Paraspinal muscle fibre structural and contractile characteristics in spinal degeneration and deformity patients

Alex M. Noonan[1], Masoud Malakoutian[2], Iraj Dehghan-Hamani[2], Stephen Lewis[3], John Street[2], Thomas R. Oxland[2], Stephen H. M. Brown[1]
[1]University of Guelph, Guelph, Ontario, Canada [2] University of British Columbia, Vancouver, Canada [3]University of Toronto, Toronto, Ontario, Canada
noonana@uoguelph.ca; amnoonan@ucsd.edu

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INTRODUCTION: Aging of the spine results in a progressive kyphotic deformity and sagittal balance disorder in 20-40% of the adult population[1]. Paraspinal and spinopelvic muscular dysfunction are often hypothesized to be a significant causative factor of spinal degeneration and deformity[2]; however, our fundamental understanding of paraspinal muscle (dys)function in this population is poorly understood. Here, we explored the intrinsic cellular level contractile and structural properties of single muscle fibers from degenerative spine patients.

METHODS: Twelve degenerative spine patients were recruited and categorized into group 1 (DEG: four patients) with no sagittal imbalance (SI) and no usage of compensatory mechanisms (CMs); group 2 (DEG-COMP: four patients) with no SI through usage of CMs; and group 3 (DEG-COMP-UNBAL: four patients) with SI despite usage of CMs. From each patient, 8 biopsies were collected at the L4-L5 level from right and left multifidus (MULT) and longissimus (LONG) and prepared for contractile and structural measurements of single muscle fibers. All data were analysed using one-way ANOVAs with patient group as the factor. Normality was confirmed using the Shapiro-Wilk test. Where appropriate, Tukey multiple comparisons were performed. Data are presented as mean (±SD) and significance was set to α=0.05. This study was approved by the University of British Columbia Clinical Research Ethics Board and Vancouver Coastal Health Research Institute, the University Health Network Research Ethics Board, and the University of Guelph Research Ethics Board. All recruited patients were informed of the study and signed consent forms.

RESULTS: Eight of 48 (17%) collected biopsies did not exhibit any contractile properties. From the remaining biopsies, a total of 175 type I fibers were statistically analyzed. In MULT, the mean specific force (kPa) values were: 106.6 for DEG; 107.2 for DEG-COMP; and 120.2 for DEG-COMP-UNBAL; no significant differences existed between groups (p=0.47). For the LONG, the mean specific force (kPa) value was 109.0 for DEG; 88.4 for DEG-COMP; and 78.6 for DEG-COMP-UNBAL patients; a significant difference existed between DEG and DEG-COMP-UNBAL (p=0.02). The mean unloaded shortening velocity (FL/s) for the MULT was: 0.92 for DEG; 0.73 for DEG-COMP; and 1.14 for DEG-COMP-UNBAL; a significant difference existed between DEG-COMP and DEG-COMP-UNBAL (p=0.02). In the LONG, the mean unloaded shortening velocity (FL/s) was: 0.89 for DEG; 0.70 for DEG-COMP; and 0.54 in DEG-COMP-UNBAL; a significant difference existed between DEG and DEG-COMP-UNBAL (p<0.05). The mean specific force, but not unloaded shortening velocity, values from all three degenerative spine patient groups are generally below average age-matched literature norms for type 1 fibers from vastus lateralis (Figure 1). For MULT, the mean optimal sarcomere length (µm) was 2.48 in DEG; 2.34 in DEG-COMP; and 2.55 in DEG-COMP-UNBAL; no significant differences existed between groups (p=0.38). The mean optimal length (µm) for the LONG was 2.47 for DEG; 2.35 for DEG-COMP; and 2.92 for DEG-COMP-UNBAL; no significant differences existed between groups (p=0.40). The mean width (µm) of the force *sarcomere-length* relationship at 90% maximum for the MULT was 1.32 in DEG; 1.36 in DEG-COMP; and 1.20 in DEG-COMP-UNBACL; no significant differences existed between groups (p=0.71). For the LONG, the mean width (µm) was 2.11 in DEG; 1.54 in DEG-COMP; and 2.12 in DEG-COMP-UNBAL; no significant differences existed between groups (p=0.27). For MULT, the mean thin filament length (µm) was: 1.14 for DEG; 1.12 for DEG-COMP; and 1.10 for DEG-COMP-UNBAL. For the LONG, the mean thin filament lengths were 1.11 for DEG; 1.05 for DEG-COMP; and 1.21 for DEG-COMP-UNBAL.

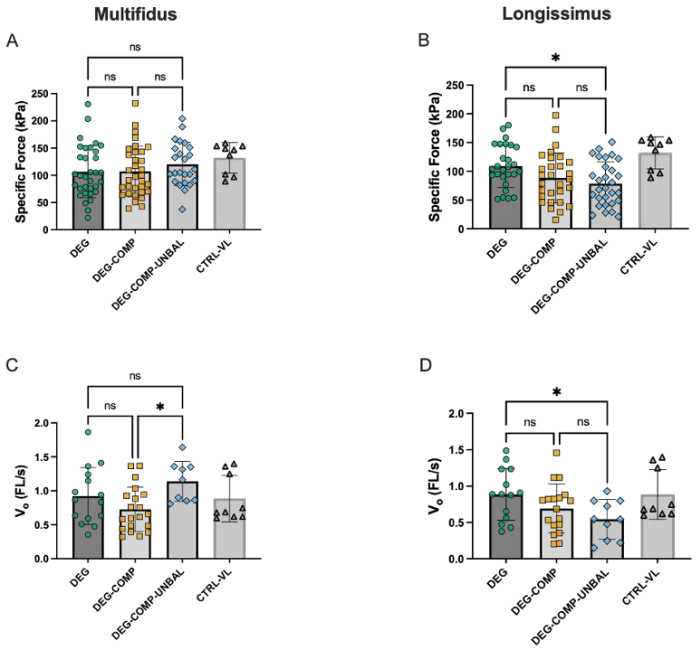

Figure 1. Specific force and unloaded shortening velocity for MULT (A&C) and LONG (B&D) muscle fibres. (Specific force: MULT; DEG: *n* = 33 fibres, DEG-COMP: n=33 fibres, DEG-COMP-UNBAL: n=24 fibres. LONG; DEG *n:* = 27 fibres, DEG-COMP: n=29 fibres, DEG-COMP- UNBAL: n=29 fibres. Unloaded shortening velocity: MULT; DEG: *n* = 15 fibres, DEG-COMP: n=20 fibres, DEG-COMP-UNBAL: n=9 fibres. LONG; DEG *n:* = 14 fibres, DEG-COMP: n=18 fibres, DEG-COMP- UNBAL: n=10 fibres). Each data point represents a single muscle fibre. The CTRL-VL data (right-most bar), represents the specific force/unloaded shortening velocity values reported for elderly human *vastus lateralis* (65-85 years old) in the literature (Trappe et al., 2003; Krivickas et al., 2006; Slivka et al., 2008; Reid et al., 2012). For the CTRL-VL each point represents an average value (male or female) from a single study. Spinal patient groups are shown as mean (±SD) with significance set to α=0.05. (ns) = not significant (*) = statistically significant.

DISCUSSION: Understanding the paraspinal muscular dysfunction that occurs in degenerative spine and spinal deformity patients is important to better understand the disease process and eventually to devise better surgical and non-surgical treatments. The present study is the first to show a heightened intrinsic contractile muscle disorder in degenerative patients who are sagittally imbalanced (compared to degenerative non-deformity patients). Additionally, there are clear indications that degenerative spine patients (all groups studied here) have intrinsic force *sarcomere-length* properties that are dysregulated, likely in part due to shorter and highly variable thin filament lengths. Patient recruitment was significantly disrupted by COVID-19, therefore the number of patients recruited for this study (four/group) was smaller than originally intended. Further, a 'true' control group of healthy individuals would be ideal for comparison to the spinal degenerative patient groups; however, as non-invasive collection of paraspinal muscle biopsies from a healthy population was not feasible, we instead defined patient group I (DEG), those who did not have a clinically meaningful spinal deformity or muscular recruitment of compensatory mechanisms to maintain sagittal balance, as the 'least severe' degenerative group for comparisons.

SIGNIFICANCE/CLINICAL RELEVANCE: General paraspinal muscle dysfunction is considered a hallmark feature of spinal degeneration and deformity, but the specific characteristics underlying this dysfunction are poorly understood. The findings from the present study provide important insight into the pathophysiology of muscle weakness in adult spinal deformity patients.

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