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26 **Abstract.**

27 **BACKGROUND:** Knee osteoarthritis (OA) impairs postural control, which may be affected

28 by how to use lower limb joints.

29 **OBJECTIVE:** To investigate how individuals with knee OA use lower limb joints for static

30 postural control.

31 **METHODS:** Ten patients with knee OA and thirteen healthy controls performed quiet

32 standing for 30 s. The standard deviation of the center of mass (COM) and lower limb joint

33 motions in the anterior-posterior (AP) and medial-lateral (ML) planes were calculated from

34 three-dimensional marker trajectories. Pearson's correlation analysis and independent *t*-tests

35 were conducted to investigate the relationship between COM and lower limb joint motions

36 and to compare group difference, respectively.

37 **RESULTS:** The AP hip angular velocity alone in the knee OA group and the AP hip and

38 knee angular velocity in the control group were significantly correlated with the AP COM

39 velocity. The ML hip angular velocity was significantly correlated with the ML COM

40 velocity in both groups. The knee OA group exhibited a significantly larger standard

41 deviation of AP COM velocity than the control group.

42 **CONCLUSIONS:** Individuals with knee OA depended solely on the contribution of the hip

43 to the AP COM velocity, which could not be successfully controlled by the knee.

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1. Introduction

Osteoarthritis (OA) of the knee is substantially prevalent in the elderly [1,2]. Patients with knee OA have knee pain [3], quadriceps weakness [4], limited knee range of motion [5], impaired proprioception of the knee [6], and structural changes (e.g., knee malalignment and cartilage degradation) [7]. Consequently, knee OA is considered one of the main causes that limits activities of daily living [8], deteriorates the quality of life [9], and increases the likelihood of falling [10]. The prevention of falls is a research priority in patients with knee OA because falls may induce incident fractures [10–12].

Poor postural control in patients with knee OA is considered to increase the likelihood of falling [13]. Patients with knee OA exhibit impairments in postural control, including increased postural sway during quiet standing compared to healthy adults [14]. Previous studies demonstrated that the amount of postural sway in this patient population is associated with knee pain [15,16], severity of knee OA [17], and knee muscle strength [18]. For the clinical assessment and rehabilitation of individuals with knee OA, it is important to understand how these factors affect joint motion during postural control.

The goal of static postural control is to stabilize the center of mass (COM) within the base of support. All joint motions, particularly those in the lower limbs, play a vital role in postural control. In general, the maintenance of equilibrium in the anterior-posterior (AP) and medial-lateral (ML) planes during quiet standing is mainly controlled by ankle and hip joint motions [19,20], while a recent study showed that knee joint motion additionally contributes to static postural control [21]. Individuals with knee OA have impaired hip and ankle joint functions as well as knee joint functions [4–6,22–25]. However, it remains unknown whether the use of lower limb joints for postural control in this patient population differs from that in healthy adults. Therefore, the purpose of this study was to investigate the changes in postural control strategies during quiet standing in individuals with knee OA. We hypothesized that

3

76 the lower limb joint motions used to control the COM motion were different between

77 individuals with knee OA and age-matched healthy controls.

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79 **2. Methods**

80   This study was a case-control type investigation. We received ethical approval from

81 the Institutional Review Board of our institute, and written informed consent was obtained

82 from all participants.

83

84 *2.1. Participants*

85   Twenty-three participants, ten individuals diagnosed with unilateral or bilateral knee

86 OA (one male and nine females) and thirteen healthy controls (three male and ten females),

87 participated in this study (Table 1). Knee pain in patients with knee OA was assessed using a

88 visual analogue scale (VAS), which ranged from 0 (no pain) to 100 mm (severe pain), and the

89 average VAS score was 23.8 mm. All participants with and without knee OA were recruited

90 from several hospitals and local communities. *A priori* power analysis was conducted using

91 the t-test model of G*Power 3.1.9.2 (Kiel University, Kiel, Germany). Based on our pilot

92 study of knee angular velocity data with seven participants (three patients with knee OA and

93 four healthy controls), the minimum sample size for this study was 10 participants per group

94 (effect size = 1.39, alpha = 0.05, and power = 0.80).

95   Participants in the knee OA group were included if they were aged 50–79 years and

96 had radiographically diagnosed knee OA with a Kellgren–Lawrence grade 2–4 in at least one

97 knee [26]. Potential participants in both groups were excluded if they had any previous lower

98 limb or back surgery, severe disability in walking and standing without an assistive device,

99 any musculoskeletal disorder other than knee OA that would influence balance, or any

100 neurological disorder.

101

2.2. Experimental protocol

All participants were instructed to stand as still as possible and to keep looking straight ahead with their feet hip-width apart and their arms folded across the chest. During testing, participants were not provided with any information on body sway. The examination was performed twice for 30 s.

All data were collected using a motion-capture system (Cortex; Motion Analysis Corporation, Santa Rosa, CA, USA), which consisted of seven cameras (Hawk cameras; Motion Analysis Corporation) with a sampling rate of 200 Hz. According to the modified Helen Hayes marker set [27], 25 reflective markers were positioned at the following anatomical landmarks: dorsum of the foot, heel, lateral and medial malleoli, lateral shank, lateral and medial femoral epicondyle, lateral thigh, anterior superior iliac spine, sacral, acromion, lateral humeral epicondyle, and wrist. All markers, except the sacral, were attached bilaterally.

2.3. Data analysis

All signals were processed using a custom MATLAB program (MathWorks Inc., Natick, MA, USA). Three-dimensional marker trajectories were low-pass filtered at a cutoff frequency of 5 Hz using a fourth-order Butterworth filter [28].

The time series of AP and ML COM positions were calculated from the marker data based on previously reported anthropometric data [29]. The time series of AP and ML COM velocities were calculated as the first-time derivatives of COM positions. The following COM-based parameters in each direction were calculated to assess the amount of body sway during quiet standing: (1) standard deviation (SD) of COM position and (2) SD of COM velocity [20,30].

126   The time series of AP and ML hip, knee, and ankle angles were calculated using

127   Visual 3D software (C-Motion Inc., Germantown, MD, USA). Subsequently, the time series

128   of AP and ML angular velocities were calculated as the first-time derivatives of each lower

129   limb joint angle. Knee joint motion analysis in the ML plane, or knee adduction-abduction,

130   was excluded based on the method presented in [31]. Additionally, the following kinematic

131   parameters in each direction were calculated to assess the amount of lower limb joint motions

132   during quiet standing: (1) SD of lower limb joint angle and (2) SD of lower limb joint angular

133   velocity [20]. The affected limb in patients with unilateral knee OA, the most affected limb in

134   patients with bilateral knee OA, and a randomly selected limb in healthy controls were

135   adopted in this study.

136   To compare the differences in standing posture, the mean values of COM positions

137   and lower limb joint angles during static standing were calculated. The mean values of COM

138   positions were defined as the distance from the midpoint of the bilateral heel markers in the

139   AP and ML planes. The mean values of COM positions in the AP plane were expressed as

140   positive if the COM position was anterior relative to the midpoint of the bilateral heel

141   markers, while those in the ML plane were expressed as positive if the COM position was in

142   the direction of the study limb relative to the midpoint of the bilateral heel markers. In

143   addition, lower limb joint angles in the AP plane were represented as positive for flexion

144   (dorsiflexion), while those in the ML plane were represented as positive for adduction

145   (inversion).

146

147   *2.4. Statistical analysis*

148   An independent *t*-test was used to compare the demographic data, the mean values of

149   COM positions, the mean values of lower limb joint angles, the SDs of COM position and

150   velocity, and the SDs of lower limb joint angles and angular velocities between the groups.

151 Then, the mean difference (the knee OA group minus the control group) and 95% confidence

152 interval (CI) for each variable was calculated. Pearson's product correlation analysis was

153 performed to investigate the relationship between lower limb joint motion and COM motion

154 in the AP and ML planes in each group. In addition, Pearson's product correlation analysis

155 was carried out to investigate the associations of the amount of body sway and lower limb

156 joint motions with knee pain in patients with knee OA. The statistical significance level was

157 set at $P < 0.05$. All statistical analyses were performed using IBM SPSS Statistics version

158 26.0 (IBM Corporation, Armonk, NY, USA).

159

160 **3. Results**

161   The mean values of COM positions for the groups in the AP (mean difference = 4.91

162 mm, 95% CI = -6.85 to 16.67 mm, $P = 0.395$) and ML planes (mean difference = -0.95 mm,

163 95% CI = -7.02 to 5.12 mm, $P = 0.748$) did not change significantly. In addition, no

164 significant difference was found between the mean values of lower limb joint angles of

165 groups in the AP (hip: mean difference = -0.58°, 95% CI = -6.76 to 5.60°, $P = 0.847$; knee:

166 mean difference = 2.71°, 95% CI = -2.97 to 8.38°, $P = 0.332$; ankle: mean difference = 0.68°,

167 95% CI = -2.92 to 4.28°, $P = 0.698$) and ML planes (hip: mean difference = -0.55°, 95% CI =

168 -2.66 to 1.57°, $P = 0.597$; ankle: mean difference = -2.43°, 95% CI = -8.21 to 3.35°, $P =$

169 0.391).

170   For the SD of AP and ML COM positions, there was no significant difference between

171 the groups (AP: mean difference = 0.06 mm, 95% CI = -0.91 to 1.02 mm, $P = 0.903$; ML:

172 mean difference = 0.51 mm, 95% CI = -0.14 to 1.16 mm, $P = 0.115$) (Fig. 1a). The SD of AP

173 COM velocity was significantly larger in the knee OA group than that of the control group

174 (mean difference = 0.79 mm/s, 95% CI = 0.24 to 1.35 mm/s, $P = 0.007$). Furthermore, there

175 was no significant difference in the ML COM velocity of groups (mean difference = 0.18

176   mm/s, 95% CI = -0.27 to 0.63 mm/s, $P = 0.408$) (Fig. 1b).

177   For the SD of lower limb joint angles in the AP plane, there was no significant

178   difference between the groups (hip: mean difference = 0.06°, 95% CI = -0.09 to 0.20°, $P =$

179   0.433; knee: mean difference = 0.02°, 95% CI = -0.08 to 0.11°, $P = 0.705$; ankle: mean

180   difference = 0.03°, 95% CI = -0.03 to 0.09°, $P = 0.296$) (Fig. 2a). Regarding the ML plane,

181   the SD of hip angle was significantly larger in the knee OA group than that of the control

182   group (mean difference = 0.07°, 95% CI = 0.02 to 0.12°, $P = 0.007$). Moreover, the SD of

183   ankle angle was not significantly different between the groups (mean difference = 0.01°, 95%

184   CI = -0.08 to 0.09°, $P = 0.853$) (Fig. 2b). As for the lower limb joint angular velocities, the

185   SD of AP hip and knee angular velocities were significantly larger in the knee OA group than

186   those of the control group (hip: mean difference = 0.22 °/s, 95% CI = 0.08 to 0.35 °/s, $P =$

187   0.003; knee: mean difference = 0.17 °/s, 95% CI = 0.04 to 0.30 °/s, $P = 0.010$). In addition,

188   the SD of AP ankle angular velocity was not significantly different between groups (mean

189   difference = 0.04 °/s, 95% CI = -0.06 to 0.15 °/s, $P = 0.372$) (Fig. 3a). The SD of ML hip

190   angular velocity was significantly larger in the knee OA group than that of the control group

191   (mean difference = 0.16 °/s, 95% CI = 0.02 to 0.31 °/s, $P = 0.030$). Furthermore, the SD of

192   ML ankle angular velocity was not significantly different between groups (mean difference =

193   -0.14 °/s, 95% CI = -0.60 to 0.31 °/s, $P = 0.523$) (Fig. 3b).

194   Pearson's correlation analysis showed that the SD of AP ankle angle was positively

195   correlated with the SD of AP COM position in both groups (OA: $r = 0.662$, $P = 0.037$;

196   control: $r = 0.777$, $P = 0.002$) (Table 2). In the knee OA group, the SD of ML hip angle was

197   positively correlated with the SD of ML COM position ($r = 0.925$, $P < 0.001$), whereas in the

198   control group, the SD of ML ankle angle was positively correlated with the SD of ML COM

199   position ($r = 0.726$, $P = 0.005$). The SD of AP hip angular velocity was positively correlated

200   with the SD of AP COM velocity in the knee OA group ($r = 0.644$, $P = 0.044$). In addition,

201 the SDs of AP hip and knee angular velocities were positively correlated with the SD of AP

202 COM velocity in the control group (hip: $r = 0.562$, $P = 0.046$; knee: $r = 0.673$, $P = 0.012$)

203 (Table 3). The SD of ML hip angular velocity was positively correlated with the SD of ML

204 COM velocity in both groups (OA: $r = 0.820$, $P = 0.004$; control: $r = 0.846$, $P < 0.001$).

205 In addition, there was no significant correlation between the SDs of COM and lower

206 limb joint motions and the VAS score for knee pain ($P = 0.092 – 0.966$) (Table 4).

207

208 **4. Discussion**

209 The current study revealed that the characteristics of the postural control strategy

210 during quiet standing in individuals with knee OA differed from those of age-matched healthy

211 adults. The knee OA group exhibited significantly larger SDs of AP COM velocity, ML hip

212 angle, AP hip angular velocity, AP knee angular velocity, and ML hip angular velocity than

213 the control group. The AP ankle angle was significantly correlated with the AP COM position

214 in both groups, while the ML hip angle and ML ankle angle were significantly correlated with

215 the ML COM position in the knee OA and control groups, respectively. In the knee OA

216 group, the AP hip angular velocity was significantly correlated with the AP COM velocity,

217 while in the control group, the AP hip and knee angular velocities were significantly

218 correlated. The ML hip angular velocity was significantly correlated with the ML COM

219 velocity in both groups.

220 Previous studies suggested that the amplitude of postural sway in the AP and ML

221 planes is generally controlled by ankle and hip joint motions [19,20]. In this study, the

222 amplitude of AP COM position was controlled by the ankle strategy in both groups, and there

223 was no significant difference in the amplitude of AP hip, knee, and ankle angles between the

224 groups. In other words, the postural control strategy for controlling the AP COM position may

225 be identical in both groups. The knee OA group used the hip strategy to control the amplitude

of ML COM position, whereas the control group used the ankle strategy. Furthermore, the

amplitude of ML hip angle of the knee OA group was larger than that of the control group;

however, there was no significant difference in the amplitude of ML ankle angles of the

groups. Compared with healthy adults, patients with knee OA have an impaired sense of

motion of the ankle joint in the ML plane; however not in the hip joint [25]. Those with knee

OA may be unable to adequately control the ML COM position with the ankle strategy.

Therefore, they may prefer to use the hip strategy to control the ML COM position.

Furthermore, the hip strategy has less COM motion than the ankle strategy even with the

same angle change because the center of rotation of hip joint is closer to the COM than that of

the ankle joint [32]. Therefore, the knee OA group may have required greater ML hip joint

motion than the control group.

It is believed that the hip strategy is preferable to the ankle strategy for fast COM

motion in postural control [33]. The hip strategy, which results in predominant hip joint

motion, includes thigh muscle activity that causes knee joint motion, although the muscles

around the ankle joint are extremely unresponsive [34]. As for the control of AP COM

velocity, the knee OA group used a postural control strategy using the hip joint alone, while

the control group used a mixed postural control strategy using the hip and knee joints. In other

words, the knee OA group depended only on the contribution of hip joint to the AP COM

velocity, while the control group depended on the contribution of hip and knee joints.

Therefore, the knee OA group exhibited increased body sway velocity and hip angular

velocity in the AP plane. In addition, the knee joint possibly cannot be used well to control

body sway velocity in the AP plane because the AP knee angular velocity in the knee OA

group was not correlated with the body sway velocity in the AP plane and was larger than that

of the control group. This may be affected by knee joint dysfunctions, such as quadriceps

weakness [4], limited knee range of motion [5], and impaired proprioception of the knee [6]

251 in patients with knee OA. Our study showed that it might be necessary to maintain hip joint

252 function and improve knee joint function for balance training in patients with knee OA. As for

253 the control of ML COM velocity, both groups used a postural control strategy using the hip

254 joint. Furthermore, the SD of hip angular velocity in the knee OA group was larger than that

255 of the control group, while there was no significant difference in the SD of ML COM velocity

256 for both groups. Postural sway velocity, such as the COM velocity, includes the direction and

257 position changes at the next moment. Therefore, this information is considered significant for

258 maintaining equilibrium [30,35]. The knee OA group may maintain postural stability because

259 of an increased sensory information resulting from an increased ML hip angular velocity to

260 compensate for an impaired sense of motion of the ankle joint in the ML plane.

261   Previous studies presented that knee pain was associated with postural sway in

262 patients with mild-to-severe knee OA whose VAS score for knee pain was greater than 30

263 mm [15,18]. However, in this study, knee pain did not correlate with the SDs of COM and

264 lower limb joint motions in patients with knee OA. This might be caused by mild knee pain

265 and mild-to-moderate knee OA in this study.

266   This study had several limitations. First, the participants in this study had mild-to-

267 moderate knee OA, and the average knee pain was less severe. The average knee pain in this

268 study may have been mild because of mild-to-moderate knee OA [9]. Therefore, the

269 participants in this study might not represent the overall characteristics of patients with knee

270 OA. Second, the task examined in this study was quiet standing, and results from dynamic

271 tasks, such as external perturbations, may differ from those in this study. Therefore, dynamic

272 tasks need to be investigated in the future. Third, we used three-dimensional motion analysis

273 to calculate the COM and lower limb joint motions. Measurement errors due to soft tissue

274 artifacts in three-dimensional motion analysis may have affected the results of quiet standing

275 performed in this study [36]. Finally, the number of participants in this study was limited.

276 Further studies with a large number of participants are needed to take into account the

277 severity of knee OA and knee pain.

278

279 **5. Conclusions**

280 We investigated the alterations in the postural control strategies during quiet standing

281 in individuals with knee OA. Both the knee OA and control groups utilized the ankle joint to

282 control the AP COM position. For the control of ML COM position, the knee OA group used

283 the hip joint, whereas the control group used the ankle joint. The knee OA group successfully

284 controlled the AP and ML COM positions because there was no significant group difference

285 in the amplitude of COM position. To control the AP COM velocity, the knee OA group

286 relied on the contribution of hip joint solely, whereas the control group relied on the

287 contribution of hip and knee joints. Both the knee OA and control groups used the hip joint to

288 control the ML COM velocity. The knee OA group exhibited postural control impairment in

289 the amplitude of COM velocity in the AP plane, and not in the ML plane though. Our findings

290 suggested that the knee OA group could not successfully control the AP COM velocity using

291 the knee joint.

292

295

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401   **Tables**

402   **Table 1**

403   Demographic data of the knee OA and control groups

	Knee OA	Control	*P*
Age (years)	67.8 (8.9)	63.5 (8.6)	0.258
Height (cm)	153.8 (7.5)	156.1 (8.2)	0.488
Weight (kg)	56.8 (15.2)	57.7 (10.3)	0.866
BMI (kg/m^2)	23.7 (4.1)	23.6 (3.1)	0.924
Kellgren–Lawrence grade (n)			
Grade 2	4		
Grade 3	6		
VAS score for knee pain (mm)[a]	23.8 (17.8)		

404   Data are reported as mean values (standard deviation).

405   OA: osteoarthritis, BMI: body mass index, VAS: visual analogue scale.

406   [a] VAS score for knee pain ranges from 0 (no pain) to 100 mm (severe pain).

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Table 2

Pearson's correlation coefficients between each lower limb joint angle and COM

position

	Knee OA		Control	
	r	*p*	*r*	*p*
SD of AP COM position				
SD of AP hip angle	0.129	0.722	0.207	0.497
SD of AP knee angle	0.311	0.382	0.418	0.155
SD of AP ankle angle	**0.662**	**0.037***	**0.777**	**0.002***
SD of ML COM position				
SD of ML hip angle	**0.925**	**< 0.001***	0.366	0.219
SD of ML ankle angle	0.239	0.507	**0.726**	**0.005***

COM: center of mass, OA: osteoarthritis, SD: standard deviation, AP: anterior-

posterior, ML: medial-lateral.

* $P < 0.05$.

433 **Table 3**

434 Pearson's correlation coefficients between each lower limb joint angular velocity and

435 COM velocity

	Knee OA		Control	
	r	*P*	*r*	*P*
SD of AP COM velocity				
SD of AP hip angular velocity	**0.644**	**0.044***	**0.562**	**0.046***
SD of AP knee angular velocity	0.539	0.108	**0.673**	**0.012***
SD of AP ankle angular velocity	0.424	0.222	0.427	0.146
SD of ML COM velocity				
SD of ML hip angular velocity	**0.820**	**0.004***	**0.846**	**< 0.001***
SD of ML ankle angular velocity	-0.023	0.949	0.212	0.488

436 COM: center of mass, OA: osteoarthritis, SD: standard deviation, AP: anterior-

437 posterior, ML: medial-lateral.

438 * $P < 0.05$.

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449 **Table 4**

450 Pearson's correlation coefficients between the COM and lower limb joint motions and

451 knee pain in patients with knee OA

	Knee OA	
	r	*P*
VAS score for knee pain		
SD of AP COM position	-0.352	0.319
SD of ML COM position	-0.196	0.588
SD of AP COM velocity	-0.182	0.614
SD of ML COM velocity	-0.105	0.772
SD of AP hip angle	0.467	0.173
SD of AP knee angle	0.449	0.193
SD of AP ankle angle	-0.322	0.364
SD of ML hip angle	-0.015	0.966
SD of ML ankle angle	0.561	0.092
SD of AP hip angular velocity	-0.196	0.587
SD of AP knee angular velocity	-0.044	0.904
SD of AP ankle angular velocity	0.145	0.689
SD of ML hip angular velocity	-0.134	0.711
SD of ML ankle angular velocity	0.495	0.146

452 COM: center of mass, OA: osteoarthritis, VAS: visual analogue scale, SD: standard

453 deviation, AP: anterior-posterior, ML: medial-lateral.

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Figure captions

Fig. 1. Group-averaged SD of COM position (a) and velocity (b) in the AP and ML planes. The white bar indicates the knee OA group and the black bar indicates the control group. * P < 0.05.

Fig. 2. Group-averaged SD of hip, knee, and ankle angles in the AP (a) and ML (b) planes. The White bar indicates the knee OA group and the black bar indicates the control group. * P < 0.05.

Fig. 3. Group-averaged SD of hip, knee, and ankle angular velocities in the AP (a) and ML (b) planes. The white bar indicates the knee OA group and the black bar indicates the control group. * P < 0.05.
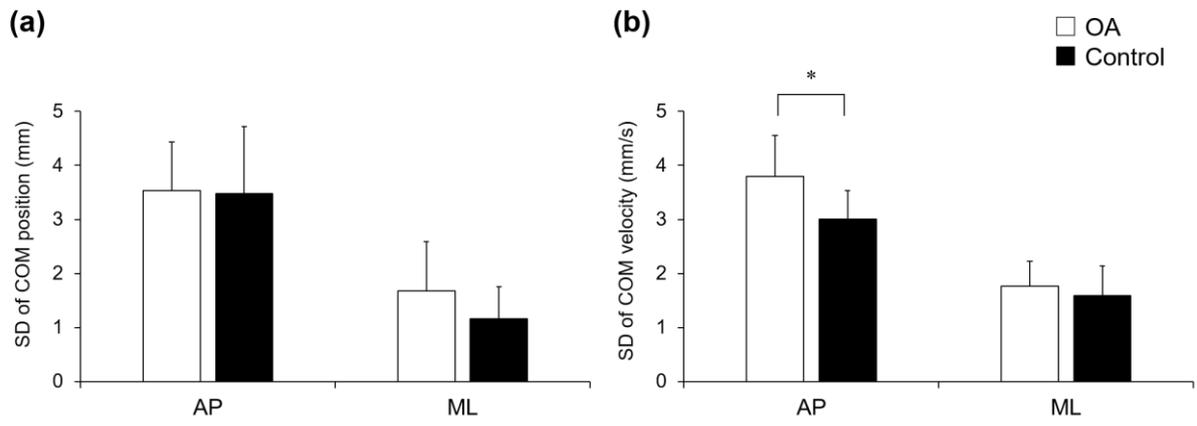
482   **Figures**

483   **Figure 1**

(a)

(b)

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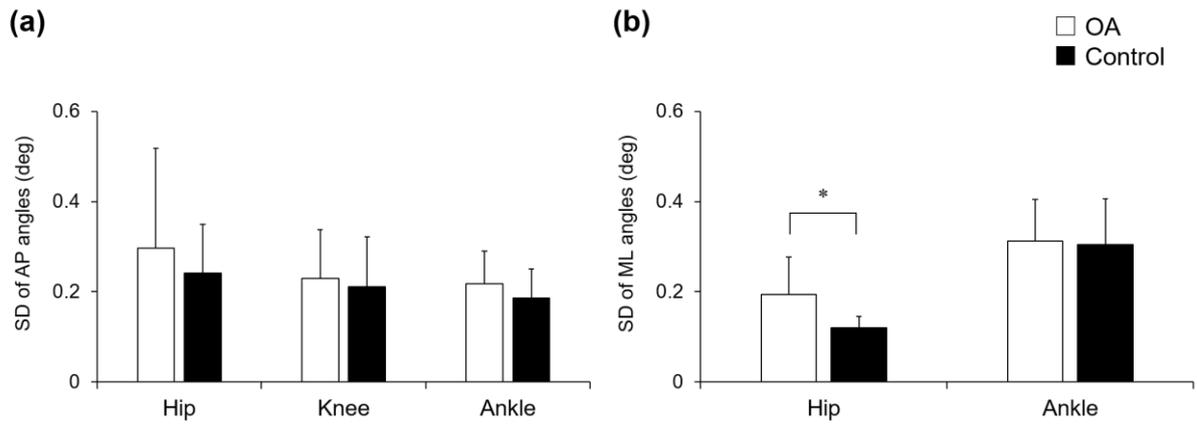
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Figure 2

522   **Figure 3**

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