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The relationship between knee joint effusion and quadriceps strength and activation after anterior cruciate ligament (ACL) injury and reconstruction.

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Online First

1 **The relationship between knee joint effusion and quadriceps strength and**
2 **activation after anterior cruciate ligament (ACL) injury and reconstruction.**

3 **ABSTRACT**

4 **Context:** Knee joint effusion and quadriceps strength and activation deficits are
5 common consequences of anterior cruciate ligament (ACL) injury and reconstruction.
6 The presence of an effusion may initiate or worsen the quadriceps dysfunction present
7 after ACL trauma. In simulated effusion studies, evidence indicates an inverse
8 relationship between effusion size and quadriceps dysfunction. While this relationship
9 was not found in patients after ACL injury, prior research was limited by a subjective
10 clinical assessment of effusion grade. **Objective:** The purpose of this study was to
11 determine if the size of the knee joint effusion, measured via ultrasound, after ACL
12 injury and reconstruction influences quadriceps strength and activation. **Design:**
13 Descriptive Laboratory Study. **Setting:** Research Laboratory. **Patients or Other**
14 **Participants:** 41 individuals (23 females, age=21.8±7.5years, height =171.7±7.9cm,
15 mass =72.3±14.2kg), with an ACL injury reported for 2 sessions [one before
16 reconstruction and one ~16 weeks after ACL reconstruction]. **Main outcome**
17 **measures:** Three ultrasound images of the suprapatellar pouch and three trials of
18 quadriceps strength and activation using the interpolated twitch technique were
19 gathered from the ACL knee. Effusion cross-sectional area was measured using
20 ImageJ, averaged and inputted into linear regression models to predict muscle strength
21 and interpolated twitch activation. Analyses were considered significant at $p \leq 0.05$.
22 **Results:** No relationship was found between effusion size and strength ($R^2 = 0.086$) or
23 activation ($R^2=0.056$) after ACL injury ($p > 0.05$). After reconstruction, however, there

24 was a small negative relationship between effusion size and activation ($R^2=0.122$;
25 Standardized $\beta=-0.349$; $p=0.025$), while no relationship was found for effusion size and
26 strength ($R^2=0.000$; $p>0.05$). **Conclusion:** The size of the effusion after ACL injury does
27 not influence strength or activation. However, after ACL reconstruction, effusion size
28 does have a small influence on quadriceps activation, with a larger effusion being
29 related to lower activation. Prior research using simulated effusions appear to
30 overestimate the effects of effusion on quadriceps function.

31 **Key Terms:** Swelling, Voluntary Activation, ITT, Muscle and Arthrogenic Muscle
32 Inhibition

33 Word Count: 292

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35 **Key points:**

- 36 • Knee joint effusion, quantitatively assessed with ultrasound imaging, is not
37 associated with quadriceps activation or strength after ACL injury
- 38 • A small negative relationship (i.e., larger effusions are related to less quadriceps
39 activation) exists after ACL reconstruction.

40 INTRODUCTION

41 Quadriceps muscle strength is often drastically reduced after anterior cruciate ligament
42 (ACL) rupture¹ and reconstruction^{2,3}. Poor quadriceps strength prior to surgery is linked
43 to poor strength and functional outcomes after surgery.^{4,5} Further, weak quadriceps
44 musculature after surgical reconstruction has not only been associated with short-term
45 functional limitations⁶ but also the development of post-traumatic osteoarthritis⁷, which
46 leads to long-term pain and dysfunction and currently has no cure. As such,
47 understanding factors that lead to quadriceps weakness after ACL injury and
48 reconstruction is paramount to combatting both its acute and chronic sequelae.

49
50 One mechanism by which quadriceps weakness develops after injury is via arthrogenic
51 muscle inhibition (AMI) or a diminished ability to voluntarily activate the quadriceps.⁸
52 AMI is thought to originate secondary to damage or altered signaling from joint
53 mechanoreceptors resulting from direct trauma caused by the injury or surgery itself
54 and/or from the pain, effusion, or inflammation occurring secondary to the trauma. AMI
55 can contribute to profound muscle weakness and is an obstacle for rehabilitation, as it
56 impairs force output during voluntary contraction rendering active exercise as a less
57 potent stimulus for inducing strength gains.⁹

58
59 AMI can be artificially induced in the quadriceps muscle using a simulated knee effusion
60 model, whereby sterile saline is injected into the knee joint capsule.¹⁰⁻¹² Simulated
61 effusions greater than 20mL in size produce some level of quadriceps muscle shutdown
62 immediately after induction.¹³ This is thought to occur because of the pressure induced

63 by the saline in the joint capsule, triggering a mechanoreceptor response. Because
64 these simulated effusions have the capability of inducing AMI, it has been assumed that
65 effusions associated with joint injury, such as after an ACL injury or surgery, also lead to
66 AMI. However, only one study has been done in ACL patients and the results refute this
67 assumption. Lynch et al.¹⁴ found that effusion size (measured/graded using the
68 stroke/sweep test) was not correlated to AMI in patients that had sustained an ACL
69 rupture. This finding would suggest that effusions, whether small or large, did not result
70 in AMI in their study population. While these results are interesting, they are limited, in
71 part, by how effusion was measured. The stroke test is a clinical examination technique
72 and raters must subjectively assess effusion grade based on the size of the “bulge”
73 produced during the test. A more quantitative approach, such as using ultrasound to
74 image and calculate the cross-sectional area of the supra-patellar pouch, should allow
75 for better sensitivity with regard to effusion size.¹⁵ Furthermore, the work by Lynch et
76 al.¹⁴ only examined persons before any surgical intervention and it remains unknown if
77 effusion size is related to AMI or quadriceps strength after ACL reconstruction.
78 Therefore, the purpose of this study is to determine if the size of the suprapatellar knee
79 joint effusion, measured via ultrasound, after ACL injury and reconstruction influences
80 quadriceps AMI and strength. We hypothesized that greater suprapatellar joint effusion
81 would be associated with greater quadriceps AMI (evaluated via the interpolated twitch
82 technique) and lesser isometric quadriceps strength in the ACL reconstructed limb.

83 **MATERIALS AND METHODS**

84 *Study Design*

85 This is a longitudinal, cohort study where individuals who had sustained an ACL tear
86 completed two testing sessions. One session occurred prior to surgery (PRE; within 90
87 days of injury) and the other occurred approximately four months after surgery
88 (POST4M). At both visits, ultrasonographic effusion measurements, isometric
89 quadriceps strength testing, quadriceps muscle activation testing, and patient-reported
90 outcomes were gathered.

91 *Participants*

92 Forty-one individuals, aged 14-45 years old, who had experienced an ACL injury and
93 were planning to undergo ACL reconstruction were recruited from a local sports
94 medicine clinic following confirmation of injury via magnetic resonance imaging (Table
95 1). An a priori power calculation for simple linear regression (i.e., predictor variable = 1)
96 in G*Power using an f^2 effect of 0.25 and a statistical power of 0.8 indicated that 34
97 subjects would be needed to demonstrate a significant relationship at an alpha level of
98 0.05. Individuals were included in this study if they had sustained a primary unilateral
99 ACL tear and were planning to use an ipsilateral autograft (bone-patellar-bone,
100 hamstring, or quadriceps tendon) for their ACL reconstruction. Individuals were
101 excluded if they: 1) sustained greater than a grade 3 concomitant collateral ligament
102 injury indicating the need for a multi-ligamentous reconstruction, 2) experienced a knee
103 dislocation at the time of their ACL injury, 3) underwent a knee aspiration following their
104 ACL injury, and/or 4) were greater than 90 days from their injury at the time of
105 recruitment.

106 *Knee Effusion*

107 Upon arriving to the lab, all participants lied supine on a table with the knees extended
108 for 15 minutes prior to any images being recorded. After which, we placed a bolster
109 under the knee providing 30° of knee flexion¹⁶ in preparation for images to be recorded.
110 A 12Mhz linear transducer connected to a ultrasound unit (Logiq e, General Electric,
111 Waukesha, WI) was then placed longitudinally, approximately 2cm proximal to the
112 patella to view the suprapatellar pouch (SPP) (Figure 1). The transducer settings were
113 set to a gain of 50dB, a frequency of 17 Hz, and a depth of 4.5cm.¹⁷ The ultrasound
114 probe was placed as lightly as possible on the skin to avoid deforming the area. Three
115 images of the SPP were taken on the ACL and contralateral limbs (contralateral limb
116 data not reported). Two raters with 3+ years of ultrasound experience collected all the
117 images for this study. The rater that collected the baseline image of a given participant
118 always collected the 4-month image of the same participant.
119 DICOM images were then exported from the ultrasound machine and processed in
120 Image J (National Institutes of Health). Echo-free space was measured (mm²) and used
121 to determine the cross-sectional area of the SPP (Figure 1). The areas of each of the
122 three images were then averaged together. A single member of the study team was
123 responsible for calculating the knee effusion area of the SPP from all images. The
124 tracing of the knee effusion area was then confirmed by a senior team member. When
125 there was disagreement between the team members, they met, discussed, and came to
126 an agreement on the image measurements. The inter-rater reliability for our two raters
127 was high (ICC_{2,1} = .981; CI = .455, 1.00) suggesting strong agreement for measuring
128 SPP effusion on our US images.



129

130 **Figure 1.**

131 *Quadriceps Strength and Activation*

132 Individuals completed isometric quadriceps strength testing and quadriceps voluntary
133 muscle activation testing (a measure of AMI) using the interpolated twitch technique.¹⁸

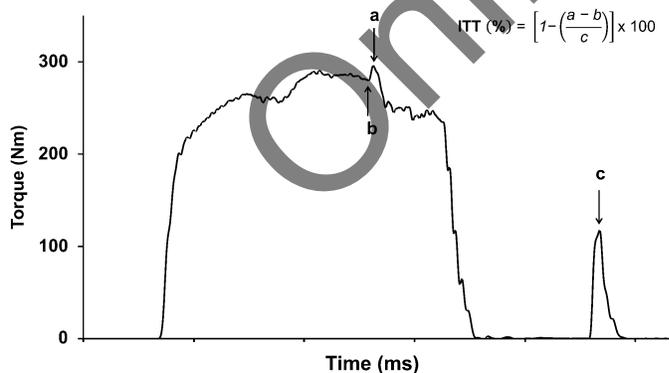
134 Briefly, individuals were seated in an isokinetic dynamometer (Humac NORM; CSMI,
135 Stoughton, MA) per manufacturer guidelines, with their ACL leg positioned at 60° of
136 flexion. Individuals were then outfitted with two self-adhesive electrodes, (2.75x5in
137 Dura-Stick Chattanooga Group) one to the proximal vastus lateralis and the second to
138 the distal vastus medialis. A series of warm-up trials at 25%, 50%, 75%, and 100% of
139 perceived maximal effort with a 1-minute rest between efforts were completed.

140 Individuals were then oriented to the electrical stimulus via the Digitimer DS7AH
141 constant current stimulator with (Digitimer Ltd., Hertfordshire, UK) low intensity pulse
142 trains (100Hz, 200- μ s pulse duration, 3 pulse train, 400V, current intensity females: 290
143 mA, males: 360mA).¹⁸ Participants were then instructed to perform three maximal
144 voluntary isometric contractions (MVIC), lasting approximately five seconds each.

145 During each MVIC, individuals would receive an electrical stimulus delivered via an
146 automated torque-based triggering approach through a custom written LabVIEW

147 program (LabVIEW 11.0; National Instruments Corp., Austin, TX).¹⁹ Following relaxation
148 from the first MVIC, an identical electrical stimulus was delivered to obtain a potentiated
149 evoked torque at rest (resting twitch torque). Individuals completed three MVICs and
150 muscle activation trials. Verbal encouragement and real-time feedback were provided
151 during each contraction.

152 The peak value from the isometric torque curve prior to the delivery of the interpolated
153 twitch was recorded as the MVIC and normalized to body mass. A limb symmetry index
154 (LSI) was also calculated for the MVIC ((ACL MVIC/Non-ACL MVIC)*100). The
155 superimposed twitch torque was recorded as the additional torque recorded after the
156 electrical stimulus was delivered, and the resting twitch torque was recorded as the
157 peak torque following the delivery of the resting twitch electrical stimulus. Overall
158 muscle activation was calculated as: % Muscle Activation = (1 - [superimposed twitch
159 torque / resting twitch torque]) × 100 (Figure 2). The average of the three trials is
160 reported for muscle activation, MVIC, and LSI.



161

162 **Figure 2.**

163 *Knee Osteoarthritis and Outcomes Scale (KOOS)*

164

165 The KOOS²⁰ was assessed at both study time points. This patient reported outcome

166 measure consists of 5 subscales: 1) Pain; 2) Other symptoms; 3) Function in daily

167 living; 4) Function in sport and recreation; and 5) Knee related quality of life.
168 Standardized answer options are given (5 Likert boxes) and each question is assigned
169 a score from 0 to 4. A normalized score (100 indicating no symptoms and 0 indicating
170 extreme symptoms) is calculated for each subscale. Each KOOS subscale score was
171 calculated and reported descriptively in Table 1.

172 *Statistics*

173 Four linear regression models were run in SPSS (Version 29, IBM Corp., Armonk, NY)
174 to determine the relationship between effusion and strength and activation before and
175 after ACL reconstruction. Effusion was entered as the independent variable and
176 normalized MVIC or interpolated twitch activation ratio were inputted as the dependent
177 variable. Supplementary multiple linear regression models were run to determine the
178 relationship between KOOS pain score, effusion, and strength. Paired t-tests were done
179 to determine differences pre-to-post ACL reconstruction for effusion size, normalized
180 strength, LSI, activation, and KOOS pain. Analyses were considered significant at $p <$
181 0.05

182 **RESULTS**

183 Demographic data for participants and descriptive data for average strength, activation
184 and effusion size can be found in Table 1. The values for each trial of activation and
185 strength can be found in Appendix 1. Two of the 41 participants were missing strength
186 data at baseline, and therefore, were not included in the baseline regression models.

187
188 No relationship was found between effusion size and isometric strength ($R^2 = 0.086$; $p =$
189 0.068) or activation ($R^2 = 0.056$; $p = 0.165$) prior to ACL reconstruction (Figure 3). After

190 reconstruction, however, there was a small relationship between effusion size and
 191 activation ($R^2 = 0.122$; Standardized $\beta = -0.349$; $p = 0.025$), while no relationship was
 192 found for effusion size strength (isometric $R^2 = .000$; $p = 0.935$) (Figure 4).
 193 Multiple linear regression models which considered pain and effusion on strength and
 194 activation revealed similar findings reported to the simple linear regression models
 195 above, whereby no relationships were noted between the variables prior to ACL
 196 reconstruction ($p > 0.05$), but after ACL reconstruction the model was significant for
 197 activation ($R^2 = .157$; Unstandardized β for pain = 0.27 and $p = 0.22$; Unstandardized β
 198 for effusion = -0.10 and $p = .04$), but not strength (Appendix 2). The size of the effusion
 199 and the quadriceps LSI decreased after ACL reconstruction compared to baseline ($p <$
 200 0.05). The KOOS pain score increased significantly from pre-surgery to post surgery (p
 201 < 0.001). No significant differences were noted between the normalized ACL leg MVIC
 202 ($p = 0.13$) and quadriceps activation ($p = 0.093$) between the pre-surgical and post-
 203 surgical time points.

204 **Table 1. Relevant Demographic Information for Participants Before and After ACL**
 205 **Reconstruction. Means (SD) unless otherwise denoted.**

	PRE-ACL Reconstruction	POST-ACL Reconstruction
N	41	
Age (yrs)	21.44 (7.59) Range: 14-45	
Height (cm)	171.73(7.95) Range: 155.4-192.5	
Mass (kg)	72.21(14.22) Range: 49.7-110.7	

Sex (N)	F = 23 M = 18	
Injury to Pre-ACL testing (days)	46.37(25.69) Range: 16-90	
Surgery to Post-ACL testing (days)		128.74 (12.19) Range: 95-157
KOOS Pain*	74.19(11.90)	83.47 11.57)
KOOS Function in Daily Living	79.84(15.75)	92.51
KOOS Sport & Recreation	36.46(24.96)	44.88(22.40)
KOOS Knee-Related Quality of Life	30.95(16.99)	45.12(19.09)
KOOS – Other Symptoms	68.47(13.48)	80.49(11.98)
Graft Type		Bone Patellar Tendon Bone = 34 Hamstring = 5 Quadriceps = 2
Effusion (mm²)*	136.18(92.44)	96.48(53.48)
ACL Limb MVIC (Nm/kg)	2.25 (0.73)	2.09 (0.63)
LSI*	83.17 (14.65)	72.22 (11.43)
Quadriceps activation (%)	68.76 (25.53)	69.27 (18.11)

* indicates significant different between pre-and post-ACL reconstruction time points

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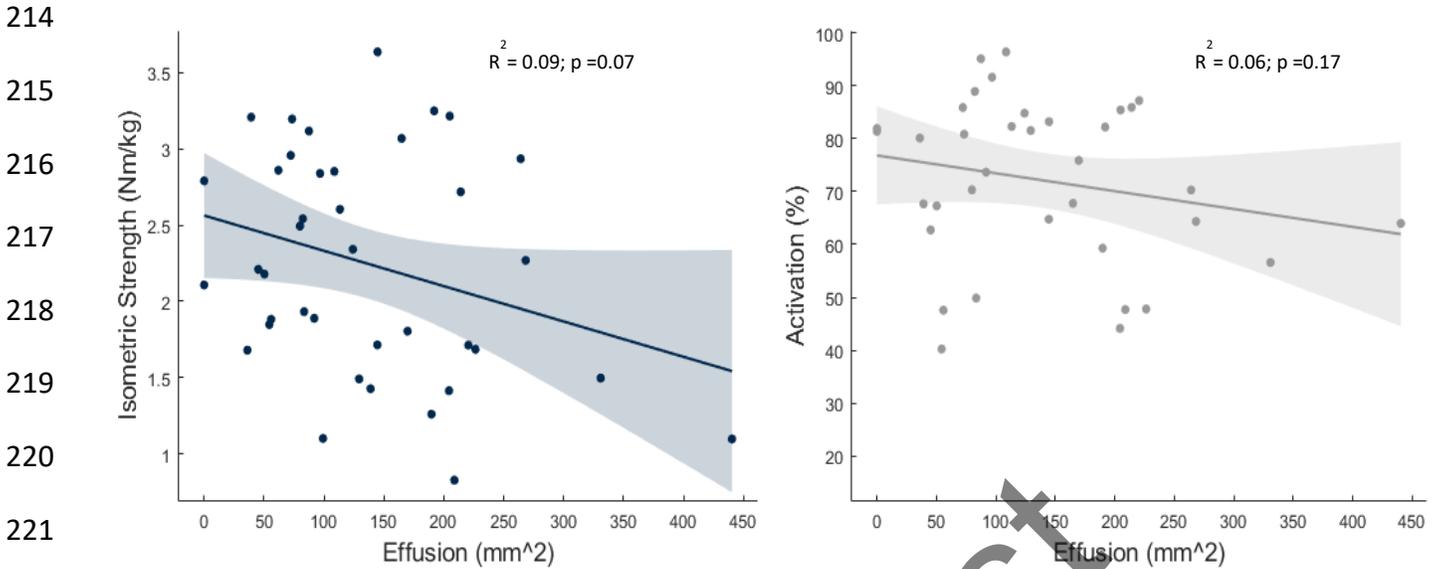
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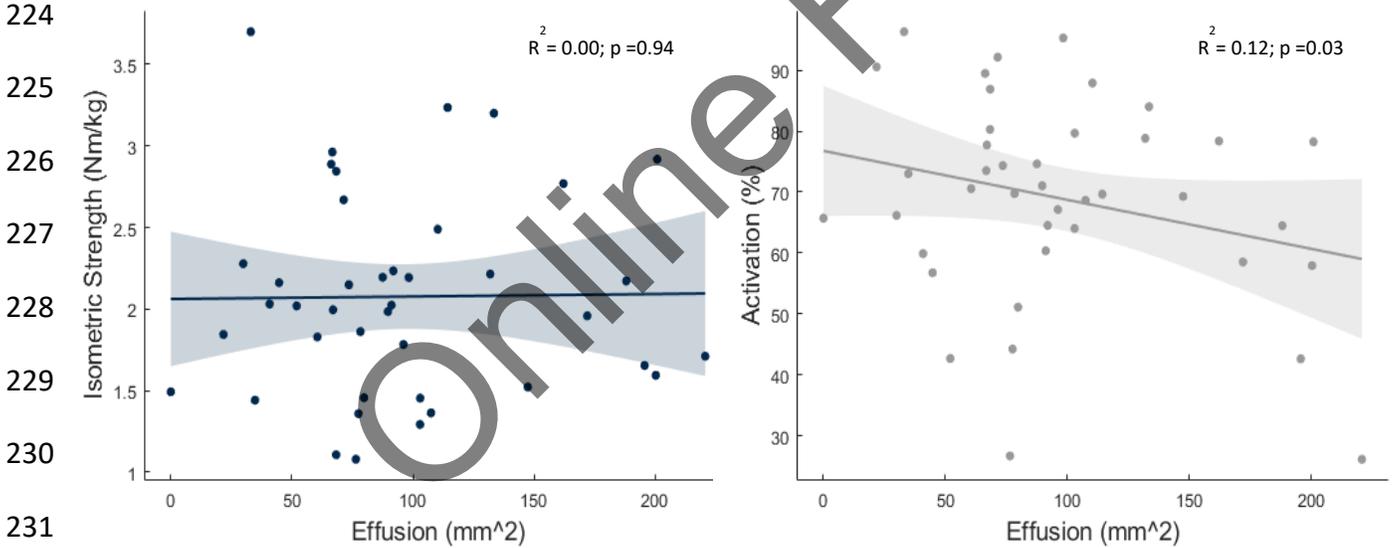
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223 **Figure 3.**



232 **Figure 4.**

233 **DISCUSSION**

234 This study aimed to examine the relationship between knee joint effusion size and
 235 quadriceps muscle strength and AMI/activation in patients before and after ACL
 236 reconstruction. Our results show that the size of the knee joint effusion after ACL injury

237 did not influence quadriceps strength or activation. However, after ACL reconstruction
238 effusion size did influence quadriceps activation, with a larger effusion being related to
239 lower quadriceps activation/more AMI. We also demonstrated no relationship between
240 effusion size and quadriceps strength after ACL reconstruction.

241 The finding that knee effusion size is not related to quadriceps strength or activation
242 after ACL injury agrees with prior literature. Work by Lynch et al.¹⁴ showed that in
243 subjects with ACL injury, clinical effusion grade measured by the stroke test was not
244 related to quadriceps activation quantified using the central activation ratio. These
245 results support that effusion grade was not related to the presence of AMI. Lynch et al.¹⁴
246 also showed that the quadriceps index did not differ among the various effusion grades,
247 suggesting patients with a variety of different effusion sizes (small, moderate, and large)
248 were not different in terms of quadriceps strength. Our findings (and those of Lynch et
249 al.¹⁴), however, are in conflict with research utilizing experimental effusion models.
250 These experimental effusion studies¹⁰⁻¹² have demonstrated that injecting fluid into an
251 otherwise healthy knee to mimic knee effusion directly results in AMI and strength
252 declines. The neuromuscular response to the experimental effusion is thought to be the
253 result of pressure applied to the mechanoreceptors from the injected fluid²¹, and this
254 pressure would also be present with effusions that develop naturally in patients after
255 ACL injury. A likely explanation for the conflict is related to the duration of time the fluid
256 is present in the joint in conjunction with the timing of when measurements were made.
257 In the experimental effusion studies, strength and activation can be captured
258 immediately after the fluid is placed inside the joint capsule, and thus, these studies
259 capture the acute response to the effusion/increase in pressure. However, in the

260 current study, we are capturing a sub-acute/chronic response to the fluid, as the
261 participants were tested on average 5-6 weeks after sustaining the injury. As such, the
262 fluid inside of the knee joint resulting from the injury may have been placing pressure on
263 the mechanoreceptors for weeks. Thus, the mechanoreceptors likely adapt to that
264 stimulus over time and their response is muted. While there isn't direct evidence to
265 support this claim inside the knee joint, research looking at cutaneous pressure
266 thresholds shows that the greater amount of time pressure is applied to the skin of the
267 hand the greater the pressure threshold becomes and supports our idea that the
268 mechanoreceptors in the knee may blunt their response to longer-term application of
269 pressure.²² Similarly, it is possible in the days since injury the magnitude of the effusion
270 inside of the knee joint for the ACL patients decreased, thereby resulting in a smaller
271 pressure-inducing stimulus, and a decreased neuromuscular response. Regardless of
272 the reason for the lack of an effect of effusion size on strength and activation, it is
273 apparent that the presence of an effusion after ACL injury is not a major driving factor
274 for initiating AMI or quadriceps strength deficits.

275 While effusion size was not related to quadriceps activation/AMI (or strength) prior to
276 surgery, it was related to AMI after surgical reconstruction. This relationship, whereby a
277 larger effusion was related to a lower activation ratio or greater AMI, was small however,
278 with effusion size only accounting for about 12% of the variance in the model. It is
279 unclear why effusion would be associated with AMI/activation after surgery, but not after
280 injury (i.e., before surgery). The average size of effusion was greater prior to surgery in
281 our cohort than it was post-surgery and as such, it is unlikely that there is a threshold
282 effect, whereby a certain magnitude of effusion is necessary to induce a neuromuscular

283 response. Along these lines, it is improbable that firing of the joint mechanoreceptors
284 responsible for pressure sensation (e.g. Ruffini endings) is responsible for the
285 relationship we see between effusion and AMI/activation after surgery given the lack of
286 relationship prior to ACL reconstruction when the pressure in the joint from the effusion
287 should be similar if not greater after injury (given the larger average effusion size at
288 baseline in our cohort). Another plausible explanation could be that pain is heightened
289 after surgery and the firing of free nerve endings in conjunction with the firing of
290 pressure receptors leads to a neuromuscular response/muscle shutdown. The KOOS
291 pain scores are significantly higher after ACL reconstruction in our study group,
292 however, when we explored the addition of pain to the activation regression model after
293 reconstruction (Appendix 2) pain did not contribute significantly, and thus, suggests pain
294 was not relevant to the relationship between effusion and AMI/activation. The only other
295 possibility we can hypothesize is that the content/makeup of the effusion (e.g., the
296 inflammatory markers, etc.) differs before and after ACL reconstruction and thereby
297 alters the body's neuromuscular response leading to AMI. This idea can be supported
298 by some literature which has shown a higher concentration of inflammatory and
299 cartilage breakdown makers in the knee four weeks after ACL reconstruction compared
300 with prior to surgery.²³ However, other research has reported no such differences.²⁴ It is
301 also possible that this observation could have been influenced by the rehabilitation
302 process, where patients with milder effusion could have been pushed harder to contract
303 their quadriceps muscle maximally than those with larger effusion during the
304 rehabilitation process. More research is necessary to understand why effusion is related
305 to quadriceps AMI/activation after ACL reconstruction. From a clinical standpoint,

306 however, it is important to discuss that while effusion and activation were related after
307 surgery the relationship was quite small, and thus, interventions that minimize effusion
308 after ACL reconstruction are unlikely to make a substantial impact on the magnitude of
309 AMI in patients who have undergone surgery. As such, researchers should continue to
310 investigate the mechanistic factors that lead to AMI after ACL injury and reconstruction
311 so that targeted intervention approaches can be developed.

312 This research is not without limitations. First, effusion was measured in the
313 suprapatellar pouch (SPP). The SPP is only one of several recesses, however, where
314 fluid could collect with an ACL injury. As such, it is important to consider that our US
315 measurements are only reflective of the fluid in the SPP and may not be reflective of
316 entire state of effusion in the knee. Secondly, while US is superior to clinical
317 examination in detecting effusion²⁵ it is not as strong as MRI²⁶. Ultrasound, however,
318 has been reported to have high specificity and sensitivity values of 100% and 66.7% for
319 detecting effusion in the SPP. Additionally, subject characteristics could have influenced
320 our results. For example, the majority of subjects had ACL reconstruction with a patellar
321 tendon autograft and as such these findings might not be relevant to participants with
322 other graft types. Next, the number of days from injury to baseline testing and from
323 surgery to 4-month testing had some variability between subjects which may have also
324 influenced our study outcomes. Lastly, the relationship between effusion and strength
325 may have been different if values were recorded at different times post-injury and post-
326 surgery.

327 In conclusion, knee joint effusion does not appear to be a driving mechanistic factor
328 leading to quadriceps strength deficits or quadriceps AMI before or after surgery. While

329 a statistically significant relationship was found between effusion and AMI/activation
330 after surgery, this was quite small and its clinical relevance is questionable.
331 Interventions targeting removal or reduction of joint effusion after ACL injury and
332 reconstruction are unlikely to mediate/reduce the quadriceps AMI.

333

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423 **Legends to Figure**

424 **Figure 1.** Placement of ultrasound probe to obtain knee effusion images (left).
425 Ultrasound image of the suprapatellar effusion (right).

426 **Figure 2.** Representative maximal voluntary isometric contraction (MVIC) trial during
427 activation testing. Letter b represents the time where the stimuli are applied at the peak
428 of the MVIC. Letter a shows the increase in torque that resulted from the stimuli. Letter
429 c represents the resting twitch torque.

430 **Figure 3.** Scatterplots depicting the relationship between effusion and quadriceps
431 isometric strength (left) and effusion and quadriceps activation
432 (right) in study participants after ACL injury.

433 **Figure 4.** Scatterplots depicting the relationship between effusion and quadriceps
434 isometric strength (left) and effusion and quadriceps activation(right) in study
435 participants after ACL reconstruction.

Online First

Appendix 1. Average Quadriceps Strength and Activation Data for Each of the Three Trials Recorded During Testing Before and After ACL Reconstruction. Reported as Mean (SD).

	PRE-ACL Reconstruction	POST-ACL Reconstruction
ACL Leg MVIC Trial 1	2.14 (0.73)	2.08 (0.63)
ACL Leg MVIC Trial 2	2.25(0.75)	2.06 (0.65)
ACL Leg MVIC Trial 3	2.23(0.74)	2.02 (0.62)
Quadriceps activation (%) Trial 1	66.94(28.45)	71.56(19.91)
Quadriceps activation (%) Trial 2	69.61(24.17)	68.13(19.28)
Quadriceps activation (%) Trial 3	67.96(27.86)	66.73(19.16)

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Appendix 2. Scatterplots depicting the relationship between the unstandardized predicted value for KOOS pain score and effusion and its relationship to pre-ACLR quadriceps isometric strength (top left), pre-ACLR quadriceps activation (top right), post-ACLR quadriceps isometric strength (bottom left) and post-ACLR quadriceps activation. (bottom right).

