Obesity and Loading during Lifting

by

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ABSTRACT

Background Obesity is associated with an increased risk of back pain, attributed to elevated mechanical load. Back injury risk is also determined by movement patterns (kinematics) and physiological factors (exertion, muscle activation). Lifting, particularly repetitive, is the most frequently cited injurious activity. However, in spite of the obvious relation, a paucity of information exists quantifying the interaction of obesity and repetition during lifting.

Purpose To determine the effects of obesity and repetition on mechanical, kinematic and physiological lifting outcomes.

Methods: An individual-specific, biomechanical model (based upon 3D photogrammetry) was developed to estimate the effect of obesity on back load during lifting (study 1). Lifting strategy and physiological outcomes related to obesity were examined in a fixed-pace, repetitive lifting task (study 2). The effect of task constraints on lifting strategy of high and normal BMI individuals were determined (study 3), followed by an evaluation of muscle activation responses during a repetitive trunk motion similar to that encountered during lifting (study 4).

Results: Obesity-specific alterations of important determinants of back load (inertia, CM_{loc}) were revealed. Obesity was related to a substantial increase in back load (M=+197.3, SE=16.8 Nm about L5/S1), however the effect differed across lifting tasks. The lifting strategy of high-BMI individuals was characterized by an increased distance to the external mass (M=+4.7, SE=1.8 cm) and shorter lift duration (M=230, SE=130 msec), with increased cardiovascular effort (M=+7.4, SE=3.4% HR_{max}) but no change in perceived exertion. Lifting frequency was not a major determinant of lifting strategy, however strategy was influenced by the presence and type of external pacing. A phase-specific, rapid alteration in muscle activation response was evident in the MMG signal during the initial repetitions of a repetitive trunk motion.

Conclusion: The effect of obesity during lifting is task-dependent, and cannot be attributable solely to mechanical factors. Future studies should consider tasks that are unconstrained, and examine the initial familiarization period of repetitive tasks, specifically the lowering phase of motions. These findings have relevance to back injury mechanisms related to obesity and the design of injury prevention programs for individuals with a high BMI.

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INTRODUCTION

Low back pain (LBP) of non-specific origin is one of the most common musculoskeletal complaints in adults[1]. While the majority of cases are of low severity and/or resolve in a short period of time[2], disability associated with more severe, longer-lasting symptoms (or recurrences) represents a considerable individual and societal burden[3]. Although the specific etiology is most often elusive[3], a plethora of factors related to the incidence[4], severity and recurrence[5] of low back pain have been identified.

Mechanical load is the most commonly cited factor involved in the etiology of low back injury[6–8]. For isolated tissues (e.g. ligaments, intervertebral discs), it is known that a load which exceeds the yield point will result in tissue failure, and that tissues are further susceptible to failure on repetitive loading[9]. However, examining tissue specific loading in the intact low back is problematic due to load-sharing[10], or partitioning of external forces amongst multiple anatomical structures. This differential loading can vary between repetitions and fatigue[11], across individuals[12], and with changes in position (e.g. shift in load from passive to active tissues at end-range lumbar flexion)[13]. This has important implications for mechanical models of back injury, as exposure to equivalent external torques could be associated with a different distribution of load amongst internal structures. Changes to load distribution can be partially deconstructed by examining patterns (timing and magnitude) of muscle activation (e.g. de-recruitment of lumbar extensors at end-range flexion)[14]. Muscle activation alterations can also be used to categorize individuals at increased risk of back injury[15], and may remain altered following 'recovery' from injury (i.e. absence of symptoms)[16]. Numerous theories of back injury mechanisms acknowledge the importance of differential or repetition-to-repetition variation in muscle activation on injury mechanisms[17,18], particularly during seemingly innocuous or low-force circumstances[19]. There is increasing recognition that a combination of biomechanics and neuromuscular activation

approaches will be required to further elucidate the mechanisms of back injury[11,16,17], and that neither approach in isolation is sufficient to explain the etiology or outcome of back pathology.

Factors that contribute to loading of the lumbar spine include such elements as external masses (e.g. a bag carried), applied forces (e.g. pushing/pulling), and the mass of body segments[20]. Modifying the determinants of back loading forms the basis of many prophylactic- and return-to-work strategies for low back pain in occupational settings[21,22]. Studies to date have focused on the obvious and readily modifiable loading parameters of external masses, applied forces, or segmental orientation[21]. Comparatively fewer have considered the influence of body segment mass[23], and none have examined other body segment parameters (e.g. mass distribution, inertia). It is well established that the mass and shape of body segments (distribution of mass) varies widely between individuals[24]. For instance, trunk mass ranges between 45 and 59% of total body mass[25,26], which is equivalent to a ~11 kg variation in trunk mass for a 34 BMI individual of average height. Relative to the recommended maximum acceptable external load of 23 kg for lifting tasks under optimal conditions[21], this represents a substantial variation. Although increased body mass, or more specifically obesity, is postulated as a causal factor in LBP[27,28], and the obvious mechanical effects on lumbar spine load are acknowledged[23], the literature is devoid of even a simple quantification of the mechanical effect of increased body mass or distribution on the lumbar spine.

In addition to body mass, back load is also influenced by kinematic factors. In particular, body segment orientation (a principle determinant of static torque) has received considerable attention during materials handling tasks as a means of quantifying lifting technique [29–31]. Despite these efforts there is an absence of evidence to support any single lifting technique as superior[31], which may explain the negligible effect of injury prevention programs that prescribe a single 'correct' technique[32] (i.e. the universal squat-style or 'lift-with-your-knees-not-your-back' advice). Numerous factors have been identified as determinants of lifting technique, including

mass lifted[33], destination height[34], strength[35], sex[36], lifting experience[37], and back pain[38]. Interestingly, with the obvious exception of the position of an object, individual-specific factors appear to exert a far greater influence on lifting technique than task-related influences[33,35], indicating individuals may have a preferred or 'natural' lifting technique[39]. Only recently has evidence emerged to support an influence of body mass on lifting technique[35]. For instance, trunk dimensions can constrain the position of the body relative to a work surface for individuals with an elevated BMI[40]. Reducing the distance between the body and external load (or work surface) is a common recommendation for minimizing load during materials handling[21], however the impact of increased body mass on lifting technique remains to be investigated. Similarly, although >40% of individual variation in trunk orientation and velocity during repetitive lifting (i.e. technique) can be accounted for using a multivariate model of numerous anthropometric variables[41], the effect of specific anthropometric parameters has not been identified (e.g. body mass, waist circumference, height). There is increasing evidence that individual-specific factors (specifically obesity) affect lifting technique, however no studies have identified BMI-dependent determinants of lifting technique (i.e. mass, body shape) nor the aspect of lifting principally affected by increased BMI (e.g. trunk or load position).

Based upon Newtonian mechanics, acceleration (another kinematic factor) is necessarily involved in back loading. In fact, loading differences of up to 190% are attributable to acceleration[42], far greater than the differences attributable to lifting techniques (0 – 33 %)[43]. One of the few studies to examine the effect of increased body mass on lifting kinematics revealed obesity-related increases in trunk acceleration and velocity[23], changes that would likely (but not necessarily) exacerbate body mass-related loading effects. Back load is a function of both static and dynamic torques (and from body segments and external loads), making it possible to alter the relative contributions of static and dynamic factors in order to maintain a given load, or a given exertion (where load and exertion may not be equivalent). Evidence to support this potential

trade-off between static and dynamic torques was provided by Faber and colleagues[44], who demonstrated that reductions in the mass lifted did not result in a proportional decrease in load, due to an increase in acceleration. In the absence of a comprehensive examination of orientation and kinematics during lifting tasks, authors can only speculate on the nature of obesity-dependent loading during materials handling[23]. Understanding the impact of obesity on lifting requires a comprehensive mechanical examination, as it is likely more complex than a simple body mass-proportional effect. The effects of increased trunk mass, variations in kinematics (orientation, acceleration) and the position of external load are important determinants of the biomechanical load on the low back in obese individuals, and remain to be studied.

Most investigations have examined relatively short duration lifting tasks (< 5 minutes)[23,45], however application of these findings to longer duration (repetitive) lifts may be problematic. Repetitive or longer duration lifting is an independent risk factor for low back pain[7], with cumulative loading effects, alterations in muscle activation, and neuromuscular fatigue cited as probable injury mechanisms[9,11]. Further, individuals often alter lifting technique during long duration lifting tasks (e.g. from a knee- to a hip-dominant lifting technique), a change that may be associated with the onset of fatigue[46,47]. When one considers the impact of obesity on longer duration lifting, it is possible that obesity will exacerbate the mechanical (increased load) or physiological (increased fatigue) effects of repetitive lifting. Elevated body mass is also associated with decreased endurance of the lumbar extensors[48], a factor that may further increase the risk of injury during repetitive lifts[49]. In other words, obesity-related changes to lifting technique/ kinematics may occur as a consequence of, or even as a strategy to minimize, fatigue or deleterious low back loading during repetitive lifts – an effect that would not be apparent during short duration lifts.

One factor that limits the generalizability of manual handling studies, even those that examine repetitive lifting, are the constraints imposed during laboratory-based investigations[50]. In

addition to foot position[50], the most frequently constrained parameter is lifting frequency[23,37], which is typically set at a fixed-pace (e.g. 1 lift every 10 - 15 seconds), intended to reflect ergonomic or workplace requirements (not athletic settings where lifting frequency is typically set at higher frequencies; e.g. 1 lift every 1.5 - 4 seconds). Although this has enabled investigators to examine the influence of individual lifting parameters (e.g. mass lifted[37]), it is uncertain whether findings from fixed-paced lifting tasks can be generalized to conditions outside the laboratory, where these constraints are not imposed. For example, the influence of the mass lifted on trunk kinematics may be dependent upon whether a lifting task is fixed-pace (constrained) or self-paced[44], indicating the imposition of a fixed-lifting frequency may alter how an individual executes a lift. The examination of lifting behavior during self-paced lifting, or similarly unconstrained tasks, has received far less consideration. It seems unlikely that, with the exception of highly-controlled occupational tasks, individuals would select a 'natural' lifting pace as low as 1 lift every 10 – 15 seconds (considered high frequency in occupational materials handling[51]). Further, increased body mass may also alter preferred lifting frequency and/or the response to changes in lifting frequency. In fact, regardless of BMI, little is known about how an individual might alter kinematics in response to changes in lifting frequency. Although it is reasonable to assume the kinematics parallel average lifting frequency[43], this is not consistent with findings revealing no effect of lifting frequency on low back loading[37]. This indicates frequency and kinetics may not be well-related across the comparatively narrow range of lifting frequencies used in many occupational research studies. An examination of the effect of lifting frequency on kinematics is needed to support the ecological validity of laboratory-based lifting tasks.

Although it is not possible to identify a single 'key' muscle or muscle group during lifting tasks[52,53], the back extensors are regarded as particularly important during lifting motions, acting to control the lowering and raising motions of the torso[54]. It is well-established that poor muscular endurance of the erector spinae is predictive of back injury risk[49], and that patients with

low back pain often demonstrate altered activation patterns in the these muscles during and following back injury[16]. However, one limitation of the task commonly used to demonstrate these alterations, the Beiring-Sorensen exercise[49] (BS), is its (lack of) task-specificity. In contrast to the isometric muscle action and static loading of the BS task, the tasks encountered during materials handling involve concentric and eccentric contraction of the back extensors and dynamic loading. These biomechanical (loading) and physiological (contraction-type) differences are important considerations as both muscle activation and mechanisms of task failure are task-specific[55], making it unlikely the activation patterns observed in the BS exercise reflect those of actual lifting motions[11]. Recent advances in surface-based techniques for evaluating muscle activation (e.g. mechanomyography[56], processing algorithms[57]) have enabled investigators to better detect alterations in activation during dynamic tasks[11], however investigations of erector spinae activation during standardized tasks (e.g. BS) have been largely restricted to EMG-based studies involving isometric tasks[15,58,59]. A next logical step from the investigations of muscle function during the BS task is to examine back extensor activation during a fatiguing task controlled by concentric and eccentric contractions – one that is more specific to muscle actions during lifting tasks.

Summary and Aims

The percentage of adult Canadians with body compositions placing them at an elevated risk for back pain exceeds 60% (37% overweight, 24% obese) and is expected to increase[60]. Injuries to the back are the leading cause of lost time occupational injuries in Manitoba and are among the most costly workplace injuries[61]. Evidence that body mass need be considered for certain vocational tasks has recently been put forth[62], but remains to be thoroughly investigated for materials handling tasks[23].

A number of areas related to obesity, repetitive lifting and back injury requiring further study were identified above. First, although it is obvious increased body mass will influence biomechanics, the magnitude of obesity-related loading effects on the low back has not been quantified[23,40]. Second, while the investigation of obesity on lifting kinematics has been initiated[23], no studies have examined whether increased BMI affects lifting technique, particularly during circumstances where the consequences of obesity may be exacerbated (repetitive lifting). It is also important for investigations aiming to generalize findings to a certain cohort (high BMI, > 30 kg/m^2) or circumstance (repetitive lifting) that experimental tasks are ecologically valid – and if there is a possibility task constraints (e.g. lifting frequency) affect lifting behavior, the influence must be known relative to that of the primary independent variable(s). Finally, it is obvious that biomechanics must be examined in the context of muscle activation, and while previous studies of muscle activation have been invaluable towards the understanding of back injury etiology and consequences[15,58], there is a discrepancy between the conditions during which activation patterns are typically examined (static) and those encountered during actual lifting tasks (dynamic), indicating a need for more specific examinations of muscle function during these tasks.

The overall aim was to examine the effects of increased body mass (obesity) and repetition on mechanical, kinematic and physiological outcomes specific to lifting. Four studies were undertaken: two considered obesity-specific factors and two examined complementary variables important to both normal weight and obese individuals. The aims were:

Study 1 *The effect of obesity on body segment parameters and lumbar spine loading*
– to quantify the mechanical effect of obesity on lumbar spine loading

Study 2 *The influence of body mass on lifting strategy during repetitive, fixed-pace lifting*
– to determine the effect of obesity on lifting during a repetitive lifting task

Study 3 *The effect of lifting frequency, cue type and BMI on kinematics*
– to determine the influence of fixed-pace lifting frequencies on kinematics

Study 4 *Activation of erector spinae during repetitive trunk motion*
– to characterize the activation of back extensor muscles during a repetitive, fatiguing task involving concentric and eccentric contractions

REVIEW OF LITERATURE

The review of literature is separated into four sections based upon the proposed studies. The first presents evidence specific to obesity and back injury/pain, and is followed by reviews of obesity biomechanics (study 1), materials handling (studies 2 and 3), and erector spinae muscle activation responses (study 4).

EFFECT OF OBESITY ON LOW BACK PAIN

Association

The possibility of an effect of body mass on the mechanism underlying low back pathology has been acknowledged as early as 1977[63], however evidence was largely equivocal. Initial cross-sectional studies often failed to detect a relationship between back pain and obesity[64], reported effects of low magnitude[65], or that lost significance in multifactorial models[27]. Not surprisingly, earlier reviews of literature found scant evidence of a causal relationship between obesity and incidence of back injury[4,28]. However, a positive association between obesity and the severity and duration of low back pain was established[3,28]. Understanding factors that predict chronicity of back pathology is important as the costs of low back pain are disproportionately distributed to the minority of patients with recurrent or long duration symptoms[3]. In fact, obesity is related to increased compensation costs of severe, work-related back pathologies requiring surgical intervention[66], perhaps due to a diminished post-surgical recovery[67].

Recent systematic reviews have provided more convincing evidence of an association between obesity and the prevalence of low back pain (OR = 1.33)[68], which is attributed primarily to longer duration symptoms and greater care-seeking behavior[69]. However, the majority of studies reviewed have been cross-sectional[3,68], a design that is not sufficient to establish causal

relationships between variables. For instance, association does not exclude the possibility of a bi-directional relationship between exposures (obesity) and outcomes (back pain), nor the influence of causal factors common to both conditions (i.e. physical inactivity)[68]. Similar to numerous other health conditions (e.g. smoking and cancer[70]), it is not practical to control the exposure variable using a randomized controlled design, and so alternate evidence of causality must be considered in order to support or refute the findings from associative studies. The most widely-accepted framework for establishing causality was provided by Bradford-Hill in 1965[71], which now forms the basis of many models of evidence-based practice[72]. Unlike other risk factors for low back pain (e.g. manual handling[73]) an examination of evidence for a causal effect of increased body mass on back pathology is absent.

Bradford-Hill Criteria

In addition to effect size (strength of an association), Sir Bradford-Hill[71] put forth 8 factors to aid clinicians in arriving at a conclusion of causality between two related variables. These included dose-response, specificity, experiment, consistency, temporality, analogy, coherence and plausibility[1]. Although the preponderance of emphasis placed on RCTs (experimental factor) in many reviews of literature suggests otherwise[74], none of the nine factors were intended to be essential towards a decision of causality. Instead the relative merit and contributions of individual factors can be considered for each specific circumstance – an approach that is not restricted to a single form of evidence (e.g. RCT) and which limits exposure to risks associated with shortcomings of any one factor (e.g. selection bias in RCTs, covariates in cross-section studies)[75]. The following is a review of findings related to obesity and back injury in the context of the Bradford-Hill criteria.

[1]The latter two factors, coherence and plausibility, reflecting the extent that evidence does not conflict with what is known about a pathology (coherence), and what is possible considering the current knowledge (plausibility), are considered here as evidence supporting the underlying mechanism of a pathology.

Dose response

Evidence for a proportional change in outcome relative to exposure (dose-response) is a simple extension of binary association (e.g. OR, RR), and in relation to body mass would be reflected as an increasing risk of back pathology across normal, overweight and obese categories. Evidence of a dose-response relationship has been established in individual studies for both the prevalence[76] and incidence[77] of back pain in the general population, and also in samples of twin cohorts[78] where the influence of genetic factors is controlled. While trends of a gradation of risk across overweight (pooled OR = 1.23) and obese individuals (pooled OR = 1.32, vs normal weight) persisted in a recent meta-analysis[68], the findings were not robust across all studies. In fact, some provided evidence of a protective effect at higher levels of obesity (i.e. A-shaped relationship)[28]. Although it is clear that some increase in body mass will elevate the risk of low back pain[68], further investigation is required regarding the nature of relationship across a continuum of body masses or BMIs, or perhaps to examine whether metabolic-based BMI cut-points are even appropriate in this circumstance. Further, BMI is an insensitive measure of the distribution (and composition) of mass across body segments, a factor that has important mechanical consequences on low back loading. For example, individuals with a greater distribution of mass in the trunk and upper extremity (android somatotype) will experience greater relative loading on the low back than those with body mass distributed in the lower extremity (gynoid somatotype), in spite of equivalent BMIs. Additional information is also needed regarding covariates that may alter the relationship between body mass and back pain, such as physical activity, fitness or inflammatory processes[68].

Specificity

A factor confounding the interpretation of many associative studies is the inability to attribute low back pain symptoms to a specific anatomical structure, as low back pain/injury is often identified via self-report rather than differential diagnosis[79]. The ability to identify specific pathological outcomes related to body mass would strengthen the causal association to back

pain[71]. As an example, elevated body mass is related to MRI-abnormalities in patients with degenerative disc disease[80] and also to the presence of Modic changes in cohorts of otherwise healthy adults[81]. Modic changes reflect degeneration to vertebral endplates and sub-chondral bone, and although the pathogenesis is still under investigation, mechanical factors are widely considered as determinants[82]. These lesions are associated with the development of degenerative disc disease[81] and more recently, chronic low back pain symptoms[83]. These pathological findings relate well to evidence associating elevated body mass to an increased duration (chronicity) of back pain symptoms[69]. Conversely, the 'specificity' factor can also relate to the exposure variable, with recent findings that symptoms of radiating low back pain can be attributed specifically to abdominal obesity, and not simply increased BMI[84]. Increased waist circumference (WC) is also associated with Modic changes (OR = +1.18 per 3 cm increase in WC)[81]. The specificity in both exposure and outcome variables also support evidence of a biomechanical basis to back injury mechanisms (see below). Establishing specificity for low back pathologies is a complex endeavor, however investigations of the relationship between specific anthropometric variables and low back pain will help establish specificity of exposure, beyond simply BMI.

Experimental

Although investigations manipulating the exposure variable (body mass) in a controlled investigation are impractical (and unethical), findings satisfying the experimental criteria of causality exist. For example, the changes to body mass during pregnancy[85] might be considered analogous to the mechanical consequences of obesity-related increases in trunk mass. It is widely accepted that the prevalence of back pain is substantially higher during pregnancy[86], and although certain physiological factors may contribute (e.g. relaxin[87]), biomechanical factors specific to increased mass (and not pregnancy per se) have been implicated[85]. More specific to obesity, large reductions in low back pain symptoms following bariatric surgery have been demonstrated[88,89] and are acknowledged, but not explicitly considered, in recent systematic reviews[68]. More recently, changes to objective outcomes such as physical function[90] and medical imaging

outcomes[91] following substantial reductions in body mass (>= 40 kg) have been shown. Reversal of pathology-specific outcomes following a reduction in the dose of exposure (body mass) provides compelling evidence for a causal association.

Temporality

Exposure prior to outcome (temporality), is the only Bradford-Hill factor that is a necessary condition for causality (i.e. exposure must occur prior to outcome). However, temporality is difficult to establish for chronic conditions, where the onset of symptoms is insidious. Only a small number of longitudinal studies[68] have examined the relationship between obesity and back pain, and limitations regarding objective evaluation of both exposure (BMI) and outcome have hindered attempts to establish temporality. For example, anthropometric variables are usually restricted to height and weight[68], yet more recent evidence indicates mass distribution may be more specific to low back pain[84]. Additionally, a variety of definitions for low back pain exist, which may reflect different underlying pathologies[79], and that might differ between normal and high BMI individuals. In short-term circumstances it is obvious that addition of mass to the body (e.g. a heavy backpack) will result in subsequent, but temporary, discomfort and pain. Establishing that increased body mass precedes the onset of low back pain is more complicated; in fact, it has been postulated that chronic back pain may lead to decreased physical activity and subsequently, an increase in body mass[68]. However, individuals with low back pain do not report different physical activity levels than asymptomatic controls[92] and a recent longitudinal study demonstrated that previous episodes of low back pain did not account for subsequent changes in BMI over an 11-year period[93]. Additionally, the incidence of back pain complaints appears to be increasing for younger individuals[94], paralleling an increase in body mass at the population level[95]. The possibility that back pain symptoms (in young Canadians at least) are an explanation for increased body mass seems remote. Although direct evidence of temporality is absent, existing evidence refutes the possibility that chronic back pain symptoms are a significant contributor to obesity in patients with low back pathology (i.e. a bi-directional relationship).

Analogy

In addition to the well-established association between obesity and metabolic/ cardiovascular health problems[96], the influence of obesity on comparable musculoskeletal pathologies is an important consideration for causality[88]. Perhaps the strongest evidence of an analogous effect is the substantial increase in risk of osteoarthritis in the lower extremity for individuals with a high BMI[97], along with an increased incidence of subsequent hip and knee arthroplasty[98]. A BMI-dependent increase in symptoms and disability in patients with existing osteoarthritis has also been established[99], which parallels the increased severity of back pain symptoms in patients with elevated BMI[69]. Consistent with the findings following bariatric surgery[89], weight loss results in reduced pain and improved (perceived) function in patients with knee osteoarthritis, even those with substantial degenerative changes[100]. Obesity is also associated with a variety of other musculoskeletal pathologies, including skeletal abnormalities[101], upper extremity disorders[102], and foot dysfunctions[103], and is associated with an increased risk of sustaining workplace injury[104]. Evidence of related obesity-dependent pathology in other body regions is particularly important if it can be postulated that a specific injury mechanism is common to all conditions.

Mechanism

Elucidating the mechanism by which an exposure produces an outcome is one of the most convincing means to support causality[71]. Although back injury is certainly multi-factorial, with genetic and inflammatory aspects accounting for some variation[68,105], increased mechanical load is the most widely-accepted primary and possibly obligatory causal mechanism for musculoskeletal pathology[9]. In the case of chronic lower extremity pathologies, mechanical load, through higher ground reaction and concomitant joint forces, influences chondrocyte activity, inflammatory mediators and resultant cartilage degeneration in synovial joints[106]. Similar mechanisms are thought to underlie degenerative changes in the lumbar spine[83]. However, the obesity-related effect size in the back region (OR = 1.32)[68] is not nearly as large as the lower

extremity (OR = 13)[99], which may indicate a differential exposure to load across body regions. For instance, the load imparted by body weight on the knee joint is largely independent of mass distribution among body segments (i.e. body shape), and cannot be avoided during ambulation. However, in the low back region, mechanical load would be dependent on the proportion of body mass distributed to the trunk, consistent with the larger effect size for waist circumference, rather than BMI, on back pain prevalence[84]. Additionally, although trunk mass increases at a greater rate than total body mass[25], individuals may alter load exposure to the low back by activity modification (e.g. avoidance or different kinematics). While an increase in body mass must result in some elevation to mechanical load[23], and the magnitude of this effect has been examined in the lower extremity[107], it remains to be quantified for the low back. A necessary step towards establishing a potential mechanism by which obesity impacts low back pathology, and to help establish or refute a causal association, is to quantify the magnitude of the obligatory increase in mechanical load that arises due to increased body mass and non-uniform mass distribution.

MECHANICAL EFFECTS OF OBESITY

Biomechanics of obesity

The forces and torques acting about a segment are widely acknowledged as a causal factor in acute and chronic musculoskeletal pathologies[9]. Independent of other factors, an increase in (body segment) mass will produce a proportional change in the force and/or torque acting about an area of interest (a joint). Although numerous authors[103,108] and clinicians consider obesity a causal and/or contributory factor for many musculoskeletal pathologies, there is a paucity of evidence on the specific mechanical effects of increased body mass (and mass distribution) during everyday motions[109].

Paralleling the strong association between obesity and degenerative changes in the lower limb[103], ambulation has been the most common experimental task to study the biomechanical effect of obesity, accounting for 10 of 12 studies in a recent systemic review[109]. Obesity is associated with altered kinematics and increased extension moments about the hip and knee joints during the stance phase of gait (e.g. ~47 Nm)[110], however an overall effect of obesity could not be established across studies due to a diversity of study outcomes. High BMI participants also execute single-limb support with greater displacement[111] and torques[110] in the adduction direction about the knee joint, alterations that are associated with pathological changes to articular cartilage of the superior tibia[112]. Two studies[113,114] have examined a sit-to-stand task, a motion similar to lifting that involves substantial vertical displacement of the body centre of mass. A clear effect of obesity on knee and hip kinematics and kinetics was apparent[113,114], however joint torques reported were normalized to body mass preventing an absolute estimate of obesity-related load increase. Interestingly, one of the few investigations to report absolute torque during a sit-to-stand task in a similar population demonstrated an increase of ~10Nm (~15%) about the hip due to an 8 kg (~12%) increase in torso mass for a participant in the 3rd trimester of pregnancy[85].

Although torques acting about the hip may give an indication of mechanical load effects about the low back[114], the extent that increased body mass influences mechanical load about the low back has not been quantified. One explanation for this paucity of information may be the assumption that obesity-related loading can be easily estimated[23] (e.g. as a simple function of body mass). A second (and more likely) contributing factor is that the influence of obesity on the mass (and mass distribution) of individual body segments involved in lifting (i.e. body segment parameters, BSIPs) has not been well established[115,116]. Valid estimates of BSIPs are necessary for accurate computation of the forces and torques acting about the low back[117]. However, the standard regression-based methods of estimating BSIPs[24,25,118] are not appropriate for individuals with elevated body mass, owing to obesity-related changes in morphology of individual body

segments – a source of variability not reflected in original study populations[24,118,119]. For example, the investigation of mechanical load during pregnancy[85] utilized subject-specific measurements to obtain estimates of body segment mass in their morphologically-atypical sample.

Anthropometrics

Accurate estimates of the inertial properties (mass, moment of intertia) of body segments are required for any kinetic analyses, but particularly during motions with high acceleration and/or where the segment of interest is freely moving[120]. Estimates of BSIPs are typically derived from predictive equations, based upon direct measures from cadavers[121], medical imaging[118] or from volumetric approaches[24]. Although a certain amount of variance can be expected from technique-specific factors[25], the most widely-acknowledged[24,116,118,121] and potentially largest source of variability can be attributed to the characteristics of the sample. The ability to generalize BSIP equations to populations with different morphologies than the original sample is not supported[116,121]. Early studies focused nearly exclusively on samples of otherwise-healthy, Caucasian, male subjects[24,118] or similar cadaver-based samples[121]. Two of the most widely-used estimates were developed by McConville[24], from a representative sample of US Air Force personnel, and Zatsiorsky (refined by de Leva[118]) on a sample of young physical education students (mean age of 24), neither of which are reflective of the Canadian population where the prevalence of overweightness and obesity is ~65%[60]. More recently, investigators have provided BSIP estimates for samples including children[122], different ethnic groups[123], and the elderly[116].

Only a small number of studies (4) have examined changes in BSIPs specific to obesity[115,116] or across a wider range of body masses[24,25]. Consistent with a central accumulation of body mass, trunk mass tends to increase at a greater extent than whole body mass[25,116]. This effect would introduce systematic error in predictive equations derived from normal weight populations, by under-estimating trunk mass in high BMI individuals. Although predictive equations have been derived from samples that include individuals with BMIs as high as 29.5[24]

and 39 kg/m^2 [25], a comparison of variance (or error) in BSIP estimates specific to high BMI participants has not been performed. For example, across all participants (including a BMI of 39 kg/m^2), predictive equations provided by Pearsall and colleagues[25] accounted for a substantial amount of variation in actual BSIPs (r^2 = .77 to .87), however large absolute errors can arise in spite of high correlation if systematic offsets[24] or heteroskedasticity are present. In fact, estimating abdomen mass as a function of height and weight would lead to under-estimations (~6.5%) for 4 of the 5 participants with a body mass above 100 kg in the Pearsall[25] sample. Obesity also affects the longitudinal centre of mass locations (CM_{loc}), characterized by a shift towards a more proximal location[116], and corresponding poor fit of predictive equations[25]. The BMI-dependent changes in mass and CM_{loc} have not been incorporated into existing BSIP equations and may lead to errors (which are currently undetermined) in BSIP estimates of high-BMI individuals, and consequently on load calculations.

The most compelling evidence for an obesity-specific error in BSIP estimates is provided by sensitivity analyses of existing BSIP models[124,125]. In addition to significant variances due to age and gender on BSIPs, Durkin and Dowling[124] reported substantial differences in BSIP estimates across predictive models and body segments. More recently, a clear indication of a systematic, body mass-dependent error in BSIP estimates was reported for whole body inertia, where predictive models over-estimated values for individuals with a BMI below 18.5 ('lean'), returned similar estimates for BMIs of 18.5 – 24.9 ('normal'), and under-estimated whole body inertia for individuals with BMIs above 30 kg/m^2 (obese)[125]. Considering the obesity-dependent increase in trunk mass distribution[116], a similar pattern of error would be anticipated for trunk mass (i.e. under-estimation at high BMIs). Further, the segment with greatest BSIP variation is the trunk, which ranges from 47 - 59% of body mass[24,25], with even larger differences for the central trunk/abdomen region[25]. The impact of obesity on predicted trunk mass has not been tested, nor is it known if this effect is consistent across other BSIP parameters (e.g. inertia, CM_{loc}

and mass). The effects of obesity-related BSIP errors on kinetics was recently demonstrated for a sample of adolescents with high BMIs (> 40 kgm2)[126], where mechanical energy expenditure derived from predictive equations was 40% lower than estimates that accounted for individual-specific variation in BSIPs. Similarly, somatotype (i.e. meso-, ecto-, endo-morph) accounted for approximately 15% of the variation in torque estimates during a kinetic analysis of reaching motions[127], and body mass-dependent effects have also been demonstrated for lower extremity torques during the swing phase of gait[120]. Although there is evidence of obesity-related BSIP errors for the whole body[125,126] and upper[127] and lower[120] extremities, a corresponding comparison has not been performed for the trunk segment. This is particularly important to establish, as trunk BSIPs are a principle determinant of back load, and the trunk is the body segment most affected by obesity-related morphological changes[25].

In addition to population-specific effects, ideal BSIP predictive equations should also account for morphological variation within a population, an effect that varies across body segments[25]. When compared to individual-specific medical imaging, no single set of predictive equations can consistently account for actual BSIP differences across all body segments[124]. Considering the limited ability of BMI to predict fat distribution or morphology, as well as the extremes of mass distribution reflected in android and gynoid somatotypes[128], it would be expected that individual variation in BSIPs will increase proportional to body mass (i.e. be even greater for high BMI individuals). One solution to morphology-specific BSIP errors is to develop predictive equations specific to each morphological variation, however this approach necessitates developing a multitude of predictive equations[124], especially if one considers specific diseases and conditions (e.g. stroke to SCI to amputation).

An alternate is to consider methods of obtaining direct, individual-specific BSIP estimates. In fact, an aim of early geometric BSIP models was to account for subject-specific morphology by modeling body segments as geometric primitives[119]. However, the ability of

simple geometric models to account for variations in morphology is limited[124], a problem exacerbated by the use of predictive equations to derive initial segment masses[129], from which density and corresponding inertial parameters were computed[119]. More recent geometric-based methods include photogrammetry[24], laser-scanning[120], direct measures[85] and advanced mathematical approaches[130]. Photogrammetric approaches offer a number of advantages for acquiring individual-specific BSIP estimates, including a non-invasive nature, good accuracy (with appropriate segment densities[25]), and low cost[24,122]. Laser-scanning may provide improved resolution of surface geometry, but has a high cost ($30 – 50k), while mathematic approaches are encumbered by numerous (> 200) direct measurements[130]. Lastly, unlike nearly all medical imaging techniques (except standing MRI) photogrammetric (and laser-scanning) approaches are not susceptible to variation in body segment dimensions arising from the shift to a supine position[116,118]. The supine orientation used for most medical imaging techniques can increase segment length[24] and causes a redistribution of (adipose) tissue[115]. Simple, low-cost methods of obtaining subject-specific BSIPs address the problem of providing population-specific predictive equations for every conceivable combination of body segment, age[116], gender[124], ethnicity[123], and body mass[115]. Considering the substantial increase in prevalence of obesity[60] and strong association of obesity and numerous health conditions[96], it is important to elucidate the mechanisms underlying obesity-related health risk. Accurate estimates of BSIPs are essential for determining the mechanical consequences of obesity.

Back biomechanics and obesity

Although the extent that an increased body mass affects lumbar spine load has not been quantified, the underlying biomechanics are well-established[131] and do not require complex models for initial estimates[23]. For the purposes of this manuscript the terms ‘mechanical load’ or ‘low back load’ will refer to the collective/general mechanical influences acting on the low back (i.e. torque and/or force), which are not well-distinguished in epidemiological literature[132].

Mechanical load acting on the lumbar spine is often estimated about the L4/L5 or L5/S1 joint, which is one of the most frequent pathological segments[21]. In addition to torque[133], common back loading parameters include the resultant forces acting parallel (shear) and perpendicular (compression) to the joint surfaces[10]. An association between mechanical load and pathology is well established[6], with corresponding estimates for the *in vitro* load tolerances of specific (healthy) anatomical structures, such as ligaments (rate dependent)[134], facet joints (~2000N)[135], and vertebral end-plates (~8000 N)[136]. These tolerances, as well as epidemiological investigation of workplace injury[137], contributed to development of mechanical thresholds for compressive (3400N) and shear forces (1000N) delineating deleterious loading exposure (during materials handling)[21]. However, the above load tolerances for specific tissues vary widely between individuals (e.g. due to age, load exposure[136], position[138]) and the validity of the mechanical thresholds has been questioned[139]. Although modification of mechanical factors is the focus of many injury prevention strategies[21], current biomechanical-based ergonomic methodologies do not take into account the effect of increased body mass, or differences in mass distribution, between individuals. Table 1 (below) lists the factors implicated in altering torque about the lumbar spine.

Table 1. Principle determinants of torque acting on lumbar spine during lifting (a 15 kg external mass from floor to table height). An estimate of the relative contribution and absolute magnitude of each factor is provided.

	contribution (%), magnitude (Nm)[1].	Description
Torque, RJM	100%, 250 Nm	Reflects the net rotary tendency of all forces acting about a joint. Computed via inverse dynamics, as the sum of static and dynamic torques. (RJM = T_w + T_d).
Static torques	70%, 180 Nm	The sum of all torques arising from weight. Includes segment weights and weights lifted. (where w = mg)
body segments	*55% (of static), 100 Nm*	The torques produced by the weight of body segment. Proportion to weight and distance to axis of rotation. (T_w = f_w x d)
external load	*45% (of static), 80 Nm*	The torques produced by the weight of the object lifted. Proportional to weight and distance to axis of rotation. (T_w = f_w x d)
Dynamic torques	30%, 70 Nm	The torques required to produce angular accelerations of limb segments and external masses. Product of angular acceleration and inertia. (where I = mr^2)
body segments	*50% (of dynamic), 35 Nm*	The torque required to accelerate body segments. Proportional to inertia and angular acceleration. (T_d = Iα).
external load	*50% (of dynamic), 35 Nm*	The torque required to accelerate the object lifted. Proportional to inertia and angular acceleration. (T_d = Iα).

[1]Anticipated effects, see *Influence of obesity on BSIPs*.
RJM = resultant joint moment, T_w = torques from weight, T_d = dynamic torques
w = weight, m = mass, g = gravitational acceleration
f_w = force of weight (or simply weight), d = moment arm
I = inertia, α = angular acceleration

Static loading

Static moments and forces account for a substantial portion of mechanical load during certain materials handling tasks, such as standing work[40] or symmetrical lifts of moderate loads[29]. In fact, a number of biomechanical investigations of materials handling have considered only static factors[29,140], and so far only static factors have been incorporated into injury prevention guidelines[21]. Contributions to static loading include the weights of body segments, external weights, or other external forces applied through cables, and the distance at which these forces act from the lumbar spine[10]. The relative contribution of these factors has been examined for normal weight individuals, and may be important considerations for selection of lifting technique[141] or for guiding injury prevention strategies. For example, a stoop-style lifting technique may minimize the moment of weight of a large or bulky external load, but at the expense of increased torques from torso weight. An elevated body mass would alter the relative contributions of torques from body segment weights and the mass lifted, however the effect of obesity on even

simple static factors is not straight forward. For individuals with high BMI, and thus a greater mass distribution to the trunk segment[116], the increase in compressive force arising from segment weight will not be a simple function of total body mass, but depend upon the masses of involved segments (head, upper extremity, trunk) – that is, the mass distribution. In the absence of accurate estimates of mass distribution amongst body segments, compressive force would be under-estimated in high BMI individuals, similar to the effect of BMI on whole body inertia[125] and energy expenditure[127]. Accurate estimates of static load are important for risk assessment during repetitive or long duration tasks that are implicated in cumulative trauma of lumbar spine segments[7].

In addition to increased torso mass, a further increase in back loading would be expected in high BMI individuals due to the increases in flexion torque imparted by the abdominal fat mass (i.e. panniculus adiposis) – similar to the effects during pregnancy[85] or when holding an external mass in front of the trunk. Torques arising from the abdominal fat mass would also increase compressive loading on the spine due to the muscle or ligament forces necessary to resist the added flexion torque[142]. In fact, a relatively small flexion torque can result in substantial increases to compressive force, due to the comparatively small moment arm of lumbar extensor muscles (7 – 10 cm)[143]. In addition to the mass (weight) of the torso, the magnitude of the flexion torque arising from the abdominal mass is also proportional to the moment arm, or distance at which the weight of the abdomen acts from the lumbar spine axes of rotation. The moment arm is derived from the anterior-posterior centre of mass location (CM_{loc}) of the torso, relative to the lumbar joint centre. Simply estimating the mass of the torso is insufficient to determine the effect of obesity on static lumbar spine loading, as the corresponding changes in mass distribution of the torso must also be considered (i.e. moment arm effects related to CM_{loc}).

Moment arm

Estimates of the lumbar joint centre are often derived based upon a fixed distance from the posterior trunk[143], however this assumption is not appropriate for high BMI participants, due to the presence of subcutaneous fat that would alter the distance between the lumbar joint centre and posterior trunk. An alternate approach to estimate the joint centre is as a percentage (33%) of the anterior-posterior dimensions of the trunk[10]. This relative estimation would provide a moment arm location proportional to trunk dimensions, however would not be sensitive to the obvious anterior shift in abdominal mass distribution (i.e. the abdominal pannus), and thus only partially account for the effect of increased body mass. In addition to an obesity-adjusted joint centre position, an estimation of the CM_{loc} is required. Two-dimensional medical imaging approaches[118] are not able to provide estimates of anterior-posterior CM_{loc} (as mass distribution is quantified in the superior-inferior and medial-lateral directions only), which is often modeled as lying along the mid-line of the trunk segment[144]. Recent photogrammetric approaches have returned CM_{loc}s that are not coincident with the longitudinal axis of the trunk segment[122], a finding that has been confirmed with 3-dimensional medical imaging techniques[115]. However, medical imaging approaches require participants to adopt a supine position, which alters the distribution of abdomen tissue (and thus CM_{loc}), particularly in high BMI individuals with a large quantity of malleable adipose tissue[115]. Valid estimates of the CM_{loc} in a standing position are necessary to estimate the effect of increased BMI during materials handling (which occurs predominantly in standing).

Moment arm of external load

When handling external masses, a central accumulation of body mass may further increase static load about the back due to physical interference (i.e. objects must be held further from the body). This has been demonstrated for a standing work task, where high BMI participants were found to stand 6 cm, or 35% farther, from the work surface compared to height-

matched, normal BMI participants[40], an effect that was attributed to a difference in torso dimensions (i.e. waist circumference difference of > 40 cm in high BMI participants). This would increase the moment of weight about the lumbar spine for any external masses handled/moved, and also for torques arising from limb segment weights, as the upper limb would necessarily be held farther from the body (or alternatively, the trunk flexed farther forward). In the standing work task, these factors contributed to a static hip joint moment that was ~15 Nm (328%) greater in high BMI participants[40]. Although similar effects would be anticipated during lifting, the task is more complex, involving multiple body segments and a horizontal load position that can be modified by the participant (e.g. a load may be handled at varying distances from the body). Load attenuation via reductions in the moment arm of external masses is a widely-used method of mitigating injury risk during materials handling[21]. In fact, for each 10 cm increase in horizontal distance at which a load is handled, current guidelines recommend a ~17% reduction in load weight[21]. It seems obvious that certain anthropometrical variations in body morphology would limit the ability of an individual to reduce the moment arm of external load, however the magnitude of this effect has not been estimated during lifting tasks. Understanding the influence of individual-specific factors (e.g. anthropometrics) on static loading is relevant when designing and delivering interventions aimed at minimizing injury risk for the Canadian population.

Dynamic loading

Lastly, dynamic torques, arising from inertia and applied accelerations, are important contributions to back torque during lifting motions. During the initial 0.1 – 0.3 seconds of a lift dynamic torques can contribute an additional 30% to net joint torques beyond static factors[29], consistent with large upward acceleration of body segments and high moment of inertia of distal segments. During lifting motions performed at high speeds, which are typically executed with high accelerations, dynamic torques can contribute an additional 190% to lumbar spine torque[42].

Failing to account for dynamic torques can lead to severe underestimations of peak torques during lifting tasks.

Similar to static load estimations, the paucity of BSIPs estimates in high BMI individuals precludes accurate estimates of the effect of body mass distribution (moments of inertia) on inertial or dynamic loading about the lumbar spine[23]. In fact, trunk inertias vary over an even larger range than trunk mass, with a 3-fold increase ($1.0 – 3.6 \times 10^7$ g*cm^2) about the somersault axis from small to large participants[25]. Lumbar loading due to dynamic factors may be even larger than anticipated by body mass alone due to the acceleration[23] and inertia[25] changes with obesity. Additionally, there is evidence high BMI participants may prefer a movement strategy involving increased trunk acceleration (51% higher than normal BMI counterparts), a choice that would further increase dynamic torque[23]. The extent that obesity increases dynamic loads about the lumbar spine during materials handling has not been quantified. Further, the additive contributions of static (weight) and dynamic (acceleration) loading in a sample of high BMI individuals have not been elucidated using even a simple model of lumbar spine loading.

Summary and Objectives

It is obvious that increased body mass affects BSIPs, and there is preliminary evidence for the pattern and magnitude of these changes[115,116], however a more complete examination of BSIPs in high BMI individuals is required. Current predictive equations are inadequate to estimate BSIPs for individuals with a high BMI[25,118], but are still the only method available - an evaluation of the extent that this may introduce error in BSIP estimates has not been performed. Further, individual variation in morphology (and thus BSIPs) is likely greater for individuals with high BMI[25,128], supporting a need for individual-specific approaches, rather than improved predictive equations. Lastly, these limitations have contributed to a paucity of estimates for the

obesity-related changes in lumbar spine loading. An investigation of the direct mechanical effects of obesity on back load will provide the necessary elucidation to understand the role that obesity has on adversely impacting back loading[23]. Determining the relative contributions of the determinants of lumbar spine load (e.g. body segments vs external load) in high BMI individuals will aid in identifying obesity-specific back injury mechanisms.

The study entitled, *The effect of obesity on body segment parameters and lumbar spine load,* has 3 objectives:

1) use an individual-specific, photogrammetric method to quantify change in BSIPs (mass, mass distribution, inertia & CM_{loc}) attributable to increased body mass,

2) compare BSIP estimates derived from individual-specific BSIPs to existing predictive equations, and

3) use individual-specific BSIPs to quantify the mechanical effect of increased BMI on the lumbar spine loading during standing, carrying and lifting tasks.

MATERIALS HANDLING

Material handling, specifically lifting, is the most widely cited environmental risk factor for back injury[6]. Although the etiology is not clear[73], lifting injuries are proposed to result from either maximal (one-time) or repetitive (submaximal) exertions[9,21,135]. Materials handling injury prevention guidelines have been developed based upon mechanical, physiological and perceptual lifting outcomes[21]. For instance, a 3400 N compressive load tolerance (mechanical) was chosen to guide the selection of weight lifted during occupational tasks and accommodations for repetitive lifts have been developed based upon energy expenditure (2.2 – 4.7 kcal/min) (physiological)[21]. These workloads are thought to be perceived as comfortable/acceptable by the vast majority of the workforce (99% of men, 90% of women)[21]. Although it is disputed which factor might contribute the majority of variance to materials handling injury (none of which account for repetition-to-repetition variation), and thus form the basis of prophylactic interventions[145], all factors are recognized as an important consideration in materials handling tasks[21].

Increased body mass is associated with a number of musculoskeletal disorders that are managed with ergonomic interventions[146]. In fact, workers with an increased body mass are more likely (4x) to report pain that restricts the ability to work than those with a normal BMI[103]. Evidence that body mass need be considered for certain vocational tasks has recently been put forth[62], but remains to be thoroughly investigated for materials handling tasks[23]. An evaluation of the effect of body mass during lifting tasks should consider physiological, perceptual and mechanical factors involved in materials handling.

Mechanical

Mechanical risk factors can be partitioned into aspects related to the external load and those specific to the individual[6]. The influence of the external load on static moments during materials handling is fairly well established[147], and workplace tasks involving large external masses are associated with an elevated risk of back injury[3]. Similarly, static forces acting about

the lumbar spine, particularly for pushing and pulling, can contribute to deleterious torsion moments[148]. Not surprisingly, the magnitude and direction of the external force created by a load carried, pushed, pulled or lifted were among the first factors considered for prophylactic material handling guidelines[21] (e.g. selection of 'stoop' versus 'squat' lift technique[43]) . Interventions that eliminate the need to handle an external load (e.g. via removal of a job task or use of assistive device) can achieve a reduction in work-related back injuries attributable to lifting[149], however avoidance of lifting is not universally applicable and there is a need to further understand factors that influence (static) loading about the lumbar spine.

For an external load, the static moment acting about the lumbar spine is directly proportional to the distance between the load (i.e. line of action of weight) and lumbar spine axis of rotation[86,150]. Gilleard and colleagues[40] demonstrated that this distance is increased for high BMI individuals during a standing work task (+35%, relative to normal BMI), with a corresponding increase in static torques about the hips (+320%). These findings demonstrate that small changes in position can result in large changes to static moments, perhaps due to the multiplicative effects of an increase to body mass and distance, or from additional contributions that have not been elucidated (e.g. torques from weight of upper limb segments). Similar effects would be anticipated during lifting tasks, however lifting involves multiple limb segments and a more complex motion than standing work, making the influence of anthropometric-dependent factors more difficult to predict and measure. For instance, unlike a static task where work position is fixed[40], the position of an external load can vary dependent upon the individual's preference[44], and only a strategy that minimizes the moment arm of an external load would result in a BMI-dependent effect (i.e. constrained by torso dimensions). Additionally, during both standing work and lifting tasks it is important to consider the orientation of all involved body segments, as an individual can reach/hold an external object utilizing either trunk or upper extremity flexion (or some combination thereof). Considering the obvious differences in the trunk

and upper extremity mass[24], approaches involving trunk versus upper extremity flexion would be associated with different loading effects. An understanding of the factors influencing the position of external loads during materials handling is important as they may limit the ability to adapt/adhere to certain ergonomic interventions (i.e. minimizing the distance of external load).

Lifting technique

The most common individual-specific mechanical factor is lifting technique[38], which is typically characterized by the absolute and/or relative orientation of body segments at initiation of a lift[34]. Although most practitioners advocate for a knee-dominant lifting technique, with an emphasis on an upright trunk position (squat-style), evidence to support this position is scant[31]. In fact, the near-opposite, hip-dominant approach involving a flexed trunk (stoop-style) is favored by some, on the basis of lower energy expenditure[151] and effort[152] per lift, as well as improved balance[153]. Certain movement patterns (e.g. twisting, end-range lumbar flexion) are independent of lifting technique[30] and can be associated with deleterious effects on the spine during lifting tasks, such as: decreased activation of agonist muscles, a shift in load to passive structures, or elevated torsional stress on discs[14]. Further, these isolated effects may influence back injury risk differentially across individuals, evident by the numerous sporting motions incorporating these movements with no corresponding increase in back injury incidence (i.e. individual factors may mitigate or exacerbate these effects). Perhaps not unexpectedly, injury prevention interventions aimed at instructing individuals on 'safe lifting' techniques (usually squat-style), have had no effect on workplace injury rates[32]. In addition to the obvious limitation of prescribing a single lifting technique, this approach may leave an individual ill-prepared for circumstances not suitable for squat-style lifts (e.g. all lifts of large objects; objects in bins/containers; awkward lifts). Injury in these circumstances may not arise simply from biomechanical factors, but also due to a lack of familiarity with the required movement pattern and/or an absence of specific muscular adaptations (conditioning), both of which may be by-products of 'single-technique' lift

training. Investigators have also postulated that the absence of benefit (from 'safe lift' training) is due to a failure of participants to adopt the techniques provided[43], which might be attributed to an absence of the necessary 'physical literacy' required to execute a variety of different lifting motions proficiently. These observations have led to an increased emphasis on examining the determinants of lifting technique, in other words, why an individual may prefer a certain lifting technique (body segment orientation) over another.

In addition to physical constraints of the task (e.g. height, obstructions), lifting technique is influenced by the weight lifted[47], the duration[35] and familiarity with the lifting task[37], muscular strength and endurance[35], and the presence or history of back pathology[38]. The influence of anthropometrics on lifting technique has received comparatively less attention. In a recent study on the effects of sex and strength, Li[35] provided predictive equations for lifting technique that also included BMI (and height) as a variable – in this case, an increase in BMI was related to a shift towards increased trunk flexion. However, the sample was not specific to individuals with high BMI (mean weight: 79.5 kg; height: 1.81 m). In a more specific sample, obese individuals were found to stand with increased trunk flexion during standing work tasks[40], perhaps as a compensatory motion for physical obstruction arising from the abdominal mass. In contrast, during a sit to stand task high BMI participants chose to move with less trunk flexion than normal BMI counterparts, a strategy that attenuates moments about the hip (and back) and increases those about the knee[113,114]. These findings suggest the effect of increased body mass on the orientation of body segments is task-dependent, and is influenced by a need for compensatory trunk flexion to accommodate for abdomen obstruction[40] and a competing attempt to minimize trunk flexion in order to spare the back from torques imparted by torso weight[114]. Lifting tasks involve both the possibility of physical interference of the abdomen, as well as a need to spare the low back from excessive loading, necessitating an exploration of possible obesity-related changes to body segment orientation during lifting tasks.

Lastly, the kinematics of materials handling tasks, specifically velocity[42], acceleration[23], and jerk[50], are important contributions to mechanical load and also reveal how an individual chooses to execute a motion[50], independent of mechanical factors. Similar to lifting technique, kinematics are affected by the magnitude of the external mass[45,47], the duration of the lifting task and fatigue[154]. Only a single study has examined kinematics in high BMI individuals during lifting[23], where increased body mass was associated with greater angular velocity (30%) and acceleration (51%) of the trunk during a short duration, standardized task (no difference was detected in trunk orientation). This strategy, which would further increase inertial loading about the spine in high BMI participants, was contrary to the authors' *a priori* hypothesis of a compensatory reduction in acceleration magnitude by high BMI individuals. Notably absent from the study was a measure of external load kinematics[23], however previous investigations have revealed differential approaches to handling external loads are possible (e.g, based on lifting experience[37]). It is possible that BMI-dependent kinematic alterations are not restricted to just a single segment (i.e. the trunk). Variations in loading arising from differences in body segment acceleration (190%)[42], are much larger those attributable to lifting technique or limb segment orientation (0-33%)[43]. Kinematic differences are also relevant for energy expenditure during lifting[21], and perhaps as an indicator of overall movement quality[39]. Considering the obvious influence of a high BMI on the body segments involved in a lift, as well as the probable indirect effects on the object lifted (i.e. increased distance), investigations examining the kinematics/orientation of both body segments and external load during lifting are needed.

Physiological

The most common physiological measures obtained during materials handling are oxygen consumption (or related measures, including HR)[21] and muscle activation[154]. While measures of muscle activation are relevant for certain biomechanical models[10] and injury mechanisms[17], both cardiovascular demand and activation are important outcomes during repetitive tasks[151]. In fact,

recommendations for materials handling tasks based upon cardiovascular rationale often differ from those obtained using a biomechanical basis. For instance, it may be more efficient to lift heavier loads once, rather than partitioning the task into multiple lighter loads[21] – a strategy that is adopted by stronger individuals during free-form handling tasks[35]. Consistent with the external work performed, cardiovascular workload depends upon lifting frequency and displacement[21], and has been shown to be sensitive to small changes in external mass (as little as 2 kg) during short duration, higher frequency lifts[155]. It would be expected that for an equivalent task, participants with a high BMI would require greater oxygen expenditure due to an increased body mass, which in the absence of cardiovascular adaptations, should manifest as an increased heart rate.

One approach to attenuate potential body-mass related cardiovascular differences might be to adopt different lifting techniques or kinematics[151]. For example, stoop-style lifting techniques are associated with approximately 13% less oxygen expenditure compared to squat-style techniques[151], and are also cited as advantageous for lifts of large or bulky objects[43]. It is possible that individuals with a high BMI elect to work at a similar cardiovascular effort as normal BMI counterparts, but adopt a lifting technique (stoop-style) that minimizes vertical displacement of body mass – in spite of potentially deleterious lumbar spine loading (due to a forward flexed trunk). In fact, cardiovascular changes, and not biomechanical factors, predict when individuals choose to reduce the load lifted[156], and are also associated with a shift towards a stoop-style lifting technique[151]. In spite of evidence supporting an association between cardiovascular effort, lifting technique, and mass, no study has examined cardiovascular, technique and kinematic differences between high and low BMI individuals during materials handling. An understanding of the relationship between physiological and mechanical lifting outcomes and body mass is relevant for ergonomic interventions that use of these relationships to guide injury prevention strategies.

Perceptual

Lastly, ratings of perceived exertion (RPEs) are often used in combination with, or as a surrogate for physiological measures[21], and may be important determinants of lifting behavior independent of objective physiological measures[151]. Perceived exertion is thought to be a task-dependent[157], non-linear combination of muscular and cardiovascular efforts and individual stimulus-perception characteristics[158]. Within a single task, whole body RPE is strongly related to cardiovascular effort[159], and tends to increase proportional to lifting frequency[160], height[161], direction[162] and distance[147] , effects consistent with the physiological and mechanical loads imparted by these factors. However, unlike objective physiological measures, whole body RPE does not vary across lifting techniques or tasks[163], suggesting individuals select a consistent level of whole body exertion, regardless of the relative contributions of underlying body regions. This is supported by findings of differences in region-specific RPEs across different lifting scenarios, but uniform whole body perceived exertions[151]. Preliminary investigations for the effect of increased body mass have reported significantly greater RPEs in high BMI individuals for certain body regions during static positions (up to 144%)[62], an effect attributable to increased torques from segment weight. It is possible that high and normal BMI individuals elect to work at similar amounts of whole body RPE, but with anthropometric-dependent differences in region-specific exertions. Similar to cardiovascular outcomes, increased RPE also influences lifting technique, and is predictive of a shift from squat- to stoop-style lifting techniques[164]. Although the effects of external work and cardiovascular effort on RPE are well-established, the influence of body mass on RPE during manual handling has not been thoroughly investigated. In fact, a discrepancy regarding the influence of mass on all lifting outcomes exists[21], dependent upon whether the mass is external (e.g. load lifted – direct effect) or internal (e.g. body mass – no effect or not considered) to the individual.

Perceptions of physical exertion are important for the safe performance of many vocational tasks and underlie formal techniques used to design workplace tasks that minimize injury risk[165,166] (i.e. maximal acceptable weight of lift, or MAWL). The premise of incorporating perceived measures in the design of manual handling tasks is that individuals are able to perceive when a lifting task would increase injury risk[156], independent of the underlying source of elevated risk (i.e. peak load, fatigue, etc). Interestingly, using perceived measures to guide weight selection is associated with comparatively higher loading on the low back[156], and results in weights lifted that exceed those based upon biomechanical criteria[21]. Further, individual-guided selection of lifting weight has been criticized for the tendency of most individuals to over-estimate lifting ability, particularly for long duration lifting tasks[167]. Only a single study has evaluated the effect of increased body mass on weight selection[158], and similar to the effects on kinematics[23], the hypothesized compensatory reduction in weight lifted was not supported. In fact, there was a tendency for high BMI participants to select a heavier weight across most frequencies, particularly at a pace of 4 lifts/minute (one of the more commonly investigated lifting frequencies[156,168]). The authors speculated that high BMI high individuals may have adopted more biomechanically advantageous lifting techniques which lowered physical stress (as has been suggested for walking[169] and sit-to-stand tasks[114]), however lifting technique was not reported. Alternatively, it was also acknowledged that increased body mass may be associated with a decreased ability to perceive physical stress[170] or judge lifting ability (i.e. stimulus-perception). Incorporating measures of RPE and cardiovascular effort during lifting tasks would enable differences in the relationship between physical load and perceived stress between high and normal BMI individuals to be considered. Knowledge of region-specific variation in RPE related to body mass may also help counsel individuals on lifting techniques that address these differences, or understand why an individual selects one lifting technique over another.

Repetitive lifting

Repetitive (> 150 lifts/day[171]) or long duration (> 2 hours[21]) lifting is often cited as a risk factor for low back injury[7,37], yet the majority of investigations of kinematics, exertion and lifting technique during materials handling have examined relatively short duration (< 20 minutes) lifting tasks[23,158]. Even at relatively low loads (25% of lifting capacity), alterations to lifting kinematics are evident during high-rate repetitive lifts (> 35 lifts/min), characterized by a decrease in angular velocity and a reduction in lifting pace[11]. Paradoxically, participants may also shift towards increased accelerations over repetitive lifts of longer duration, evident by a 2-fold increase in distal segment peak acceleration[154] - a strategy that would increase low back load with all other factors held constant. Alterations in lifting technique (increased lumbar flexion and a shift to a stoop-style technique) also occur over the course of a repetitive lifting trial[11,172]. Even among studies examining repetitive lifting, most have focused on high-paced, short duration tasks (+ 20 lifts/min, < 2 minutes) intended to elicit muscular fatigue[173], and comparatively fewer exist evaluating longer duration, slower-paced lifts that are more representative of vocational lifting tasks[174].

Additionally, the effects of obesity during materials handling tasks has only been examined during short duration lifting tasks[23,158]. It is plausible that mechanical influences of obesity on physiological (heart rate, fatigue) and perceptual outcomes are exacerbated during long duration, repetitive lifting tasks. For instance, elevated body mass increases the rate of fatigue of lumbar musculature[48,154], yet relatively little is known about the manifestation and consequences of lumbar fatigue during repetitive lifting tasks. Body mass- dependent alterations in technique have also been detected over multiple repetitions during a sit-to-stand task, where high BMI individuals execute the initial repetitions with a relatively upright trunk posture[113], but shift towards a more flexed trunk posture by the end of trial. This was also associated with a substantial redistribution in moments about the hip (increased) and knee (decreased)[113], which

likely influenced the activation (and effort) in the surrounding musculature (i.e. hip and back extensors). Although the sit-to-stand findings for high BMI participants are similar to the alterations observed during repetitive lifting in normal BMI individuals (i.e. increased trunk flexion)[154], the change by high BMI participants occurred in a small number of repetitions (10) and was not observed in the normal BMI participants. Only a limited number of studies have considered the effect of body mass on lifting technique, kinematics or outcomes[23,62,158]. An investigation of longer duration repetitive lifting may help confirm the existence of BMI-specific lifting alterations (e.g. increased acceleration) and provide important information regarding the effect of obesity during circumstances where the risk of injury may be elevated.

Summary and Objectives

Mechanical, physiological and perceived factors are important outcomes during materials handling[21], however the changes between variables and across situations is not always predictable[44]. Evidence supporting an effect of obesity has been provided for certain outcomes (e.g. kinematics)[23,40], but remains to be explored for others. The direction of the obesity-related effects can be hypothesized based upon principles of mechanics and physiology, however the actual manifestations are often contradictory (e.g. increased mass lifted[158], higher acceleration[23]). Additionally, the effects of increased body mass would likely be exacerbated during repetitive lifts, yet most studies have been restricted to short duration tasks. Understanding how individual factors influence lifting outcomes is important, as there is growing emphasis on providing individual-specific ergonomic interventions[146] and shifting the emphasis of interventions towards individual, rather than environmental, factors[145].

The study entitled, *The influence of body mass on lifting strategy during repetitive, fixed-pace lifting*, has two objectives:

1) determine the effect of increased body mass on lifting technique and kinematics of the principle body segments and the external load during a long-duration, repetitive lifting task, and

2) determine the effect of increased body mass on cardiovascular effort and perceived exertion during a long-duration, repetitive lifting task.

FREE-FORM VERSUS PROMPTED LIFTING

Individual (e.g. body mass[23]) and task-related variables (e.g. load lifted[168]) are not the only factors that exert an influence on lifting technique and kinematics during materials handling tasks. For example, lack of knowledge of the mass lifted can influence lifting technique, muscle activation and the corresponding load (increased) about the lumbar spine[175]. Similarly, performing mental tasks concurrent with manual handling alters the kinematics and corresponding kinetics[176]. Another near ubiquitous factor that has not been considered is the presence and nature of the prompting used to guide lifting tasks.

While a certain amount of standardization of lifting tasks is required to examine the influence of specific external factors (e.g. mass), the intended reduction in variance often occurs at the expense of ecological validity and/or generalizability[50]. For example, during a free-form, self-paced lifting task, Faber and colleagues[44] demonstrated that reducing the mass lifted did not result in a proportional reduction in lumbar load, due to a concomitant increase in acceleration of the trunk. In contrast, during more standardized tasks (i.e. foot position constrained) the alterations in trunk acceleration associated with changes to mass lifted are negligble[177]. Similarly, Puniello[50] attributed weak associations between strength and lifting kinematics in older adults to the selection of a free-form lifting task. Free-form lifting tasks enable participants to execute a lifting motion using their preferred technique and kinematics, without being constrained by lifting pace[168], foot position[23] or even technique[152]. In fact, Puniello[50] suggests "it is unlikely subjects used their natural lifting strategy" in experiments that have "imposed specific strategies on test subjects". Although this has been offered as an explanation for certain results[39,50], no deliberate examination of changes to 'natural lifting strategy' has been performed for any of the impositions during materials handling investigations. Therefore, it is not known if controlling certain task variables causes participants to adopt strategies that are different than those used during activities of daily living or occupational tasks performed outside the laboratory setting.

Lifting frequency

Lifting frequency is likely the most commonly constrained aspect of lifting[37,168], with fixed-pace tasks providing important knowledge regarding the effects of numerous ergonomic variables (e.g. the relationship between lifting pace and cardiovascular exertion)[21]. Although authors have presumed that lifting frequency will be a substantial determinant of lifting kinematics[43] (i.e. a faster pace necessitates faster individual lifts), the actual relationship between frequency and kinematics (and the corresponding kinetics) is likely more complex. For example, Marras[37] reported that lumbar spine compressive load was not affected by alterations to lifting frequency (2 - 12 lifts/min), particularly for inexperienced lifters. These findings are consistent with an individual preference towards certain techniques or kinematics (i.e. a 'natural lifting strategy')[50], which is not altered unless specific constraints of the task demand otherwise (e.g. a dramatic increase in lifting pace). As another example of dissociation between frequency and kinematics, the instructions provided during rehabilitative exercise can alter limb acceleration, independent of movement frequency or cadence, resulting in a change in joint torque[178].Therefore, it is unlikely that lifting frequency is simply associated to the kinematics of individual lifts (which influence loading)[43], however this has not been evaluated.

The vast number of materials handling investigations that have examined a fixed lifting frequencies is in contrast with other areas of human movement science, such as gait analysis where it is common practice to examine individuals at both self-selected and fixed-paces[109]. In fact, during ambulation, a significant effect of BMI on self-selected movement pace has been shown, where high BMI individuals ambulate at slower speeds, perhaps as an attempt to minimize the effect of ground reaction forces on the lower extremity[179]. During materials handling, only strength-dependent differences in lifting pace have been shown, with stronger individuals lifting heavier loads in fewer repetitions[180]. Although high BMI individuals adopt different movement strategies during certain self-paced tasks (e.g. ambulation[109]) compared to

their normal BMI counterparts, the possibility of similar effects during manual handling has not been investigated.

Cue type

A second laboratory-related influence on lifting that has not been explored is the method used to prompt (or cue) the initiation of the lift. Three main types of prompts are used during fixed-paced tasks, consisting of tone, verbal and motion cues. In the majority of studies participants are prompted to lift by an audible tone generated by a metronome[37,175] or computer[156]. In contrast, during vocational lifting tasks, prompts are provided either by verbal command (e.g. a coworker) or the physical arrival of the object to be lifted (a 'motion' cue). Arrival of an object to cue initiation of the lift has only been used in a small number of lab-based studies[37]. Although it has received little attention, evidence that cue-type may influence lifting kinematics exists. For instance, when individuals are able to observe a second person moving an object (as might of occur during two-person lifts), cues regarding the characteristics of the load are obtained from the kinematics of the observed motion as well as the state of an individual's muscles[181]. However, the effects of these cues on the resultant movements executed by the observer have not been demonstrated (i.e. does an individual adjust the subsequent lift based upon perceptions of the motion executed by another person?). An examination of the effects of tone, verbal and motion cues on lifting strategy may help support the ecological validity of lifting tasks performed in the laboratory setting, by providing an estimation of the magnitude (or lack) of the effect on lifting kinematics. Should an effect of cue type be apparent, this may have implications for the design of workplace lifting tasks that occur at fixed lifting frequencies.

Summary and Objectives

Constraining certain aspects of lifting tasks is a necessary approach for isolating the effect of specific independent variables and to control extraneous sources of variability. However, it is possible these approaches cause participants to move in a manner that is not reflective of that which would be used use during everyday tasks[50]. One of the most commonly-encountered laboratory constraints is the imposition of a fixed lifting pace or frequency. Although it is likely that kinematics do not vary proportional to overall lifting frequency[37], no description of how a fixed lifting frequency might alter kinematics relative to those used during 'preferred' or self-paced tasks has been performed. This has relevance for between-group comparisons derived from fixed-pace lifting tasks (e.g high vs low BMI), particularly if the kinematics are not representative of an individual's 'natural lifting strategy'[50]. Additionally, a fixed lifting pace must be controlled by an external prompt, yet no study has examined differences between existing cue types. An investigation of these factors, as well as a comparison in self-paced lifting between high and normal BMI individuals, will strengthen the ability to generalize findings of fixed-pace lifting tasks by establishing the extent these factors (may) influence lifting kinematics. External factors, such as prompt-effects that might alter low back loading beyond the characteristics of the external load or body segments, are important to understand for injury prevention interventions that incorporate such factors to alter lifting behavior.

The study entitled, *The effect of lifting frequency, cue type and BMI on preferred lifting strategy*, has 3 objectives, to determine the effects of:

1) lifting frequency (fixed-pace) on kinematics,

2) BMI on kinematics during fixed- and self-paced lifting, and

3) cue type (verbal, tone, motion) on kinematics during externally-paced lifting.

ACTIVATION OF LUMBAR ERECTOR SPINAE DURING REPETITIVE MOTION

Characterization of muscle activation during materials handling tasks is a complementary, and in fact essential, approach to investigations of kinematics. On a simple level kinematics are the result of muscle activation, however each joint in surrounded by multiple muscles, leaving numerous possibilities for the specific combination of individual muscle activation to produce a given motion[182]. For instance, increased recruitment of antagonists, such as that which occurs during fatiguing lumbar spine contractions[183], would necessitate greater agonist activation to produce the same torque, an alteration that would also impact compressive load (increase). Similarly, the relative contributions of agonist-synergist muscles around certain joints may differ depending upon contraction type[184]. Advanced biomechanical models of lumbar spine loading incorporate patterns of muscle activation in order to enhance accuracy of prediction of lumbar loading during fatiguing conditions[11], revealing that increases in lumbar spine load do not accompany the elevated activation of agonist and antagonist muscles during fatiguing conditions. Further, the relative contributions of muscles may change during repetitive lifting tasks[57], suggesting that injury mechanisms during repetitive lifting tasks may be due to factors other than cumulative loading effects. In an effort to elucidate the mechanism underlying injury risk during repetitive lifting, muscle activation during fatiguing tasks has received considerable attention[58,174,185].

Electromyography

The majority of studies examining the effect of fatigue on lumbar muscles have utilized the Beiring-Sorensen test (BS), a task requiring the trunk be held horizontal to the ground, in a prone position[49], involving isometric contraction of the lumbar (and hip) extensor muscles[58,59,186]. The BS task is considered a submaximal task for most individuals, based upon estimates of lumbar extension strength (~400 Nm) compared to (average) moment of upper body limb segments (~160 Nm)[58]. During a BS task performed to volitional fatigue, lumbar extensor muscle

activation increases through the first 40 – 50% of the task[56,187], consistent with a submaximal fatiguing task (i.e. increase in MU recruitment and or firing rate[185]). Additionally, the activation of antagonist and synergist muscles may also increase throughout the task[183], concomitant with the decrement in torque production ability of the agonist erector spinae (ES) muscles.

Perhaps the most well-established (and relevant) characteristic of muscle activation during the BS task is the decrement in frequency domain of the electromyogram[58]. Median frequency changes are considered better indicators of back extensor function (endurance) than time to failure, due to the independence from motivational factors[15]. Although alterations to both time to failure and median frequency changes are associated with back pain[15,49], median frequency changes are more strongly related to low back pain compared to endurance time[59,188]. However, task failure cannot always be attributable to a single variable[55], and studies examining the association between median frequency and endurance time have produced variable results. For instance, the decrement in median frequency of the thoracic erector spinae[59], multifidus/semitendinosis[189], lumbar portion of ES[187], and most fatigued muscle[58] have all been identified as the best predictor of endurance time during the BS task. No single factor has emerged to explain the diversity of findings, with differences in relative loading (due to body weight), methodology, and position of electrodes offered as explanations[59,189]. However, the overall association of median frequency changes during the BS task (and endurance time) with back pathology remain consistent[15,49]. In spite of the differences between muscles, BS task remains a useful method for identifying pathology-related alterations in muscle activation[15] and perhaps revealing factors underlying injury mechanisms[187].

However, one limitation of the BS task is its lack of specificity to daily lifting (and therefore external validity) – it involves a purely static loading scenario, negligible trunk motion and isometric muscle contractions. In contrast, the majority of materials handling tasks involve dynamic loading factors[131], considerable trunk motion, and are controlled by shortening

(concentric) and lengthening (eccentric) muscle contractions[11]. It is well established that contraction type can have a substantial influence on muscle activation, both within a single muscle[190] and between agonist-synergist muscle groups[184,190]. Median frequency changes of erector spinae muscles were shown to be much more variable during a fatiguing stoop-lifting task compared to an isometric task, a phenomenon that was hypothesized to reflect a fatigue-attenuation strategy of load-sharing amongst agonist-synergist muscles[11]. It is unlikely the patterns of muscle activation during the BS task reflect those of materials handling tasks, which is also consistent with the task-dependent nature of muscle activation and fatigue-failure[55]. Although this may not diminish the predictive ability of the BS task, it also does not exclude the possibility that an individual may demonstrate alterations in muscle activation that are not apparent during isometric tasks – or display normal activation during the BS task, but altered muscle activation during actual lifting tasks (i.e. the BS is unlikely 100% sensitive to low back pain or related dysfunction).

An alternative task, one that is more specific to lifting and that would isolate the effect of contraction type, is the roman-chair (RC) exercise[191]. While it is also possible to investigate activation patterns during fatiguing conditions with dynamometry[192], the RC exercise is simple to administer and relatively reliable, similar to the BS[59]. Additionally, the RC task involves a similar position (trunk horizontal) and loading (weight of trunk) as the BS task[191], which facilitates comparisons between the tasks (e.g. to consider the effect of contraction type only, rather than position or other lifting-related factors). Although loading based upon trunk weight (present in both RC and BS) will result in some variation between individuals[58], it is reflective of the loading experienced during lifting. In contrast to the BS task, during the RC exercise increases to load (additional weight)[193,194] or the onset of fatigue[193] have a smaller effect on the activation of back extensor muscles, instead manifesting as changes to the hip extensor muscles[193,194]. The primary explanation for these differences is that the hips/pelvis are free to rotate during the RC task[193],

enabling torque contributions from the hip extensors. In fact, even though the RC exercise involves movement, the majority of motion occurs at the hip joint[195], which may lead to relatively little low back motion (and muscle contractions closer to isometric, rather than concentric or eccentric). This is supported by findings of no change to back extensor activation following a fatiguing RC exercise[193], which has led some to conclude the RC exercise is a poor task to train (or perhaps study) the back extensor muscles[191]. A logical change is to execute the RC task in an identical position as the BS exercise, with straps over hips and calf (rather than just the hips) and the pelvis restrained against a plinth, instead of a padded bar. These alterations should shift the emphasis from hip extensors to lumbar extensors, by minimizing the motion of the pelvis – a change that could easily be confirmed by evaluation of the time and frequency domain changes of muscle activation throughout the task. These modifications will improve the specificity of the RC exercise to lifting motions performed as part of activities of daily living or job tasks, which involve concentric and eccentric contractions of back extensors.

Erector Spinae

The erector spinae muscle group contribute the majority of extension torque during both the BS and RC tasks, producing substantially more force (up to 10-fold) than surrounding musculature (obliques, rectus abdominus, lats)[192]. Combined with an easily accessible location for surface recording techniques, this has made the ES among the most frequently examined of all back muscles. The ES can be partitioned into four parts based upon medial-lateral and superior-inferior divisions[196]. The more medial 'longissimus' portion undergoes greater changes in activation than the more lateral 'iliocostalis' during fatiguing contractions, and thus is considered to play a more prominent role in back extension torque production[187]. The erector spinae can also be divided into the more cephalad *pars thoracis* (muscle bellies at the levels of T3 – T12), and the more caudal *pars lumborum* at L1 to L5[196]. The lumborum-thoracis distinction has received much more attention than the longissimus-iliocostalis, with considerable mechanical and physiological

differences identified between the muscles[188,197]. For instance, although both are composed predominantly of type 1 fibres, the proportion of slow twitch fibres is greater in pars thoracis[197] – a disparity often used to explain fatigue-related differences between muscles[58,187]. The most notable mechanical difference concerns the distal attachment of pars thoracis – inserting on the posterior iliac crest and thoracolumbar fascia via a tendinous attachment passing dorsal to pars lumborum[196]. This provides pars thoracis with a large moment arm (>7 cm) about the lumbar spine, indicating a capacity to provide extension torques about the lumbar spine, in spite of its location in the thoracic region. In contrast, pars lumborum fibres tend to be aligned at a more oblique direction relative to the longitudinal axis of the spine[198], indicating a potential role in resisting shear forces on the lumbar vertebrae[199]. Pars lumborum and thoracis may both contribute to extension of the lumbar spine, yet differ in architecture, fibre type and attachment points[196,197]. It is unclear whether these differences may influence the respective activation during the RC task.

Mechanomyography

A complementary approach to electromyography is to consider the signal generated by the mechanical events associated with muscle contraction, or mechanomyogram[200](aka sound myogram, acoustic myography, muscle sound)[201].This mechanical signal manifests from three phenomena: a) gross lateral fluctuations of the muscle; b) oscillations at a muscle's resonant frequency; c) dimensional changes of contracting muscle fibres[200,202]. Mechanomyograms can be acquired using accelerometers[200], microphones, or laser distance sensors, which produce similar signals related to the vibration of the muscle[203]. The majority of the MMG signal is considered to lie between 10 and 100 Hz[201], in contrast to the EMG signal where the majority of bandwidth is above 100 Hz[154]. Similar to EMG, the MMG is frequently examined in both the time and

frequency domains[200], which have received increasing attention in recent years, with over 450 published studies in the past 5 years[2].

Motor unit activation strategy and MMG

A principle factor for the resurgence in MMG interest is its (potential) ability to reveal alterations in the mechanical response to muscle activation that are not apparent in (surface) EMG recordings (electrical response). Some of the most convincing examples of dissociation between EMG and MMG signals are from studies of activation patterns during isometric contractions at varying torque outputs. For instance, Orizio[204] was among the first to clearly demonstrate that the time domain (magnitude) changes in MMG signal related to isometric torque generation differ from the EMG signal. Specifically, MMG magnitude tends to increase up to ~80% of peak torque and then decreases to 100% MVC. In contrast, after a gradual increase to 80% MVC, the median frequency of the MMG increases at a much greater rate up to 100% MVC. The most popular explanation of these changes is that the MMG amplitude reflects motor unit recruitment – as recruitment of larger (and more superficial)[200] motor units would tend to generate larger dimensional changes, consistent with the increase through 80% MVC above[205]. The MMG median frequency, which is typically 2 – 3 times lower than that of the EMG[201], is thought to reflect firing rate – more specifically, an indicator of the aggregate firing rate of all motor units, rather than an absolute measure of firing rate[206]. At particularly high firing rates, it is suggested the amplitude of the MMG signal may be attenuated due to a near tetanus of motor unit firing rate/mechanical oscillations[205].

Findings of similar MMG changes in a variety of muscles[205,207,208], but with time- and frequency-domain changes related to muscle-specific physiological properties, have also provided support for the popular interpretation of MMG magnitude (recruitment) and frequency (firing rate). For instance, in the 1st dorsal interossei, a muscle with a small cross sectional area and

[2] PubMed query with terms "mechanomyogram OR acoustic myography OR mechanomyography", May 8 2013.

motor units comprised of fewer muscle fibres, the decrement in MMG amplitude occurs earlier and median frequency increase is more prominent, changes consistent with an increased reliance on motor unit firing rate to modulate force[208]. In contrast, in the quadriceps muscle group, MMG amplitude tends to increase up to 100% MVC, with comparatively less change in median frequency – consistent with increased recruitment of larger motor units[207]. Perhaps the strongest evidence that MMG reflects specific features of motor unit activation strategy are the findings by Yoshitake[209], demonstrating the MMG frequency changes are related to the rate of electrical stimulation applied to a muscle. In contrast to MMG changes during non-fatigued conditions, numerous authors have demonstrated a decline in MMG amplitude and frequency during fatiguing conditions[202]. In addition to de-recruitment of high-threshold motor units, these changes are often attributed to the increase in twitch duration and reduction in motor unit firing rate during submaximal fatigue tasks[204,210] (i.e. 'muscle wisdom). Lastly, the relative synchronization of motor unit depolarization/ contraction, may also result in an increase in MMG amplitude, with an unclear (or no) effect on the median frequency[206]. Although it is clear that the MMG and EMG represent different (independent) phenomena associated with muscle activation[202,206], with the exception of electrical stimulation studies[209], evidence the MMG reflects specific aspects of motor unit activation strategy is based on the association of known or probable activation strategy changes to specific features of the MMG signal[200].

Non-physiological factors and MMG

Numerous authors have acknowledged that 'non-physiological' influences, and not necessarily related to activation, may also influence the MMG signal[201,206,211](Table 2). The most obvious is the initial gross lateral fluctuation of a muscle as it changes from a relaxed (or elongated) state to a contracted (or shortened) one, however this large dimensional change occurs at relatively low frequencies (< 1 Hz) and is easily excluded via digital filtering techniques[206]. The most commonly-debated factor is muscle compliance (the inverse of stiffness), which is

thought to decrease (stiffness increase) during a sustained exertion, particularly one involving an isometric contractions[211] . Multiple factors have been identified as mechanisms of compliance/stiffness changes, including fluid accumulation, muscle thickness, and intramuscular pressure[202,206,211]. In an isolated material, a decrease in compliance/increase in stiffness would attenuate oscillations arising from a given applied force. In the case of an individual muscle, this would result in a decrease in MMG amplitude (Table 2), a change that has been shown to occur during submaximal fatigue involving concentric and isometric contractions[201,202]. However, the influence of muscle compliance may vary between tasks involving different contraction types, with the effect greatest during isometric contractions[212]. In fact, Sogaard[211] demonstrated comparatively little effect of intramuscular pressure on MMG amplitude during muscle contractions where blood flow was occluded. Although it is widely acknowledged as an influence on the MMG signal[206], the extent (and variation) of compliance-related changes to the MMG signal is not well known. Lastly, interstitial fluid turbulence has also been postulated to alter the MMG signal, however this possibility has been restricted to high-velocity motions (300 deg/s)[210]. Further, the MMG amplitude during passive limb motions (cycling) is negligible[213] indicating the influence of these passive movement artifacts is likely minimal.

Table 2. Physiological and non-physiological influences on the mechanomyogram (MMG) signal. The (proposed) effect on the amplitude and frequency domain of the MMG is indicated, with decreases (↓) left-aligned, increases (↑) right-aligned and no change(--) and unknown (?) effects aligned to centre in the respective columns (see legend).

Category	Factor	effect on MMG signal	
		amplitude	frequency
Physiologicial - activation strategy[206,209]	Firing rate, increase	↓	↑
	Recruitment, increase	↑	--
Physiological – other[200,202,206]	Wisdom – firing rate, decrease	↓	↓ ,--
	Wisdom – twitch duration, increase	↓	--
	Synchronization, increase	↑	?
Non-physiological [205,211,214]	Compliance – fluid accumulation, increase	↑	?
	Compliance – muscle thickness, increase	↑	?
	Compliance – pressure, increase	↑	?
	Interstitial fluid turbulence, increase	↑	--
	Gross lateral dimensional changes, increase	↑	--

↑ = increase; ↓ = decrease; -- = no effect; ? = unknown effect

Although direct evidence supporting an association between the MMG and specific aspects of motor unit activation is absent, the influence of a number of non-physiological factors have been excluded (movement artifact[213]) or considered secondary determinants (stiffness[206,211]). The association of MMG amplitude and frequency domain changes to predicted motor unit activation strategies (during isometric contractions[201,205]), and numerous reports of a differential change in MMG and EMG signals[202,207,212], indicate the MMG signal may reflect aspects of muscle activation not apparent from surface EMG. Although a number of authors have advocated for concurrent measurement of EMG and MMG, few investigations have reported changes to both signals within a single task[56,207].

Task-specific changes to MMG

Numerous studies have investigated MMG signal changes during non-fatiguing contractions, across varying intensities, velocities and contraction types[200,201,207]. Briefly, the MMG signal tends to vary proportional to torque production during both concentric and eccentric contractions[200], with lower amplitudes during eccentric contractions. In contrast, far fewer studies have considered fatiguing (Table 3). Of the small number of investigations of fatigue-related MMG changes, the majority have considered only isometric conditions during maximal[213,215] or submaximal tasks[56,201,216,217]. Similar to EMG, a decrease in MMG magnitude is apparent during maximal fatiguing tasks, attributed to de-recruitment of high threshold motor units[202,206]. As expected, during submaximal fatiguing tasks, the MMG signal varies depending upon the intensity of the contraction. For instance, nearly all studies in peripheral muscles groups examining submaximal, isometric tasks (< 50% MVC) report an increase in MMG amplitude over the course of a fatiguing trial[56,201,216], however the rate and duration of the increase varies dependent upon initial load. At lower intensities/loads, the increase in amplitude is more gradual and occurs later in the trial, consistent with increasing recruitment of motor units, while at higher intensities, the increase is rapid and may even plateau or decrease prior to termination of the

task[201]. As mentioned, a plateau in MMG amplitude may reflect a shift towards increased firing rate or a fatigue-related contraction change ('wisdom'), however few studies have reported both frequency and amplitude changes which might be used to distinguish these possibilities. Studies reporting only frequency domain changes have reported either no change or a decrease over the course of fatigue, which is speculated to reflect the decline in firing rate associated with the onset of fatigue[206].

Only a small number of studies have MMG signal changes during fatiguing concentric (4 studies) or eccentric (1 study) conditions, and 4 of 5 have involved maximal contractions (Table 3). Regardless of intensity, most have reported a decline in MMG amplitude, with de-recruitment or muscle wisdom[202,212] offered as explanations for the change during maximal and submaximal conditions, respectively. As is evident in Table 3, only a single study[207] has reported both time and frequency domain changes of the MMG during fatiguing contractions involving concentric or eccentric contractions. In light of the possibility that both derecrutiment of motor units and an increase in firing rate may result in a decrement to MMG amplitude, but have (potentially) differential effects on frequency, interpretation of MMG signal changes is limited without both time and frequency domains (and complementary EMG).

In contrast to peripheral muscle groups, only 3 studies have considered MMG signal of erector spinae muscles during fatigue (shaded, Table 3), with all considering the isometric/static conditions of the BS task. Unlike peripheral muscle groups, MMG changes reported were much more variable, with two authors reporting an increase[56,216] (but at different rates), and one reporting no change[217]. No convincing arguments for the discrepancy between studies have been offered, with effects such as sensor location or inter-individual variation in loading (due to trunk weight)[216] unlikely to be large enough to explain the dramatic differences between studies. Similar to peripheral muscle groups, no study has considered the MMG signal of erector spinae muscles during a fatiguing, submaximal task involving concentric and eccentric contractions.

Table 3. Summary of MMG signal changes during submaximal fatigue, organized by contraction type and sorted in descending order by intensity. The indicators of amplitude and frequency changes (↑, ↓, --) are aligned to reflect any non-linear changes, which is also indicated with suffixes (40, linear, etc)(see legend). Erector spinae studies are highlighted.

			MMG signal changes	
contraction type	intensity (%mvc)	muscle group	amplitude	frequency
Isometric	100	elbow flexors[215]	↓, linear	
Isometric	100	knee extensors[213]	↓60, --end	↓40, --end
Isometric	60	elbow flexors[201]	--	--
Isometric	40	elbow flexors[201]	↑40, --end	--
Isometric	20	elbow flexors[201]	↑, linear	--
Isometric	25	elbow flexors[201]	↑, linear	↓, linear
Isometric	~45 (BW)	erector spinae[56]	↑40, ↓end	--
Isometric	~45 (BW)	erector spinae[216]	--50, ↑end	↓, linear
Isometric	~45 (BW)	erector spinae[217]	--	
concentric	100	elbow flexors[200]		↓, quadratic
concentric	100	knee extensors[202]	↓, linear	
concentric	100	knee extensors[207]	↑30, ↓end	↓, linear
concentric	95 (MW)	knee extensors[210]	--	
concentric	80 (MW)	knee extensors[210]	--	
concentric	65 (MW)	knee extensors[210]	↓, linear	
concentric	50 (MW)	knee extensors[210]	↓, linear	
eccentric	100	knee extensors[212]	↓, linear	

↓/↑xx = increase/decrease to xx% of trial duration; -- = no change
BW = body weight; MW = % max power output, maximum wattage (cycling)

Although an MMG investigation of erector spinae will contribute to the body of knowledge on mechanomyogram signal changes and methodology, the overall aim of MMG-based studies is to further the understanding of task-specific changes in muscle activation. The strength of MMG-based techniques is the ability to identify change in activation not apparent from surface-based EMG[200]. Alterations to muscle activation have received considerable attention as a mechanism of back injury, underlying a possible role of transverse abdominus dysfunction in back injury[52], or more recently (and better supported), the concept of a motor control error[17]. An error in muscle activation (failure in appropriate timing or magnitude) could lead to an alteration in load distribution or loss of stability in the lumbar spine, which might explain how back injury arises during a seemingly 'random' movement (i.e. a single repetition of a highly practiced motion or one involving low external loads)[17,19]. However, alterations may not be restricted or manifest only in the temporal and spatial patterns of muscle activation detectable by

EMG. For instance, deviations in how motor units are activated within a single muscle (e.g. synchronization, firing rate) or dissociations between mechanical (MMG) and electrical (EMG) aspects of muscle contraction might also be indicative of dysfunctional activation. Only a single study has considered MMG as a prognostic or predictive tool for patients with low back pain[218], which established the reliability of MMG amplitude changes (i.e. coefficient of variation) but reported only limited task-specific effects (mean values). Establishing MMG-signal changes during dynamic conditions (concentric/eccentric) will add to the understanding of paraspinal muscle function during fatiguing conditions and establish normative responses useful for future studies of symptomatic individuals.

Summary and Objectives

An understanding of muscle activation, particularly during fatiguing conditions and across repetitions, has important implications for back injury mechanisms[187]. Although the ES has received considerable attention, studies have been largely restricted to EMG-based approaches, which leave a large proportion of the variation in injury risk and muscle activation unaccounted[15]. Changes in the mechanical events associated with muscle contraction (MMG) have been shown to vary independent of electrical events (EMG), and may reveal aspects of motor unit activation that cannot be detected using conventional surface-EMG[200]. However, only a limited number of studies have examined MMG-related changes in erector spinae muscles during fatiguing conditions[56,217], and none have considered tasks involving concentric and eccentric muscle actions similar to those encountered in actual lifting tasks. An EMG- and MMG-based examination of erector spinae muscle during a standardized task will contribute to mechanomyogram methodology and the understanding of muscle function during materials handling tasks.

The study entitled, *The activation of erector spinae during repetitive trunk motion*, has 2 objectives:

1) describe the activation changes of erector spinae during submaximal fatigue using electrical- (EMG) and mechanical- (MMG) based techniques, and

2) compare activation of erector spinae during concentric and eccentric phases of a repetitive trunk motion.

STUDY 1. EFFECT OF OBESITY ON BODY SEGMENT PARAMETERS AND LUMBAR SPINE LOAD

METHODS

1. PARTICIPANTS

A sample of 8 male participants, 5 with a BMI > 30 kg/m^2 and 3 with a normal BMI (< 25 kg/m^2), was obtained. The mean (SD) age was 29.9 (7.2) years (range: 23 – 44). A ninth 'virtual' subject was also constructed (subject #9, normal BMI group)[3]. Each high BMI participant was matched to a normal BMI individual based upon height, providing 5 high-normal BMI pairs. Anthropometric measurements were obtained with a caliper anthropometer (60 cm, Lafayette Instrument Company, IN, USA) and steel tape. Body weight was measured on a digital weigh scale (BWB-800S, Tanita Corp, Ill, USA). Ethical approval was obtained from Bannatyne Campus Research Ethics Board, University of Manitoba (HREB#: H2010:408).

2. PROCEDURE

A stepwise overview of the procedure is provided in Table 4.

2.1. Image Acquisition

Low and high angle digital images (30 images, 2592 x 3456 pixels, Casio Exilim EX-100, Casio USA) were acquired simultaneously (1 fps) for each participant. Two cameras were mounted at a distance of 325 cm from a moveable backdrop at heights of 82.5 and 152.5 cm (Figure 1, c). Participants were positioned midway between backdrop and cameras. The moveable backdrop provided a high-contrast background for image processing and consistent camera

[3]The 3D topography of subject 9 was scale-adjusted to the correct height (1.70m); estimates of body segment parameters were derived based upon the segment lengths of two participants in the high BMI group of similar height (mean of subjects 4 and 5).

orientation (Figure 1, a and b). Correction for lens distortion was provided by the 3D model-generation software (Strata Foto 3D CX, Santa Clara, Utah, US.).

Table 4. Overview of protocol and methodology for acquisition of individual-specific body segment parameter estimates, evaluation of variance (relative to predictive models) and computation of back loading.

Procedure	Description	Outcome
Image Acquisition	Digital images obtained from high + low points of view	2 x 30 digital images (.jpg), 9 megapixels.
Image Processing	Delineation of subject from background	2 x 28 images (.tiff), subject outline stored as an alpha channel mask
3D Wireframe	Generation of 3D wireframe	3D topography represented by polygon mesh (20,000 polygons)
Virtual dissection	Partitioning of whole body into segments using 3D modeling software	11 segment model: head + neck, upper arm, forearm, hand, thigh, shank, foot, whole trunk and upper-, middle- & lower-trunk segments
Estimation of body segment parameters	Inertial properties derived via volumetric integration	Mass, density, centre of mass location, moments of inertia for each segment
Comparison to existing models	Relative difference between current and historical models	Variation (%) in BSIPs relative to 4 existing models
Application to lumbar spine loading	Use of existing model to estimate back load during material handling tasks	Static/dynamic torques + tangential/normal components of force acting about L5/S1.

Subjects stood in a relaxed standing position with feet 35 cm apart and arms abducted ~30 degrees in the frontal plane. This position minimized tissue approximation between upper arms, trunk and lower extremities. Subjects stood on a 122 x 122 cm calibration mat (Figure 1, c) used by the software to determine camera position, subject orientation, scaling, and primary face (front) for texture mapping. Joint centres were determined by visual inspection and palpation, with markers affixed indicating the proximal and distal joint centres (see below).

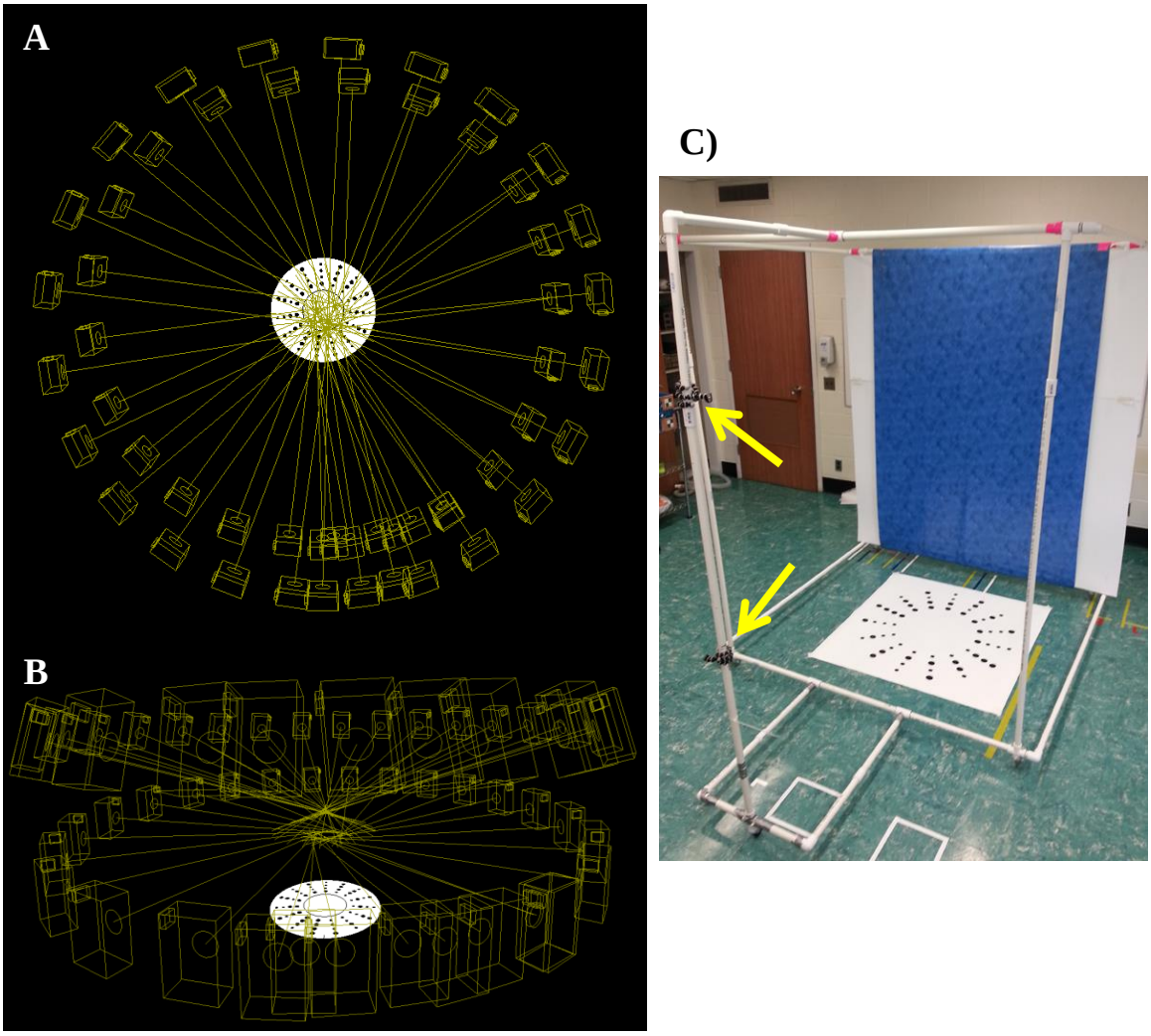


Figure 1. Camera position during data collection:A) top and B) front views of camera position (yellow) relative to calibration mat (white circle) in 3D environment; C) backdrop and point of attachment of cameras (yellow arrows).

2.2. Image Processing and 3D wireframe

Images (.JPEG) were imported to photo editing software (Adobe Photoshop CS5, Adobe USA). An image mask delineating the subject from the image background was created using the 'quick select' tool and refined manually. Masks were saved to the alpha channel (transparency layer) of each image and exported in a .TIFF file format. The original .JPEG and masked .TIFF images were imported to 3D model-generation software (StrataFoto CX, Santa Clara, Utah US). A 3D polygon model was constructed by the software, using the subject outline (masked .TIFF files) and computed camera position (derived from calibration mat). Post-processing was

performed to specify the number of polygons in the model (20,000) and further refine object boundaries using proprietary algorithms (StrataFoto CX). Post-processing settings were determined iteratively by visual inspection of generated models (Appendix: *StrataFoto CX Settings*).

A texture map was generated for each participant's polygon model, a process that 'mapped' the image data to the faces of 3D polygon model, effectively 'wrapping' the 2D digital images to the 3D polygon model, allowing visualization of anatomical features. This provided a method of locating markers and anatomical features for determination of joint centres and segmentation planes. Relevant anatomical landmarks were determined via palpation by a trained health care practitioner (Athletic Therapist), with markers affixed to the skin for landmarks not easily determined by visual reference from the texture map (e.g. a joint line on certain high BMI individuals). The joint centres were estimated on the 3D models by intersection of the medial-lateral and anterior-posterior landmarks. The completed wireframe and texture map was exported as a Virtual Reality Modeling Language file (.WRL), a text formatted file which specifies the coordinates of vertices and faces of the 3D polygonal model with an embedded texture map.

2.3. Virtual dissection

Models were imported into 3D modeling software (Blender 2.63a, Blender Foundation, Amsterdam Netherlands) for segregation of individual body segments ('virtual dissection'). The whole body wireframe was 'dissected' into 11 individual body segments using the Boolean intercept tool: a geometric primitive (usually a cube) was created in the 3D environment and positioned over the segment of interest, with the superior and inferior faces aligned with the proximal and distal ends of the segment (i.e. the segmentation planes). Execution of the Boolean intercept tool created a wireframe of the common volume between the body segment and

primitive, effectively ‘dissecting’ a body segment (see Figure 2). This approach was advantageous to splitting the model at edges/vertices, providing a more precise segmentation plane (not constrained to existing vertices) and returning segments that were ‘water-tight’ (required for computation of inertial properties). The body was segmented consistent with comparative models, described below.

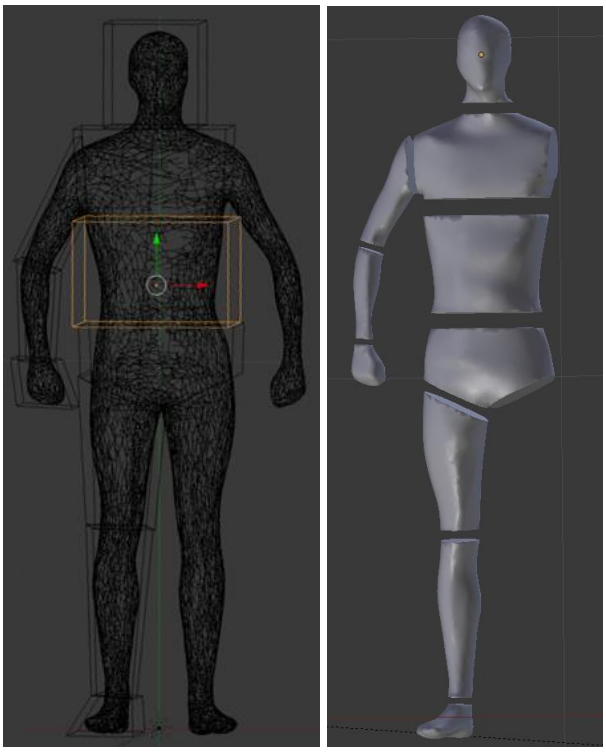


Figure 2. Segmentation planes and geometric primitives used for 'virtual dissection'. Highlighted (orange) is the primitive used for segregation of middle trunk segment. Shown is wireframe prior to (left) and post (right) segmentation.

Segments were translated and rotated such that the proximal joint centre was positioned at the origin (0,0), with the Z-axis extending longitudinally to the distal joint centre. The y-axis was orthogonal and in the anterior-posterior direction and x-axis extended medial-lateral, defining a consistent local coordinate system for each limb segment. Segments (and whole body) wireframes were exported as an open-source, text-based object modeling file format (.OBJ).

2.4. Estimation of body segment parameters

Whole body volume was calculated using a direct integration method, returning an exact volume for the provided 3D polygon mesh[219]. Inertial properties (volume, mass, centre of mass location and moments of inertia) of body segments were derived from the same algorithm[219]. Segment densities were assumed to be uniform[122] and were derived from previously reported values: the head, arms, legs and hands from cadaver-based estimates[26,129,220] and the trunk segments from MRI estimates [25]. Densities were scale-adjusted such that the sum of individual segment masses was equal to the total body mass. Whole body density was estimated from total body volume and body mass, providing a means of estimating total body fat percentage[221].

2.5. Estimation of Error

Models of two objects, a rectangular cuboid (58 x 27 x 16 cm) and a sphere (37 cm diameter), were constructed using the methods described above. Error was evaluated by comparing measured height (z-axis), width (y-axis) and depth (x-axis) of each object to those derived from the 3D models. Texture map error was estimated from incremental gradations (10 cm) on each face of the box surface.

The effect of potential movement artifacts during image capture was estimated for one participant (height 1.89 m, mass 80 kg), by comparing actual anthropometric dimensions (segment length, breadth) to those derived from the 3D model. The absolute value of each difference (3D dimension - actual) was computed.

3. COMPARATIVE MODELS

Three comparative models of body segment inertial parameters (BSIPs) were chosen based upon similar segmentation methods (for ease of comparison) and representing typical methodological approaches in the literature (Table 5). The first utilized medical imaging (gamma

mass scanning) for a population of young adults, with predictive equations of BSIPs based upon a) height and body mass[222], and b) body mass and segment lengths[118]. In the second, segments were represented as geometric primitives proportioned to basic anthropometric dimensions (e.g. height, breadth, depth)[119], with BSIPs derived using mathematical formulae for inertial properties of geometric solids and estimates of mass distribution amongst body segments[129]. The third model estimated trunk parameters from MRI images on a sample of individuals with a wider range of BMIs than previous investigations, with BSIPs modeled as a function of height and weight[25].

Table 5. Description of segmentation planes and corresponding BSIP models.

Segment	Proximal and distal endpoints	Models
Whole trunk	suprasternal notch – distal pelvis	P,D
Upper trunk (chest)	suprasternal notch – xyphoid	P,D,Z,H
Middle trunk (abdomen)	Xyphoid – umbilicus	P,D,Z,H*
Lower trunk (pelvis)	Umbilicus – distal pelvis	P,D,Z,H*
Thigh	Hip – knee JC	P,D,Z,H
Shank	Knee – ankle JC	P,D,Z,H
Foot	Ankle JC – distal foot	P,D,Z,H
Upper arm	Shoulder – elbow JC	P,D,Z,H
Forearm	Elbow – radiocarpal JC	P,D,Z,H
Hand	Radiocarpal JC – distal hand	P,D,Z,H

P – Pearsall 1994[25]; Z – Zatsiorksy 1983[222]; D – de Leva 1996[118]; H = Hanavan 1964[119]; JC – joint centre
* - custom Hanavan model

A segmentation approach was chosen that enabled comparison across all three models (Table 5). The trunk was partitioned into three segments, an upper (suprasternal notch to xyphoid), middle (xyphoid to navel), and lower segment (navel to hip joint centres), and whole trunk, consistent with three comparative models[118,222,223]. The geometric model[119] shared a similar upper trunk segment, but considered the middle and lower trunk as a single segment. To preserve consistency across models, the lower segment of this model was partitioned into separate middle and lower segment using a similar approach as the original study – the depth and breadth of the

trunk was measured at the appropriate levels (xyphoid, umbilicus, hips) to construct corresponding geometric primitives, and inertial parameters were computed from the original equations[119], with mass distribution derived from the current 3D model. The difference between current photogrammetric model and comparative models was computed, (current – existing)/current, where positive values indicated greater estimates for the current model.

4. MECHANICAL MODEL

Load about L5/S1, represented as the resultant joint moment (RJM) and normal/tangential forces, was estimated using a 5-link model consisting of a torso, head/neck, upper arm (2) and forearm/hand (2) segments, and external load[224]. Model assumptions included rigid body segments, frictionless joints, and negligible muscle co-contraction (of trunk flexors) and intra-abdominal pressure. The lumbar extensors were modeled as a single muscle with a fixed moment arm of 7.0 cm, parallel to the L5/S1 joint centre[196] . The position of the L5/S1 joint centre was estimated at a distance of 33% of trunk depth from the posterior trunk[225]. This provided an estimate of L5 location that scaled with the (probable) accumulation of adipose tissue around the trunk, rather than a fixed distance from the posterior trunk. The loading parameters and model is depicted in Figure 3, with computation described below.

Load was estimated for 3 conditions: 1) quiet standing, 2) load carrying/holding, and 3) the beginning of a stoop-style lift (trunk flexed at 45 degrees). The external mass in the latter conditions was a box of the same dimensions used in subsequent studies (37.5 cm x 36 cm x 25 cm), with a mass of 18.5 kg (the average mass selected by participants in *The influence of body mass on lifting strategy during repetitive, fixed-pace lifting*. Segment inertial parameters were determined from the 3D models described above.

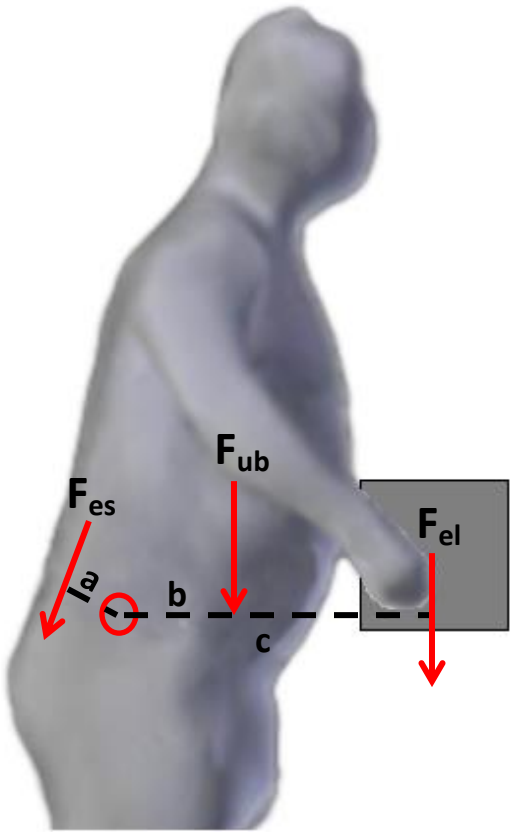


Figure 3. Representation of biomechanical model and parameters for estimation of load about L5/S1 joint (circle). The arrows indicate the forces from the weights of upper body segments (Fub: trunk + upper arm + forearm + head) and external load (Fel: 18.5 kg box) and the force from back extensors (Fes). The relevant moment arms are shown (a: back extensors, b: body segments, c: external load). Resultant joint moment was computed as the extension torque required to resist the flexion torques imparted by external load and limb segment weights (static), as well as accelerate the upper body and external mass (dynamic). Compressive (perpendicular) and shear (tangential) forces where derived from net force acting about L5/S1 (Fes + Fub + Fel).

The resultant joint moment about L5/S1 was estimated using an inverse dynamics approach[133]. Net static torque represented the sum of the moments of weight arising from body segments and external load:

$$T_{L5\ static} = -(M_{seg\ i} \times g) \times d_{seg\ i} - (M_{box} \times g) \times d_{box}$$

Where $T_{L5\ static}$ is the static torque about L5/S1 for an individual segment, $M_{seg\ i}$ is the mass of the *i*-th segment, g the gravitational acceleration, $d_{seg\ i}$ the moment arm of the *i*-th segment, and M_{box} and d_{box}, the mass and moment arm of the external load, respectively.

Dynamic, or inertial torque, was that required to accelerate the body segments and external load:

$$T_{L5\ dynamic} = -(I_{seg\ i} \times \alpha_{seg\ i}) - (I_{box} \times \alpha_{box})$$

with the same conventions as above and 'I' representing moment of inertia and α, angular acceleration.

Components of net force included the weights of body segments ($F_{w\ seg}$) and external load ($F_{w\ box}$), as well as the erector spinae muscle force (F_{ES}) required to resist external flexion moments:

$$F_{L5} = -F_{ES} - F_{w\ seg\ i} - F_{w\ box}$$

The normal and tangential components of the net force were derived based upon a 30 degree inclination of the L5/S1 joint relative to horizontal[144].

Lastly, two models were developed for each condition. In the first, the positions of limb segments and box were derived from actual position (*The influence of body mass on lifting strategy during repetitive, fixed-pace lifting*) and kinematic data (angular acceleration[23]) specific to normal and high BMI subjects. A second 'conservative' model incorporated limb orientations specific to body type, but positioned the external load as close to the body as measured trunk dimensions would allow (i.e. minimized the moment arm of external load) and assumed similar trunk angular acceleration (i.e. eliminated the effect of movement strategy based upon body type).

5. STATISTICAL ANALYSIS

The effect of increased body mass on segment inertial parameters was evaluated using a matched-pairs design. The difference in BSIPs attributable to elevated BMI was calculated for

each pair of subjects, matched by height (high/normal). This approach was preferred over a comparison of absolute values (i.e. failing to control for height), which would skew the results towards taller and/or heavier participants. The hypothesized increase in BSIPs was tested using a one-sample t-test on the relative difference between high and normal BMI participants (high/norm, $H_0 = 1$). As an alternative to reporting post hoc tests across all models (4), segments (4) and parameters (8), a threshold for the ratio of mean:SD (effect size:variance) for significance was derived (>=1)[4], which is easily evaluated across all parameters and body segments. The effect of increased body mass on lumbar loading parameters was tested with the same approach.

Differences between the BSIPs from the current study and predictive equations were evaluated with an analysis of variance. For high BMI participants, this represents the error in BSIPs attributable to an increased body mass (i.e. beyond the original sample). Omnibus tests were performed using a repeated measures model (MODEL, PARAMETER) with GROUP as a between-subject effect. Both 3-way (all models) and 2-way ANOVAs are reported, the latter evaluating individual models separately. The F-statistic and effect size (n_p^2) for the main effects of PARAMETER, MODEL, and also the GROUP*PARAMETER and GROUP*MODEL*PARAMETER interactions are reported. Post hoc comparisons were performed by group (high and low BMI separately).

Significance was set at $p < .05$. Statistical analysis was carried out using SPSS 19.0 (IBM Corporation, Somers NY).

[4]For a give sample size (n = 5), the equation for a one-sample t-test, t = (x-u)/(s/sqrt(n)), can be reduced to a simple ratio of effect size (mean, x) to variability (standard deviation, s), accomplished factoring out the 'sqrt (n)' term . The corresponding critical value for this one sided test was t = 2.13, which when factored by the 'sqrt(n)' (2.23), leaves a value of near 1. Thus a ratio of mean/SD greater than the factored t-critical value indicates the probability of such an effect by chance is less than the chosen α (.05) - – in other words, as long as the mean is at least as large as the SD, the test is significant (for this sample size and α). The actual value of the factored t is 0.96 (i.e. 2.13/2.23), which indicates the mean can be slightly less than the SD – a mean/SD ratio of 1 corresponds to an α of 0.045. Lastly, the population value (u = 1) must be subtracted prior to the estimation. All tests were confirmed with SPSS v19.

RESULTS

1. ERROR

The mean (SD) resolution was 15.1 (2.1) pixels/cm for the superior-inferior direction (z-axis), 8.64 (2.1) pixels/cm for the medial-lateral direction (x-axis) and 3.2 (1.1) pixels/cm for the anterior-posterior direction (y-axis) [0.6 (0.1), 1.2 (0.3), and 3.1(1.1) mm/pixel, respectively].

Actual (cm) and relative (%) errors of the 3D geometry for inert and human models are presented in Table 6. No systematic under- or over-estimation was detected in model estimates. The texture map had an absolute error of mean (SD) error 0.12 (0.08) cm, corresponding to a relative error of 1.27 (0.93) %.

Table 6. Actual (cm) and relative (%) error in 3D geometry for inert objects and a human subject, absolute values are shown. The x, y, and z directions correspond to the medial-lateral, anterior-posterior, superior – inferior directions, respectively.

	Difference, abs(actual – measured)	
	cm, mean (SD)	% (/100), mean (SD)
Cuboid		
X	0.33 (0.15)	1.24 (0.57)
Y	0.27 (0.06)	1.63 (0.34)
Z	0.15 (0.10)	0.25 (0.17)
total	*0.25 (013)*	*1.05 (0.72)*
Sphere		
radii	0.20 (0.01)	0.52 (0.01)
Human		
X	0.45 (0.27)	1.39 (0.87)
Y	0.59 (0.31)	2.79 (1.49)
Z	0.59 (0.54)	0.61 (0.24)
total	*0.45 (0.24)*	*1.54 (1.05)*

Error magnitude was proportional to the computed resolutions – lowest for the Z-axis (superior-inferior) and greatest for the Y-axis (anterior-posterior). Total error was slightly higher in the human subject (1.54%) compared to either of the inert objects (0.53 and 1.05%).

Movement artifacts are the most likely sources of the additional variance in the human subject, particularly in the anterior-posterior direction (i.e. due to respiration).

The coefficients of variation across multiple measures were 0.42% for the cube (n = 9) and 1.29% for the texture map (n = 13). The bilateral variance in upper and lower limbs was also compared (greater in upper compared to lower model segments, $t(7) = 2.48$, $p < .05$) and is provided along with a case study of convenient limb asymmetry illustrating the sensitivity of the technique (Appendix: *Limb Asymmetry*).

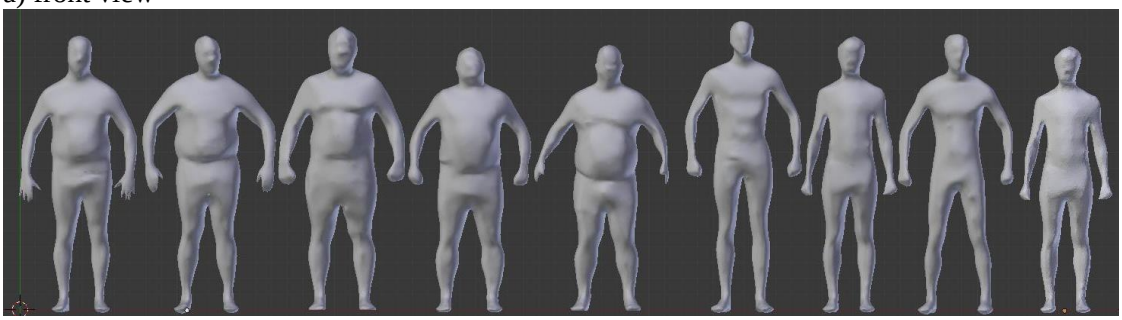
2. INDIVIDUAL-SPECIFIC BODY SEGMENT PARAMETERS

2.1. Anthropometrics

Frontal and sagittal views of participants are shown in Figure 4. Whole body anthropometrics are reported in Table 7, with the pairing of participants for high-normal BMI comparisons indicated in the last column. As expected, differences between groups were substantial, with the exception of height, for which the participants were matched. The high BMI participants were a mean (SD) 1.54 (0.20) times heavier than the normal BMI group, and consistent with an android-like somatotype (Figure 4). The mean (SD) waist circumference and waist:hip ratio of the high BMI participants were 115.1 (10.2) cm and 0.99 (0.06), respectively, while those of the normal BMI participants were 78.2 (3.1) cm and 0.78 (0.04).

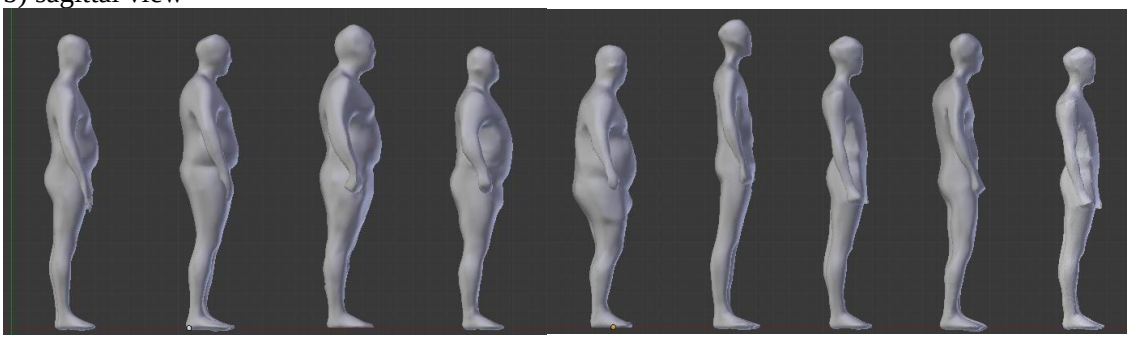
Figure 4. Front and sagittal views of study participants, rendered 3D topography. P = participant number, M = matched pair.

a) front view



P	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
M	*7*	*7*	*8*	*9*	*9*	--	*1,2*	*3*	*4,5*

b) sagittal view



P	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
M	*7*	*7*	*8*	*9*	*9*	--	*1,2*	*3*	*4,5*

Table 7. Whole-body anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All parameters, except height, were significantly different based on relative differences (high/norm), p < .05.

Participant	height (m)	mass (kg)	BMI ($kg*m^2$)	volume (cc)	density (g/cc)	fat mass (%)	density scale (%)**	pair
1	1.76	89.9	29	88,100.1	1.035	27.29	0.987	2
2	1.79	103.8	32.4	104,044.2	1.009	38.37	0.968	2
3	1.85	126.7	37.0	127,419.1	1.004	40.89	0.973	3
4	1.71	108.6	37.1	107,342.8	1.023	32.25	0.979	4
5	1.71	112.7	38.5	113,046.8	1.005	42.13	0.973	4
6	1.91	81.9	22.5	77,056.6	1.080	8.80	1.027	-
7	1.79	72.5	22.6	69,693.0	1.059	17.23	1.007	5,6
8	1.84	72.8	21.5	69,747.7	1.073	11.70	1.019	7
9*	1.71	67.0	22.4	64,600.3	1.057	17.91	1.004	8,9
high, M	*1.76*	*108.34*	*34.82*	*107,990.4*	*1.018*	*35.79*	*0.976*	-
SD	*0.06*	*13.39*	*3.99*	*14,271.3*	*0.012*	*5.83*	*0.007*	
normal, M	*1.81*	*73.73*	*22.42*	*70,274.4*	*1.068*	*13.91*	*1.014*	-
SD	*0.08*	*6.15*	*0.51*	*5125.4*	*0.011*	*4.40*	*0.011*	
high/norm, M	*1.00*	*1.54*	*1.54*	*1.60*	*0.96*	*2.27*	*0.97*	-
*SD****	*0.01*	*0.20*	*0.18*	*0.23*	*0.02*	*0.74*	*0.01*	

*subject 9 is a derived model, with segment lengths taken as average of subjects 4 & 5 and 3D topography scaled from subject 2.
**represents the relative difference from mean values reported in literature, used to scale-adjust segment densities; calculated as (actual / derived body mass), where derived body mass is computed from segment volume and estimated density.
*** As an alternate to reporting results of all possible comparisons, the ratio of mean:SD (i.e. effect size:variance) for a p-value < .05 was computed. In the current sample, a ratio of 1 or greater (i.e. mean > = SD) indicates significance for a one-sided test at p < .05 (or .045 exactly, see methods).
normal = normal BMI group, high = high BMI group, high/norm = relative difference based upon matched pairs.

2.1.1. Mass distribution amongst body segments

The difference in body segment mass (g) and volume (cc) between normal and high BMI participants was not uniform across segments (Table 8). Relative to the difference in total body mass (g, cc; one-sample testing, $H_0 = 1.54$), high BMI participants had more body mass distributed to the whole trunk, middle trunk, and upper arm segments ($p < .05$). Compared to normal BMI participants, the high BMI group had a greater portion of body volume and mass distributed in the trunk segment (excluding the upper trunk), a similar amount in the arm segments, and relatively less in lower extremity, hand and head segments (Table 8, __ $\%_{body}$).

Table 8. Summary of relative differences in segment anthropometry between high and normal BMI participants (high/norm). Mean (SE) is reported and significant differences (p < .05) are indicated (* & ____); 95% confidence intervals can be constructed from the SE and t-critical value (2.78).

Segment	Volume (cc)	Volume ($\%_{body}$)	Volume ($\%_{trunk}$)	Mass (g)	Mass ($\%_{body}$)	Mass ($\%_{trunk}$)	length (cm)	depth (cm)
Whole Trunk, M	1.89*	1.19*	--	1.84*	1.20*	--	1.06*	1.63*
(SE)	(0.11)	(0.03)		(0.11)	(0.02)		(0.01)	(0.08)
Upper Trunk, M	1.74*	1.09	0.92*	1.68*	1.09	0.91*	1.14*	1.33*
(SE)	(0.13)	(0.06)	(0.04)	(0.12)	(0.06)	(0.04)	(0.05)	(0.07)
Middle Trunk, M	2.25*	1.40*	1.18*	2.17*	1.40*	1.17*	1.09	1.63*
(SE)	(0.20)	(0.06)	(0.04)	(0.19)	(0.06)	(0.04)	(0.04)	(0.08)
Lower Trunk, M	1.75*	1.10*	0.93	1.70*	1.11*	0.93	1.00	1.34*
(SE)	(0.09)	(0.04)	(0.04)	(0.08)	(0.04)	(0.04)	(0.04)	(0.04)
Upper Arm, M	1.77*	1.12	--	1.71*	1.13	--	0.95	1.35*
(SE)	(0.08)	(0.07)		(0.07)	(0.07)		(0.04)	(0.04)
Forearm, M	1.68*	1.06	--	1.62*	1.06	--	1.06*	1.24*
(SE)	(0.09)	(0.07)		(0.09)	(0.07)		(0.02)	(0.05)
Hand, M	1.29*	0.81*	--	1.25*	0.81*	--	1.06*	0.85*
(SE)	(0.09)	(0.04)		(0.09)	(0.04)		(0.02)	(0.10)
Thigh, M	1.29*	0.80*	--	1.25*	0.81*	--	0.99	1.15*
(SE)	(0.11)	(0.02)		(0.10)	(0.02)		(0.02)	(0.05)
Shank, M	1.37*	0.86*	--	1.33*	0.86*	--	1.05	1.09
(SE)	(0.11)	(0.03)		(0.11)	(0.03)		(0.02)	(0.05)
Foot, M	1.05	0.66*	--	1.02	0.67*	--	1.02	0.93*
(SE)	(0.04)	(0.02)		(0.04)	(0.02)		(0.03)	(0.03)
Head, M	1.20	0.75*	--	1.16	0.75*	--	1.01	0.99
(SE)	(0.15)	(0.06)		(0.14)	(0.07)		(0.05)	(0.07)

* & ____ p < .05; t = 2.78

The superiority of 3D compared to planar (2D) geometry to characterize anthropometric differences between groups was also evident by significant differences between groups in the volume (and mass) of the shank, but an absence of differences in the uni-dimensional measures of the same segment (i.e. length or depth) (Table 8, *shank*). The correlation between segment depth and volume for individual segments ranged from r = 0.09 for the lower trunk to r = 0.88 for the head. When considered across all segments, the correlation was higher, r = .74, but still left a large proportion (45%) of variability unaccounted (r^2 = .55), indicating uni-dimensional measures are insufficient to account for variation in volume or mass of body segments across individuals.

Individual-specific anthropometrics of the whole trunk are provided in Table 9 and for the extremities in Appendix: *Upper and Lower Limb Anthropometrics*. Not surprisingly,

estimated trunk density was lower in the high BMI group, consistent with an increased accumulation of adipose tissue relative to body mass (Table 9, *density)*. The difference in density also had the effect of attenuating differences in segment mass (and corresponding BSIPs) between groups (compared to an assumption of uniform density across all subjects). The estimated location of the L5 vertebral body in the superior-inferior direction, expressed as a percentage of segment length, did not differ between groups (L5 z, Table 9), consistent with uniform trunk length between groups.

Table 9. Whole trunk anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All differences between high and normal BMI pairs were significant, $p < .05$, with the exception of the estimated location of L5 in the superior-inferior direction.

	Volume		mass*		Density	length	depth	circ	waist/	L5, z
Participant	(cc)	(%$_{body}$)	(g)	(%$_{body}$)	(g/cc)	(cm)	(cm)	(cm)	hip	(%)
1	50,273.6	57.1	49.223.8	54.8	0.979	74.1	28.3	98.0	0.91	.67
2	57,077,9	54.9	54,388.0	52.4	0.953	73.8	32.9	117.5	0.98	.67
3	68,284.3	53.6	65,254.6	51.5	0.955	73.6	33.8	116.5	0.97	.69
4	60,372.4	56.2	58,531.5	53.9	0.969	72.5	33.5	118.0	1.01	.66
5	66,344.8	59.2	63,482.2	56.3	0.957	73.8	36.3	125.5	1.07	.65
6	39,656.9	51.5	40,389.6	49.3	1.018	75.4	21.1	80.7	0.72	.68
7	32,701.9	46.9	32,479.4	44.8	0.993	71.1	19.9	80.9	0.79	.64
8	34,182.8	49.0	33,814.6	46.0	0.989	68.4	22.8	76.1	0.80	.70
9	30,295.3	46.9	29,974.1	44.7	0.989	67.7	19.5	75.0	0.82	.65
high, M	*60,470.5*	*56.2*	*58,176.0*	*53.8*	*0.963*	*73.5*	*32.9*	*115.1*	*0.99*	*0.67*
SD	*7263.9*	*2.2*	*6,574.2*	*1.9*	*0.011*	*0.6*	*2.9*	*10.2*	*0.06*	*0.02*
normal, M	*34,209.2*	*48.6*	*34,164.4*	*46.2*	*0.998*	*70.7*	*20.8*	*78.2*	*0.78*	*0.68*
SD	*3696.4*	*2.2*	*4,444.9*	*2.2*	*0.014*	*3.5*	*1.5*	*3.1*	*0.04*	*0.04*
high/norm,	*1.89*	*1.19*	*1.84*	*1.20*	*0.97*	*1.06*	*1.63*	*1.49*	*1.23*	*1.01*
M, SD	*0.25*	*0.06*	*0.24*	*0.05*	*0.01*	*0.02*	*0.18*	*0.17*	*0.06*	*0.04*

*trunk mass and density are derived from the scale-adjusted values for the upper, middle and lower trunk (see respective tables). Values derived using scale-adjusted whole trunk density are reported in accompanying text.

**length was measured from 3D geometry as distance from most proximal (C7) to distal aspect of mid-pelvis; depth was also measured from the 3D geometry as the maximum of the middle trunk depth; waist circumference and waist:hip ratio are from physical measurements; L5, z is the location of the L5 vertebral segment from the proximal trunk as a percentage of trunk length.

2.1.2. Mass distribution within the trunk

Subject-specific anthropometrics of the upper, middle and lower trunk segments are reported in Table 10, Table 11, and Table 12, respectively. For normal BMI participants, the distribution of volume within the trunk segment was slightly greater in the upper and lower trunk segments, compared to the middle trunk (Table 10, Table 11, and Table 12, *volume*, $\%_{trunk}$,). Correcting for density (e.g. of the lungs) shifted the largest distribution of mass to the lower trunk segment, which included a portion of the hips and pelvis.

In contrast, for high BMI participants, the middle trunk segment contained the greatest distribution of both volume and mass. In fact, although the upper and lower trunk accounted for more of the total body volume (and mass) in the high BMI participants (Table 10, Table 11, and Table 12, *high/norm*, $\%_{body}$), the segments represented a lower proportion of total trunk mass and volume (Table 10, Table 11, and Table 12, *high/norm*, $\%_{trunk}$), indicating a substantial shift towards a central accumulation of body volume (and mass).

Table 10. Upper trunk anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All parameters were significantly different based on relative differences (high/norm), p < .05, with the exception of the body-mass normalized mass and volume distribution (%body).

	Volume			Mass			density	length*	depth
Participant	(cc)	($\%_{body}$)	($\%_{trunk}$)	(g)	($\%_{body}$)	($\%_{trunk}$)	(g/cc)	(cm)	(cm)
1	15,399.2	17.5	30.6	13,228.8	14.7	26.9	0.859	23.1	25.6
2	19,588.5	18.8	34.3	16,496.7	15.9	30.3	0.842	24.6	31.2
3	24,017.9	18.8	35.2	20,322.0	16.0	31.1	0.846	23.6	34.9
4	18,0.32.5	16.8	29.9	15,357.5	14.1	26.2	0.852	22.3	31.9
5	22,337.2	19.9	33.7	18,891.5	16.8	29.8	0.847	24.0	31.1
6	12,696.7	16.5	32.0	11,349.0	13.9	28.1	0.894	24.1	19.4
7	11,182.9	16.0	34.2	9,800.8	13.5	30.2	0.876	20.1	23.9
8	14,531.7	20.8	42.5	12,880.9	17.5	38.1	0.886	24.7	23.0
9	10,354.7	16.0	34.2	9,040.3	13.5	30.2	0.873	19.3	23.1
high, M	*19,875.1*	*18.4*	*32.7*	*16,863.9*	*15.5*	*28.9*	*0.849*	*23.5*	*30.9*
SD	*3418.2*	*1.2*	*2.3*	*2818.8*	*1.1*	*2.2*	*0.006*	*0.9*	*3.4*
normal, M	*12,191.4*	*17.4*	*35.7*	*10,767.8*	*14.6*	*31.6*	*0.882*	*22.0*	*22.3*
SD	*1837.0*	*2.3*	*4.6*	*1705.2*	*1.9*	*4.4*	*0.009*	*2.7*	*2.0*
high/norm, M, SD	*1.74*	*1.09*	*0.92*	*1.68*	*1.09*	*0.91*	*0.97*	*1.14*	*1.33*
	0.28	*0.13*	*0.08*	*0.27*	*0.13*	*0.08*	*0.01*	*0.11*	*0.16*

*Upper trunk length was derived from the 3D model and represents the distance from the C7 joint centre to the xyphoid process; depth is the maximum anterior-posterior distance.

The effect of density estimates on segment mass was apparent in small but differential effects across trunk segments and groups. As expected, the estimated density of the upper trunk was < 1 for both normal and high BMI groups, due to the effect of pulmonary void space (i.e. density < 1) (Table 10, *density*), and similarly, the density of the lower trunk was > 1 for all participants in both groups (Table 12, *density*). In contrast, the density of the middle trunk was >1 for all normal BMI participants and < 1 for (near) all high BMI participants (Table 11, *density*), an effect that actually attenuated the differences in trunk mass between groups.

Table 11. Middle trunk anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All parameters (except length) were significantly different based on relative differences (high/norm), p < .05.

	Volume			Mass			Density	length*	depth
Participant	(cc)	($\%_{body}$)	($\%_{trunk}$)	(g)	($\%_{body}$)	($\%_{trunk}$)	(g/cc)	(cm)	(cm)
1	17,242.5	19.6	34.3	17,366.2	19.3	35.3	1.007	21.4	28.3
2	19,398.0	18.6	33.9	19,152.9	18.5	35.2	0.987	20.0	32.9
3	23,277.2	18.2	34.1	23,091.0	18.2	35.4	0.992	21.7	33.8
4	23,993.7	22.4	39.7	23,957.5	22.1	40.9	0.998	23.0	33.5
5	25,955.5	23.2	39.1	25,767.7	22.9	40.6	0.993	23.9	36.3
6	11,639.3	15.1	29.4	12,197.6	14.9	30.2	1.048	20.0	21.1
7	10,279.5	14.7	31.4	10,562.8	14.6	32.5	1.028	20.9	19.9
8	9,609.6	13.8	28.1	9,986.6	13.6	29.5	1.039	18.7	22.8
9	9,521.2	14.8	31.4	9,745.8	14.6	32.5	1.024	20.2	19.5
high, M	*21,973.4*	*20.4*	*36.3*	*21,867.1*	*20.2*	*37.5*	*0.996*	*22.0*	*32.9*
SD	*3558.5*	*2.2*	*2.9*	*3489.6*	*2.1*	*3.0*	*0.008*	*1.5*	*2.9*
normal, M	*10,262.4*	*14.6*	*30.1*	*10,628.2*	*14.4*	*31.2*	*1.035*	*20.0*	*22.3*
SD	*978.4*	*0.6*	*1.6*	*1104.2*	*0.6*	*1.6*	*0.011*	*0.9*	*2.0*
high/norm,	*2.25*	*1.40*	*1.18*	*2.17*	*1.40*	*1.17*	*0.97*	*1.09*	*1.63*
M, SD	*0.44*	*0.13*	*0.09*	*0.43*	*0.13*	*0.09*	*0.01*	*0.10*	*0.18*

*Middle trunk length was derived from the 3D model and represents the distance from the xyphoid process to the umbilicus; depth is the maximum anterior-posterior distance.

Table 12. Lower trunk anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All parameters were significantly different based on relative differences (high/norm), p < .05, with the exception of segment length and the trunk-normalized volume and mass distributions (%trunk).

	Volume			Mass			Density	length*	depth
Participant	(cc)	($\%_{body}$)	($\%_{trunk}$)	(g)	($\%_{body}$)	($\%_{trunk}$)	(g/cc)	(cm)	(cm)
1	17,631.9	20.0	35.1	18,628.8	20.7	37.9	1.057	29.5	28.2
2	18,091.4	17.4	31.7	18,738.5	18.1	34.5	1.036	29.3	33.5
3	20,988.8	16.5	30.7	21,841.6	17.2	33.5	1.041	28.3	33.1
4	18,346.2	17.1	30.4	19,216.5	17.7	32.8	1.048	27.1	31.7
5	18,052.1	16.1	27.2	18,800.0	16.7	29.6	1.041	25.9	33.1
6	15,321.0	19.9	38.6	16,843.0	20.6	41.7	1.099	31.4	24.2
7	11,239.9	16.1	34.4	12,115.8	16.7	37.3	1.078	30.1	23.8
8	10,041.5	14.4	29.4	10,947.0	14.9	32.4	1.090	25.0	24.0
9	10,419.4	16.1	34.4	11,188.0	16.7	37.3	1.074	28.1	23.7
high, M	*18,622.1*	*17.4*	*31.0*	*19,455.1*	*18.1*	*33.6*	*1.044*	*28.0*	*31.9*
SD	*1347.6*	*1.5*	*2.8*	*1358.0*	*1.6*	*3.0*	*0.008*	*1.5*	*2.2*
normal, M	*11,755.4*	*16.6*	*34.2*	*12,773.4*	*17.2*	*37.2*	*1.085*	*28.6*	*23.9*
SD	*2429.1*	*2.3*	*3.8*	*2759.4*	*2.4*	*3.8*	*0.012*	*2.7*	*0.2*
high/norm, M, SD	*1.75*	*1.10*	*0.93*	*1.70*	*1.11*	*0.93*	*0.97*	*1.00*	*1.34*
	0.21	*0.09*	*0.10*	*0.19*	*0.09*	*0.10*	*0.01*	*0.08*	*0.09*

*Lower trunk length was derived from the 3D model and represents the distance from the umbilicus to the most inferior portion of the lower trunk segment (mid-line of pelvis); depth is the maximum anterior-posterior distance at the level of the buttocks.

2.2. Body segment inertial parameters

2.2.1. BSIPs of whole body segments

A significant anterior displacement in the CM_{loc} of the whole trunk was detected for the high BMI participants (M = 3.1, SD = 1.2 cm), compared to the normal BMI group (M = 0.5, SD = 0.7 cm) (Table 13, CM_y). The CM_{loc} in the superior-inferior direction (Z-axis) did not differ between groups, and depended upon the mass of individual trunk segments (see Table 10, Table 11, Table 12, *mass*). For instance, the CM_{loc} was farther superior in subject 8 compared to the rest of the sample (not shown), due to increased muscle mass in the upper torso (see Figure 4). Individual-specific body segment parameters of the whole trunk, and upper and lower extremities are provided in Appendix: *Trunk and Extremity BSIPs*.

In the lower extremity, as well as head/neck, the longitudinal CM_{loc} was situated closer to the proximal joint centre in the high BMI participants (Table 13, CM_z), reflecting a more proximal mass distribution.

Table 13. Summary of relative differences (high/norm) in inertial parameters between high and low BMI participants. The mean (SE) is reported and significant differences (p < .05) are indicated (* & ____); 95% confidence intervals can be constructed from the SE and t-critical value (2.78).

	CMx* (%length)	CMy (%length)	CMz (%length)	Ixx (kgcm^2)	Iyy (kgcm^2)	Izz (kgcm^2)	Rxx (%length)	Ryy (%length)	Rzz (%length)
Whole Trunk, M (SE)	1.35 (0.81)	6.89* (0.59)	0.94 (0.04)	2.09* (0.16)	2.08* (0.15)	3.24* (0.32)	1.00 (0.01)	1.00 (0.01)	1.24* (0.02)
Upper Trunk, M (SE)	0.49 (0.63)	1.88* (0.24)	1.05* (0.01)	2.53* (0.28)	2.41* (0.31)	2.56* (0.30)	1.07 (0.05)	1.04 (0.04)	1.08 (0.06)
Middle Trunk, M (SE)	3.09 (1.66)	3.69* (1.06)	1.06* (0.03)	3.68* (0.58)	3.43* (0.42)	4.27* (0.56)	1.17* (0.02)	1.15* (0.03)	1.28* (0.04)
Lower Trunk, M (SE)	0.93 (0.09)	1.38* (0.16)	0.96 (0.03)	2.42* (0.24)	2.28* (0.20)	3.02* (0.28)	1.20* (0.04)	1.16* (0.02)	1.34* (0.06)
Upper Arm, M (SE)	2.27 0.73)	1.80 (0.74)	0.96 (0.04)	1.56* (0.21)	1.46* (0.20)	2.87* (0.36)	0.99 (0.03)	0.95 (0.04)	1.35* (0.05)
Forearm, M (SE)	0.42* (0.17)	0.65 (0.17)	1.02 (0.02)	1.81* (0.23)	1.73* (0.21)	2.49* (0.28)	0.98 (0.03)	0.96 (0.04)	1.16* (0.04)
Hand, M (SE)	2.16 (0.49)	0.68 (0.35)	0.84* (0.05)	1.43 (0.23)	1.71* (0.25)	1.64* (0.21)	0.90 (0.05)	0.99 (0.04)	1.00 (0.12)
Thigh, M (SE)	1.99 (0.84)	0.58* (0.20)	0.89* (0.02)	1.12 (0.10)	1.08 (0.09)	1.90* (0.29)	0.96 (0.13)	0.94* (0.02)	1.22* (0.04)
Shank, M (SE)	0.94 (0.38)	0.96 (0.19)	0.94* (0.01)	1.38* (0.13)	1.40* (0.13)	1.84* (0.25)	0.97* (0.01)	0.98 (0.01)	1.11* (0.05)
Foot, M (SE)	1.23 (0.38)	1.13 (0.08)	0.89* (0.04)	1.06 (0.08)	1.02 (0.06)	1.09 (0.08)	0.99 (0.01)	0.98 (0.03)	1.00 (0.01)
Head, M (SE)	1.61 (0.69)	0.94 (0.10)	0.85* (0.01)	1.35 (0.38)	1.32 (0.36)	1.49 (0.40)	1.03 (0.02)	1.02 (0.01)	1.09 (0.37)

CM = centre of mass location relative to proximal joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, kgcm^2
R = radius of gyration, as a percentage of segment length
* & ____ p < .05; t = 2.78

As expected, moments of inertia for the whole trunk segment were substantially greater in the high BMI group, over double that of the height-matched normal-BMI participants (and triple about the Z-axis) (Table 13, I_{xx}, I_{yy}, I_{zz}). The radii of gyration were greater in the high BMI group (M = 12.6, SD = 0.7 cm) only about the Z-axis (twist) – which takes into account mass distribution in both the anterior-posterior and medial-lateral directions (normal BMI R_{zz}: M = 9.8,

SD = 0.4 cm). The absolute radii (cm) about the X- (somersault) and Y-axes (cartwheel) were greater for the high BMI group (M = 20.2, SD = 0.6 cm and M = 20.7, SD = 0.7, respectively) than those of the normal BMI group (M = 18.9, SE = 0.5 cm and M = 19.7, SE = 0.6 cm), $p < .05$, however the relative differences (Table 13, R_{xx}, R_{yy}) were not.

Similarly, with the exception of the foot/hand/head, the moments of inertia about the Z-axis of all primary limb segments were greater in the high BMI participants (Table 13, I_{xx}, I_{yy}, I_{zz}). The radii of gyration about the Z-axis for most limb segments was also greater in the high BMI participants, indicating that mass distribution about a transverse axis did not scale with body mass (Table 13, R_{zz}). Comparisons of inertial properties of the hand segment may not be reflective of group differences, as some participants held the hands relaxed while others held the hand in a fist (see Figure 4 and Appendix: *Trunk and Extremity BSIPs*).

2.2.2. BSIPs of trunk segments

Similar to the whole trunk, the CM_{loc} was farther anterior in the high BMI participants compared to the normal BMI for the upper (high BMI: M = 3.0, SD = 0.6 cm, normal BMI: M = 1.5, SD = 0.2 cm), middle (high BMI: M = 4.1, SD = 0.2 cm, normal BMI: M = 1.9, SD = 1.0 cm) and lower trunk segments (high BMI: M = 2.5, SD = 0.4 cm, normal BMI: M = 3.0, SD = 0.6 cm), with the largest difference in the middle trunk segment (Table 14, Table 15, Table 16, CM_y). However, the relative differences in anterior-posterior CM_{loc}s of individual trunk segments (M = 1.4 to 3.7) were lower than that of the whole trunk (M = 6.9), an effect that may be attributed to the relative orientation of multiple trunk segments. The Z-direction CM_{loc} was more inferior in the high BMI group for both the upper and middle segments (Table 14, Table 15, CM_z), and tended to be farther superior in the lower segment (Table 16, CM_z), consistent with a more central accumulation of trunk mass.

Table 14. Upper trunk inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All moments of inertia and CMloc in the vertical (CMz) and anterior-posterior (CMy) were significantly different based on relative differences (high/norm), p < .05.

Participant	CMx (%L)	CMy (%L)	CMz (%L)	Ixx ($kgcm^2$)x10^2	Iyy ($kgcm^2$)x10^2	Izz ($kgcm^2$)x10^2	Rxx (%L)	Ryy (%L)	Rzz (%L)
1	1.1	14.5	58.2	10.02	17.72	17.62	37.7	50.1	49.9
2	0.0	9.4	58.9	15.84	22.70	24.51	39.8	47.7	49.6
3	1.2	16.2	59.1	21.46	33.52	38.46	43.6	54.5	58.3
4	0.2	13.2	60.2	13.45	21.18	23.89	41.9	52.6	55.8
5	3.1	10.0	57.2	17.69	31.42	33.01	40.3	53.7	55.5
6	0.5	7.3	59.8	7.09	16.11	14.62	32.9	49.5	47.2
7	0.6	6.7	55.2	5.94	10.05	10.32	38.7	50.3	51.0
8	1.2	6.2	57.9	9.33	16.58	15.37	34.4	45.9	44.2
9	0.2	7.2	55.7	5.18	8.76	9.14	39.2	51.0	52.0
high, M	*1.1*	*12.7*	*58.7*	*15.70*	*25.31*	*27.50*	*40.7*	*51.7*	*53.7*
SD	*1.2*	*2.9*	*1.1*	*4.32*	*6.82*	*8.22*	*2.2*	*2.8*	*3.8*
normal, M	*0.6*	*6.8*	*57.2*	*6.89*	*12.88*	*12.36*	*36.3*	*49.2*	*48.6*
SD	*0.4*	*0.5*	*2.1*	*1.81*	*4.04*	*3.10*	*3.1*	*2.3*	*3.6*
high/norm, M, SD	*0.49* *1.40*	*1.88* *0.52*	*1.05* *0.03*	*2.53* *0.63*	*2.41* *0.70*	*2.56* *0.68*	*1.07* *0.11*	*1.04* *0.09*	*1.08* *0.14*

CM = centre of mass location relative to C7 joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$
R = radius of gyration, as a percentage of segment length

Table 15. Middle trunk inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All parameters (except CMx) were significantly different based on relative differences (high/norm), p < .05.

Participant	CMx (%L)	CMy (%L)	CMz (%L)	Ixx ($kgcm^2$)x10^2	Iyy ($kgcm^2$)x10^2	Izz ($kgcm^2$)x10^2	Rxx (%L)	Ryy (%L)	Rzz (%L)
1	1.9	17.5	49.2	15.47	21.83	23.91	44.0	52.3	54.7
2	3.5	21.0	50.5	18.93	24.91	30.83	49.8	57.1	63.6
3	0.4	17.9	50.7	27.04	32.33	40.77	49.9	54.5	61.2
4	2.4	18.8	50.8	28.03	33.75	40.88	47.0	51.5	56.7
5	0.8	17.3	52.6	32.76	37.11	45.81	47.2	50.3	55.9
6	4.7	13.9	51.3	7.59	13.42	12.96	39.4	52.4	51.5
7	0.4	3.1	46.2	7.43	9.53	9.10	40.1	45.4	44.3
8	3.7	12.9	52.9	6.12	8.68	8.89	42.0	50.0	50.6
9	1.9	7.7	47.3	6.47	8.35	8.05	40.3	45.8	44.9
high, M	*1.8*	*18.5*	*50.7*	*24.44*	*29.99*	*36.43*	*47.6*	*53.1*	*58.4*
SD	*1.2*	*1.5*	*1.2*	*7.06*	*6.38*	*8.87*	*2.4*	*2.7*	*3.8*
normal, M	*2.7*	*9.4*	*49.4*	*6.90*	*10.00*	*9.75*	*40.4*	*48.4*	*47.8*
SD	*1.9*	*5.0*	*3.2*	*7.2*	*2.33*	*2.19*	*1.1*	*3.4*	*3.7*
high/norm, M,SD	*3.09* *3.71*	*3.69* *2.36*	*1.06* *0.06*	*3.68* *1.30*	*3.43* *0.93*	*4.27* *1.25*	*1.17* *0.05*	*1.15* *0.07*	*1.28* *0.09*

CM = centre of mass location relative to T9 joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$
R = radius of gyration, as a percentage of segment length

In contrast to the differences in CM_{loc}, partitioning the trunk into segments revealed larger differences in moments of inertia for the middle trunk segment (Table 15, I_{xx}, I_{yy}, I_{zz}) – being over 4x greater for the high BMI group. The pattern of differences across axes for the moments was similar to that of the whole trunk (i.e. greatest about the z-axis, followed by the somersault or x-axis). The radii of gyration in the middle and lower trunk segments were also farther from the centre of mass in the high BMI participants (Table 15, Table 16, R_{xx}, R_{yy}, R_{zz}) indicating that unlike the whole trunk, the distribution of mass about all axes for individual segments (excluding the upper trunk) does not scale with mass. In other words, the increase in moment of inertia for the middle and lower trunk segments can be attributed to both an increase in mass and mass distribution (i.e. farther from the centre of mass).

Table 16. Lower trunk inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. All parameters (except CMx and CMz) were significantly different based on relative differences (high/norm), p < .05.

Participant	CMx (%L)	CMy (%L)	CMz (%L)	Ixx ($kgcm^2$)x10^2	Iyy ($kgcm^2$)x10^2	Izz ($kgcm^2$)x10^2	Rxx (%L)	Ryy (%L)	Rzz (%L)
1	0.9	9.3	40.7	20.35	22.21	21.01	35.4	37.0	36.0
2	1.4	11.9	36.2	21.12	21.31	24.33	36.2	36.4	38.9
3	2.7	11.6	39.3	26.18	26.99	32.21	38.7	39.3	42.9
4	1.7	8.0	36.1	21.19	21.02	27.41	39.4	38.6	44.0
5	1.6	13.3	35.1	22.91	19.38	28.60	42.7	39.2	47.7
6	1.5	8.6	43.6	15.41	19.63	16.28	30.5	34.4	31.4
7	1.5	7.3	38.4	10.56	10.80	9.53	31.1	31.4	29.5
8	2.7	11.6	40.9	7.96	8.80	8.58	34.1	35.8	25.4
9	1.6	7.1	38.8	9.15	9.41	8.49	32.2	32.6	31.0
high, M	*1.7*	*10.8*	*37.5*	*22.50*	*22.19*	*26.71*	*38.5*	*38.1*	*41.9*
SD	*0.7*	*2.1*	*2.4*	*2.27*	*2.88*	*4.26*	*2.9*	*1.3*	*4.5*
normal, M	*1.8*	*8.7*	*40.4*	*10.77*	*12.16*	*10.72*	*32.0*	*33.6*	*31.8*
SD	*0.6*	*2.1*	*2.4*	*3.27*	*5.05*	*3.74*	*1.6*	*1.9*	*1.5*
high/norm, M,SD	*0.93*	*1.38*	*0.96*	*2.42*	*2.28*	*3.02*	*1.20*	*1.16*	*1.34*
	0.20	*0.36*	*0.06*	*0.54*	*0.45*	*0.63*	*0.08*	*0.04*	*0.14*

CM = centre of mass location relative to L3 joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$
R = radius of gyration, as a percentage of segment length

3. COMPARISON TO EXISTING ESTIMATES

3.1. Whole trunk segment

The difference in whole trunk BSIPs between the current and historical models differed across models (MODEL effect, Table 17). More importantly, the magnitude of the difference was dependent upon body mass index (main GROUP effect) and was non-uniform across BSIPs (MODEL*GROUP*PARAMETER and GROUP*PARAMETER interactions).

Table 17. Analysis of variance for difference between individual-specific and regression-based estimates of the whole trunk. The 3-way ANOVA evaluates for differences between models, and the 2-way ANOVA evaluates the effect of BMI, indicated by main effect (GROUP) and interaction across parameters.

	Df	F	P	η_p^2*
3-way ANOVA (MODEL, PARAMETER, GROUP)				
MODEL	1, 7	102.08	< .001	.94
MODEL*PARAMETER*GROUP	4, 28	3.10	< .05	.31
2-way ANOVA (PARAMETER, GROUP)				
Pearsall				
GROUP	1, 7	18.59	< .01	.73
GROUP*PARAMETER	4, 28	15.70	< .001	.69
De Leva				
GROUP	1, 7	33.97	< .001	.83
GROUP*PARAMETER	4, 28	32.16	< .001	.82
Zatsiorsky**				
GROUP	7	t = 2.98	< .05	--
Hanavan**				
GROUP	7	t = 6.20	<.01	--

*effect size
**one parameter only, effect of GROUP evaluated with an independent samples t-test

3.1.1. Main effects

As expected, the individual-specific model of the current study returned greater estimates of whole trunk mass compared to historical regression or geometric models across all subjects (range, 6.4 – 13.5%, Table 18, *overall*, * mass). Similarly, estimates of CM_{loc} were farther superior relative to existing models (Table 18, *overall*, CM_z), but with relatively small absolute differences (Pearsall model, M = 1.7 SD = 0.3 cm; DeLeva model, M = 6.6, SD = 0.5 cm).

Across all subjects (Table 18, *overall*, all parameters), significant differences were detected for 8 of 12 possible comparisons (mass: 3 of 4; CM_{loc} : 2 of 2; inertia: 3 of 6).

Table 18. Difference in individual-specific (current) and predicted inertial parameters of the whole trunk, computed as (current - predicted)/current *100. Positive values indicate higher estimates for the current model, mean (SE) shown. The difference was tested across all subjects and for each group (*), as well as between groups (shaded), p < .05; t-critical values are provided.

	Mass	CMz	Ixx	Iyy	Izz
Pearsall (1994)					
overall	9.4 (1.7)*	7.4 (1.3)*	-22.9 (3.2)*	-27.0 (3.3)*	1.5 (6.1)
normal BMI	6.1 (3.0)	7.3 (2.7)	-30.5 (2.9)*	-33.5 (3.7)*	-16.9 (3.9)*
high BMI	12.1 (1.2)*	7.4 (1.4)*	-16.8 (3.2)*	-21.8 (3.9)*	16.2 (2.0)*
De Leva (1996)					
overall	13.5 (2.4)*	27.0 (1.8)*	17.3 (5.3)*	11.5 (5.4)	20.8 (10.0)
normal BMI	6.6 (1.9)*	23.8 (3.0)*	3.1 (5.7)	-3.0 (5.6)	-9.3 (4.3)
high BMI	19.1 (2.4)*	29.6 (1.6)*	28.7 (3.0)*	23.1 (3.0)*	44.8 (4.9)*
Zatsiorsky (1983)					
overall	12.0 (1.6)*	--	--	--	--
normal BMI	8.1 (2.0)*	--	--	--	--
high BMI	15.1 (1.4)*	--	--	--	--
Hanavan (1964)					
overall	6.4 (3.4)	--	--	--	--
normal BMI	-3.5 (2.9)	--	--	--	--
high BMI	14.4 (1.1)*	--	--	--	--

*p < .05, two-sided for $H_0 = 0$. ___ = difference between normal and high BMI groups (GROUP effect), p < .05.
CMz = distance from L5/S1 joint centre
x = medial-lateral; y = anterior-posterior; z = superior-inferior
t-distribution critical values at α = .05 (two-sided) for overall: df(8) = 2.31; normal BMI: df(3) = 3.18; high BMI: df(4) = 2.78

3.1.2. Effect of high BMI

Evidence for a BMI-specific variance between current and predicted BSIPs was supported by findings of significantly different variances between normal and high BMI groups (8 of 12 comparisons, Table 18, __). Additionally, individual-group differences were also detected for the high, but not normal, participants (2 additional comparisons & 6 of 12 across all parameters, Table 18, *). In fact, 3 of the 8 differences detected across all subjects above (Table 18, *overall*, *) could be attributed to differences in the high BMI and not the normal BMI participants (Pearsall: 2; de Leva: 1, Table 18, *).

The current model returned greater estimates of trunk mass in high BMI participants relative to all comparative models (M = 15.2, SE = 0.5 %) (Table 18, *mass*, high BMI *), while the difference was much lower for normal BMI participants (M = 4.3, SE = 1.3 %) and significant for only two of four models. Additionally, the differences between high and normal BMI groups were significance for 3 of the 4 models (Table 18, *mass*, __), and although the between-group comparison was not significant for the fourth (Table 18, *mass*, Pearsall), the estimate of trunk mass differed for the high-BMI group (*) and not the normal BMI group.

The effect of increased body mass on the moment of inertia estimates was substantial, with significantly greater predicted moments for the high BMI participants in 5 of 6 parameters (Table 18, *Ixx*, *Iyy*, *Izz*, __). The current model returned greater estimates for all moments of inertia in high BMI participants in one comparative model (M = +32.2, SE = 3.6%, Table 18, *de Leva*), with very similar estimates for the normal BMI participants (M = -3.1, SE = 5.1%, ns). For the second model (Pearsall), estimates of whole trunk inertia were lower for both high (except the z-axis) and normal BMI participants (all axes).

Regardless of the variation amongst predictive models, a strong influence of body mass on BSIP estimates was apparent, and for many parameters and models the variance in overall comparisons (all participants) was due exclusively to the high BMI group. No predictive models accounted for the difference in estimated CM_{loc} in anterior-posterior direction detected in the current model (Table 9, CM_y).

3.2. Individual Trunk Segments

The results of omnibus comparisons for individual trunk segments were similar to that of the whole trunk, with significant main effects of MODEL ($p < .001$) and GROUP*MODEL ($p < 01$), and GROUP*MODEL*PARAMETER ($p < .01$) interactions (see Appendix: *Comparison of Trunk Segments and Extremities* for ANOVA tables).

3.2.1. Main effects

When compared across all subjects, only 5 of 12 differences in predicted mass (Table 19, Table 20, Table 21, *overall*, mass *) reached significance, which tended to be localized to the upper (2) and lower (2) trunk segments. With the exception of the Zatsiorsky and De leva models for the lower trunk (Table 21, *mass*), the mean (SD) differences in predicted mass of individual segments was relatively low, 4.0 (8.0) %, indicating a relatively good fit of predictive equations when compared across all subjects. The increased mass estimates for the lower trunk compared to the Zatsiorsky (M = 36.5, SE = 0.8%) and De Leva (M = 36.5, SE = 0.7%) models can likely be attributed to differences in the upper thigh/gluteal mass included in each model (a function of the segmentation angle), as the current model returned correspondingly lower estimates for the mass of the thigh (see Appendix: *Comparison of Trunk Segments and Extremities*, Zatsiorsky: M = -24.6, SE = 4.8%; DeLeva: M = -26.7, SE = 5.0%).

Table 19. Difference in individual-specific (current) and predicted inertial parameters of the upper trunk, computed as (current - predicted)/current *100. Positive values indicate higher estimates for the current model, mean (SE). The difference was tested across all subjects and for each group (*), as well as between groups (shaded), p < .05; t-critical values are provided.

	Mass	CMz	Ixx	Iyy	Izz
Pearsall (1994)					
overall	15.0 (3.3)*	29.2 (2.1)*	15.5 (7.5)	6.7 (7.6)	-2.8 (6.9)
normal BMI	12.2 (6.6)	26.3 (4.4)*	-2.6 (9.8)	-8.8 (12.2)	-17.9 (9.3)
high BMI	17.3 (3.2)*	31.5 (0.6)*	29.9 (5.1)*	19.1 (5.5)*	9.2 (6.1)
De Leva (1996)					
overall	-6.2 (3.4)	36.1 (2.8)*	-6.3 (12.9)	-51.3 (14.7)*	-25.5 (13.5)
normal BMI	-9.8 (6.6)	30.1 (2.9)*	-40.2 (13.5)	-87.6 (11.0)*	-63.0 (9.0)*
high BMI	-3.3 (3.2)	41.0 (3.3)*	20.7 (8.9)	-22.2 (14.5)	4.6 (9.9)
Zatsiorsky (1983)					
overall	-7.1 (3.4)	0.8 (3.3)	6.4 (5.8)	-29.5 (8.5)*	-8.1 (6.2)
normal BMI	-6.1 (7.0)	4.6 (6.6)	-1.0 (10.6)	-38.8 (16.9)	-16.3 (11.0)
high BMI	-7.9 (3.5)	-5.0 (1.2)*	12.3 (5.6)	-22.0 (7.7)*	-1.6 (6.5)
Hanavan (1964)					
overall	17.4 (5.1)*	27.2 (4.1)*	21.4 (6.0)*	33.9 (5.3)*	32.8 (3.8)*
normal BMI	10.0 (7.6)	22.2 (4.6)*	14.2 (11.1)	27.9 (8.1)*	30.6 (5.6)*
high BMI	23.4 (6.2)*	31.1 (6.3)*	27.1 (6.3)*	38.7 (6.9)*	34.6 (5.6)*

*p < .05, two-sided for $H_0 = 0$. ___ = difference between normal and high BMI groups, p < .05.
CMz = distance from proximal joint centre
x = medial-lateral; y = anterior-posterior; z = superior-inferior
t-distribution critical values at α = .05 (two-sided) for overall: df(8) = 2.31; normal BMI: df(3) = 3.18; high BMI: df(4) = 2.78

The CM_{loc} in the current model was located farther distal than the predictive models for most trunk segments (Table 19, Table 20, *overall*, CM_z *). Although the relative differences appear large, the absolute differences were of fairly low magnitudes, owing to the short segment lengths – in the lower trunk segment, the average variance across models was 16.2%, which corresponded to a mean (SE) distance of 1.78 (0.22) cm; in the middle trunk the mean variance was 11.0%, a 1.54 (0.22) cm distance; and in the upper trunk the values were 24.3% and 3.2 (0.3) cm, respectively.

Across all subjects, half of all differences (18 of 36) in estimated moments of inertia between current and predictive models reached significance (Table 19, Table 20, Table 21, *overall*, I_{xx}, I_{yy}, I_{zz} *). Interestingly, in 11 of 12 possible cases the current model returned a larger difference for moments about the X-axis (somersault) compared to the Y-axis (cartwheel), an effect that was also apparent in 22 of 24 possible cases for normal- and high-BMI comparisons.

Table 20. Difference in individual-specific (current) and predicted inertial parameters of the middle trunk, computed as (current - predicted)/current *100. Positive values indicate higher estimates for the current model, mean (SE) shown. The difference was tested across all subjects and for each group (*), as well as between groups (shaded), p < .05; t-critical values are provided.

	Mass	CMz	Ixx	Iyy	Izz
Pearsall (1994)					
overall	8.6 (3.0)*	11.7 (3.0)*	18.7 (8.2)	1.2 (6.8)	6.9 (8.2)
normal BMI	1.6 (1.4)	16.0 (2.4)*	1.1 (12.9)	-13.8 (8.9)	-15.8 (7.8)
high BMI	14.1 (3.6)*	8.3 (4.6)	32.8 (5.6)*	13.1 (6.2)	25.0 (4.7)*
De Leva (1996)					
overall	4.6 (5.9)	16.7 (4.1)*	33.7 (10.1)*	26.8 (8.7)*	30.9 (11.5)*
normal BMI	-12.6 (2.2)*	12.8 (3.7)*	7.5 (5.9)	5.4 (2.4)	0.5 (9.0)
high BMI	18.4 (3.8)*	19.9 (6.8)*	54.7 (10.1)*	43.8 (10.2)*	55.3 (9.6)*
Zatsiorsky (1983)					
overall	1.5 (3.8)	5.1 (3.0)	9.7 (7.5)	-3.9 (6.6)	9.1 (7.0)
normal BMI	-8.6 (1.5)*	-1.3 (0.8)	-11.2 (5.7)	-21.8 (5.5)*	-11.4 (4.9)
high BMI	9.5 (3.6)	10.2 (4.1)	26.4 (5.2)*	10.5 (4.6)	25.5 (3.3)*
Hanavan (1964)					
overall	2.1 (5.2)	10.2 (3.0)*	22.0 (7.1)*	11.9 (7.4)	9.8 (7.7)
normal BMI	-11.3 (6.0)	8.3 (6.1)	6.9 (10.7)	-8.8 (7.1)	-8.5 (11.8)
high BMI	12.7 (3.6)*	11.6 (3.0)*	34.1 (5.3)*	28.5 (3.1)*	24.4 (2.9)*

*p < .05, two-sided for $H_0 = 0$. ___ = difference between normal and high BMI groups, p < .05.
CMz = distance from proximal joint centre x = medial-lateral; y = anterior-posterior; z = superior-inferior
t-distribution critical values at α = .05 (two-sided) for overall: df(8) = 2.31; normal BMI: df(3) = 3.18; high BMI: df(4) = 2.78

Table 21. Difference in individual-specific (current) and predicted inertial parameters of the lower trunk, computed as (current - predicted)/current *100. Positive values indicate higher estimates for the current model, mean (SE) shown. The difference was tested across all subjects and for each group (*), as well as between groups (shaded), p < .05; t-critical values are provided.

	Mass	CMz	Ixx	Iyy	Izz
Pearsall (1994)					
overall	3.5 (1.1)	19.5 (0.9)*	26.0 (4.6)*	-19.1 (7.5)*	-12.6 (6.5)
normal BMI	2.9 (3.3)	22.6 (2.1)*	17.8 (8.6)	-23.5 (14.7)	-26.8 (11.1)
high BMI	3.9 (1.7)	17.0 (1.5)*	32.5 (2.6)*	-15.5 (8.2)	-1.2 (2.7)
De Leva (1996)					
overall	36.5 (0.7)*	8.9 (1.7)	45.3 (9.1)*	35.1 (9.1)*	41.8 (10.2)*
normal BMI	34.8 (2.1)*	10.8 (4.9)	29.9 (16.9)	20.5 (19.4)	20.6 (16.5)
high BMI	37.9 (1.0)*	7.3 (2.5)	57.6 (6.4)*	46.8 (7.2)*	58.8 (6.7)*
Zatsiorsky (1983)					
overall	36.5 (0.8)*	16.5 (0.9)*	53.1 (3.2)*	44.7 (4.2)*	50.8 (3.7)*
normal BMI	32.3 (2.2)*	20.1 (2.1)*	46.9 (5.8)*	37.6 (7.8)*	41.3 (5.1)*
high BMI	39.9 (0.8)*	13.5 (1.6)*	57.9 (1.6)*	50.4 (2.9)*	58.4 (0.7)*
Hanavan (1964)					
overall	-0.2 (1.4)	16.5 (1.4)*	-0.6 (10.4)	-10.5 (7.1)	-29.3 (9.6)*
normal BMI	-11.0 (4.3)	22.5 (4.7)	-22.5 (17.5)	-25.5 (11.5)	-41.3 (21.3)
high BMI	8.4 (0.8)*	19.6 (0.9)*	16.9 (5.8)*	1.5 (4.7)	-19.8 (2.9)*

*p < .05, two-sided for $H_0 = 0$. ___ = difference between normal and high BMI groups, p < .05.
CMz = distance from proximal joint centre
x = medial-lateral; y = anterior-posterior; z = superior-inferior
t-distribution critical values at α = .05 (two-sided) for overall: df(8) = 2.31; normal BMI: df(3) = 3.18; high BMI: df(4) = 2.78

3.2.2 Effect of high BMI

Consistent with the whole trunk segment, the effect of BMI on estimates of segment mass was evident by both between-group comparisons (5 of 12, Table 19, Table 20, Table 21, *mass* __) and differences for high, but not normal BMI participants (3 additional comparisons, Table 19, Table 20, Table 21, *mass*, *), demonstrating a significant effect of body mass in the majority (75%) of segments and models examined.

The influence of mass on predicted BSIPs was most apparent for the middle trunk segment, with evidence of the effect in 19 of 20 BSIPs comparisons (Table 20, __ & *). In fact, the between-group differences in whole trunk mass (Table 18, *overall*) could be attributed exclusively to this segment for 2 of the 4 models (*mass*, De Leva and Zatsiorsky, * Table 20 vs

Table 19, Table 21). Similarly, more than half of the BSIP comparisons in the middle trunk (12 of 20) across all subjects failed to detect a difference (Table 20, *overall*, *).

The effects of BMI on estimated moments of inertia were numerous (30 of 36 comparisons - Table 19, Table 20, Table 21, I_{xx}, I_{yy}, I_{zz}, __ or *), with the current model returning greater differences for the high BMI group in all cases. Similar to above, 11 of the 18 significant inertia differences detected across all subjects (Table 19, Table 20, Table 21, *overall*) could be attributed to a difference in high, but not normal BMI participants. Additionally, the effect of body mass, via either between (__) or within (*) comparisons (Table 19, Table 20, Table 21, *high BMI*), revealed a further 16 differences in inertia estimates that were not apparent in overall comparisons.

Far fewer differences in BSIPs in normal BMI reached significance, with only 19 of 75 comparisons reaching significance (Table 19, Table 20, Table 21, *normal BMI,* *), albeit with a small sample. In particular, the MRI-based BSIP estimates returned similar values for normal BMI participants as the current subject-specific approach, particularly for the upper and middle trunk segments, and about the somersault (X) axis (Table 19, Table 20, *Pearsall*). The two estimates derived from gamma scanning provided similar estimates for mass across segments, but variable differences in inertia, with the equations based on body mass and height providing smaller differences in the upper trunk (Table 19, *Zatsiorsky*), and those derived from segment length less in the middle trunk (Table 20, *De Leva*). Interestingly, the inclusion of a model based on simple geometric assumptions provided adequate estimates for the normal BMI participants (only 3 of 15 significant differences), but was not sufficient to account for the effect of BMI, with 12 of 15 estimates of BSIPs in this model influenced by body mass (Table 19, Table 20, Table 21, *Hanavan*, High BMI *).

3.3. Relationship between body mass and inertial properties of the trunk

The relationship between body mass and BSIPs could be fit by linear regression, significant in 19 of 24 cases ($p < .05$) in this small sample (Figure 5). The slope was > 1 in all cases, indicating a larger proportional increase in BSIPs for each unit increase in body mass, a finding consistent with the contributions of mass (linear) and mass distribution (squared) to underlying parameters. The use of logarithmic scaling resulted in modest improvements to variability (r^2) compared to a linear scale (not shown).

Slopes were not uniform across segments, indicating that body-mass related increases in BSIPs are specific to different body segments. For instance, slopes were greatest for the middle trunk segment, which parallels (and is dependent upon) the increased accumulation of trunk mass in this segment as body mass increased (see Table 11). The negative slope for the CM_Z of the lower segment reflects a more proximal CM_{loc} as body mass increased, consistent with the segmentation plane of the middle and lower segments (umbilicus), which left a portion of the abdominal fat mass in the superior aspect of the lower trunk segment.

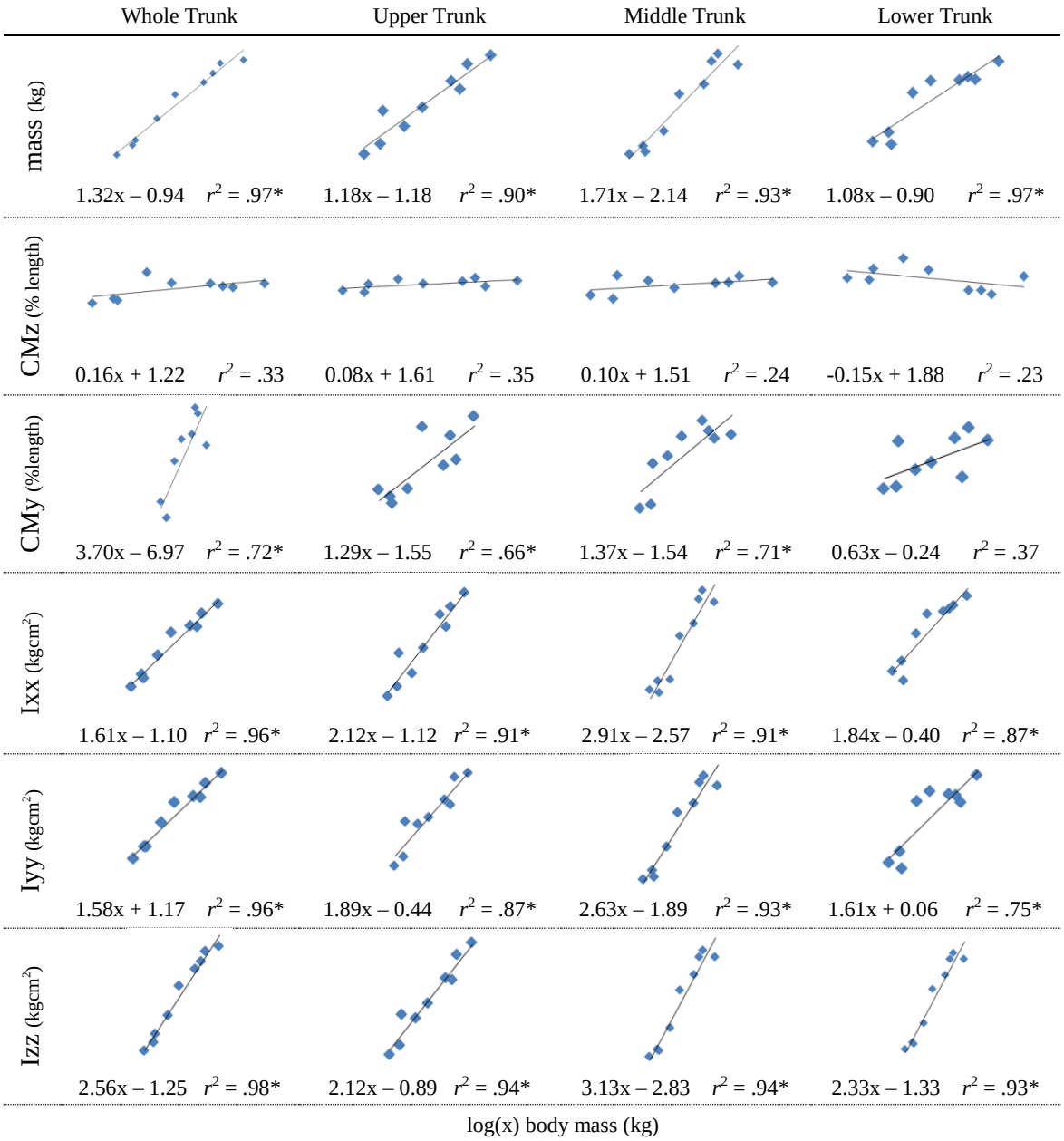


Figure 5. Relationship between body mass and segment inertial parameters, pearson correlation. Equation for a best-fit linear regression line and effect size (r^2) are provided. All variables are log-transformed.

4. THE EFFECT OF BMI ON LUMBAR SPINE LOAD

4.1. Static conditions

4.1.1. Standing

As expected, a high BMI was associated with much larger estimates of torque (95%CI: 7.8, 17.8 high/norm) and force (95% CI: 1.60, 3.26 high/norm) about L5/S1 (Table 22, *Torque, Force*), effects that can be attributed to both the increase in trunk mass and anterior-shifted CM_{loc} (Table 9). The increase in back extensor ES muscle force necessary to counter the flexion torque (M = 260.9, SE = 56.8 N, Table 22, *erector spinae*) accounted for more than half of the BMI-dependent increase in compressive force (M = 496.3, SE = 75.9, Table 22, *total*).

Table 22. Torque (Nm) and force (N) acting on L5/S1 during standing for normal and high BMI individuals. Differences are based upon height-matched pairs.

	Torque		Force – Normal			Force – Tangential
	Head & Trunk	Upper Extremity	Body Segments	Erector Spinae	Total	Total
normal M (SD)	2.7 (2.4)	--	347.9 (36.2)	38.0 (34.1)	385.9 (68.2)	291.1 (30.4)
high, M (SD)	19.8 (8.2)	--	563.8 (61.8)	282.3 (116.5)	846.1 (142.3)	473.0 (51.9)
difference, M (SE)*	18.3 (1.8)	--	235.4 (31.8)	260.9 (56.8)	496.3 (75.9)	197.5 (26.2)
high/norm, M (SE)*	12.81 (1.8)	--	1.72 (0.1)	12.81 (1.8)	2.43 (0.3)	1.72 (0.1)

*all differences and high/norm significant at $p < .01$.
t-critical value, df(4) = 2.78 for $p < .05$, two-sided

4.1.2. Load carrying

The addition of an external load (18 kg box) reduced the relative BMI-dependent increase to torque (95%CI: 1.16, 1.72 high/norm) and force (95%CI: 1.18, 1.74 high/norm) compared to standing, but resulted in much larger increases in absolute magnitudes (Table 23, *torque,* total). In spite of equivalent external load masses, the high BMI model predicted an increase in torque arising from box weight (M = 8.8, SE = 1.6 Nm, Table 23, *torque,* box) and upper extremities holding load (M = 8.9, SE = 2.1 Nm, Table 23, *torque,* upper). In the case of the external load, this arose exclusively due to a difference in box moment arms between groups (+4.8 cm in high

BMI group, see *Influence of body mass on lifting strategy*). Although a similar moment arm difference was predicted for the upper extremity (M = 4.5, SD = 2.1 cm), the difference in segment masses between high and normal BMI participants (M = 1.6 SD = 0.2 high/norm) accounted for more of the torque than the relative difference in moment arms (M = 1.2, SD = 0.2 high/norm).

Table 23. Torque (Nm) and force (N) acting on L5/S1 during load carrying for normal and high BMI individuals. Differences are based upon height-matched pairs.

	Torque				Force – Normal			Force – Tangential
	Head & Trunk	Upper Extremity	Box	Total	Weights	Erector Spinae	Total	Total
normal, M (SD)	2.7(2.4)	10.4(1.2)	71.2(1.8)	84.3(3.3)	489.5(36.2)	1203.9(47.3)	1693.4(82.6)	410.7(30.4)
high, M (SD)	19.8(8.2)	18.7(4.8)	80.0(3.6)	118.4(7.7)	705.5(61.8)	1691.6(109.9)	2397.0(155.6)	591.9(51.9)
difference, M (SE)*	18.3(1.8)	8.9(2.1)	8.8(1.6)	35.9(3.8)	235.4(31.3)	513.6(54.9)	749.1(80.1)	233.0(98.7)
high/norm, M (SE)*	12.8(1.9)	1.90(0.2)	1.12(0.1)	1.44(0.1)	1.50(0.1)	1.44(0.1)	1.46(0.1)	1.50(0.1)

*all differences and high/norm significant at $p < .01$.
t-critical value, df(4) = 2.78 for $p < .05$, two-sided

A model in which the moment of external load was minimized (based upon trunk dimensions) reduced the net torque for both the normal (M = 73.1, SD = 4.3 Nm) and high BMI groups (M = 113.5, SD = 10.7 Nm), but exacerbated the differences in absolute (M = 42.8, SE = 5.5 Nm) and relative (M = 1.61, SE = 0.1 high/norm) loading between groups.

4.1.3. Bending

As expected, flexion of the trunk to 45 degrees (as in lift initiation) further increased the absolute difference in static torque (95% CI: 69.0, 114.6 Nm) and forces (95% CI: 1053.4, 1780.0 N) between groups (Table 24, *torque, force*, total). Similar to the standing and load carrying conditions, the relative difference in net torque (95% CI: 1.24, 1.80 high/norm) could not be

predicted from the increased weight of body segments (i.e. the relative difference in segment weights was 1.72, while the difference in torque was 1.52).

Table 24. Static torque (Nm) and force (N) acting on L5/S1 during lifting for normal and high BMI individuals. Differences are based upon height-matched pairs.

	Torque				Forces – Normal			Force – Tangential
	Head & Trunk	Upper Extremity	Box	Total	Weights	Erector Spinae	Total	Total
normal, M (SD)	59.8(7.6)	21.9(3.2)	100.5(2.7)	182.2(12.6)	155.4(16.2)	2602.2(180.2)	2820.8(193.7)	600.5(44.4)
high, M (SD)	99.3(14.5)	45.6(8.0)	122.4(3.2)	267.3(23.1)	251.8(27.6)	3818.6(330.5)	4133.6(357.2)	865.3(75.8)
difference, M (SE)	43.0(6.4)	25.5(3.3)	23.3(1.1)	91.8(8.2)	105.1(13.8)	1311.5(117.2)	1416.7(130.7)	288.8(38.6)
high/norm, M (SE)	1.77(0.2)	2.3(0.2)	1.24(0.1)	1.52(0.1)	1.72(0.1)	1.52(0.1)	1.52(0.1)	1.50(0.1)

*all differences and high/norm significant at $p < .01$.
t-critical value, df(4) = 2.78 for $p < .05$, two-sided

A model that minimized the moment arm of the external load reduced the net torque in both normal (M = 155.4, SD = 13.8 Nm) and high BMI participants (M = 208.6, SD = 20.6 Nm). Unlike the previous conditions, this assumption also reduced the absolute (M = 60.1, SE = 7.2 Nm) and relative (M = 1.41, SE = 0.1 high/norm) differences between groups.

4.2. Dynamic conditions

4.2.1. Dynamic torques

The absolute difference in dynamic (inertial) torques between high and normal BMI participants was approximately 1/3 of the difference in static load (95% CI: 23.9, 53.9 Nm) (Table 25, *torque*, constant), however accounting for (potential) effect of BMI on trunk acceleration[23] resulted in a substantial increase (95%CI: 79.8, 132.0 Nm, Table 25, *torque*, differential). Similar to static conditions, the relative difference in dynamic torque for the constant (95% CI: 1.19, 1.75 high/norm) or differential acceleration models (95% CI: 1.98, 2.54 high/norm) was not proportional to the differences in body mass (M = 1.52, Table 7) or inertia (M = 2.10, Table 9).

Table 25. Dynamic torque (Nm) and force (N) acting on L5/S1 during lifting for normal and high BMI individuals. Differences are based upon height-matched pairs. Angular acceleration (rad/s^2) was modeled as equivalent (constant) and differential between groups.

	Angular Acceleration		Torque		Forces – Normal	
	Constant	Differential	Constant	Differential	Constant	Differential
normal BMI, M (SD)	9.6	9.6**	86.3 (6.4)	86.3 (6.4)	1233.2 (91.6)	1233.2 (91.6)
high BMI, M (SD)	9.6	14.8**	122.1 (14.9)	188.7 (22.9)	1744.2 (212.5)	2695.6 (328.5)
*difference, M (SE)	--	5.4	38.9 (5.4)	105.5 (9.4)	555.1 (78.3)	1506.5 (134.8)
*high/norm, M (SE)	--	1.48	1.47 (0.1)	2.26 (0.1)	1.47 (0.1)	2.26 (0.1)

*all differences and high/norm significant at $p < .01$.
**from Xiang et al 2010
t-critical value, df(4) = 2.78 for $p < .05$, two-sided

A model minimizing inertia (radii of gyration) of upper limb and external load resulted in lower estimates of net torque for both normal (M = 72.3, SD = 6.4 Nm) and high BMI (M = 117.0, SD = 16.4 Nm) participants, but increased the relative difference between groups (M = 1.68, SE = 0.08 high/norm).

4.2.2. Lifting - complete model

The additive contributions of static and dynamic (peak) torques during lifting was a mean (SE) 1.76 (0.1) times greater in the high, compared to normal, BMI participants (Figure 6, *net torque*, difference), corresponding to mean (SE) absolute differences of 197.3 (16.8) Nm for torque (95% CI: 150.8, 243.7 Nm) and 2923.2 (253.0) N for normal force (95% CI: 2219.6, 3626.8 N).

Static torques were the primary contributions to net torque in both normal and high BMI participants (Figure 6, *normal BMI, high BMI*, static torque). Static torques made a substantially greater contribution to net torque for normal BMI (M = 67.9, SE = 2.5%), compared to high BMI participants (M = 58.7, SE = 5.8%), $t(7) = 13.2$, $p < .001$. The contribution to static torque from body segments was greater in high BMI participants (M = 54.0, SE = 1.2%) compared to normal BMI (M = 44.7, SE = 1.4%), $t(7) = 5.00$, $p < .01$ (Figure 6, *normal BMI, high BMI*, body segments). As a comparison, in the load carrying model (Table 23) torques from

segment weight accounted for a mean (SE) 32.5 (3.8)% of static torque in high BMI participants and only 15.5 (0.9)% in the normal BMI model. For dynamic torques, body segments accounted for a mean (SE) 52.2 (1.0)% and 59.8 (1.8)% of dynamic torque in normal and high BMI participants, respectively, $t(7) = 3.40$, $p < .05$. Torques from body segments accounted for the majority of both static and dynamic torque in high, but not normal BMI participants (Figure 6, 'crossed lines').

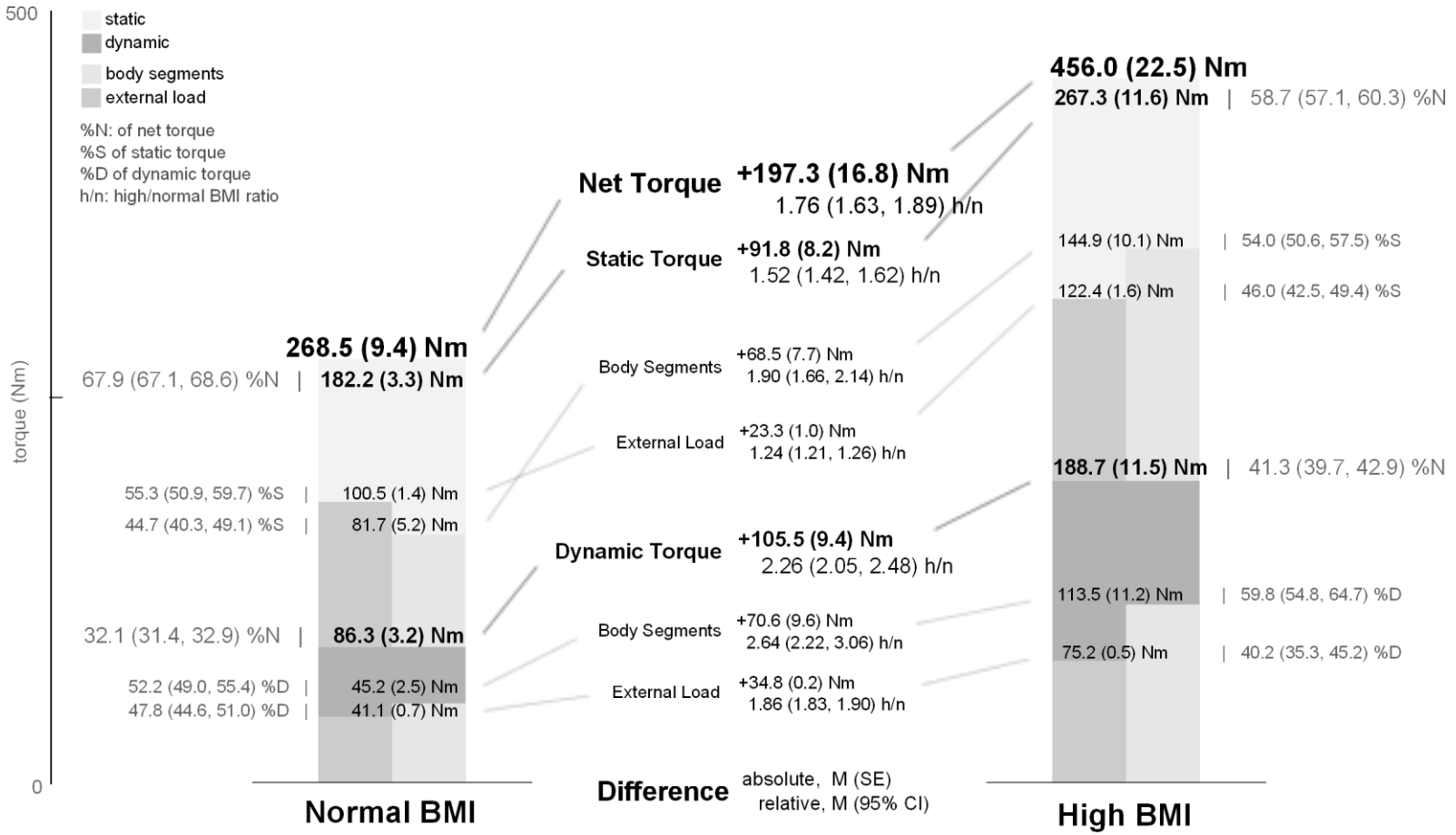


Figure 6. Torques acting about L5/S1 in normal and high BMI participants during a sagittal-plane lift (18.5 kg, floor to table height). Shown are the absolute (M, SE) estimates (black text) and relative contributions (M, 95%CI, grey text). Contributions are provided for static ( ) and dynamic torques ( ), as a percentage of net torque (%N), and also for torques from body segments ( ) and external load ( ), as a percentage of static (%S) and dynamic torques (%D). The difference between high and normal BMI participants is provided in the centre column, as absolute values (Nm) and relative difference (h/n).

A model assuming equivalent acceleration between groups (and minimal radii of gyration/moment arms) reduced the mean (SD) torque estimates to 227.8 (20.2) and 325.6 (36.2) Nm for the normal and high BMI groups, respectively. These assumptions reduced the relative difference to a mean (SE) of 1.50 (0.1) high/norm, a value similar to the difference in body mass (M = 1.54, Table 7). However, this was well below the difference in body segments parameters incorporated in the model (i.e. trunk, upper extremity: M = 1.62 – 2.17, Table 8), and thus arose coincidentally. Estimates of normal force in this model remained substantially greater in the high BMI group, with mean (SD) absolute and relative differences of 2923.2 (253.1) N and 1.75 (0.1)%, respectively.

As a case example for the effect of existing BSIP equations, use of the de Leva model to derive loading parameters during the standing position resulted in an estimated net torque difference that was 14.2% lower than the current model (M = 11.0, SD = 2.9 high/norm), with a similar reduction (18.9%) in compressive force (M = 2.0, SD = 0.4 high/norm). This effect accumulated in a complete model (static + dynamic), where the de Leva BSIP equations returned differences in torque (M = 1.27, SD = 0.3 high/norm) and compressive force (M = 1.26, SD = 0.3 high/norm) that were 28.1% and 27.9% lower than those obtained from individual-specific estimates. Similar effects would be anticipated for the other BSIP models.

5. SUMMARY OF FINDINGS

A simple, photogrammetric method for estimating individual-specific BSIPs was demonstrated. The approach provided accurate renderings of 3D surface geometry, with good resolution and reliability.

The inertial properties of trunk segments were substantially greater in high BMI participants, compared to height-matched, normal BMI subjects (up to 4x greater). This effect was due not only to elevated mass, but also a shift in mass distribution to the trunk segment. The effect of BMI on BSIPs was not proportional to body mass, and varied across body segments.

The estimates of individual-specific BSIPs in the current study for normal BMI participants were similar to existing predictive equations, but not for high BMI participants. Compared to individual-specific estimates for high BMI participants, a systematic under-estimation was demonstrated across all predictive models.

Back load was greater in high- compared to normal BMI participants. However, the difference varied across conditions and was not proportional to the increase in body mass or mass of principle body segments, precluding simple estimates for the effect of BMI on back load. Previous predictive BSIP models would likely have under-estimated these effects.

The relative contributions of static and dynamic torques, and those from body segments and external masses, differed between normal and high BMI participants.

These findings have implications for biomechanical investigations of individuals with high BMI (and other morphologically atypical populations) and for the design of ergonomic interventions that target back load.

DISCUSSION

Individual-specific body segment parameters and obesity

A photogrammetric method of acquiring subject-specific body segment parameters was demonstrated. Improvements to existing techniques[24,122] included absolute scaling, addition of surface features to the topography, inclusion of multiple camera views, more complex geometries, and corrections for lens distortion. These attributes were shown to be beneficial for acquiring 3D topography and estimating inertial parameters in a sample of participants with different body morphology (due to obesity) than the ubiquitous sample of healthy young males[24,118].

The error in dimensions of inert objects (1.05%) was consistent with existing estimates for similar parameters and techniques (e.g. error of 0.9 – 1.5% for CM_{loc})[122]. Unlike prior photogrammetric approaches, no evidence of a systematic over-estimation in body volume was apparent in derived estimates for body density[24], which were within expected ranges[26] – an improvement that cannot be attributed to (increased) resolution in the current study, which was similar to previously reported photogrammetric methods[24]. Recent photogrammetric approaches have also reported low error, but only for volume estimates of an isolated limb segment[122]. A comparison of whole body (rather than segmental) volume to other volumetric approaches (e.g. air displacement plesthysmography[226]) is needed to further establish the accuracy of photogrammetric techniques, particularly for populations with atypical or complex body morphology.

Mass distribution

Contrary to a simplistic proportional change, the volume (and therefore mass) changes attributable to a 54% (~35 kg) increase in body mass were not uniform across body segments. Similar findings have been reported for the trunk[25], however the current study is one of few examinations of multiple body segments in a sample of high BMI participants[115,116]. Consistent

with a central accumulation of body mass, the increases for most trunk segments exceeded that of the whole body. Independent of other mechanical factors (e.g. orientation, CM_{loc}, inertia) this would expose high BMI participants to a proportionally greater mechanical penalty than would be predicted by body mass alone. These estimates may aid in quantification of cumulative loading effects associated with degenerative lumbar spine changes in high BMI individuals[227]. The non-uniform, BMI-dependent distribution of body mass was also evident by a lower proportion of body mass in the lower extremity of high BMI participants, compared to height-matched normal BMI participants. In contrast to the trunk segment, this would attenuate predicted mechanical effects of BMI for lower extremity segments (e.g. swing phase of gait[228]). Lastly, segments with relatively less adipose tissue (foot, head, hand) were found to vary less with body mass[24]. Aside from mechanical influences, the ability to account for mass distribution amongst body segments is important for metabolic considerations[60] in high BMI individuals and also for special populations, such as individuals with spinal cord injury, where the effect of increased body/trunk mass is exacerbated by atrophy below the lesion site.

Inertial parameters

Similar to mass distribution, the obesity-related increase in BSIPs varied across body segments (largest for trunk) and parameters (largest for inertia), and were substantially different than the change in total body mass. Changes to radii of gyration, reflecting mass distribution within a segment (i.e. overall shape), were of lower magnitude than the change in body mass, and were most evident about a longitudinal (twist) axis[115]. In combination with segment mass, the relatively small increase in trunk radii of gyration (~24%) resulted in substantial increases to moments of inertia (up to 400%). These estimates are consistent with the range of trunk inertias established in previous publications[24,25,229] which had not yet controlled for the influence of segment length (i.e. the effects of mass and height on moments of inertia were not distinguished). Increased segment inertia would impact the ability to control angular motion of the trunk segment

(e.g. speeding up and slowing down), requiring increased muscle force and/or an alteration to kinematics (consistent with Newtonian mechanics, $\sum M = I\alpha$). These results, along with previous investigations[115,229], provide evidence that mass distribution within and between segments cannot be predicted from whole body mass, and that obesity has substantial, but non-proportional, effects on the BSIPs required for computation of static and dynamic loading parameters.

In addition to an obesity-related proximal shift in longitudinal CM_{loc} of most body segments[115], a BMI-dependent increase in the trunk anterior-posterior CM_{loc} was also demonstrated, consistent with the accumulation of adipose tissue in anterior abdomen. This effect had not been detected in previous investigations of high BMI individuals, perhaps due to the substantially larger body mass differences examined in the current study (35 kg), compared to previous (13 kg[115]), but may also be due to postural differences between studiess. A shift from a standing (current study) to supine posture (for MRI-based methods) can affect segment length[115,129] and may alter tissue distribution within a segment, which would attenuate anterior-posterior dimensions and increase the lateral. The difference between current and predicted moments of inertia about the somersault (bigger) and cartwheel (smaller) axes are consistent with such an effect, however the current sample was insufficient to test the significance of this trend. The influence of a supine posture for BSIP estimations has been acknowledged by other authors[24,115,223], but still remains to be directly tested. This effect would be expected to be greatest in high BMI individuals, as the quantity of malleable adipose tissue would be greater (e.g. panniculus adiposis). The moment of inertia and CM_{loc} differences demonstrated in this study support this phenomenon, warranting further investigation. Accurate CM_{loc}s are essential for estimations of static load during upright postures, particularly for the anterior-posterior direction in which the majority of ADLs occur.

Effect of BMI on predicted BSIPs

A significant effect of BMI on the difference in BSIPs acquired using individual-specific and predictive equations was apparent across nearly all models and segments, particularly the middle trunk. The BSIP estimates for normal BMI participants were similar to previously reported between-model variances[25,229], and were also within the range of actual BSIPs for similar populations[24]. In contrast, compared to the individual-specific method of the current study, predictive equations returned consistently lower BSIP estimates for high BMI participants. Further, the relatively good fit of the overall comparisons (all participants) demonstrate that low error can be achieved across a range of BMIs, in spite of larger error for specific individuals (e.g. high BMI). These findings are consistent with body mass-dependent effects reported for predicted versus actual whole body inertia[125]. The most obvious explanation are differences in the sample populations for two predictive models[117,218], which in this case occurred due to increased body mass, with a corresponding systematic underestimation of 15 – 30% demonstrated. No obvious improvements were apparent for the estimates based on simple geometric assumptions[119], nor the model derived from a larger range of body masses[25]. Although the geometric model[119] offered a rudimentary means of accounting for changes in morphology (body shape), the underlying equations for mass distribution (and density estimates) were also derived from normal BMI samples[129] which likely introduced similar under-estimations as the regression-based models.

The choice of simple predictive equations, which modeled BSIPs as constant functions of height/mass, would also be expected to contribute to the variation in estimates[25,229]. The differences in predicted BSIPs paralleled the differences in mass distribution between normal and high BMI participants, demonstrating the (probable) error arising from constant-function/linear predictive equations will increase as the mass distribution of the sample departs from that of the original population. Although the proportion of the population with elevated body mass is quite large (>60%)[60], it is unlikely a single predictive model will suffice for all overweight or obese

participants, as the morphology of obesity varies substantially across individuals (e.g. android vs gynoid somatotypes)[128]. Similar effects have been demonstrated for children[122], different ethnic groups[123], the elderly[229], and would also be anticipated for other populations (e.g. SCI, high lean body mass, amputees, etc). The effect of variation in mass distribution was also apparent within the low-BMI group, evident by the large increase to upper torso BSIPs in subject 8 (due to lean body mass), an effect not represented in predictive equations. These findings support the use of individual-specific anthropometrics, rather than predictive equations, in biomechanical studies involving high-BMI individuals and other morphologically-atypical populations. In fact, as methods of obtaining individual-specific BSIP estimates improve (e.g. in accuracy, ease of use), the need for predictive equations may become unnecessary.

Although estimates for the range of trunk BSIPs[25] and other segments have been published[229], few estimates with associated anthropometrics exist specific to high BMI participants[115,116], which somewhat limits comparisons. The distribution of body mass to the trunk in the current study (56%) is consistent with samples of similar age and BMI[115], but was slightly higher than estimates in older adults of similar BMI (49%)[116]. Similarly, Pearsall[25] included a sample with 5 participants of high body mass (>100kg) and BMIs (up to 39 kgm^2), but reported only maximum trunk mass – lower than here (56 vs 68 kg and 48 vs 56% body mass), a difference that appeared to be restricted to the upper and lower trunk segments. Lastly, trunk radii of gyration were lower than Matrangola and colleagues[115], but the difference was small (28% vs 33% of segment length). In light of the relatively small samples of high BMI participants in this and other studies, it is difficult to determine the extent these differences reflect normal variations in obesity-related morphology[128] and how much may be attributed to methodological differences between studies.

Limitations of individual-specific body segment parameters

The assumption of uniform density in volumetric techniques necessitates a centre of mass that is coincident with centre of volume, which may not be accurate if tissue density varies within a segment[24]. However, a strong relationship has been shown between volumetric, uniform-density models and tissue-specific models (MRI-derived, $r > .95$), along with a relatively low error for most body segments (4.7%) provided appropriate density estimates are used[223] (e.g. accounting for void space in upper trunk). Additionally, accounting for differences in mass distribution amongst segments will likely explain a larger proportion of variance (24%, based upon trunk masses of 46 – 58%BM) than the variation in individual segment densities (8%, based upon trunk densities of 0.89 - 0.97 g/cm^3)[223]. The aim of the density-correction in the current study was to partially adjust for BMI-related differences in segment density, however it is highly unlikely that density changes are proportional across all segments (as was modeled). Alternatively, the volumes could be scaled with constant density assumed.. Although this approach (scaling volumes) may be suitable for normal BMI individuals (from which the density estimates are derived[25]), it would result in unrealistic segment volumes for high BMI participants, as the volume would have to be reduced in order to fit a (higher) segment density derived from a normal BMI individual (i.e. higher proportion of lean mass to fat mass). Additionally, scaling densities would not take into account the normal variation in mean segment densities of normal BMI participants, an effect that can be partially detected using measured body/segment volume and total body mass. Another method of reducing density-related error is use of segment density profiles[220], which might actually be less relevant to high BMI populations, as body segments become more homogenous via increased proportions of fat mass[128]. Further understanding of BMI-dependent segment density changes are needed to improve these estimates.

It is also possible the values here represent the worst performance of predictive BSIPs, as more advanced multivariate equations are available that may improve accuracy across a wider

range of body morphologies[24,25]. However, certain predictors (e.g. subcutaneous fat thickness) are not feasible for participants with high amounts of adipose tissue and the simple predictive equations are still widely used, including for samples with high BMI[40]. The potential error in discriminating the participant from the image background is a methodological limitation specific to photogrammetry. This is typically minimized by using a well-lit, open space and minimal (or tight fitting) clothing, however these practices may contribute to discomfort in participants that are body-conscious. Also, as the overlap of body segment contours increases (due an increase in segment girth) the ability to discriminate segment boundaries is affected, an effect that was minimized using a wide stance with arms abducted. Additionally, the moments of inertia reported were assumed to be principle, but may not be, however this would tend to increase the reported values and corresponding effects[24].

Lastly, the effect of increased BMI on BSIPs could have been tested using alternate statistical approaches, and it is important to ensure the findings are robust to variations in statistical approaches. First, the approach of height-matching was selected in order to control for the influence of variations in segment lengths – to which obesity does not (likely) contribute – on BSIP differences; the moments of inertia about the somersault and cartwheel axes would be particularly sensitive to this effect.

Second, the decision to create a 'virtual subject' (#9) may have reduced independence/variation within the normal BMI comparison group (being a function of subject #7), however the BSIPs of the virtual subject were within the published values for similar individuals[25] and it is well-established that BSIPs can be scaled linearly for young, healthy male participants[118] making it highly likely these BSIPs are reflective of a normal sample. Additionally, the reduction in variability arising from a virtual subject is still substantially less than comparisons based upon normative values, which reflect only population means and not between-person variation. The BSIP differences were also tested using an independent t-test

between high and normal BMI groups (excluding subject #9), with only a small reduction in effect size and minimal change in significant differences (see Appendix – Study 1, Table 61.), reflecting that either methodology produces ostensibly the same results. Additionally, a portion of the reduction in effect size could be attributed to an near significant difference in height (rather than obesity) between high (M = 1.76, SD = 0.06 m) and normal BMI groups (M = 1.85, SD = 0.06), p = .06, which would have a tendency to increase inertia estimates for normal BMI participants, thus reducing the between group difference (and supporting the process of heigh-matching)(see table xx). .

Conclusion of individual-specific body segment parmeters

The increase in BSIPs attributable to a high BMI was quantified and shown to vary in magnitude (12 – 400%) across body segments and parameters. The non-uniform distribution of body mass across body segments impacts the ability of predictive equations to account for elevated body mass, with a systematic underestimation of BSIPs demonstrated for high BMI individuals. In part due to complex body morphologies (shape) and also to within-group anthropometric variations, the use of individual-specific methodologies for deriving BSIPs, rather than population-specific regression equations, was supported. Future research is needed to improve estimates of segment density, examine the effect of position (supine vs standing) and quantify BSIPs in other morphologically atypical populations (e.g. athletes, SCI, etc). Accurate BSIP estimates for obese individuals are relevant for biomechanical investigations examining the effect of increased body mass, and also for metabolic considerations that may be associated with the distribution of body volume or mass.

Effect of obesity on lumbar spine loading

A significant effect of BMI on lumbar spine loading was demonstrated across a variety of material handling scenarios, as anticipated[23]. More importantly, the increase to body mass resulted in non-proportional changes in loading, with differential effects across scenarios and mechanical variables (e.g. static vs dynamic; segments vs external mass). The mechanical influence specific to an abdominal fat mass was quantified and shown to arise from both direct (mass, CM) and indirect factors (interference, other limb segments). These results support the notion that lumbar spine loading will vary non-linearly with body mass[25,115], but can still be attributed to specific features of obesity (direct and indirect factors).

Effect of panniculus adiposis (static load during standing)

The mechanical consequences of a obesity during standing arose from a (trunk) mass-dependent change in compressive force (as expected) and also from an additional flexion torque imparted by the abdominal fat mass. Although these 'direct' factors are easily estimated with simple static calculations, neither the magnitude nor relative contributions can be predicted *a priori*. For instance, even the simple influence of trunk mass on compressive load cannot be modeled without appropriate BSIP estimates, as mass distribution to the trunk segment varies across a wide range (above,[25,116]). Additionally, the increased flexion torque (18Nm) associated with a 13 kg/m^2 change in BMI was attributable to an anteriorly-shifted trunk CM_{loc}, which had not been modeled previously, as population-specific CM_{loc} estimates did not exist until now (above). For participants with a 'normal' BMI (20 – 25 kg/m^2) this additional torque would be equivalent to a 6 kg mass held/placed on the anterior trunk, and parallels the increase in torque from segment weight during pregnancy[85]. Although the magnitude of these effects are below thresholds for elevated back injury risk[51], the cumulative load would certainly be greater in high BMI participants, contributing to increased degenerative changes[230] or perhaps neuromuscular adaptations (dependent upon dose)[68] . It is also possible that high BMI participants adopt

compensatory postural strategies in standing[85], which was accounted, but not specifically evaluated, by using a whole-trunk BSIP model. Additionally, high BMI individuals have been shown to adopt a more flexed posture during standing work[40], a change that would exacerbate, rather than counter, the flexion torque imparted by the abdominal fat mass.

The addition of an external load revealed 'indirect' consequences of increased body mass, which manifested as changes to the moment arms of the external load and involved body segments. The increased external load torque (+12%) arose exclusively due to physical interference of the abdominal fat mass, a relative difference that was even greater when a minimal box moment arm strategy was modeled (as occurs during long-duration lifting and safe-lifting advice)(*Influence of body mass on lifting strategy*)[51]. Likewise, the necessary changes in orientation of upper body segments contributed an additional 20% increase to moments of segment weight beyond the obvious differences in segment mass. BMI-dependent postural changes have been shown to occur during standing work involving the upper extremity[40], and without substantial change to work environment, cannot be altered by simple behavioural approaches (i.e. handling technique) as they are necessitated by the morphology of the participants. These effects would also lead to similar increases in loading about the shoulder, an area susceptible to degenerative musculoskeletal pathology[231]. Although high BMI participants report increased perceived exertions during static postures similar to certain workplace tasks (presumably due to elevated torques from limb segments)[62], relatively little is known about how these effects might manifest during actual material handling tasks. For instance, increased trunk flexion will increase static moments about the lumbar spine, but would tend to reduce static moments about the shoulder via a reduction in the moment arm of external loads and body segments about the shoulder

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Effect during lifting (dynamic loading)

The magnitudes of peak torque and force estimated for normal BMI participants during the lifting motion are consistent with those previously reported[42,232]. The task chosen reflected a typical posture encountered during activities of daily living (45 degrees of trunk flexion) and represented lift initiation, a period of relatively high inertial moments[42]. Excluding potential BMI-dependent movement strategy differences[23], the increase in torque across static and dynamic factors happened to be similar to the increase in body mass, however this arose coincidentally and did not reflect the actual increase in mass or inertia of involved segments. The substantial difference in torque and force reported for high BMI participants extend previous findings of BMI-dependent kinematic changes[23] and (no change in) external load[233], by providing a simple approximation of the magnitude of load attributable to each effect: a +200% increase in dynamic torque and 24% increase in static moment of external load (in spite of equivalent mass). Exposure to high lumbar loads is a well-established risk factor for back injury[7,51], and the magnitudes provided here for high BMI participants exceed these thresholds. The effects of body mass on prophylactic load guidelines[51] has not been considered, in spite of evidence of an association between obesity, back pain[68] and mechanical load[7].

Relative contributions of mechanical variables

Compared to dynamic factors, static load made a larger contribution to overall load for both normal and high BMI groups, which supports interventions aimed at reducing static load[51], however the contributing factors to static load were not uniform across participants. In high BMI participants, the moments of body segment weight accounted for the majority of static torque compared to moments of external load, while the inverse was shown for normal BMI participants. Although not unexpected, this indicates prophylactic reductions in weights lifted/handled will have differential effects on relative load reduction across individuals, and supports individual-specific material handling advice[146]. For instance, an increase in segment weight may contribute

to a reduction in the ability to discriminate external masses[170], the tendency to select larger external loads[23], or even increased confidence when lifting equivalent masses (*Influence of body mass on lifting strategy*). These factors do not account for the potential of neuromuscular adaptations (increased strength/endurance) in high BMI participants, however current evidence does not support this as a possibility[48] and it runs contrary to the typical etiology of obesity (i.e. physical inactivity). These individual-specific effects (e.g. BMI) may also contribute to the near-universal failure of prophylactic interventions aimed at altering movement technique[234], as the ability to alter posture (e.g. moment arm of external load) and influences on lifting technique differ across individuals. Obviously, reduction of body mass is supported not just for mechanical effects, but is not a feasible short-term solution, and so interventions incorporating BMI-dependent factors (e.g. moment arms) may be warranted.

Although the absolute magnitude of dynamic torques was dependent upon modeled movement strategy differences, body segments contributed a substantially greater proportion to dynamic torque in high BMI participants (independent of strategy), similar to static torques. If the BMI-dependent movement strategies modeled in the current study are applicable to other lifting tasks, modification of movement strategy may be indicated to reduce peak torque in high BMI participants. While the potential for risk reduction from modifications of dynamic torque is low compared to static torques (1/3)[42], it may be one of the simpler loading factors to alter, especially considering that the orientation of body segments is often constrained by task demands (e.g. initial height) or individual-specific factors (anthropometry). This is supported by the finding that the reduction in torque expected from lifting a lighter mass is attenuated by changes to movement strategy (increased acceleration)[44]. It contrast, is also possible that high acceleration strategies are not necessarily detrimental in certain circumstances (multi-segmental motion, light loads[235]), and so more detailed dynamical models of lifting in high BMI participants are required.

Effect of BSIP estimates

Consistent with the variances in BSIP estimates, the effect of extrapolating existing BSIP predictive equations to a high-BMI mechanical model was shown to attenuate the effect of obesity on lumbar spine loading, by way of a substantial under-estimation of the obesity-related increase in torque and force. However, similar to the effect of obesity on actual BSIP estimates (above,[229]) this effect is unlikely to be consistent across BSIP models, body segments and tasks. For instance, the head/neck segment is likely impacted less by increased body mass (vs the trunk)(above,[24]), yet can contribute a significant amount to mechanical load during trunk flexion, due to a comparatively large moment arm. It is possible this effect (reduced or variable error across segments) might offset under-estimations for the trunk, and similar to the relationship between body mass and mechanical load, may coincidentally result in reasonable estimates (in spite of inaccuracies across constituent segments). Although a systematic sensitivity analysis was not undertaken across all models and conditions, illustration of under-estimation in a single (commonly used) BSIP model is sufficient to support the findings particularly since the underlying mechanism was established (BSIP variances). Systematic, body-mass dependent under-estimations would not likely impact within-group comparisons in high BMI samples, but are certainly relevant for between-group comparisons of high- and normal-BMI samples. In these circumstances, the use of BMI-specific BSIP estimates would be expected to further increase effect size. Lastly, although the accuracy and relevance of estimates for absolute load about the lumbar spine are complex, current absolute estimates are likely under-representative of the actual mechanical impact of obesity on the lumbar spine.

Limitations of effect of obesity on lumbar spine loading

While the use of a fixed trunk segment, derived from a whole trunk 3D scan was ideal for controlling the influence of postural effects in static positions (standing), it would not be sensitive to orientation changes that might occur during trunk flexion (e.g. during bending/lifting)[40]

(*Influence of body mass on lifting strategy).* However, this source of variance is likely lower than error in BSIPs (above) or perhaps even the redistribution of adipose tissue within a segment due to postural changes. The mechanical model could be further improved by inclusion of intra-abdominal pressure, muscle activation, and muscle moment arms[123], however there is no evidence (or rationale) these factors vary systematically with body mass. The large increases in obesity-related static torques arose from relatively small moment arm increases (4-5 cm), and while this is within the margin of error for techniques used here[122] and is consistent with expected morphological changes, more complex multi-segmental models may further refine this estimate. Lastly, the estimation of lumbar joint centre was likely the most conservative available for estimating the mechanical effect of obesity, which assumed a uniform accumulation of adipose tissue around the abdomen, as opposed to the disproportionate anterior distribution often apparent in central adiposity.

An additional benefit of the photogrammetric techniques used in the current study is as a means to investigate dynamic changes to body segment parameters across positions, and potentially during movement. While the influence of tissue redistribution on BSIP estimates between supine (photogrammetry) and standing (medical imaging) conditions was considered above, a similar, or perhaps even greater effect is plausible between the start and end positions of many lifting tasks. For instance bending forward to initiate a lift may result in a significant anterior displacement of abdominal fat mass, which would further increase the BMI-related torques – an effect not accounted for by assumptions of constant BSIPs during a task/movement. It is also possible that tissue distribution changes during a movement, for example due to the inertial properties of large quantities of fat mass not rigidly attached to the axial skeleton may present an additional challenge to high BMI individuals, particularly during high-acceleration movements (e.g. as a perturbation to the body centre of mass following deceleration of the axial skeletal). Lastly, increased trunk girth may even offer a means of minimizing the load imparted

by the torso mass, if the abdominal mass contacts other body segments (i.e. the anterior trunk resting on the thighs during a deep squat motion). Recent advances in cine-photogrammetry (e.g. Microsoft Kinect), which obtain 3D imaging at frequencies as high as 30 Hz (640 x 480 pixels), offer a method to quantify the potential changes in body segment parameters during the actual execution of a lifting task, rather than simply standing or supine positions.

Conclusion of effect of obesity on lumbar spine loading

This study adds to evidence that high BMI individuals experience differential loading about the lumbar spine, beyond that which might be predicted based upon whole body mass, or even trunk mass. In the case of a materials handling task, the increase in load was shown to arise from two 'direct' factors that could not be predicted from existing literature – a non-proportional increase in trunk mass and the effect of anterior trunk mass on the CM_{loc} (i.e. moment arm). The added influence of 'indirect' factors arising from physical obstruction and necessary changes in orientation of upper body limb segments were also shown to contribute to obesity-related mechanical load. The preliminary estimations for obesity-related mechanical load provided here necessitate further examination, particularly for the influence of obesity on other determinants of mechanical load (e.g. postural compensations, muscle activation, intra-abdominal pressure). Estimates of lumbar spine load in high BMI individuals are relevant for injury-prevention strategies and etiological considerations of degenerative back pathologies.

Overall Conclusions

The aim of this study was to quantify the mechanical effect of obesity on lumbar spine loading. This required obtaining estimates for the effect of increased BMI on body segment parameters (objective 1) – as existing predictive equations were shown to under-estimate most parameters (objective 2) – prior to deriving loading estimates for standing, carrying and lifting tasks (objective 3). The results support the use of individual-specific BSIPs for samples of high-BMI participants, in order to account for variations in BSIPs that are not easily estimated by

mass- or BMI-based algorithms. This approach was used to confirm obesity-specific BSIP changes from prior studies (e.g. proximal shift in longitundal CM_{loc}[115]) and provided additional BSIP estimates requisite for biomechanical investigations involving individuals with a central accumulation of body mass (e.g. anterior-posterior CM_{loc} of trunk). The corresponding magnitude and determinants of BMI-dependent back load during lifting were quantified and shown to be attributable to direct (inertial parameters) and indirect factors (physical intereference). In addition to materials handling, these findings may be relevant to other areas of study (e.g. sport/ recreation), however further study is required, as the biomechanical effects of obesity are dependent not only on mass distribution, but also the movement patterns used for specific tasks.

STUDY 2. THE INFLUENCE OF BODY MASS ON LIFTING STRATEGY DURING REPETITIVE, FIXED-PACE LIFTING

METHODS

1. Participants

Two groups of participants were recruited, one with a normal body mass index (< 25 kg/m^2, n = 10) and the other with a high body mass index (>28 kg/m^2, n = 13). Participants were predominantly male (n = 20), and distributed evenly between groups (n=2 and n=1 female participants in the normal and high BMI groups, respectively). Participant characteristics are described in Table 26, differences in age and height were not significant. Ethical approval was obtained from Bannatyne Campus Research Ethics Board, University of Manitoba (HREB#: H2010:408).

Table 26. Participant characteristics.

	age (yrs)	height (cm)	mass (kg)**	BMI (kg/m^2)**	WC* (cm)**	W:H$^+$ **
normal BMI (M, SD)	35.6 (10.1)	180.2 (5.5)	75.7 (6.0)	23.3 (2.1)	80.5 (6.2)	0.84 (0.08)
high BMI (M, SD)	38.8 (10.9)	176.1 (5.4)	99.2 (12.7)	32.3 (3.4)	108.7 (9.4)	0.96 (0.06)

*WC: waist circumference (umbilicus)
+W:H: waist:hip ratio calculated as ratio of circumferences of waist and hips (largest circumference)
**differences in mass, BMI, WC and waist:hip ratio significant, p < .001

2. Protocol

Participants performed one hour of lifting, consisting of three, 20-minute sets (5 min inter-set rest). A box (37.5 x 36 x 25 cm; bilateral handles, 7 cm from top) was lifted from floor level to a shelf positioned at a height of 76 cm (table height). The box was returned to the initial position (centroid of box 30 cm from front of shelf) following each lift by a research assistant. The lifting pace was set at 1 lift every 15 seconds (4 lifts/min), cued by an audible tone (250 msec @ 1000Hz). Each set consisted of 80 lifts (20 mins @ 4 lifts/min).

No specific instructions were provided regarding lifting technique or position of limb segments, and subjects were instructed lift in a manner that "felt most natural or they would normally use". However, participants initiated the lift facing towards the shelf and grasped the box with both hands, constraining the lifting motion to sagittal plane. A series of 6 – 10 lifts were performed for familiarization prior to the start of the sets.

3. Lifting Strategy

The term 'lifting strategy' was chosen to represent the parameters an individual manipulates during a lifting task. Although no clear definition or consistent use of 'lifting strategy' exists in the literature, lifting strategy can encompass: a) kinematics of body segments/external load[39], b) frequency (lifts/min) and pacing (fixed or irregular) of individual lifts[44], c) characteristics of the load lifted (e.g. mass[180]). It is common practice to investigate the effect of an independent change on outcomes in one or two of these categories, while controlling the influence of the others[236].

For this investigation, frequency (4 lifts/min) and pace (fixed) were controlled, and 'lifting strategy' was characterized by: 1) lifting technique (orientation of body segments at lift initiation); 2) kinematics of the trunk and box (acceleration and derived measures); 3) the mass an individual selects to lift.

3.1. Self-selected mass

The box mass in the first two sets was self-selected by each participant to an amount they perceived being able to lift for one hour[237]. Masses of 1.0, 2.0, 5.0 and 10.0 kg increments were provided for adjustments; the 1.0 and 2.0 kg masses were bags of sugar (density: 1.59 g/cm^3) and the 5 and 10 kg masses consisted of lead shot (density: 11.4 g/cm^3). A standardized written script

regarding mass selection criteria was provided (Appendix: *Lifting Instructions*), and participants were encouraged to make as many adjustments as needed. Initial box mass in the first set (5 kg) was varied in the second set (12.5 kg) to minimize recall of adjustment procedure. The box mass for the third set was the average of the two. The final mass lifted in each set was recorded.

3.2 Lifting technique

Lifting technique was quantified from digital video, using a measure of postural index (PI) – the ratio of displacements causing trunk extension (knee) to those causing trunk flexion (sum of hip and ankle), PI = knee / (hip + ankle)[34]. The PI provides a single, static outcome reflecting aggregate lifting technique, independent of total lift displacement. A value near 0.1 indicates a more hip-dominant (stoop) technique, while a value > .80 is associated with a knee-dominant (squat) technique.

3.2.1. Video

Two digital cameras (1280 x 720 pixels, 30 fps, Casio EX-100, Casio USA) were aligned such that the image planes were parallel to the anterior-posterior and medial-lateral planes of the lifting shelf, corresponding approximately to the sagittal and frontal planes of the lifting motion. An alignment rig consisting of two orthogonally mounted mirrors, with a marker positioned at centre, was used to align the image plane of each camera parallel to the respective plane of the shelf. Four high-contrast markers were affixed in a known orientation to the lateral and front surfaces of the box, used for image-scaling.

Lifting motions were assumed to be constrained to the sagittal plane (verified from frontal-plane camera) and error in joint angle estimation due to out of plane motion was considered negligible. The variability attributable to this (potential) measurement error was estimated to be well below the hypothesized effect sizes: the difference in hip angle between

stoop and squat-style techniques was anticipated to be 40 degrees and the error from off-axes rotation was estimated to be ~4 degrees for a 15 degree rotation (proportional to the cosine of the off-axis angle). This error is consistent with existing estimates of error due to off-axis rotation in 2D analysis of kinematics and kinetics[238].

3.2.2. Processing

Video was imported to an open-source video-analysis program (Kinovea Motion Tuner, GNU General Public License, Free Software Foundation, Inc). Individual video frames of the initiation and termination of 3 successive lifts, at the beginning and end of each set, were extracted (.JPEG images). Additionally, the 3 lifts immediately following the period of mass-adjustment in the first two sets were extracted (i.e. beginning of constant-weight lifting), for a total of 48 video images (start and end of 24 lifts) for each subject.

Images were subsequently imported into a numerical computation program (GNU Octave) for extraction of joint angles and other measures, where each participant was modeled as a 5-link, rigid body with segments corresponding to the shank, thigh, lower torso, upper torso and upper arm. The end-points of each segment were taken as the x-y coordinates of the respective joint centres and joint angles were calculated using the cosine law (Table 27). Pixel coordinates were converted to metres using the scale affixed to the box. Moment arms of the external load were defined as the perpendicular distance between the weight vector (centroid of the box, parallel to gravity) and the appropriate joint axis (e.g. hip and lateral malleolus). The centroid of box was used as the CM based upon the assumption of uniform mass distribution within box.

Table 27. Orientation outcomes extracted from video frames during a sagittal plane lifting task (15 kg, floor to table height). Angles were derived based upon visually determined joint centres or bony landmarks and the centroid of the box was computed from actual dimensions.

joint angle	description (proximal – middle – distal endpoint)
knee	hip – knee – lateral malleolus
trunk	knee – hip – C7 spinous process
torso	hip – T7 spinous process – C7
hip	knee – hip – T7 spinous process
glenohumeral, (start + upright)	T7 spinous process – glenohumeral – elbow
distance (start)	box centroid – lateral malleolus (perpendicular distance)
moment arm (upright)	box centroid – PSIS (perpendicular distance)

3.3. Kinematics

3.3.1. Accelerometry during lifting motions

A system of wireless, micro electro-mechanical accelerometers was used (47 g, 58 x 43 x 26 mm, resolution 9mg, range +/- 20g, G-link mXRS, Mictrostrain USA) to acquire acceleration of the trunk and box. One accelerometer was mounted to the distal trunk, affixed at the manubrium using adhesive tape, and a second to the front of the box (Figure 7). The accelerometers were positioned such that the axes were manually aligned to a consistent local coordinate system of the trunk and box – the x-, y- and z- axes (channels 1,2,3) represented the anterior-posterior, medial-lateral and superior-inferior directions, respectively.

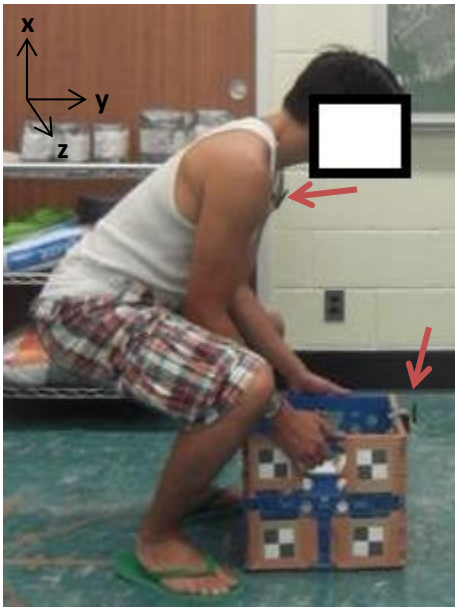


Figure 7. Accelerometer placement during lifting task - two wireless, tri-axial accelerometers were used: one attached to the superior sternum and one to the anterior box (arrows). The axes of the accelerometers (indicated) were manually aligned to the principal axes of the trunk and box.

The signal from an accelerometer is proportional to the net force acting upon the unit's proof mass – during static conditions the net force reflects the gravitational acceleration only (i.e. the weight of the mass), with the output dependent upon the component of the weight vector acting parallel to the sensitive axis of the accelerometer (i.e. varies with the COS of the angle between gravity and the sensitive axis). In an orthogonally-mounted, triaxial accelerometer the magnitude of the resultant force vector [$\sqrt{(\text{axis one}^2 + \text{axis two}^2 + \text{axis three}^2)}$] from all 3 axes will always be 1g under static conditions (assuming correct calibration). Periods where external forces are applied to the accelerometer (i.e. dynamic conditions) are easily detected as deviations in the magnitude of the resultant vector from 1g.

A predictable pattern of acceleration has been shown to arise in the dynamic component of the acceleration signal during a variety of different human motions, including lifting[39]. The initial starting acceleration (to speed up a body segment) and the second slowing acceleration (to

slow down the segment) have been referred to as the P1 and P2 phases, respectively[239], and would represent the initial lowering-phase of a lift. An object or limb segment that moves back to the original starting position (the actual lifting-motion) will undergo (at minimum) two additional phases of acceleration – the P3 and P4 phases, speeding up and slowing down accelerations, respectively (Figure 8). For a continuous motion (i.e. no pause between the lowering and raising phases), the P2 and P3 phases typically manifest as one distinct peak (rather than two individual peaks).

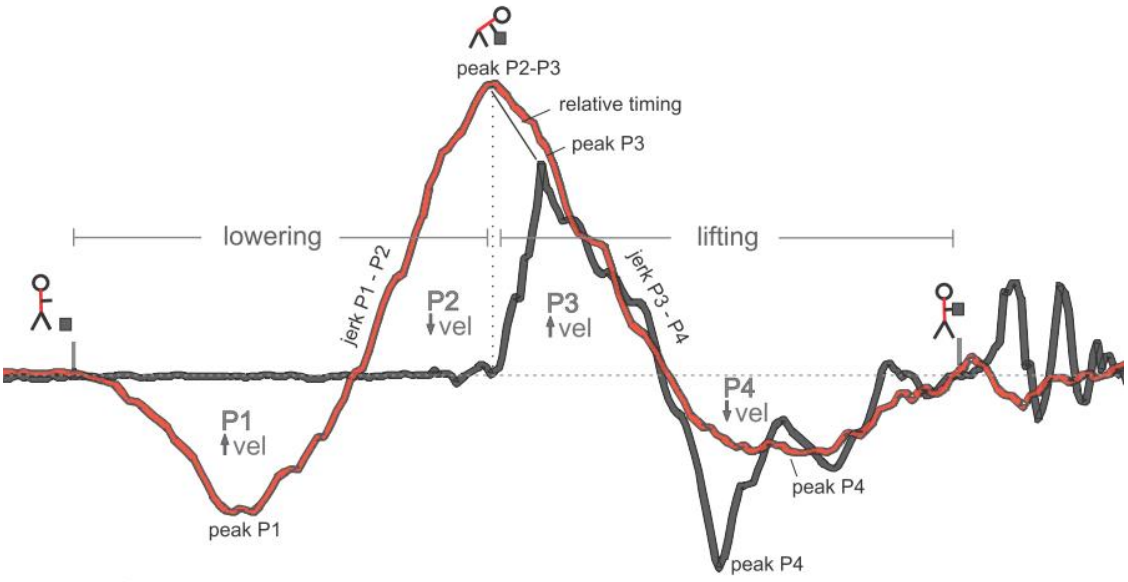


Figure 8. Trunk and box resultant acceleration during lifting. The trunk experiences 4 phases of acceleration, two in each half of the task (lowering: P1 + P2; lifting: P3 + P4), while the box experiences 2 phases (P3 + P4). The effect of the acceleration on trunk velocity is indicated (i.e. speeding up or slowing down) as are kinematic parameters (i.e. velocity, jerk). This signal was derived from a single repetition of a relatively faster lift (pilot data), compared to the time normalized (experimental) data presented in figure 13, and does not display the characteristic inflection point between P2 and P3 observed during slower, heavier mass lifts.

3.3.2. Accelerometry Derived Measures

The signal was sampled at 256 Hz and transmitted wirelessly and time-synchronized (+/- 32 μsec) to a personal computer.

A series of 3 mechanical-visual artifacts (hand-taps) applied to the accelerometers at the beginning of each set were used to time-synchronize the accelerometers and video. The accelerometers were factory-calibrated, converting the voltage changes to output in gravitational acceleration units (g's), and the calibration of the accelerometers was confirmed prior to each session by rotating each axis 180 degrees relative to gravity (e.g. +1g, 0g, -1g). The gravitational acceleration was taken as 9.81 m/s^2.

Accelerometer output was processed using programs written for GNU Octave (GNU General Public License, Free Software Foundation, Inc). Accelerometer signals (3 channels; trunk + box) were imported in .CSV format and digitally filtered to remove high frequency artifacts (recursive, low pass, 4^{th} order Butterworth). The cutoff frequency (7.5 Hz) was refined by visual comparison of filtered and raw signal (negligible attenuation of peak values). The resultant was computed from the filtered signals ($\sqrt{(x_1^2+x_2^2+x_3^2)}$). Accelerations of the P1 and P4 phases were < 1g for all subjects and repetitions, validating the use of the resultant to identify onset of acceleration phases[5].

The initiation of each (acceleration) phase was determined as the first sustained (+150 msec) period of acceleration below (P1, P4) or above (P2,P3) the baseline, and were confirmed by visual inspection. Duration was defined as the time between the initiations of two successive phases, the peak as the local maximum (P2,P3) or minimum (P1,P4), and rate of change of acceleration (jerk) was as the derivative (discrete) of the leading edge (increasing) acceleration signal. Jerk was reported as peak values, which were highly covariant with average jerk across all phases, $r = 0.91 – 0.95$.

[5] The magnitude and direction of the acceleration signal (e.g. increasing or decreasing about 1g) is explained by the equation ACC = DYN – G (where ACC = acceleration sensed by accelerometer, DYN = dynamic acceleration of trunk, and G = gravitational acceleration) (Luinge 2004), after Newton's Second Law. When the DYN component is < 1g during the P1 and P4 phases, accelerometer output will be proportional to the dynamic trunk component. If DYN is >1g, the resultant accelerometer output will tend to deviate back towards 1g, in spite of a largely DYN acceleration (e.g. 0.5 g can arise from DYN of -0.5 or -1.5 as, abs (0.5) or abs(-0.5) is equivalent).

Duration, acceleration and jerk variables were computed across all reps and phases within each set (80 reps x 4 phases x 3 sets for each variable). Repetitions were time-normalized for each set by binning data to 20 equally-spaced bins per set, representing 5% intervals for the duration of the set. The low-pass, orientation signal from the trunk mounted accelerometer (reflecting trunk inclination) was also derived and used to confirm the temporal characteristics of the resultant acceleration (e.g. inflection between P2 and P3 phases). No between group differences in this signal were observed and it did not provide additive information to the measures of trunk inclination extracted from video analysis.

4. Exertion

Prior to the first set, and following each successive set, participants rated their perceived exertion (RPE) at the low back, lower and upper extremities[164], and whole body[161] using a modified Borg scale (6-20 scale) (Appendix: *Perceived Exertion*). During each set, RPE of the whole body was also reported at 5 minute intervals. Heart rate (HR) was sampled throughout (R-R interval, RS-800x, Polar Electro USA).

Data processing was performed using spreadsheet software (Excel 2010, Microsoft). Heart rate (R-R) was imported from a tab-delimited .TXT file (Polar software) and low-pass filtered using a 10-sample moving average. Spurious data points, arising from brief communication loss between transmitter and receiver, were identified (mean + 3SD) and replaced using linear interpolation prior to filtering. Mean heart rate was calculated for 30 second epochs at 5 minute intervals throughout each set (corresponding to the samples of whole body RPE). To account for the effect of age on maximal heart rate, heart rate was represented as percent of (estimated) age-adjusted maximum[240]. No additional processing was required for independent

variables, ratings of perceived exertion and other outcome measures (weight lifted, questionnaire responses).

5. Other variables

Prior to the lifting task, each participant's mass and height were measured using an electronic scale (BWB-800S, Tanita Corp, Ill, USA) and steel tape. Grip strength was measured for the right and left hand with a grip dynamometer (Almedic, Montreal, Canada). Following the lifting task, participants completed a questionnaire on their perception of the lifting task (accuracy), previous history of back injury, lifting experience, and physical activity.

6. Statistical Analysis

The effect of BMI on lifting strategy and exertion variables was tested using repeated measures ANOVA for each set of variables. The main effect of GROUP (high, normal BMI) was modeled as a between-subject factor, in addition to the within-subject effects of SET and TIME, the latter representing the variation in outcomes within a set. The TIME effect was not tested for the mass lifted (i.e. BMI, SET factors only). Post hoc tests were two-sided, with the exception of kinematics which are one-sided (high > normal), as hypothesized. Effect size is reported as partial eta-squared (n_p^2), representing the proportion of variance (error + effect) attributable to each factor. Sphericity was assessed with Mauchly's test and a Greenhouse-Geisser correction applied where appropriate (adjusted degrees of freedom and F-ratios reported), providing a more conservative estimate of effect size for situations where variance was not equivalent across groups.

The relationship amongst dependent (e.g. heart rate, RPE) and independent variables (e.g. mass lifted, BMI) was evaluated with parametric correlation (Pearson). The sum of squares used

for calculation of the Pearson *r*-statistic was sensitive to circumstances where participants reported constant RPE values across measures – this resulted in a sum of squares (SS) of zero and a corresponding *r*-statistic (ratio of SSs) of zero or undefined (depending upon whether RPE was the numerator or denominator). As an alternate, a two-way (SET, TIME) repeated measures test was carried out on the ratio of normalized perceived exertion (whole body RPE) to heart rate. This approach was not susceptible to the SS effects (did not rely upon correlation), allowed for evaluation of differences in relative contributions of RPE and hearth rate (i.e. $H_0 <> 1$), and a means of detecting change within a set (i.e. by way of a TIME effect).

Stepwise regression analysis was performed to identify linear combinations of predictors for whole body perceived exertion from constituent region-specific exertions, heart rate and other independent variables. Binary regression models were also estimated to identify normal and high BMI participants from selected strategy and exertion outcomes. Although the sample size in this study is below the recommended *n* for generalizability of such regression models, the results of this exploratory analysis will be valid for the current sample of participants (i.e. it provides a mathematical approach to partitioning variance to selected predictor variables).

Statistical significance was set to a $p < .05$ for all tests, and a $p < .10$ was used to retain variables in stepwise regression models. Statistical analysis was carried out using SPSS 19.0 (IBM Corporation, Somers NY).

RESULTS

The self-selected mass of the normal (M = 17.7, SD = 6.9 kg) and high BMI groups (M = 20.0, SD = 5.5 kg) was similar, $t(20) = .91$, $p > .37$. Both groups rated the mass as relatively safe (M = 91, SD = 11 mm) using a visual analogue rating-scale (0 – 100 mm), with no difference between groups, $t(20) = 0.31$, $p > .76$. The high BMI participants were more confident in the accuracy of their selection (M = .84, SD = .10) than the normal BMI group (M = .74, SD = .10), $t(20) = 2.21$, $p < .05$. Participants made a mean (SD) 6.5 (4.1) number of adjustments to mass lifted, taking 3.0 (1.0) minutes to reach 80% of final mass selected (i.e. completed within the first 15.0% of the trial). The number and duration of adjustment period did not differ between high and normal BMI individuals, $p > .28$.

There was no difference in the number of participants that reported receiving 'safe-lift training' (n = 15) between high and normal BMI groups, χ^2 (1, n = 22) = 1.94, $p > .16$, nor was there a difference in those that reported lifting as part of occupation or recreation activities (n = 14) (e.g. lifting weights), χ^2 (1, n = 22) < 1, $p > .85$. The number of participants that had sought medical care for prior (+1 yr) back pain or injury (n = 13) was also equivalent between groups, χ^2 (1, n = 22) < 1, $p > .69$, with a mean (SD) of 2.5 (1.9) years prior. The grip strength of high (M = 53.8, SE = 2.4 kg) and normal BMI (M = 51.1, SE = 2.8) did not differ, $t(20) < 1$, $p > .47$. One participant (normal BMI) did not complete the questionnaires.

1. LIFTING TECHNIQUE

1.1. Relative displacement

Normal (M = 0.61, SD = .19) and high BMI (M = .53, SD = .19) participants executed the lifts using similar relative displacements (postural index, PI) about the knee, ankle and hip joints (M = 0.56, SD = 0.19), $t(20) < 1$, $p > .34$, consistent with a technique intermediary between

knee- (0.80) and hip-dominant (0.20) style. For the majority of participants (n = 18), the individual technique was representative of the group mean (i.e. 0.4 < PI < 0.8), however 3 participants choose a clear hip-dominant style (PI < .20) and one, a knee-dominant technique (PI > .80) (Figure 9).

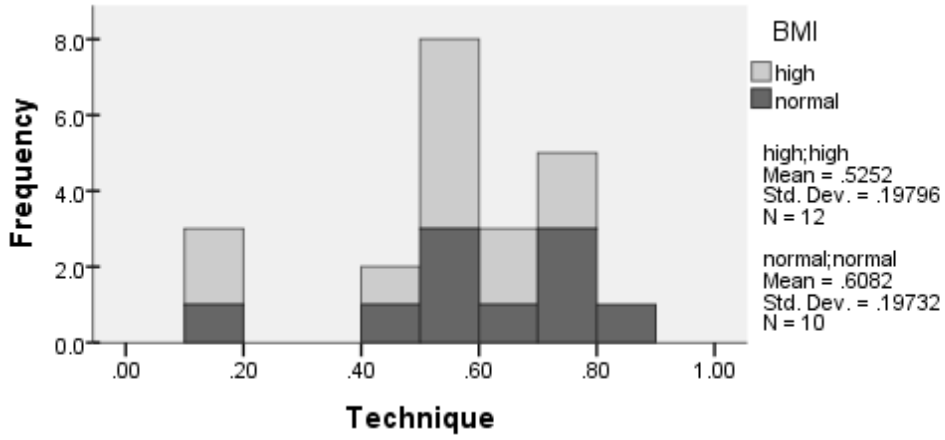


Figure 9. Distribution of lifting techniques in normal and high BMI participants. Lifting technique is represented by the relative displacement of the hip, knee and ankle joints: knee/(ankle + trunk). A value near 0.1 indicates a hip-dominant (stoop) technique, while a value > .80 is associated with a knee-dominant (squat) technique.

No difference in the postural index was detected between, $F(2,40) < 1$, $p > .72$, or within sets, $F(1,20) = 2.13$, $p > .16$; no BMI-dependent interactions were apparent, all $F < 1$, $p > .27$.

1.2. Absolute orientation

1.2.1 BMI effects

Consistent with the relative displacements, no effect of body mass was detected for the orientation of the knee, ankle or trunk at lift initiations, all $F(1,20) < 1.90$, $p > .18$. However, to achieve similar trunk inclinations, high BMI participants used comparatively more mid-torso flexion (M = +9.4, SE = 4.1 deg), $F(1,20) = 5.34$, $p < .05$, $\eta_p^2 = .21$, and less hip flexion (M = -8.0, SE = 3.0 deg), $F(1,20) = 6.86$, $p < .05$, $\eta_p^2 = .26$ (Figure 10, *torso, hip*).

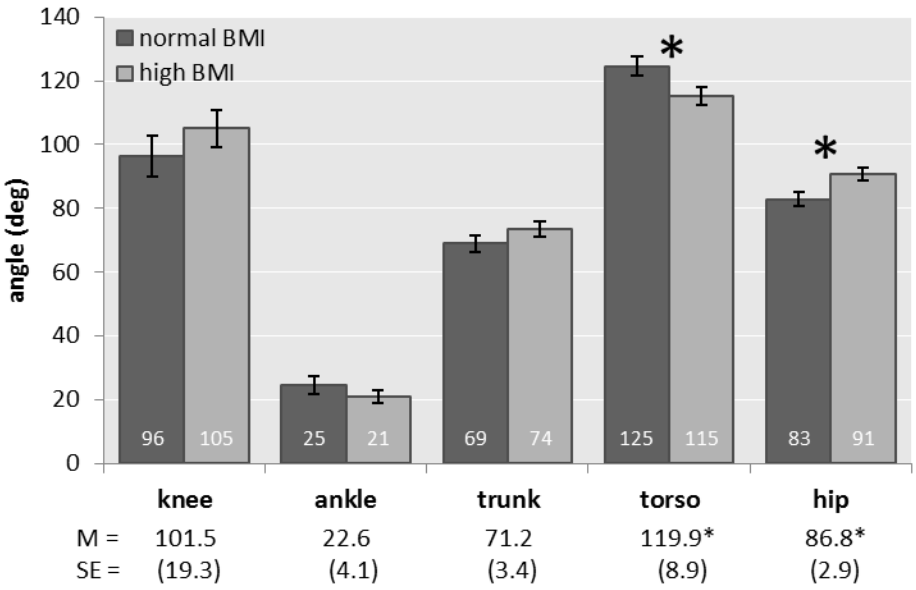


Figure 10. Joint angle at lift initiation (M, SE) of normal and high BMI participants. The average angle across all participants is provided below the table. *between-group effect, p < .05.

Consistent with the reduction in hip flexion of high BMI participants, hip range of motion (ROM) was also reduced (M = -8.7, SE = 3.1 deg), $F(1,20) = 9.45$, $p < .01$, $\eta_p^2 = .32$, however torso ROM did not differ between groups, $F(1,20) < 1$, $p > .54$. Instead, high BMI participants used less trunk ROM, (M = -9.3, SE = 3.4 deg), $F(1,20) = 7.89$, $p < .05$, $\eta_p^2 = .28$, with a corresponding increase in mid-torso flexion at upright (M = 9.5, SE = 2.6 deg), $t(20) = 3.61$, $p < .01$ (i.e. the trunk was more flexed at upright).

1.2.2. Time effects

Across all participants, a small increase (M = 2.6, SE = 1.2%) in knee angle was detected within each set, $F(1,20) = 4.50$, $p < .05$, $\eta_p^2 = .18$. With the exception of an increase in hip angle between set 1 and set 2 (M = 1.76, SE = 0.81%), $t(21) = 2.09$, $p < .05$, no other main effects or interactions were detected for the knee, $F < 1$, $p > .39$, ankle, $F < 1$, $p > .35$, trunk, $F < 2.8$, $p > .12$, torso, $F < 1.84$, $p > .19$ or hip angles, $F < 1$, $p > .47$.

1.3. External load

1.3.1. BMI effects

High BMI participants held the box farther from the hip joint centre (M = +4.7, SE = 1.8 cm) than participants with a normal BMI, $F(1,20) = 7.16$, $p < .05$, with a corresponding increase in shoulder flexion at upright (M = +20.0, SE = 4.3 deg), $F(1,20) = 21.78$, $p < .001$, $\eta_p^2 = .52$ (Figure 11, *box-hip*, *shoulder top*). The difference in shoulder flexion at lift initiation (M = 11.6, SE = 6.3 deg) was approaching significance, $F(1,20) = 3.37$, $p = .08$.

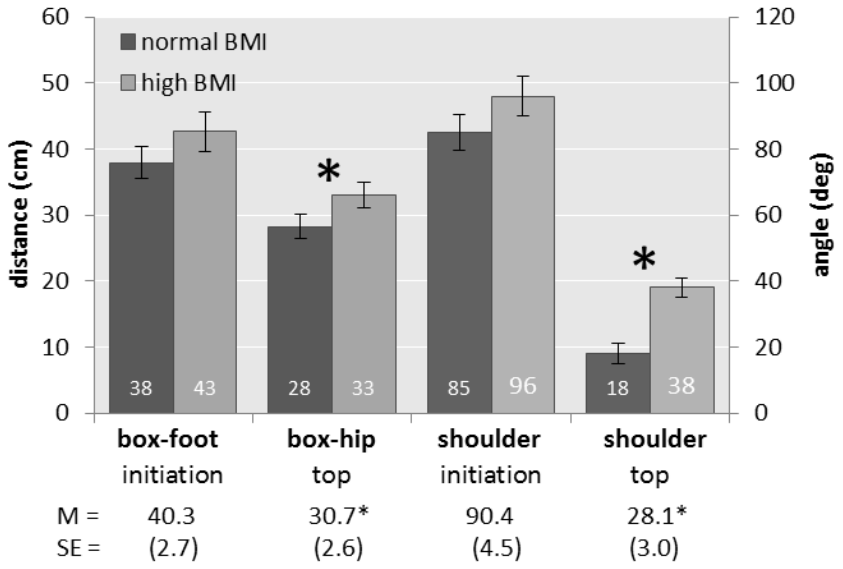


Figure 11. External load position (M, SE) relative to the foot (initiation) and hip (top) and accompanying shoulder position of normal and high BMI participants. Average across all participants provided below the table. *between-group effect, p < .05

No main effect of BMI was apparent for foot position at lift initiation, $F(1,20) = 1.66$, $p > .21$, however a BMI*SET interaction was detected, $F(2,40) = 78.55$, $p < .05$, $\eta_p^2 = .16$, along with a near significant BMI*TIME interaction, $F(1,20) = 3.44$, $p > .08$, $\eta_p^2 = .15$.

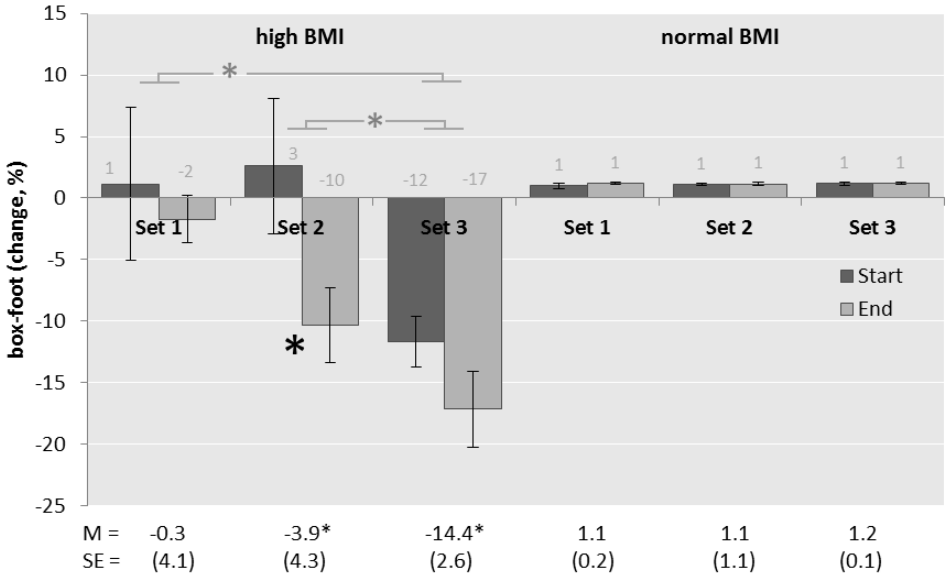


Figure 12. Change in foot position (M, SE) across (set 1 – 3) and within (start – end) sets of normal and high BMI participants during 3 – 20 minute sets of lifting (4 lifts/min). * p< .05

High BMI participants maintained consistent foot placement during the first set, $t(11) < 1$, $p > .68$, but moved closer to the box during set 2 (M = -12.5, SE = 5.32 %), $t(11) = 2.43$, $p < .05$, and likely set 3 (M = -5.5, SE = 3.0 $\%_{start}$), $t(11) = 2.12$, $p = .06$ (Figure 12, *high BMI*). By end of the last set, high BMI participants were standing 18.3 (5.7) $\%_{start}$ closer to the box, $t(11) = 3.68$, $p < .01$, compared to the start of set 1. Normal BMI participants used consistent box-foot placement across all sets, all $t(9) < 1$, $p > .34$ (Figure 12, *normal BMI*).

No other BMI interactions were detected, all $F < 1$ $p > .38$.

1.3.2. Time effects

Both groups decreased the box moment arm between sets (box-hip), $F(2,40) = 5.44$, $p < .01$, with the change detected only between set 1 and set 2 (M = -4.8, SE = 2.0 %), $t(21) = 2.25$, $p < .05$. A similar within-set reduction in moment arm (M = -2.2, SE = 1.1%) was approaching

significance, F(1,20) = 4.06, p = .06. By the end of the last set, participants were holding the box 7.5 (1.1) $\%_{start}$ closer than at the beginning of the task (not shown).

No change in shoulder position (initial or top) was observed across, F(1,20) = 2.27, p >.13, or within sets, F(1,20) < 1, p > .41. No BMI-dependent interactions were detected, all F < 2.2, p > .15

2. KINEMATICS

As expected, 4 distinct phases of acceleration were apparent for the trunk segment (P1-P4) and two for the external load (B1, B2) (Figure 13). The peaks of the time-normalized, trunk-lowering accelerations (P1, P2) were of higher magnitude (M = +0.07, SE = .01 g) than the trunk-raising accelerations (P3,P3), *t*(21) = 4.99, p < .001, and the accelerations of the external load (B1, B2) were larger (M = +0.12, SE = 0.02 g), *t*(21) = 5.97, p < .001, than those of the trunk (P3, P4)

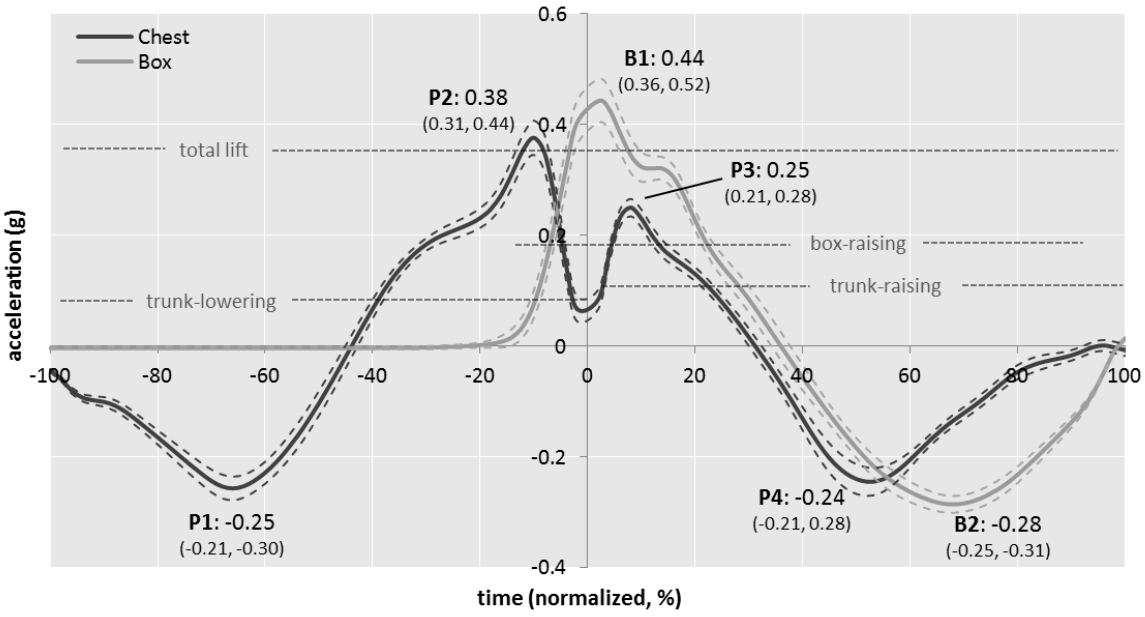


Figure 13. Time-normalized accelerations (mean +/- SE plotted) during lifts (4/min) of a self-selected mass. Signals were time-normalized to the start of the 2nd upwards acceleration of the trunk (P3). The mean (95% CI) peak acceleration is indicated, as well as delineation of each lifting phase (e.g. total lift, lowering, raising). The normalization procedure used here (independent, or two-point, normalization of trunk and box signals) is intended to reflect the magntidue of each signal, rather than differences in the relative timing of each signal.

2.1. BMI effects

2.1.1. Duration

The effect of BMI on lift duration was best fit with a time-dependent model, with significant BMI*SET interactions for the total lift, $F(1.6, 32.6) = 1.63$, $p < .01$, $\eta_p^2 = .21$,and trunk-raising durations, $F(2,40) = 4.58$, $p < .01$, $\eta_p^2 = .19$. The interaction for trunk-lowering duration was approaching significance, $F(1.4, 27.3) = 2.45$, $p = .11$, but the box-raising was not, $F(1.3, 26.5) < 1$, $p > .58$.

Table 28. Duration of lifting phases for normal and high BMI participants, across and within sets during 3-20 minute sets of lifting (4 lifts/min). Differences between (shaded) and within (*) groups are indicated; p < .05.

	set 1		set 2		set 3	
	normal BMI	high BMI	normal BMI	high BMI	normal BMI	high BMI
total lift						
duration, sec (M, SD)	2.32 (0.31)	2.23 (0.23)	2.43 (0.40)*	2.20 (0.20)	2.39 (0.40)	2.19 (0.22)
difference, sec (M, SE)	ns		0.23 (0.13)		ns	
t(20) =	< 1, p > .21		1.78, p < .05		1.48, p > .07	
trunk-raising						
duration, sec (M, SD)	1.11 (0.19)	1.04 (0.12)	1.18 (.026)*	1.02 (0.10)	1.15 (0.24)	1.02 (0.08)
difference, sec (M, SE)	ns		0.15 (0.08)		0.14 (0.08)	
t(20) =	1.11, p > .19		1.85, p < .05		1.87, p < .05	
trunk-lowering						
duration, sec (M, SD)	1.21 (0.14)	1.19 (0.12)	1.25 (0.13)	1.17 (0.13)	1.24 (0.16)	1.18 (0.16)
difference, sec (M, SE)	ns		ns		ns	
t(20) =	< 1, p > .69		1.44, p > .17		< 1, p > .72	
box-raising						
duration, sec (M, SD)	1.33 (0.18)	1.30 (0.17)	1.36 (0.21)	1.31 (0.07)	1.33 (0.25)	1.29 (0.08)
difference, sec (M, SE)	ns		ns		ns	
t(20) =	< 1, p > .69		< 1, p > .40		< 1, p > .66	

Between-group comparisons revealed that normal BMI participants were lifting slower that the high BMI participants by the second set (M = +0.23, SE = 0.13 sec), with a similar trend persisting (but not significant) for the last set (Table 28, *total lift* __). The effect was more apparent in the trunk-raising phase with a mean (SE) difference of +0.15 (0.08) seconds in the last two sets (Table 28, *trunk-raising* __).

Within-group comparisons revealed these effects could be attributed to an increase in duration (slowing) of the total lift (M = -4.5, SE = 1.8%), $t(9) = 2.55$, $p < .05$, and trunk-raising phases (M = -2.8, SE = 1.2%), $t(9) = 2.42$, $p < .05$, by the normal BMI participants (Table 28, *normal BMI*, *) rather than a change (decrease) by the high BMI participants, $t(11) < 1$, $p > .32$.

2.1.2. Acceleration magnitude

No main effect of BMI was detected for the acceleration magnitude of any lifting phase, all $F(1,20) < 1.1$, $p > 0.20$, however within-set interactions were detected for the P3, $F(4.1, 82.8) = 2.10$, $p < .05$, $\eta_p^2 = .10$, and B1 phases, $F(4.9, 97.7) = 2.19$, $p < .05$, $\eta_p^2 = .10$.

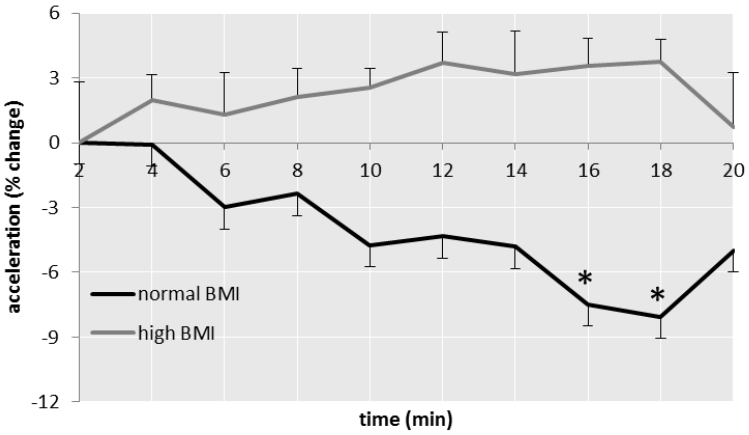


Figure 14. Change in box acceleration (B1) during a single 20 minute set of lifting for normal and high BMI individuals (M, SE), *p < .05 for both between- (high vs normal) and within-group (vs start) differences.

Normal BMI participants decreased the magnitude of the B1 (box) acceleration within each set, evident by a significant repeated-measures linear contrast, $F(1, 9) = 8.04$, $p < .05$ $n_p^2 = .47$, (main effect: $F(9, 99) = 2.45$, $p < .05$) . Post hoc tests (vs start of set) were significant (M = -7.7, SE = 2.4%) for the two-largest deviations (minute 16 and 18), $t(9) = 2.14$, $p < .05$ (Figure 14 *). The acceleration magnitude differed between-groups (M = 11.0, SE = 4.6%) over the same interval, $t(20) > 2.39$, $p < .05$. No within-group effect was detected for high BMI participants, $F(1,11) < 1$, $p > .87$.

The interaction for the P3 phase could not be attributed to either between-, all $t(20) < 1.18$, $p > .25$, or within-group effects. The apparent increase in high BMI acceleration over the course of a set (M = 5.9, SE = 3.4 %)(Figure 15, *high BMI*), did not reach significance as either a main effect, $F(9, 99) = 1.56$, $p < .14$, or post-hoc comparison $t(11) = 1.87$, $p > .09$.

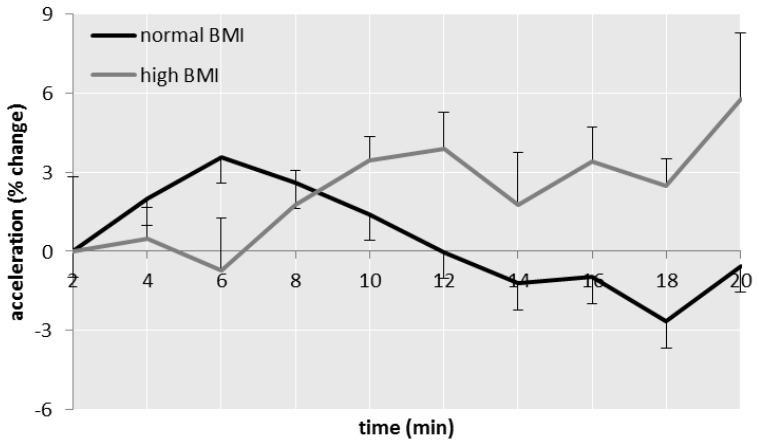


Figure 15. Change in trunk acceleration (P3) during a single 20 minute set of lifting for normal and high BMI individuals (M, SE).

2.2 Time effects

A significant SET effect for B1 acceleration was detected across all participants, $F(1.2, 29.9) = 4.97$, $p < .05$, $\eta_p^2 = .20$, attributable to a mean (SE) increase of 4.1 (1.2)% between set 2 and set 3, $t(21) = 3.40$, $p < .01$ (not shown). No other effects were detected, all $F < 1$, $p > .41$.

3. EXERTION

3.1. BMI effects

Both normal (M = 54.4, SE = 2.7 $\%HR_{max}$) and high BMI participants (M = 61.8, SE = 2.5 $\%HR_{max}$) maintained relatively low cardiovascular efforts, however heart rate was greater (M = +7.4, SE = 3.7 $\%HR_{max}$) in the high BMI group, $F(1,20) = 4.15$, $p < .05$, $\eta_p^2 = .17$. The increase

in heart rate throughout each set (%start) also differed between normal (M=16.4, SE = 2.3%) and high BMI participants (M = 21.1, SE = 2.5%).

In contrast, normal and high BMI participants reported similar ratings of whole body perceived exertion (RPE), $F(1,20) < 1$, $p > .97$, which was perceived as 'light' (M = 10.4, SD = 2.3, Borg scale 6-20). No BMI main effects were detected for the region-specific perceived exertion measures, $F(1,20) < 1$, $p > .45$, however normal and high BMI participants differed in RPE across sets for the back, $F(1.5, 30.5) = 4.09$, $p < .05$, $\eta_p^2 = .17$, and shoulder regions, $F(2, 40) = 3.52$, $p < .05$, $\eta_p^2 = .15$ (Figure 16).

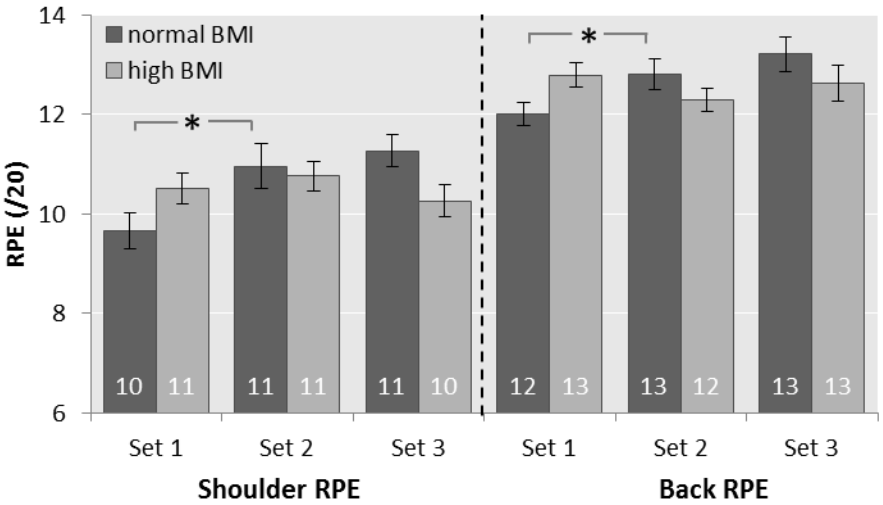


Figure 16. Perceived exertion of the shoulder and back regions for normal and high BMI participants throughout 3 – 320 minute sets of lifting (4 lifts/min). Shown are mean and within-subject SD. *p < .05, within group difference. No difference was detected between groups, all p > .30.

Post hoc tests revealed a significant increase in RPE by normal BMI participants between the first and second sets for both the back (M = +1.30, SE = 0.60), $t(9) = 2.20$, $p < .05$ and shoulder regions (M = +0.80, SE = 0.20), $t(9) = 4.00$, $p < .01$. No difference was detected for the high BMI participants, $t(11) < 1.32$, $p > .22$, and the (lower-power) between-group post-hocs were not significant, $t(20) < 1.04$, $p > .31$ (Figure 16).

3.2. Region and Set effects

Across all participants, perceived exertion differed by body region, $F(4, 80) = 16.13$, $p < .001$, $\eta_p^2 = .45$, with the highest exertions reported for the back (M = 11.5, SD = 2.2) and whole body (M = 11.1, SD = 2.4), which were not different, $t(20) = 1.10$, $p > .29$. The remainder of regions varied in a hierarchal manner, $t(20) > 2.21$, $p < .05$ (Figure 17, *mean SD* *).

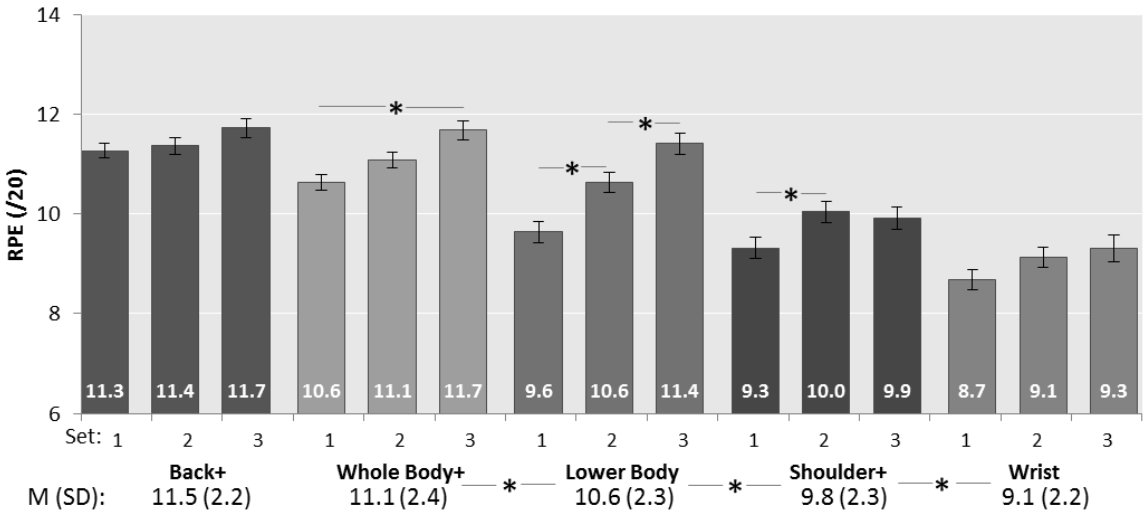


Figure 17. The effect of SET on perceived exertion. Error bars depict within-subject variance only (SD). * p < .05; +BMI interaction (see above).

Perceived exertion also varied across sets, $F(2, 40) = 7.27$, $p < .01$, $\eta_p^2 = .27$, however the variation was not uniform across regions, $F(8,160) = 2.50$, $p < .05$, $\eta_p^2 = .11$. Lower body RPE increased across all three sets (M = 0.9, SE = 0.4), $t(21) > 2.1$, $p < .05$, with a similar trend in whole body RPE, however post hocs were significant only between the first and last set (M = 1.05, SE = 0.32), $t(21) = 3.28$, $p < .01$ (between-sets: $p > .07$)(Figure 17 *). Participants also reported increased shoulder exertion between the first and second set (M = 0.73, SE = 0.34), $t(21) = 2.07$, $p < .05$, but not change for the back, $t(21) < 1.50$, $p > .14$, effects that were better described with a BMI-dependent model (Figure 16). No change was detected for the wrist, $t(21) < 1.52$, $p > .14$. At the end of the third set, perceived exertions of the whole body, lower body and back regions were similar (M = 11.6, SE = 0.7), $t(21) < 1$, $p > .43$.

3.3. Time effects

As expected, heart rate increased within each set, $F(1.9, 38.7) = 146.3$, $p < .001$, $\eta_p^2 = .88$, by a mean (SE) of 13.4 (0.9) $\%HR_{max}$ (Figure 18, *top*) and with significant increases at each 5-minute interval, $t(21) > 5.11$, $p < .001$. Average heart rate also varied across sets, $F(2, 40) = 208.41$, $p < .001$, $\eta_p^2 = .91$, increasing between the first and second ($M = 8.6$, $SE = 0.5$ $\%HR_{max}$), $t(21) = 16.71$, $p < .001$, but not second and third sets, $t(21) < 1$, $p > .38$ (Figure 18, *bottom*). Between-group differences were significant at all intervals, $t(20) > 2.15$, $p < .05$, and did not vary across, $F(1.6, 31.8) < 1$, $p > .79$, or within sets, $F(1.9, 38.7) = 1.1$, $p > .34$.

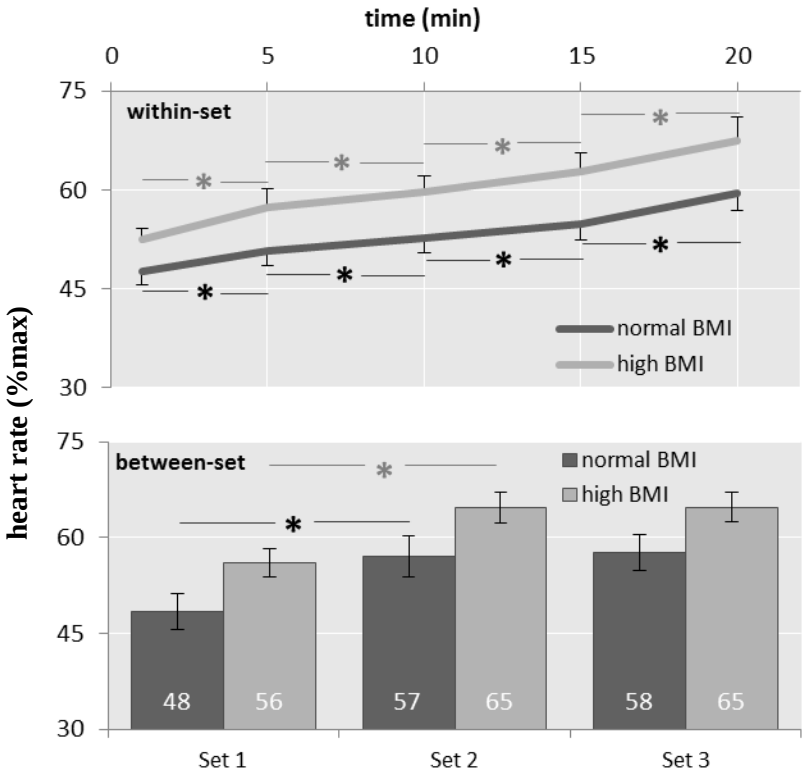


Figure 18. Change in cardiovascular effort (heart rate) within (top) and between (bottom) sets for high and normal BMI participants during 3 – 20 sets of lifting (4 lifts/min). *p < .05, within-group effect; the difference between groups was significant at all intervals (not indicated, p< .01).

Similar to heart rate, whole body RPE increased within each set, $F(1.4, 28.2) = 38.7$, $p < .001$, $\eta_p^2 = .66$, by a mean (SE) of 5.0 (0.2) (Borg scale, 6 – 20; ~45.5%) and was also significant

across each 5-minute interval, $t(20) > 2.52$, $p < .05$ (Figure 19, *top*). Although the BMI*SET interaction did not reach significance, $F(1.3, 25.4) = 1.77$, $p > .20$, a SET effect was detected using one-way ANOVA for the normal BMI participants, $F(2,18) = 4.35$, $p < .05$, $\eta_p^2 = .33$, but not the high BMI group, $F(2, 22) = 1.40$, $p > .27$. Post hoc tests revealed an increase ($M = +1.3$, $SE = 0.4$) in whole body perceived exertion for normal BMI participants between set one and set two, $t(9) = 2.80$, $p < .05$ (Figure 19, *bottom)*.

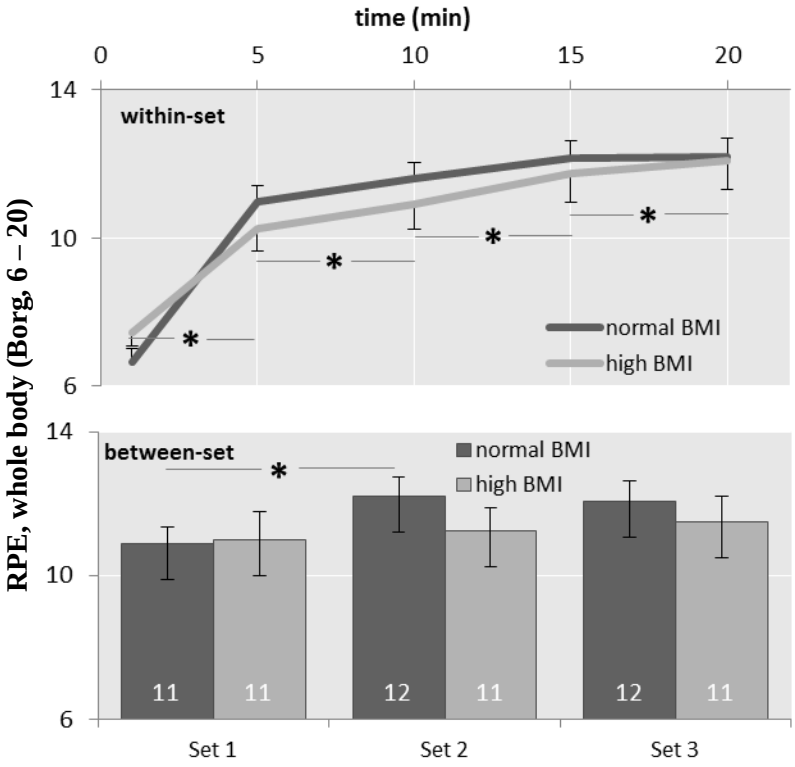


Figure 19. Change in perceived exertion (whole body) within (top) and between (bottom) sets for high and normal BMI participants during 3 – 20 minute sets of lifting (4 lifts/min). *p < .05, within-group effect; no differences were detected between groups (p > .30).

4. RELATIONSHIP BETWEEN HEART RATE AND PERCEIVED EXERTION

4.1. RPE:HR ratio

As confirmation of the apparent BMI-dependent variation in perceived exertion relative to heart rate (Figure 18, Figure 19), the ratio of RPE:HR was found to be greater in normal (M = 1.11, SE = 0.06) compared to high BMI participants (M = 0.92, SE = 0.06), $F(1,20) = 5.92$, $p < .05$, $\eta_p^2 = .23$. The ratio also varied across and within sets, $F(3.5, 70.4) = 41.8$, $p < .001$, $\eta_p^2 = .67$.

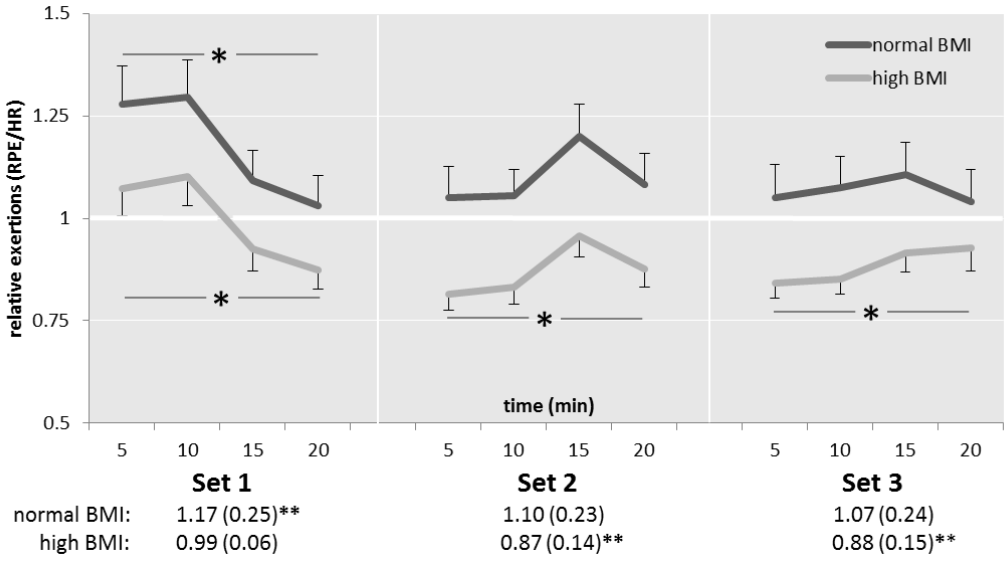


Figure 20. Relative change in perceived exertion and cardiovascular effort (RPE/HR ratio) of high and normal BMI participants throughout 3 – 20 minutes sets of lifting (4 lifts/min). *within-group effect, p< .05t. Mean (SD) RPE/HR value by set provided below table, **H_0 <>1, p < .05 (i.e. group mean significantly different from 1).

For normal BMI participants, the RPE:HR ratio was greater than 1 during the first set, indicating relatively greater perceived effort compared to heart rate, $t(9) = 2.20$, $p < .05$, but not the second, $t(9) = 1.35$, $p > .21$ or third sets, $t(9) = 1.07$, $p > .39$ (Figure 20, *normal BMI* **). Conversely, in high BMI participants, the ratio was not different from 1 during the first set, $t(11) < 1$, $p > .55$, but less than 1 during both set 2 (M = 0.87, SD = 0.14), $t(11) = 3.21$, $p < .01$, and set 3 (M = 0.88, SD = 0.15), $t(11) = 2.74$, $p < .05$ (Figure 20, high *BMI* **).

The RPE:HR ratio decreased for both normal (M = 0.25, SE = 0.05), $t(9) = 4.92$, $p < .001$, and high BMI participants (M = 0.19, SE = 0.04), $t(11) = 4.59$, $p < .001$, during set 1 (Figure 20, *set 1*,*). In the second and third sets, it remained constant for the normal BMI participants, $t(9) < 1.22$, $p > .25$, but increased for the high BMI group (M = 0.08, SE = 0.03), $t(11) > 2.18$, $p < .05$, indicating an elevation in HR without a concomitant increase in RPE.

4.2. Predictors of whole body perceived exertion

Consistent with involved body segments, perceived exertions of the lower body ($\beta = .24$, $p < .05$) and back regions ($\beta = .49$, $p < .001$) were retained in a stepwise (backwards), linear regression model for whole body RPE ($R^2 = 0.54$), along with mass lifted ($\beta = .34$, $p < .001$)(Table 29). In support of a BMI-effect on perceived exertion, BMI was also retained, but with a negative β-coefficient ($\beta = -0.22$, $p < .05$). The correlation between BMI and whole body perceived exertion was $r = -.23$; the complete stepwise model (all steps) and correlation among predictors is provided in Appendix: *Relationship Among Lifting Outcomes*.

Table 29. Model predicting whole body perceived exertion from region-specific exertion, mass lifted and BMI during a 1-hour repetitive lifting task (sagittal plane lift, self-selected mass). Backwards, stepwise regression was used.

	R^2 (adjusted)	B	SE B	B	P
Final model	.54				
Constant		4.57	1.66		< .01
Lower		0.22	0.10	0.24	< .05
Back		0.50	0.11	0.49	< .001
Mass lifted		0.12	0.03	0.34	< .001
BMI		-0.10	0.04	-0.22	< .05

5. *Predictors of group (high vs normal BMI) by lifting outcome*

Regression models developed to predict group membership were single-variable when modelled using only technique or exertion outcomes (Table 30), indicating little additional variability was explained using more than one predictor in each category. No model was returned

for kinematic (acceleration) variables. The technique model (shoulder angle) provided the best fit across models (R^2 = .70), and heart rate, rather than RPE, was retained in the exertion model (R^2 = .23).

Table 30. Models predicting BMI (high or normal) based upon technique a) and exertion b) variables during a 1-hour repetitive lifting task (sagittal plane lift, self-selected mass). Backwards, stepwise regression was used.

a)		95% CI for odds ratio		
	B(SE)	lower	odds ratio	Upper
Technique				
Constant	-6.2 (2.7)			
Shoulder angle	0.2 (0.1)	1.04	1.28	1.56

R^2 = .52 (Cox and Snell), .70 (Nagelkerke). Model $_X^2$ = 16.27, p < .001
variables entered: box moment arm; shoulder, hip, trunk, torso angles; trunk & knee ROMs

b)		95% CI for odds ratio		
	B(SE)	lower	odds ratio	Upper
Exertion				
Constant	-6.2 (3.5)			
Heart rate	0.1 (0.1)	0.99	1.12	1.26

R^2 = .17 (Cox and Snell), .23 (Nagelkerke). Model $_X^2$ = 4.12, p < .05
variables entered: heart rate; RPEs: whole body, lower, back, shoulder

Using both technique and exertion predictors, a two-term model incorporating heart rate and shoulder angle (Table 31) was returned, indicating exertion and technique variables make independent (and additive) contributions to the explained variance across individuals. This model was estimated using linear, rather than logistic, regression as the logistic model identified all participants, producing inflated regression coefficients; the adjusted r^2 (0.65) is comparable to the Naglekerke r^2 in binary models.

Table 31. Model predicting BMI based upon exertion and technique variables during a 1-hour repetitive lifting task (sagittal plane lift, self-selected mass). Backwards, stepwise regression was used.

	B(SE)	β	P
All			
Constant	-0.52 (0.44)		
heart rate	0.026 (0.005)	0.72	< .001
shoulder angle	0.023 (0.007)	0.41	< .01

R^2 = .65 (adjusted)
variables entered: heart rate, shoulder angle

6. Summary of Findings

The effect of BMI on lifting strategy was most apparent on how individuals handled the external mass (rather than self-selected mass or lifting technique), where high BMI participants held the external load farther from the body and with greater shoulder flexion.

Participants adopted a 'free-form' lifting technique, however in order to assume a similar trunk orientation (technique) as normal BMI participants, the high BMI participants initiated the lift with increased flexion at the mid-torso (rather than the hips).

All participants reduced the moment arm of the external load as sets progressed and high BMI participants also moved closer to box to initiate each lift across sets.

High BMI participants executed the lifts with shorter durations than normal BMI participants. This effect increased as lifting progressed, but was due to a change in lift duration (slowing) by normal BMI participants only.

High BMI individuals performed the lifting task with greater cardiovascular effort, but equivalent perceived effort as normal BMI participants. As lifting progressed, variations (or lack of) in perceived effort of high BMI participants were independent of changes in cardiovascular effort.

Normal and high BMI participants could be identified based upon the orientation of the upper extremity and cardiovascular effort outcomes.

DISCUSSION

Body mass index was shown to have a significant effect on lifting strategy and exertion outcomes during a repetitive, fixed-pace lifting task. This was most apparent on how individuals handled the external mass, rather than the orientation of body segments or the mass lifted. Repetitions had a BMI-dependent influence on lifting kinematics, but not in the direction anticipated, characterized by alterations in normal BMI participants only. It was possible to identify normal and high BMI participants based upon orientation variables specific to the external load and exertion (cardiovascular) measures, revealing aspects of lifting attributable to increased BMI.

External load

The main effect of BMI on lifting strategy was on the external load, where the most pronounced alteration was a BMI-dependent increase in the distance between the external load and hips (moment arm), along with a corresponding increase in upper arm orientation (shoulder flexion). The increased distance can likely be attributable to morphology-specific factors (i.e. waist circumference), and is consistent with recent findings from standing work tasks[40], suggesting this effect might be expected in other materials handling tasks. Reducing the moment arm of an external load or force is one of the most common approaches to minimizing injury risks, and the results study indicate the effectiveness of this approach to mitigate risk will vary across individuals. Preliminary estimates for the mechanical consequences of this effect on lumbar spine load during a load carrying task, which is similar to the upright position of the lifting task studied here, have been provided (44%)(*The effect of obesity on BSIPs*). Although the BMI-dependent effect on external load position appears relatively straightforward, a number of potential covariates require further study. For instance, environmental constraints (e.g. a bin or shelf) may prevent participants from adopting an external load position proportional to their morphology. Similarly, neither group of participants elected to adopt a strategy that minimized

the moment arm of the box (evident by the decrease across sets), a strategy that would actually exacerbate the loading differences between groups (*The effect of obesity on BSIPs*). The proportion of the population with an elevated body mass is increasing[60], however a paucity of studies exist that examine the effect of morphology on manipulation of external loads during materials handling tasks[241] (e.g. pushing or pulling tasks). Considering the significant BMI-dependent effects demonstrated here and in a small number of other studies[23,40], and the reliance of injury prevention recommendations on underlying kinematic and kinetic principles[51], further study is indicated. In particular, thorough biomechanical investigations are required to quantify the time-dependent aspects of external load position, and to determine the relevance of these effects to peak/cumulative loads and corresponding injury-risk thresholds[51].

In addition to the lumbar spine, an increased external load (or work surface[40]) distance would also contribute to elevated loading on interposed body segments (e.g. the shoulder). Although the impact of obesity on upper extremity musculoskeletal pathology has received comparatively less attention than the lower extremity[102], individuals with an elevated BMI are at an increased risk of many upper-extremity musculoskeletal disorders[242]. High BMI individuals also account for a disproportionate number of patients undergoing surgical repair for rotator cuff tendinopathy, and tend to have poorer outcomes[243]. While the underlying mechanisms are likely multi-factorial[243], mechanical factors have been implicated, but not well elucidated. However, the seemingly obvious mechanical impact of obesity may not necessarily manifest during real life tasks if participants adopt a strategy that attenuates these effects. For example, a BMI-dependent increase in trunk flexion (as shown here and previously[40,241]) may serve to position the shoulder joint closer to the external load and preferentially reduce mechanical load about the shoulder (at the expense of the low back). In fact, although much attention has been placed on back-specific factors as explanations for lifting strategy[31,152], it is possible that consequences at peripheral joints (e.g. glenohumeral) may also exert an influence on lifting strategy. Identification of the

determinants of upper-extremity materials handling strategies has important implications for manual lifting advice (e.g. individual-specific interventions), and may also help clarify the extent that mechanical factors contribute to the increased incidence of upper-extremity musculoskeletal pathologies in high BMI individuals.

Lifting technique

No clear effect of body mass on the orientation of primary body segments was detected, and similar to previous investigations[30,43] most participants adopted a free-style technique, supporting the hypothesis that external demands of the task (e.g. lifting height) are the principle determinants of lifting technique (rather than individual factors)[30]. However, most studies (including this one) have examined relatively simple lifting tasks[23,233,244] (e.g. sagittal or diagonal lifts), and it is possible the influence of individual factors (e.g. body mass, height) are more apparent during complex or high-demand tasks (e.g. loading/unloading a large bin, etc). Increased body mass has been shown to impact the range of motion at certain joints[241] and is related to increased effort during sustained reaching motions[62]. These effects would be unlikely to manifest during free-style, sagittal plane lifts, but would be relevant during manual handling tasks that involve extended reaching or terminal ranges of motion. Although more complex or high exertion manual handling tasks are seldom performed, they contribute disproportionately to materials handling injuries[245]. Understanding the influence of individual-specific factors in these high-risk circumstances may be a more fruitful approach than examination of comparatively simple lifting tasks.

Although the effect BMI on the position of principle body segments (trunk, thigh, shank) was negligible, the increased mid-trunk flexion apparent in high BMI participants has a number of important considerations. In addition to a potential means of minimizing shoulder load, increased trunk flexion may also arise due to physical obstruction of the abdominal fat mass during hip-initiated flexion motions. Support for this was provided by Galli[113] who observed a

similar forward flexion during a sit-to-stand task. Importantly, this task did not involve an external load, and so the need to minimize loading about the shoulder was likely inconsequential. Flexion at the mid-trunk, rather than hips, may serve to keep the abdominal fat mass closer to the lumbar spine/hips. Minimizing the torques arising from the weight of the abdominal pannus may be a particularly advantageous/preferable strategy since the majority of trunk mass is located in this region, and the centre of mass is already more anteriorly located (*The influence of obesity on BSIPs*). Although increased flexion about the mid-trunk appeared to be an intentional strategy of high BMI individuals, both during lifting and sit to stand tasks[113], future study is required to differentiate this change from more chronic postural changes related to obesity. For instance, increased mid-trunk flexion reflect a more chronic postural adaptation in order to minimize flexion torques about the lumbar spine or maintain the centre of gravity within the base of support (i.e. static tasks). Alternatively, it may reflect a more acute adaptation to performance of a lifting task with increased torso mass. Regardless of the rationale, increased torso (versus hip) flexion can have deleterious consequences for the lumbar spine, independent of external load. For instance, increased lumbar flexion is associated with a reduction in lumbar extensor muscle activation, re-distribution of load to passive tissues[14], a decreased ability to resist shear forces, and elevated compressive load secondary to reductions in erector spinae moment arms[198]. Lastly, the differential torso flexion between high and normal BMI individuals is relevant to biomechanical models that consider the trunk as a single fixed segment, or that use whole trunk BSIP estimates, which may not account for changes in torso flexion throughout a lifting motion

Self-selected mass

In spite of performing more physical work and with elevated cardiovascular exertion, individuals with increased body mass did not adjust the mass lifted to compensate over the course of a 1-hour repetitive lifting task. Although the effect of BMI on self-selected mass has only been examined for a relatively narrow range of lifting frequencies, durations and tasks[23,233] (e.g.

sagittal plane lifts, 1 – 12 lifts/min or less), body mass does not appear to be a significant contribution to the mass individuals choose to lift during simple lifting tasks. Instead factors such as strength[180] or lifting experience[37,246] may explain a larger proportion of the variance in self-selected mass. Individuals with high BMI may also elect to compensate for the mechanical penalty of increased body mass by modifying different aspects of lifting strategy (e.g. kinematics, foot position). Alternatively, high BMI individuals may be less sensitive to increases in mechanical or cardiovascular effort that arise due to the increased mass moved[233], and therefore would be less likely to alter the load based upon perceived exertions. Alterations in mass lifted would be expected for individuals with greater strength[180] or endurance, however no evidence exists to support the presence of beneficial neuromuscular adaptations secondary to obesity (in fact, the alternate is more probable[48]). Similar to the lifting technique outcomes above, it is also possible the one-hour duration (and 4 lift/min frequency) examined here was not sufficient to elicit the effects of BMI on self-selected lifting mass, and subsequent studies may wish to consider testing at durations greater than one hour, higher frequencies and/or in more complex (or even self-paced) lifting tasks. In fact, it is also possible high BMI participants elected to lift a heavier mass, evident by the trend towards a greater mass lifted (+13%). Similar trends were also observed by Singh et al (2009)[158] across a range of lifting frequencies and heights. No clear explanations are apparent to explain the potential increase in mass lifted, however it is possible that certain high BMI individuals have musculoskeletal adaptations (e.g. increased muscle mass, ligament strength) secondary to the increased load imparted by obesity that facilitate lifting heavier loads. A difference in perceptions of load/effort may also explain the difference, particularly if an individual feels they are being evaluated on the amount of mass lifted. Further study across a wider range of lifting tasks (e.g. self-paced, fixed-workload, higher frequency, etc) are required to confirm this potential effect.

Kinematics

This study provides further support that high BMI participants execute single lifts faster than normal BMI individuals, which was most apparent in the duration of lifting phases rather than acceleration magnitude[23]. More importantly, the kinematic differences increased as the lifting progressed, but contrary to the original hypotheses, were due to changes by the normal- rather than high-BMI participants. With the exception of a small increase in acceleration of the external load across sets by all subjects (which coincidentally may offset any decrement in loading arising from a reduction in moment arm), an increase in lifting duration (slowing) across sets was detected for normal BMI participants only. The increases in lift duration and external load acceleration over the course of a repetitive lifting task are consistent with previous investigations[11] that had not deliberately compared high and normal BMI groups. Findings from multiple studies indicate that both changes (acceleration and duration) are likely proportional to the extent of neuromuscular fatigue (i.e. greatest for high frequency, high load lifts[11,247,248]), and may be a movement strategy adaptation by normal BMI individuals to account for decreased torque production of prime movers (or as a direct result of fatigue). The comparatively small effects for acceleration magnitude and duration observed here are consistent with these findings (i.e. the task was not deliberately fatiguing), however the impact of these changes may manifest in lifts at higher frequencies or longer durations, and requires further study.

In contrast, high BMI participants maintained relatively consistent kinematics as sets progressed, failing to support the hypothesis of a cumulative, mechanical-based reduction in lifting kinematics for high BMI individuals. In other words, the BMI-specific adaptation in lifting strategy appears to be to maintain certain lifting kinematics (duration), rather than to alter movement strategy as lifting progresses. It is possible that high BMI participants chose to maintain a shorter lifting time in order to limit the duration of muscular exertion or for other as yet unknown benefit. Regardless of the underlying rationale, this lifting strategy likely occurs at

the expense of peak mechanical load, which generally varies inversely with the duration of individual lifts[46]. A lifting strategy that does not alter exposure to peak load to account for the time-dependent effects on fatigue (increases) or tissue yield points (decreases), may increase the probability the loading of a single repetition will exceed the yield point of lumbar tissues, due merely to the normal variability in lifting kinetics between repetitions[249]. Unless lumbar tissues have adapted to withstand greater peak forces, which are already up to 76% greater in high BMI individuals (*The effect of obesity on BSIPs*), this may increase the probability of injury during a single lift. Future studies are needed to quantify the specific mechanical consequences of these effects and to evaluate the response of high BMI participants to lifting tasks that are deliberately intended to elicit fatigue – either after performing a fatiguing task or over the course of a deliberately fatiguing lift, where the much larger alterations in kinematics would be expected[11]. Additional studies should also consider temporal differences between high and normal BMI individuals, as participants may also differ in the relative timing of joint motions and not just total displacement.

Constrained tasks

An additional contribution to the absence of BMI-dependent effects in the current study may be the unconstrained nature of the task, where high BMI participants may have been free to alter another aspect of lifting strategy (that is usually held constant). Although it is not well-understood, the influence of task constraints on observed effect sizes has been acknowledged by other authors [46,50], particularly with respect to foot position. In addition to lifting frequency, foot position is one of the most commonly constrained aspects of lifting in material handling investigations[23,177], and although it may aid in reducing between-subject variability (which presumably must vary enough to warrant control), it can limit the generalizability of findings. Principles of evidence-based practice necessitate that experimental conditions replicate actual circumstances encountered in real life (i.e. ecologically validity) or are externally valid[250], in

order for practitioners to be confident the effects will manifest outside the laboratory. Obviously, if a previously constrained variable was shown to vary across individuals, as was observed by the BMI-dependent change in foot position, this would require further scrutiny. It is well-established that the distance between the feet and external load at lift initiation influences torque during lifting tasks (via the moment arm of mass lifted), and may interact differently across lifting techniques and object size[31]. Although the implications of a BMI-dependent shift closer to the load require further investigation, it is possible that this may be an attempt to minimize the mechanical effects of the external load as lifting progressed, particularly since high BMI participants have a limited ability to move the external load closer to the trunk during the lift. Future studies should examine 'unconstrained' lifting tasks to ensure these effects, and any injury prevention strategies derived from them, can be generalized to lifting circumstances encountered during activities of daily living and occupation.

Effort (cardiovascular)

Considering that high BMI participants did not elect to reduce the mass lifted, it was not surprising to find a corresponding increase in cardiovascular exertion. Previous investigators have acknowledged that body weight influences cardiovascular exertion[51,163], but have not specifically evaluated this effect. Although the cardiovascular effort of all participants were well below recommended cardiovascular thresholds[51], it is evident that high BMI participants do not possess sufficient cardiovascular adaptations to compensate for the increased workload arising from body mass (i.e. in spite of lifting an equivalent mass, heart rate was higher than normal BMI individuals). Further, at the relatively low lifting frequency examined here, cardiovascular effort did not appear to be a sufficient influence to necessitate a reduction in mass lifted, however this effect would likely increase proportional to lifting frequency. In the absence of improvements to cardiovascular fitness, lifting tasks performed at higher frequencies (or durations) would require high BMI participants to alter some aspect of lifting strategy (e.g. mass, frequency) if they wish

to remain below proposed thresholds for cardiovascular exertion[51]. In fact, current algorithms for weight adjustment during repetitive lifting do not take into account body mass[51], and as was shown here, would result in high BMI participants working at higher cardiovascular workloads than their lower BMI counterparts. The use of direct/maximal measures of cardiovascular fitness to derive effort (rather than predictive equations) in future studies may help determine if BMI also impacts aerobic work capacity, which is assumed to be equivalent in current lifting recommendations (and the estimations used here). It is also possible that high BMI individuals would rather alter lifting frequency (e.g. fewer lifts per minute), instead of mass lifted, in order to compensate for cardiovascular issues, however this would only manifest during self-paced and not fixed pace lifting tasks. Participants did not differ with respect to self-reported comorbidities and so it is unlikely the exertion or kinematic differences observed here are due to underlying pathology (e.g. osteoarthritis), however this may become a factor in older participants where the prevelance of certain health conditions increases with obesity. Considering the well-established effect of increased external mass on cardiovascular effort[151], it seems likely that increased body mass will be a complicating factor to the safe and effective design of occupational lifting tasks with respect to frequency, duration and mass lifted. The influence of body mass should be considered as an obligatory covariate for future studies of cardiovascular effort during materials handling.

Effort (perceived exertion)

Whole body perceived exertion was similar to previously reported values[159,164], and in contrast to cardiovascular effort, did not differ between normal and high BMI participants. The inferred differential contributions of perceived and cardiovascular effort between groups was confirmed by a significantly lower RPE:HR ratio in high BMI individuals. Whole body perceived effort is a function of physiological effort and individual stimulus-perception characteristics of the individuals[251]. Although perceived exertion was not significant across groups, BMI was found

to be negatively related to perceived exertion in multiple regression models – a finding that could only be expected if increased body mass arose from training effects (e.g. lean body mass) rather than accumulation of adipose tissue, suggesting that stimulus-perception characteristics may also vary with body mass[233]. A BMI-dependent stimulus-perception effect is supported by findings that high BMI individuals are less able to discriminate between external masses of varying magnitude[170], perhaps because equivalent changes in external load will produce smaller relative differences in total mass lifted as body mass increases. Decreased sensitivity to sources of physiological effort could expose an individual to elevated risk of injury (e.g. detecting onset of fatigue), however this hypothesis should be evaluated over a wider range of perceived and cardiovascular efforts. Alternatively, normal BMI participants may have adopted a more cautious interpretation of effort during initial lifting sets, evident by the relatively greater perceived exertion compared to cardiovascular effort. Perceptions of lifting effort are also important for certain methods of establishing 'safe' lifting masses[166], however the accuracy of these approaches have been questioned[167], and a BMI-dependent difference in perceived effort would further contribute to variance in these tasks.

The change in perceived effort across body regions was also consistent with previous findings[159,164], being greatest for the principle regions involved in the lift (low back, legs). Similar to heart rate, most perceived exertion measures increased across sets, however this effect occurred almost exclusively in normal BMI participants. This is contrary to expectations of elevated effort or fatigue secondary to increased body mass. This effect was particularly apparent for the perceived exertion of the shoulder region, where based upon external load and limb segment positions, increased effort would have been expected in high BMI individuals, not normal BMI participants. It is unknown whether the shift closer to the external load by high BMI participants (foot position) was sufficient to attenuate all increases in perceived effort, and it is possible high BMI individuals are either less sensitive to increases in effort, or that they began the

task with elevated effort in these regions. Although there appeared to be a trend towards the latter possibility, the (lower-powered) between-group post hoc tests did not support this difference. Individual differences in strength may also be expected to explain a portion of variation in perceived effort, however no findings of a BMI-dependent difference in strength have been reported, and the strength measure used here (grip) was equivalent between groups.

Overall Conclusions

The aim was to determine the effect of obesity on lifting during a repetitive lifting task, quantified by both lifting strategy (objective 1) and outcomes related to physiological and perceived exertion (objective 2). The effect of BMI on lifting strategy manifested more through alterations in the position of external load and the duration of lifting phases, rather than in the orientation of body segments (lifting technique). These findings support the notion that high BMI individuals will be differentially exposed to mechanical load during materials handling and may identify areas of importance for ergonomic interventions. However, simple modifications of specific variables may be premature until the underlying rationale for changes is elucidated (e.g. compensatory or fatigue-induced). BMI-dependent differences (and lack of) in effort also suggest high BMI individuals may be less-sensitive to cumulative effects of loading and cardiovascular exertion. These findings are relevant to individuals in occupational health and to the design of injury prevention materials handling programs. Future investigations are indicated to examine more challenging/high-risk lifting scenarios and also unconstrained lifting tasks.

STUDY 3. THE EFFECTS OF LIFTING FREQUENCY, CUE TYPE AND BMI ON PREFERRED LIFTING STRATEGY

METHODS

1. Participants

Two groups of participants were recruited, one group (n = 25) with a normal BMI (M = 22.5, SD = 2.6 kg/m^2, and the other (n = 12) with a high BMI (M = 34.0, SD = 3.4 kg/m^2), for a total sample of 37 participants. The mean (SD) age of all participants was 31.2 (7.9) years, which did not differ between the normal (M = 29.7, SD = 8.1 years) and high BMI participants (M = 31.2, SD = 7.9 years), $p > .20$. The mean (SD) BMI across all participants was 26.0 (6.1) kg/m^2. Participants were otherwise healthy and free of musculoskeletal injury for > 6 months. Ethical approval was obtained from Bannatyne Campus Research Ethics Board, University of Manitoba (HREB#: H2010:408).

2. Protocol

Participants performed 3 sets of 12 lifts, in random order, interspersed by 4 minutes of rest. The parameters of the sets varied across participants: all participants (n = 37) performed one set of self-paced lifts of a light mass (8kg), as well as one set of low-frequency, fixed-pace (4 lifts/min) lifts prompted by a standardized audible cue (tone: 250 msec @ 1000Hz) [effects of BMI and frequency]. Two different prompts were examined in the low-frequency, fixed-pace condition: a verbal cue (the recorded phrase "start your lift now", n = 18) and a motion cue (n = 10), where the mass lifted remained on the shelf after each lift and was lowered by the research assistant as an indication to start the next lift (versus lowering the mass immediately following each lift) [effect of cue type]. Two additional repetitions were performed in the motion cue, where the box was lowered at a maximal rate by the research assistant. A subset of participants (n = 9) performed a set of lifts at a high-frequency, fixed-pace prompted by the standardized cue (12

lifts/min) [effect of frequency]. The distribution of participants, statistical method (i.e. within-subject or between-group) and comparison (i.e. self-paced, tone, or BMI) are provided in Table 32.

Table 32. Distribution of participants and objectives for evaluation of the effects of fixed-pace lifting (frequency and cue type influences). Statistical tests and comparison conditions are indicated.

objective	participants	statistical tests	comparison
frequency			
self-selected	n = 37	within-subject	vs self-selected
low frequency (4 lifts/min)	n = 37		
high frequency (12 lifts/min)	n = 9		
cue type			
tone	n = 37	within-subject	vs tone
verbal	n = 18		
motion	n = 10		
BMI			
normal BMI	n = 25	between-group	high vs normal
high BMI	n = 12		

2.1. Lifting Task

Participants lifted an a box (37.5 cm x 36 cm x 25 cm, handles bilateral 7 cm from top edge) from floor level to a shelf positioned at average table height (76 cm). After each lift the box was lowered by a research assistant to the starting position (centroid of the box 30 cm from front shelf). No instructions were provided regarding lifting technique or the position of limb segments, however participants were instructed to lift in a manner that "felt most natural or they would normally use". The lift was initiated from a position facing towards the shelf (i.e. shelf parallel to frontal plane) and the box was lifted with both hands, constraining the lifting motion to the sagittal plane. A series of 6 – 10 lifts were performed for familiarization.

3. Kinematics

3.1. Accelerometers

Kinematics of the distal trunk and box were acquired using a system of wireless, tri-axial accelerometers (+/- 10 g, G-Link mXRS, Microstrain Inc). Techniques and application were identical as in the previous study (see 'Kinematics' in previous study). Briefly, accelerometers were mounted relative to the standard local coordinate system of the trunk and box, and acceleration signals were obtained for 3 axes (256 Hz, synchronized between box units). Video of each set was recorded with a digital camera (30 fps, 640 x 480 pixels; Casio EX100, Casio USA) positioned parallel to the anterior-posterior plane of the lifting shelf, corresponding approximately to the sagittal plane of the lifting motion. The video was used to confirm the lifting motions occurred in the sagittal plane (visual inspection) and to ensure acceleration signals reflected the appropriate phases of motion during the high frequency conditions (e.g. synchronize acceleration signal to video).

3.2. Processing

Data processing was carried out using programs written for GNU Octave (GNU General Public License, Free Software Foundation, Inc), as in the previous protocol. The resultant acceleration was computed for the trunk and box accelerometers, with the duration, peak and rate of change of the resultant acceleration extracted for each phase of lifting, across all repetitions. Filtering parameters, computation of variables and identification of lifting phases are as detailed previously (see 'Kinematics'). Variables for each set were time-normalized to 20% intervals (5 equally-spaced bins) and exported to a spreadsheet program (Microsoft Excel 2010, Microsoft Corp) prior to statistical analysis. Kinematic variables were exported as absolute values for between-group comparisons and as normalized-values for evaluation of within-subject effects. To evaluate the within-subject effect of frequency, values were normalized to the self-selected pace

condition (preferred strategy), and for the within-subject effect of cue type, to a standardized cue (the tone condition).

4. Statistical analysis

To evaluate the within-subject main effects of lifting frequency and cue type, a repeated measures analysis of variance (repeated measures) was used. Tests were carried out on values normalized to self-selected pace (frequency effect) or the standardized tone prompt (cue type effect). The kinematics of specific lifting phases (e.g. lowering, raising, etc) were modeled as a within-subject factor (PHASE), while the main CONDITION effect, representing the difference across frequencies or cue type, was obtained from the model intercept (e.g. %self or %tone, $H_0 = 1$). Post hoc comparisons on the normalized values are equivalent to paired tests on non-normalized values, with the added advantage of reducing between-subject variability due to individual differences in movement strategy. The interaction of BMI with these effects was not tested. Confidence intervals (95%) were constructed for significant within-subject effects.

The effect of BMI on was modeled as a between-subject factor (GROUP) using ANOVA; variations within a single set (TIME) and across lifting phases (PHASE) were set as within-subject factors. Post-hoc comparisons are two-sided, unless otherwise indicated. The difference in variability between groups (sphericity) was tested with Mauchly's test, and adjusted degrees of freedom/F-ratios are reported where appropriate (Greenhouse-Geisser correction), along with an estimate of effect size (n_p^2).

Significance was set at $p < .05$ for all tests. Statistical analysis was performed using SPSS 19.0 (IBM Corporation, Somers NY).

RESULTS

1. DESCRIPTIVE STATISTICS AND BMI EFFECTS

Normal (M = 9.1, SD = 1.9 lifts/min) and high BMI (M = 9.8, SD = 1.2 lifts/min) participants lifted at similar self-selected lifting frequency, $t(35) = 1.1$, $p > .28$.

1.1. Self-paced lifts

Although lifting frequencies were similar, high BMI participants executed the self-paced lifts a mean (SE) 0.23 (0.12) seconds faster than the normal BMI group, $F(1,33) = 3.25$, $p < .05$, $\eta_p^2 = .09$ (Figure 21, *total lift*).

Differences for individual lifting phases were significant for the trunk-lowering (M = -0.16, SE = .09 sec), $F(1,33) = 3.09$, $p < .05$, $\eta_p^2 = .09$, box-raising (M = -0.08, SE = 0.03 sec), $F(1,33) = 6.81$, $p < .01$, $\eta_p^2 = .17$, and upwards acceleration (B1) of the box (M = -0.07, SE = .02 sec), $F(1,33) = 7.21$, $p < .01$, $\eta_p^2 = .18$. The duration of the trunk-raising phase did not differ, $F(1,33) = 1.51$, $p = .12$ (Figure 21, *duration*).

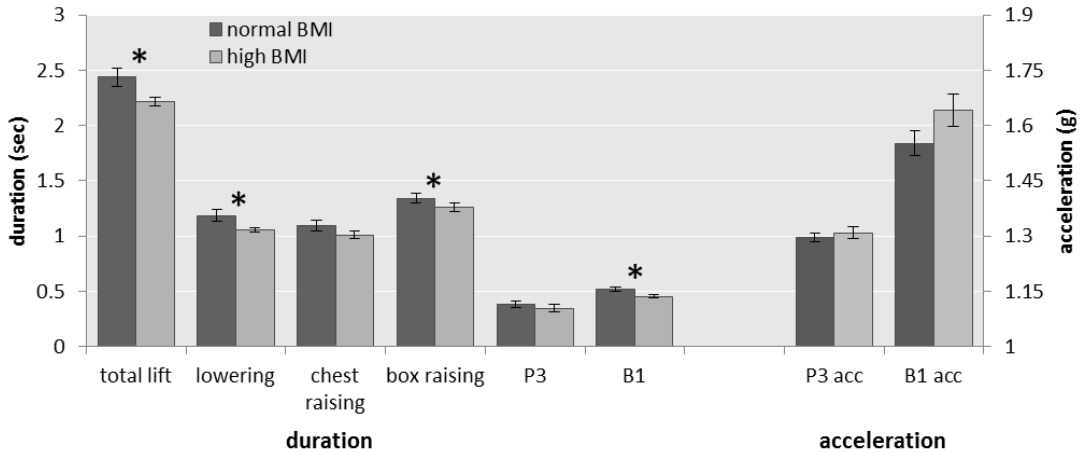


Figure 21. Duration and acceleration magnitude of lifting phases in high- and normal-BMI participants during self-paced (~9 lifts/min). * p < .05. The difference in B1 acceleration did not reach significance, p = .05.

Consistent with duration differences, there was a trend towards increased B1 magnitude for the high (M = 0.64, SE 0.05 g) compared to the normal BMI group (M = 0.54, SE = 0.18 g), $F(1,33) = 2.84$, $p = .05$, $\eta_p^2 = .08$ (two-sided) (Figure 21, *acceleration)*. This persisted for the difference in jerk (not shown) of the box between high (M = 9.50. SE = 2.50 g/sec) and normal BMI participants (M = 7.49, SE = 3.23 g/sec), $F(1,33) = 3.36$, $p > .08$ (two-sided). No differences were detected in the acceleration or jerk of other phases, $F(1,33) < 1$, $p > .32$.

1.2. Fixed-pace

Unlike the previous study (*The influence of body mass on lifting strategy)*, the effect of BMI on lift duration was limited to a shorter duration of the box upward acceleration (B1 phase) in the high (B1, M = 0.46, SD = 0.10 sec) compared to normal BMI participants (B1, M = 0.53, SD = 0.09 sec), $F(1,33) = 3.86$, $p < .05$, $\eta_p^2 = .10$. (Figure 22, *B1*). No differences were detected in the acceleration, $F(1,33) < 1.35$, $p > .25$, or jerk, $F < 1.3$, $p > .22$ (not shown).

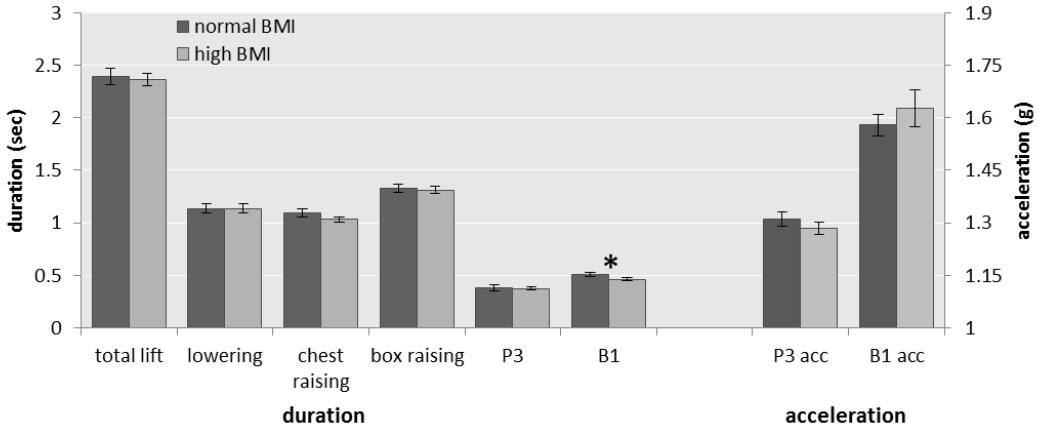


Figure 22. Duration and acceleration magnitude of lifting phases in high- and normal-BMI participants during fixed-pace lifts (4 lifts/min). * p < .05

Within-group post hoc comparisons revealed that high BMI participants increased the duration of lifts during the slower, fixed-pace condition (M = +0.14, SE = 0.05 sec), $t(9) = 2.75$, $p < .05$, while normal BMI participants maintained consistent lift durations across conditions, $t(24) < 1$, $p > .91$. In other words, the difference in BMI-related kinematics between self- and fixed-

paced conditions could be attributed to a change in the high BMI group, rather than normal BMI participants.

2. LIFTING FREQUENCY

As expected, lifting strategy varied across frequencies, evident by main effects for duration, $F(2,40) = 22.53$, $p < .001$, $n_p^2 = .53$, acceleration, $F(2,40) = 12.83$, $p < .001$, $n_p^2 = .38$, and jerk, $F(2,40) = 17.56$, $p < .001$, $n_p^2 = .47$.

The participants' self-selected lifting pace was intermediary (M = 9.2, SD = 1.8 lifts/min) of the low- (M = 4.2 SD = 0.1 lifts/min) and high-frequency, fixed-pace conditions, 12.0 (0.4) lifts/min ($p < .001$). Statistical tests for the effect of frequency are presented using normalized data (to the self-paced condition), which controls for the effect of BMI on kinematics (see above). The effect size and variance of post hoc comparisons are shown using 95% confidence intervals, where significant effects (relative to preferred pace) can be observed by intervals that do not cross zero.

2.1. Duration

A reduction in lifting frequency below the self-selected pace was not associated with a change in lift duration, $F(5,125) = 1.31$, $p > .27$. An increase in lifting frequency was sufficient to alter lift duration (i.e. speed up, M = -17.1, SE = 1.5%), $F(1,7) = 69.61$, $p < .001$, where the reduction was uniform across all phases, $F(2.1, 15.1) = 1.20$, $p > .33$ (Figure 23, *high pace*).

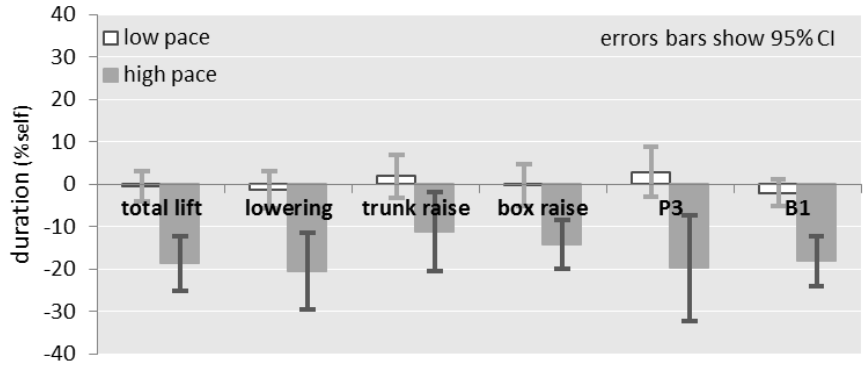


Figure 23. Effect of an increase (high-pace) and decrease (low-pace) in lifting frequency (relative to self-paced lifts) on total lift duration, and the duration of individual lifting phases (lowering, raise, P3, B1). The mean (95% CI) is shown, with significant effects evident by error bars that do not cross 0 (i.e. all high-pace effects comparisons)

2.2. Acceleration

Alterations to acceleration magnitude were also independent of frequency changes, however unlike duration, a trend towards altered acceleration magnitude during the low frequency condition was apparent (all phases, M = +6.5, SE = 3.6%), $F(1,25) = 3.50$, $p = .08$. Contrary to expectations of a reduction in acceleration due to lower lifting frequency, a substantial increase in acceleration of the P1 phase was detected (M = +26.4, SE = 6.8 %), $t(25) = 3.90$, $p < .001$ (Figure 24, *low pace*, P1). The effect was not uniform across phases, $F(2.4, 65.7) = 9.63$, $p < .001$, and did not persist for the subsequent portions of the lift, $t(25) < 1.31$, $p > .21$.

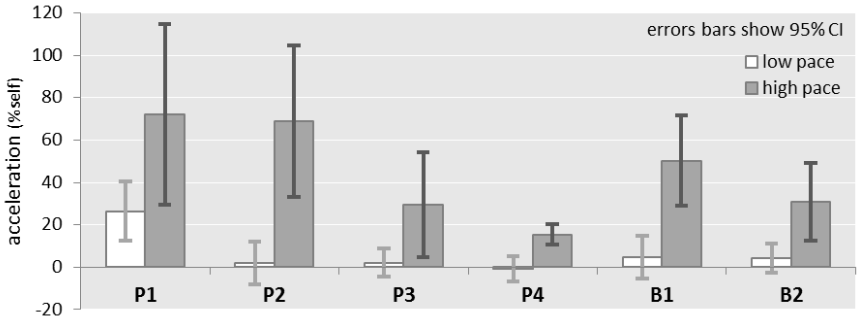


Figure 24. Effect of an increase (high-pace) and decrease (low-pace) in lifting frequency (relative to self-paced lifts) on acceleration magnitude of individual lifting phases. The mean (95% CI) is shown, with significant effects evident by error bars that do not cross 0 (i.e. all high-pace effects comparisons and P1 low-pace).

Not surprisingly, an increase in lifting frequency was accompanied by a sizeable increase in acceleration magnitude, (M = +44.5, SE = 9.4%), $F(1,7) = 43.97$, $p < .001$, however the

changes were not uniform across lifting phases, $F(2.27, 20.4) = 5.19$, $p < .05$, $n_p^2 = .37$. The increase was greatest for the lowering portion of the lift (P1 & P2, M = 70.4, SE = 12.4%), with a much smaller increase detected for the raising phases, (P3 & P4, M = 22.4, SE = 5.64%), $t(7) = 3.88$, $p < .01$. Similarly, participants increased the acceleration of the box (B1, M = 50.2, SE = 9.1%) to a greater extent than the trunk (P3, M = 29.5, SE = 10.4 $\%_{self}$), $t(7) = 4.67$, $p < .01$ (Figure 24, *high pace*).

2.3. Jerk

Similar to the acceleration response, a (contradictory) increase in jerk was apparent during the low-frequency condition, $F(1,25) = 13.30$, $p < .001$. This effect was more persistent than acceleration changes, with increases apparent in the initial acceleration phase (P1, M = +23.4, SE = 7.3 %), $t(25) = 3.2$, $p < .01$, as well as the subsequent slowing (P2, M = +15.2, SE = 6.5%) and speeding up phases (P3, M = +19.3, SE = 8.1%) phases (not shown). As expected, a substantial increase in jerk was detected across all phases during the high-frequency lifts (M = 67.5 , SE = 9.5%), $F(1,7) = 19.28$, $p < .01$ (not shown).

2.4. Effect of external pacing

In addition to the obvious contradictory increases in the acceleration (P1) and jerk (P1 – P3) during the low-frequency condition (relative to the higher frequency, self-paced condition), the effect of externally-paced lifting, independent of frequency, was tested for the high-frequency condition. Analysis of covariance revealed that controlling for the increased frequency, did not eliminate the changes to kinematics associated with high-pace condition, evident by significant main effects for the duration of trunk-raising, $F(1,6) = 9.99$, $p < .05$, $\eta_p^2 = .62$, and box-raising phases, $F(1,6) = 10.03$, $p < .05$, $\eta_p^2 = .63$. A significant effect also remained for the box acceleration, B1, $F(1,6) = 7.00$, $p < .05$, $\eta_p^2 = .54$, indicating that lifting frequency was insufficient to account for the variation in lifting strategy. The effect was near significant for the total lift duration, $F(1,6) = 3.36$, $p = .12$, $\eta_p^2 = .36$, as well as the magnitudes of other acceleration

phases (P1, P2 and P4, $F(1,6) > 3.11$, $p < .13$). The ANCOVA models were not significant for jerk, $F(1,7) < 1$, $p > .61$.

2.5. Variation across repetitions

Participants maintained a consistent strategy within sets, with the exception of a small reduction in lift duration (M = 4.4, SE = 1.6%) in the first 20% of the low-frequency, fixed-pace condition, $F(2.0, 16.3) = 3.77$, $p < .05$, $\eta_p^2 = .32$. Failing to account for this effect did not alter the significance of any main effects.

3. CUE TYPE

In comparison to a standardized cue (tone), a significant change in lifting kinematics was evident in response to the motion cue, $F(1,9) = 7.44$, $p < .05$, $n_p^2 = .45$ [duration], $F(1,9) = 11.13$, $p < .01$, $n_p^2 = .55$ [acceleration, jerk]. A near significant main effect was detected for the verbal cue, $F(1,17) = 3.60$, $p = .08$, $n_p^2 = .18$ [duration], $F(1,17) = 2.23$, $p = .15$ [acceleration, jerk], indicating the effect of external pacing on lifting kinematics was not equivalent across cue types.

3.1. Duration

When prompted by a motion cue (box arrival) participants executed the lifts a mean (SE) 4.7 (1.7)% slower (all phases) compared to a standardized cue (tone), however the effect was not equivalent across phases, $F(2.0, 17.7) = 4.30$, $p < .05$, $n_p^2 = .32$ (Figure 25, *motion cue*). Post hoc tests revealed a significant increase in duration for the total lift, (M = 5.9, SE = 2.0%), $t(9) = 2.90$, $p < .05$, which was restricted to the lowering, (M = 7.0, SE = 1.9%), $t(9) = 3.66$, $p < .01$, and not raising phase of the trunk, $t(9) < 1$, $p > .70$. The increase in box lifting duration (M = 5.1, SE = 2.5%) was near significant, $t(9) = 2.01$, $p = .07$.

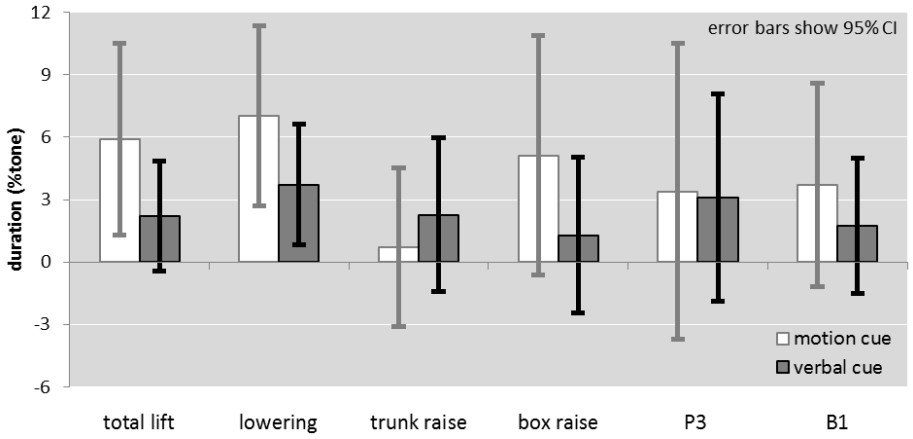


Figure 25. Effect of cue tupe (motion and verbal) relative to standardized cue (%tone) on total lift duration, and the duration of individual lifting phases (lowering, raise, P3, B1). The mean (95% CI) is shown, with significant effects evident by error bars that do not cross 0.

The near significant overall effect for the verbal cue (M = +2.4, SE = 1.2%) could be attributed to an increase in duration for the lowering (M = +3.7, SE = 1.4%), $t(17) = 2.72$, $p < .05$, and not the lifting phases of the trunk, $t(17) = 1.30$, $p > .21$, or the box, $t(17) < 1$, $p > .48$ (Figure 25, *verbal cue*, lowering).

3.2. Acceleration

Similar to the impact on duration, in response to a motion cue participants reduced the acceleration by a mean (SE) of 8.7 (2.2)%, which was not equivalent across phases, $F(2.4, 21.1) = 5.43$, $p < .01$, $n_p^2 = .38$ (Figure 26, *motion cue*). The reductions were significant for both lowering accelerations (P1: M = 22.0, SE = 4.4%; P2: M = 6.6, SE = 2.6%), $t(9) > 2.57$, $p < .05$, as well as the subsequent lifting acceleration (P3: M = 7.8, SE = 3.0%), $t(9) = 2.61$, $p < .05$, and were near significant for the final box acceleration (B2: M = 7.3, SE = 3.3%), $t(9) = 2.17$, $p = .06$. No change was detected for the final trunk (P4: $t(9) < 1$, $p > .63$) or initial box accelerations (B1: $t(9) < 1.43$, $p > .19$).

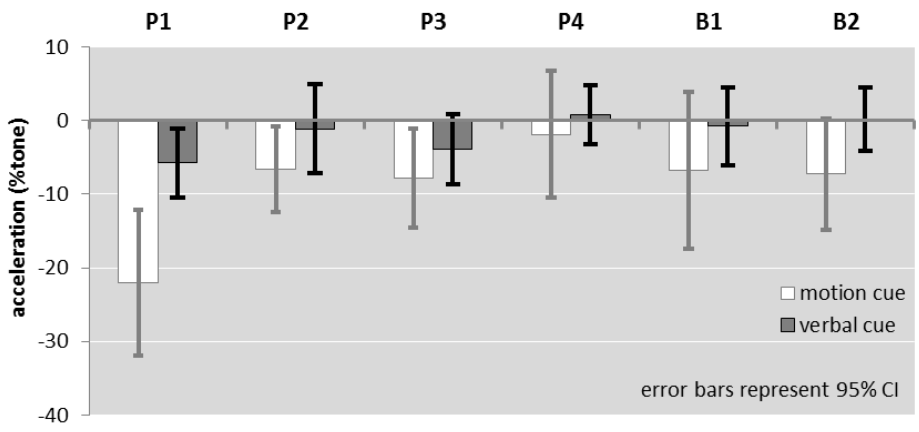


Figure 26. Effect of cue tupe (motion and verbal) relative to standardized cue (%tone) on acceleration of lifting phases. The mean (95% CI) is shown, with significant effects evident by error bars that do not cross 0.

Although the main effect for the verbal cue did not reach significance (across all acceleration phases), $F(5,85) = 1.37$, $p > .24$, individual tests revealed a significant reduction for the initial lowering acceleration (P1: M = +5.8, SE = 2.2), $t(17) = 2.61$, $p < .05$ (Figure 26, *verbal cue*, P1). This is consistent with the duration effects, but is not supported by the omnibus tests (above). The P3 phase was near significant, $t(17) = 1.72$, $p = .10$, while the P2 and P4 phases were not, $t(17) < 1$, $p > .71$.

3.3. Jerk

The mean (SE) reduction in jerk for the motion cue was 7.8 (2.6)%, an effect that, consistent with the magnitude, could be attributed to the lowering (P1: M = 21.0, SE = 5.3%; P2: M = 14.1, SE = 3.9%), $t(9) < 2.90$, $p < .01$) and initial lifting phases (P3: M = 9.1, SE = 3.6%), $t(7) = 2.54$, $p < .05$ (not shown).

Similar to above, the main effect was not significant for the verbal cue condition, $F(4, 68) < 1$, $p > .44$, but individual comparisons revealed differences for the P1 (M = 7.5, SE = 2.7%) , $t(17) = 2.78$, $p < .05$, and P2 phases (M = 5.9, SE = 2.5%), $t(17) = 2.33$, $p < .05$ (not shown).

3.4. Rate of box arrival

An evaluation of the effect of the rate of box arrival during the motion cue (i.e. how quickly the research assistant lowered the box) revealed a transient effect, localized to the lowering/initial portion of the lift. When the box was lowered at a maximal rate (last 2 reps), participants executed the lowering-phase of the subsequent lift a mean (SE) 5.9 (2.7)% faster, $t(9) = 2.59$, $p < .05$, and with trunk accelerations 12.0 (5.4)% higher, $t(9) = 2.22$, $p < .05$, than the lifts preceding (not shown). Similar to above, this effect did not persist into the subsequent phases, with participants reverting back to their preferred strategy for the raising-phase of the motion, all $t(9) < 1.14$, $p > .28$.

4. Summary of Findings

Lifting frequency was not a major determinant of lifting kinematics. Participants chose to maintain a preferred lifting duration and did not appear to deviate until task constraints demanded otherwise (i.e. an increase in frequency), however this effect was not uniform across all lifting phases.

A portion of the frequency effect could be attributed to the external pacing, independent of frequency. Further, the effect of external pacing was not consistent across cue types – the responses to both motion and verbal cues were of lower magnitude than a standardized audible tone prompt.

High and normal BMI individuals lifted at similar self-selected lifting paces, however the kinematics of individual lifts differed between groups, with high BMI participants preferring a shorter trunk-raising duration and increased acceleration of the distal segment/external load. An externally-paced, low-frequency lifting condition attenuated the difference between groups.

DISCUSSION

Alterations to lifting frequency did not have the anticipated effects on lifting kinematics, providing evidence that within a certain range of frequencies, individuals elect to lift with preferred set of kinematics. Not surprisingly, an increase to lifting frequency above a self-selected pace necessitated alterations to kinematics, however this was not apparent for lifts performed below the self-selected pace, and in fact, a portion of the external-pacing effect was independent of the lifting frequency. Further, the influence of external pacing was dependent upon the nature of the prompt used, where cues that enabled an individual to anticipate lift timing were found to have both attenuating and exacerbating effects, depending of specific cue parameters (i.e. rate of box arrival). The effects of external pacing on lifting kinematics is relevant to design of ergonomic interventions where individuals may not be lifting at a self-selected pace, and may also challenge the generalizability of certain findings from studies where lifting pace was externally-prompted.

Effect of frequency

Changes to lifting strategy, represented by accelerometer-derived kinematics, were independent of alterations to frequency (i.e. pace), particularly for reductions below an individual's self-selected pace. Although manipulation of lifting frequency for injury prevention efforts are generally intended to modify the work-rest ratio and minimize fatigue[51], it is not unreasonable to expect that individuals also alter lifting kinematics proportional to frequency[43], however this was not supported. The relatively invariant kinematics demonstrated in this study are similar to the findings of Marras[37], who found that back load did not differ across a range of lifting frequencies. These findings are relevant for design of injury prevention interventions – for instance, a reduction in lifting frequency might be desired to minimize fatigue, however if individuals maintained consistent lifting kinematics they may continue to be exposed to relatively high load during individual lifts. In fact, if the reduction in frequency was accomplished by

external pacing, the load during certain portions of the lift might actually be increased, as was shown for the acceleration and jerk magnitudes during the initial phases of the lift. Such an effect could increase the probability of an individual lift exceeding the yield point of lumbar tissue[249], resulting in injury, or might also be involved in increasing the probability of a motor control error[17]. It is increasingly apparent that modifications of task demands will not always have the intended effects, as has also been shown to occur in response to reductions in mass handled[44]. This supports the notion that individuals have a preference towards a fixed set of lifting kinematics (or effort/load) that they tend to adopt, regardless of changes to task constraints. These effects may partly explain why interventions intended to change lifting strategy (e.g. 'safe lifting' techniques) often have little impact on injury rates[32,234] (i.e. individuals revert to a preferred lifting strategy). Further investigations are required to identify specific lifting parameters that an individual seeks to maintain (e.g. kinematics, kinetics, exertion) in spite of changes to task demands.

Effect of external pacing

In spite of the widespread use of external-pacing during laboratory-based investigations[152,246,252], a paucity of information exists regarding the specific effect of external-pacing on lifting kinematics. While it was completely expected that a sufficient increase to lifting frequency (e.g. 12 lifts/min) would necessitate an alteration, the corresponding increases to kinematics reported here could not be explained by frequency alone – an effect that was even more apparent during low-frequency lifts where kinematic changes were in the opposite direction as anticipated (i.e. increased acceleration). The possibility of an external-pacing effect that is independent, or in addition to, changes in lifting frequency is relevant to any fixed-pace occupational task, where it might expose individuals to increased loading. This effect was particularly large for the initial lowering-phase of the lift, where a substantial (+25%) increase in acceleration magnitude was detected. The changes to kinematics did not persist into the

remainder of the lift (i.e. was not equivalent across lifting phases), with the most obvious explanation being that the effect of external pacing is temporally-mediated/of short duration. However, if temporal factors underlie the changes, then a similar increase would be expected (in the lifting phase) had participants initiated the motion from a crouched position – or more realistically, had the task involved lowering, rather than lifting. This is an important delineation to explore, as lowering tasks contribute to at least as many workplace injuries as lifting tasks[253,254] and an understanding of the factors that might increase injury risk in these tasks is important, particularly if they differ from those of raising tasks. Alternatively, the effect of external pacing may be reduced due to the addition of an external mass, however the increase in box acceleration observed here does not support this as an explanation for the reduction in external-pacing related kinematics. Further study is required to determine the impact of external-pacing kinematic changes to loading (one-time and/or cumulative), particularly if the effects persist beyond the small number of lifts examined here. Additionally, determining whether the effect is mediated by the addition of mass, temporal factors, lifting experience, or is dependent on other phase-specific (e.g. raising vs lowering) aspects of the lift is important to determine.

Effect of cue type

In addition to the main external pacing effect, the effect on kinematics was also dependent upon the type of prompt chosen. A variety of different prompts, or cue types, are utilized in investigations and daily tasks involving fixed-pace lifts (and daily tasks), including arrival of the external load[37], verbal prompts, or most often, audible tones[252]. Amongst the cue types examined here, the ubiquitous audible tone was associated with higher acceleration/jerk magnitudes and shorter lift durations. Considering the variety of cue types used in the literature, the influence of these effects on comparisons across studies is not known, but would be largest for comparisons between tasks using a 'motion cue' (load arrival) to those using a more standard audible tone. Although motion cues are less often used in the literature[37], they more closely

resemble the nature of repetitive lifting tasks in occupational or daily activities (e.g. when working in pairs, conveyor belts), reflecting that this is may be a more ecologically valid method of constraining lift frequency. While the changes in kinematics were minimal for the motion cue, further study is needed to determine whether this cue type eliminates the influence of external-pacing, thus enabling participants to lift using their preferred lifting strategy, or if it merely alters the effect of external pacing. The finding that an increase to the rate of box arrival produced an increase in kinematics refutes the possibility that motion cues eliminate external pacing effects. In fact, previous studies have demonstrated that individuals use kinematic characteristics of an object's arrival to make judgments about the object[181], and this study extends these findings by demonstrating that the kinematic characteristics (or associated judgments) can exert an influence on subsequent lifts.

Effect of BMI

The shorter lift duration and increased acceleration observed in high BMI participants relates well to previous investigations, however in the current study this effect was observed only at the higher, self-paced frequency and not the lower, fixed-pace frequencies that have been previously studied[23](*The influence of body mass on lifting strategy*). Considering the findings of the previous study, the most obvious explanation for the absence of a BMI-effect at lower-frequency lifts is the duration of the lifting task (12 repetitions vs 1-hour). However, a distinct difference in the underlying within-group effects was observed in the current study (a change by high-BMI participants only), which suggests BMI interacts differently to changes in task duration (*Influence of body mass on lifting strategy*) and lifting frequency (current study). Unfortunately, the influence of BMI on lifting kinematics has been examined only across a relatively narrow ranges of frequencies (4 to 12 lifts/min) and for relatively short durations (1 to 20 minutes) [23], which hinders attribution of these effects to a specific task parameter. This study, as well as those previous[23], indicate that the effect of BMI on lifting strategy is not a simple main effect, but is a

more complex interaction between task demands and body mass. This context-dependent effect of BMI may partly explain the findings of a positive association between BMI and low back injury in some epidemiological investigations[68] and not others[105]. Additionally, the external pacing studied here may be another example of an experimental constraint (similar to foot placement[46]), that may impact generalizability (i.e. attenuate or exacerbate experimental effects) and perhaps attenuate between-group effects (high vs normal BMI). Finally, an alternative explanation for the decreased lift duration in the high BMI individuals observed in this study and the one previous may be discomfort associated with observation – it is possible individuals with a high BMI may be psychologically, rather than physiologically, uncomfortable in certain positions (e.g. that might emphasize an abodominal pannus).

In addition to frequency, subsequent investigations of BMI (and cuing) should consider a wider range of external masses, which for studies examining BMI have been restricted to relatively low masses[23] (current study). In fact, the most prominent BMI-dependent difference in lifting strategy observed was for the external mass/distal segment, a finding that was apparent even during the low frequency, fixed-pace condition where the overall BMI-dependent influence was lower. Few studies have examined the effect of increased body mass on lifting [23,233], and none have reported on kinematics of the external load, however our findings indicate aspects of the external load are directly affected by morphology (*Effect of obesity on BSIPs* & *Influence of body mass on lifting strategy*). Similar to above, a differential increase in external load kinematics may be a strategy adopted to protect the low back region from increased mechanical load, however this appears to be at the expense of the upper extremity (i.e. increased torque at the glenohumeral joint). One explanation for increased kinematics of the distal segment is a greater reliance on inter-segmental torques to move the external mass, however this alteration has only been demonstrated in experienced materials handlers and for relatively light masses[235]. This hypothesis could be confirmed with more detailed kinematic and kinetic analyses of multiple

body segments (now that the effect of BMI on BSIPs has been considered)(*The influence of obesity on BSIPs*). Lastly, a change in mass (or frequency) might also be sufficient to elicit a difference in the self-selected pace between normal and high BMI individuals, as has been shown to exist during ambulation[109]. Future studies may want to consider heavier masses, longer durations, or perhaps even a fixed workload (rather than frequency) that an enable individual's to lift using their preferred strategy (e.g. a pallet unloading task[44]).

Limitations

One aspect of the current study that limits direct comparisons to existing literature is the methodological approach used to derive kinematics. Although wireless accelerometers can provide accurate, time-synchronized measures of net acceleration across multiple segments, contributions to the acceleration of individual body segments cannot be easily determined with systems of single sensors under dynamical conditions, as used here (i.e. a distal segment may be undergoing both angular and translational motions, the product of multiple interposed limb segments). As an example, Xu 2008[23] reported large BMI-dependent differences in peak trunk acceleration that were not detected here, however the investigators reported angular acceleration while those reported here were the resultant of linear and angular accelerations. These differences may not be inconsistent if participants altered the kinematics of interposed joints (e.g. the knee, as in a shift from a squat to stoop style lift), which could conceivably influence the duration of the acceleration (found here), independent of the magnitude.

Although this appears to be a limitation of using the resultant acceleration as an outcome, it is important to acknowledge resultant acceleration is a distinct parameter from angular acceleration, reflecting the net force acting on the segment/sensor (i.e. the end result of kinematics of individual joints). It is possible net force/resultant acceleration may be a variable individuals seek to maintain at a fixed level, regardless of changes to kinematics or task demands (and may be an interesting aspect of lifting in its own right). An important next step towards

understanding the effects of obesity during materials handling tasks is to perform a comprehensive biomechanical analysis on the distribution and magnitude of joint torques across multiple body segments, which would help elucidate the relevance of differences in kinematics reported here and in other studies[23].

Future studies may also wish to examine the influence of externally-paced lifts that occur at random intervals, rather than the fixed-pace lifts, as it is possible participants were able to anticipate the next lift, which may have attenuated the effects of cuing. More importantly, a direct comparison of external pacing and cuing effects should be performed at self-selected paces, which would better control for the effects of lifting frequency on preferred lifting strategy. While the indirect comparisons here support both the external-pacing and cue type effects, the findings would be strengthened (and verified) by matching externally-paced frequency to that preferred by the individual. Lastly, this design did not distinguish between the context of the verbal cue, and it is possible that similar to the motion cue, the specific parameters of the cue type (i.e. instructions) would impact the lifting strategy.

Overall Conclusions

The aim was to determine the influence of fixed-pace lifting on kinematics. The potential effects of fixed-pace lifting were partitioned into the effect of lifting frequencies (objective 1) - which was compared between high and normal BMI participants (objective 2) - and influence of the cue type used to prompt the lift (objective 3). The results of this study are consistent with the notion of preferred lifting kinematics, however the kinematics were influenced by the presence and format of external pacing/prompts. Two important implications were discussed: the effect of this previously unacknowledged source of variability on comparisons between studies, and the impact on generalizability/external validity of experimental tasks. Additionally, although only a small number of studies have considered the effect of BMI on lifting, it appears increased BMI is related to a preference for faster and/or higher acceleration kinematics, however this is unlikely to

be a simple main effect evident across all lifting scenarios. Future studies are required for a larger range of frequencies, masses and constrained/unconstrained circumstances. Additional studies are indicated on external pacing/cuing, particularly to determine whether the effects are persistent across a larger number of repetitions, and to better understand how cuing- or pacing-effects might manifest during both lowering and lifting tasks.

STUDY 4. ACTIVATION OF ERECTOR SPINAE DURING REPETITIVE TRUNK MOTION

METHODS

1. Participants

The sample consisted of 17 participants (male = 15, female = 2) a mean (SD) 27.3 (4.9) years of age, with a body mass of 76.6 (10.6) kg, height of 1.79 (0.08) m and body mass index of 23.9 (2.2) kg/m^2. Ethical approval was obtained from Bannatyne Campus Research Ethics Board, University of Manitoba (HREB#: H2010:408).

2. Protocol

Participants were positioned prone on a plinth (height of 65 cm), with the top of the iliac crest aligned with the edge of a table. Repetitive flexion and extension of the lumbar spine was performed by lowering (flexing) the trunk from a neutral position (0 degrees, midline of torso parallel to horizontal plane) towards a marker positioned at 45 degrees of flexion. The lower extremity was secured with a padded strap across the distal shank[59].

Participants were instructed to perform the motion at a 'smooth and steady' pace, with a visual demonstration of the movement frequency (0.33 Hz; 1.5 second raising and lowering phases)[6]. Motions began from the flexed position, with the upper extremities held against the upper torso and repetitions were performed to volitional fatigue. The protocol was terminated prior to volitional fatigue in two participants – due to a failure to maintain the range of motion and pace, respectively. No encouragement was provided, with the exception of verbal prompts regarding the top position of the trunk.

[6]An audible cue proved ineffective, with participants either drastically altering movement strategy (attempting to 'catch up' or 'wait' for the tone) or ignoring the cue and moving at a self-selected pace, an effect that became more apparent as fatigue progressed. Instead, the appropriate movement pace was demonstrated and a small number of repetitions, supported by the upper extremity, were performed for familiarization.

Prior to the trunk flexion-extension motions, a measure of maximal voluntary trunk extension torque was obtained - a padded strap affixed to a strain gauge (Intertechnology Model 60001-200) was secured over the mid-torso (T5 level) such that subjects were able to exert an extension torque with the torso slightly flexed (~10 degrees). Participants performed 3 maximal exertions in the trunk extension direction (5 seconds duration), interspersed with 2 minutes of rest, and followed by a further 5 minutes of rest. The protocol was repeated immediately following the flexion-extension trial.

3. Muscle Activation

Surface electromyogram signals were acquired from the lumbar and thoracic portions of the erector spinae, positioned at the L3 and T8 vertebral levels, corresponding to pars lumborum and thoracis, respectively. Electrode pairs were applied 3cm lateral to the spinous processes, parallel to the erector spinae muscle fibres and separated by approximately 10 cm (mid-line of electrodes aligned at vertebral level)[255]. Mechanomyogram signals (MMG) were also obtained for pars thoracis and lumborum using miniature uniaxial accelerometers (8 g, 15 x 15 x 7 mm, EGAS3, Measurement Specialties USA). Accelerometers were affixed using double-sided adhesive tape and positioned at the mid-point between the electromyogram electrode pairs, at the level of the L3 and T10 vertebral bodies. The MMG and EMG signals were sampled at 1000 Hz (Data Translation 21 Board 9800 series, 12 bit A-D converter) using programmable data acquisition software (Scope, version 2.2, Data Translation, Marlboro, Mass, USA).

4. Kinematics

A wireless tri-axial accelerometer (+/- 10 g, G-Link mXRS, Microstrain Inc, USA) was affixed to the upper trunk at the level of the C7 vertebral body. The orientation of the accelerometer was such that the superior-inferior axis was aligned parallel to the superior-inferior

axis of the trunk. The acceleration signal was sampled from each channel/axis at 512 Hz and stored in the device's onboard memory, prior to transfer to a computer following the trial (.CSV format).

Video was recorded with a digital camera (30 fps, 640 x 480 pixels; Casio EX100, Casio USA) positioned parallel to the sagittal plane, at the level of plinth. The video was used to synchronize the tri-axial accelerometer signal to the EMG and MMG signals (via a visual-mechanical artifact), and also to verify motion was occurring at the lumbar spine.

5. Processing

All data processing was carried out using programs and scripts written for GNU Octave (GNU General Public License, Free Software Foundation, Inc).

The EMG and MMG signals were high-pass filtered using a zero-lag (recursive), 4th order Butterworth filter at cutoff frequencies of 100 and 10 Hz, respectively[56]. The magnitude was determined as the root mean square (RMS) value for each phase of the motion, and the median frequency was derived from a fast fourier transform. Data from each subject was normalized to the maximal value obtained during MVC testing in order to derive a measure of absolute magnitude, and normalized to the value at the start of the trial in order to quantify the rate of change[56].

Tri-axial accelerometer data was band-pass filtered at 0.75 – 5 Hz to extract kinematics, and low-pass filtered at 0.75 Hz to extract orientation[239] (zero-lag, 4th order Butterworth). Orientation was converted from linear acceleration units (g's) to orientation using trigonometric functions (atan2 of vertical and horizontal axes) and used to derive range of motion, as well as the beginning of the raising (minimum orientation) and lowering phases (maximum orientation). The acceleration signal was converted from linear to angular units based upon the distance between

the sensor and axis of rotation (L3 spinous process) as measured with a cloth tape. Kinematic variables extracted for each phase (raising and lowering) included peak accelerations for speeding up and slowing down, velocity and duration.

Measures of muscle activity and kinematics were time-normalized to 15 equally-distributed bins across the duration of the trial. Time-normalized values were exported to a spreadsheet (Microsoft Excel 2010, Microsoft Corp) prior to statistical analysis.

Peak force was taken as the highest 1 second average obtained across the three repetitions and normalized to the maximal force of each participant.

6. Statistical Analysis

Differences in trunk kinematics between raising and lower phases were tested with a paired t-test. Two approaches were used to test and quantify changes in muscle activation. First, to test for a change in muscle activation, repeated measures ANOVA was performed. Repetitions were modeled as a within-subject effect (TIME) and muscle activity for the raising and lowering phases added as a PHASE effect. Estimates of effect size (n_p^2) are provided for significant main effects, interactions and contrasts. Sphericity was assessed with Mauchly's test and a Greenhouse-Geisser correction applied where indicated. In addition to standard post-hoc tests (e.g. across repetitions and/or versus the first repetition), a comparison of first and last repetition was also performed.

The second approach involved estimating the coefficients (slope and intercept) of linear regression models for each signal (EMG, MMG), phase (raising, lowering) and participant (Ebenichler), which provided a method of quantifying the magnitude and direction of the changes. Trials were partitioned based upon the contrasts and post-hoc tests from repeated measures, and regression parameters were estimated for each portion of the trial with a significant

effect detected with ANOVAs. The partitioning enabled non-linear variation across repetitions to be estimated with linear regression. Differences in activation were tested using paired t-tests on regression coefficients[207].

Significance was set at $p < .05$. Statistical analysis was carried out using SPSS 19.0 (IBM Corporation, Somers NY).

RESULTS

The mean (SD) repetitions to failure was 34.7 (12.5), corresponding to a mean (SE) exertion of (SE) 75.8% (6.8) of pre-trial isometric torque, $t(16) = 3.54$, $p < 0.001$, $r = 0.66$.

Participants moved the trunk through a mean (SD) 61.9 (6.3) degrees. The raising-phase had a slightly shorter duration than the lowering-phase (M = -0.38, SE = 0.08 sec), $t(15) = -4.51$, $p < .001$, $r = .76$ (Table 33). Increases were also detected for the raising-phase velocity (M = +9.2, SE = 1.7 deg/s), $t(15) > 5.57$, $p < .001$, $r > .82$, and acceleration (M = +48.7, SE = 7.4 and M = 28.7, SE = 5.7 deg/s^2), $t(15) = 6.74$, $p < .001$, $r = .87$. Although there was slight asymmetry between phases, the durations of each phase remained consistent throughout the trial, $F(1.5, 22.2) < 1.48$, $p > .25$.

Table 33. Kinematics (mean, SD) of the raising and lowering phases for a repetitive trunk flexion-extension exercise performed in a prone position.

	Raising	Lowering
duration (sec)	1.38 (0.22)	1.76 (0.47)**
velocity (deg/s)	46.4(7.5)	37.8 (10.3)**
acceleration, speed up (deg/s^2)	178.2 (55.6)	129.5 (44.7)**
acceleration, slow down (deg/s^2)	217.7 (55.0)	189.1 (59.0)*

mean (SD) shown; between phase differences as *p < .01 & **p < .001

The changes in position, velocity (decreased) and acceleration (decreased) over repetitions were consistent with fatigue (all p <.05), see Appendix: *Effect of repetitions on kinematics*)

1. ELECTROMYOGRAPHY

1.1. Frequency domain

Consistent with submaximal fatigue characteristics, EMG frequency during the raising (concentric) phase decreased across the trial for both pars lumborum [7], $F(1.79, 28.7) = 67.2$, $p < .001$, $\eta_p^2 = .81$, and thoracis , $F(1.4, 22.9) = 10.8$, $p < .001$, $\eta_p^2 = .40$, with substantial effect sizes for the respective linear contrasts, $F(1,16) = 93.3$, $p < .001$, $\eta_p^2 = .85$ and, $F(1,16) = 25.1$, $p < .001$, $\eta_p^2 = .61$ (Table 34, *raising*, [b]).

Table 34. Change in EMG frequency (median) of back extensor muscles (pars lumborum and thoracis) during the raising and lowering phases of a repetitive trunk flexion-extension task.

	start (M, SD)	end (M, SD)	change, % (M, SE)
Raising			
Thoracis (Hz)	98.2 (16.5)[a]	65.4 (7.4)	-32.1 (3.1)[b]
Lumborum (Hz)	115.7 (20.4)[a]	68.3 (12.7)	-37.1 (3.6)[b]
Lowering			
Thoracis (Hz)	94.1 (13.7)[a]	68.5 (8.4)	-26.1 (3.2)[b]
Lumborum (Hz)	116.5 (21.4)[a]	74.9 (15.5)	-31.9 (4.7)[b]

a: between-muscle difference, $p < .05$
b: within-muscle difference, $p < .001$

Similar effects were apparent for the lowering-phase in both pars lumborum , $F(1.9, 29.8) = 35.5$, $p < .001$, $\eta_p^2 = .69$ and thoracis $F(3.3, 52.5) = 37.7$, $p < .001$, $\eta_p^2 = .71$, with large effects for the linear contrasts, $F(1,16) = 81.3$, $p < .001$, $\eta_p^2 = .84$ and $F(1,16) = 53.1$, $p < .001$, $\eta_p^2 = .77$ indicating a substantial, uniform reduction over the course of the trial (Table 34, *lowering*, [b]).

The change in activation (EMG frequency) differed between pars thoracis and lumborum for both the raising, $F(1,16) = 13.7$, $p < .01$, $\eta_p^2 = .46$, and lowering-phases, $F(1,16) = 5.26$, $p < .05$, $\eta_p^2 = .25$. The absolute frequency (Hz) of pars lumborum EMG was greater than pars thoracis

[7] Sparkline of trend (*cf* Tufte, E 2006 *Beautiful Evidence* pp8-20, Graphics Press **ISBN-10:** 0961392177)

at the start of both the raising (M = +17.6, SE = 2.5 Hz) and lowering phases (M = +22.4, SE = 4.0 Hz), $t(15) > 6.99$, $p < .001$, but similar at the end, $t(15) < 1$, $p > .44$ (Table 34, [a]).

The mean (SE) rate of decline, represented by the mean (SE) slope of individual regression lines (b1), was slightly greater in pars lumborum compared to pars thoracis (b1, M = +0.7, SE = 0.3), during the raising phase, $t(15) = 2.72$, $p < .05$, $r = .57$ (Table 35, *raising*, b1 *), but did not reach significance for the lowering phase, $t(15) = 1.4$, $p > .17$. This was also associated with a better fit for past lumborum during the raising phase (Table 35, r^2). Consistent with normalized data, the intercept (Table 35, b0), was near 100 for both muscles and did not differ.

Table 35. Regression coefficients describing the frequency domain changes of back extensors (pars lumborum and thoracis) for the raising and lowering phases of a repetitive trunk flexion-extension task.

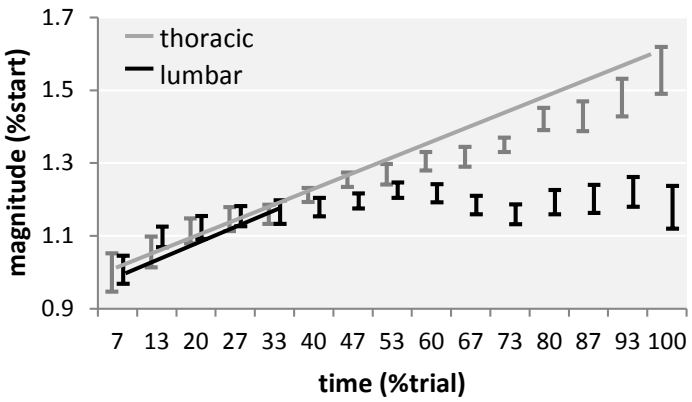
	raising	lowering
b1, *thor*$^+$	-2.0 (0.4)*	-1.9 (0.2)*
b1, *lumb*$^+$	-2.7 (0.3)*	-2.4 (0.3)*
b0, *thor*$^+$	100.3 (1.5)	102.9 (2.1)
b0, *lumb*$^+$	98.7 (1.6)	101.0 (1.9)
r^2, *thor*$^+$	.64 (.01)	.66 (.01)
r^2, *lumb*$^+$	.80 (.01)	.68 (.01)

*p < .05; +values are M (SE)

1.2. Time domain

1.2.1. Raising-phase

The raising-phase (concentric) EMG magnitude (RMS) of pars thoracis increased throughout the trial  , $F(2.1, 33.1) = 17.6$, $p < .001$, $\eta_p^2 = .52$, (linear contrast: $F(1,16) = 26.6$, $p < .001$, $\eta_p^2 = .63$). No main effect was detected for pars lumborum  , $F(1.8, 29) = 1.3$, $p > .29$, $\eta_p^2 = .07$, however it could be fit with a cubic model, $F(1,16) = 9.5$, $p < .01$, $\eta_p^2 = .37$, with significant between-repetition contrasts for the initial third (33%) of the trial, $F(1,15) > 5.3$, $p < .05$, $\eta_p^2 > .26$ (Figure 27, *left*).



	start – 33%	67 - end
b1, *thor*$^{+}$	3.6 (0.7)*	
b1, *lumb*$^{+}$	3.7 (0.9)*	-0.1 (0.9)
b0, *thor*$^{+}$	98.7 (3.5)	
b0, *lumb*$^{+}$	99.7 (1.6)	119.9 (6.8)
r^2, *thor*$^{+}$	.58 (.01)	
r^2, *lumb*$^{+}$	.33 (.02)	.02 (.03)

*p < .05; +values are M (SE)

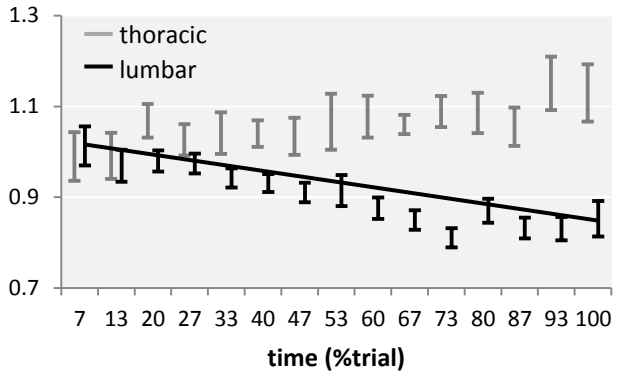
Figure 27. Change in EMG magnitude of back extensors (pars lumborum and thoracis) for the raising phase of a repetitive trunk flexion-extension task. Mean (SE) are plotted (left) and regression parameters are provided (inset table).

The increases in raising-phase EMG of pars lumborum (M = 15.0, SE = 3.6 $\%_{start}$) and thoracis (M = 16.0, SE = 4.3 $\%_{start}$) during the initial 33% of the trial were similar, $t(15) < 1$, $p > .75$ (Figure 27, *right*, b1), after only pars thoracis continued to increase, ending a mean (SE) 55.5 (10.6)% higher than the start of the trial, $t(15) = 5.3$, $p < .001$, $r = .82$.

1.2.2. Lowering-phase

Conversely, pars thoracis EMG remained unchanged for the lowering (eccentric) phase , $F(2.9, 47.4) = .87$, $p > .59$, while pars lumborum decreased , $F(4.4, 70.3) = 5.6$, $p < .001$, $\eta_p^2 = .26$, (linear contrast: $F(1,16) = 18.7$, $p < .001$, $\eta_p^2 = .54$).

Consistent with ANOVA, a significant regression model was returned for the lowering-phase EMG of pars lumborum only (b1, M = -1.3, SE = 0.3, $r^2 = .14$), and not for pars thoracis ($r^2 = .02$). The decrement in pars lumborum was a mean (SE) 16.6 (5.7) %, over the trial, $t(15) = 2.8$, $p < .01$, $r = .59$ (Figure 28, *right*, b1).



	start – end
b1, *thor*$^{+}$	0.8 (0.7)
b1, *lumb*$^{+}$	-1.3 (0.3)*
b0, *thor*$^{+}$	99.4 (3.8)
b0, *lumb*$^{+}$	100.9 (3.2)
r^2, *thor*$^{+}$	.02 (.02)
r^2, *lumb*$^{+}$	.14 (.01)

*p < .05; +values are M (SE)

Figure 28. Change in EMG magnitude of back extensors (pars lumborum and thoracis) for the lowering phase of a repetitive trunk flexion-extension task. Mean (SE) are plotted (left) and regression parameters are provided (inset table).

1.2.3. Absolute magnitudes

At the beginning of the trial, the raising-phase EMG magnitude of pars lumborum (M = 80.4, SD = 32.8 $\%_{MVC}$) was greater than pars thoracis (M = 64.8, SD = 24.7 $\%_{MVC}$) as expected, $t(15) = 4.68$, $p < .001$, $r = .77$, however the activation at the end of the trial was not different, $t(15) = 1.4$, $p > .17$ (Table 36, *raising*, [a]).

Table 36. Absolute value of EMG magnitude of back extensors (pars lumborum and thoracis) for the lowering and raising phases of a repetitive, trunk flexion-extension motion. The change in activation of pars lumborum through initial 33% of the trial is provided, based upon post hoc comparisons (above).

	start (M, SD)	33% (M, SD)	end (M, SD)
Raising			
thoracis (%mvc)	64.8 (24.7)[a, b]	*na*	101.6 (34.2)[b]
lumborum(%mvc)	80.4 (32.8)[a, b]	93.2 (41.5)[b]	91.3 (33.9)[b]
Lowering			
thoracis (%mvc)	39.9 (19.0)[a, b]	*na*	40.8 (16.6)[b]
lumborum(%mvc)	46.5 (20.6)[a, b]	*na*	37.4 (19.3)[b]

a: between-muscle difference
b: within-muscle difference

Similarly, the lowering-phase activation (EMG) of pars lumborum (M = 46.5, SE = 20.6 $\%_{MVC}$) was greater than pars thoracis (M = 39.9, SE = 19.0 $\%_{MVC}$) at the start of the trial, $t(15) = 2.35$, $p < .05$, $r = .52$, but not the end, $t(15) < 1$, $p > .47$ (Table 36, *lowering*, [a]). The activation of

pars lumborum and thoracis during the lowering-phase were a mean (SE) 50.8 (5.4) and 42.6 (4.2)% less than during the raising-phase.

2. MECHANOMYGRAPHY

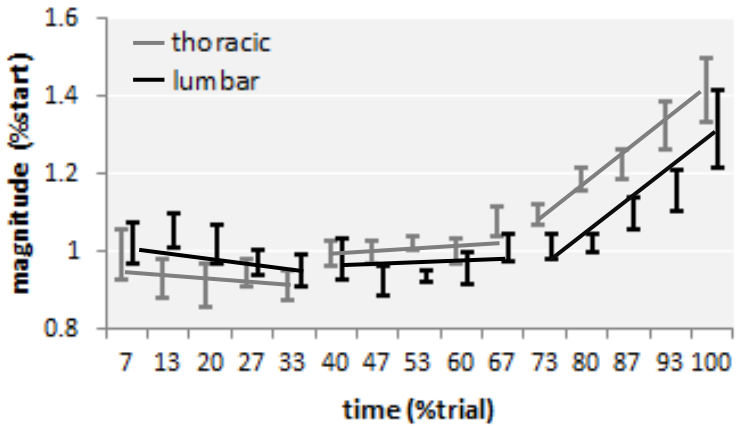
2.1. Time domain

2.1.1. Raising-phase

A significant increase was detected in the raising-phase MMG magnitude across repetitions for pars lumborum ~, $F(2.1, 32.9) = 4.2$, $p < .05$, $\eta_p^2 = .21$ and thoracis ~, $F(3.2, 33.1) = 11.2$, $p < .001$, $\eta_p^2 = .41$. Between-repetition contrasts for pars lumborum were significant in the final 28% of the trial, $F(1,16) > 2.5$, $p < .05$, $\eta_p^2 = .13$ to .22, and the final 47% for pars thoracis, $F(1,16) > 2.1$, $p < .05$, $\eta_p^2 = .12$ to .44.

Partitioning the trial into thirds (based upon between-repetition contrasts) revealed no change in pars thoracis MMG ($p > .55$), and a small reduction for pars lumborum (M = 6.9%, SE = 3.8) through the initial 33% of the trial (Figure 29, *bottom*, b1). The activation of both muscles remained unchanged through the mid-third of the trial ($r^2 < .02$), and increased in the final third ($r^2 > .30$), with pars lumborum increasing by a mean (SE) of 30.1 % (8.8), $t(15) = 3.42$, $p < .01$, $r = .66$ and pars thoracis by 31.9 % (7.8), $t(15) = 4.11$, $p < .001$, $r = .73$.

The change in EMG magnitude of pars thoracis and lumborum during the raising phase differed, $F(1,15) = 5.9$, $p < .05$, $\eta_p^2 = .27$, however the effect was localized to the initial 33% of the trial, with no difference in regression parameters detected for the middle and final portions, $t(15) = 1.08$, $p > .30$ (Figure 29, *bottom*, b1, r^2).



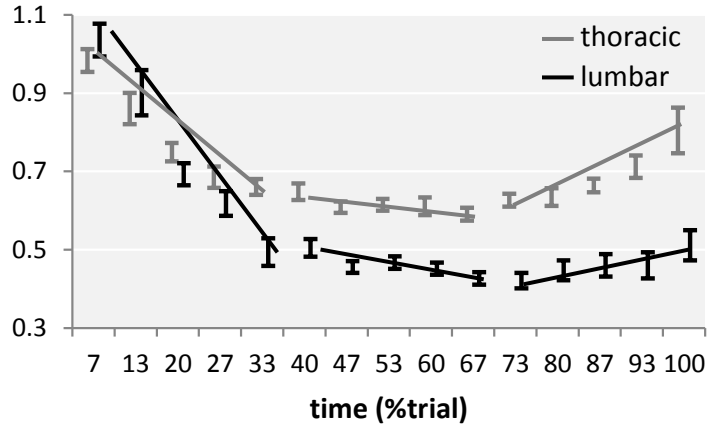
	start – 33%	40 – 67%	73 – 100%
b1, *thor*$^{+}$	-1.5 (1.4)	1.6 (1.5)	6.7 (1.4)*
b1, *lumb*$^{+}$	-2.2 (1.0)*	0.9 (1.8)	.4 (2.1)*
b0, *thor*$^{+}$	98.4 (4.2)	88.8 (7.8)	24.2 (6.7)*
b0, *lumb*$^{+}$	107.1 (3.2)*	88.9 (12.3)	16.5 (7.4)*
r^2, *thor*$^{+}$	.05 (.03)	.02 (.03)	.30 (.01)*
r^2, *lumb*$^{+}$	.09 (.02)*	.01 (.03)	.35 (.01)*

*p < .05; +values are M (SE)

Figure 29. Change in MMG magnitude of back extensors (pars lumborum and thoracis) for the raising phase of a repetitive trunk flexion-extension task. Mean (SE) are plotted (top) and regression parameters are provided (bottom) for each third of the trial.

2.1.2. Lowering-phase

A significant decrement was detected in the lowering-phase MMG magnitude of both pars lumborum ⌣, $F(3.6, 35.8) = 33.7$, $p < .001$, $\eta_p^2 = .68$ and thoracis ⌣, $F(1.5, 24.5) = 3.7$, $p < .05$, $\eta_p^2 = .19$. Between-repetition contrasts revealed it was localized to the initial 33% of the trial, $F(1,16) > 7.2$, $p < .05$, $\eta_p^2 > .31$ (both muscles), evident by substantial quadratic models for lumborum, $F(1,16) = 192.2$, $p < .001$, $\eta_p^2 = .93$, and thoracis, $F(1,16) = 106.9$, $p < .001$, $\eta_p^2 = .87$. The change in activation differed between muscles, $F(1,15) = 9.6$, $p < .01$, $\eta_p^2 = .38$.



	start – 33%	40 – 67%	73 – 100%
b0, *thor*$^{+}$	103.4 (4.3)*	70.5 (7.8)*	12.5 (15.9)*
b0, *lumb*$^{+}$	115.8 (6.3)*	58.9 (12.3)*	20.8 (8.8)*
b1, *thor*$^{+}$	-8.2 (0.8)*	-1.1 (0.8)	4.3 (1.3)*
b1, *lumb*$^{+}$	-13.7 (1.2)*	-1.6 (0.7)*	1.9 (0.7)*
r^2, *thor*$^{+}$	.66 (.01)*	.04 (.02)	.25 (.02)*
r^2, *lumb*$^{+}$	.85 (.01)*	.22 (.01)*	.16 (.02)*

*p < .05; +values are M (SE)

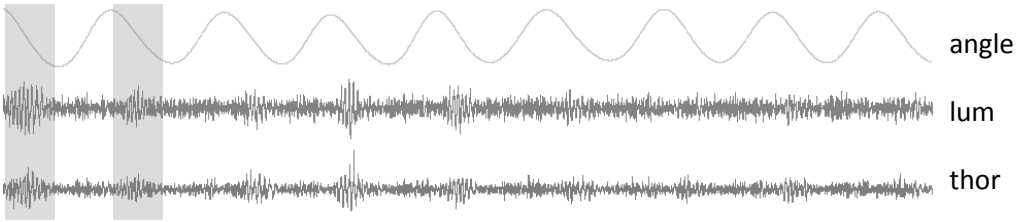


Figure 30. Change in MMG magnitude of back extensors (pars lumborum and thoracis) for the lowering phase of a repetitive trunk flexion-extension task. Mean (SE) are plotted (top) and regression parameters are provided (middle) for each third of the trial. A representative sample of raw MMG data is provided for the first 8 repetitions (bottom), where a visible reduction in amplitude during the lowering phase (highlighted) can be observed.

The reduction in MMG magnitude during the initial third of the trial was substantial for both pars lumborum (M = -53.1, SE = 4.8 %) and pars thoracis (M = -33.5, SE = 3.5 %), $t(15) > 9.59$, $p < .001$, $r > .93$ (Figure 30, *top*). The rate of reduction was greater in pars lumborum, (b1, M = +4.9, SE = 0.1), $t(15) = 4.51$, $p < .001$, $r = .76$, and was more persistent, taking a mean (SE) 2 (1) more repetitions to achieve an 80% decline (M = 12, SD = 4 reps / M = 38.7, SE = 3.6 $\%_{trial}$) compared to pars thoracis (M = 10, SD = 4 reps / M = 32.3, SE = 3.6 $\%_{trial}$), $t(15) > 2.48$, $p < .05$.

A small decline in pars lumborum MMG (M = -7.8, SE = 2.8%) persisted through the middle third of the trial (Figure 30, b1), $t(15) = 2.79$, $p < .05$, $r = .58$, but not for pars thoracis ($r^2 = .04$). This was followed by a moderate increase for both pars lumborum (M = 9.0, SE = 2.8 $\%_{rep1}$) and thoracis (M = 17.8, SE = 6.3 $\%_{rep1}$), $t(15) > 2.81$, $p < .05$, $r > .58$, with the rate of change (b1) greater in pars thoracis, $t(15) = 1.97$, $p < .05$, $r = .45$ (Figure 30, b1).

2.1.3. Absolute magnitude

Similar to the EMG signal, pars lumborum MMG magnitude was greater than thoracis during both the raising (M = +33.5, SE = 13.3 $\%_{MVC}$), $F(1,14) = 4.73$, $p < .05$, $\eta_p^2 = .25$, and lowering-phases (M = +57.2, SE = 20.5 $\%_{MVC}$, $F(1,15) = 4.73$, $p < .05$, $\eta_p^2 = .25$, with the differences significant at each third of the trial, $t(15) > 2.52$, $p < .05$ (Table 37, *raising*, [a]). However, unlike EMG, the magnitude of MMG signal was greater during the lowering-phase in both pars lumborum (M = +139.4, SE = 22.2 $\%_{MVC}$) and thoracis (M = +80.5, SE = 9.3 $\%_{MVC}$), compared to the raising-phase (Table 37, *lowering*, [a]).

Table 37. Change in absolute MMG magnitude of back extensors (pars lumborum and thoracis) during the raising phase and lowering phase of a repetitive, trunk flexion-extension motion.

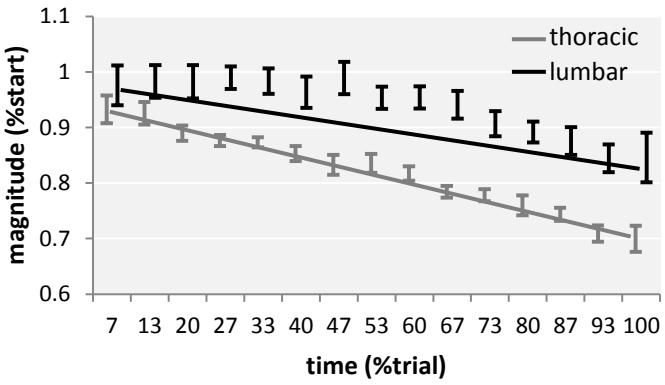
	start (M, SD)	33% (M, SD)	67% (M, SD)	end (M, SD)
Raising				
thoracis (%mvc)	77.7 (40.2)[a]	75.5 (37.3)[a]	78.1 (32.9)[a]	103.4(45.2)[a,b]
lumborum(%mvc)	118.7(65.1)[a,b]	110.1(59.1)[a]	108.7(53.5)[a]	134.7(60.1)[a,b]
Lowering				
thoracis (%mvc)	113.5 (52.8)[a,b]	73.3 (36.9)[a]	65.5 (27.5)[a]	90.2 (44.8)[a,b]
lumborum(%mvc)	268.1 (160.7)[a,b]	126.9 (90.5)[a,b]	98.9 (63.7)[a,b]	114.6 (69.1)[a,b]

a: between-muscle difference
b: within-muscle difference

2.2. Frequency domain

2.2.1. Raising-phase

The raising-phase frequency changes were the inverse of the magnitude changes, with decrements for both pars lumborum, ⁓, F(3.0, 48.5) = 3.42, p < .05, η_p^2 =.18 and thoracis ⁓, F(14, 210) = 18.6, p < .001, η_p^2= .55. Linear contrasts returned the greatest effect for pars lumborum, F(1,16) = 5.9, p < .05, η_p^2 = .24 and thoracis, F(1,16) = 85.3, p < .001, η_p^2 = .85).



	start – end
b1, *thor*$^+$	-1.6 (0.2)*
b1, *lumb*$^+$	-1.1 (0.4)*
b0, *thor*$^+$	95.1 (3.5)
b0, *lumb*$^+$	102.4 (3.0)
r^2, *thor*$^+$	.58 (.01)*
r^2, *lumb*$^+$	.12 (.02)*

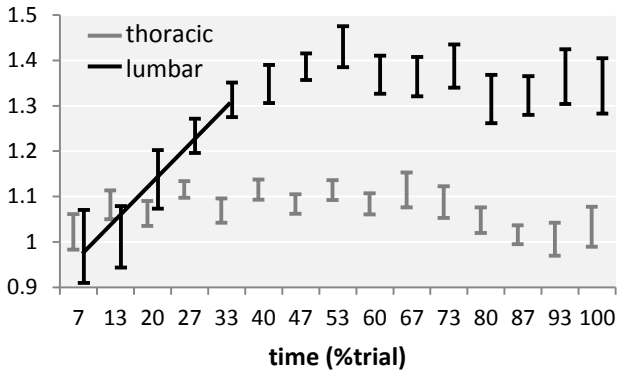
Figure 31. Change in MMG frequency of back extensors (pars lumborum and thoracis) for the raising phase of a repetitive trunk flexion-extension task. Mean (SE) are plotted (left) and regression parameters are provided (inset table).

The decrement in raising-phase MMG frequency was apparent throughout the trial, evident by the single regression models and linear contrasts (Figure 31, *left*). A significant between-muscle effect was detected in ANOVA, F(1,15) = 10.55, p < .01, η_p^2 = .41, however the slighthly greater reduction in rate (b1, M = +0.5, SE = 0.2) and magnitude for pars lumborum (M = 10.3, SE = 7.0 $\%_{start}$) did not reach significance, *t*(15) = 1.70, p = .06 and *t*(15) = 1.89, p = .08, respectively.

2.2.2. Lowering-phase

During the lowering phase, only MMG frequency of pars lumborum changed ⁓, F(3.2, 51.3) = 6.3, p < .001, η_p^2 = .28, with between-repetition contrasts significant through the first 33% of the trial, F(1,16) > 3.0, p < .05, η_p^2 > .16 , and a corresponding quadratic contrast of

$F(1,16) = 12.2$, $p < .01$, $\eta_p^2 = .43$). Pars thoracis remained constant ~~~~, $F(3.9, 59.2) = 1.40$, $p > .25$.



	start – 33%	67 - end
b1, *thor*$^+$	2.6 (0.3)	
b1, *lumb*$^+$	8.0 (2.2)*	-0.5 (0.8)
b0, *thor*$^+$	108.9 (2.6)	
b0, *lumb*$^+$	89.3 (4.0)*	141.8 (12.7)*
r^2, *thor*$^+$	.03 (.01)	
r^2, *lumb*$^+$	.36 (.01)*	.01 (.02)

*p < .05; +values are M (SE)

Figure 32. Change in MMG frequency of back extensors (pars lumborum and thoracis) for the lowering phase of a repetitive trunk flexion-extension task. Mean (SE) are plotted (left) and regression parameters are provided (inset table) for the initial 33% and final 67% of the trial (pars lumborum) and entire trial (pars thoracis).

Consistent with ANOVA, a significant regression model was returned for the lowering-phase pars lumborum MMG frequency in the initial third of the trial (Figure 32, *right*, b1), with a mean (SE) increase of 35.8 (9.9) $\%_{rep1}$, $t(15) = 3.61$, $p < .01$, $r = .68$. No significant models were returned for pars thoracis ($r^2 = .03$), nor for the remainder of the trial in pars lumborum ($r^2 = .01$).

2.2.3. Absolute magnitude

Similar to the time domain signals, the raising-phase pars lumborum MMG frequency (M = 45.2, SD = 7.6 Hz) was greater than pars thoracis (M = 40.1, SD = 10.6), $F(1,15) = 5.39$, $p < .05$, $\eta_p^2 = .26$. The absolute frequencies of the muscles were similar at the start, $t(15) < 1$, $p > .85$, with a near significant difference at the end of the trial, $t(15) = 1.8$, $p = 0.08$ (Table 38, *raising* ,[a]). During the lowering-phase, pars lumborum frequency (M = 48.0, SE = 21.4 Hz) was also higher than pars thoracis (M = 36.7, SE = 7.1 Hz), $F(1,15) = 4.37$, $p < .05$, $\eta_p^2 = .23$, with no between-muscle effects at the start, $t(15) = 1.1$, $p > .30$, but a mean (SE) difference of 16.5 (6.4) Hz at the end of the trial, $t(15) = 2.59$, $p < .05$ (Table 38, *lowerin*, [a]).

Table 38. Change in absolute MMG frequency of back extensors (pars lumborum and thoracis) during the raising and lowering phase of a repetitive, trunk flexion-extension motion.

	start (M, SD)	33% (M, SD)	end (M, SD)
Raising			
thoracis (Hz)	46.7 (13.0)[b]	*NA*	34.0 (5.9)[b]
lumborum (Hz)	47.0 (12.1)[b]	*NA*	39.4 (12.4)[b]
Lowering			
thoracis (Hz)	37.7 (14.3)	*NA*	35.8 (12.2)[a]
lumborum (Hz)	32.9 (10.4)[b]	46.5 (21.5) [b]	50.7 (33.1)[a]

a: between-muscle difference
b: within-muscle difference

2.2.5 Alternative models and summary

Although the frequency delineations for partitioning the trial were based upon statistical methods (i.e. within-rep contrasts), visual inspection of the pars lumborum MMG signal revealed (perhaps) different cut-points. For instance, the raising-phase MMG magnitude appears flat for the initial half (b1, M = 0.00, SE = 0.71, r^2 = .01), at which point an observable decrease is apparent (b1, M = -1.80, SE = 0.66, r^2 = .22) (Figure 29). This effect increases the between-muscle difference during the first half, $t(15) = 4.12$, $p < .01$, but not for the latter, $t(15) < 1$, $p > .89$. Likewise, during the eccentric-phase (Figure 32), visual cut-points would extend the increase through to 53% of the trial, resulting in a small reduction in the slope and slight improvement in the regression model (M = 6.7, SE = 1.7, r^2 = .44).

A summary of the time and frequency domain changes in the EMG and MMG signals of pars thoracis and lumborum, partitioned by phase/contraction type (raising/concentric; lowering/eccentric) is provided in Figure 33. The independence of EMG and MMG signals and substantial reduction in MMG magnitude during the initial portion of the trial is clearly illustrated. Additionally, for the majority of the periods with MMG magnitude changes (e.g. end

of trial raising/concentric, beginning of trial lowering/eccentric) inverse changes in the frequency domain are apparent (and supported based upon the ANOVA and regression results above).

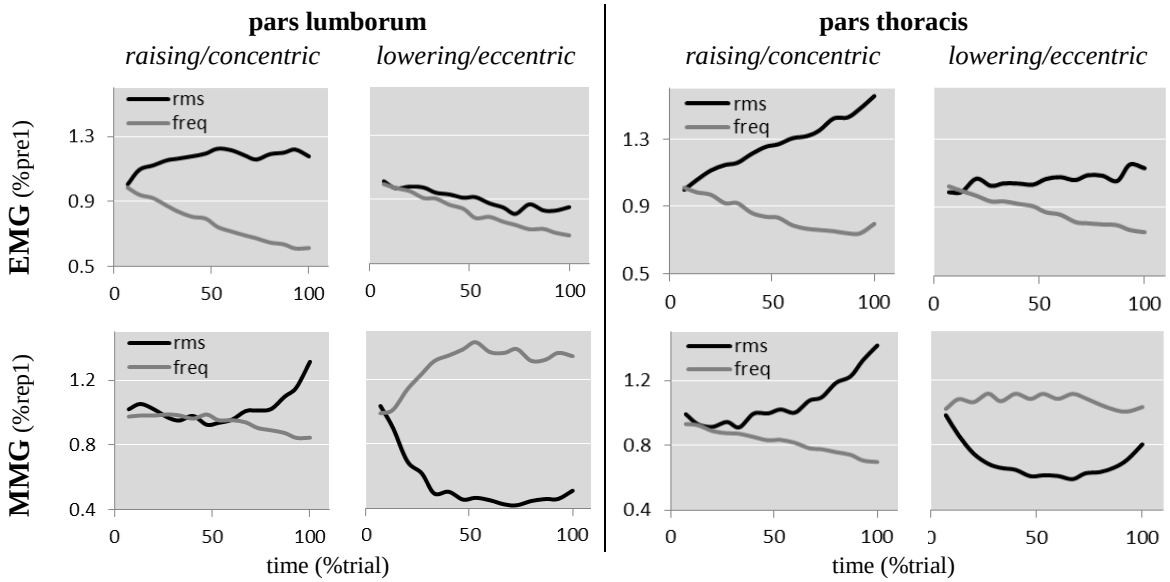


Figure 33. Summary of EMG (top row) and MMG (bottom) changes of erector spinea muscles during repetitive trunk motions, partitioned by phase (raising/lowering). Time domain (RMS) changes are shown in black and frequency domain (median frequency) in grey. Series are time-normalized, with magnitudes are normalized to beginning of trial (%rep1).

3. Summary of Findings

A repetitive trunk flexion-extension task was effective at eliciting fatigue in lumbar and thoracic portions of the erector spinae during a motion involving concentric and eccentric contractions.

A substantial difference in electrical (EMG) and mechanical (MMG) signals associated with muscle contraction was demonstrated during a repetitive trunk extension task.

The dissociation was due largely to a time- and contraction-type dependent alteration in MMG amplitude, particularly evident by a large reduction in the initial 1/3 of trial for the portion of the motion involving eccentric contractions.

The change in activation (EMG and MMG) differed between muscles, which was evident by larger magnitude and greater reduction in EMG median frequency in pars lumborum. This was associated with a correspondingly greater initial decrement in MMG signal amplitude, but a lower end-of-trial increase.

DISCUSSION

Activation of back extensor muscles during repetitive trunk motion was quantified using concurrent electrical (EMG) and mechanical (MMG) techniques, which revealed differential changes over the course of the trial. The most notable instance was a substantial reduction in MMG magnitude for the lowering-phase of the motion (eccentric), localized to the initial portion of the trial and not evident in surface EMG. A standardized experimental task was chosen that enabled the effect of contraction-type to be examined, reflecting the function of back extensor muscles during many daily lifting tasks. The results demonstrate that activation of lumbar extensor muscles during dynamic conditions is dependent upon contraction type and temporal factors. This has relevance for applications dependent upon knowledge of task-specific muscle activation, such back injury mechanisms, identification of disordered motor control related to back injury/pain, and injury prevention interventions.

MMG

Initial change

In contrast to EMG changes which followed relatively predictable, but task-dependent patterns (see below), analysis of the MMG revealed a substantial dissociation between the electrical and mechanical events associated with muscle contraction. This was particularly evident for the lowering (eccentric) phase, where the MMG amplitude of both pars lumborum and thoracis declined substantially during the initial 33% of the trial. Previous investigations of erector spinae during submaximal (isometric) fatigue have also reported dissociation between EMG and MMG, however the discrepancy arose due to an increase in EMG magnitude and an absence of change in MMG[217]. In contrast, the MMG changes during the raising (concentric) phase were similar to previous isometric conditions (i.e. a gradual, non-linear increase)[216]. Substantial reductions in MMG magnitude have been demonstrated previously during both

concentric[202] and eccentric[212] (maximal) fatiguing contractions of peripheral muscle groups, however unlike the submaximal conditions considered here, the decrement in MMG was accompanied by the expected reduction in EMG amplitude and paralleled the decline in voluntary torque production. A decrement in quadriceps MMG has also been reported during submaximal cycling tasks[210], however in this case the rate of reduction was gradual and persisted over the entire trial, which involved concentric contractions and a substantially longer time to fatigue (9 – 15 minutes). In fact, when participants pedaled at higher intensities (time to fatigue of ~4 min), the reduction in MMG amplitude was no longer observed[210]. The reduction in ES MMG magnitude in the present study was shown to be independent of EMG, specific to contraction type (eccentric only) and relatively rapid (occurring by 33 – 39% of trial duration). A similar reduction has not been reported in previous studies of MMG changes during fatigue, which have considered only maximal dynamic conditions[202,212] or submaximal isometric contractions[56].

Frequency domain

The linear decline in MMG frequency observed for the concentric phase is consistent with findings from isometric fatigue tasks for the erector spinae[216], as well as peripheral muscles[206,256]. No reports of MMG frequency changes during fatiguing concentric or eccentric contractions exist, with the small number of studies examining MMG signal during these conditions reporting only time-domain changes[202,212]. Although the time and frequency domains of MMG signal are interesting independently, the primary strength of MMG is that (potential) alterations in motor unit activation strategy not apparent in EMG will manifest differently in amplitude and frequency aspects of the MMG[204]. For example, it is hypothesized that both a reduction in motor unit recruitment (decreased activation) or an increase in motor unit firing rate (increased activation) can result in a decrement to MMG amplitude[202,204,206]. Differentiating between the two circumstances is not possible without consideration of MMG frequency domain changes, which may reflect aggregate motor unit firing rate[206]. Alternatively (or in addition),

changes in the magnitude of surface-EMG can also be used to rule out within- or between-muscle changes in muscle activation as influences on the MMG magnitude. In fact, a remarkable aspect of MMG changes reported in the current study is that 7 of the 8 significant alterations to MMG magnitude were accompanied by an inverse change in median frequency (i.e. the start and end of the trial x 2 muscles x 2 contraction types). This effect would not be evident in studies that considered only frequency[206] or time domains[202,212], and is relevant for interpretation of the MMG signal.

Influences on MMG

Muscle stiffness (the inverse of compliance) is the most frequently considered non-physiological influence on the MMG signal, and may explain the initial decrement in MMG amplitude shown here. Increasing stiffness is thought to result in a decrement to MMG amplitude[200,202] and tends to increase during sustained contractions[204,211]. However, the influence of muscle stiffness changes may be lower during eccentric conditions[201,212], and are not sufficient to explain the localization of the MMG decline to the beginning of the trial, nor the absence of change during the raising phase (concentric). In fact, the opposite effect would be hypothesized, with the effect of stiffness (and thus MMG decrement) increasing over the course of the trial[202,214]. The inverse changes in frequency (increases) are also not consistent with the hypothesized effect of stiffness on MMG frequency (no change or uniform change)[206] . Lastly, motion artifacts are easily minimized via digital filtering techniques (i.e. < 1 Hz) and are generally considered for movements at much higher velocities (300 deg/sec)[210]. Although the influence of non-physiological factors cannot be completely excluded, current evidence suggests they are unlikely to be primary determinants of the MMG changes observed here. No existing factors are sufficient to explain the temporal variation in magnitude, isolation to the eccentric phase of motion, nor the inverse changes in frequency and time domains. Alternatively, if stiffness (or other related factors) can account for the MMG changes, it necessitates a difference

in relative contributions of non-physiological (stiffness) and physiological factors (activation) over the course of a trial and between contraction types. Both are interesting possibilities for future study.

Activation changes

The absence of a change in EMG magnitude in the current study allows the possibilities of motor unit de-recruitment (as might occur during maximal fatigue) or a load-shift to other muscles (e.g. hip extensors) to be eliminated as explanations for the initial reduction in MMG magnitude. Instead, the reduction in MMG magnitude of the lowering phase may reflect a shift towards increased firing rate, which is consistent with the accompanying frequency changes (increase) in pars lumborum, but not for pars thoracis (no change). However, this possibility differs from known firing rate changes during submaximal fatigue which may decrease[257] – an effect that may explain frequency changes of the raising (concentric) phase only. This explanation (muscle wisdom) has been widely considered as an explanation for a reduction in MMG amplitude[202,210], however the reductions tend to be more gradual[210] and persist over the course of trial[202], rather than the rapid decline and subsequent increase observed for the lowering phase. Muscle activation has been shown to be more variable during lengthening contractions[190], which may explain why the initial MMG changes were observed for the lowering, and not raising phase. One seldom considered factor that may account for a reduction in MMG amplitude is the synchronization of motor unit firing (i.e less synchronized)[206,258], which would tend to attenuate MMG amplitude via 'temporal' fusion of individual oscillations (as firing becomes less synchronized). In fact, if the MMG frequency is more reflective of aggregate motor unit firing rate (and not that of any single motor unit)[206], it is possible a shift to a more asynchronous firing rate (and therefore 'smoother' force output) could manifest as an increase in the number of oscillations per second (i.e. increase MMG frequency as seen here), independent of a change in firing rate(s). While it is not unsurprising to find that muscle activation differs between

contraction-types[184,190] and between fatigued and non-fatigued[259,260] conditions, the MMG signal provided a method of detecting activation changes not apparent in surface EMG. Although it is unlikely any single factor (i.e. stiffness versus synchronization) will be sufficient to account for the MMG changes, based upon a consideration of known mechanisms it seems probable that an eccentric-specific alteration in motor unit activation, in terms of a shift to a more asynchronous firing rate, of ES is at least partially explanatory for the reduction in MMG magnitude observed here.

Implications

Alterations in back muscle activation are implicated in current theories of back injury (i.e. motor control errors), where aberrations in activation may contribute to loss of stability of a spinal segment, resulting in injury[17]. Identifying circumstances where motor control errors are more likely to occur is important, particularly for back injuries that cannot be explained by load exposure alone[19]. The substantial change in ES activation observed in the current study occurred within a small number of repetitions, suggesting the state of the neural input to the low back (i.e. the controller[17] or neural control subsystem[261]) is changing rapidly, which would complicate the ability to incorporate feedback and/or predict the state of the spine system[17,261] (e.g. potentially decreasing robustness, or tolerance to perturbations). In contrast to the results of *The influence of body mass on lifting strategy* and other studies[7], where deleterious changes were linked to repetitive and cumulative loading effects, these results indicate specific attention is also required for the initial repetitions of a lifting task, and particularly the phase controlled via eccentric contractions (lowering). Factors that contribute to injury during a short number of repetitions may be as important as those attributable to fatigue[11,195], as most daily lifting tasks involve only a small number of repetitions and are rarely performed to fatigue, yet still result in injury.

These effects may also be due to the absence of a ‘warm-up’ or familiarization period (performing an unfamiliar/novel task), which based upon the MMG changes in the current study,

might increase the likelihood (and magnitude) of repetition-to-repetition variation in muscle activation. These changes would not be apparent in many experimental lifting tasks, which typically allow for familiarization to the experimental task[11,23,249], however potential familiarization-related changes may be as relevant to injury as the subsequent fatigue- or load-related effects. Additionally, changes to eccentric control are particularly important to understand, as lowering tasks account for a substantial proportion of back injuries[253,262], and aberrations in muscle activation may explain injuries that occur during the lowering phase of innocuous task (i.e. bending to pick up the morning newspaper). Although there is still a need to establish the implications of the MMG-related changes shown here, two circumstances requiring further study are the initial repetitions/familiarization period and the lowering/eccentric phase of lifting motions, areas that have received less attention in lifting and back injury literature.

EMG

Consistent with the load imparted by the weight of the upper body (i.e. 45%MVC)[58], the erector spinae were recruited at sub-maximal levels to begin the task (EMG, magnitude). Fatigue was apparent by substantial compression in EMG frequency domain and concurrent increases in the time domain (magnitude) of the concentric/raising phase, changes that are consistent with previous reports of ES activity during isometric conditions[58,59]. These findings differ from existing studies of ES activation during dynamic fatiguing conditions, where relatively constant[195] or even decreasing[193] ES activation has been reported, along with increased hip extensor activation[191]. This may be attributable to differences in analysis techniques, with previous investigations[193,195] reporting only pre- vs post-set amplitude changes (and not frequency changes), an approach that may fail to reveal a change if the EMG magnitude increased initially and was followed by a decline to the end of the trial, as has been shown during isometric tasks[56]. As well, most have focused exclusively on pars lumborum[56,193,195], rather than thoracis, which were shown to differ in this study – in fact, the pars thoracis EMG changes were similar to those

reported for hip extensors[193], while lumborum changes (during the concentric phase) were of lower magnitude.

The EMG signal, particularly the time-domain changes, was also essential for interpretation of the MMG signal. For instance, the absence of change in EMG magnitude allowed the possibilities of between-muscle substitution and within-muscle derecruitment to be ruled out as explanations for the reduction in MMG magnitude, further supporting an alteration in motor unit firing/synchronization. Until the effect of motor unit firing rate on MMG frequency domain is better established, the use of concurrent MMG-EMG will remain a necessary approach for interpretation of MMG magntidue and frequency changes. Additionally, surface EMG can provide important information regarding the intensity of muscle contraction (activation level) and state of fatigue, which may be useful for study of the determinants of MMG changes.

Pelvis restraint (specificity)

One of the most appealing explanations for the difference in ES activation between studies of involving dynamic tasks is the influence of pelvis restraint. During typical Roman Chair (RC) tasks, the pelvis is free to rotate[193,195], which may result in trunk motion produced predominantly by rotation about the iliofemoral joint, rather than the lumbar spine – an alteration that would account for increased activation of hip extensors[193,263], and less use of the ES (particularly if relatively little motion occurred at the lumbar spine). In contrast, the task in this study was clearly effective at eliciting fatigue of both lumbar and thoracic ES muscles, evident by frequency compression and increased recruitment. It is unclear which RC modification in the current study – the additional strap across posterior pelvis or more inferior position on a plinth (i.e. iliac crest vs ASIS) – was sufficient to shift the emphasis to ES muscles (although a strap in isolation appears ineffective[263]). The ability to target the ES (rather than hip extensors) using a dynamic task has relevance to therapeutic exercise (e.g. to increase back extensor endurance), as the use of an unrestrained pelvis appears insufficient to elicit fatigue in ES muscles[193]. Of equal

importance, confirming that a task performed to volitional fatigue results in neuromuscular fatigue of specific muscles is (obviously) necessary to investigate fatigue-related changes in those muscles. Task failure and neuromuscular fatigue, particularly of individual muscles, are not necessarily synonymous[259]. For instance, even during the isometric BS task, patient discomfort (due to an unpadded plinth) rather than neuromuscular fatigue, has been cited as a possible explanation for differences in endurance time between studies[264]. Failing to restrain pelvis motion during the RC task could reduce lumbar spine motion to such an extent that the ES muscles are contracting isometrically to 'stabilize' an immobile torso rotating about the hips, rather than acting as agonists to the motion (via concentric/eccentric contractions). Understanding ES fatigue during concentric/eccentric (dynamic) conditions is important since this reflects the contraction type and loading encountered during activities of daily living. If inferences are to be made from activation patterns regarding an individual's injury risk[15], it is important the tasks (and corresponding muscle actions) replicate the demands of real life.

Unlike the BS exercise which is used to investigate ES fatigue of both healthy and back-injured individuals during static/isometric conditions[58] , no dynamic equivalent has been established to examine the effect of contraction type. A comparison of MMG responses and task outcomes (e.g. endurance time) between healthy and back-injured patients using the current experimental task is an important next step. In light of the increased specificity to daily lifting tasks (e.g. concentric/eccentric contractions), it is possible that pathology-related activation changes will manifest differently between static (i.e. Biering-Sorensen) and dynamic fatiguing tasks (current study). Additionally, with the exception of an investigation by Lee and Stokes[218] which established the reliability of MMG in low back pain patients, mechanomyography has not been used to investigate pathology-related alterations in motor control of back muscles. Investigating the extent the rapid changes in MMG magnitude are impacted by previous or existing back injury may be one method of elucidating whether these MMG-related changes are

deleterious. Comprehensive evaluation techniques (e.g. concurrent EMG-MMG[202,265]) and standardized tasks that more closely resemble muscle function during daily tasks (e.g. modified RC used here) are needed, and may identify additional predictors of task failure and back pathology beyond EMG frequency domain factors already established[15].

End of trial

The end of trial increase in MMG amplitude is consistent with previous submaximal fatigue tasks involving isometric contractions of peripheral muscles[201,256], as well as the ES[216]. The partitioned regression models of the current study relate particularly well to Madeleine[216], who also observed a non-linear change in MMG magnitude over the course of a fatiguing task. In contrast to the initial reduction in MMG magnitude the end-of-trial increase is more readily explained, and may reflect increased motor unit recruitment (most likely), twitch potentiation, or motor unit synchronization related to fatigue[216,266]. Additionally, fatigue-related tremor associated with sporadic recruitment of high threshold motor units[185,260] may also contribute, which is typically observed in joint torque fluctuations[267] rather than MMG. It is possible MMG amplitude carries information regarding the local manifestations of fatigue-related tremor, which likely sums across muscles to produce the fluctuation (tremor) observed in joint torque (however this has not been established). Consistent with this possibility, the median frequencies of the MMG were much higher (40 – 50Hz) than fatigue-related tremor (6 – 20 Hz)[267], which would be expected due to the low-pass filtering effect of limb mass and temporal summation of force fluctuations from individual muscles. The increase in MMG at the end of trial was much greater during the concentric phase, consistent with increased motor unit recruitment, but was still apparent during the eccentric phase where the recruitment of additional motor units may not be (as) necessary. This suggests multiple factors, such as tremor and recruitment, may contribute to the end of trial increase in MMG amplitude. An examination of other EMG parameters (e.g. incidence of high amplitude EMG bursts[185,260] or spike-triggered averaging techniques[208]) and the

corresponding MMG signal changes may help to support or refute this possibility. Subsequent studies are needed to determine the relationship of MMG changes (from multiple muscles) to torque fluctuations (tremor) during isometric and dynamic conditions.

Between muscle comparisons

Significant between-muscle effects were apparent for nearly all conditions, characterized by increased EMG magnitudes and frequency reductions in pars lumborum, which is similar to previously investigated (isometric) conditions[58]. The single largest explanation for the between muscle effect is the difference in load (torque) imparted by the torso weight on each muscle[201], however the influence of other factors cannot be excluded. For instance, pars thoracis contains a greater proportion of slow twitch fibres[197], which may also account for a portion of the difference in fatiguability (EMG frequency change) and perhaps even MMG signal between muscles[209]. In fact, the difference in mechanical load between muscles may not be as large as predicted based upon simple moment of weight estimations, as pars thoracis can make significant contributions to lumbar extension torque via its tendinous attachment to the sacrum/iliac crests[196]. Explanations based upon mechanical factors would also need to account for the difference in muscle moment arms between muscles (greater in pars thoracis[143]), which affects the muscle force required to produce a given torque (i.e. muscle force should be estimated, not simply torque). In contrast to the EMG changes, the magnitude and duration of the initial MMG amplitude changes was greatest for pars lumborum, indicating the eccentric-specific MMG reduction may be proportional to the intensity of the contraction. Similar to EMG, the end of trial MMG changes were greater in pars thoracis, perhaps explained by increased motor unit recruitment, which would be more apparent in muscles contracting at lower intensity. Differential change in MMG between agonist-synergist muscles has been reported elsewhere[212], however relatively little is known about the factors underlying muscle-specific changes in MMG, and additional research is required. Understanding the relative loading and activation changes of back extensor muscles can help

guide back injury rehabilitation, either by identifying specific muscle groups requiring intervention (e.g. endurance training for pars thoracis) or as explanations for symptoms/injury involving thoracic muscle groups (e.g. due to torque generation in lumbar spine).

Contributions to task failure

Identifying the muscle most responsible/predictive of task-failure has achieved much attention[58,59,193], with numerous muscles identified during isometric tasks, including pars thoracis[59], multifidus/semitendinosis[189], the superior aspect of lumbar ES[187], or the most fatigued of either lumborum or thoracis[58]. Based upon frequency domain changes in the current study, pars lumborum would be identified as the best predictor of fatigue, having both the strongest relationship to endurance time (r^2) and greatest decrement in frequency. Although frequency-based approaches of identifying the most fatigued muscle are pervasive[187,189], some have questioned the validity[195,268]. Alternatively, using time domain changes, these results indicate task failure during dynamic trunk motions occurred when activation of pars thoracis reached maximum during the concentric phase of the motion, whereas pars lumborum was at maximal activation earlier (~30% of task time). The natural solution to disparate results of frequency- and time- based approaches, is the use of regression models incorporating both approaches, however the number of participants (and sites measured) in the current study were insufficient for regression analysis. The obvious discrepancy in time- and frequency- domain changes of the current study support the importance of both aspects for identifying predictors of task-failure in dynamic tasks, which has implications for injury mechanisms, therapeutic exercise and the design of injury prevention interventions. It is possible the mechanisms of task failure differ for dynamic (i.e. inability to raise trunk) and static conditions (i.e. failure to maintain trunk position). Dynamic conditions also involve more complex requirements for muscle activation, such as the need to maintain dynamic stability (vs static) or attenuate perturbations while moving (vs stationary), which may increase the probability of injury. Future studies are required that incorporate multiple

muscles (e.g. hip extensors, trunk flexors) to identify mechanisms of task failure during dynamic trunk motion, and regression models should be multivariate, including both frequency and time domain changes.

Future study

This study provides clear evidence the activation of erector spinae muscles during fatigue is dependent upon contraction type, supporting the task-specific nature of fatigue[269]. The most obvious next steps are to replicate the findings, perhaps under isovelocity conditions (dynamometer controlled) where greater control can be obtained (range of motion, velocity, contraction type, force). For example, although the initial reduction in MMG was clearly demonstrated, little is known about the determinants of the change (e.g. intensity, velocity, etc). Unlike peripheral muscle groups[200], there is a paucity of information regarding erector spinae MMG changes under varying torque outputs – all studies have been restricted to load imparted by torso weight[266]. Examination of wider range of load (e.g. an external mass or via dynamometry), will help elucidate the effects of activation intensity and load, which could only be speculated in the current study based upon anatomical and rudimentary mechanical rationale. Similarly, it is not known to what extent the initial change in MMG was related to fatigue or merely the execution of a relatively novel task, which could be considered by examining more experienced lifters, a second bout, terminating the task prior to task failure, or eliciting fatigue of the ES muscles prior to the task. Future studies should also consider antagonist (rectus abdominus, obliquues) and synergist (hamstrings, glutes) muscle groups, which play an important role in stability and moment generation about the lumbar spine. Comparisons between muscle groups may also reveal whether the changes in MMG signal reported here are related to muscle fibre type, as the erector spinae group is composed of a larger proportion of type 1 fibres compared to large muscle groups of the lower extremity (i.e. gluteus maximus).

Limitations

The influence of activation changes amongst multiple muscles was not considered, and so no clear conclusions can be made regarding the generalizability of these findings to other muscle groups. For instance, it is unclear whether the lowering-phase MMG changes will be restricted to the prime movers (studied here) or if they will also manifest in synergistic muscles (also contracting eccentrically) or antagonist muscles (undergoing concentric contractions). It is well-established that the contributions of hip extensors increase during dynamic trunk extensions[191,263], and while steps were taken to minimize this effect, it unknown if this shifted the emphasis away from the hip extensor muscles or if that of the ES simply increased, which may be relevant for identifying predictors of task failure. Although body mass-dependent loading provides high ecological validity, it does not account for variations in strength or anthropometrics between individuals, which may hinder comparisons across studies that incorporate different sample populations[189]. Additionally, sex has been shown to have an effect on neuromuscular fatigue[185,269], and the findings of the current sample (near exclusively normal BMI males), may not be generalizable to female participants. Surface EMG and MMG signals may be limited by effects of subcutaneous fat[58], which may preclude the use of these techniques for examination of pathology-related activation changes in high BMI populations. Further, the experimental task was not suitable for high BMI individuals, due to intereference by the abdominal pannus to trunk flextion in a prone position. However, it is important to establish these changes (in MMG/EMG) in order to understand the role/implications of muscle activation differences during lifting – although these findings are limited to individuals with a normal BMI, they serve as a starting point to investigations of repetition-based variations in muscle activation during dynamic tasks. Lastly, surface recordings of muscle activity can be impacted by a variety of methodological issues, such as cross talk or non-stationary effects[57]. Although these effects can be partially controlled by standardized electrode position[59] and use of a consistent range of motion[270], future studies may wish consider more advanced methods of frequency domain analysis[57].

Overall Conclusions

The aim was to characterize the activation of back extensor muscles during a dynamic, repetitive fatiguing task. This was quantified using the electrical (EMG) and mechanical (MMG) signals associated with muscle activation (objective 1), and compared between the concentric and eccentric portions of the task (objective 2). The most remarkable finding was a large decrement in MMG magnitude that was not easily explained by known influences of the MMG signal or EMG-related changes. One explanation is of an eccentric-specific change in synchronization of motor unit firing rates, independent of alterations to recruitment or firing rate. Additionally, modifications to the roman-chair exercise were shown to be effective at eliciting fatigue of the back extensors, providing a means of evaluating the effect of fatigue during concentric and eccentric contractions. These findings demonstrate the utility of composite MMG-EMG analysis[265] for detecting change in muscle activation, however further study is required to examine the influence of covariates (repeated bouts, relative loading) and explore the MMG signal in patients with back pathology. The lowering-phase of initial repetitions during a novel or unfamiliar task may be an important area to consider for injury prevention.

SYNTHESIS DISCUSSION

While a primary aim of these studies (*Effect of obesity on BSIPs and lumbar spine load* & *Influence of body mass on lifting strategy during repetitive fixed-pace lifting*) was to quantify the mechanical effect of obesity during lifting, the results of both studies indicate that the contributions of obesity to low back pain are most likely multi-factorial. Multiple contributing factors is consistent with proposed etiology of back injury in the general population[17,21], and factors beyond mechanical influences have also been postulated for obesity-related back pain (e.g. inflammation, dyslipidemia)[68]. The current investigations provide specific examples, related to existing biomechanical and physiological risk factors, of factors that may underlie obesity-related back injury.

It was obvious that elevated BMI would increase the load imparted on the low back[23], however the actual extent and determinants of the load increase had not been elucidated. The study *Effect of obesity on BSIPs and lumbar spine load* clearly demonstrated that obesity-related loading imparted on the low back is not due simply to the direct effects of increased body mass, but also indirect effects related to how an individual's morphology impacts movement (e.g. hold a box, flex the trunk, accelerate body segments). Evidence to support the most pronounced effect – an increased distance between the external load and the trunk – had been provided in previous studies[40], but had not been tested for materials handling tasks. It was also unknown whether these direct and indirect effects of obesity would impact other determinants of injury risk, such as cardiovascular factors, perceived effort or mass lifted. While previous investigators also confirmed that high BMI individuals do not likely elect to alter the mass lifted to compensate for increased body mass[158], it was possible that high BMI individuals might alter another aspect of lifting to accommodate (such as technique). The study *Influence of body mass on lifting strategy* revealed that as a result of increased loading (and perhaps obesity-related deconditioning), high

BMI individuals worked at elevated cardiovascular effort when performing the same materials handling task as normal BMI counterparts, and that perceptions of exertion were insensitive to the elevated mechanical or cardiovascular factors (in fact, BMI was negatively associated to perceived effort). These factors could be postulated to further increase injury risk beyond increased loading, via an earlier onset of fatigue during long duration tasks (cardiovascular) or an insensitivity to consequences of elevated loading (perceived exertion). The mechanisms underlying obesity-related back injury are due to multiple factors, not just the obvious and direct effect of increased trunk mass or inertia, and future studies should consider non-biomechanical consequences of increased body mass during investigations of materials handling tasks.

These findings may also have relevance for current injury prevention guidelines, such as the 3400 N compressive load threshold that guides weight selection during materials handling tasks, or the energy expenditure recommendations for repetitive lifting tasks (i.e. 2.2 – 4.4 kcal/min)[21]. Based upon the current findings, it is likely that high BMI individuals will exceed these thresholds during a number of different materials handling tasks –which may explain a portion of the elevated injury risk related to obesity[68]. This may also indicate that existing guidelines are not appropriate for high BMI individuals (or alternatively, that high BMI individuals can tolerate increased back load with less ill-effect). Injury-prevention guidelines related to characteristics of the mass lifted (loading) or frequency of lifts (energy expenditure) were developed based upon data indicating these parameters would be acceptable for a large proportion of the workforce (80 – 95%)[21]. Accommodations for increased body mass were not directly addressed, rather were incorporated into the model based upon the anthropometrics of the working population at that time. This is problematic if the characteristics of the population changes over time. It is well known the North American population is heavier (thus imparting greater lumbar loading), possess larger waist circumference (loads must be held farther from the body) and is in worse cardiovascular condition (working at a higher cardiovascular effort) than

two decades previous[60]. This suggests that if a reduction in back injury can be achieved through a reduction in load exposure, existing guidelines may no longer be protective to a substantial portion of the population (i.e. > 60% Canadians overweight or obese). Additionally, the full benefit of interventions that focus on altering aspects of the external environment may not be realized if deleterious changes to the individual (e.g. obesity) occur concomitant with any beneficial environmental changes, particularly if the environmental modifications eliminate a substantial portion of daily physical activity. It is well-established the response of biological tissue to load is U-shaped (i.e. an optimal and moderate amount of loading), indicating elimination of load exposure/activity (via a shift towards more sedentary work tasks) may leave an individual ill-prepared for circumstances requiring greater physical exertions. In addition to confirming that current injury prevention guidelines/thresholds are appropriate for individuals with high BMI, increased attention is required for interventions that address individual-specific factors (e.g. deconditioning, work capacity, obesity, etc)[145].

The BMI-related effect sizes for mechanical and physiological factors in the current studies are substantially greater than effects sizes for BMI-related back injury risk (e.g. back pain prevalence OR = 1.32)[68]. This is consistent with contributions from other BMI-dependent factors (e.g. inflammation, activity avoidance, deconditioning), but may also reflect a lack of specificity in either exposure (BMI) or outcomes (back injury) in existing epidemiological studies. For instance, the majority of obesity-related back pain odds ratios are based upon BMI cut-points[68] which will not reflect individual-specific differences in morphology (e.g. two people with equivalent BMI but different mass distriution – android and gynoid somatotypes) . The direct (CM_{loc}) and indirect loading effects (load distance) identified in the current study (*The effect of obesity on BSIPs and lumbar spine load)* would be greatest for individuals with central adiposity, consistent with more recent findings of stronger associations between waist circumference and back pain (vs body mass)[27]. Similarly, BMI does not account for differences in lean body mass

(or strength). Differences in strength (or lean body mass) have been postulated as an explanation for the increased BMI-dependent back injury risk in females compared to males[68], which might also be explained by sex-dependent morphological differences. It is also possible that obesity-related back injury is pathology- or mechanism-specific, factors that are challenging to control in epidemiological studies. For instance, the BMI-dependent increase in static load during standing (*Effect of obesity of BSIPs)* reflects the cumulative loading effects that high-BMI individuals would be exposed to, and would be expected to contribute to increased degenerative changes in the low back[230]. On the other hand, the BMI-dependent risk for acute back injury may not be as high, particularly if high BMI individuals avoid certain (high risk) activities and/or perhaps are afforded some protection from unexpected perturbations due to an increased body mass (e.g. tolerating greater force prior to a loss of stability). These BMI-specific factors need to be considered in epidemiological studies, since the effect of elevated BMI may not emerge in the high prevalence of self-reported, non-specific back pain without adequate control of these important covariates.

The BMI-related findings of the current studies were apparent in spite of examining a relatively narrow range, and somewhat constrained, lifting tasks (e.g. by frequency). The results of *The effects of lifting frequency, cue type and BMI* indicate these constraints may change how an individual executes a lift, and perhaps even attenuate between-group differences. This has implications for the results of the first two studies (*Effect of obesity on BSIPs* & *Influence of body mass on lifting strategy*), suggesting there are aspects of lifting behavior related to BMI that were not detected (e.g. pacing) and/or that the actual BMI-dependent kinematic effects might be larger than detected (as was apparent in the self-paced condition of the subsequent study). Although the findings of these studies identify potential mechanisms for BMI-dependent injury risk (e.g. load, cardiovascular, perceived effort), it is still unclear if these aspects can be generalized to

conditions outside the laboratory, where most lifting tasks are self-paced, free-form, and of a fixed workload (rather than a fixed weight).

Unconstrained tasks have received some attention in materials handling literature [44,180], with clear indications that lifting pace/frequency is an important parameter of preferred lifting strategy. If high BMI individuals choose to alter the pace of lifting tasks due to mechanical or physiological factors related to an elevated BMI, studies that impose a fixed-lifting pace would not detect these changes. Additionally, if the fixed-pace imposed was below a threshold that might exacerbate BMI-related effects (i.e. provided substantial rest), the magnitude of any potential BMI-dependent compensatory changes would be minimal. Data on functional consequences of obesity (e.g. increased effort at end-range of motion, decreased muscular endurance, increased lifting distance)[241] also suggests the impact of obesity on movement may not manifest during relatively simple sagittal plane lifting tasks, and that more challenging circumstances should be considered. This might also explain a portion of the relatively low odd ratios for obesity and back pain – obesity effects may only be specific to certain situations (e.g. non-occupational lifting tasks). Identification of the situations that predispose an individual to an elevated risk of back injury are important to elucidate (particularly if they vary across individuals), as these would provide the best potential 'return' on injury prevention efforts.

One such area may be the lowering phase of materials handling tasks, which has received less attention than the lifting or raising phase. The importance of the lowering phase was demonstrated in *Activation of erector spinae during repetitive trunk motion*, where mechanical responses to muscle activation (MMG) changed dramatically during the initial repetitions, but only for the eccentric (lowering) portion of the motion. Although studies identifying the determinants of the changes in activation have yet to be undertaken, these findings may have implications for back injury, particularly since it appeared to be independent of fatigue and attributable to execution of a novel/unfamiliar task. Familiarization is a commonly-used

experimental technique to minimize 'unwanted' within-participant variability, however this variability could be an essential consideration for back injury. For instance, variability in repetition-to-repetition loading (related to fatigue) has been acknowledged as a means in which the load tolerance of a specific tissue might be exceed on a given repetition[11]. In the current study variation in repetition-to-repetition activation was suggested to impose additional challenge to neural control of the lumbar spine. If the novelty of a task is implicated, these results support training individuals to be proficient in a larger number of movement patterns – not just as a means to spare tissue from cumulative loading effects, but also due to motor control-related issues related to performance of unfamiliar tasks. Risk factors related to initial repetitions may also impact task rotation (i.e. differential familiarization/adaptation across tasks, dependent upon the number of repetitions performed). Although surface-based muscle activation techniques are problematic for samples of individuals with high BMI, it is likely that similar (MMG) findings would be apparent in high BMI participants. There is a complete absence of studies on the effect of BMI during lowering tasks, where perhaps the kinematic consequences of these activation changes may manifest. A further understanding of risk factors related to neural control is required, and may be particularly important for back injury sustained during seemingly innocuous tasks.

FUTURE STUDY

Body segment parameters

Although obesity is becoming increasingly prevalent[60], the study of obesity biomechanics is relatively new[109] and has been limited by the absence of a reliable method of obtaining obesity-specific BSIPs. Considering the strong relationship between increased body mass and musculoskeletal disorders[271] there is a need to understand the mechanical consequences of increased body mass. The studies *Effect of obesity on BSIPs* and *Influence of body mass on lifting*

strategy identified several areas requiring further study. For instance, BMI-dependent changes in BSIPs need to be considered for more diverse morphologies than those considered here. One of the most obvious examples is to consider the effect of obesity on BSIPs for female individuals, as body mass distribution clearly differs between sexes. Additionally, the effect of sex may be partially related to somatotype[8] (i.e. android vs gynoid shape), which was not compared in this study. Studies examining the effects sex and morphology on obesity-related BSIPs will help identify which aspects of BMI-dependent BSIPs can be generalized (and accounted for by predictive models – e.g. proximal shift in longitudinal centre of mass[115]) and which cannot (requiring individual-specific approaches – e.g. effects of central vs peripheral adiposity). These studies may help strike a balance between the need for individual-specific approaches (advocated in study 1 and study 2) versus the actual practicality of such an approach. Similar to other aspects of anthropometrics already encorporated into ergonomic design (e.g. height), a more appropriate approach will be to identify factors accounting for a large proportion of the variance in load and movement strategy across individuals (e.g. waist circumference) as primary targets for delivery of 'individual-specific' ergonomic interventions. While each individual may differ with respect to the combination of morphological, fitness and health-related constraints to material handling capability (and injury risk), it is not unrealistic to better understand how each category of factors influences lifting and design ergonomic training with these in mind (rather than the near ubiquitous approach of assuming that each individual is identical with respect to lifting ability).

Back loading

The next step after understanding how obesity impacts BSIPs is to examine the interaction with the environment, as obesity-related loading is a function of how an individual moves and not simply body segment mass or inertia. Load exposure for high BMI individuals during daily tasks will be a function of: a) how an individual executes a specific motion

[8] **Future study:** to determine the effect of sex and central versus peripheral mass distribution on body segment parameters

(kinematics and kinetics), and b) the frequency the motions are performed (activity patterns). It is possible normal and high BMI individuals may be exposed to similar cumulative loading over the course of a day, but with different contributions from body mass and activity-related factors. Investigations using more detailed kinematic and kinetic analyses are required, as this might reveal temporal differences in kinetics not apparent in the peak measures used here[9]. For instance, individuals may initiate a lift with similar body segment orientations (constrained by the environment), but execute the actual motion with different inter-segmental coordination.

Similar to approaches used for population-based physical activity[272], the patterns of occupational and daily lifting tasks should be evaluated in normal and high BMI individuals. This could include factors such as the total number of lifts (analogous to step count), but more importantly, variables like the number of lifts in succession (bout length), rest period, weight lifted (intensity), and time-of-day analyses[10]. Recent advances in accelerometer-based physical activity monitoring[273], as well as the stereotypical acceleration pattern of many lifting motions (see Figure 8), indicate these aspects could be objectively measured in field studies. Regardless of whether the total lifts differ, it is important to determine whether strategies such as altered bout length, rest breaks or time of day are dependent on individual-specific factors (e.g. body mass), which might have a similar (or even greater) effect on back load as BMI-dependent changes in lifting strategy[23]. Currently, epidemiological studies of occupational back injury operate under the assumption that individuals performing the same job are exposed to the same load, but this has not been thoroughly tested and may not hold for jobs that are self-paced or performed as part of a large group. Unlike ambulatory-based physical activity, objective evaluations of the pattern of daily and occupational manual lifting have not been considered.

[9] **Future study:** to examine differences in peak and temporal loading characteristics and kinematics between normal and high BMI individuals

[10] **Future study:** to objectively evaluate the pattern of daily and occupational lifting tasks and the influence of BMI

No attempt was made in the current series of studies to evaluate or control for the rate of increase in body mass or the number of years the parctipants had been obese. This is an important consideration as the chronicity of obesity may influence the movement strategies and any potential underlying (mal)adaptations to the increased body mass. For instance, it is possible that with a more gradual accumulation of body mass and some minimal amount of physical activity, an individual may develop beneficial adaptations to obesity, such as increased bone density, muscle mass or ligament strength in the lumbar region (i.e. high BMI and physically active). Alternatively, an increase in body mass that is more rapid or with an sub-optimal amount of activity (i.e. high BMI and sedentary) may leave an individual with underlying tissues that are not adatpatedadapted to the increased body mass-related load. These effects are very likely evident during pregnancy, which is associated with a relatively rapid weight gain and a high incidence of low back complaints[86]. Although other factors (e.g. relaxin) certainly contribute[86], it is obvious that throughout the pregnancy term women make postural changes in order to compensate for the increased loading, and that the opportunity for tissues to adapt is relatively short (particularly if the proportion of sedentary time increase). Elucidating the effect of obesity on underlying tissue tolerances is an important aspect of obesity-related back pathology, particularly in light of the substantially greater effect sizes for BMI-dependent mechanical loading compared to BMI-dependent back injury risk. It is well-established that individuals can adapt to tolerate substantial loading about the low back with minimal injury (e.g. Olympic weight lifters), however the extent that similar adapations might occur in high BMI individuals is not known. It is likely the movement strategies displayed by the high BMI individuals may also have developed concurrent with the increase in body mass (i.e. gradual changes of a long time period), rather than as acute changes to increased body mass.

Unconstrained lifting

Controlled examinations of kinetics and kinematics are necessary to identify factors that may be related to an elevated BMI (e.g. cardiovascular effort, load distance) and for comparisons to existing literature, but have poor ecological validity and may not be generalizable to conditions outside the laboratory. It is increasingly clear (*Effect of lifting frequency, cue type and BMI)*[44,50] that evaluations of unconstrained lifting tasks are required, in both normal and high BMI individuals. For instance, ergonomic interventions and job tasks are designed under the assumption that workplace behavior reflects that observed in lab, but more evidence is gathering this may not always be the case[44,50] (which may contribute to the poor outcome for some prophylactic interventions[234]). One area of priority is to examine lifting technique and related outcomes (RPE, HR) during tasks that are constrained only by workload (e.g. pallet of bricks[44], a pile of dirt, driveway full of snow), where differences in lifting pace, work-rest ratio and the load lifted may be elucidated[11]. Lastly, studies should also consider repetition-to-repetition variation in muscle activity (and related kinematics) during the initial repetitions of a lifting task, particularly for the eccentric phase of motion and/or lowering tasks[12], which may be important considerations for injuries that are not easily explained by loading or fatigue-related factors. Although mechanical factors are important to understand, there is increasing evidence that alterations in muscle activation[17] (i.e. motor control errors) are implicated in back injury mechanisms. Investigations of unconstrained lifting tasks will help identify situations where high BMI individuals may be at increased risk (versus conditions that are uniformly high risk) and aid in design of individual-specific interventions that more directly address the natural tendencies of an individual when executing lifting tasks.

[11] **Future study**: to determine lifting strategy (kinematics, pace, load) and exertion (HR, RPE) of normal and high BMI individuals during self-paced, manual lifting of a fixed workload.

[12] **Future study**: evaluate between-repetition variation in phase-specific kinematics and muscle activation during novel lifting tasks.

OVERALL CONCLUSION

Back injury and obesity are widespread health concerns associated with substantial individual and societal costs[3]. Establishing factors that underlie obesity-related back pathology, as well as identifying circumstances where injury risk is elevated, will enable practitioners to better treat and prevent back pathology. This thesis undertakes some initial steps toward our understanding of the effects of obesity during lifting, and provides initial estimations for obesity-dependent loading and an in-depth evaluation of the subsequent effects during a repetitive lifting task, where BMI-dependent factors other than mechanical load were identified. Evidence for an effect of external constraints on lifting strategy was provided, which, combined with the paucity of studies on unconstrained lifting tasks, aided in identification of areas for future study related to the generalizability of experimental findings. One specific area identified, based upon novel changes in back extensor activation, was the lowering-phase of the initial repetitions of a repetitive task. It is unlikely that the health complications associated with back injury and/or unhealthy body composition will be eliminated in the near future, making it imperative to elucidate the relationship between obesity and musculoskeletal injury and establish effective methods to mitigate obesity-related pathology.

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APPENDICES – STUDY 1

Appendix: StrataFoto 3D CX Settings

The following settings were used for generation of 3D models using Strata Foto CX:

Texture quality: 100

Silhouette decimation: 1.0

Texture map size: 1600

Maximum number of images to use for mesh refinement: 60

Number of polygons: 20,000

Use sub-sampled images: Yes

Model fit: Exact

Constrain mesh to lie within current geometry: Yes

Appendix: Limb Asymmetry

The mean (SD) variance in volume distribution between right and left upper extremity segments was 234.3 (155.0) cc across all participants, equivalent to 6.2 (4.4) % of average upper limb volume, $p < .001$. No differences were detected between sides, with the upper extremity of greatest volume evenly divided between the right (n = 4) and left (n = 5) sides. In the lower extremity the difference was 376.3 (203.4) cc, or 2.7 (1.6) percent of average limb volume, $p < .001$, with all but two models returning greater volume for the right side. A paired t-test for the difference in asymmetry between the upper and lower extremity was near significant at $t(8) = 1.97$, $p = 0.08$ (two-sided), revealing a trend for less (relative) variance bilaterally in the lower, compared to the upper extremities.

Interestingly, the largest asymmetry in the lower extremity was found for participant 3 at 778.9 cc ($6.1\%_{limb}$), who happened to be < 3 months post-repair for an Achille's tendon rupture. This asymmetry was near triple the bilateral variance in the other 3 normal BMI models, 270.6 cc ($2.1\%_{limb}$). Partitioning the limb into segments for subject 3 revealed asymmetries for both the shank (189.6 cc or $7.0\%_{limb}$) and thigh + foot segments (589.3 cc, or $5.9\%_{limb}$), however the difference was proportionally greater in the shank ($+1.1\%_{limb}$). Excluding participant 3, the lower extremity asymmetry was similar between normal (M = 2.1, SD = 0.1 $\%_{limb}$) and high BMI groups (M = 2.3, SD = 1.3 $\%_{limb}$), while the test for the difference in asymmetries between the lower and upper extremities became, $t(7) = 2.48$, $p < .05$ (two-sided), indicating greater bilateral volume variability in the upper compared to lower extremities of the models. The effect of the increased variance on the between-group differences in BSIPs is considered negligible, as the magnitude of this difference (~700 cc) is far below the magnitude of the difference between groups (i.e. it represents < 1kg of lean body mass).

Appendix: Upper and Lower Limb Anthropometrics

note: participant numbers in the following appendix are inverse of those presented above (i.e. high-BMI participants are 5-9, while normal BMI are 1-4)

Table 39. Upper arm anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	volume (cc)	volume (%body)	mass (g)	mass (%body)	density (g/cc)	length* (cm)	depth (cm)
1	2136.0	2.8	2359.2	2.9	1.104	30.2	10.5
2	1819.7	2.6	1970.6	2.7	1.083	28.3	10.4
3	1974.6	2.8	2162.7	2.9	1.095	28.6	10.5
4	1685.5	2.6	1818.3	2.7	1.078	26.4	10.4
5	2839.6	3.2	3014.1	3.4	1.061	27.6	12.2
6	3637.5	3.5	3785.2	3.6	1.041	27.2	14.3
7	3642.0	2.9	3807.7	3.0	1.045	28.2	15.3
8	3073.5	2.9	3234.3	3.0	1.052	27.6	14.0
9	2736.6	2.4	2863.3	2.5	1.046	20.8	14.3
normal, M	*1903.9*	*2.7*	*2077.7*	*2.8*	*1.090*	*28.4*	*10.4*
SD	*194.6*	*0.1*	*234.7*	*0.1*	*0.012*	*1.5*	*0.1*
high, M	*3185.8*	*3.0*	*3340.9*	*3.1*	*1.049*	*26.3*	*14.0*
SD	*431.9*	*0.4*	*436.3*	*0.4*	*0.008*	*3.1*	*1.1*
high/norm, M	*1.77*	*1.12*	*1.71*	*1.13*	*0.97*	*0.95*	*1.35*
SD	*0.18*	*0.16*	*0.16*	*0.16*	*0.01*	*0.10*	*0.10*

*Upper arm length is derived from 3D model and represents the distance from the shoulder joint centre to the elbow joint centre; depth is the maximum anterior-posterior dimension distal to the axilla.

Table 40. Forearm anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	volume (cc)	volume (%body)	mass (g)	mass (%body)	density (g/cc)	length* (cm)	depth (cm)
1	882.5	1.2	1020.0	1.2	1.156	24.5	7.4
2	767.1	1.1	869.4	1.2	1.133	23.8	8.5
3	875.7	1.3	1003.8	1.4	1.146	23.8	8.8
4	756.2	1.2	853.8	1.3	1.129	23.0	8.2
5	1151.4	1.3	1279.0	1.4	1.111	25.8	9.4
6	1380.6	1.3	1503.5	1.4	1.089	26.6	9.9
7	1714.9	1.4	1876.4	1.5	1.094	26.0	11.5
8	1253.3	1.2	1380.2	1.3	1.101	23.5	11.3
9	1109.0	1.0	1214.3	1.1	1.095	22.9	10.0
normal, M	*820.4*	*1.2*	*936.7*	*1.3*	*1.141*	*23.8*	*8.2*
SD	*68.0*	*0.1*	*87.3*	*0.1*	*0.012*	*0.6*	*0.6*
high, M	*1321.8*	*1.2*	*1450.7*	*1.3*	*1.098*	*24.9*	*10.4*
SD	*243.4*	*0.1*	*261.9*	*0.2*	*0.008*	*1.7*	*0.9*
high/norm, M	*1.68*	*1.06*	*1.62*	*1.06*	*0.97*	*1.06*	*1.24*
SD	*0.21*	*0.15*	*0.20*	*0.15*	*0.01*	*0.05*	*0.11*

*Forearm length is derived from 3D model and represents the distance from the elbow joint centre to the radio-carpal joint centre; depth is the maximum anterior-posterior dimension.

Table 41. Hand anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs*).

Participant	volume (cc)	volume (%body)	mass (g)	mass (%body)	density (g/cc)	length* (cm)	depth (cm)
1	441.7	0.6	524.1	0.6	1.187	13.3	7.8
2	379.4	0.5	441.5	0.6	1.164	13.0	8.6
3	425.5	0.6	500.7	0.7	1.177	13.9	8.2
4*	351.4	0.5	407.3	0.6	1.159	11.9	8.4
5**	399.5	0.5	455.6	0.5	1.140	18.9	5.4
6**	485.5	0.5	542.8	0.5	1.118	20.5	6.4
7	688.3	0.5	773.2	0.6	1.123	13.7	9.9
8	473.8	0.4	535.7	0.5	1.131	11.5	8.0
9**	401.3	0.4	451.1	0.4	1.124	12.1	6.3
normal, M	*399.5*	*0.6*	*468.4*	*0.6*	*1.172*	*13.0*	*8.3*
SD	*41.5*	*0.1*	*53.4*	*0.1*	*0.013*	*0.8*	*0.4*
high, M	*489.7*	*0.5*	*551.7*	*0.5*	*1.127*	*15.3*	*7.2*
SD	*117.9*	*0.1*	*131.1*	*0.1*	*0.009*	*4.0*	*1.8*
high/norm, M	*1.29*	*0.81*	*1.25*	*0.81*	*0.97*	*1.06*	*0.85*
SD	*0.21*	*0.09*	*0.20*	*0.09*	*0.01*	*0.05*	*0.23*

*hand length is derived from the 3D model and represents the distance from the radio-carpal joint to the distal end of the hand. centre; depth is the maximum anterior-posterior dimension.

**indicates hand held with fingers extended; all others with hand formed in a fist-shape

Table 42. Thigh anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	volume (cc)	volume (%body)	mass (g)	mass (%body)	density (g/cc)	length* (cm)	depth (cm)
1	8276.4	10.7	8928.5	10.9	1.079	40.4	18.3
2	9049.1	13.0	9571.9	13.2	1.058	38.8	19.1
3	8900.5	12.8	9521.8	13.0	1.070	40.4	16.1
4	8380.7	13.0	8830.7	13.2	1.054	37.6	18.3
5	8397.8	9.5	8706.8	9.7	1.037	37.0	18.9
6	10,941.9	10.5	11,121.4	10.7	1.016	37.0	22.0
7	13,926.4	10.9	14,221.3	11.2	1.021	42.3	21.1
8	11,329.0	10.6	11,644.6	10.7	1.028	37.7	20.9
9	11,636.6	10.4	11,892.2	10.6	1.022	37.2	20.9
normal, M	*8651.7*	*12.4*	*9213.2*	*12.6*	*1.065*	*39.3*	*18.0*
SD	*380.4*	*1.1*	*387.9*	*1.1*	*0.011*	*1.4*	*1.3*
high, M	*11,246.3*	*10.4*	*11,517.3*	*10.6*	*1.025*	*38.2*	*20.7*
SD	*1971.6*	*0.5*	*1970.1*	*0.6*	*0.007*	*2.3*	*1.2*
high/norm, M	*1.29*	*0.80*	*1.25*	*0.81*	*0.97*	*0.99*	*1.15*
SD	*0.24*	*0.04*	*0.22*	*0.05*	*0.01*	*0.04*	*0.11*

*Thigh length is derived from the 3D model and represents the distance from the hip joint centre to the knee joint centre; depth is the maximum anterior-posterior dimension distal to the axilla.

Table 43. Shank anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	volume (cc)	volume (%body)	mass (g)	mass (%body)	density (g/cc)	length* (cm)	depth (cm)
1	3789.0	4.9	4243.3	5.2	1.120	45.7	12.4
2	2904.1	4.2	3188.9	4.4	1.098	36.5	12.7
3	2718.2	3.9	3018.7	4.1	1.111	38.1	11.6
4	2689.8	4.2	2942.2	4.4	1.093	35.4	12.4
5	2867.5	3.3	3086.3	3.4	1.076	37.5	11.9
6	3700.8	3.6	3904.8	3.8	1.055	40.7	13.5
7	4355.6	3.4	4617.3	3.6	1.060	40.1	14.5
8	4184.9	3.9	4465.4	4.1	1.067	37.6	14.0
9	3898.2	3.5	4135.6	3.8	1.061	35.3	13.5
normal, M	*3025.3*	*4.3*	*3348.2*	*4.5*	*1.106*	*38.9*	*12.3*
SD	*517.9*	*0.4*	*605.5*	*0.5*	*0.012*	*4.7*	*0.5*
high, M	*3801.4*	*3.5*	*4041.9*	*3.7*	*1.064*	*38.2*	*13.5*
SD	*580.1*	*0.2*	*602.3*	*0.3*	*0.008*	*2.2*	*1.0*
high/norm, M	*1.37*	*0.86*	*1.33*	*0.86*	*0.97*	*1.05*	*1.09*
SD	*0.25*	*0.06*	*0.24*	*0.06*	*0.01*	*0.04*	*0.11*

*Shank length is derived from the 3D model and represents the distance from the knee joint centre to the ankle joint centre; depth is the maximum anterior-posterior dimension distal to the axilla.

Table 44. Foot anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs*

Participant	volume (cc)	volume (%body)	mass (g)	mass (%body)	density (g/cc)	length* (cm)	height (cm)
1	1184.7	1.5	1332.8	1.6	1.125	26.6	10.0
2	998.6	1.4	1101.6	1.5	1.103	24.8	9.7
3	1050.7	1.5	1172.2	1.6	1.116	25.8	9.4
4	924.9	1.4	1016.3	1.5	1.099	24.2	9.2
5	909.6	1.0	983.5	1.1	1.081	23.1	9.6
6	1056.8	1.0	1120.1	1.1	1.060	27.0	9.6
7	1167.4	0.9	1243.2	1.0	1.065	27.9	8.0
8	952.3	0.9	1020.8	0.9	1.072	24.3	8.8
9	1062.1	1.0	1131.9	1.0	1.066	24.3	8.0
normal, M	*1039.7*	*1.5*	*1155.7*	*1.6*	*1.111*	*25.3*	*9.6*
SD	*109.6*	*0.1*	*134.2*	*0.1*	*0.012*	*1.0*	*0.4*
high, M	*1029.6*	*1.0*	*1099.9*	*1.0*	*1.069*	*25.3*	*8.8*
SD	*1001.4*	*0.1*	*102.2*	*0.1*	*0.008*	*2.0*	*0.8*
high/norm, M	*1.05*	*0.66*	*1.02*	*0.67*	*0.97*	*1.02*	*0.93*
SD	*0.09*	*0.05*	*0.08*	*0.05*	*0.01*	*0.07*	*0.06*

*Foot length is derived from the 3D model and represents the distance from the posterior calcaneus to the most distal phalange; height is the maximum superior-inferior dimension.

Table 45. Head anthropometrics. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	volume (cc)	volume ($\%_{body}$)	mass (g)	mass ($\%_{body}$)	density (g/cc)	length* (cm)	height (cm)
1	4116.5	5.3	4694.6	5.7	1.140	25.1	20.0
2	5126.8	7.4	5732.8	7.9	1.118	27.6	20.8
3	4355.6	6.2	4925.9	6.7	1.131	25.6	18.3
4	4748.0	7.4	5288.9	7.9	1.114	26.4	20.3
5	5132.8	5.8	5625.6	6.3	1.096	26.9	19.4
6	5078.2	4.9	5456.4	5.3	1.075	25.6	21.2
7	7750.9	6.1	8367.3	6.6	1.079	30.6	22.6
8	5067.7	4.7	5506.5	5.1	1.087	24.6	17.8
9	5406.6	4.8	5841.1	5.1	1.080	26.5	18.3
normal, M	*4586.7*	*6.6*	*5160.5*	*7.1*	*1.126*	*26.2*	*19.8*
SD	*444.3*	*1.0*	*453.2*	*1.1*	*0.012*	*1.1*	*1.1*
high, M	*5687.2*	*5.3*	*6159.4*	*5.7*	*1.083*	*26.8*	*19.9*
SD	*1161.9*	*0.6*	*1243.2*	*0.7*	*0.008*	*2.3*	*2.0*
high/norm, M	*1.20*	*0.75*	*1.16*	*0.75*	*0.97*	*1.01*	*0.99*
SD	*0.33*	*0.14*	*0.31*	*0.15*	*0.01*	*0.11*	*0.15*

*Head length is derived from the 3D model and represents the distance from the superior head to the C7 joint centre; depth is the maximum anterior-posterior dimension at the forehead.

Appendix: Trunk and Extremity BSIPs

note: participant numbers in the following appendix are inverse of those presented above (i.e. high-BMI participants are 5-9, while normal BMI are 1-4).

Table 46. Whole trunk inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2$)x10^3	Iyy ($kgcm^2$)x10^3	Izz ($kgcm^2$)x10^3	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	1.1	1.8	18.6	15.17	16.96	4.32	25.7	27.2	13.7
2	1.2	0.5	19.6	12.04	12.71	2.96	27.1	27.8	13.4
3	0.3	0.5	27.7	11.52	12.60	3.36	27.0	23.2	14.6
4*	0.8	0.7	19.7	10.40	10.99	2.63	27.5	28.3	13.8
5	0.2	3.1	20.1	19.99	21.57	6.61	27.2	28.3	15.7
6	0.2	3.5	19.7	21.72	22.91	8.41	27.1	27.8	16.8
7	1.3	2.7	21.8	28.37	30.27	11.69	28.3	29.3	18.2
8	1.4	6.4	18.3	21.59	22.81	9.31	26.5	27.2	17.4
9	0.2	5.6	19.3	25.38	26.99	10.82	27.1	28.0	17.7
normal, M	*0.8*	*0.9*	*21.4*	*12.29*	*13.31*	*3.32*	*26.8*	*27.9*	*13.9*
SD	*0.4*	*0.7*	*4.2*	*2.04*	*2.56*	*0.73*	*0.8*	*0.5*	*0.5*
high, M	*0.7*	*4.2*	*19.8*	*23.41*	*24.91*	*9.37*	*27.2*	*28.1*	*17.2*
SD	*0.6*	*1.7*	*1.3*	*3.41*	*3.63*	*0.20*	*0.7*	*0.8*	*0.9*
high/norm, M. SD	*1.35*	*6.89*	*0.94*	*2.09*	*2.08*	*3.24*	*1.00*	*1.00*	*1.24*
	1.82	*1.33*	*0.10*	*0.36*	*0.34*	*0.72*	*0.03*	*0.03*	*0.04*

CM = centre of mass location relative to L5 joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior I = moment of inertia, $kgcm^2$ R = radius of gyration, % segment length

Table 47. Upper arm inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2$)x10^2	Iyy ($kgcm^2$)x10^2	Izz ($kgcm^2$)x10^2	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	0.1	0.1	-42.9	2.19	2.11	0.28	31.9	31.4	11.4
2	1.4	0.7	-41.5	1.63	1.55	0.24	32.2	31.3	12.4
3	1.1	1.5	-46.4	1.78	1.72	0.37	31.8	31.2	14.5
4	2.6	3.5	-40.2	1.41	1.34	0.21	33.4	32.5	12.9
5	4.2	3.1	-34.7	2.52	2.36	0.54	33.2	32.1	15.3
6	6.1	1.7	-39.3	3.04	2.79	0.93	32.9	31.5	18.2
7	2.7	1.8	-42.7	3.28	2.86	0.98	32.9	30.7	18.0
8	2.7	1.1	-41.9	2.56	2.16	0.76	32.2	29.6	17.5
9	0.8	2.5	-42.8	1.07	0.90	0.43	29.4	26.9	18.7
normal, M	*1.3*	*1.4*	*-42.7*	*1.76*	*1.68*	*0.27*	*32.3*	*31.6*	*12.8*
SD	*1.0*	*1.5*	*2.7*	*0.33*	*0.33*	*0.07*	*0.7*	*0.6*	*1.3*
high, M	*3.3*	*2.0*	*-40.3*	*2.49*	*2.21*	*0.73*	*32.1*	*30.2*	*17.5*
SD	*1.9*	*0.8*	*3.4*	*0.86*	*0.79*	*0.24*	*1.6*	*2.0*	*1.3*
high/norm, M, SD	*2.27*	*1.80*	*0.96*	*1.56*	*1.46*	*2.87*	*0.99*	*0.95*	*1.35*
	1.64	*1.66*	*0.09*	*0.47*	*0.45*	*0.81*	*0.07*	*0.08*	*0.11*

CM = centre of mass location relative to glenohumeral joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior I = moment of inertia, $kgcm^2$ R = radius of gyration, % segment length

Table 48. Forearm inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2$)x 10^2	Iyy ($kgcm^2$)x 10^2	Izz ($kgcm^2$)x10^2	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	1.2	2.4	-36.3	0.45	0.44	0.07	27.3	26.9	10.8
2	3.3	1.5	-37.0	0.39	0.39	0.06	28.1	28.2	10.6
3	5.3	4.5	-38.7	0.41	0.42	0.07	27.0	27.3	11.0
4	0.9	2.4	-37.3	0.38	0.38	0.05	29.1	29.0	10.7
5	1.4	0.3	-35.4	0.66	0.65	0.10	27.8	27.6	10.9
6	3.5	0.9	-38.3	0.79	0.77	0.14	27.2	26.9	11.6
7	0.0	2.0	-41.2	1.04	1.00	0.23	28.5	28.1	13.4
8	0.2	2.9	-40.3	0.64	0.60	0.15	29.0	28.0	13.9
9	0.3	1.9	-36.4	0.43	0.42	0.10	26.1	25.8	12.5
normal, M	*2.7*	*2.7*	*-37.3*	*0.41*	*0.41*	*0.06*	*27.9*	*27.9*	*10.8*
SD	*2.0*	*1.3*	*1.0*	*0.03*	*0.03*	*0.01*	*0.9*	*0.9*	*0.2*
high, M	*1.1*	*1.6*	*-38.3*	*0.71*	*0.69*	*0.14*	*27.7*	*27.3*	*12.5*
SD	*1.4*	*1.0*	*2.5*	*0.22*	*0.21*	*0.05*	*1.1*	*0.9*	*1.2*
high/norm, M SD	*0.42*	*0.65*	*1.02*	*1.81*	*1.73*	*2.49*	*0.98*	*0.96*	*1.16*
	0.38	*0.38*	*0.05*	*0.51*	*0.47*	*0.63*	*0.06*	*0.10*	*0.10*

CM = centre of mass location relative to elbow joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$ R = radius of gyration, as a percentage of segment length

Table 49. Hand inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*. Estimates may not reflect actual differences as some participants held the hands in a fist and others with fingers splayed.

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2$)	Iyy ($kgcm^2$)	Izz ($kgcm^2$)	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	8.2	2.1	-55.9	6.50	7.81	3.94	26.4	28.9	20.6
2	1.5	3.6	-52.9	5.90	5.43	2.58	28.2	27.0	18.6
3	0.7	6.5	-53.9	7.43	7.41	3.24	27.7	27.7	18.3
4	2.5	2.5	-48.0	5.13	4.61	2.39	29.8	28.3	20.3
5	4.4	0.8	-35.4	7.80	9.72	3.34	21.9	24.5	14.4
6	1.2	1.7	-40.8	12.87	14.03	3.64	23.8	24.9	12.7
7	2.5	2.1	-51.0	11.76	12.42	7.93	28.5	29.3	23.4
8	6.1	0.8	-44.7	6.42	6.56	4.04	30.1	30.4	23.9
9	3.3	5.2	-42.3	4.06	5.10	3.24	24.7	27.8	22.1
normal, M	*3.2*	*3.7*	*-52.7*	*6.24*	*6.32*	*3.04*	*28.0*	*28.0*	*19.5*
SD	*3.3*	*1.9*	*3.4*	*0.97*	*1.54*	*0.70*	*1.4*	*0.8*	*1.2*
high, M	*3.5*	*2.1*	*-42.8*	*8.58*	*9.57*	*4.44*	*25.8*	*27.4*	*19.3*
SD	*1.9*	*1.8*	*5.7*	*3.68*	*3.78*	*1.98*	*3.39*	*2.64*	*5.3*
high/norm, M SD	*2.16*	*0.68*	*0.84*	*1.43*	*1.71*	*1.64*	*0.90*	*0.99*	*1.00*
	1.10	*0.78*	*0.12*	*0.51*	*0.55*	*0.48*	*0.11*	*0.08*	*0.26*

CM = centre of mass location relative to radio-carpal joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$
R = radius of gyration, as a percentage of segment length

Table 50. Thigh inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2 \times 10^2$)	Iyy ($kgcm^2 \times 10^2$)	Izz ($kgcm^2 \times 10^2$)	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	2.4	2.9	-40.1	12.96	12.58	3.29	29.8	29.4	15.0
2	1.3	2.8	-41.5	15.68	14.96	3.84	33.0	32.2	16.3
3	1.3	1.9	-43.7	16.31	15.76	3.66	32.4	31.8	15.3
4	1.3	2.0	-44.9	13.58	12.94	3.34	33.0	32.2	16.4
5	6.3	0.9	-35.5	12.24	11.73	3.47	32.1	31.4	17.1
6	3.7	2.4	-38.3	16.16	15.01	6.26	32.6	31.4	20.3
7	1.2	0.9	-41.8	22.50	20.84	9.45	29.8	28.7	19.3
8	0.7	2.4	-37.8	15.35	14.15	6.98	30.5	29.3	20.6
9	1.0	0.2	-39.4	17.30	15.39	7.66	32.4	30.6	21.6
normal, M	*1.6*	*2.4*	*-42.6*	*14.63*	*14.06*	*3.53*	*32.0*	*31.4*	*15.8*
SD	*0.6*	*0.5*	*2.2*	*1.61*	*1.54*	*0.26*	*1.5*	*1.4*	*0.7*
high, M	*2.6*	*1.3*	*-38.6*	*16.71*	*15.43*	*6.76*	*31.5*	*30.3*	*19.8*
SD	*2.4*	*1.0*	*2.3*	*3.74*	*3.34*	*2.19*	*1.3*	*1.7*	*1.7*
high/norm, M SD	*1.99*	*0.58*	*0.89*	*1.12*	*1.08*	*1.90*	*0.96*	*0.94*	*1.22*
	1.87	*0.44*	*0.05*	*0.23*	*0.20*	*0.65*	*0.30*	*0.04*	*0.10*

CM = centre of mass location relative to hip joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$ R = radius of gyration, as a percentage of segment length

Table 51. Shank inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2 \times 10^2$)	Iyy ($kgcm^2 \times 10^2$)	Izz ($kgcm^2 \times 10^2$)	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	1.1	3.3	-41.2	6.45	6.38	0.66	27.0	26.8	8.6
2	2.3	2.1	-42.4	2.26	3.32	0.46	28.1	28.0	10.4
3	0.1	2.6	-41.9	3.28	3.24	0.40	27.3	27.2	9.5
4	2.9	4.2	-41.9	2.90	2.86	0.41	28.0	27.9	10.5
5	1.2	3.4	-38.9	3.15	3.14	0.46	26.9	26.9	10.3
6	1.8	1.1	-39.0	4.45	4.43	0.70	26.2	26.2	10.4
7	1.9	2.3	-38.7	5.52	5.49	0.95	27.3	27.2	11.3
8	0.9	4.2	-40.8	4.52	4.52	0.82	26.8	26.8	12.1
9	1.8	3.3	-41.0	4.09	4.10	0.84	28.2	28.2	12.7
normal, M	*1.8*	*3.0*	*-41.9*	*4.00*	*3.95*	*0.48*	*27.6*	*27.5*	*9.8*
SD	*1.0*	*0.9*	*0.5*	*1.65*	*1.63*	*0.12*	*0.6*	*0.6*	*0.9*
high, M	*1.5*	*2.9*	*-39.7*	*4.35*	*4.34*	*0.77*	*27.1*	*27.1*	*11.4*
SD	*0.5*	*1.2*	*1.1*	*0.85*	*0.84*	*0.19*	*0.7*	*0.8*	*1.06*
high/norm, M SD	*0.94*	*0.96*	*0.94*	*1.38*	*1.40*	*1.84*	*0.97*	*0.98*	*1.11*
	0.84	*0.42*	*0.03*	*0.28*	*0.29*	*0.57*	*0.03*	*0.03*	*0.11*

CM = centre of mass location relative to knee joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$ R = radius of gyration, as a percentage of segment length

Table 52. Foot inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2 \times 10^2$)	Iyy ($kgcm^2 \times 10^2$)	Izz ($kgcm^2 \times 10^2$)	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	1.8	26.4	-22.1	0.63	0.14	0.62	26.0	12.2	25.7
2	0.7	20.3	-23.5	0.46	0.11	0.43	26.0	12.7	25.3
3	1.3	17.2	-21.7	0.51	0.11	0.50	25.5	12.0	25.3
4	1.2	20.4	-22.5	0.40	0.09	0.39	25.9	12.4	25.4
5	1.3	21.5	-24.2	0.37	0.09	0.35	26.4	13.4	25.7
6	1.2	22.9	-19.2	0.53	0.10	0.53	25.5	11.1	25.3
7	2.8	24.4	-17.9	0.62	0.12	0.63	25.4	11.2	25.5
8	0.2	21.6	-20.6	0.39	0.09	0.39	25.4	12.5	25.3
9	0.6	19.9	-19.0	0.44	0.11	0.45	25.8	12.8	26.0
normal, M *SD*	*1.3* *0.4*	*21.1* *3.9*	*-22.4* *0.8*	*0.50* *0.10*	*0.11* *0.02*	*0.48* *0.10*	*25.9* *0.2*	*12.3* *0.3*	*25.4* *0.2*
high, M *SD*	*1.2* *1.0*	*22.1* *1.7*	*-20.2* *2.4*	*0.47* *0.10*	*0.10* *0.01*	*0.46* *0.11*	*25.7* *0.4*	*12.2* *1.00*	*25.6* *0.3*
high/norm, M *SD*	*1.23* *0.84*	*1.13* *0.17*	*0.89* *0.09*	*1.06* *0.17*	*1.02* *0.13*	*1.09* *0.19*	*0.99* *0.02*	*0.98* *0.07*	*1.00* *0.01*

CM = centre of mass location relative to ankle joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$ R = radius of gyration, as a percentage of segment length

Table 53. Head inertial parameters. The mean (SD) of individual groups and the relative difference (high/norm) is provided. The effect of BMI can be evaluated by comparing the ratio of mean (minus 1): standard deviation of the high/norm parameter (see methods: *Effect of obesity on BSIPs)*

Participant	CMx (%length)	CMy (%length)	CMz (%length)	Ixx ($kgcm^2 \times 10^2$)	Iyy ($kgcm^2 \times 10^2$)	Izz ($kgcm^2 \times 10^2$)	Rxx (%length)	Ryy (%length)	Rzz (%length)
1	0.0	9.1	52.5	2.67	2.34	1.48	30.0	28.1	22.3
2	1.9	10.9	54.9	4.24	3.91	2.04	31.2	29.9	21.6
3	0.9	6.1	51.2	3.03	2.65	1.59	30.7	28.7	22.2
4	0.4	8.2	55.6	3.68	3.38	1.80	31.6	30.3	22.2
5	0.5	12.8	47.0	3.98	3.55	1.96	31.3	29.6	21.9
6	2.5	12.8	46.7	3.84	3.23	2.18	32.8	30.1	24.7
7	1.2	4.8	45.7	8.54	7.23	4.86	33.0	30.4	24.9
8	1.8	7.2	44.9	3.16	3.04	2.08	30.8	30.2	25.0
9	0.3	5.6	46.0	4.40	4.20	2.18	32.7	32.0	23.1
normal, M *SD*	*0.8* *0.8*	*8.6* *2.0*	*53.5* *2.1*	*3.40* *0.69*	*3.07* *0.71*	*1.73* *0.25*	*30.9* *0.7*	*29.3* *1.0*	*22.1* *0.3*
high, M *SD*	*1.3* *0.9*	*8.6* *3.9*	*46.1* *0.9*	*2.8* *0.9*	*4.25* *1.72*	*2.65* *1.24*	*32.1* *1.01*	*30.5* *0.9*	*23.9* *1.4*
high/norm, M *SD*	*1.61* *1.55*	*0.94* *0.23*	*0.85* *0.03*	*1.35* *0.84*	*1.32* *0.80*	*1.49* *0.89*	*1.03* *0.04*	*1.02* *0.03*	*1.09* *0.60*

CM = centre of mass location relative to C7 joint centre, as a percentage of segment length
x = medial-lateral; y = anterior-posterior; z = superior-inferior
I = moment of inertia, $kgcm^2$ R = radius of gyration, as a percentage of segment length

Appendix: Comparison of Trunk Segments and Extremities

ANOVA

Table 54. ANOVA results for effect of model, BMI (group) and parameter on upper trunk inertial parameters.

	df	F	p	η_p^2*
3-way (MODEL, PARAMETER, GROUP)				
MODEL	1, 7	22.21	< .001	.77
MODEL*PARAMETER*GROUP	4, 28	8.34	< .01	.54
2-way (PARAMETER, GROUP)				
Pearsall				
GROUP	1, 7	5.54	=.051	.44
GROUP*PARAMETER	4, 28	7.48	< .01	.52
De Leva				
GROUP	1, 7	16.06	< .001	.70
GROUP*PARAMETER	4, 28	12.41	<.001	.64
Zatsiorsky				
GROUP	1, 7	1.07	> .34	Ns
GROUP*PARAMETER	4, 28	1.48	> .26	Ns
Hanavan				
GROUP	1, 7	1.17	> .32	Ns
GROUP*PARAMETER	4, 28	1.00	> .40	Ns

*effect size

Table 55. ANOVA results for effect of model, BMI (group) and parameter on middle trunk inertial parameters.

	df	F	p	η_p^2*
3-way (MODEL, PARAMETER, GROUP)				
MODEL	1, 7	5.16	< .05	.43
MODEL*PARAMETER*GROUP	4, 28	1.52	> .13	Ns
2-way (PARAMETER, GROUP)				
Pearsall				
GROUP	1, 7	7.15	< .05	.51
GROUP*PARAMETER	4, 28	13.87	< .001	.66
De Leva				
GROUP	1, 7	16.44	< .01	.70
GROUP*PARAMETER	4, 28	7.40	< .001	.52
Zatsiorsky				
GROUP	1, 7	31.04	< .001	.82
GROUP*PARAMETER	4, 28	8.71	< .001	.55
Hanavan				
GROUP	1, 7	9.72	< .05	.58
GROUP*PARAMETER	4, 28	11.51	< .001	.62

*effect size

Table 56. ANOVA results for effect of model, BMI (group) and parameter on lower trunk inertial parameters.

	df	F	p	η_p^2*
3-way (MODEL, PARAMETER, GROUP)				
MODEL	1, 7	83.93	< .001	.92
MODEL*PARAMETER*GROUP	4, 28	2.69	< .01	.44
2-way (PARAMETER, GROUP)				
Pearsall				
GROUP	1, 7	< 1	> .35	Ns
GROUP*PARAMETER	4, 28	4.84	< .01	.41
De Leva				
GROUP	1, 7	2.01	> .20	Ns
GROUP*PARAMETER	4, 28	6/17	< .01	.47
Zatsiorsky**				
GROUP	1, 7	2.60	> .15	Ns
GROUP*PARAMETER	4, 28	14.68	< .001	Ns
Hanavan**				
GROUP	1, 7	2.81	> .14	Ns
GROUP*PARAMETER	4, 28	6.16	< .01	.47

*effect size
**one parameter only, effect of GROUP evaluated with an independent samples t-test

Comparisons of body segments and inertial parameters

Note: negative numbers indicate greater relative estimates for the individual-specific model used in the current study

Table 57. Difference in upper arm inertial parameters between individual specific and predictive equations. Mean (SD) shown. Negative numbers indicate higher estimates for individual-specific measures.

	Mass	CMz	Ixx	Iyy	Izz
De Leva (1996)					
overall	-8.3 (9.7)	37.6 (13.2)	-37.7 (12.7)	-23.0 (19.1)	0.3 (38.0)
normal BMI	-4.3 (3.8)	28.3 (7.4)	-40.1 (7.2)	-29.7 (8.4)	34.4 (30.9)
high BMI	-11.4 (12.2)	45.0 (12.4)	-35.8 (16.5)	-17.6 (24.4)	-27.0 (8.6)
Zatsiorsky (1983)					
overall	-7.0 (9.9)	27.0 (17.6)	-24.7 (31.8)	-31.5 (28.3)	6.2 (44.3)
normal BMI	-5.2 (4.0)	12.9 (7.3)	-29.9 (2.1)	-17.9 (3.2)	47.3 (28.0)
high BMI	-8.4 (13.3)	38.2 (14.9)	-20.6 (44.4)	-42.4 (35.5)	-26.8 (17.0)
Hanavan (1964)					
overall	14.1 (13.1)	32.2 (11.0)	48.3 (52.2)	64.3 (67.0)	-42.3 (40.9)
normal BMI	13.5 (5.5)	26.3 (9.3)	32.6 (12.7)	38.7 (12.4)	-58.7 (11.3)
high BMI	14.6 (17.8)	36.8 (10.8)	60.8 (69.9)	84.9 (87.6)	-29.2 (52.6)

Table 58. Difference in foream inertial parameters between individual specific and predictive equations. Mean (SD) shown. Negative numbers indicate higher estimates for individual-specific measures.

	Mass	CMz	Ixx	Iyy	Izz
De Leva (1996)					
overall	24.4 (13.1)	32.1 (8.8)	36.1 (24.7)	50.1 (26.9)	63.7 (46.1)
normal BMI	26.7 (7.6)	38.9 (9.7)	48.1 (25.2)	60.6 (28.6)	105.7 (35.3)
high BMI	22.6 (17.0)	26.6 (1.4)	26.5 (22.1)	41.8 (25.2)	30.1 (11.8)
Zatsiorsky (1983)					
overall	21.0 (13.1)	54.3 (16.4)	44.1 (33.8)	58.0 (36.9)	83.6 (38.7)
normal BMI	25.1 (7.4)	69.1 (4.6)	52.7 (11.4)	63.1 (12.4)	97.3 (20.4)
high BMI	17.7 (16.5)	42.6 (11.4)	37.3 (45.4)	54.0 (50.5)	72.6 (48.5)
Hanavan (1964)					
overall	48.8 (16.6)	20.9 (7.8)	82.7 (34.5)	85.8 (34.3)	-59.2 (16.9)
normal BMI	49.4 (10.5)	27.5 (7.0)	93.5 (31.0)	93.1 (30.2)	-66.3 (5.1)
high BMI	48.2 (21.7)	15.6 (2.7)	74.1 (38.1)	79.9 (39.7)	-53.5 (21.4)

Table 59. Difference in thigh inertial parameters between individual specific and predictive equations. Mean (SD) shown. Negative numbers indicate higher estimates for individual-specific measures.

	Mass	CMz	Ixx	Iyy	Izz
De Leva (1996)					
overall	24.6 (14.4)	10.2 (18.8)	59.7 (53.0)	69.5 (54.4)	6.5 (46.0)
normal BMI	12.6 (11.6)	2.3 (12.7)	38.2 (51.4)	43.6 (52.1)	16.3 (39.3)
high BMI	34.2 (7.4)	16.5 (21.8)	76.9 (53.0)	90.2 (51.8)	-1.4 (53.8)
Zatsiorsky (1983)					
overall	26.7 (14.9)	62.7 (17.5)	72.5 (33.3)	83.1 (36.5)	20.7 (23.9)
normal BMI	14.5 (13.0)	48.6 (11.3)	47.8 (35.2)	53.3 (34.8)	13.9 (20.9)
high BMI	36.5 (7.0)	73.9 (12.7)	92.2 (13.8)	106.9 (12.6)	26.1 (27.0)
Hanavan (1964)					
overall	-10.9 (9.2)	2.3 (19.3)	-18.1 (26.1)	-12.8 (27.6)	-13.0 (27.1)
normal BMI	-18.1 (7.5)	-4.9 (14.3)	-34.9 (23.2)	-32.4 (23.6)	-39.5 (11.4)
high BMI	-5.2 (5.7)	8.1 (22.3)	-4.5 (21.1)	2.8 (20.3)	8.2 (10.3)

Table 60 Difference in shank inertial parameters between individual specific and predictive equations. Mean (SD) shown. Negative numbers indicate higher estimates for individual-specific measures.

	Mass	CMz	Ixx	Iyy	Izz
De Leva (1996)					
overall	7.4 (13.4)	22.5 (6.9)	11.5 (20.6)	17.8 (21.5)	28.1 (28.8)
normal BMI	-4.3 (8.8)	19.5 (5.4)	-1.7 (15.3)	4.4 (16.2)	43.8 (16.1)
high BMI	16.7 (7.5)	24.8 (7.7)	22.1 (19.0)	28.4 (20.2)	22.8 (37.1)
Zatsiorsky (1983)					
overall	5.3 (10.8)	0.3 (6.3)	19.8 (18.3)	24.2 (18.8)	41.6 (22.4)
normal BMI	-2.0 (9.0)	3.8 (6.2)	10.5 (22.1)	15.6 (23.4)	41.8 (20.7)
high BMI	11.2 (10.8)	-2.4 (5.3)	27.3) (12.)	31.0 (12.8)	41.4 (26.2)
Hanavan (1964)					
overall	28.8 (18.0)	9.2 (8.8)	55.0 (25.5)	56.1 (25.4)	-42.2 (23.8)
normal BMI	12.4 (11.50	14.2 (5.0)	47.2 (22.5)	49.0 (22.8)	-65,1 (6.2)
high BMI	41.9 (8.1)	5.3 (9.6)	61.3 (28.5)	61.7 (28.4)	-23.8 (12.4)

Appendix – Between-group comparisons in body segment paratmers

Table 61. Differences in principle BSIPs between high and normal BMI groups (high/normal) tested with two-sample, independent t-test. The normal BMI group comprised participants 6 - 8, but not the virtual subject (#9). A small reduction in effect size can be observed compared to the height-matching process used above. Normal BMI participants were also likely taller (M = 0.09, SD = 0.06 m), p = .06, an difference that likely attenuated certain between group differences (i.e. Ixx, Iyy). For comparison see tables 8 and 13.

	mass			CMy	CMz	Ixx	Iyy	Izz
	(g)	($\%_{body}$)	($\%_{trunk}$)					
Whole Trunk, M (SE)	1.64* (0.10)	1.15* (0.02)	--	4.41* (1.10)	0.89 (0.07)	1.81* (0.14)	1.77* (0.14)	2.64* (0.30)
Upper Trunk, M (SE)	1.49* (0.12)	1.04 (0.06)	0.90 (0.06)	1.88* (0.20)	1.02 (0.01)	2.11* (0.30)	1.77* (0.24)	2.05* (0.31)
Middle Trunk, M (SE)	2.00* (0.15)	1.41* (0.06)	1.22* (0.04)	1.85* (0.28)	1.02 (0.03)	3.47* (0.55)	2.84* (0.34)	3.53* (0.48)
Lower Trunk, M (SE)	1.46* (0.11)	1.04 (0.07)	0.91 (0.05)	1.18 (0.12)	0.92* (0.03)	1.99* (0.18)	1.70* (0.22)	2.33* (0.25)
Upper Arm, M (SE)	1.54* (0.09)	1.09 (0.06)	--	2.67* (0.73)	0.92 (0.05)	1.33 (0.19)	1.23 (0.18)	2.44* (0.42)
Forearm, M (SE)	1.50* (0.12)	1.06 (0.05)	--	0.57 (0.16)	1.03 (0.02)	1.70* (0.24)	1.64* (0.23)	2.21* (0.39)
Thigh, M (SE)	1.23* (0.06)	0.79* (0.04)	--	0.53* (0.39)	0.92* (0.02)	1.11 (0.10)	1.07 (0.09)	1.88* (0.28)
Shank, M (SE)	1.16 (0.10)	0.87* (0.04)	--	1.08 (0.19)	0.95* (0.01)	0.99 (0.16)	1.00 (0.16)	1.53* (0.21)

APPENDICES – STUDY 2

Appendix: Lifting Instructions

Self-Assessed Lifting Ability Instructions

Instructions for Adjusting Workload

We want you to imagine that you are on piece work, getting paid for the amount of work that you do, but working a normal 1 h shift that allows you to go home without feeling bushed.

In other words, we want you to work as hard as you can *without straining yourself, or without becoming unusually tired, weakened, overheated or out of breath.*

YOU WILL ADJUST YOUR OWN WORKLOAD. You will work only when you hear the beep. *Your job will be to adjust the load;* that is, to adjust the weight of the tote box that you are handling.

Adjusting your own workload is not an easy task. Only *you* know how you feel.

IF YOU FEEL YOU ARE WORKING TOO HARD, reduce the load by taking some weight out of the box.

WE DON"T WANT YOU LOAFING EITHER. If you feel that you can work harder, as you might on piece work, put in more weight.

DON'T BE AFRAID TO MAKE ADJUSTMENTS. You have to make enough adjustments so that you get a good feeling for what is too heavy and what is too light. You can never make too many adjustments – but you can make too few.

REMEMBER....

THIS IS NOT A CONTEST

EVERYONE IS NOT EXPECTED TO DO THE SAME AMOUNT OF WORK.

WE WANT YOUR JUDGMENT ON HOW HARD YOU CAN WORK WITHOUT BECOMING UNUSUALLY TIRED.

Appendix: Perceived Exertion

Ratings of Perceived Exertion

We want you to rate your perception of exertion. This feeling should reflect how heavy and strenuous the task feels to you, combining all sensations and feelings of physical stress, effort, and fatigue.

The scale ranges from 6 to 20, where 6 means "no exertion at all" and 20 means "maximal exertion". Choose the number that best describes your level of exertion. Try to appraise your feeling of exertion as honestly as possible, without thinking about what the actual physical load is.

6 No exertion at all
7 Extremely light
8
9 Very light - (a comfortable pace)
10
11 Light
12
13 Somewhat hard (it is quite an effort; you feel tired but can continue)
14
15 Hard (heavy)
16
17 Very hard (very strenuous, and you are very fatigued)
18
19 Extremely hard (you cannot continue for long at this pace)
20 Maximal exertion

Indicate your effort for each body part in the space below:

Whole Body: _____ Lower Body: _____

Low Back: _____ Shoulder: _____

Wrist: _____

Appendix: Relationship Amongst Lifting Outcomes

Correlation

Whole body perceived effort was correlated to exertion of all other regions, $r > .48$, $p < .01$, however a substantial amount of variation remained unaccounted (> 60%) (Table 62). Predictably, perceptions of effort at the shoulder were highly covariant with those at the wrist, $r = .84$, but surprisingly, also the lower body, $r = .80$.

The addition of objective measures (heart rate, mass lifted, BMI) explained very little of the variability in perceived effort ($r < .30$). There was a trend towards a negative relationship between BMI and perceived effort across all regions, however the effect size was relatively small and reached significance only for the wrist ($r = -.28$).

Table 62. Correlation matrix for relationship between region-specific perceived exertions, heart rate, mass lifted and BMI.

	Whole body	Lower body	Back	Shoulder	Wrist	Heart rate	Mass lifted	BMI
Whole body		.50**	.63**	.48**	.56**	.30*	.28*	-.23
Lower body	.50		.59**	.80**	.68**	.15	-.19	-.16
Back	.63	.59		.59**	.61**	.26*	.01	.01
Shoulder	.48	.80	.59		.84**	.24	-.04	-.14
Wrist	.56	.68	.61	.84		.21	.12	-.28*
Heart rate	.30	.15	.26	.24	.21		.51	.21
Mass lifted	.28	-.19	.01	-.04	.12	.51		.07
BMI	-.23	-.16	.01	-.14	-.28	.21	.07	

threshold for $p < .05$* was r = .25, for $p < .01$** was $r = .40$

Regression

Stepwise regression analysis to predict whole body RPE returned a model including the back and wrist regions, which accounted for 43% of the variability (Table 63).

Table 63. Regression model predicting whole body perceived exertion based upon region-specific measures. A backwards, stepwise approach was used.

	R^2 (adjusted)	B	SE B	β	P
Step 1	.42				
Constant		4.25	1.17		< .001
Lower		0.17	0.15	0.18	> .27
Back		0.45	0.13	0.44*	< .001
Shoulder		-0.22	0.20	-0.23	> .28
Wrist		0.35	0.17	0.36*	< .05
Step 2	.42				
Constant		4.17	1.17		< .001
Lower		0.08	0.12	0.08	> .54
Back		0.45	0.13	0.44*	< .001
Wrist		0.27	0.13	0.24	> .09
Step 3	.43				
Constant		4.33	1.13		< .001
Back		0.47	0.12	0.46*	< .001
Wrist		0.27	0.11	0.28*	< .05

The addition of objective measures returned a model that included BMI and mass lifted as predictors, and accounted for an additional 11% of variability in whole body RPE (Table 63). The negative β for BMI in the final model (-0.22) indicates that for every unit change in BMI, perceived effort decreases by ~1/5 of a unit. A model with heart rate in the place of the retained objective measures (mass lifted and BMI) returned a non-significant model (adjusted r^2 = .44, with $p > .17$).

Table 64. Regression model predicting whole body perceived exertion based upon region-specific measures, heart rate, mass lifted and BMI. A backwards, stepwise approach was used.

	R^2 (adjusted)	B	SE B	β	P
Step 1	.52				
Constant		4.34	1.79		<.05
Lower		0.26	0.14	0.28	> .07
Back		0.49	0.12	0.49	< .001
Shoulder		-0.10	0.19	-0.11	> .60
Wrist		0.05	0.18	0.05	> .77
Heart rate		0.01	0.03	0.04	> .73
Mass lifted		0.11	0.04	0.31	< .01
BMI		-0.10	0.04	-0.22	< .05
Step 2	.53				
Constant		4.43	1.76		< .05
Lower		0.26	0.14	0.28	> .07
Back		0.51	0.11	0.49	< .001
Shoulder		-0.06	0.14	-0.07	> .65
Heart rate		0.01	0.02	0.03	> .75
Mass lifted		0.12	0.04	0.32	< .01
BMI		-0.10	0.04	-0.23	< .05
Step 3	.54				
Constant		4.58	1.67		< .01
Lower		0.26	0.14	0.28	> .07
Back		0.51	0.11	0.50	< .001
Shoulder		-0.06	0.14	-0.06	> .68
Mass lifted		0.13	0.03	0.34	< .001
BMI		-0.10	0.04	-0.23	< .05
Step 4	.54				
Constant		4.57	1.66		< .01
Lower		0.22	0.10	0.24	< .05
Back		0.50	0.11	0.49	< .001
Mass lifted		0.12	0.03	0.34	< .001
BMI		-0.10	0.04	-0.22	< .05

APPENDICES – STUDY 4

Appendix: Effect of Repetition on Kinematics

No difference in range of motion was detected across all repetition, $F(3.8, 58.0) = 1.41$, $p > .24$, however the last repetition was 10.3% (SE = 5.0) less than the beginning, $t(15) = 2.73$, $p < .05$, $r = .49$. This was due to a decrement in top position, $F(2.8, 43.3) = 16.49$, $p < .001$, $\eta_p^2 = .52$, apparent by 42% of the trial, $F(2.8, 43.3) < 2.1$, $p > .16$., with a last repetition 18.1% (SE = 2.8) lower than at the start, $t(15) = 6.56$, $p < .001$, $r = .86$. No change in the bottom position was detected, $F(4.2, 62.7) = 0.77$, $p > .55$.

Consistent with an attenuation in position, but not duration, velocity was decreased at the end of both the raising (M = 13.8, SE = 7.1%), $t(15) = 1.95$, $p < .05$, $r = .48$, and lowering phases (M = 18.8, SE = 8.9%), $t(15) = 2.1$, $p < .05$, $r = .48$.

Lastly, alterations in acceleration differed by phase, with no change detected during the raising phase for either the positive (speeding up), $F(2.5, 37.3) = 1.34$, $p > .28$, or negative (slowing down) accelerations, $F(1.8, 27.3) = 1.32$, $p > .28$. In contrast, participants reduced the slowing (positive) acceleration of the lowering phase, $F(3.9, 59.0) = 3.32$, $p < .05$, $\eta_p^2 > .21$, by the end of the trial (M = -14.8, SE = 7.6%), $t(15) = 1.95$, $p < 0.05$, $r = .45$, but maintained a constant speeding up (negative) acceleration, $F(2.1, 31.0) = 0.49$, $p > .62$.