High-Intensity Resistance Training Does Not Produce Immediate Ultrasonographic Changes in Muscle Tendons

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**Background:** Chronic overload injuries to tendons can be visualized using ultrasonography, with characteristics such as tendon thickening and darkening.

**Purpose:** To investigate whether these characteristics are evident in the patellar and Achilles tendons immediately after 1 session of high-intensity resistance training.

**Study Design:** Controlled laboratory study.

**Methods:** A total of 18 volunteers were randomized to an experimental group (n = 10) and a sham group (n = 8). The experimental group performed 5 circuits at maximum effort consisting of 5 weighted front squats, 10 box jumps (60/50 cm), and 15 double-under jump-rope jumps. The sham group performed a similar circuit consisting of 5 weighted shoulder presses, 10 push-ups, and 15 weighted biceps curls. Ultrasonograms were obtained before and after exercise, for a total of 30 minutes at intervals of 2.5 minutes for the first 10 minutes and 5 minutes for the remaining time. Tendon thickness and tendon matrix signals were measured. Statistics were performed using repeated-measures mixed analysis of variance (ANOVA).

**Results:** Tendon thickness did not increase significantly over 30 minutes after both circuits. The mean grayscale value for the patellar and Achilles tendons increased for both the experimental and the sham groups. ANOVA showed that the experimental group was not a significant explanatory variable; however, the increased work of both groups was. A post hoc analysis found that the maximum increase in the tendon signal was a grayscale value of 10.8 for the patellar tendon (99.4% CI, 3.7-17.9; \( P = .002 \)).

**Conclusion:** This trial failed to reproduce an earlier study in which tendon thickness increased after high-intensity training. The tendons produced a hyperechoic signal after high-intensity resistance training, regardless of loading to the tendon. Chronic overload characteristics on ultrasonography were not evident immediately after acute loading of tendons.

**Clinical Relevance:** There is a need for prognostic and diagnostic markers of tendinopathy especially because of the protracted course of subclinical development of an injury. This study assessed whether clinical findings for a chronic overload injury can be detected during acute overloading.

**Keywords:** knee patellar tendon; Achilles tendon; imaging; diagnostic ultrasound; biology of tendon

The high prevalence of tendinopathy among athletes and in the general population makes it a condition of great significance.\(^{16}\) The incidence rate of Achilles tendinopathy has been reported as 1.85 per 1000 patients who present to general practitioners, and the prevalence among athletes has been reported as 1.8%.\(^{2,4}\)

It has been hypothesized that overload injuries are the result of multiple, minor single overloads during exercise, which in turn produce a chronic overload injury.\(^{12,13,18}\) These stimuli are found to induce inflammation or degeneration,\(^{17,24}\) which in turn weakens the tendon and increases the risk of ruptures. Most sports produce both eccentric and concentric loads on the tendons. Eccentric loads are often the cause of both tendinopathy and ruptures.\(^{10}\) However, rehabilitation regimens have been successful in applying eccentric loads as a mode of therapy, which could imply that the tendons do not only react to the forces applied but also the quality of the loads.\(^{21}\)

These chronic injuries can be assessed by ultrasonography and are characterized by changes such as increased tendon thickness and hypoechoic tendon signals.\(^{1,6,11}\) Asymptomatic chronic changes in tendon morphology observed by ultrasonography have been shown to be predictive of the later development of...
tendinopathy.\(^2\) Whether these characteristics are observable immediately after loading and potential overloading of the tendons is not known. An acute alteration in tendon thickness could potentially be representative of the early-stage development of a chronic overload injury. Thus, this study applied ultrasonography as a modality of investigation for the pathophysiology of such injuries.

The objective of this blinded, randomized trial was to reproduce the findings of Fisker et al\(^5\) and hence elucidate the pathophysiology of the overloading of tendons. The Fisker et al\(^5\) study found that the patellar tendon increased in thickness by 0.47 mm immediately after a high-intensity resistance training session. Patellar tendon thickness was measured in 34 volunteers by ultrasonography before and immediately after the session. Their study did not include a control group.

We set out to examine the potential development of these characteristics in the first 30 minutes after high-intensity tendon loading. We hypothesized that tendon thickness would increase immediately after high-intensity resistance training involving the patellar and Achilles tendons. Second, we hypothesized that tendon morphology would change immediately after the exercises and that this would lead to an altered distribution of the ultrasonographic signal of the tendon matrix.

**METHODS**

The study was approved by the regional ethics committee, and volunteers signed informed consent forms before participating. The study was designed as a randomized, investigator-blinded trial between an experimental group and a sham group.

**Power Analysis**

A priori power analyses were computed using G\(^*\)Power 3.1 (Heinrich Heine Universität Düsseldorf) on the basis of the results of Fisker et al.\(^5\) An a priori power analysis for repeated-measures analysis of variance (ANOVA) (effect size [$\eta^2$] = 0.69; correlation between repeated measures = 0.5; power = 0.90) demonstrated that 16 volunteers would be needed in total. An a priori power analysis for the $t$ test, comparing the first time point against baseline in the experimental group alone (mean difference = 4.70 ± 0.34; power = 0.90), demonstrated that 8 volunteers would be needed for reproducing the findings in the experimental group.

**Participants**

A total of 18 healthy volunteers were included in the study, producing an actual power of 0.95. The inclusion criteria were age between 18 and 40 years and ability to complete the experimental and sham exercises. Volunteers with clinically diagnosed tendinopathy or with subjective experiences of pain or any other subjective symptom of the knees or ankles were excluded. Baseline tendon thickness above 5 mm was not an exclusion criterion because increased tendon thickness is only suggestive of the future development of tendinopathy and is not diagnostic.

Volunteers were recruited from a local gymnasium offering high-intensity resistance CrossFit training. To ensure that the volunteers had sufficient capacity to perform at the required intensity, the experimental exercises included a particular difficult jump-rope exercise, double-under jump-rope jumps, meaning that only experienced athletes would be able to participate. Age, weight, and height were registered. The 18 volunteers were randomized into 2 groups using block randomization, ensuring not exact but close to equally (±2) large groups.

**Experimental and Sham Exercises**

The experimental exercises were designed to evaluate the effects of high-intensity resistance training involving knees and ankles. The sham exercises were designed as a comparable high-intensity resistance training protocol without any involvement of knees or ankles. The purpose of the sham exercises was to induce the physiological response to exercise (increased sweating, increased cardiac output, etc). This enabled the study to distinguish between effects elicited by high-intensity loading of particular tendons and the general physiological changes from high-intensity resistance exercise. The investigators (M.M.T., I.J.) who operated the scanners were blinded to the randomization and were not present during the performance of the exercises.

**Experimental Exercises.** Participants completed 5 rounds of 5 weighted front squats (weight: male, 50 kg; female, 30 kg), 10 box jumps (height: male, 60 cm; female, 50 cm), and 15 double-under jump-rope jumps. This protocol is identical to the experimental protocol applied in an earlier pilot study.\(^5\) For the front squat (Figure 1A), a barbell was supported on the chest and shoulders, with the shoulders at $90^\circ$ of flexion and the elbows at full flexion. From a standing position, the participant descended by flexion of the hip and knees. The descent was continued until the femurs were horizontal. The participant then immediately returned to the standing position. For the box
Figure 1. Experimental and sham exercises: (A) weighted front squat, (B) box jump, (C) double-under jump-rope jump, (D) weighted shoulder press, (E) push-up, and (F) weighted biceps curl. (G) Diagram illustrating the acquisition of ultrasonography data, enabling the imaging of Achilles and patellar tendons simultaneously. (H) Display showing how a paper was taped to the screen to blind the results of the measurements. (I) Ultrasonography screenshot showing measurements of the patellar tendon thickness and placement of the region of interest (ROI) in the tendon matrix. (J) Ultrasonography screenshot showing measurements of the Achilles tendon thickness and placement of the ROI in the tendon matrix. The distances from the bony prominence on the calcaneus and patella to the ROI center are 20 mm and 5 mm, respectively.
jump (Figure 1B), from a standing position in front of the designated box, the participant jumped onto and stood on the box, with the hips and knees in full extension. He or she then jumped down from the box and immediately jumped onto the box again in a plyometric manner. For the double-under jump-rope jump (Figure 1C), the participant performed jump-rope jumps, which were high enough for the rope to pass twice for each jump.

Sham Exercises. The participants in the sham group completed 5 rounds of 5 weighted shoulder presses (weight: male, 40 kg; female, 20 kg), 10 push-ups (body weight), and 15 weighted biceps curls (weight: male, 20 kg; female, 10 kg). For the weighted shoulder press (Figure 1D), in a standing position with the barbell supported on the chest, the participant grabbed the barbell and pressed it to full elevation. The knees were fully extended for the entire movement. From full elevation, the barbell was lowered until contacting the chest again, completing 1 repetition. For the push-up (Figure 1E), in a prone position with fully extended knees, the participant pushed to full extension with the elbows and 90° of flexion in the shoulders. He or she descended until the chest made contact with the floor, completing 1 repetition. For the weighted biceps curl (Figure 1F), with the barbell grasped in a supine grip, the participant lifted the barbell with fully extended elbows until contact with the chest.

This protocol was designed to avoid any loading of the Achilles and patellar tendons while aiming for a similar duration.

Ultrasonography

Two identical scanners (Noblus [Hitachi] with a 44-mm EUP-L75 linear probe and 5- to 18-MHz transducers) were operated by 2 investigators (M.M.T. and I.J.) to simultaneously obtain sonograms of the patellar and Achilles tendons. Each operator was dedicated to either the Achilles tendon or patellar tendon and had 2 to 3 years of experience in ultrasonography of tendons through everyday practice and earlier ultrasonography studies.

The protocol was arbitrarily designed so that the left tendons were always scanned first and the right tendons second. This was to ensure approximately the same amount of time between the scans of each tendon. The alternative would have been to alternate between which tendon was scanned first, which in turn would change the intervals between the scans of each respective tendon.

Longitudinal scans were performed, and tendon thickness was measured during the scanning session. The result of each measurement was blinded to the investigator during scanning. Hence, the investigator would not be able to deliberately and consistently overestimate or underestimate any measurements. This was achieved by taping a piece of paper to the display, covering the caliper measurements. The measurements were then afterward noted by another investigator (Figure 1H).

Scans were obtained at baseline. Two scans of each tendon were performed. The ultrasonography operators moved to an adjacent room, while a third investigator supervised the exercises being performed as prescribed. Immediately on completion, the 2 operators were called to the room, and the participant was then scanned immediately at a time point defined as 0 minutes. From here, the participant was again scanned at the following time points: 2.5, 5, 7.5, 10, 15, 20, 25, and 30 minutes. The reason for the increase in interval length was to accommodate the need for extending the legs and sitting up to drink water after 10 minutes. Participants were not allowed to stand or stretch during the 30 minutes.

With the participant in a supine position with the knees bent at 90°, a longitudinal patellar tendon scan was obtained. The measurement of thickness was performed 5 mm distal to the patellar apex. A longitudinal scan of the Achilles tendon was obtained simultaneously, with the participant in the same position and with the ankles in a supported in situ position at 90°. The measurement of thickness was performed 20 mm proximal to the tendon insertion on the calcaneus. The setup is illustrated in Figure 1G, and the measurements are shown in Figures 1I and 1J. The protocol was equal to that of the pilot study and was based on the evidence of its reproducibility.5,7

Data Analysis

Intraoperator Variability. We performed blinded intraoperator analyses for each operator using the 2 baseline scans of the same tendon. Because each operator was dedicated to either the Achilles or patellar tendon, we did not perform interoperator analyses.

Thickness Analysis. The thickness of the tendon was measured during the scanning session. The baseline thickness was defined as the mean of the 2 baseline scans of the same tendon. The baseline thickness was subtracted from the thickness at each time point to yield the increase in tendon thickness.

Signal Analysis. On the basis of the marks of the caliper on the sonogram, a circular region of interest (ROI) was defined (Figures 1I and 1J). The circle was drawn so that the diameter corresponded to the 2 marks from the caliper, meaning that the diameter was the same as the tendon thickness in that given sonogram. This eliminated any potential influence of the analyzing investigator. The distribution of pixels in the 8-bit grayscale image in the ROI was then obtained, yielding histograms and mean values. The grayscale ranged from 0 to 255, with 0 being black (no signal) and 255 being white (strongest echo signal). Letting the ROI size depend on the tendon thickness ensured that it sampled the entire tendon matrix for analysis.

Statistical Analysis

All image analyses were performed using Fiji software (Laboratory for Optical and Computational Instrumentation, University of Wisconsin–Madison).25 Calculations, statistical analyses, and charts were made in Excel (Microsoft) and XLSTAT (Addinsoft).
TABLE 1
Participant Characteristics

|                | Experimental Group | Sham Group | P   |
|----------------|--------------------|------------|-----|
| Sex, male/female, n | 8/2                | 5/3        | —   |
| Age, y          | 24.89 ± 2.89       | 28.00 ± 6.00 | .19 |
| Height, cm      | 175.89 ± 6.57      | 179.29 ± 12.08 | .48 |
| Weight, kg      | 75.63 ± 4.10       | 77.42 ± 15.90 | .76 |
| Training per week, h | 7.50 ± 2.56       | 6.60 ± 2.61 | .55 |

aData are presented as mean ± SD unless otherwise specified. P values are for the Student t test.

Statistical analysis was performed as repeated-measures mixed ANOVA with 2 factors, time and group (experimental vs sham), with 10 and 2 levels, respectively. After performing ANOVA, post hoc analyses were performed between the given time point and baseline within the given group.

To correct for multiple linear comparisons and type I errors, the Bonferroni correction was performed for this part of the analysis. Correction for 9 comparisons yielded a significance level of .006; hence, P values < .006 were considered statistically significant. Confidence intervals in the analyses of tendon thickness and mean grayscale value are therefore also corrected. Left and right tendons were averaged before performing statistics, meaning that n values in all tests are equal to the number of volunteers and not double.

Analyses were performed on Achilles and patellar tendons independently. The Student t test was performed for height, age, weight, and workout times between groups.

RESULTS

Of the 18 volunteers, 10 were randomized to the experimental group and 8 to the sham group. Participants’ age ranged from 21 to 39 years, with a mean of 26.5 years. Their weight ranged from 55 to 96 kg, with a mean of 76.5 kg, and their height ranged from 161 to 195 cm, with a mean of 177.4 cm. Hours of weekly training ranged from 3 to 12 hours, with a mean of 7.1 hours. Volunteers were recreational athletes, with 2 competing at an amateur level. Comparisons between groups on these data are presented in Table 1. Sham exercises were performed in a nonsignificantly shorter time (time difference: 108 seconds; 95% CI, −0.02 to 103.01; P = .068).

Intraoperator Variability

For tendon thickness, there was a mean error of 0.05 ± 0.22 mm for the patellar tendon operator and 0.06 ± 0.28 mm for the Achilles tendon operator. As for the signal change, the patellar tendon operator had a mean error of 4.38 ± 10.37, and the Achilles tendon operator had a mean error of 5.30 ± 8.05. No tendencies were found from Bland-Altman plots.

Patellar Tendon Thickness

The analysis found that there was no interaction between time and group (P = .957). Neither were there any significant main effects of group (P = .055) or time (P = .742), and hence neither were significant explanatory variables. The Bonferroni-corrected post hoc analysis showed that the patellar tendon thickness did not change significantly from baseline at any time point in either group (Figure 2). The baseline patellar tendon thickness was not significantly different between the 2 groups (P = .082).

Achilles Tendon Thickness

The analysis found that there was no interaction between time and group (P = .513). Neither was there any significant main effect of group (P = .402). However, there was a significant main effect of time (P = .007), suggesting that time could be responsible for the change in signal alone.

The Bonferroni-corrected post hoc analysis showed that the mean grayscale value of the ROI in the experimental group increased after the exercises at all time points, reaching a significant level at 7.5 minutes with an increase of 10.8 (99.4% CI, 3.7–17.9; P = .002). The baseline patellar tendon grayscale distribution was not significantly different between the 2 groups (P = .520).

In the sham group, there was no significant increase in the mean value of the ROI. However, there might have been a tendency toward an increase in the signal of the sham group as well.

Patellar Tendon Matrix Signal Distribution

The analysis found that there was no interaction between time and group (P = .513). Neither was there any significant main effect of group (P = .402). However, there was a significant main effect of time (P = .007), suggesting that time could be responsible for the change in signal alone.

The Bonferroni-corrected post hoc analysis showed that the mean grayscale value of the ROI in the experimental group increased after the exercises at all time points, reaching a significant level at 7.5 minutes with an increase of 10.8 (99.4% CI, 3.7–17.9; P = .002). The baseline patellar tendon grayscale distribution was not significantly different between the 2 groups (P = .520).

Achilles Tendon Matrix Signal Distribution

The analysis found that there was no interaction between time and group (P = .101). Neither was there any significant main effect of group (P = .853). However, there was a significant main effect of time (P = .0001), suggesting that time could be responsible for the change in signal alone.

From the Bonferroni-corrected post hoc analysis, we found that the signal of the ROI did not change significantly in the Achilles tendon during the 30-minute time course when compared with baseline within the given group. The baseline Achilles tendon grayscale distribution was not significantly different between the 2 groups (P = .799).
DISCUSSION

This trial was designed to reproduce the results of Fisker et al., who showed that tendon thickness increased during a high-intensity resistance training protocol in volunteers. Our trial failed to reproduce the same increase in tendon thickness immediately after replicating the same exercise protocol. In contrast to the Fisker et al., study, which was a nonblinded cohort study without any control group, the present study applied blinding and contained a sham group. Furthermore, the Fisker et al. study only had measurements at 1 time point after the intervention, whereas our study had 9 measurements after the session and 2 at baseline. Because of the randomized, blinded trial design in which operators were completely blinded to the groups and to the caliper on the sonogram, this trial has a markedly lower potential for involuntary operator bias, making type I errors very unlikely. Obviously, it does not exclude the possibility of type II errors. However, our power calculations suggest that we were well powered to reproduce the effects on tendon thickness, making type II errors less likely as well.

A small tendency toward a decrease in thickness of the patellar tendon was observed. This could be in concordance with the results of Kristiansen et al. and Syha et al. These studies have shown decreased cross-sectional areas (CSAs) after 4 hours of heavy resistance training. Their protocol is obviously not directly comparable with ours, but the mechanisms that produce the effects could be the same. Mechanical deprivation of fluids from the tendon is a likely explanation.

Habitual loading of tendons through resistance training and sports has been shown to increase CSAs of the tendons in the long term. This implies that an increase in tendon CSA or thickness could be a physiological beneficial adaptation to training, which might augment strength as a result of the increased requirements of heavy loads. However, this also leaves us with a paradox: When are increases in tendon CSA or thickness pathological, and when are they physiological responses? Hence, more complex measures than thickness and CSA are needed to differentiate between the 2 states.

From our analysis, we found that the variable of time is explanatory of the changes observed. We did not find that

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**Figure 2.** Thickness values measured at all time points for the (A) patellar tendon and (B) Achilles tendon. Grayscale values measured at all time points for the (C) patellar tendon and (D) Achilles tendon. The first time point is baseline. Gray: experimental group; black: sham group. Error bars: 99.4% CI. The star in (C) indicates significant difference from baseline for the experimental group.
the experimental group itself significantly explained the changes. Within the time variable lie the effects of immediate physiological adaptations to increased work, which were present in both groups. Our study design did not allow for further differentiation of this variable, and we can only observe that completing the work prescribed in both groups seems to affect the tendon signal.

We found a significant increase in the signal of the patellar tendon after the experimental protocol. The lack of significant changes in the Achilles tendon and the sham group is probably the consequence of our strict Bonferroni correction and a type II error because ANOVA clearly did not find any significance of the experimental protocol.

A potential explanation of the observed changes could be artifacts from increased blood flow in the skin, changing the ability of the skin to transmit ultrasonic waves. However, one would expect this change to be evident immediately after the completion of work, if the duration of work exceeds the time for these skin adaptations to occur. Our data suggest that the increase in tendon signal is delayed and occurs approximately 20 minutes after the exercises start. This might reflect the fact that adaptations in skin blood flow occur at this time point.

Another explanation could be stretching of the tendons with mechanical pressure, depriving the tendons of water. Again, this would be expected as an immediate mechanical effect, which then should be at its maximum at the first time point.8

Studies using magnetic resonance imaging (MRI) have shown changes in tendon matrix signals as well.25,26 Unfortunately, these studies did not include sham controls, and comparing signal changes between these 2 modalities should be done with caution; however, these studies found that water content in the tendons is probably lowered because of exercise.

Solid matters are physically more echogenic than water, meaning that proteins and other structural molecules not in solution are hyperechoic. The hyperechoic signal could be explained by infiltration of the tendon by inflammatory nuclear cells because the nuclei would increase the echo signal. However, this would not be consistent with histopathological findings, suggesting that acute inflammation is not part of the pathophysiology.6 Furthermore, it would be expected that such infiltration would be accompanied by edema, which would result in a hypoechoic signal. Our findings are therefore in support of the hypothesis that acute inflammation is not part of the pathophysiology. Obviously, our study does not draw conclusions on this matter because it is outside the scope of the study design.

The change in signal could also be the result of changes in metabolism and potentially autoregulatory mechanisms changing the water content. This hypothesis could be explained by the fact that tendon tissue is metabolically active.9

A study by Rutkauskas et al22 suggested that strenuous exercise might produce intratendinous bubbles, which are visualized as hyperechoic areas. We did not observe macroscopically visual bubbles in our sonograms, but microscopic air bubbles could explain the hyperechoic signal.

Strengths and Limitations

Even though the sham exercises were designed to bypass the use of the patellar and Achilles tendons, one may argue that the tendons were loaded compared with a resting state. Both tendons were loaded to some extent because of the standing position of 2 of the sham exercises and especially the prone position of the push-ups. However, the tendons were not loaded in a concentric/eccentric manner. This might explain the inability of our study to show statistical differences between groups because the tendons of the sham group may have been sufficiently loaded to diminish a difference between groups. A redesign of the study exercises, letting the participants sit down for the entire study, could have been applied, and this presents a clear limitation to the current study design.

The study participants were highly selective group of well-trained persons, which means that they will have a higher degree of adaptations to the loads to which they are subjected. The greater overall tendon strength of our participants could explain why we did not see any changes in tendon thickness. Conducting the study in a cohort of non-trained persons might yield a different result. However, we chose to include well-trained persons, who regularly subjected themselves to comparable workloads, to minimize the risk of inflicting acute injuries to our study volunteers.

The general hydration state of the participants could in theory affect signal change. Nevertheless, both groups underwent exercise for such a short duration that it seems very unlikely that sweat production would produce a significant decrease in the systemic hydration state, which in turn again would decrease the water content of the tendon.

Ultrasonography is both an operator- and observer-dependent modality, meaning that operating the scanner and the observing part including caliper measurements are prone to variability. Our intraoperator analyses showed this as well and found that the mean error was lower than the changes observed. However, the mean error was found to be in the magnitude of the changes observed, and hence we cannot reject that the observed changes could be artifacts of noise and insecurity. These are obvious disadvantages of the modality. To compensate for this inherent variance in the modality, this study was designed as a randomized, blinded study with multiple repeated measures.

In contrast to other imaging modalities such as MRI, ultrasonography can be performed immediately after exercise and with a high resolution in time, which makes it optimal for imaging acute physiological and pathological changes within the tendon matrix. A comparable study could have been conducted using MRI. However, MRI has some obvious limitations in terms of latency from workout completion because of shimming and planning of the scan. Furthermore, the duration of the scan itself would produce a problem, not allowing for the same resolution in time.

In our pilot study,5 we decided to apply fixed weights for all volunteers for the front squats, only adjusting for sex. One could argue that a protocol with the weight defined by a percentage of 1-repetition maximum would yield a higher degree of standardization. However, because our primary
end point is more likely to be dependent on tendon strength rather than muscle power, and because 1-repetition maximum would merely be a surrogate marker of tendon strength, this standardization would not be optimal either. Optimal, one would standardize the weight to tendon strength, which, however, is not a measurement we were able to obtain.

CONCLUSION

Patellar and Achilles tendon thickness did not increase immediately after 1 session of high-intensity resistance training. The tendon tissue matrix produced a hyperechoic signal after high-intensity resistance training. This effect seems to be independent of loading of the tendon and could be an artifact of noise or a consequence of immediate physiological adaptations to increased work. The design of this study and the modality used did not allow for further explanations of our findings beyond the above and should only be regarded as observations that need further studies to be explained.

There is a need for prognostic and diagnostic markers of tendinopathy, particularly because of the protracted course of the subclinical development of injuries. A basic quantitated description of changes using readily accessible modalities, such as those shown in this study, could be of great importance in developing future prognostic markers or tests of potential tendinopathy. With still better ultrasonography equipment being developed and with the possibility of conducting 3-dimensional arrays, sensitivity and reproducibility will increase, making ultrasonography a prime candidate for exploring this field of study.

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