ESTIMATING MUSCLE FORCES AND JOINT CONTACT FORCES IN THE ANTERIOR CRUCIATE LIGAMENT DEFICIENT KNEE

by

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ABSTRACT

Many of the estimated 200,000 individuals who sustain anterior cruciate ligament (ACL) injuries annually in the U.S. experience characteristic knee instability with daily activities ("noncopers"). The risk for developing knee osteoarthritis (OA) increases after injury, and within 20 years half of ACL-injured patients will have radiographic OA, significant pain and functional limitation. The noncoper gait strategy has been described in terms of aberrant movement and altered muscle activity, but neither muscle forces nor joint loads have been investigated. Further characterization of their neuromuscular strategy and knee joint loading will ultimately expand our understanding of the onset and progression of knee OA after ACL injury.

Therefore, the first aim of this work was to identify the well-described decreased injured limb peak extensor moment in a group of noncopers and to identify the muscle force adaptation which drove that gait asymmetry. We used an EMG-driven modeling approach to partition flexor and extensor group forces during normal walking. We found that noncopers accomplished the decreased peak moment with decreased muscle force from both flexor and extensor muscle groups. This strategy may be explained by muscle weakness which frequently accompanies ACL injury, or by apprehension, low confidence and fear of further injury.

The second aim of this work was to determine whether medial and lateral joint contact forces are altered acutely after ACL rupture. Medial and lateral compartment loads were calculated using muscle forces obtained from EMG-driven modeling. Noncopers unloaded their injured knees. This adaptation may cause

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cartilage deconditioning, resulting in an increased potential for cartilage insult with the resumption of normal or excessive loading.

The lower muscle force in the injured limb of these patients affirms the existence of neuromuscular asymmetries following ACL injury. The loading analyses provide a basis from which to compare longitudinal changes in knee joint loading.

Chapter 1

INTRODUCTION

Consequences of ACL Injury

The majority of individuals with ACL deficiency fail to dynamically stabilize their knee, even during daily activities [1]. These individuals with characteristic knee instability and poor function are called <u>noncopers</u> [2], and represent the majority of individuals with ACL injury.

Loss of quadriceps strength and injured limb function are nearly universal consequences of ACL injury. In acutely injured patients, quadriceps strength deficits for the injured limb range from 14-25%, when compared to the uninjured limb [3-9]. Quadriceps strength deficit is linked to poor knee function after injury [3, 10]. Hamstring strength deficits for the injured limb may also exist and range from 14-19% [8, 11].

After injury, individuals with ACL-deficiency exhibit altered movement patterns during various tasks [7, 12-15]. Movement patterns during walking may have the greatest impact on long-term joint health because an individual may take more than a million steps each year. After injury, noncopers walk with decreased knee excursions and moments [7, 14, 15]. These altered movement patterns may be related to quadriceps weakness [6].

The major long-term consequence of ACL injury is the development of early-onset osteoarthritis (OA). Within 10-20 years of injury, approximately half of all ACL-injured knees exhibit radiographic OA with significant pain and functional

limitation [16]. The risk for OA is no less for individuals who have their ligament reconstructed when compared to those who do not [17-19].

The estimated direct medical costs associated with symptomatic knee OA range from \$1,000 to 4,100 per person, per year in the U.S. [20-22]. Total knee replacement remains the eventual treatment for painful and debilitating knee OA, incurring direct medical costs exceeding \$11 billion annually [23].

Muscles: The Effectors of Aberrant Movement Patterns

Muscles are the force-producing and primary energy-absorbing elements of the human musculoskeletal system. The aberrant movement patters observed after ACL injury are driven by muscle forces. Numerous studies report the persistence of aberrant gait patterns following reconstructive surgery [24-29], which casts importance on understanding the muscle actions which cause them. The key to restoring normal movement lies with re-training and restoring strength in the muscles affected by injury. A better understanding of the noncopers' muscular strategy may promote more successful rehabilitation programs to resolve gait asymmetries in these individuals.

Muscles also provide the majority of the load to the articular surface of the knee [30]. Altered muscular strategies will result in not only altered movement patterns, but also altered joint loading.

Loading and the Pathogenesis of Osteoarthritis

Joint loading is related to the development of osteoarthritis. This conclusion follows *in vitro* and *in vivo* animal studies which show that cartilage responds negatively to excessive loading [31]. Additionally, epidemiological studies linked obesity to development of knee osteoarthritis in humans [32, 33]. Consequently, the mechanism for joint degeneration is widely thought to be overloading. Specifically, after ACL injury, the initiation of the OA has been ascribed to altered kinematics and the progression to altered loading [34, 35]. However, the loading in the ACL-injured knee is not known.

Animal models of ACL transection reflect the natural history of ACL rupture in humans: progressive erosion of the cartilage which ends in OA [36-38]. The importance of joint loading in the onset of cartilage degeneration is supported by the fact that OA results even from surgical transection, which does not create concomitant cartilage lesions seen with natural ACL injury in humans. Although concomitant joint trauma (i.e. articular cartilage defects, meniscal lesions) is an independent risk factor for OA development in humans [39], altered joint loading remains the common denominator associated with cartilage degeneration in ACL-deficient animals and humans.

The importance of neuromuscular compensations for ACL injury to the initiation and progression of knee OA is not well understood. Many of the estimated 200,000 individuals who sustain ACL injuries annually in the US [40] experience a considerable degree of instability with daily activities ("noncopers" [2]). Their early compensation strategy consists of altered sagittal knee excursions and moments as well as elevated muscular co-activation [7, 14, 15]. One or more of these components may contribute to an altered internal loading environment at the knee, posing an insult

to articular cartilage. Knowledge of joint loads in ACL-injured individuals will further characterize the potential impact of their aberrant movement strategies on long-term joint health.

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Chapter 2

SPECIFIC AIMS

The effects of neuromuscular compensations for anterior cruciate ligament (ACL) rupture on long-term joint health are unknown. Risk for developing knee osteoarthritis (OA) increases with ACL injury, regardless of whether or not the ligament is reconstructed. In particular, those individuals who compensate poorly for injury (noncopers) demonstrate altered muscle activity and kinematics acutely after injury. Their neuromuscular strategy implies altered joint loading and may be the precipitating cause of OA in the injured joint. However, in vivo joint loads are difficult to measure and have not been evaluated among individuals with ACL deficiency.

Muscles are the shock-absorbing and force-producing elements which control joint motion and loading. The characteristic decreased knee moment in ACLdeficient gait is a significant gait asymmetry, though it may be accomplished by several muscular strategies. Relative contributions of opposing muscle groups which coordinate to produce the decreased knee moment are unknown.

Several significant questions remain unanswered in the current body of literature. What combination of muscle forces result in the decreased peak knee moments observed during walking? What are the consequences of the non-coper neuromuscular strategy on knee joint loading? Do non-copers adopt a gait strategy which shifts load from one compartment to the other?

The **overall goal** of this work is to further characterize the noncoper neuromuscular strategy and to describe joint loading patterns acutely after ACL injury.

The **specific aims** of this work are to investigate:

- 1) Whether the decreased peak moment on the injured limb of noncopers is accomplished with decreased quadriceps force or increased hamstring force
 - Hypothesis 1.1 Noncopers exhibit similar extensor force on their injured limbs compared to their uninjured limbs
 - Hypothesis 1.2 Noncopers exhibit greater flexor force on their injured limbs compared to their uninjured limbs

2) Whether medial and lateral joint contact forces during gait are altered acutely after ACL rupture among noncopers

Hypothesis 2.1 – Noncopers exhibit greater medial compartment loading on their injured limbs than on their uninjured limbs

- Hypothesis 2.2 Noncopers exhibit greater lateral compartment loading on their injured limbs than on their uninjured limbs
- Hypothesis 2.3 Load sharing between the medial and lateral compartments will be similar between the injured and uninjured limbs

Chapter 3

MUSCLE FORCES AND GAIT ASYMMETRIES AFTER ACUTE ACL RUPTURE

Abstract

Background: The decreased peak knee extensor moment is a significant gait asymmetry among patients with anterior cruciate ligament (ACL) deficiency, yet the muscular strategy driving this altered moment for the injured limb is unclear. Hypothesis: We hypothesized that our noncopers would demonstrate a decreased knee moment, elevated flexor force and non-different extensor force for their injured limb when compared to their uninjured limb (a hamstring facilitation strategy). Study Design: Cross-Sectional Study; Level of evidence, 3. Methods: Gait analysis was performed on 30 athletes with acute ACL rupture. An EMG-driven modeling approach was used to estimate muscle forces which were grouped for analysis, normalized to bodyweight and compared between limbs. Variables of interest were peak knee moment, force at peak knee moment and peak muscle group force. Results: As expected, noncopers demonstrated a decreased peak knee moment for their injured limb, but they exhibited neither an isolated decrease in quadriceps force (quadriceps avoidance), nor an isolated increase in hamstring force (hamstring facilitation) at the point of peak knee moment. Instead, they exhibited decreased muscle force from both flexor and extensor groups. Likewise, peak extensor, hamstring and gastrocnemius muscle group forces were all significantly lower for the injured limb. Conclusions: The results of this study identify the neuromuscular asymmetries which drive the

aberrant movement patterns observed after ACL injury. The noncopers' strategy of decreased muscle force may be explained in part by muscle weakness which frequently accompanies ACL injury, or by apprehension, low confidence and fear of further injury. Clinical Relevance: The lower muscle force in the injured limb of these patients affirms the existence of neuromuscular asymmetries following ACL injury. Noncopers may benefit from a rehabilitation program which restores muscle strength and confidence in the function of their injured limb.

Introduction

Rupture of the anterior cruciate ligament (ACL) is a frequent, traumatic knee injury [1, 2] that often results in dynamic knee instability with daily activity. Those without dynamic knee stability acutely after injury are called noncopers [3] and represent the majority of ACL-injured patients [4]. ACL-deficient noncopers adopt an asymmetrical gait pattern which includes a well-described decreased peak knee extensor moment for the injured limb during gait [5-7]. This is an important gait asymmetry among patients with ACL injury [8]; however the muscular strategy driving this gait asymmetry is unclear. Numerous studies report the persistence of aberrant gait patterns following reconstructive surgery [9-14], which casts importance on understanding the causative muscle actions. The key to restoring normal movement lies with re-training and restoring strength in the muscles affected by injury. A better understanding of the noncopers' muscular strategy may promote more successful rehabilitation programs to resolve gait asymmetries in these individuals.

Several muscular stabilization strategies which could produce the decreased knee extensor moment have been proposed. These are based on the theory that ACL-injured individuals attempt to reduce anterior tibial translation (ATT), a

motion restrained by the intact ACL. One such strategy is a "quadriceps avoidance" [15] or a "quadriceps reduction" strategy [16]. The quadriceps muscles are responsible for generating the knee extensor moment early in stance; they also draw the tibia anteriorly on the femur. An isolated reduction in quadriceps force may represent an attempt to reduce ATT in the absence of normal ligamentous restraint and would also produce a smaller peak knee extensor moment. Alternatively, a "hamstring facilitation" strategy [17] may accomplish the smaller peak knee moment seen in ACL-deficient gait. The hamstrings oppose the knee extensor moment and apply a posteriorly-directed, restraining force to the tibia. An isolated increase in hamstring muscle force may represent an attempt to restrain ATT and would also result in a decreased peak knee extensor moment.

The net joint moment has limited usefulness in determining which muscular stabilization strategy noncopers employ because muscle activity patterns are altered after injury [5, 6, 17-19]. Relative contributions of opposing muscle groups which coordinate to produce the observed extensor moment are unknown. Muscle cocontraction has been observed in ACL-deficient knees [5, 6, 18, 19], which suggests that noncopers may employ the "hamstring facilitation" strategy. However, electromyography (EMG) cannot explicitly represent the timing and magnitude of muscle force (although it is a useful measure of neural command). Muscle force contributions from antagonistic groups cannot be evaluated with either the net joint moment or EMG alone.

Estimation of muscle forces in a population which exhibits neuromuscular compensation for injury requires a computational model which takes into account individual muscle activation patterns. Use of an EMG-driven musculoskeletal model

[20] provides estimates of muscle force from EMG. Subsequently, the net joint moment can be partitioned into flexor and extensor components and the relative contributions to the net joint moment can be examined. Analysis of muscle forces will enhance our understanding of the noncoper neuromuscular strategy and lead to improved rehabilitation design for individuals with ACL injury.

The purpose of this study was to 1) to evaluate the sagittal plane knee moments in 30 acutely-injured noncopers in order to determine whether they exhibited a decreased peak knee moment, 2) to partition flexor and extensor muscle forces for each limb in order to determine the source of the asymmetrical knee moment and 3) to compare peak muscle forces between limbs in order to further characterize the compensation strategy adopted by this group. We hypothesized that our patients would demonstrate a decreased knee moment, elevated flexor force and similar extensor force for their injured limb when compared to their uninjured limb.

Methods

Subjects

Thirty individuals (17 men and 13 women) with complete unilateral ACL rupture and who were functionally classified as noncopers [3] participated in this study (demographics listed in Table 3.1). Inclusion criteria were: complete, unilateral ACL rupture within 7 months of testing, regular participation in IKDC level I/II activities [21] and 14-55 years of age. Exclusion criteria were bilateral knee involvement, concomitant or symptomatic grade III injury to other knee ligaments, repairable meniscus tear or full-thickness articular cartilage defect greater than 1cm2.

Knee range of motion, effusion, pain and obvious gait impairments were treated and resolved prior to testing. ACL rupture was confirmed with MRI and through clinical examination with a KT 1000 arthrometer side-to-side difference less than 3mm. This work was approved by the institutional Human Subjects Review Board and all patients provided informed consent prior to testing.

29.3 ± 10.1 Age (yrs) Time from Injury (wk) 8.7 ± 8.1 Body mass (kg) 82.5 ± 14.9 Height (m) 0.09 1.75 <u>+</u> Walking speed (m/s) 1.54 ± 0.11

 Table 3.1. Patient demographics. Values represent means ± standard deviations.

Motion Analysis

Motion analysis was performed using an eight-camera video system (VICON, Oxford Metrics Ltd., London, UK) with a sampling rate of 120 Hz and force platform (Bertec Corporation, Worthington, OH) with a sampling rate of 1080 Hz. Walking speed varied within 5% of each subject's self-selected pace. Joint angles and moments were calculated using Visual3D software (C-motion, Germantown, MD). For analysis, knee joint moments were time-normalized to 100 percent of stance phase.

EMG was recorded at 1080 Hz using an MA-300 EMG system (Motion Lab Systems, Baton Rouge, LA) from 7 lower extremity muscles, bilaterally: vastus medialis and lateralis (VM and VL), rectus femoris (RF), semitendinosus (ST), long head of biceps femoris (BFL) and medial and lateral gastrocnemius (MG and LG). Subjects performed unilateral squats and isolated maximum voluntary isometric contractions for normalization. Linear envelopes for each muscle were normalized to the maximum value found during isometric or dynamic trials, which included a unilateral squat task and walking trials. Activity for semimembranosus (SM) and short head of biceps femoris (BFS) were assumed to be identical to activity recorded from SM and BFL, respectively. Activity for vastus intermedius (VI) was assumed to be the average of activities recorded from VM and VL.

Estimation of Muscle Forces

Muscle forces were estimated using an EMG-driven musculoskeletal model, which is described in detail elsewhere [20]. The analysis steps are briefly described here: anatomical scaling, EMG-driven model calibration and muscle force prediction.

The pelvis, femur, tibia and foot segments of the anatomical model (SIMM 4.0.2, Musculographics, Inc. Chicago, IL) were scaled according to subject-specific anatomical dimensions captured by the camera system during quiet standing. Stance phase kinematics and kinetics were input to the scaled model in order to obtain subject-specific muscle-tendon lengths and muscle moment arms for each gait trial.

During model calibration, muscle parameters that define the conversion of EMG to muscle force were allowed to vary within physiological bounds in order to characterize the EMG-to-force relationship in a subject-specific manner. In addition to two global strength gains (one for knee flexors and one for extensors), there were 6 parameters per muscle that uniquely define the EMG-to-force relationship. (For a detailed discussion of muscle parameters, see [20].) During model calibration, the

governing assumption was that the net flexor/extensor moment generated by muscle forces times their moment arms should equal the net sagittal plane joint moment which was calculated using inverse dynamics. In accordance with this assumption, the muscle parameters were iteratively adjusted within physiological bounds so that the sagittal plane moments from forward and inverse methods were in good agreement. A simulated annealing search strategy was used to obtain an optimal solution in which the squared difference between the two moment curves was minimized.

After calibrating the model and defining a subject-specific EMG-to-force relationship, muscle forces were predicted from EMG for novel gait trials. Each predicted knee joint moment (the sum of all predicted muscle forces times their moment arms) was compared to the measured moment calculated via inverse dynamics in order to ensure that the modeled joint moment converged to the measured joint moment from inverse dynamics.

Muscle forces were normalized to body weight (BW) and time-normalized to 100 percent of stance phase. Three trials per subject were averaged in this manner for each limb (in 7 limbs, only 2 trials were averaged). Muscle forces were grouped as knee flexors (FLEX) or extensors (EXT) for comparisons at peak knee moment. Subsequently, the FLEX group was subdivided into the hamstring group (HAMS) and gastrocnemius group (GAST) for comparison of muscle force peaks between limbs.

Data Analysis

Paired t-tests were performed (PASSW 18.0, SPSS Inc., Chicago, IL) to compare the following measures between limbs: measured peak knee extensor moment, EXT force at the point of peak knee extensor moment, FLEX force at the

point of peak knee extensor moment, peak EXT force during stance, peak HAMS force during stance, and peak GAST force during stance. Significance was set at 0.05.

Results

As expected, our ACL-deficient noncopers displayed a significantly lower peak knee extensor moment (p=0.0002) in the injured limb (Fig 3.1). Extensor force was significantly lower at the point of peak knee moment (p=0.0001) (Fig 3.2). Likewise, FLEX force was significantly lower in the injured limb at the point of peak knee moment (p=0.0188) (Fig 3.3).



Figure 3.1 Measured net knee moment during stance phase for both limbs (whiskers represent standard deviations). Internal extensor moments are positive. Asterisk (*) marks the occurrence of peak knee moment.



Figure 3.2 Extensor muscle group force for both limbs (whiskers represent standard deviations). The EXT group included VM, VL, VI and RF muscles. Asterisk (*) marks the occurrence of peak knee moment.



Figure 3.3 Flexor muscle group force for both limbs (whiskers represent standard deviations). The FLEX group included ST, SM, BFL, BFS, MG and LG muscles. Asterisk (*) marks the occurrence of peak knee moment.

Muscles of the injured limb exhibited a global decrease in peak forces when compared to the uninjured limb; however the timing of peak forces for each group was similar for both limbs (Table 2). Peak HAMS force occurred early in stance and was significantly lower in the injured limb (p=0.0011) (Fig 4). Similarly, peak EXT force occurred early in stance phase and was also significantly lower in the injured limb (p<<0.0001). Peak GAST force occurred late in stance and was significantly lower in the injured limb (p<<0.0001).

Discussion

Muscles are the effectors of aberrant gait strategies observed among ACLinjured individuals and better understanding of the muscular stabilization strategy adopted after injury will aid in rehabilitation design for these patients. The aims of this study were to 1) to determine whether our patients exhibited the typical decreased peak knee moment for the injured limb, 2) determine the muscular strategy causing the asymmetrical knee extensor moment and 3) further characterize the muscular compensation strategy adopted by this cohort by comparing peak muscle forces between limbs.

Consistent with previous reports [5-8], our patients with ACL deficiency demonstrated decreased peak knee extensor moments in their injured limbs during gait. This, together with a decreased knee flexion excursion, is the biomechanical hallmark of the noncoper [6].

	Limb	Peak Force	Occurrence
		(BW)	(% Stance)
EXT	Ι	1.36	23
	U	1.95	22
HAMS	Ι	1.18	8
	U	1.58	7
GAST	Ι	1.35	72
	U	1.77	75

 Table 3.2
 Peak EXT, HAMS and GAST force and their occurrence during stance phase. (*I*=injured, *U*=uninjured)



Figure 3.4 Ensemble muscle force curves for HAMS, EXT and GAST groups.

Noncopers accomplished the decreased peak knee moment for their injured limbs with decreased muscle force from both EXT and FLEX groups, rather than either of the expected strategies: isolated decrease in EXT force or isolated increase in FLEX force. Their strategy may be explained, in part, by muscle weakness, which frequently accompanies ACL injury. In acutely injured patients, quadriceps strength deficits for the injured limb range from 14-25%, when compared to the uninjured limb [22-25]. Hamstring strength deficits have also been reported for the injured limb, ranging from 14-19% [24, 26]. Aberrant gait patterns are associated with quadriceps strength deficits of as low as 20% for the injured limb in patients with ACL deficiency [27], which demonstrates the potential for muscle weakness to impact the biomechanics of low demand activities. Although only about 30% of maximal thigh muscle strength is utilized during normal walking, strength deficits observed acutely after ACL injury may explain the low muscle force utilization during gait.

The decreased EXT and FLEX forces at the point of peak knee moment were unexpected, given other studies which report that patients with ACL deficiency exhibit elevated muscle co-contraction in an attempt to stiffen and stabilize their injured knee [5, 6, 17, 18]. However, the muscle force results in this study are not necessarily contradictory of the co-contraction findings previously reported. Cocontraction utilizes EMG, which is a measure of neural command to muscle, whereas muscle force is the cumulative actuation of muscle. Furthermore, EMG is often normalized to reference value obtained during a volitional maximum test. As strength and activation deficits (esp. quadriceps) are common following ACL injury, one cannot assume that muscles of the injured limb generate as much force as those of the uninjured limb during volitional strength testing. Therefore, the normalization of EMG

to a maximal reference value does not necessarily accomplish normalization with respect to muscle force. By including flexor and extensor group strength gains in our model, our approach allows muscle actions to be adjusted to match the magnitude of the measured joint moment. Additionally, our approach provides a set of muscle forces that are constrained in timing and relative magnitude to recorded muscle activity.

Our patients demonstrated a global decrease in muscle force for the injured limb during stance (HAMS, EXT and GAST; Fig 4). This overall decrease in muscle forces may be explained by patients' apprehension after injury. Noncopers are those who lack functional knee stability and their experience after injury is one which is punctuated by low confidence in knee function and/or multiple episodes of givingway [3]. Despite having resolved obvious gait impairments, they may be simply unwilling to place normal demands on their injured limb and consequentially select a gait strategy which 'favors' their injured limb and requires less muscle force. While restoring muscle strength is an important prerequisite for symmetrical movement [27] and is important for joint function the long term [28], restoring confidence in knee function may also be an important component of rehabilitation after ACL injury. Fears associated with re-injury exist after reconstructive surgery [29, 30] and are coincident with low function [29]. However, there is no evidence which directly relates psychosocial obstacles to altered movement patterns after ACL injury. Apprehension and lack of confidence may be a factor which distinctly impacts movement strategies following ACL injury.

Muscle forces have been estimated for gait in uninjured individuals using forward methods [31], however explicit values for peak muscle force are surprisingly

scarce. Muscle force peaks reported by Shelburne et al. [32] were of similar shape but lower in magnitude compared to the uninjured limbs in this study. These were obtained using a cost function during optimization which assumes that the goal adopted by the central nervous system during walking is to minimize energy expenditure [33]. Although this cost function provides reasonable results for healthy gait, it may not be valid for the case of ACL injury. Movement patterns after ACL rupture are variable [6, 34, 35] which implies that the neuromuscular strategies are likewise diverse. The modeling approach used here partitions force among muscles using recorded muscle activity, and is uniquely suited to investigate muscle forces following ACL injury because it is sensitive to individual neuromuscular strategies.

Predictions of muscle force for ACL-deficient gait are few. Noyes et al. [36] predicted muscle forces for varus-aligned, ACL-deficient knees during slow walking (1.1 m/s) and reported higher EXT and lower FLEX peak forces for the injured limb than the present study. Muscle forces were obtained by reducing the number of muscles spanning each joint until the muscle force-joint moment problem could be solved uniquely. The present study is unique in that EMG was used to partition forces among muscles, incorporating in vivo muscle activity data in order to resolve the muscle redundancy problem. The higher muscle forces reported by Noyes et al. were likely a direct result of higher adduction moments measured in their varusaligned patients.

The lower muscle force for the injured limbs of these patients affirms the existence of neuromuscular asymmetries following ACL injury. Considering that biomechanical mal-adaptations can persist even after reconstruction, an important goal of rehabilitation is to restore normal movement by re-training and strengthening the

muscles affected by injury. Noncopers may benefit from a rehabilitation program which restores muscle strength and confidence in the function of their injured limb.

Muscles function primarily as joint accelerators, and as such, they apply loads to the articular surfaces. The adaptation of lower muscle forces during walking has the potential to impact joint health in the long term, considering that the lower muscle forces are likely accompanied by decreased loading and altered cartilage stresses. With time, decreased joint loads may result in de-conditioning of articular cartilage. Future work should investigate joint loading after ACL injury in order to determine the impact of their muscular strategy on joint loads, and subsequently on long-term joint health.

Conclusions

ACL-deficient noncopers employed a gait strategy which requires less global muscle force from their injured limb. They exhibited neither a 'quadriceps avoidance' nor a 'hamstrings facilitation' muscular stabilization strategy. Instead, they walked with decreased muscle force for both flexor and extensor muscle groups, as well as lower peak muscle forces for their injured limb. This strategy may be explained in part by muscle weakness which frequently accompanies ACL injury. Also, noncopers represent the majority of those with ACL injury and have low confidence in their knee and/or experience repeated episodes of giving-way on their injured limb. These individuals may be unwilling to place normal demands on their injured limb and therefore select a gait strategy that requires less global muscle force

from their injured limb. The results of this study identify the neuromuscular asymmetries that drive the aberrant movement patterns observed after ACL injury.

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Chapter 4

ALTERED LOADING IN THE INJURED LIMB AFTER ACL RUPTURE

Abstract

Articular loading is an important factor in the joint degenerative process for individuals with anterior cruciate ligament (ACL) rupture. Evaluation of loading for a population that exhibits neuromuscular compensation for injury requires an approach which can accommodate individual muscle activation strategies in its estimation of muscle forces. The purpose of this study was to evaluate knee joint loading for patients with ACL deficiency using an EMG-driven modeling approach to estimate muscle forces. Thirty athletes with acute, unilateral ACL rupture underwent gait analysis. Electromyography was recorded bilaterally from a total of 14 lower extremity muscles and input to a musculoskeletal model for estimation of muscle forces and joint loads. Patients loaded their injured knees significantly less than their uninjured knees. If excessive loads are the mechanism for onset of osteoarthritis, our results indicate that they are not widely present early after injury. The joint unloading observed in these patients after injury may cause cartilage deconditioning, resulting in an increased potential for cartilage insult with the resumption of normal or excessive loading.

Introduction

Articular loading is an important factor in joint degeneration for individuals with anterior cruciate ligament (ACL) rupture. The risk for developing

premature knee osteoarthritis (OA) increases with ACL injury, and within 10-20 years, approximately 50% of those with ACL-injured knees have radiographic knee OA, significant pain and functional limitation [1]. ACL insufficiency allows changes in tibiofemoral contact mechanics and dynamic loading—two factors which influence cartilage health and remodeling.

Cartilage tissue morphology is unmistakably related to the nature of the mechanical loads regularly sustained by the joint [2]. It is known from animal models that cartilage health suffers with repetitive over-loading [3], restriction of normal joint movement [4] and unloading [2], yet the mechanisms driving the onset of OA after ACL rupture are not well-defined. The early stages of cartilage degeneration in ACL deficiency are thought to be marked by the overloading of normally unloaded joint contact areas [5]. The magnitude of joint loads in the ACL-deficient knee is largely unknown. Characterizing the joint motion and loading environment soon after injury will help us to better understand the mechanical factors which are involved in the initiation of OA after ACL rupture.

The importance of neuromuscular compensations for ACL injury to the initiation and progression of knee OA is not well understood. Many of the estimated 200,000 individuals who sustain ACL injuries annually in the US [6] experience a considerable degree of instability with daily activities ("noncopers" [7]). Their early compensation strategy consists of altered sagittal knee excursions and moments as well as elevated muscular co-activation [8-10]. One or more of these components may contribute to an altered internal loading environment at the knee, posing an insult to articular cartilage. Knowledge of joint loads in ACL-injured individuals will further

characterize the potential impact of their aberrant movement strategies on long-term joint health.

The knee adduction moment serves as an estimate of the load distribution between the medial and lateral compartments and correlates with the severity and progression of knee OA [11]. Recently, use of the adduction moment as a measure of OA progression after ACL injury has been proposed [12]. Nonetheless, the relationship between net moment and joint loading is not straightforward [13], particularly when agonist/antagonist muscle groups are co-activated, as in ACLdeficiency [8, 9]. Muscle forces are important contributors to total joint contact force, and consequently, altered muscle activity can substantially affect the load of the articular surface. Since noninvasive measurement of muscle forces in vivo is impractical, a computational model must be used to estimate forces indirectly. Evaluation of joint loading for a population that exhibits neuromuscular compensation for injury requires an approach which can accommodate individual muscle activation strategies in its estimation of muscle forces.

Therefore, the purpose of this study was to evaluate articular loading for a group of acutely injured patients with ACL deficiency using an EMG-driven modeling approach. We hypothesized that peak medial, lateral and total tibiofemoral loads would be greater for the injured knee, compared to the uninjured knee.

Methods

Subjects

Thirty athletes with acute, unilateral ACL rupture (17 men, 13 women) participated in the study (mean \pm SD; age = 29.3 \pm 10.1 yrs, time from injury = 8.7 \pm 8.1 wks, BMI = 27.0 \pm 0.1 kg/m2, gait speed = 1.54 \pm 0.1 m/s). All patients were functionally classified as noncopers using the University of Delaware screening examination [14]. Inclusion criteria were: 13 to 55 years of age, regular pre-injury participation in level I or II activities [15] and complete ACL rupture within the past 7 months. Exclusion criteria were: bilateral knee involvement, presence of repairable meniscus tear, symptomatic grade III injury to other knee ligaments, full-thickness articular cartilage defect greater than 1 cm2, or presence of any other injuries which prevented completion of the screening exam. This work was approved by the University of Delaware Human Subjects Review Board, and all participants provided informed consent prior to testing.

Testing

Patients were tested upon gaining full knee range of motion, minimal effusion, and the ability to walk on the injured limb without pain or obvious gait impairments. Patients were asked to walk at their self-selected intentional speed and were required to maintain that speed throughout the testing session. Gait speed was monitored using two photoelectric beams which spanned the collection volume. Passive retro-reflective markers were used to define anatomical coordinate systems and track segment motion. Stance phase kinematics and ground reactions were recorded using an 8-camera video system (sampling rate 120 Hz) (VICON, Oxford Metrics Ltd., London, UK) and force platform (sampling rate 1080 Hz) (Bertec

Corporation, Worthington, OH). Joint angles and moments were calculated using inverse dynamics, performed with commercial software (Visual3D, C-Motion, Germantown, MD).

Electromyography (EMG) was recorded bilaterally using an MA-300 EMG System (sampling rate 1080 Hz) (Motion Lab Systems, Baton Rouge, LA) for a total of 14 lower extremity muscles: (bilaterally) the medial and lateral vasti, rectus femoris, medial and lateral gastrocnemeii, semitendinosus and long head of biceps femoris. Subjects performed unilateral squats and isolated maximum voluntary isometric contractions for normalization purposes. Raw data were high-pass filtered (2nd order Butterworth, cutoff 30 Hz), rectified and subsequently low-pass filtered (2nd order Butterworth, cutoff 6 Hz) to create a linear envelope. Linear envelopes were normalized to the maximum value found during isometric, unilateral squat or walking trials. EMG for vastus intermedius was assumed to be the average of the medial and lateral vasti linear envelopes. EMG for semimembranosus and short head of biceps femoris were assumed to be equal to that recorded from semitendinosus and long head of biceps femoris, respectively.

EMG-Driven Modeling

Muscle forces were estimated using a patient-specific EMG-driven model that is described in detail elsewhere [16]. Briefly, joint kinematics and EMGs were used as input to estimate muscle forces and muscle moments during gait. Analysis steps included: 1) anatomical scaling, 2) EMG-driven model calibration, 3) muscle force prediction, and 4) contact force calculation.

<u>Anatomical Scaling.</u> Anatomical dimensions captured for each patient during quiet standing, using the video system. These data were used to scale the

pelvis, femur, tibia and foot segments of the anatomical model (SIMM 4.0.2, Musculographics, Inc. Chicago, IL). Patient-specific muscle-tendon lengths and muscle moment arms were calculated for the stance phase of each gait trial.

<u>Model Calibration.</u> During model calibration, muscle parameters which characterize the EMG-to-force conversion (6 parameters per muscle, plus 2 global strength factors) were allowed to vary within physiological constraints. For a full discussion of model parameters modified during calibration, see the original publication [16]. Throughout the calibration process, it was assumed that the sum of the individual muscle moments (muscle forces × moment arms) obtained from EMGdriven forward methods should equal the net joint moment calculated from inverse dynamics. Muscle parameters were iteratively adjusted so that the sagittal plane moments from forward and inverse methods were in best agreement. A simulated annealing search strategy was used to find an optimal solution in which the squared difference between the two moment curves was minimized.

<u>Muscle Force Prediction.</u> Upon calibration, the solution set of muscle parameters was fixed within the model. When given a set of EMGs for a novel movement trial, the model predicted muscle forces and muscle moments for that movement. Each predicted knee joint moment (the sum of all predicted muscle forces times their moment arms) was compared to the measured moment from motion analysis in order to ensure that the modeled moment converged to the measured joint moment from inverse dynamics. A typical set of predicted muscle forces is shown in Figure 4.1.



Figure 4.1 Representative set of muscle forces for the stance phase of gait. Muscles are semimembranosus (SM), semitendinosus (ST), long and short heads of biceps femoris (BFL, BFS), rectus femoris (RF), vastus medialis (VM), vastus lateralis (VL), vastus intermedius (VI), medial and lateral gastrocnemius (MG, LG). The hamstrings and quadriceps are majorly active during loading response, with VL carrying the most force. Gastrocnemeii generate force primarily in the propulsive phase, with peak MG force being greater than that of LG.

Contact Force Calculation. A frontal plane moment balancing algorithm [17] was used to compute medial and lateral compartment contact forces. Medially and laterally offset segmental coordinate systems were constructed so that the knee adduction moment could be expressed about the contact point within each compartment. These points were fixed within subjects but varied between subjects according to individual anthropometry (25% of trans-epicondylar distance, measured medially and laterally from the joint center). The knee adduction moment calculated from inverse methods was expressed about each contact point. A static equilibrium was then assumed about the lateral contact point; the knee adduction moment about

the lateral contact point was balanced by muscle forces and by contact force acting at the medial contact point. Contact force for the medial compartment was the only unknown and could be determined at every point of stance. The same equilibrium was constructed for the medial compartment, allowing resolution of the lateral compartment contact force.

Variables of Interest

Contact forces for each gait trial were normalized to bodyweight (BW) and time-normalized to 100 samples. Three trials per subject were averaged in this manner for each limb (in 7 limbs, 2 trials were averaged).

Statistical analyses were performed with PASSW 18.0 software (SPSS Inc, Chicago, IL). Paired t-tests were used to compare peak loads (for tibiofemoral, medial and lateral compartments) between limbs. Significance was set at 0.05.

Results

Tibiofemoral contact force for the stance phase of gait was biphasic in shape, with the largest peak occurring near the end of weight acceptance phase and a second, lesser peak occurring in late stance (Table 4.1, Fig 4.2). Peak tibiofemoral load was significantly lower for the injured limb (p<0.0001).

	Limb	Peak Load (BW)		SD	Peak Load (N)		SD	Occurrenc (% Stance	e e)	SD
Tibiofemoral	Inj.	3.62	±	0.89	2924	±	706	23.1	±	2.8
	Un.	4.42	±	0.95	3597	±	939	21.8	±	3.1
Medial	Inj.	2.37	±	0.50	1916	±	397	24.2	±	3.3
	Un.	2.79	±	0.56	2279	±	604	22.9	±	2.3
Lateral	Inj.	1.56	±	0.5	1270	±	411	13.1	±	5.7
	Un.	2.01	±	0.71	1625	±	554	13.5	±	5.5

Table 4.1Peak loads for tibiofemoral, medial and lateral compartments and
their occurrence during stance phase.



Figure 4.2 Mean tibiofemoral contact forces (whiskers represent standard deviations) for the stance phase of gait.

Medial compartment contact force was also biphasic in shape. Peak medial load occurred early in stance and was significantly lower for the injured limb (p=0.002) (Fig 4.3). The lateral compartment exhibited only one consistent loading peak, which occurred early in stance. Peak lateral load was also significantly lower for the injured limb (p=0.0001) (Fig 4.4).



Figure 4.3 Mean medial compartment contact forces the stance phase of gait.



Figure 4.4 Mean lateral compartment contact forces for the stance phase of gait.

Discussion

The purpose of this study was to evaluate articular loading for a group of acutely injured patients with ACL deficiency using an EMG-driven modeling approach. Our major finding was that patients unloaded their injured knee compared to their uninjured knee. This result was unexpected in light of the high incidence of OA among individuals with ACL injury coupled with the dominant hypothesis that excessive joint loading is the mechanism by which cartilage degeneration is initiated [3, 18]. This study provides some of the first direct in vivo evidence in support of a theory of cartilage degeneration [19] which implicates changes in both contact mechanics and dynamic loading in the initiation of OA after ACL injury.

The etiology of osteoarthritis is multi-factorial, but the evidence points to mechanical factors as responsible for cartilage gains and losses as well as the site and severity of OA [2]. One mechanism for cartilage failure is elevated stress [18], which can be caused by excessive loads. Our findings indicate that elevated joint loading is not widely present in the acute period after injury (< 7 months).

The relative unloading of the injured knee which was observed in this study prompts the question of whether the onset of OA following ACL injury may be related to the acute unloading of articular cartilage. Cartilage, like other musculoskeletal tissues exhibits a unique structure-function relationship and appears to be conditioned to the loads it experiences. It has been demonstrated in studies of animal models that joint unloading is detrimental to cartilage health. Complete hind limb unloading in rats resulted in advancement of the calcification tidemark at the chondroosseous interface and cartilage thinning [20], and hind limb immobilization in dogs resulted in a reduced glucosaminoglycan concentration in un-calcified cartilage

[21]. These atrophic adaptations to joint unloading represent cartilage de-conditioning, and the reversibility of them with the return of normal loading is uncertain.

Most likely, a threshold magnitude and frequency of loading exists for maintenance of healthy cartilage. To our knowledge, no studies have investigated the effects of partial unloading on long-term articular cartilage health. Therefore, it is difficult to place the unloading observed among our patients into context. Effects of complete unloading (as done in animal studies) may be different than those of partial unloading (as observed in the ACL-deficient knees in this study). Determining the incidence of OA among these patients who demonstrate unloading acutely after injury may provide insight to the threshold loading magnitude required for the maintenance of healthy articular cartilage.

Decreased joint loading in the ACL-deficient knee has been suggested previously. Unloading appears to precede the onset of OA in the ACL-transected cat knee [22]. Two weeks following ACL transection, the animals walked with significantly decreased ground reactions and muscle forces for the injured limb, which strongly implies decreased joint loading. Other studies report decreased ground reaction forces [23-26] in ACL-transected animals. Degenerative changes are also observed early after transection, suggesting that the initiation of OA after ACL disruption may be associated with decreased rather than excessive joint loading.

Greater peak knee adduction moments have been identified in ACLreconstructed knees (ranging approximately 1-10 years post-surgery) compared to matched, uninjured knees [27]. These higher moments suggest that joint loading may be increased in the years following ACL injury and reconstruction. Unloading in the acute phase after injury may decrease the loading tolerance of articular cartilage in the

injured knee. The potential for cartilage insult is increased with the resumption of normal or excessive loading. More longitudinal studies are needed to investigate the loading profile in the ACL-injured knee in the years after injury in order to determine whether injurious cartilage over-loading occurs at a later time.

In this study, we strove to capture gait strategies in our patients acutely after injury, while controlling for the confounding effects of inflammation and conscious pain avoidance. Pain, effusion and range of motion impairments were resolved in all individuals prior to data collection, which allowed us to evaluate knee loading in the absence of trauma-related afferent activity from the joint capsule. Therefore, we propose that the unloading observed in these "quiet" ACL-deficient knees captures the effect of these patients' neuromuscular strategies on articular loading.

This is the first study to estimate medial and lateral compartment contact forces in patients with acute ACL rupture and normal knee alignment. There are a limited number of in vivo studies which report measured knee joint loading during walking.

Loading magnitudes for both limbs were higher in this study, compared to internal contact force measurements for an elderly individual with an instrumented total knee arthroplasty (TKA) [28]. However, our subjects were younger and walked faster and consequently, higher loading was expected. The shapes of the predicted loading patterns for medial and lateral compartments in our subjects were similar to those reported for the instrumented TKA [28]. Medial loading curves had two peaks occurring near 25% and 80% of stance, respectively. The lateral compartment did not become unloaded at any point during stance, for any of our subjects (Fig. 4). Other

modeling approaches [29, 30] predict lateral compartment unloading, which does not correspond to measurements from the instrumented TKA.

The approach used here is not without limitations. Ligaments were not included in our model, and consequently we expect that joint loads reported here may be under-estimates of the loading which occurs in vivo. Also, the fixation of medial and lateral tibiofemoral joint centers as discrete points throughout stance is a simplification incorporated into our model. We chose to fix medial/lateral compartment contact points at $\pm 25\%$ of epicondylar width (as measured from the joint center), as has been done previously [31].

Conclusions

Noncopers unload their injured knee acutely after injury, during normal walking. If excessive loads are the mechanism for onset of OA, these results indicate that they are not widely present acutely after injury. As cartilage is conditioned to the loads it regularly experiences, the partial unloading in the injured knee observed in these patients may result in cartilage deconditioning, and thereby play a role in the initiation of OA after ACL injury. Future work should investigate whether this unloading resolves with pre-operative rehabilitation and surgical intervention, and whether the duration and magnitude of unloading represents a significant deconditioning of articular cartilage in individuals with ACL rupture.

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Chapter 5

LOAD SHARING REMAINS UNCHANGED AFTER ACL INJURY

Introduction

The medial compartment bears the majority of the load at the knee during normal walking. Knee joint loads which were recorded using an instrumented total knee arthrosis during walking show that the medial compartment bears about 60% of the total load throughout stance phase [1]. Various computation models predict a similar result, with the medial compartment bearing 60-100% of the total load during stance [2-5].

After anterior cruciate ligament (ACL) rupture, altered muscle activity and movement patterns occur during gait. Because muscles are the major determinant of load the articular surface of the knee [5], there is a potential for altered loading after injury. Specifically, there is a potential for a shifting of load from one compartment to the other. Cartilage may fail to adapt to acute changes in its mechanical environment following injury. Thus, altered load sharing may play a role in the initiation and progression of joint degeneration after ACL injury.

The purpose of this study was to determine whether the percentage of load borne by the medial compartment was significantly altered in the injured limb of ACL-deficient noncopers (individuals with characteristic knee instability [6]).

Methods

Subjects

Thirty athletes with acute, unilateral ACL rupture (17 men, 13 women) participated in the study (mean \pm SD; age = 29.3 \pm 10.1 yrs, time from injury = 8.7 \pm 8.1 wks, BMI = 27.0 \pm 0.1 kg/m2, gait speed = 1.54 \pm 0.1 m/s). All patients were functionally classified as noncopers using the University of Delaware screening examination [6]. Inclusion criteria were: 13 to 55 years of age, regular pre-injury participation in level I or II activities [7] and complete ACL rupture within the past 7 months. Exclusion criteria were: bilateral knee involvement, presence of repairable meniscus tear, symptomatic grade III injury to other knee ligaments, full-thickness articular cartilage defect greater than 1 cm2, or presence of any other injuries which prevented completion of the screening exam. This work was approved by the University of Delaware Human Subjects Review Board, and all participants provided informed consent prior to testing.

Testing and EMG-Driven Modeling

Kinematics, kinetics and electromyography were obtained for each subject during standard gait analysis testing (as described in Chapter 4, p. 33-4). Muscle forces were estimated using an EMG-driven modeling approach, which is sensitive to individual muscular activation patterns (as described in Chapter 4, p. 34-6). Joint contact forces were calculated for the medial and lateral compartment, according to an algorithm described by Winby et al. [5] (as described in Chapter 4, p. 36-7).

Variables of Interest

Total tibiofemoral load equaled medial contact force plus lateral contact force. Medial and lateral compartment loads from three walking trials were averaged and summed to obtain total tibiofemoral load. The percentage of total load borne by the medial compartment at the point of peak tibiofemoral loading was calculated for each limb. A paired t-test was used to compare the percentage of total load borne by the medial compartment between limbs. Significance was set at 0.05.

Results

For both limbs, the medial compartment bore approximately 60% of the total load throughout stance (Fig 5.1). For the uninvolved limb, the medial compartment bore 62.9% (SD 7.9%) of the knee compressive load at the point of peak total load. For the involved limb, the medial compartment bore 65.8% (SD 7.5%) of the knee compressive load at the point of peak total load. There was no significant difference in medial/lateral load sharing between limbs (p=0.134).

Discussion

The purpose of this study was to determine whether the percentage of load borne by the medial compartment was significantly altered in the injured limb. We found that patients distributed load similarly between medial and lateral compartments in their injured limb when compared to the distribution in their uninjured limb and compared to load sharing data from an instrumented arthrosis.

Muscles contribute substantially to the loading of the articular surface, and a shift in load from one compartment to the other may be an important factor in the risk for degeneration after ACL injury. Because ACL deficiency is associated primarily with anterior-posterior knee instability and because walking is a sagittal plane motion, perhaps the lack of altered load sharing immediately after injury is not surprising.

The knee adduction moment serves as an estimate of the load distribution between the medial and lateral compartments and correlates with the severity and progression of knee OA [8]. Greater peak knee adduction moments have been identified in ACL-reconstructed knees (ranging approximately 1-10 years postsurgery) compared to matched, uninjured knees [9]. These higher moments suggest that load sharing may be altered in the years following ACL injury and reconstruction, with more load being placed on the medial compartment. Future work should assess load sharing in the years following ACL reconstruction and rehabilitation in order to determine whether increased medial load is associated with the onset of OA in these patients.

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Chapter 6

CONCLUSION

The **overall goal** of this work was to further characterize the noncoper neuromuscular strategy and to describe joint loading patterns acutely after ACL injury.

Aim 1 – Muscle Forces

The results from Aim 1 (described in Chapter 3) identify the neuromuscular asymmetries which drive the aberrant movement patterns observed after ACL injury. As expected, noncopers demonstrated a decreased peak knee moment for their injured limb, but they generated it with neither an isolated decrease in quadriceps force (quadriceps avoidance), nor an isolated increase in hamstring force (hamstring facilitation) at the point of peak knee moment. Instead, they exhibited decreased muscle force from both flexor and extensor groups.

The noncopers' strategy of decreased muscle force may be explained in part by muscle weakness which frequently accompanies ACL injury, or by apprehension, low confidence and fear of further injury. The lower muscle force in the injured limb of these patients affirms the existence of neuromuscular asymmetries following ACL injury. Noncopers may benefit from a rehabilitation program which restores muscle strength and confidence in the function of their injured limb.

Aim 2 – Joint Contact Forces

The results from Aim 2 (described in Chapters 4 and 5) identify the effect of the noncoper neuromuscular strategy on joint loading. Patients loaded their injured knees significantly less than their uninjured knees after ACL injury. This unloading occurred in both medial and lateral compartments; however the distribution of load between compartments was unchanged. If excessive loads are the mechanism for onset of osteoarthritis, these results indicate that they are not widely present early after injury. The joint unloading observed in these cause cartilage deconditioning, resulting in an increased potential for cartilage insult with the resumption of normal or excessive loading at a later time.

Future Direction

This work provides a basis from which to compare the effects of longitudinal changes in neuromuscular strategy and knee joint loading. Such future work will increase our understanding of the initiation and progression of knee OA after ACL injury.

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APPENDIX: HUMAN SUBJECTS REVIEW BOARD APPROVAL

IIUMAN	SUBJ	ECTS REVI University of B Newark, DE	EW BOA Delaware 19716	RD ACTION				
Protocol title-	Can Neuromuscular Training Alter Movement Patterns?							
Principal investigator(8):	Lynn Snyder-Mackler, Wendy Hurd, Thomas Buchanan, Kurt Manal & Michael Axe							
HSRP number:	HS 04-326							
Type of review:	П	Expedited	\boxtimes	Full Boatu				
The Human Subject respect to (i) the rights and be used to secure informed and has taken the following	s Revie i welfar consen ; action	w Board has rev te of the subjects t; and (3) the ris :	iewed the abo : (2) the appro- ks and potenti	ove-referenced protocol with opriateness of the methods to ial benefits of the investigation,				
Approval w	šchout, r	eservation						
L Approved as	revise	3						
E Disapproved	for res	sons noted below	×					
Approval date:	July	20, 2004						
Approvai period:	12 m	woths						
Expiration date:	July 19, 2005 (1 year from conveneal meeting date)							
Submittal date for continuing review;	May	19, 2005						
Changes in the prof Comments:	acol ton	ist be approved i	n advance by	the HSR5.				
Dr. Richard D. Holsten, A Chairman, Human Subject 210 Hudhhen Hall 302-831-2383, Jax: 302 83	ssociata Reviet 1 2828,	- Provost fer Res # Board , rheisten@udel.e	.D carch xtu	uto, 712164				

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