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Exposure to internal muscle tissue loads under the ischial tuberosities during sitting is elevated at abnormally high or low body mass indices

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ABSTRACT

Deep tissue injury (DTI) is a severe pressure ulcer characteristic of chairfast or bedfast individuals, such as those with impaired mobility or neurological disorders. A DTI differs from superficial pressure ulcers in that the onset of DTI occurs under intact skin, in skeletal muscle tissue overlying bony prominences, and progression of the wound continues subcutaneously until skin breakdown. Due to the nature of this silently progressing wound, it is highly important to screen potentially susceptible individuals for their risk of developing a DTI. Abnormally low and high values of the body mass index (BMI) have been proposed to be associated with pressure ulcers, but a clear mechanism is lacking. We hypothesize that during sitting, exposure to internal muscle tissue loads under the ischial tuberosities (IT) is elevated at abnormally high or low body mass indices. Our aims in this study were: (a) to develop biomechanical models of the IT region in the buttocks that represent an individual who is gaining or losing weight drastically. (b) To determine changes in internal tissue load measures: principal compression strain, strain energy density (SED), principal compression stress and von Mises stress versus the BMI. (c) To determine percentage volumes of muscle tissue exposed to critical levels of the above load measures, which were defined based on our previous animal and tissue engineered model experiments: strain $\geq 50\%$, stress ≥ 2 kPa, SED ≥ 0.5 kPa. A set of 21 finite element models, which represented the same individual, but with different BMI values within the normal range, above it and below it, was solved for the outcome measures listed above. The models had the same IT shape, size, distance between the IT, and (non-linear) mechanical properties for all soft tissues, but different thicknesses of gluteus muscles and fat tissue layers, corresponding to the BMI level. The resulted data indicated a trend of progressive increase in internal tissue loading, particularly in volumetric exposure to critical loading for BMI values outside the $17 \leq \text{BMI} \leq 22$ kg/m^2 range, supporting our hypothesis for this study. We concluded that exposure to internal muscle tissue loads under the IT during sitting is optimally reduced at the low-normal BMI range, which is important not only in the context of DTI research, but also for understanding general sitting biomechanics.

1. Introduction

Deep tissue injury (DTI) is a severe pressure ulcer characteristic of chairfast or bedfast individuals with impaired motosensory capacities (Black, 2005). A DTI differs from superficial pressure ulcers in that the onset of DTI occurs under intact skin, in skeletal muscle tissue overlying bony prominences, and progression of the wound continues subcutaneously until skin breakdown (Black, 2005). A DTI is therefore difficult to diagnose timely, and may cause death by sepsis, myocardial infarction, renal failure or multiple organ collapse (Agam and Gefen, 2007). A sitting-acquired DTI typically develops in the gluteus maximus muscles which envelop the ischial tuberosities (IT) during sitting (Linder-Ganz et al., 2007).

Due to the nature of this silently progressing wound, it is important to pre-screen patients for their risk of developing DTI. Several factors have been proposed in the epidemiological literature as being associated with an increased risk of developing pressure ulcers at large; a consensus exists regarding impaired mobility, poor nutrition manifested by a low body mass index (BMI=bodyweight/squared-height [kg/m^2]) and loss of subcutaneous fat (Garcia and Thomas, 2006). However, clinical studies aimed at identifying risk factors specific for DTI are limited in sizes of patient groups and involvement of co-morbidities (Nixon et al., 2006). There is also little scientific evidence regarding risk factors specific for DTI (Stekelenburg et al., 2007; Elsner and Gefen, 2008; Linder-Ganz and Gefen, 2009), though our group has previously suggested obesity as such (Elsner and Gefen, 2008).

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Finite element (FE) modeling for studying the effects of bodyweight on internal tissue loading is an efficient research methodology, as it allows isolating just the effect of bodyweight, or BMI, unlike experimental studies. Recently, we used FE modeling to explore the effect of obesity on tissue strains/stresses in the gluteus muscles during sitting, and found that there is a trend of increase in peak tissue loads with a rise in BMI (Elsner and Gefen, 2008). However, underweight effects were not studied, and also, only 4 overweight BMI values were simulated, yielding a rough estimation of trends. Additionally, the study of Elsner and Gefen (2008) did not consider sizes of tissue regions exposed to critical strain/stress levels. Using a rat model, we previously showed that muscle tissue can tolerate internal compression stresses below 2 kPa for up to 5 h without loss of viability, but higher stresses may induce permanent damage (Linder-Ganz et al., 2006). In a later study, employing tissue-engineered muscle constructs, we found that the corresponding critical (engineering) compression strains which can be safely tolerated by muscle over that time period were 50% (Gefen et al., 2008). The present study utilizes these data for calculating % muscle volume exposed to critical loading as function of BMI.

Based on the literature reviewed above, we hypothesize that during sitting, exposure to internal muscle tissue loads under the ischial tuberosities (IT) is elevated at abnormally high or low BMI. Our objectives were therefore to develop biomechanical models of the IT region in the buttocks that represent an individual who is gaining or losing weight drastically, and then to determine changes in internal tissue loading and exposures to loading versus the BMI.

2. Methods

In order to determine the effect of BMI on internal muscle tissue loads during sitting for a person who is losing or gaining weight, we used a set of 21 FE models. These models represent the same individual, but with different BMI values within the normal range ($18.5 < \text{BMI} \le 25\ \text{kg/m}^2$, 8 models), above it (overweight: $25 < \text{BMI} \le 30\ \text{kg/m}^2$, 4 models; obese class I: $30 < \text{BMI} \le 35\ \text{kg/m}^2$, 1 model; obese class II: $35 < \text{BMI} \le 40\ \text{kg/m}^2$, 2 models), and below it (underweight: $16.5 \le \text{BMI} \le 18.5\ \text{kg/m}^2$, 3 models; severely underweight: $\text{BMI} < 16.5\ \text{kg/m}^2$, 3 models). Specifically, the models had the same IT shape, size, distance between the IT, and mechanical properties for all soft tissues, but different thicknesses of gluteus muscle and fat tissues, corresponding to a BMI level, as detailed below (Fig. 1, Table 1).

One model within the normal BMI range, $\text{BMI} = 19\ \text{kg/m}^2$, was built from real MRI anatomy of a subject (female, age 29 years, bodyweight 55 kg, height 1.7 m, no known orthopaedic or neuromuscular disorders), and was defined as the "reference model" (Fig. 1c). All other 20 models were artificial variants of this reference model, and simulated altered BMI. The MRI scanning protocol, the development of the reference model, and the methodology for altering the soft tissue anatomy in each variant model were described elsewhere (Linder-Ganz et al., 2007; Elsner and Gefen, 2008), and are summarized for completeness as follows.

A mirror-symmetric three-dimensional (3D) FE model of a 4 mm-thick slice through the seated buttocks of the subject, i.e. the reference model, was developed from coronal MRI scans taken during sitting in an open-MRI. The model geometry, including IT, skeletal muscle (gluteus maximus), smooth muscle, adipose tissue and 2 mm-thick skin (Fig. 1c), was built based on a scan at a non-weight-bearing posture, obtained while the subject was sitting on a rubber tire. A second MRI scan was conducted weight-bearing at comparable scan conditions, and yielded vertical sagging of the IT of this subject as 17.3 mm, which was consequently taken as the displacement boundary condition for the reference model (Linder-Ganz et al., 2007) (Fig. 2).

The geometry of the reference model was then modified using SolidWorks 2008 (ver. SP2.1, Dassault Systèmes SolidWorks Corp., MA, USA) to represent the altered anatomy and tissue loading changes associated with the variant BMI levels. The volume of fat tissue in each model was adjusted with respect to the reference model by uniformly shifting the superficial boundary of fat (Elsner and Gefen, 2008) (Fig. 1). We calculated percentage body fat in each BMI model by proportionally adjusting % body fat with % fat volume in the buttocks. The BMI characteristic of the change in buttocks anatomy was estimated using the empirical relationship (Jackson et al., 2002):

$$\%\text{body fat} = 4.35 \times \text{BMI} - 0.05 \times \text{BMI}^2 - 46.24 \quad (1)$$

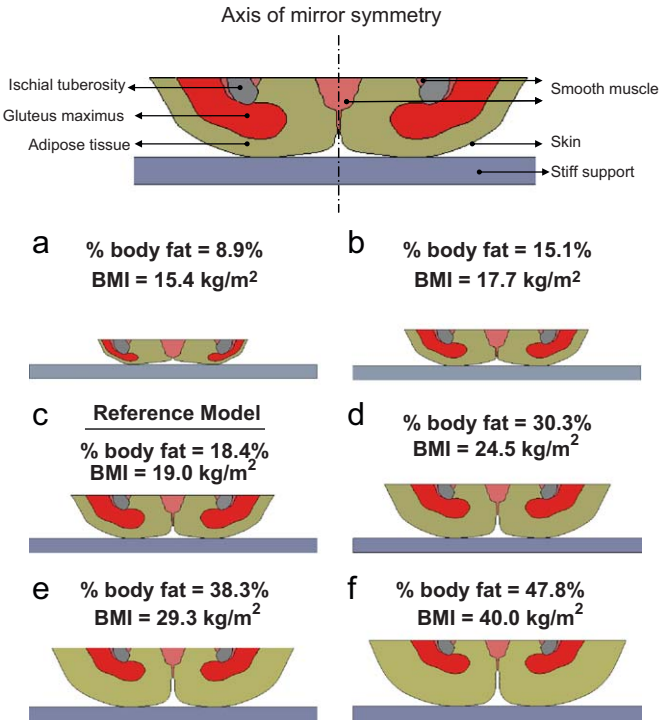

Fig. 1. Example of finite element model geometries simulating a severely underweight buttocks anatomy (a), underweight buttocks (b), normal buttocks (c,d), overweight buttocks (e), and class II obese buttocks (f). % body fat per each simulation case was calculated by proportionally adjusting % body fat with % fat volume in the buttocks per each model. The body mass index (BMI) was then estimated from Eq. (1), and BMI were categorized consistent with the World Health Organization BMI categories.

For the underweight models, we also simulated loss of muscle mass that is physiologically coupled with fat loss (Fig. 1a and b) by correspondingly shifting the superficial border of the gluteus muscles so that the ratio between the glutei and fat tissue thicknesses under the IT was kept constant at 1.8, based on our previous MRI studies (Linder-Ganz et al., 2008). The resultant fat and muscle tissue thicknesses corresponded with experimental data (Chan et al., 2006; Zaybak et al., 2007).

Constitutive laws and properties for tissues were adopted from previous studies. Specifically, the IT were considered as an isotropic linear-elastic material with elastic modulus of 7 GPa and Poisson's ratio of 0.3 (Elsner and Gefen, 2008). Muscle, fat and skin tissues were assumed to be nearly incompressible (Poisson's ratio 0.495), non-linear isotropic materials, which undergo stress relaxation under the sustained tissue deformations caused by sagging of the weight-bearing IT (Fig. 2). The elastic behavior of muscle and fat was described using a neo-Hookean material model, with strain energy density (SED) function $W_{f,m}$ (Linder-Ganz and Gefen, 2009):

$$W_{f,m} = \frac{G_{ins}}{2}(\lambda_1^2+\lambda_2^2+\lambda_3^2-3)+\frac{k}{2}(J-1)^2 \quad (2)$$

where G_{ins} is the instantaneous shear modulus, taken as 7.1 kPa for muscle (Palevski et al., 2006) and 0.286 kPa for fat (Gefen and Haberman, 2007), k the bulk modulus, λ_i the principal stretch ratios, and $J=\det(F)$ where F is the deformation gradient tensor. The elastic behavior of skin was described using a 2nd-order-polynomial (Mooney) SED function:

$$W_s = C_{10}(\lambda_1^2+\lambda_2^2+\lambda_3^2-3)+C_{11}(\lambda_1^2+\lambda_2^2+\lambda_3^2-3)(\lambda_1^{-2}+\lambda_2^{-2}+\lambda_3^{-2}-3) \quad (3)$$

where $C_{10}=9.4$ kPa and $C_{11}=82$ kPa are the instantaneous stiffness coefficients (Hendriks et al., 2003).

Since long-term viscoelastic behavior of muscle, fat and skin is evident experimentally within an order of minutes (Palevski et al., 2006; Gefen and Haberman, 2007; Liu and Yeung, 2008), whereas onset of DTI takes an order of hours (Gefen, 2008), only long-term tissue stresses were calculated using (Linder-Ganz et al., 2007):

$$S \cong (1-\delta)\frac{\partial W}{\partial E} \quad (4)$$

where S is the 2nd-Piola–Kirchhoff stress tensor; E the Green–Lagrange strain tensor; and δ the percentage difference between the instantaneous and long-term shear moduli, taken as 0.9 for muscle (Palevski et al., 2006), 0.64 for fat (Gefen and Haberman, 2007), and 0.73 for skin (Liu and Yeung, 2008).