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Improving Highly Accelerated Fat Fraction Measurements for Clinical Trials in Muscular Dystrophy: Origin and Quantitative Effect of R2* Changes

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### Purpose:
To investigate the effect of R2* modeling in conventional and accelerated measurements of skeletal muscle fat fraction in control subjects and patients with muscular dystrophy.

### Materials and Methods:
Eight patients with Becker muscular dystrophy and eight matched control subjects were recruited with approval from the Newcastle and North Tyneside 2 Research Ethics Committee and with written consent. Chemical-shift images with six widely spaced echo times (in 3.5-msec increments) were acquired to correlate R2* and muscle fat fraction. The effect of incorporating or neglecting R2* modeling on fat fraction magnitude and variance was evaluated in a typical three-echo protocol (with 0.78-msec increments). Accelerated acquisitions with this protocol with 3.65×, 4.94×, and 6.42× undersampling were reconstructed by using combined compressed sensing and parallel imaging and fat fraction maps produced with R2* modeling.

### Results:
Muscle R2* at 3.0 T (33–125 sec⁻¹) depended on the morphology of fat replacement, the highest values occurring with the greatest interdigitation of fat. The inclusion of R2* modeling removed bias, which was greatest at low fat fraction, but did not increase variance. The 95% limits of agreement of the accelerated acquisitions were tight and not degraded by R2* modeling (1.65%, 1.95%, and 2.22% for 3.65×, 4.94×, and 6.42× acceleration, respectively).

### Conclusion:
Incorporating R2* modeling prevents systematic errors in muscle fat fraction by up to 3.5% without loss of precision and should be incorporated into all muscular dystrophy studies. Fat fraction measurements can be accelerated fivefold by using combined compressed sensing and parallel imaging, modeling for R2* without loss of fidelity.

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**Online supplemental material is available for this article.**
M uscular dystrophies are a heterogeneous group of genetic diseases characterized by progressive muscle weakness and wasting. Over the course of the disease, skeletal muscle is progressively replaced by fat and fibrosis. There are currently no curative treatments, but several therapeutic approaches for Duchenne muscular dystrophy, the most common form of muscular dystrophy, have entered clinical trials (1). The primary end points of these trials are presently based on assessments of muscle function and strength, but these tests depend on patient motivation and engagement. Quantitative magnetic resonance (MR) chemical-shift imaging techniques, on the other hand, allow accurate, objective fat fraction analysis of muscle fat replacement (2) and are starting to be specified as trial end points. Quantitative MR imaging has been applied in cross-sectional cohort studies (3–9) and longitudinal studies of disease progression (10,11) to measure muscle fat fraction, and multicenter trial protocols have been issued (12).

Despite the increasing number of studies in muscular dystrophy, none of these model the effects of R2* relaxation (the reciprocal of T2*) on the fat fraction (3–7,10–12), though investigators in two studies have used a global R2 relaxation correction (8,9) extrapolated from values measured in control muscle. It has generally been assumed that R2* is relatively low compared with the echo times used and does not change greatly in these slow-progressing diseases; however, this has not been specifically evaluated, even in those studies, by using multipake fat modeling (6,8,9). This situation possibly arises because the research groups performing the clinical studies in muscular dystrophy are distinct from those who developed the algorithms to compute fat fractions that incorporate R2*, who concentrated on the quantification of hepatic steatosis (13–15). When measuring fat fraction in the liver by using gradient-echo sequences, it is recognized that iron overload can alter R2* by more than an order of magnitude (16) but that increasing diffuse hepatic steatosis without iron overload is not associated with R2* change where multipake models of lipid are used (13).

Recently, fivefold accelerated acquisition of whole-muscle fat fraction data has been demonstrated in muscular dystrophy, combining compressed sensing and parallel imaging reconstruction (17) but not modeling R2* relaxation. These techniques have the ability to save expensive imaging unit time, improve patient compliance (important for children), and make the incorporation of quantitative MR imaging into trial protocols more feasible. In our study, we considered the effect of incorporating R2* modeling when reconstructing fat fraction maps from accelerated imaging. We considered the relationship between fat fraction and R2* in healthy volunteers and patients with muscular dystrophy, illustrating the origins of R2* change in muscular dystrophy and the effect on fat fraction measurements in clinical trials, where multipake models of lipid are used. The aim of our study was to investigate the effect of R2* modeling in conventional and accelerated measurements of skeletal muscle fat fraction in control subjects and patients with muscular dystrophy.

Materials and Methods

Patients
Eight subjects (necessarily all men; mean age ± standard deviation, 31 years ± 12; age range, 21–56 years; weight, 76 kg ± 15; body mass index, 25 kg/m² ± 4) with a genetically proven diagnosis of Becker muscular dystrophy were recruited through the clinics of the Medical Research Council Centre for Neuromuscular Diseases at

Advances in Knowledge

- MR imaging fat fraction measurements in muscular dystrophy can be prospectively highly accelerated to save time with modeling of R2* relaxation (95% limits of agreement, 1.65%, 1.95%, and 2.22% for 3.65×, 4.94×, and 6.42× accelerations, respectively).
- Neglecting R2* modeling leads to protocol-dependent inaccuracy in fat fraction, which is a strong function of the fat fraction (the difference in fat fraction obtained when neglecting R2* modeling has a slope of −0.053 and an intercept of 3.41, both P < .001).
- Modeling R2* relaxation in dystrophic muscle does not increase the variance of the calculated fat fraction compared with the underlying physiological variance (ratio of fat fraction standard deviation calculated with R2* modeling compared with no R2* modeling is 0.95 ± 0.19 [not significant]).

Implications for Patient Care

- Volumetric muscle fat fraction measurements with R2* modeling can be significantly accelerated with combined compressed sensing and parallel imaging reconstruction, reducing imaging cost and patient burden in muscular dystrophy clinical trials.
- In clinical trials of muscular dystrophy, investigators who use MR imaging as an outcome measure need to model for R2* relaxation rate to eliminate bias in fat fractions.
Newcastle. Inclusion criteria included independent ambulation and no contraindications to MR imaging (e.g., pacemaker, aneurysm clip, or claustrophobia). Eight age-matched healthy male control subjects (mean age, 32 years ± 12; range, 21–55 years; weight, 78 kg ± 9; body mass index, 24 kg/m² ± 4) were also recruited. The study, performed between May 2013 and April 2014, obtained a favorable opinion from the Newcastle and North Tyneside 2 Research Ethics Committee, with all subjects giving written informed consent. K.G.H. had control of the study data. Some of the data obtained from the eight patients with Becker muscular dystrophy have been analyzed previously without R2* modeling (17) in a technical study that concentrated on optimizing parameters in the reconstruction of the under-sampled data: The overlap is detailed in the following subsections. Subjects lay supine, and their legs and feet were immobilized. D.M.H., an employee of Philips Healthcare, provided specialist advice to K.G.H. on reprogramming the MR imaging unit to perform undersampling. K.G.H., an employee of Newcastle University, had complete control of and responsibility for the modifications to the imaging system, data acquisition, processing, and study inclusion.

**Relationship between R2* and Fat Fraction of Dystrophic and Control Muscle**

A 3.0-T Philips Achieva and six-channel cardiac coil (Philips, Best, the Netherlands) were used. A three-dimensional gradient-echo acquisition was used for patients with Becker muscular dystrophy and control subjects, with a matrix size of 256 read × 190 phase × 48 sections, field of view of 320 mm (right-left) × 190 mm (anterior-posterior) × 240 mm (foot-head) (or 285-mm anterior-posterior for four larger subjects), and resolution of 1.25 × 1 × 5 mm (or 1.25 × 1.5 × 5 mm). Six unipolar echoes per repetition time were acquired, with repetition time of 22 msec and echo times of 2.0, 5.5, 9.0, 12.5, 16.0, and 19.5 msec; flip angle, 5°; receiver bandwidth, 111 kHz; and acquisition time, 198 seconds. The widely spaced echo times used (with 3.5-msec increments) allowed accurate characterization of a wide range of R2* values and water-fat separation. Two imaging blocks were acquired: The first was centered in the mid-lower leg, defined as the part of the calf muscle with the largest circumference, and the second was in the thigh, determined by locating the superior border of the patella, and the anterior superior iliac spine, centering one-third of the distance superior to the patella. Data were acquired from patients and control subjects for both legs with the read direction left-right.

The chemical-shift separation into water and fat images was performed by using the “graphcut” algorithm previously described by Hernando et al (18,19) with a single R2* component. This algorithm was robust to water/fat swapping when using wide echo spacing. It should be noted that we only proposed to model one R2* component, and it is more generally true that the fat and water present in a pixel may have independent R2* values. However, it has been shown for liver steatosis measurement that modeling two independent components led to instability and noise breakthrough in the resulting fat fraction maps and that the remaining bias in a one-component fit was small (20,21), even if the R2* of water and fat species were markedly different. A six-component fixed-spectrum model was used to represent fat (22). Proton-density fat fraction maps were generated, avoiding noise bias (23).

By using ImageJ (National Institutes of Health, Bethesda, Md), regions of interest (ROIs) were drawn by K.G.H. on 10 axial sections per anatomic area at midsection on the left leg. The areas delineated at these levels included the tibialis anterior, soleus, medial and lateral gastrocnemius muscles, and bone marrow of the tibia, as a structure composed principally of fat. In the thigh, we outlined the rectus femoris, vastus lateralis, vastus intermedius, biceps femoris long head, semitendinosus, semimembranosus, and sartorius muscles. The regions were defined on the sum of the water and fat images. The mean fat fractions and R2* relaxation rates were evaluated and averaged for each muscle group for each subject, providing 192 data points for a comparative plot. The mean fat fractions and R2* relaxation rates of the healthy control subjects and patients with Becker muscular dystrophy were tested for significance by using a Mann-Whitney U test, for both individual muscles and overall.

Intrarater (repeatability) and interrater (reproducibility) variation of the ROI analysis was assessed in all regions of four randomly chosen patients with Becker muscular dystrophy. T.L. and K.G.H. marked and evaluated all the regions independently; K.G.H. additionally made a further marking of all the ROIs 3 weeks later without reference to his original regions. In both cases, Bland-Altman analysis (24) was used to evaluate the bias and 95% limits of agreement between the paired fat fractions and R2* values.

**Comparison of Fat Fractions Evaluated with and without R2* Modeling**

A further three-dimensional spoiled gradient-echo stack was acquired in the patients and control subjects by using the field of view and matrix size detailed earlier, with three interleaved gradient echoes with narrow echo spacing (0.78-msec increments) to optimize the fat fraction measurement (repetition time of 10 msec and echo times of 4.40, 5.18, and 5.96 msec; flip angle, 3°; one echo acquired per repetition time; bandwidth, 55.5 kHz; and acquisition time, 273 seconds). Such narrow echo spacing is typical of protocols used in muscular dystrophy studies (3–11).

Fat fraction maps were produced by using the algorithm of Tsao and Jiang (19,25), a fast algorithm robust for fat water separation with narrow echo time spacing, with the same fat model and noise bias correction as given earlier, both with and without single component R2* compensation. ROI analysis was performed as detailed earlier. A Bland-Altman plot was constructed for the difference in fat fraction with and without R2* modeling and the mean fat fraction for all muscle groups. For comparisons of differences in fat fraction for specific
The Relationship between $R_2^*$ and Fat Fraction of Dystrophic and Control Muscle

The $R_2^*$ relaxation rates plotted against the fat fraction of each muscle group for the patients with Becker muscular dystrophy and control subjects are shown in Figure 1, with representative proton-density fat fraction and $R_2^*$ maps shown in Figure 2. The $R_2^*$ value increased and then decreased again as fat fraction increased from 0% to 100%. Note that tibial bone marrow has fat fraction close to 100% for both control subjects and patients. For control muscles (the letter A on Fig 2) and patient muscles with no fat replacement, the $R_2^*$ was approximately 40 sec$^{-1}$. The mean fat fraction and $R_2^*$ ($\pm$ standard deviation) for control subject muscles were 3.2% $\pm$ 1.2 and 41.5 sec$^{-1} \pm$ 3.8 and for patients were 39.4% $\pm$ 35.4 and 54.2 sec$^{-1} \pm$ 16.6, respectively (both $P < .001$ compared with control subjects).

In dystrophic muscle with substantial interdigitation of fat and water, $R_2^*$ was increased: In the thigh of a patient with Becker muscular dystrophy, especially high values of $R_2^*$ occurred (17), summarized in Appendix E1 (online). Three echoes with narrow echo spacing (0.78-msec increments) were used, as in the previous section. Data with net accelerations of 3.65×, 4.94×, and 6.42× were acquired prospectively. The acceleration factors were determined through choosing three different minimum mean separations of k-space points in the sampling pattern; the same undersampling pattern was used for each echo time. The undersampled acquisition times were 75 seconds, 55 seconds, and 43 seconds, respectively. A coherent (regular) 3.36× undersampling for reconstruction with parallel imaging only was acquired.

The undersampled data were reconstructed with combined compressed sensing and parallel imaging (26), where the optimization of the algorithm parameters required has been demonstrated in detail previously (17), summarized in Appendix E1 (online). Fat-water separation involved use of the algorithm of Tsao and Jiang (19,25) with single-component $R_2^*$ modeling. The data from the patients with Becker muscular dystrophy were previously processed (17) without $R_2^*$ modeling to demonstrate reconstruction optimization.

The fat fractions generated by the reconstructions from undersampled data were compared with those from the fully sampled data with standard reconstruction (inverse Fourier transform and phase-sensitive coil combination). By using Bland-Altman analysis (24), measures of bias and the 95% limits of agreement between the measurements were calculated.
with diffuse fat infiltration. Similar patterns of interdigitated fat and significantly elevated R2* were seen in the vastus intermedius (the letter E on Fig 2, 85.8 sec⁻¹) and vastus lateralis of another patient (the letter F on Fig 2, 81.5 sec⁻¹).

Once the fat replacement increased beyond approximately 75%, the fat appeared as a continuous area, and the R2* decreased once more (such as the letter G on Fig 2, 39.9 sec⁻¹, where the lateral and medial gastrocnemii have been completely replaced by fat). The R2* of muscles mostly replaced by fat, bone marrow, and subcutaneous fat tended toward 40 sec⁻¹ or lower (Fig 1). With 192 data points from 16 individuals, there will be within-patient correlation especially in control subjects, though the muscle groups of patients with Becker muscular dystrophy can show marked variation.

The repeatability assessment of fat fraction showed a negligible bias between the two occasions (0.04%, not significant), and the 95% limit of agreement was 1.40% (±0.4 sec⁻¹ and 2.0 sec⁻¹ for R2*, respectively). The reproducibility assessment again shows negligible bias between the two analysts (0.04%, not significant), and the 95% limit of agreement was 1.52% (±0.03 sec⁻¹ and 2.8 sec⁻¹ for R2*, respectively).

Comparison of Fat Fractions Evaluated with and without R2* Modeling

Figure 3 shows the fat fractions for the patients with Becker muscular dystrophy derived with R2* compensation compared with those derived without, presented against the line of equality (Fig 3, A) and as a Bland-Altman plot (Fig 3, B). The overall trend was that neglecting R2* modeling led to overestimation of fat fraction at low fat fraction by as much as 3.5%, but the effect disappeared at moderate fat fractions with underestimation at high fat fraction. Table 1 shows the effect of R2* modeling for the muscles that we highlighted in Figure 1. While Figure 3, B, shows nonlinear differences for fat fractions close to 0% or 100%, a regression to the linear portion of the plot (between 10% and 90%) yields \( y = -0.0527x + 3.41 \) (both coefficients, \( P < .001 \)). The effect on the fat fraction was most significant for the lowest fat fraction (the letter A on Fig 2) with an R2* value of 36.4 sec⁻¹ (\( P < .005 \)), where there was 2.3% overestimation. The muscle group with the highest R2* observed (the letter C on Fig 2) showed no significant difference in fat fraction whether R2* was modeled or not.

The mean ratio of the standard deviation of fat fraction within each ROI with R2* fitting compared with no R2* fitting was 0.95 ± 0.19 (\( F \) test results, not significant), indicating that the fitting of the extra parameter did not significantly increase the variance of fat fraction within an ROI compared with the underlying physiological variability in the measurement, as has been theoretically demonstrated for uniform tissue (14). R2* modeling can be added to the precision of the resulting fat fractions in muscular dystrophy without substantial cost.

Accelerated Fat Fraction Measurements with Combined Compressed Sensing and Parallel Imaging Reconstruction and R2* Modeling

Figure 4 shows an example of the reconstructed fat fraction images for two patients with muscular dystrophy, showing a high quality of reconstruction with R2* modeling with combined compressed
**Table 1**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>$R^2$ (sec$^{-1}$)</th>
<th>Fat Fraction without $R^2$ Modeling (%)</th>
<th>Fat Fraction with $R^2$ Modeling (%)</th>
<th>Difference in Fat Fraction (%)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>36.4</td>
<td>3.8</td>
<td>1.5</td>
<td>2.3</td>
<td>.005</td>
</tr>
<tr>
<td>B</td>
<td>90.5</td>
<td>27.0</td>
<td>25.9</td>
<td>1.1</td>
<td>.005</td>
</tr>
<tr>
<td>C</td>
<td>128.1</td>
<td>66.8</td>
<td>66.7</td>
<td>0.1</td>
<td>NS</td>
</tr>
<tr>
<td>D</td>
<td>83.0</td>
<td>52.5</td>
<td>51.5</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>E</td>
<td>85.8</td>
<td>62.0</td>
<td>61.3</td>
<td>0.7</td>
<td>.007</td>
</tr>
<tr>
<td>F</td>
<td>81.5</td>
<td>73.7</td>
<td>75.2</td>
<td>1.5</td>
<td>.005</td>
</tr>
<tr>
<td>G</td>
<td>59.9</td>
<td>82.5</td>
<td>83.9</td>
<td>1.4</td>
<td>.005</td>
</tr>
</tbody>
</table>

Note.—The $P$ value shows the significance associated with the difference in fat fraction by using a Wilcoxon paired test. NS = not significant. The muscle groups A–G are annotated in Figures 1 and 2.
TECHNICAL DEVELOPMENTS: Improving Highly Accelerated Fat Fraction Measurements in Muscular Dystrophy

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Figure 4: Fat fraction maps in two patients with Becker muscular dystrophy were generated from data acquired with full sampling and 3.65×, 4.94×, and 6.42× accelerated acquisition.

with control subjects. This stood in marked contrast to measurements in diffuse hepatic steatosis, where, for patients unaffected by hepatic iron overload, R2* was independent of the degree of steatosis (13), provided that a multi-peak lipid model was used.

As demonstrated, not modeling for R2* relaxation introduced bias, which was particularly acute at low values of fat fraction, where the greatest overestimation occurred. This bias will be dependent on the choice of echo times and field strength used, preventing comparison between study sites where identical acquisition conditions cannot be used. This point has not been previously recognized in muscular dystrophy research studies, where it has been assumed that R2* is low and that effects of not modeling R2* on fat fraction are negligible. Our study shows that it is the muscles with low R2* values at low fat fractions that generate the largest errors, while the highest muscle R2* values tend to lie in a fat fraction region that is least sensitive to R2* correction. This has important implications for the measurement of early stage disease and indicates that R2* correction should be performed routinely.

For midrange fat fractions, the effect of R2* correction was modest, and the conclusions of previous cross-sectional cohort and longitudinal disease progression studies are likely to be correct, since the change in the fat striations is generally small between longitudinal time points. The largest overestimations, and indeed differences in calculated fat fraction if R2* changes, will occur in control muscle and dystrophic muscles with low fat fractions. Since we have shown that modeling R2* does not increase the variance of the fat fraction measurements on a practical ROI basis, it should not affect the precision of longitudinal measurements and should always be used. Moreover, the fact that structures that contain only water or fat had an effective R2* of 35–45 sec⁻¹ means that making a global correction based on R2* values (as performed in references 8 and 9) of fat and water from a control subject, approximately 6.7 sec⁻¹ and 26.3 sec⁻¹ at 3.0 T, respectively, is likely to produce systematic errors in fat fraction. R2* modeling will benefit harmonization in multicenter trials, where echo times and field strengths used may vary.

The bounds of agreement between the fully sampled data and the accelerated data were similar to those without R2* compensation (17), and, taken together with improved fat fraction accuracy and no loss of precision, represent an improvement on that study. Our previous study also demonstrated that the intrinsic image quality of the undersampled reconstructions permitted all ROIs to be distinguished up to 4.94× acceleration but that at the higher acceleration (6.42×), not all desired ROIs could be delineated. Such an acceleration reduces the time to acquire volumes covering both upper and lower legs with 48 sections of 5-mm thickness from a total of 9 minutes to less than 2 minutes. The total unaccelerated acquisition time is similar to
two-dimensional protocols that cover 25 sections of 5-mm thickness (8,9). The ability to highly accelerate MR imaging fat fraction measurements in dystrophic muscle has the potential to decrease MR imaging trial costs, increase patient compliance, and decrease patient movement artifacts, particularly where children are studied. In general, it may make the acquisition of MR imaging end points more acceptable within the context of a clinical trial.

The limitations of our study included the fact that our fat fraction measurements have not been validated with biopsy, but this was not routinely clinically indicated for our patients, and hepatic proton-density chemical-shift techniques with multipake lipid modeling and R2* fitting have already been shown to have excellent agreement with biopsy results (28). While a modest number of patients were studied, the heterogeneity of fat replacement between muscle groups permitted the full dynamic range of the technique to be studied. Our measure of fat fraction variance, based on 10 axial sections covering 50 mm of muscle in the foot-head direction, reflects common practice in clinical trials of averaging several results from adjacent sections to reduce longitudinal sensitivity to small variations in patient position. Within-subject correlation was not dealt with in this analysis. For that reason, the presented robust-test inference might be slightly more optimistic. In the accelerated acquisitions, only three echoes were collected, one per repetition time interval. This has been found to be sufficient in liver studies by using R2* fitting and multipake fat modeling (13) and permitted close echo spacing (0.78 msec) for accurate B0 field map determination. The qualitative feasibility of an alternative approach to accelerating fat-water separation with R2* modeling has been demonstrated in the skeletal muscle of a control subject (29). While there have been skeletal muscle studies with reports of fat fraction in type 2 diabetes, spina bifida, and rotator cuff injuries that have modeled R2* (27,30,31), in none of these studies was dystrophic muscle considered or was a particular significance ascribed to the R2* values obtained. Owing to the nonmonotonic relationship of R2* relaxation rates with disease progression, R2* is unlikely to be a useful trial end point, though it is essential for estimating fat fractions.

In summary, our study demonstrated that R2* varies during fat replacement in the muscular dystrophy disease process, and R2* variation needs to be accounted for in fat fraction analysis. Furthermore, inclusion of an R2* modeling term in either fully sampled or highly accelerated fat fraction measurements does not degrade the precision.

### Table 2

<table>
<thead>
<tr>
<th>ROI</th>
<th>Patients with Becker Muscular Dystrophy</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone marrow</td>
<td>99.4 ± 0.4</td>
<td>99.4 ± 0.2</td>
</tr>
<tr>
<td>Biceps femoris long head</td>
<td>76.7 ± 29.7</td>
<td>76.9 ± 30.1</td>
</tr>
<tr>
<td>Semimembranosus</td>
<td>68.7 ± 34.8</td>
<td>68.9 ± 34.9</td>
</tr>
<tr>
<td>Semitendinosus</td>
<td>59.9 ± 38.4</td>
<td>60.0 ± 38.2</td>
</tr>
<tr>
<td>Vastus intermedius</td>
<td>47.8 ± 29.7</td>
<td>48.4 ± 29.8</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>47.2 ± 31.0</td>
<td>47.4 ± 31.1</td>
</tr>
<tr>
<td>Vastus lateralis</td>
<td>43.6 ± 28.2</td>
<td>44.4 ± 28.2</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>32.8 ± 28.4</td>
<td>32.3 ± 27.8</td>
</tr>
<tr>
<td>Lateral gastrocnemius</td>
<td>28.4 ± 32.8</td>
<td>28.1 ± 32.5</td>
</tr>
<tr>
<td>Sartorius</td>
<td>20.0 ± 30.3</td>
<td>20.4 ± 30.6</td>
</tr>
<tr>
<td>Soleus</td>
<td>13.8 ± 10.2</td>
<td>13.4 ± 9.6</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>4.2 ± 2.0</td>
<td>4.1 ± 1.6</td>
</tr>
</tbody>
</table>

Note.—Data are means ± standard deviations.

### Table 3

<table>
<thead>
<tr>
<th>Reconstruction</th>
<th>Bias Compared with Fully Sampled Reconstruction (%)</th>
<th>95% Limit of Agreement Compared with Fully Sampled Reconstruction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compressed sensing and parallel imaging 3.65×</td>
<td>-0.05</td>
<td>1.65</td>
</tr>
<tr>
<td>Compressed sensing and parallel imaging 4.94×</td>
<td>-0.13</td>
<td>1.95</td>
</tr>
<tr>
<td>Compressed sensing and parallel imaging 6.42×</td>
<td>0.17</td>
<td>2.22</td>
</tr>
<tr>
<td>Parallel imaging 3.36×</td>
<td>0.21</td>
<td>2.99</td>
</tr>
</tbody>
</table>
REFERENCES


