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Review

Potential contributions of skeletal muscle contractile dysfunction to altered biomechanics in obesity

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ABSTRACT

Background: Obesity alters whole body kinematics and joint kinetics during activities of daily living which are thought to contribute to increased risk of musculoskeletal injury, development of lower extremity joint osteoarthritis (OA), and physical disability. To date, it has widely been accepted that excess adipose tissue mass is the major driver of biomechanical alterations in obesity. However, it is well established that obesity is a systemic disease affecting numerous, if not all, organ systems of the body. Indeed, obesity elicits numerous adaptations within skeletal muscle, including alterations in muscle structure (ex. myofiber size, architecture, lipid accumulation, and fiber type), recruitment patterns, and contractile function (ex. force production, power production, and fatigue) which may influence kinematics and joint kinetics. This review discusses the specific adaptations of skeletal muscle to obesity, potential mechanisms underlying these adaptations, and how these adaptations may affect biomechanics.

1. Background

Obesity directly afflicts over one-third of American adults [1] and induces numerous kinematic alterations during activities of daily living (ADLs) [2–5] which are thought to contribute to development of musculoskeletal injuries [4,6], lower extremity osteoarthritis (OA) [7–9], and physical disability [10–13]. Indeed, it has been estimated that for every kg increase in body mass, risk of OA increases by up to 13% [14]. Numerous studies have examined the effects of obesity on biomechanics during various ADLs [2–4,7,15–22]. These studies have largely focused specifically on the effects of obesity on lower extremity kinematics and kinetics during gait, and to a lesser extent, rising from a chair. The specific kinematic and kinetic adaptations to obesity during these ADLs have been reviewed previously [23] and are summarized in Table 1. The purpose of this review is to discuss skeletal muscle adaptations to obesity, potential mechanisms underlying these adaptations, and how these may contribute to altered biomechanics.

Currently, weight loss is the major recommended intervention for improving biomechanics and reducing risk of joint injury, OA, and disability in obesity. However, a recent meta-analysis of 18 wt loss studies (N = 1636) indicates that 3–24 months of intervention resulted only in 4.9 kg (diet alone) and 6.7 kg (diet + exercise) weight loss which corresponded to 1.9 and 2.5 kg/m^2 reduction in body mass index (BMI), respectively [24]. Although this degree of weight loss will likely decrease risk of complications of obesity, it is likely that weight-reduced individuals remain at substantially higher risk of physical disability and lower extremity OA than their normal weight peers. Furthermore, weight regain is common following weight loss interventions [24]. Indeed, Kramer et al. [25] reported that less than 3% of their 152 subjects maintained their reduced body mass five years after a 15 wk weight loss intervention. Therefore, weight loss interventions are unlikely to completely ameliorate risk of developing injury or OA, especially in long-term interventions. Therefore, it is imperative to explore and understand mechanisms other than body mass per se which may contribute to obesity-induced biomechanical alterations.

2. Effects of obesity on skeletal muscle contractile function

In addition to increasing body mass, obesity elicits numerous skeletal muscle adaptations including altered contractile function (force, power, and fatigue), muscle structure (myofiber size, architecture, lipid accumulation, and fiber type), and recruitment patterns. Table 2 summarizes previously published effects of obesity on skeletal muscle.

2.1. Skeletal muscle strength

Absolute muscle strength is increased in obesity [26–31], at least in younger adults. Indeed, it has previously been suggested that increased muscle strength is a beneficial adaptation to obesity [29]. It should be

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Table 1
Previously reported biomechanical adaptations to obesity. Previous studies examining biomechanics during gait and sit-to-stand are presented. The effect of obesity (relative to lean) and the approximate magnitude of the effect of obesity is indicated. O = obese; L = lean; GRF = ground reaction forces; sag = sagittal plane.

Study	Activity	Subjects		Outcomes	Effect of Obesity	Magnitude of Effect
Browning and Kram *Med Sci Sports Exerc* 2007 [10]	Treadmill walking (0.50–1.75 m/s)	Lean BMI 20–25 Age: 25–32 n = 10	Obese BMI 30–43 Age: 25–32 n = 10	GRF Stance duration Double support Swing time Step width Joint angles Hip moment (sag) Knee moment (sag) Ankle moment (sag)	O > L O > L O > L O < L O > L O = L O > L O > L O = L	60% 5–7% 15–20% 7–15% 30% 70% 50%
DeVita and Hortobagyi *J Biomech* 2003 [17]	Overground Walking (Self-selected pace)	Lean BMI 16–27.3 Age 20.8 n = 18	Obese BMI 32.4–58.7 Age 39.5 n = 21	Stance duration Swing time Hip extension Knee extension Ankle plantarflexion	O > L O < L O > L O > L O > L	3% 5% 5° 4° 5.5–7.5°
Lai et al. *Clin Biomech* 2008 [41]	Overground walking (Self-selected pace)	Lean BMI 21.3 Age 27.6 n = 14	Obese BMI 33.1 Age 35.4 n = 14	Walking speed Stride length Stance duration Double support Hip adduction Knee adduction Ankle eversion	O < L O < L O > L O > L O > L O > L O > L	12% 8% 3% 18% 3° 4.5–8° 5°
Lerner et al *Gait Posture* 2014 [43]	Treadmill walking (1.25 and 1.50 m/s)	Lean BMI 22.1 Age 26 n = 10	Obese BMI 35.0 Age 35 n = 9	Pelvis obliquity Hip extension Knee extension	O > L O = L O > L	N/A N/A
McMillan et al. *Gait Posture* 2010 [50]	Overground walking (Self-selected pace)	Lean BMI 20.3 Age 12–17 n = 18	Obese BMI 44.6 Age 12–17 n = 18	Hip extension Hip abduction Knee extension Knee abduction Ankle plantarflexion Ankle eversion Hip flexion moment Hip adduction moment Knee extension moment Knee adduction moment Ankle dorsiflexion Ankle eversion	O > L O = L O > L O > L O > L O < L O > L O > L O > L O > L O > L O > L	8–12° 6° 6–9° 3° 2° 0.15–0.29 Nm/kgm 0.7–0.13 Nm/kgm 0.8–0.20 Nm/kgm 0.15 Nm/kgm 0.20 Nm/kgm 0.04 Nm/kgm
Spyropoulos et al. *Arch Phys Med Rehabil* 1991 [74]	Overground walking (Self-selected pace)	Lean Age 30–47 n = 12	Obese (70–99% ideal Body mass) Age 30–47 n = 12	Walking speed Stride length Step width Hip flexion Hip abduction Knee flexion Ankle dorsiflexion	O < L O < L O > L O = L O < L O = L O > L	33% 25% 200% N/A N/A
Galli et al. *Int J Obes* 2000 [22]	Sit-to-stand	Lean BMI 22 Age 28.0 n = 10	Obese BMI 40 Age 39.4 n = 30	Trunk flexion Hip moment Knee moment	O > L O < L O > L	17° 0.240 Nm/kgm 0.437 Nm/kgm
Huffman et al. *Gait Posture* 2015 [32]	Sit-to-stand	Lean BMI 22.1 Age 24.9 n = 10	Obese BMI 31.2 Age 33.4 n = 9	Sit-to-stand duration Trunk flexion Hip flexion Hip abduction Hip abduction moment Medial GRF	O = L O = L O = L O > L O > L O > L	5° 0.073 Nm/kgm 0.204 N
Schmid et al J Appl Biomech 2013 [68]	Sit-to-stand	Lean-overweight BMI < 17.5–29.9 Age: 50.9 n = 10	Obese BMI > 30, > 35 Age: 48.9, 48.5 n = 10, 16	Peak velocity Mean velocity Total time (5 cycles) Toe-out angle Interheel distance	O < L O < L O > L O = L O = L	23% 20% 37%
Sibella et al. *Clin Biomech* 2003 [72]	Sit-to-stand	Lean BMI 23.0 Age 26.5 n = 10	Obese BMI 37.9 Age 48.5 n = 40	Trunk flexion Hip moment Knee moment	O < L O < L O > L	24° 0.40 Nm/kgm 0.37 Nm/kgm