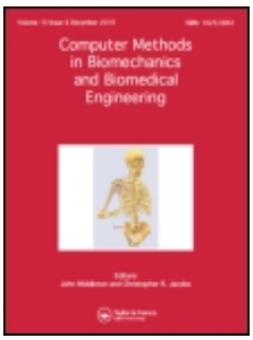

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# The relationship between trunk muscle activation and trunk stiffness: examining a non-constant stiffness gain

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# The relationship between trunk muscle activation and trunk stiffness: examining a non-constant stiffness gain

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The relationship between muscle activation, force and stiffness needs to be known to interpret the stability state of the spine. To test the relationship between these variables, a quick release approach was used to match quantified torso stiffness with an EMG activation-based estimate of individual muscle stiffnesses. The relationship between activation, force and stiffness was modelled as $k = q \times F/l$, where $k$, $F$ and $l$ are muscle stiffness, force and length, respectively, and $q$ is the dimensionless stiffness gain relating these variables. Under the tested experimental scenario, the 'stiffness gain', $q$, which linked activation with stiffness, demonstrated a decreasing trend with increasing levels of torso muscle activation. This highlights the likelihood that the choice of a single $q$ value may be over simplistic to relate force to stiffness in muscles that control the spine. This has implications for understanding the potential for spine instability in situations requiring high muscular demand.

**Keywords:** spine; stability; abdominal muscles; muscle force; muscle stiffness

## 1. Introduction

Measurement of low back loads and structural stability is helpful in understanding injury risk together with prevention and rehabilitation of painful backs. Muscle activity is needed to create moments and stiffen the torso and consequently imposes loads on the spine joints. As low back moment demands rise, torso muscular activation rises accordingly. This rise in activation leads to an increase in muscle force and stiffness, which dictate the rotational stiffness and stability of the individual joints of the lumbar spine. The relationship between muscular activation, force and stiffness will determine the ability of a muscle to appropriately stiffen the spine to bear load and prevent 'buckling'-type injuries. A long-held assumption in the spine stability literature has been that muscle force and stiffness increase proportionately one another. However, recently, Brown and McGill (2005) have hypothesised that this may not be the case in all situations and demonstrated that if muscle stiffness levels off as the muscle force continues to increase, the spine-stiffening potential of the said muscle may be compromised at high force levels. Brown and McGill (2008a) suggested that this phenomenon may lead to instances of impaired stiffness or stability control in highly demanding spine loading situations, creating the potential for catastrophic injury scenarios.

Bergmark (1989), in developing a spine model, first suggested a relationship between the muscle force ($F$), length ($l$) and stiffness ($k$), $k = qF/l$, where $q$ represents a stiffness gain. Subsequent researchers have attempted to determine a value for $q$, documenting ranges from 0.5 to 50, with an average of approximately 10 (Crisco and Panjabi 1991; Cholewicki and McGill 1995). Determining $q$ is important since those trying to quantify spine stability must assume a value. However, if the relationship between muscle-tendon force and stiffness is non-linear, as demonstrated in the muscle literature (Joyce and Rack 1969; Ettema and Huijing 1994), the proportionality value or stiffness gain ($q$) should not be a constant but rather should vary with muscle activation or force. This theory was the basis for our previous conceptual modelling paper (Brown and McGill 2005); the current study was thus designed to experimentally test the relationship between torso muscle activation and lumbar spine stiffness, in an attempt to further the hypotheses put forth in the earlier work.

## 2. Methods

Experimental procedures have been published previously (Brown and McGill 2009). Briefly, nine healthy males were secured, lying either on their right side for flexion trials or on their back for lateral bend trials, on an apparatus that allows for movement of the upper body about the lumbar spine with near-frictionless outside resistance (Figure 1).

Participants began each trial in their position of neutral elastic equilibrium. They were then instructed to generate either a flexor or a right-side lateral bend moment to one of

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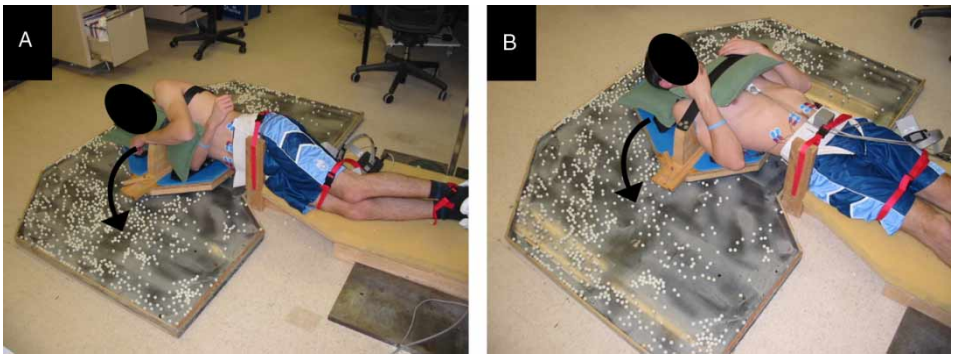


Figure 1. Picture of the participant positioning for the experimental trials. (A) Position for the flexion condition and (B) position for the lateral bend condition. Curved arrows represent the direction of muscularly generated moment and subsequent torso movement post release. Nylon balls between plexi-glass surfaces allow for minimised friction between the upper body cradle and surface base.

the three distinct target activation levels (5, 10 and 15% termed light, moderate and heavy, respectively) as monitored from biofeedback of their right external oblique (EO) muscle site. A subset of six of these participants also performed a fourth target activation, which was the maximum flexor or lateral bend moment that they could produce in the test position (termed maximum). The internally generated moments were resisted (so as to maintain the participants in their neutral position) by the experimenter via a cable, instrumented with a force transducer (Transducer Techniques, Inc, Temecula, CA, USA), oriented perpendicular to the upper body cradle. Once the target activation was achieved and held steady for a period ranging between 1 and 3 s, the cable was rapidly released via a latch mechanism, thus causing a rotational perturbation of the participants' trunk in either the flexion or right-side lateral bend direction. Participants were instructed to react in a natural manner to the perturbation. Participants performed two trials for each condition. Previous work has indicated that muscular reflex activity was minimal under this perturbation paradigm (Brown and McGill 2009), and thus a linear time-invariant model of the kinematic response, described below, was considered appropriate for the analysis.

### 2.1 Instrumentation

Twelve channels of EMG were collected from the following muscles bilaterally: rectus abdominis, EO, internal oblique (IO), latissimus dorsi (LD) and two levels of the erector spinae (EST9 and ESL3). Blue Sensor bi-polar Ag–AgCl electrodes (Ambu A/S, Denmark, intra-electrode distance of 2.5 cm) were placed over the muscle belly of each muscle in line with the direction of muscle fibres. Signals were amplified (± 2.5 V; AMT-8, Bortec, Calgary, Canada; bandwidth 10–1000 Hz, CMRR = 115 db at 60 Hz, input impedance = 10 GΩ), captured digitally at 2048 Hz, low-pass filtered at 500 Hz, rectified and low-pass filtered at 2.5 Hz (single-pass second order) and normalised to the maximum voltage produced during isometric maximum voluntary contraction (MVC) trials to produce a linear envelope.

Three-dimensional lumbar spine motion was recorded using an electromagnetic tracking system (Isotrak, Polhemus, Colchester, VT, USA). The trunk motion data were sampled digitally at 32 Hz and dual-pass filtered (effective fourth-order 3 Hz low-pass Butterworth).

The torso moments were calculated by the product of the force applied in the cable (perpendicular to the distal end of the upper body cradle) and the moment arm from the location of the applied force to the level of L4/L5. Both the linear enveloped EMG and force signals were downsampled to 32 Hz to match the trunk motion data.

### 2.2 Kinematic model

A second-order linear representation of the trunk was used to model the rotational motion of the trunk post-perturbation. The form of the model was as follows:

$$I\ddot{\theta} + B\dot{\theta} + K(\theta - \theta_0) = 0, \quad (1)$$

where $I$ is the moment of inertia of the upper body and cradle (kg m$^2$), $B$ the trunk rotational damping (Nm s/rad), $K$ the trunk rotational stiffness (Nm/rad), $\theta_0$ the trunk angle offset (release angle of the trunk in the plane of interest) and $\theta$ the trunk rotational displacement.

The moment of inertia of the upper body cradle was quantified using the pendulum method (Dowling et al. 2006) and subsequently transferred to the L4/L5 joint, while the upper body moment of inertia about the L4/L5 joint was calculated using participant-specific anthropometrics (Winter 2004; combined mean (standard deviation) of the cradle and upper body about L4/L5 was 9.7(1.2) kg m$^2$). The length of post-perturbation data analysed in order to obtain trunk characteristics of $K$, $B$ and $\theta_0$ was taken from the time of quick release to the time of maximum trunk deflection (mean 1150 ms).

An optimisation algorithm was utilised to solve for the three equation unknowns by minimising the root-mean-square (RMS) difference between the measured and modelled trunk angular displacements. This second-order time-invariant model performed very well in matching the predicted to the measured kinematic responses (average errors never exceeding 4.2%).

### 2.3 EMG-based model

Linear enveloped EMG signals and lumbar spine angles, averaged over the 50 ms prior to quick release, were entered into an anatomically detailed model representing 58 muscle lines of action crossing the L4/L5 spinal joint. An estimate of the force generated by each of these muscle lines was made by the following equation:

$$F_m = \mathrm{NEMG}_m * \mathrm{PCSA}_m * \sigma_m * l_m * G, \qquad (2)$$

where $F_m$ is the force in muscle $m$ (N), $\mathrm{NEMG}_m$ the normalised EMG signal for muscle $m$ (% MVC), $\mathrm{PCSA}_m$ the physiological cross-sectional area of muscle $m$ ($\mathrm{cm}^2$), $\sigma_m$ the maximum stress generated by the muscle $m$ (set at $35\ \mathrm{N/cm}^2$), $l_m$ the length coefficient of the muscle $m$ (unitless) and $G$ the participant-specific multiplier (unitless).

The participant-specific multiplier was obtained by finding a best match between the experimentally determined moment and the moment estimated by the combined agonist EMG-driven muscles (the abdominals in the flexor trials and the right-side muscles in the lateral bend trials). In this way, the differences in the size of muscles between individuals could be accommodated by the model.

Lumbar spine muscular rotational stiffness was then estimated with the following equation (Potvin and Brown 2005):

$$S(m)_z = F\left[\frac{A_X B_X + A_Y B_Y - r_z^2}{\ell} + \frac{q r_z^2}{L}\right], \qquad (3)$$

where $S(m)z$ is the rotational stiffness contribution of a muscle about the $z$-axis of the joint in question; $F$ the muscle force (N); $\ell$ the 3D length of the muscle vector that crosses the joint in question; $L$ the full 3D length of the muscle; $r$ the 3D muscle moment arm; $A_X$, $A_Y$ and $A_Z$ the origin coordinates with respect to the joint of interest at $(0, 0, 0)m$; $B_X$, $B_Y$ and $B_Z$ the initial deflection or insertion (without deflection points) coordinates with respect to the joint and $q$ the stiffness gain relating muscle force and length to stiffness.

A unique stiffness gain ($q$) was calculated (lumped for all muscles) to minimise the RMS difference between the rotational stiffness estimated using the EMG-based (muscle) model and the kinematic-based lumbar spine model, for each of the four activation levels for both the flexor and lateral bend conditions. Finally, at each level of moment resistance, the average activation level was calculated for the agonist muscles (i.e. abdominal muscles in the flexion condition and right-side torso muscles in the lateral bend condition). A non-linear power function was then computed to link the stiffness gain with this average muscle activation.

## 3. Results

The activation–stiffness gain, relating to stiffness in both the flexion and lateral bend directions, showed a marked decrease as torso muscle activation increased (Figure 2). This divergence between torso muscle activation and its subsequent stiffening effect can greatly impact the stability of the spinal column, as high forces in the absence of equivalent rises in stiffness can create scenarios of spinal instability (Brown and McGill 2005).

Mathematical power functions, linking the activation levels with the stiffness gain, were developed for both the flexion and lateral bend conditions (Figure 3). These

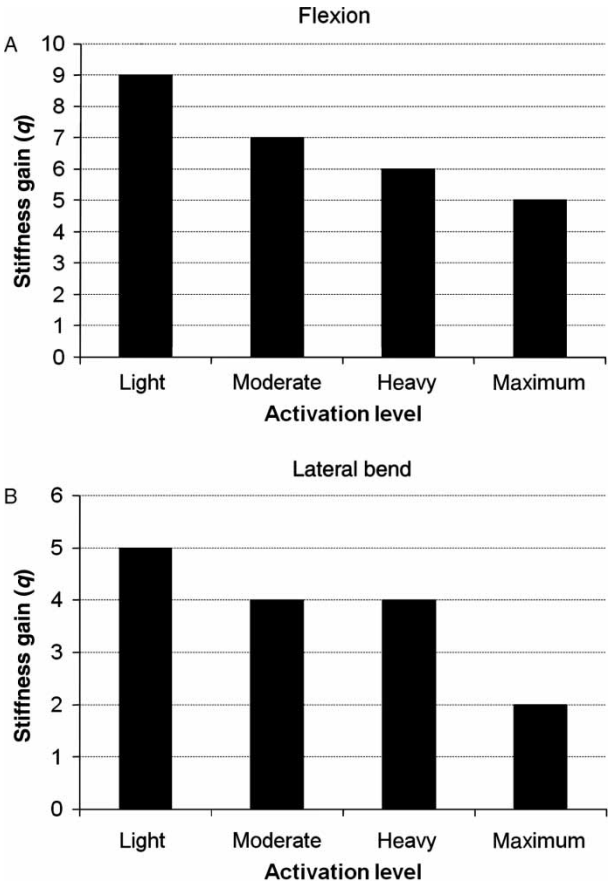


Figure 2. Stiffness gain ($q$), representing the relationship between muscle force and muscle stiffness, for each of the four torso activation levels in the flexion (A) and lateral bend (B) conditions.

functions were fit and are displayed only through the average levels of activation studied in the current experimental protocol.

Because our main interest here is in relating torso stiffness to muscle activation, and ultimately muscle force, it is imperative that the nature of the changing levels of muscular activation is presented. There was a trend of increasing activation between each of the targeted activation levels for the majority of muscles; however, the increases were only statistically significant ($p < 0.05$) in the maximum activation level when compared to each of the light, moderate and heavy activation levels. The EO and IO muscles displayed the highest level of activation in the maximum flexor condition, with averages of approximately 27 and 25% MVC, respectively; the right IO was by far the most active of the muscles in the lateral bend trials, with an approximate average of 50% MVC, followed by the right LD and EO at approximately 18% MVC each. This confirmed the expected general trend of increasing activation at each successive increase in moment resistance and thus substantiates the hypothesis that the stiffening effect (gain) of torso muscle activation is most pronounced at low levels of activation and tapers off as activation levels increased in the range 25–50% of MVC.

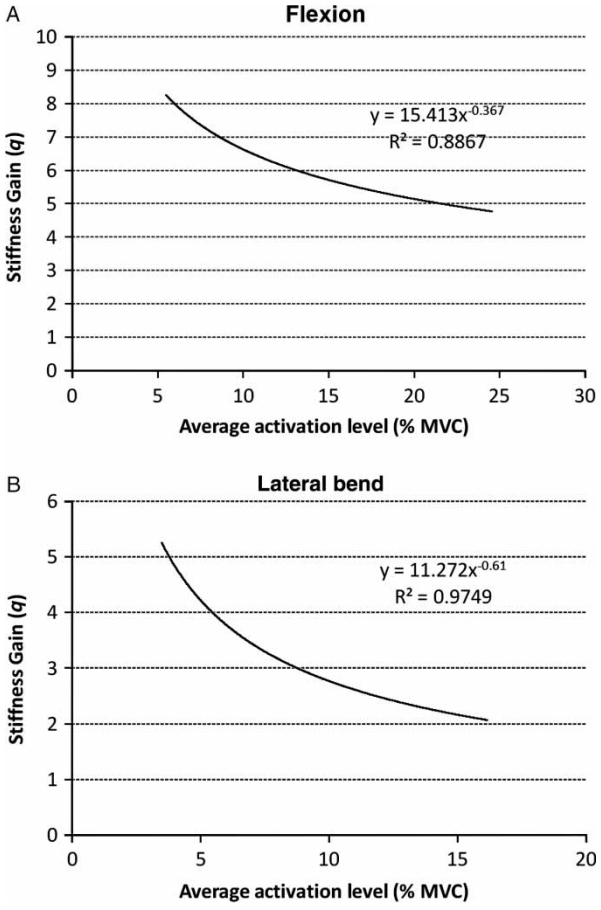


Figure 3. Power functions describing the relationship between muscle activation level and stiffness gain in the flexion (A) and lateral bend (B) conditions. Muscle activation levels were averaged across the muscles generating the desired moment (i.e. abdominal muscles in the flexion condition and right-side torso muscles in the lateral bend condition). Equations of best fit and $R^2$ values are shown.

## 4. Discussion

The relationship between torso muscle activation, force and stiffness dictates in large part the ability and success of the muscles in stiffening the spine to prevent unwanted and potentially harmful, kinematic displacements at individual vertebral levels. The current study has demonstrated that for the current experimental protocol, the stiffness gain ($q$), representing the spine-stiffening effect of the combined torso musculature, is highest at low levels of activation and decreases with subsequent increases in activation. This indicates that the choice of a single $q$ value for relating muscle force to stiffness might be an oversimplification when modelling spine stability, as this value can vary depending on the magnitude of muscle activation.

Previous work in our laboratory reported a potential degrading stiffening or stabilising effect produced by increases in muscle force beyond an optimal level, which was governed by the shape of the muscle's force–stiffness relationship (Brown and McGill 2005). The non-linear force–stiffness relationship simulated in that study was based on previous evidence of muscle–tendon units demonstrating a plateau of increasing stiffness at the highest level of force increases (Joyce and Rack 1969; Ettema and Huijing 1994). The simulated non-linear force–stiffness relationship was characterised by a stiffness gain that peaked at the lowest level of activation and decreased non-linearly as the muscle force increased. The current study has provided evidence, based on a lumping of the complete torso muscle activation and stiffness profile, that the stiffness gain can indeed decrease as muscle activation levels increase. Our use of discrete levels of torso muscle activation, driven by increases in moment generation/resistance, precludes any definitive conclusions as to the complete shape of the activation–stiffness relationship (linear versus non-linear), but this has no immediate bearing on the confirmation that the stiffness gain may not be constant across activation levels. It is thus important to note that the functions displayed here (Figure 3), linking muscle activation level with stiffness gain, only hold across the levels of activation studied in the current experimental protocol. The increasing force–decreasing stiffness gain relationship has the potential to compromise spine stability in high load/demand situations (Brown and McGill 2008a), as increasing muscle forces in the absence of proportionally

increasing stiffness may lead to instances of inadequate stiffness under high destabilising loads. Future studies will have to determine whether the stiffness gain continues to decrease at higher levels of torso muscle activation, which would confirm this potential for compromised spine stability. Further, this stiffness gain relationship needs to be explored across a wider range of trunk posture/perturbation conditions to fully verify its form.

The relationship reported here details the changes in stiffness gain with changes in muscle activation. The stabilising effect of muscle activation ultimately relies on the relationship between muscle force and stiffness development. The association between torso muscle activation and force output has been studied in the past, with differing conclusions regarding the shape of this relationship; some studies reporting a linear relationship (Seroussi and Pope 1987; Dolan and Adams 1993), others reporting a non-linear relationship with reduced increases in force as activation increases (Stokes et al. 1987; Thelen et al. 1994; Potvin et al. 1996). A recent study has been published examining this linear versus non-linear phenomenon (Brown and McGill 2008b), where it was concluded that the torso muscle force–stiffness relationship is predominantly linear when considering the complete moment (including the antagonist muscle moment) being generated by the muscle group in question. This linearity indicates that muscle activation can be used to approximate muscle force, and thus, the relationship between muscle activation and torso stiffness demonstrated in the current study is most likely representative of the muscle force–torso stiffness relationship that is crucial in determining the stabilising potential of the trunk muscles.

The decreasing stiffness gain observed in the current study may arise from a number of potential sources. The first relates to the previously mentioned non-linear muscle force–stiffness relationship that has often been reported in the literature. This non-linearity arises from the stiffening dominance of different tissues that lie in series with one another, namely muscle fibres and tendinous connective tissues. As muscle fibres increase the number of actin–myosin cross-bridge links, they increase force and stiffness fairly proportionally. However, as the stiffness of the fibres increases, the compliance of the connective tissues with which they lie in series begins to dominate movement (Rack and Westbury 1984; Kawakami and Lieber 2000). Because these connective tissues display a fairly constant stiffness (Proske and Morgan 1987), the overall muscle–tendon force–stiffness relationship levels off beyond this point. Studies of animal limb muscles generally display the connective tissue compliance dominating at the higher ends of force development, while we have shown changes at relatively low levels of activation. It is possible that the abdominal musculature generates a substantial stiffening effect, thereby approaching the stiffness of its in-series connective tissues, at low levels of activation. Studies at higher levels of abdominal muscle force and stiffness development will be needed to test this hypothesis. Further, the spine displacements in the current study are large enough to exceed the so-called short-range stiffness of the active muscle fibres; therefore, the muscular stiffness assessed here represents the stiffness effective over fairly large changes in length and thus would be less than the fibre short-range stiffness. A second potential source of the decreasing stiffness gain may exist within the intrinsic stiffness of the passive spine tissues. This passive stiffness makes up a greater proportion of the overall trunk stiffness at instances when muscle activation is low and subsequently decreases as activation increases. The stiffness calculation in Equation (3), dictating the stiffness gain ($q$), accounts only for muscular contributions to stiffness; thus, the gain may be highest at low levels of activation because it accounts for the greater overall proportion of the intrinsic stiffness that resides outside the active musculature. Further, the muscle stiffness gain reported here does not separate the direct rotational stiffening effect of the muscle from the additional stiffening effect that will be contributed by the muscle compressing the spinal joints (Janevic et al. 1991; Stokes and Gardner-Morse 2003). These studies indicate that compression of the spine increases its rotational stiffness in a highly non-linear manner, with the majority of stiffness being contributed at relatively low ($< 1000$ N) forces. This could also represent a portion of the reduced stiffness gain at higher force levels.

The muscle stiffness estimated in the current study represented the stiffness inherent to the muscular apparatus itself, in the absence of substantial reflexive responses. Reflexes have the ability to increase the effective stiffness that a muscle can provide to a joint (Sinkjaer et al. 1988; Moorhouse and Granata 2007) and thus increase the effective $q$ value. The $q$ values documented at higher force levels in the current study approached the critical levels that have been estimated in previous work (Gardner-Morse et al. 1995; Granata and Marras 2000) to be necessary to ensure a stable spine. It is highly likely that the presence of muscular reflexes would serve to increase the $q$ values calculated in the current study to levels well above these critical marks. Thus it must also be considered that if the reflex response is enhanced at higher activation levels, the presence of reflexive activity has the potential to temper the non-linearity between muscle activation and trunk stiffness.

The current study employed a time-invariant model, similar to previous groups (e.g. Cholewicki et al. 2000; Hodges et al. 2009), to determine the kinematic stiffness from the quick release trials. Lawerence et al. (2005, 2006) have shown that an adaptive time-varying model can account for a changing stiffness over the course of a trunk perturbation in the upright position. However, the time-invariant model employed here is considered adequate for

the current protocol because of the suppressed reflexive muscle response (as the muscular response would be the primary cause of the changing stiffness) and because of the excellent fit that was obtained between the measured and predicted trunk displacements when considering a time-invariant stiffness. However, some voluntary muscle responses were detected in the current study, a bulk of which occurred after more than 500 ms post release. In order to ensure that these muscle responses were not the source of the non-linearity between muscle activation and stiffness, the model analyses were re-done examining the kinematics over a course of 500 ms post release (as opposed to the full-time course to maximum displacement). These additional analyses uncovered a similar non-linearity as in the primary analysis, albeit with slightly lower $q$ values (from 8 to 4 in flexion and from 5 to 2 in lateral bend).

Finally, it is important to note that while a main biological interest lies in the stiffness of the spine at individual vertebral levels, a grosser assessment of lumbar trunk stiffness was used here as a surrogate kinematic measure. It is thus possible that the actual gain relating muscle force to the stiffness at individual spine joints does not demonstrate the same decreasing trend as it does to lumped lumbar trunk stiffness.

To conclude, the purpose of this paper was to test the relationship between torso muscle activation and the resulting torso-stiffening effect that is inherent to the muscular apparatus itself. Results indicate that for the experimental scenario tested here, the stiffness gain ($q$) relating muscle activation to stiffness was not constant across all levels of activation and in fact, decreased as activation level increased. This has very relevant implications in understanding the potential for instability-related injury occurring in situations requiring high levels of muscular demand and loading, especially when reflex responses are compromised. From a clinical point of view, it appears plausible that individuals trained and experienced in strength-related endeavours develop motor strategies (e.g. intense breath holding to generate intra-abdominal pressure and stiffen the abdominal wall) to combat the potential divergence between muscular load and stiffness.

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## Note

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