harridge et al., 1999; Reeves et al., 2009), intervention studies targeting TKA patients
78 months after surgery are scarce. Investigating the effect of aquatic training 4-18 months after
79 TKA with a 12 months follow-up, Valtonen et al. (2010) found significant increases in leg
80 extensor and flexor power, improvements in functional tasks and gains in thigh muscle CSA.
81 However, all improvements but the increases in muscle power disappeared at 12 months follow-
82 up (Valtonen et al., 2011). These results demonstrate not only the need of muscle strengthening
83 for this population but also maintenance of an active lifestyle after TKA.

84 Previous publications have shown that loading of the m. quadriceps femoris during recreational
85 skiing is an effective stimulus to increase muscle mass (Narici et al., 2011) and strength (Müller
86 et al., 2011) in healthy older individuals. Furthermore, skiing also requires a high level of motor
87 control for hamstring muscles. Their activation must be carefully tuned to stabilize the knee
88 without impairing the knee extension during the turning phase (Hintermeister et al., 1995;
89 Hintermeister et al., 1997). The optimization of co-activation patterns expected after a skiing
90 intervention might therefore translate into a normalization of antagonist muscle coordination, via
91 a reduction of the excessive co-activation found in TKA patients. However, a training
92 intervention based on alpine skiing with TKA older patients has never been studied before.
93 Therefore, the purpose of this study was to determine the effect of 12 weeks of guided skiing 2-3
94 times per week on knee extensor muscle architecture, strength and antagonistic co-activation 1-5
95 years after TKA. We hypothesized that this kind of intervention would mitigate the loss of
96 muscle mass and strength associated with ageing and disuse and reduce excessive co-activation.

97

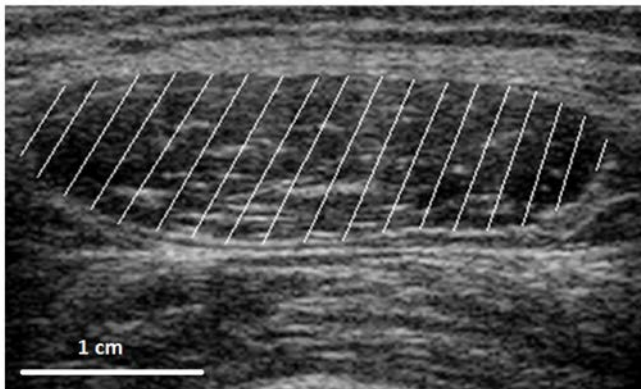
99 **Materials and methods**

100 Details regarding the overall study design, the patients and the skiing intervention protocol, are
101 presented in the companion paper by Kösters et al. (2015). Briefly, 31 older adults
102 (70.4 ± 4.7 years) with unilateral TKA, 2.7 ± 0.9 years after surgery, were assigned either to an
103 intervention group (IG) or a control group (CG). The IG followed a guided skiing program for 12
104 weeks, whereas the CG did not change their daily routines over the same time span and were not
105 allowed to ski. Muscle architectural parameters, maximum isometric knee extension and flexion
106 torque and hamstrings co-activation were measured before and after the intervention period and
107 after an eight week retention phase, respectively.

108 Muscle architecture

109 Muscle architecture was measured at rest with ultrasonography (LA523, 10- to 15-MHz
110 transducer, MyLab25, Esaote, Genoa, Italy) at the beginning of each testing day. Patients were
111 lying in a supine position while muscle scans of m. rectus femoris (RF) and m. vastus lateralis
112 (VL) were taken from both legs at a thigh region corresponding to 40% of femur length relative
113 to the femoral condyles (measured manually as the distance between the femoral condyles and
114 the trochanter major). At this location, the width of the RF did not exceed that of the ultrasound
115 field of view. All ultrasound scans were done by the same examiner. The pressure of the probe on
116 the skin was kept to a minimum to prevent deformation of the muscles. Transparent acetate paper
117 was used to record the location of measurements, as well as some anatomical landmarks (e.g.
118 moles, small scars) that were used in subsequent testing sessions to ensure a consistent
119 positioning of the template. Recordings of RF CSA, VL fascicle length (Lf), VL muscle

120 thickness (T_m) and VL pennation angle (θ) were analyzed offline using digitizing software
121 (ImageJ 1.41, NIH, Bethesda, USA). Fascicles were segmented manually but portions running
122 out of the scanning frame were extrapolated as straight lines (Muraoka et al., 2001). Pennation
123 angle was defined as the angle between the deep aponeurosis while the fascicles and muscle
124 thickness was measured as the distance between the deep and superficial aponeuroses. An
125 average of three measurements was obtained for each parameter for the statistical analysis. Due
126 to the limiting range of the ultrasound probe, muscle CSA could only be measured in the RF. In
127 VL muscle, T_m was used as a surrogate. The reliability of ultrasound based measurements of
128 human muscle architecture and CSA has been reported in previous publications (Sipilä &
129 Suominen, 1993; Muraoka et al., 2001).



130
131 Cross sectional area (CSA) of m. rectus femoris (RF).
132 Maximum voluntary contraction (MVC)
133 Maximum isometric torque of knee extensors and flexors was measured in a seated position on an
134 isokinetic dynamometer (IsoMed 2000 D&R Ferstl GmbH, Hemau, Germany). The knee angle
135 was set on 90° (180° corresponding to full extension) and patients had to perform two maximum

136 voluntary knee extensions followed by two knee flexions with a two-minute resting period
137 between contractions. Patients were instructed to progressively exert a torque within 3-4 seconds.
138 The same procedure was repeated for the other leg. Afterwards, the highest MVC values were
139 retained and normalized to body weight for further analysis. The reliability of these
140 measurements has recently been published by Dirnberger et al. (2012).

141 Hamstrings co-activation

142 Electromyographic activity (EMG) of m. biceps femoris (BF) was measured during isometric
143 knee extension and flexion MVCs to quantify hamstrings co-activation. Standard procedures for
144 skin preparation were followed (Hermens et al., 1999) before placing EMG surface electrodes
145 (Ag/AgCL; 120 dB, input impedance: 1200 GOhm; 10 mm diameter, 22 mm spacing, Biovision,
146 Wehrheim, Germany) over the BF. The reference electrode was placed on the lateral epicondyle
147 of the femur. The activity of this muscle was assumed to be representative of the hamstring group
148 (Macaluso et al., 2002). During MVCs, EMG activity of BF and torque were recorded and
149 synchronized with a sampling frequency of 2000 Hz (biovision, Wehrheim, Germany). To
150 process EMG data, the signal recorded during the highest MVC in each condition (knee extension
151 and flexion) was filtered offline using a second order butterworth filter with cut off frequencies of
152 10 and 300 Hz. EMG activity was calculated as the root mean square (RMS) over a one-second
153 period around peak torque. Hamstrings co-activation was determined by normalizing BF RMS
154 during knee extension to BF RMS during knee flexion (BF RMS during knee extension / BF
155 RMS during knee flexion; Stevens-Lapsley et al., 2010). The resulting values were multiplied by
156 100 afterwards to obtain percental relations.

157 Statistics

158 Normality of the data distribution was tested using the Kolmogorov-Smirnov test. Baseline
159 differences between groups and legs were assessed for each variable using a two way ANOVA
160 with factors group (IG / CG) and leg (OP / NOP). To determine possible effects of the
161 intervention a two way ANOVA with repeated measures of factors time (PRE / POST) x leg
162 (OP / NOP) x group (IG / CG) was performed for each variable separately. If group x time or
163 group x time x leg interaction effects were found to be significant further paired sample t-tests
164 were performed as post-hoc tests for each group and leg to identify possible changes.
165 Additionally, paired sample t-tests were calculated between post- and retention-test for each
166 group and leg to analyze if there were any changes during the eight week retention period.
167 Strength and co-activation data were not included in the retention-test analyses due to the
168 reduction of the sample size at these time point and for these variables. The level of significance
169 was set at $P < 0.05$. Data are presented as means and standard deviations (SD).

170 **Results**

171 Twenty-six (12 IG; 14 CG) out of the original 31 (14 IG; 17 CG) TKA patients remained after
172 the 12-week intervention period (see Kösters et al., 2015). Data from three patients (1 IG; 2 CG)
173 had to be excluded following the screening of ultrasound recordings due to poor image quality,
174 while data of two other patients (CG) were refused for strength and co-activation analysis due to
175 defective EMG signals. This reduction led to different sample sizes for muscle architecture (11
176 IG; 12CG) and strength/ co-activation (12 IG, 12 CG) analyses. There was a further loss of one
177 patient (IG) for muscle and four patients (1 IG; 3 CG) for strength/ co-activation analyses from
178 post- to retention-test. Due to the reduced sample size for strength and co-activation data, no
179 statistical post-retention-test comparisons were performed. Baseline characteristics of the patients
180 used in these analyses are presented in Table 1.

181 Baseline characteristics of patients

182

	IG		CG	
	OP	NOP	OP	NOP
	<i>n</i> = 11	<i>n</i> = 11	<i>n</i> = 12	<i>n</i> = 12
Muscle architecture				
RF CSA (cm ²)	2.8 ± 0.9	2.5 ± 0.9	2.2 ± 1.0	2.3 ± 0.9
VL Tm (cm)	2.0 ± 0.3	1.9 ± 0.4	1.9 ± 0.4	1.9 ± 0.3
VL Lf (cm)	8.8 ± 2.1	8.9 ± 2.2	9.4 ± 1.9	8.8 ± 2.4
VL θ (°)	15.9 ± 2.3	16.1 ± 2.9	14.6 ± 3.3	14.5 ± 2.3
	<i>n</i> = 12	<i>n</i> = 12	<i>n</i> = 12	<i>n</i> = 12
Maximum torque				
n torque extension (Nm/kg)	1.47 ± 0.26	1.73 ± 0.44	1.46 ± 0.44	1.37 ± 0.42
n torque flexion (Nm/kg)	0.56 ± 0.18 [#]	0.73 ± 0.23 [#]	0.57 ± 0.11 [#]	0.63 ± 0.23 [#]
BF coactivation (%)	11 ± 6*	8 ± 4*	13 ± 10*	15 ± 6*

Values are presented as mean ± SD; IG = intervention group; CG = control group; OP = operated leg; NOP = non-operated leg; RF = m. rectus femoris; VL = m. vastus lateralis; CSA = cross sectional area; Tm = muscle thickness; Lf = fascicle length; θ = pennation angle; n torque = maximum torque normalized to body weight; BF = m. biceps femoris; significant differences between groups: * $P < 0.05$; tendency for differences between legs: [#] $P = 0.066$.

183

184 **Muscle architecture**

185 There were no significant group x time x leg or group x time interactions for VL Tm, VL Lf or
186 VL θ (all $P > 0.05$). These data are summarized in Table 2. There were also no significant group
187 x time x leg interactions for RF CSA ($P = 0.964$; $F_{(1,21)} = 0.00$). However, the group x time
188 interaction for RF CSA was significant ($P = 0.000$; $F_{(1,21)} = 24.06$). Post-hoc tests revealed
189 RF CSA of the IG increased significantly in the OP (+10%; $P < 0.05$) and NOP leg
190 (+12%; $P < 0.01$), whereas no changes occurred in the CG (Figure 2). There were no significant
191 post-retention-test differences in any muscle variables for the two groups or legs (all $P > 0.05$).

192 **M. vastus lateralis architecture at PRE-, POST- and RET-test**

193

194 M. rectus femoris cross sectional area (CSA) at PRE-, POST- and RET-test

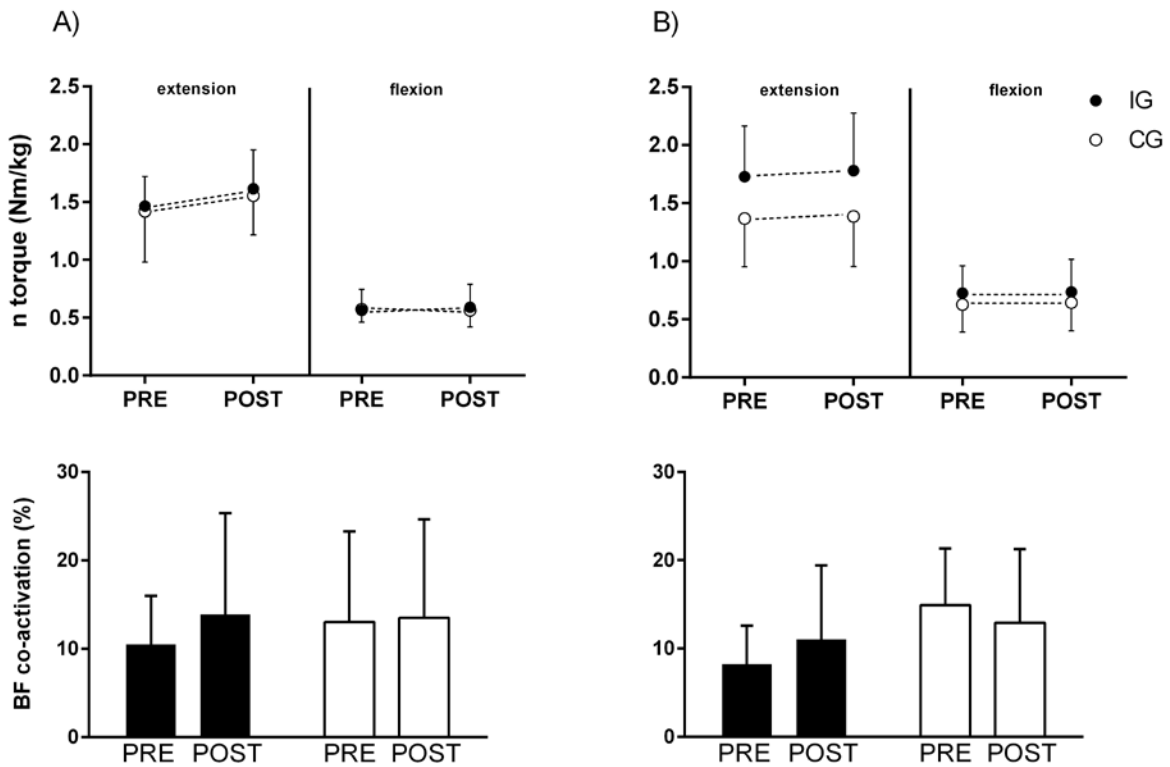
195

196 Maximum torque and hamstrings co-activation

197 There were no significant group x time x leg ($P = 0.847$; $F_{(1,22)} = 0.04$) or group x time
198 ($P = 0.738$; $F_{(1,22)} = 0.11$) interactions for maximum knee extension torque. In addition, no
199 significant group x time x leg ($P = 0.410$; $F_{(1,22)} = 0.706$) or group x time ($P = 0.549$;

200 $F_{(1,22)} = 0.37$) interactions were found for maximum knee flexion torque. Body weight did not
 201 change in the IG (85 ± 12 kg vs. 84 ± 12 kg) and CG (84 ± 15 kg vs. 83 ± 15 kg). There was no
 202 significant group x time interaction effect for this parameter ($P = 0.882$; $F_{(1,22)} = 0.23$). BF co-
 203 activation also showed no significant group x time x leg ($P = 0.628$; $F_{(1,22)} = 0.24$) or group x
 204 time ($P = 0.160$; $F_{(1,22)} = 2.11$) interactions. These data are summarized in Figure 3.

205 Maximum torque and m. biceps femoris (BF) co-activation at PRE- and POST-test



206

207

208 **Discussion**

209 The present results demonstrate the effectiveness of an alpine skiing intervention to increase
210 muscle mass in TKA older patients, 1-5 years after surgery. After the 12-week skiing program
211 RF CSA was significantly increased in the OP and NOP leg, although contrary to our hypothesis,
212 the skiing intervention had no influence on VL architecture, 90° isometric strength or BF co-
213 activation.

214 Muscle morphological adaptations

215 The skiing intervention led to a significant RF CSA gain of 10% in the OP and 12% in the NOP
216 leg of TKA patients. These adaptations were still visible at retention-test. A hypertrophic muscle
217 response to training, more than one year after surgery, has to date only been reported once
218 previously in these patients (LaStayo et al., 2009). The increase in quadriceps volume (11%)
219 observed by these authors after eccentric resistance training are in line with the results obtained in
220 our study and are similar to quadriceps CSA increases (~10%) found in healthy older individuals
221 after resistance training (Harridge et al., 1999; Kryger & Andersen, 2007). Although our study
222 found a training effect for the RF only, these results reflect the resistive exercise imposed to the
223 muscle during skiing. Downhill skiing predominantly involves eccentric work of knee extensor
224 muscles (Berg & Eiken, 1999). Previous publications have shown eccentric loading is effective in
225 inducing hypertrophy of type II muscle fiber and of the whole quadriceps muscles, in particular
226 the RF muscle (Hortobágyi et al., 1996; Narici et al., 1996). Taken together, the above studies
227 and our results suggest eccentric work induced by skiing training elicit a similar hypertrophic
228 response in TKA patients than that obtained with resistance training in healthy elderly.

229 Surprisingly, we were not able to find any change in VL muscle architecture after the
230 intervention period. This is in contrast to the findings of Narici et al. (2011) reporting increases in
231 all architecture parameters (VL Tm: +7.1%; VL Lf: +3.4%; VL θ : +5.4%) after 12 weeks of
232 recreational skiing in older adults. Furthermore, since eccentric tensile load seems particularly
233 effective to increase Lf (Reeves et al., 2009), such an increase was expected with downhill skiing
234 training. The explanation for this discrepancy is unclear but our ultrasound measurements are
235 consistent with the lack of evidence of VL muscle hypertrophy in biopsy samples collected in the
236 same patients (Kristensen et al., 2015). A possible explanation may be related to regional
237 differences in muscle hypertrophy (Wells et al., 2014) and the different muscle areas that were
238 scanned by Narici et al. (2011) and in the present protocol. Alternatively, the lack of change in
239 VL architecture in TKA elderly could reflect different postural strategies during skiing, which
240 would reduce the load of the VL muscle. Further investigations on these aspects are needed to
241 investigate the influence TKA upon muscle architecture and function.

242

243 Muscle functional adaptations

244 Despite the RF hypertrophy, the 12-week skiing intervention had no appreciable effects on
245 isometric torque. This result, especially for extension torque, was not expected since LaStayo et
246 al. (2009) reported concomitant gains in quadriceps volume and in isometric extension strength in
247 a comparable population following eccentric resistance training. Yet changes in muscle size and
248 isometric strength are not always coupled. For instance, the hypertrophic response previously
249 observed in healthy older individuals was accompanied by an increase in isokinetic leg press
250 without any change in isometric knee extension torque (Müller et al., 2011). Scheiber et al.

251 (2012) demonstrated that a knee joint range of motion spanning 115-140° is required during
252 recreational skiing. The lack of change in isometric torque produced at 90° may therefore be
253 linked to the lack of specificity of this test. This explanation is further supported by results of
254 another experiment in this same study reporting strength gains in isokinetic unilateral leg press
255 and isometric 120° knee extension torque in the OP leg, following the intervention (Pötzelsberger
256 et al., 2015).

257 Results of former publications demonstrate a persistent strength deficit in the OP leg when
258 compared to healthy controls (Walsh et al., 1998). Recently, a skiing intervention study with
259 healthy individuals, with a similar age to the present patients, was performed by our group
260 (Müller et al., 2011). Using the same methodology and equipment (knee angle 90°), higher knee
261 extension and flexion torques were measured in these subjects compared to the OP leg of our
262 patients (1.81 Nm/kg and 0.93 Nm/kg vs. 1.47 Nm/kg 0.57 Nm/kg, respectively). However, these
263 measurements were not performed within the same study and results have to be considered with
264 caution. Additionally, baseline flexion torque of the OP leg tended to be weaker compared to the
265 NOP one. These results indicate that strength deficits of lower limb muscles may exist years after
266 TKA surgery. Accordingly, future rehabilitation/training interventions should indifferently target
267 all major muscle groups of the lower limbs in this population.

268 An excessive hamstrings co-activation (~40%) has been proposed as a contributing factor to the
269 reduction in quadriceps strength of TKA (Stevens-Lapsley et al., 2010) and healthy older
270 individuals (Macaluso et al., 2002). However, the values (~12%) obtained in this study were
271 lower than previously reported, without any statistical difference between legs. If high levels of
272 hamstrings co-contractions were initially limiting knee extension torque, this problem could no

273 longer be detected more than two years post-surgery. On the contrary, co-activation in this study
274 was markedly lower than previously measured in healthy individuals (~20%; Macaluso et al.,
275 2002; Stevens-Lapsley et al., 2010) and did not increase significantly after training. This
276 discrepancy could be related to methodological differences (e.g. EMG electrodes positioning)
277 between studies.

278 **Perspective**

279 Physical activity remains reduced in many TKA patients after surgery (Naal & Impellizzeri,
280 2010). The risk of adverse effects to which impaired muscular function and sarcopenia expose
281 this ageing population is alarming (Narici & Maffulli, 2010). Our results showed that recreational
282 skiing as intervention is effective in eliciting RF hypertrophy. Although no increases in isometric
283 torque were observed at 90° of knee flexion, gains in isokinetic and isometric extension torque at
284 120° of knee flexion were measured and are reported in a companion paper (Pötzelsberger et al.,
285 2015). In addition to conventional training therapies, alpine skiing appears as a feasible
286 alternative to increase muscle mass and strength after TKA. However, VL architecture and
287 flexion torque remained unchanged after the intervention. Further longitudinal studies with
288 bigger samples and longer periods are needed to optimize the parameters of skiing training in this
289 population.

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386

387 **FIGURES:**

388 **Figure 1:** Cross sectional area (CSA) of m. rectus femoris (RF).

389 Representative ultrasound image of RF CSA (white shaded area).

390

391 **Figure 2:** M. rectus femoris (RF) cross sectional area (CSA) at PRE-, POST- and RET-test.

392 RF CSA at PRE-, POST- and RET-test in the intervention group (IG: black) and the control

393 group (CG: white) for the A) operated and B) non-operated leg. Significant group x time

394 interaction $P < 0.01$. . Significant differences from PRE- to POST-test: * $P < 0.05$; ** $P < 0.01$.

395

396 **Figure 3:** Maximum torque and m. biceps femoris (BF) co-activation at PRE- and POST-test.

397 Maximum normalized torque and BF co-activation at PRE- and POST-test in the intervention

398 group (IG: black) and control group (CG: white) for A) the operated and B) non-operated leg.

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