Diffusion Tensor Imaging of the Calf Muscles at 1.5T: Diffusion Property Differences between Athletes and Non-Athletes

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Introduction

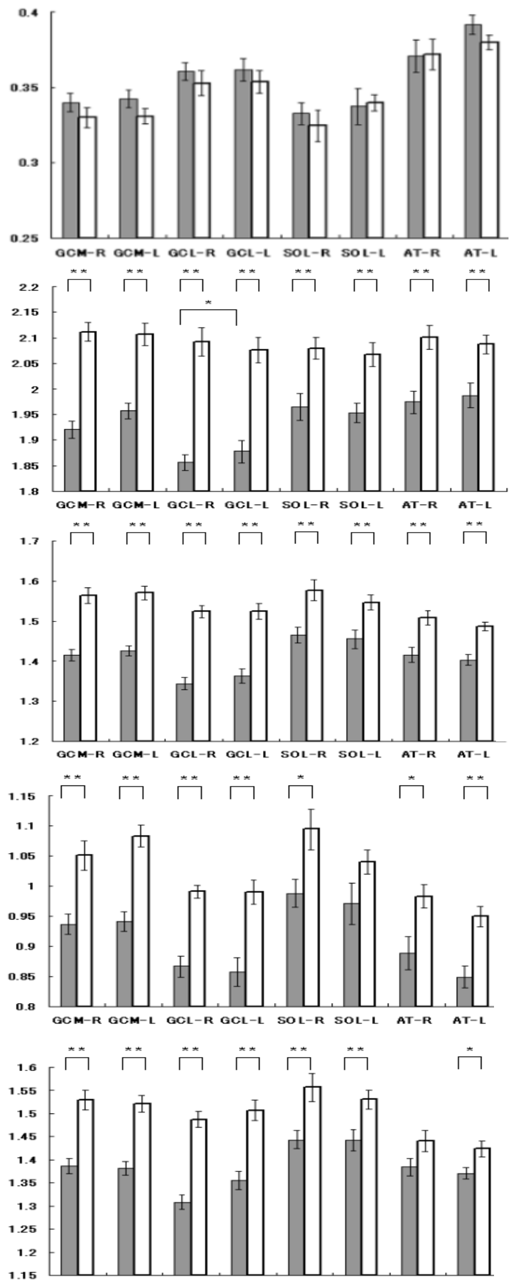
Skeletal muscle changes its microstructure markedly due to physical training [1-3]. It is well known that skeletal muscle hypertrophy occurs due to repeated contraction and laxity of the muscle during long-term training [1-4].The purpose of this present study is to compare the diffusion properties between the trained and non-trained muscle of volunteers belonging to athlete and non-athlete group, respectively.

Materials & Methods

Twelve athletes (Group A) and 11 non-athletes (Group B) were recruited for this study. They were all healthy females in their 20s. All 12 athletes were from the department of physical education of our university. They were all recruited to the university with sports scholarships. They were all active, well-trained athletes including 4 tennis players and 8 *Kendo* (modern Japanese sword-fighting) practitioners, regular class members and with high athletic achievement levels. Their average age was 20.3 years. All 11 non-athletes were also healthy, but did not do physical training in the daily life. Their average age was 21.6 years. We scanned the proximal portion of bilateral calves (including the largest part in diameter) using a 1.5 T clinical MR machine (Nova Dual release 2.6, Philips, Best, the Netherlands). Subjects were set in the supine position with feet first. The 4 channel SENSE body coil (sized 45×30 cm for parallel imaging) was convolved around the anterior and posterior aspects of their bilateral calves. Diffusion-weighted images were acquired using a single-shot spin-echo echo planner imaging (EPI) sequence with the following parameters: b-values of 0 and 500 seconds/mm^2, field of view (FOV) 350 (mm), rectangular FOV 51.79%, matrix size 224×224, slice thickness 6 mm without gap, internal number of slices 12 (7.2 cm of the length of scan range), TR = 4000 ms, TE = 60 ms, SENSE factor 2.2, number of motion probing gradient (MPG) directions 6, number of excitation 6 and total scan time 5 minutes 20 sec. Before the DTI, we scanned T1-FFE images as anatomical mapping using the following parameters; matrix size 256×192, slice thickness 6 mm, internal number of slices 12, TR = 13 ms, TE = 2.3 ms, SENSE factor 1.4, and total scan time 3 minutes, 4 sec.

We measured λ1-3, FA, and ADC for the right and left gastrocnemius medialis (GCM), gastrocnemius lateralis (GCL), soleus (SOL), and anterior tibialis (AT) for each volunteer. We compared each averaged λ1-3, FA and ADC from eight muscles, between groups A and B by Student's t-test.

Figure 1 Comparison of FA (a), λ1 (b), λ2 (c), λ3 (d), and ADC (e) values of eight bilateral calf muscles between group A (Athletes) and group B (Non-athletes)



The gray bar means group A, and white bar means group B in (a), (b), (c), (d), and (e).

(a) Each FA value of A and B in the right GCM, left GCM, right GCL, left GCL, right SOL, left SOL, right AT and left AT was 0.34 and 0.33, 0.34 in A and 0.33 in B, 0.36 and 0.35, 0.36 and 0.35, 0.33 and 0.32, 0.34 and 0.34, 0.37 and 0.37, 0.39 and 0.38, respectively. All FA values showed no statistically significant differences between group A and B in all eight muscles.

(b) Each λ1 value of A and B was 1.92 and 2.11, 1.96 and 2.11, 1.86 and 2.09, 1.88 and 2.08, 1.97 and 2.08, 1.95 and 2.07, 1.97 and 2.10, and 1.99 and 2.09, respectively. In all eight muscles, the value of λ1 of A showed lower than that of B with statistically significant difference (** $P<0.01$).And the value of GCL-R showed lower than that of GCL-L (* $P<0.05$) in group A reflecting laterality.

(c) Each λ2 value of A and B was 1.39 and 1.53, 1.38 and 1.52, 1.31 and 1.49, 1.36 and 1.51, 1.44 and 1.56, 1.44 and 1.53, 1.38 and 1.44, and 1.37 and 1.42, respectively. There were also significant differences with P value were less than 0.01 (** $P<0.01$) in six muscles including bilateral GCM, bilateral GCL, and bilateral SOL, and less than 0.05 (* $P<0.05$) in left AT.

(d) Each λ3 value of A and B was 0.94 and 1.05, 0.94 and 1.08, 0.87 and 0.99, 0.86 and 0.99, 0.99 and 1.09, 0.97 and 1.04, 0.89 and 0.98, and 0.85 and 0.95, respectively. There were also significant differences with P value were less than 0.01 (** $P<0.01$) in five muscles including bilateral GCM, bilateral GCL, and left AT, and less than 0.05 (* $P<0.05$) in right SOL and right AT inλ3.

(e) Each ADC value of A and B was 1.41 and 1.56, 1.34 and 1.52, 1.34 and 1.52, 1.36 and 1.52, 1.47 and 1.58, 1.46 and 1.55, 1.41 and 1.51, and 1.40 and 1.49, respectively. And there were significant differences with P value were less than 0.01 (** $P<0.01$) in all eight muscles.

Results & Discussion

In all eight muscles of bilateral calves, all three eigenvalues and ADC were lower in A than in B. As for λ1 and ADC, there were significant differences in all muscles ($P<0.01$). There were also significant differences in all muscles ($P<0.05$) except for right AT in λ2,and left SOL in λ3. FA values showed no statistically significant differences in all muscles (Figure 1). Human skeletal muscle is grossly and microstructurally hypertrophied by physical training. Skeletal muscle hypertrophy is basically characterized by such as the following some histologic changes [4] 1. Thickening of the cross section of the muscle fiber 2. Increase in the number of the muscle fiber 3. Increase in the collagen fiber networks. Although there was no apparent difference in FA between the athlete and non-athlete groups, the trained muscle showed lower λ1, λ2, λ3 and ADC values compared to the non-trained muscle. These results may indicate the passive narrowing of the extracellular space due to chronic muscle hypertrophy. The DTI of the skeletal muscle in our study mainly demonstrated the extracellular water diffusion. This is apparently different from published data which assert that the DTI of the skeletal muscle reflects intracellular water diffusion [5, 6].

Conclusion

Our results indicated that training caused a decrease of three eigenvalues and ADC. We hypothesize that it is due to a decrease of the extracellular space of the skeletal muscle at the microstructural level as a result of chronic muscle hypertrophy.

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