Frontal Plane Pelvic Drop in Runners: Causes and Clinical Implications

Evie Neff Burnet
Virginia Commonwealth University

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FRONTAL PLANE PELVIC DROP IN RUNNERS: CAUSES AND CLINICAL IMPLICATIONS

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University.

by

EVIE NEFF BURNET

Bachelor of Science, University of North Carolina at Chapel Hill, 2000
Doctor of Physical Therapy, Virginia Commonwealth University, 2003

Director: Peter E. Pidcoe, P.T., Ph.D.
Associate Professor, School of Physical Therapy

Virginia Commonwealth University
Richmond, Virginia

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List of Abbreviations

BW = body weight
COG = center of gravity
FFT = Fast Fourier Transform
FS = foot-strike
GM = gluteus medius muscle
ICC = intra-class correlation coefficient
LE = lower extremity
OGA = observational gait analysis
ΔPelvic Drop = difference between average ending and beginning pelvic drop
PSIS = posterior superior iliac spine
RE = running economy
ΔRE = difference between average ending and beginning RE
SEM = standard error of the mean
sEMG = surface electromyography
vGRF = vertical ground reaction force
VCO₂ = volume of carbon dioxide
VO₂ = volume of oxygen
Running is becoming an increasingly popular sport; however, runners have a high rate of injury and are therefore often treated in the orthopedic or sports medicine setting. One current focus of these patients’ treatment is gluteus medius muscle (GM) strengthening and gait retraining, with the goal of decreasing frontal plane pelvic drop. Unfortunately, there is a research void assessing the role of GM function on pelvic drop, and the effect of an increased pelvic drop on running performance. The specific aims of this research were to investigate a link between frontal plane pelvic drop and (1) isometric GM torque, (2) GM surface electromyography (sEMG) peak amplitude and
onset timing, and (3) GM fatigue; and (4) to study the relationship between frontal plane pelvic drop and increased metabolic energy demands. Subjects were recreational runners who ran an average of five or more miles per week. Data from an initial ten subjects were collected, followed by an additional eleven subjects tested for Specific Aims #1, 2, and 3. GM maximal isometric torque was obtained prior to the run. Subjects ran on a treadmill for thirty minutes while three-dimensional pelvic kinematics, GM sEMG, and metabolic data were collected. Pearson’s Correlations and scatter plots of the variables showed no relationship between GM maximal isometric strength, GM peak amplitude and onset timing, or GM fatigue rate and frontal plane pelvic drop. The change in pelvic drop also had no effect on the change in running economy (RE) from the start to end of the run. Clinicians should not employ a GM centered treatment approach when treating frontal plane pelvic instability in runners. Future research into additional core stabilizing muscles and their interactions could provide insight into which muscles should be the focus of treatment in runners with proximal instability. These studies should also include kinetic as well as lower extremity (LE) kinematic analysis of running gait to investigate the link between these variables, their relationship to muscle performance, as well as to running performance.
CHAPTER 1 – INTRODUCTION AND LITERATURE REVIEW

Running is becoming an increasingly popular fitness activity, with an estimated 37.8 million Americans participating at some level in 2005.¹ Meanwhile, the combination of running’s repetitive loading and the increasing number of runners contributes to running-related injuries. The overall incidence of injuries has been reported as 19.4% to 92.4% in long-distance runners, with an average of 49.6%.²⁻²⁰ The majority of musculoskeletal running injuries have been classified as overuse in nature, and can be traced to training errors, or anatomical or biomechanical factors.⁸, ²¹, ²² However, there is a research void linking altered running mechanics to overuse injuries. To begin to address this void, the overall aim of this research was to investigate one running gait deviation, frontal plane pelvic drop, and its relationship to gluteus medius muscle (GM) isometric strength and activity, as well as metabolic energy demands.

This introduction will provide: an overview of the running gait cycle, methods for running assessment, causes of gait deviations, and specific aims and hypotheses for this research.

RUNNING GAIT CYCLE

Distinct from walking, running consists of two float phases occurring at the beginning and end of swing phase. Running is therefore an alternation of single limb stance and nonsupport phases. As speed increases, less time is spent in the stance phase.

¹
Slocum and James\textsuperscript{23} were the first to describe the running gait cycle, which they broke up into two phases: support and forward recovery. The support phase is the period of single leg stance from initial contact until the foot leaves the ground to begin swing phase, and consists of foot-strike, mid-support, and takeoff. The forward recovery phase occurs during swing as the leg is advancing prior to foot-strike, and is comprised of follow-through, forward swing, and foot descent.

**Foot-strike**

Foot-strike (FS) begins when the foot hits the ground and continues until the foot becomes firmly fixed. At the point of FS, the phase of support is initiated, and the stance limb must absorb the body weight, maintain balance, and continue forward progression.\textsuperscript{23}

In the sagittal plane, the leg moves in a rearward direction at FS secondary to the concentric contraction of the gluteus maximus and hamstring muscles; eccentric quadriceps contraction controls this motion. Ground contact occurs slightly ahead of or below the center of gravity (COG).\textsuperscript{24-27} The pelvis is anteriorly rotated approximately 15°, the hip is flexed 30° to 40°, and the knee is flexed 30° to 35° at FS.\textsuperscript{23, 25, 28-30} Ankle position will depend on the runner’s style of foot contact (heel, midfoot, or forefoot).\textsuperscript{23, 25} Depending on the location of foot contact, the ground reaction force vector will vary. The gravitational line of weight, however, must fall through the stance foot and divide the body weight equally if balance is to be maintained.\textsuperscript{27}

In the frontal plane, the pelvis is obliquely aligned, with the stance side slightly elevated compared to the swing side, corresponding to hip adduction.\textsuperscript{28-30} Schache et al.\textsuperscript{30} found the average pelvic obliquity to be $2.3° \pm 1.2°$ elevated on the stance side at FS in a
group of twenty healthy, conditioned male runners running at 4.0 m/s. Pelvic obliquity, controlled by the GM and tensor fascia which eccentrically stabilize the hip joint, is theorized to aide in shock absorption and control descent of the COG.\textsuperscript{23, 25, 26}

Additionally, the base of support is decreased in running compared to walking. In the transverse plane, this will be apparent as foot strikes will occur along a line of progression, as compared to two to four inches apart as in walking.\textsuperscript{31} The hip will also be externally rotated corresponding to the slight external rotation of the pelvis in the transverse plane, which Schache et al.\textsuperscript{30} quantified as $3.9^\circ \pm 2.5^\circ$\textsuperscript{23, 28, 29}

\textbf{Mid-support}

The period of mid-support begins when the foot is fixed and continues until the heel begins to rise from the ground.\textsuperscript{23, 25} During this phase, momentum takes the body over the foot, while the stance leg supports the body and continues the body’s forward motion. In the sagittal plane, the COG is at its lowest point, as the stance extremity is in a flexed position in preparation for an extension thrust in the latter phase.\textsuperscript{25, 27} The quadriceps muscles continue their eccentric control of knee flexion.\textsuperscript{26} Meanwhile, the pelvis moves posteriorly to a position of minimal anterior tilt, followed by anterior movement.\textsuperscript{28-30}

Frontal plane pelvic motion begins with horizontal alignment that transitions toward downward obliquity of the swing leg, followed by a shift toward upward pelvic motion by the end of midsupport. This corresponds to a transition from eccentric to concentric contraction of the GM and tensor fascia.\textsuperscript{24, 26, 28, 29} The pelvic movement corresponds to hip position, which is slightly adducted in the frontal plane due to pelvic
sag on the swing leg in an effort to provide shock absorption, vertical height adjustment to clear the swing leg, and lateral balance; followed by hip abduction during late midstance.\textsuperscript{23,28} In the transverse plane, pelvic external rotation increases and reaches a maximum point of $7.2^\circ \pm 3.5^\circ$.\textsuperscript{28-30}

**Takeoff**

Takeoff begins when the heel begins to rise and continues until the toes leave the ground. At the end of takeoff, there is an extension of the stance leg to provide forward motion in an effort to maximally accelerate the body in preparation for a float phase. In the sagittal and transverse planes, the pelvis is at approximately $20^\circ$ of maximal anterior tilt, the hip extends and internally rotates, and the knee also extends.\textsuperscript{23,25,28-30} The leg passes over the metatarsal heads in the sagittal plane, allowing the heel to rise.\textsuperscript{23,25}

In the frontal plane, the pelvis reaches a point of maximum downward obliquity on the takeoff side, and the hip is in slight abduction.\textsuperscript{28-30} Schache et al.\textsuperscript{30} found the average downward pelvic obliquity to be $5.4^\circ \pm 2.6^\circ$ at takeoff. The pelvis also begins to internally rotate in the transverse plane during this period, such that it is neutral when the toes leave the ground.\textsuperscript{28-30}

**Follow-through**

Follow-through begins as the trail foot leaves the ground and continues until the leg finishes decelerating during rearward motion. At the same time the contralateral limb is undergoing foot descent. In the sagittal plane, the hip, knee, and ankle reach a point of greatest extension; while, the pelvis begins to posteriorly tilt.\textsuperscript{23,25,28,29} Flexion of the hip and knee, however, will occur near the end of follow-through.\textsuperscript{23,25,28}
Further hip abduction occurs to aid in clearance of the contralateral swing limb, and the pelvis begins to rise in the frontal plane until a slight lowering occurs corresponding to FS on the contralateral side. The pelvis also continues to internally rotate in the transverse plane.\textsuperscript{28-30} Deceleration occurs in a proximal to distal progression until a point of zero velocity at the foot is reached.\textsuperscript{23, 25}

**Forward Swing**

Forward swing begins as the thigh moves forward in the sagittal plane to a point of maximum hip flexion.\textsuperscript{23, 28} Hip flexion during this phase is eccentrically controlled via the hamstring muscle group.\textsuperscript{26} In the transverse plane, the pelvis continues to internally rotate until a point of maximum internal rotation of $6.7^\circ \pm 3.5^\circ$ is reached to lengthen the stride; while, in the frontal plane the pelvis on the ipsilateral side rises and the hip continues abduction.\textsuperscript{28-30} A proximal to distal forward motion occurs in the sagittal plane, angular momentum is created, and the knee is able to passively flex to clear the foot.

In the sagittal plane, the pelvis reverses from relative posterior tilt to anterior tilt, and knee flexion reaches its greatest point once the thigh has past the trunk’s vertical line.\textsuperscript{23, 25, 28, 29} Knee flexion also enables the COG to rise, until the hip nears full flexion.\textsuperscript{23, 25} During early forward swing, the body is in a double float, or airborne phase. Once the thigh is just behind the COG, the contralateral leg bears weight as FS occurs. The contralateral leg will continue to be in support phase until the thigh of the swing leg reaches its highest point. Then, takeoff will occur on the contralateral leg, and the body again enters a period of double float.\textsuperscript{23}
Foot Descent

The period of foot descent begins when the hip has reached a point of maximum hip flexion, and ends at the point of FS. In the sagittal plane, the thigh begins posterior movement allowing continued knee extension via quadriceps concentric contraction. The gluteus maximus and hamstrings increase eccentric muscle activity during foot descent to decelerate the forward motion of the swing thigh to zero velocity in preparation for FS. At this point, the knee is at 30° of flexion, and the tibia is perpendicular to the ground. As the knee extends, the foot reaches a point where it is furthest from the body. The swing leg begins to move posteriorly in preparation for FS, and the running cycle repeats.

In the frontal plane, the pelvis continues to rise and maximal upward pelvic obliquity is obtained in an effort to allow foot clearance of the swing limb while the hip and knee are extending, and the hip begins to adduct again in preparation for accurate FS. The pelvis also externally rotates in the transverse plane. This has been hypothesized to prepare the body for FS by: first decreasing the posterior vector of the ground reaction force (GRF), and second by decreasing the horizontal linear distance between the point of contact and the body’s COG. An externally rotated pelvis may therefore decrease horizontal braking forces and avoid deceleration.

RUNNING ASSESSMENT

Methods to assess running performance vary from observational clinical methods to computerized, gait labs. The purposes for the spectrum of performance assessment methods differ; however, the goal of using the tool is to improve running gait.
Observational Gait Analysis

Observational gait analysis (OGA) is cost efficient, fast, and easy to use as compared to computerized gait analysis techniques. To evaluate a patient, clinicians may employ OGA to determine impairments, establish goals, and/or assess progress. OGA can either occur in person (real-time) or via videotape (real-time, slow motion, or frame by frame). Depending on availability, a clinic may or may not have a video camera. A clinician without a camera is at a disadvantage, because the clinician is not able to replay or break the running down into slow motion or frame by frame in order to better analyze gait. Without this capability, the clinician could miss gait deviations that are not grossly abnormal or that occur quickly. The clinician in either instance could observe the runner on a treadmill or on land. The difficulty with treadmill running is that it may not replicate real-life conditions for the runner. The runner may feel uncomfortable on the treadmill, and therefore the observed running gait may not be representative. The advantage is that the runner is in one place, and so videotape analysis is more feasible. If the clinician were to watch the runner on land, the clinician might have difficulty detecting deviations as the runner moves away from the clinician.

The use of OGA is not new to research; however, assessments of observational analysis of running gait are. Therefore, information related to the reliability and validity of OGA primarily comes from research related to pediatrics, amputees, and neurologic patients. The interrater reliability of OGA studies has ranged from poor to good, possibly due to differences in design and severity of the gait deviation.33
One of the few studies assessing OGA reliability in the orthopedic population was conducted by Brunnekreef et al.\textsuperscript{33} Patients studied had a mild to severe gait deviation secondary to an orthopedic impairment. Walking gait deviations were assessed using videotaped OGA, and raters were allowed to view the videotape in slow motion, freeze frame, and as many times as needed. The authors reported moderate reliability of OGA. Decreased reliability could be attributed to inconsistencies or variability in the gait deviation. The highest reliability was obtained for easily observed items, rather than minute gait deviations. Additionally, in-experienced and experienced raters showed little difference in interrater reliability (intra-class correlation coefficient (ICC) 0.40 and 0.42, respectively) or intrarater reliability (ICC 0.57 and 0.63, respectively). Reliability did, however, improve in expert raters, with ICC of 0.54 for interrater and 0.72 for intrarater reliability.

Eastlack et al.\textsuperscript{34} studied the interrater reliability among fifty-four physical therapist using videotaped OGA to assess walking in three patients with stage 2 or 3 rheumatoid arthritis. An investigator was present during the rater’s assessments to slow and uniformly stop the video at predetermined points within the gait cycle. The investigators found that kappa coefficients were in the low to moderate range for the ten variables tested, and there was no significant difference in the interrater reliability based on the rater’s clinical experience. Decreased reliability was noted on kinematic measures of temporospatial assessment and those requiring judgement on “normal” ranges of motion. The most reliable results occurred during frontal plane assessments. The authors
point out that this contrasts with previous findings by Krebs et al.\textsuperscript{35} who found sagittal plane assessments to be more reliable.

Eastlack et al.\textsuperscript{34} state that although OGA is a quick and cost-efficient method to assess gait, findings of poor to moderate reliability bring into question the validity of the technique. If reliability of OGA is poor, findings would be inaccurate, and clinical decision-making could be jeopardized. Although the authors conclude that OGA has the potential to be a useful clinical assessment tool, the clinician should first recognize OGA’s reliability limitations and not base treatment regimes or clinical decisions solely on OGA results. The authors indicate that research is needed to identify normative values to be used in OGA, and suggest that education on gait analysis techniques might improve clinician expertise and therefore reliability.

**Laboratory Gait Analysis**

Kinematic, kinetic, and EMG variables are more commonly assessed in a laboratory than clinic setting, due to cost and therefore equipment restrictions. The advantage to these methods is that they supply more information than simple OGA. One can obtain computerized force and kinematic data, from which computer processing and statistics supply information on relations between planar motions and forces. Computer manipulation allows the researcher to view the runner in three dimensions, and therefore increases the ease of viewing kinematics in the sagittal, transverse, and frontal planes. Additionally kinematic, kinetic, and EMG methods permit the researcher to analyze multiple, quantitative variables, rather than subjective assessments made with OGA. The
reliability and accurate reflection of these measures is important if the measure will be used to guide clinical decisions.

Researchers often employ reflective markers or electromagnetic tracking systems for kinematic analysis. These markers are placed over anatomical reference points from which the computer creates an anatomical coordinate system to represent the subject. Problems arise, however, with the markers/sensors due to either movement, distance from the anatomical landmark, or poor placement. Reinschmidt et al.\textsuperscript{36} studied the effect of skin movement in three subjects on the validity of knee joint motion during running. Kinematic data collected from skin markers was compared to Hofmann bone pins, which were used as the gold standard. Error from the greater trochanter marker was twice as great as the other markers, for which error averaged 2.5 mm. The difference for knee flexion/extension averaged 20.8\%, abduction/adduction 70.4\%, and internal/external rotation 63.3\%. The authors concluded that the skin markers yielded a good representation of skeletal motion for flexion/extension; however, other knee motions may not be valid measures. Differences seen between skeletal and external motions were attributed to skin movement of the thigh and muscle activity. Therefore, the authors recommend that marker placement over a muscle belly should be avoided.

Using a larger sample size of forty normal adults, Kadaba et al.\textsuperscript{37} evaluated the reliability of kinematic, kinetic, and EMG data. Subjects’ walking gait was analyzed three times on three testing days that were at least one week apart as the subjects walked at a natural speed along a 6 m walkway. For kinematic, kinetic, and EMG variables, repeatability was greater during within-day versus between-day testing (ranges of
coefficient of multiple correlations for within-day versus between-day testing, respectively: kinematic 0.643 to 0.996, 0.240 to 0.983; kinetic 0.856 to 0.997, 0.817 to 0.995; and EMG 0.746 to 0.899, 0.661 to 0.875). Kinematic analysis of joint angle motion other than pelvic tilt showed excellent reliability (> 0.90) in the sagittal plane; however, transverse and frontal plane repeatability was lower (> 0.40 and 0.60, respectively). Kinetic measures of vGRF (vertical ground reaction force) and fore-aft shear were more repeatable (> 0.98) than mediolateral shear or torque about the center of pressure (> 0.89). Force moments at the knee showed lower repeatability (> 0.93) than moments at the hip and ankle (> 0.97), and sagittal plane moments were more reliable (> 0.93) than transverse or frontal plane variables (> 0.81 and 0.88, respectively). Yet, all kinetic variables for within-day and between-day testing showed good repeatability (> 0.80). EMG activity was more reliable for distal than proximal muscles (> 0.83 and 0.66, respectively), and EMG data was less repeatable compared to kinetic and kinematic measures. The authors attributed the decreased EMG between-day reliability to difficulty placing electrodes over the same volume of muscle on different days. Subtracting out a neutral position obtained from a static trial could reduce offsets in data between testing days. However, between-day repeatability remained lower than within-day. Based on the findings of adequate reliability (> 0.60), the authors concluded that for a normal population it may be feasible to make clinical decisions from a single gait analysis. This finding should not be generalized to injured populations or patients with gait deviations.

In a similar study, Mills et al. studied the repeatability of three-dimensional gait kinematics using an electromagnetic tracking system during treadmill walking in ten
subjects. Intra-trial repeatability was excellent (coefficient of multiple determination ranging from 0.819 to 0.995), with repeatability of sagittal plane motions the highest (≥ 0.967). These findings were similar to those reported by Kadaba et al.37, who found average coefficients of multiple determinations of the hip, knee, and ankle angles to be 0.983, compared to 0.985 found by Mills et al. Although frontal and transverse plane repeatability were lower, coefficients of multiple determinations averaged 0.919 and 0.922, respectively. These findings were superior to those reported by Kadaba et al. The results concurred with Kadaba et al.; repeatability was more affected by inter-day testing than inter-tester set-up.

Gait analysis of running presents specific factors than can affect reliability, such as individual differences in mechanics, speed of testing, and comfort level under testing conditions. Therefore, studies related to the reliability of running variables specifically are important when evaluating running gait analysis. In a study by Diss39, the reliability of kinematic and kinetic variables and the number of trials required to obtain accurate data were evaluated in five runners. Kinematic data based on the mean of five trials and kinetic data from the mean of ten trials showed good reliability (R > 0.93). Unfortunately, multiple trials are often time consuming in the clinic, and reliability would therefore decrease if fewer trials were analyzed. Three trials, which might be more obtainable, produced an 80% confidence level for kinematic and kinetic variables. Additionally, efforts to disguise or divert attention away from the force plate location improved reliability secondary to decreased alterations in running gait.
In a similar study, Ferber et al.\textsuperscript{40} compared the within and between-day reliability of discrete three-dimensional kinematic and kinetic variables in twenty uninjured runners. The investigators found that most of the variables maintained high reliability (> 0.80) for both within and between-day testing. Between-day reliability was consistently lower except for GRF’s. For between-day variables with an ICC of < 0.70, the corresponding within-day variables had ICC values > 0.87. The authors point out that between-day reliability remained lower despite attempts to control intertester reliability by employing one investigator to apply the markers. Between-day variables’ peak angular excursion and velocity ICC’s were higher than the corresponding peak angle ICC (using hip abduction as an example, 0.84 and 0.86 versus 0.69). The average difference in peak angle was 9.04° for all variables combined; while, the average difference in the respective angular excursion was 3.63°. This finding suggests that when assessing a runner’s gait over time, relative variables such as joint angular excursion and velocity are more reliable than absolute variables such as joint angle. In this study, sagittal plane ICC’s were more reliable (> 0.8) than those in either the frontal or transverse planes (> 0.5 and 0.6, respectively) for between-day testing only. GRF variables had high within and between-day reliability (≥ 0.88) and were found to be more reliable than either kinetic or kinematic variables. The authors conclude that although within-day testing was more reliable when looking at the variables as a whole, between-day testing could also produce similar reliability if variables consisted of GRF’s and relative kinematic variables.

Hunter et al.\textsuperscript{41}, similar to Diss\textsuperscript{39} and Ferber et al.\textsuperscript{40}, assessed the reliability of kinematic and kinetic variables during sprint running in twenty-eight males subjects. For
all variables, improvements in reliability were directly correlated to increases in the average of multiple trials, up to five in this study. Variables related to horizontal velocity of the COM yielded the highest reliability (ICC > 0.9). Whereas, variables calculated from vertical displacement of the COM produced the lowest reliability (ICC > 0.5). No matter the variable, the trend remained that increases in the number of trials analyzed increased reliability.

Based on the above findings there are several consistent conclusions which should be considered when assessing gait in a laboratory setting. First, the researcher should recognize that between-day reliability would likely be lower than within-day secondary to extraneous variables introduced by testing on multiple days. Second, when using reflective markers or electromagnetic sensors, skin movement and muscle contractions will change the position of the marker/sensor relative to its anatomical landmark. Additionally, attaching the markers/sensors to the same location each day is difficult, and will therefore introduce reliability issues. Third, a neutral, static data collection should be used to negate offset that occurs between days or trials. Lastly, if it is possible to run multiple trials, at least three will aide in increasing the reliability of measures.

**GAIT DEVIATIONS**

**Epidemiology**

The current debate in running literature centers on the epidemiology of running injuries and gait deviations. Does the runner develop a gait deviation that causes an injury, or does the runner become injured and compensates through a gait deviation? Does age, activity level, or gender impact running gait or the likelihood of injury?
Because of a lack of longitudinal gait analysis studies, there is not a conclusive answer to these questions.

**Age and Activity Level**

There is little information on the long-term changes in running gait associated with aging secondary to a lack of longitudinal studies from childhood to later in life. During adolescence there are many body changes both structural and hormonal. It is possible that these changes could predispose an adolescent to running gait deviations or overuse injuries, similar to tendencies in sports such as soccer and gymnastics. Studies do exist, however, comparing younger to older runners. Aging is associated with a decline in muscle force and coordination, and these changes in muscle function are associated with gait deviations. Deficits in mobility include gait velocity, step length, and range of motion of the lower extremity (LE). A linear decrease in running speed in adults correlates with aging up to eighty years-old, thereafter, speed progressively decreases.\(^{42, 43}\)

In a study assessing the age-related differences in 100 m sprint performance, Korhonen et al.\(^ {42}\) evaluated the sprinting of seventy subjects aged forty to eighty-nine years old. The authors found the decline in velocity to be exponential rather than linear; this difference was more apparent after sixty-five years of age. The velocity deficit was similar throughout the phases of running gait for both genders. Associated with a decline in velocity, stride length decreased and stance time increased linearly with increasing age for both males and females. Bus\(^ {43}\) found that these compensations in fifty-five to sixty-five year-old men kept peak impact force and loading rate similar to that in twenty to
thirty-five year-old male runners when running at a self-selected speed. When the older men ran at a controlled running speed of 3.3 m/s, higher impact loads were observed. Increased forces would suggest a decreased ability to absorb shock and a resulting increased stress on LE bones, joints, and soft tissues. Without age-related running gait compensatory techniques, the older runner in theory could be at higher risk of overuse injury and/or gait deviations.

Savelberg et al.44 studied the effects of running on counterbalancing gait performance changes in the inactive and aging populations. Forty male subjects comprised groups of young active, young inactive, old active, and old inactive adults. Active was defined as having run at least twice a week for more than two years. The researchers found that activity level did not affect walking gait velocity, and cadence was not significantly affected by age. When subjects were able to walk at a comfortable self-selected pace, the age-activity interaction remained; young active subjects walked faster than subjects in the other groups. In terms of kinematics, knee range of motion was inversely related to age. The support torque was similar between the young and active elderly. However, the inactive elderly subjects’ differences were due to work production. Inactive elderly subjects were found to generate less knee extension work than the active elderly. Additionally, the shift from an ankle to hip postural strategy in the active elderly allowed compensation for a decreased ankle torque, which maintained the overall support torque pattern similar to that in the young adults. This shift in increased hip torque production is not, however, affected by activity level. Running in the active elderly did not counterbalance the ankle to hip torque shift, rather running enlarged the shift.
In a similar study, Dorner et al. studied the effect of running on the age-related changes in gait symmetry of Sprague-Dawley rats. Following three months of training, the stride length increased and gait symmetry improved. Stance width, however, was unaffected. Running appeared to prevent age-related gait changes in rats. From these age and activity studies, it appears that running can prevent associated gait changes through compensations. Although the older population may decrease stride length and increase stance time to compensate for a decreased running velocity, overall torque generation remains relatively unchanged. Despite these compensations, one might hypothesize that a shift from an ankle to hip postural control strategy combined with a decrease in muscle force and control could lead to injuries and/or gait deviations in older or inactive runners. There are, however, no studies to suggest that increased age or inactivity may lead to a propensity toward running gait deviations or injury because of a decrease in muscle activity and coordination.

Gender

In a fifteen year prospective-longitudinal study, Rauh et al. tracked the injury rate among 199 high school cross country teams from twenty-three high schools during the athletes’ high school career (1,202 females and 2,031 males). The researchers found that injury rates were significantly higher for females for both initial and subsequent injuries. Additionally, injuries in females resulted in greater length of absence from running secondary to the injury, and the injury rate for out-of-season injuries was almost two times greater for females than males. Females also had a significantly higher injury
rate than males at the hip, shin, and foot. The authors did not attempt to link incidence to epidemiology.

Studies have found a correlation between decreased hip abduction strength, gender, and injury.\textsuperscript{42, 46} Ferber et al.\textsuperscript{47} studied gender differences between kinetic and kinematic data for twenty male and female recreational runners. In the frontal plane of stance phase, females had a significantly greater peak hip adduction angle, hip frontal plane negative work, and peak hip adduction velocity as compared to males. The authors hypothesized that the greater peak hip adduction angle and greater stance phase hip adduction in females could be attributed to structural differences. These findings may suggest that strength imbalances may be associated with or predispose an athlete to injury, injuries may lead to strength imbalances, or this cascade of events may differ between genders.

**Pathology**

There is a gap in research linking running gait deviations to overuse injuries. Theory exists in the field supporting the idea that abnormal gait mechanics could increase forces and lead to injury, or vice versa. One possibility for a gait deviation could be asymmetry caused by limb alignment. For instance, if a runner’s right leg was shorter he might exhibit right pelvic drop to compensate for the decreased leg length. One might hypothesize that the compensatory hip drop could increase impact forces on the right LE and predispose the runner to right LE injury. Lun et al.\textsuperscript{6}, however, found that static lower limb alignment did not differ between 153 injured and non-injured recreational runners. These findings were consistent with Wen et al.\textsuperscript{20}, who concluded that LE alignment
asymmetries did not appear to be a conclusive major risk factor for overuse injuries in 255 runners training for a marathon.

James et al.\textsuperscript{21} followed 180 patients with 232 injuries. Again, no single anatomic variation correlated with specific diagnoses. The authors could trace the majority of injuries to improper training. However, the authors also concluded that most injuries in distance runners could be resolved with sufficient rest, but may recur if the exact etiology was not identified and addressed. These studies fail to assess running gait, and therefore it is unknown whether there is a correlation between gait deviations and pathology. Therefore, longitudinal studies are needed to track runners and their running gait to test the theory of a link between gait deviations and injury.

**Frontal Plane Pelvic Drop**

As therapists and researchers expand their focus in injury prevention and treatment to proximal joint mechanics and core stability, the lumbo-pelvic hip complex and its role in closed kinetic chain activities have become a focus. Core stability could be defined as the lumbo-pelvic hip muscle strength and endurance yielding a coordinated activation of muscles and maintenance of alignment throughout the kinetic chain, thereby positioning the distal body segment in the most optimal position for a particular athletic task.\textsuperscript{48-51} When core instability exists, due to strength and/or endurance deficits, the body may not be optimally aligned. For example, frontal plane pelvic drop is a sign of core instability that could be identified as a weak link in the running kinetic chain that could explain some running deviations and injuries.
Pelvic drop occurs when the stance leg hip abductors are weak, leading to hip drop or a Trendelenburg sign on the ipsilateral side, such that there is a resulting downward obliquity of the opposite hip during its swing phase. Pelvic drop is named based on the stance leg (see Figure 1).

Electromyography has shown that a hip abduction moment is created by the GM, and to some extent the tensor fascia, during the stance phase of running. At FS, these muscles eccentrically contract to control hip adduction, and then concentrically contract from the support phase into propulsion to create hip abduction. Weakness in the hip musculature, especially the abductors, may impair efficient transference of forces, increase thigh adduction, and lead to pelvic drop.

**Frontal Plane Pelvic Drop and Gluteus Medius Muscle Performance**

One specific biomechanical factor that may be associated with frontal plane pelvic drop is the presence of GM weakness (Specific Aim #1). Research appears to
indicate a relationship between hip abduction weakness and injury. Fredericson et al.\textsuperscript{52} studied 24 distance runners (14 females, 10 males) with iliotibial band syndrome (ITBS) to assess differences in hip abduction strength as compared to 30 controls (14 females, 16 males). They found that runners with ITBS had weaker hip abductors on their injured side compared to their noninjured side and controls. After six weeks of physical therapy, hip abduction strength of the injured side was at least equal to that of the noninjured and control limbs, and 22 of the 24 athletes were pain free. Additionally, Leetun et al.\textsuperscript{51} found significant differences between hip abduction for 60 male and 90 female collegiate basketball and track athletes; males demonstrated increased strength and core stability. Athletes who were injured during the season showed statistically significant deficits in hip abduction strength.

Niemuth et al.\textsuperscript{53} also identified an association between hip abductor muscle group strength imbalances and LE overuse injuries in thirty recreational runners with a single leg overuse injury. Cichanowski et al.\textsuperscript{54} identified injured limb hip abductors to be significantly weaker (p = 0.003) than the noninjured side and the control group (p = 0.010) in a group of thirteen female collegiate athletes with unilateral patellofemoral pain syndrome. These findings may suggest that strength imbalances may be associated with or predispose an athlete to injury, injuries may lead to strength imbalances, or this cascade of events may differ between genders. Deficits in hip abduction strength could in theory correspond to pelvic drop on the contralateral side. Therefore, the presence of pelvic drop and/or hip abduction weakness could be a significant evaluative finding.
If, however, factors exist other than GM strength that impact frontal plane pelvic drop, and the treatment plan focuses solely on strengthening, the clinician may not reduce pelvic drop or decrease the risk of injury. Unfortunately, there is a research void assessing the underlying causes of frontal plane pelvic drop in runners. Factors such as GM activation pattern and changes in frontal plane pelvic drop have not been addressed in the running population (Specific Aim #2). Previous research has identified changes in GM EMG latency and amplitude in subjects with chronic conditions.\textsuperscript{55-58} Beckman et al.\textsuperscript{55} reported a decreased latency of GM muscle recruitment in subjects with hypermobile ankles as compared to a healthy control group. In subjects with unilateral hip osteoarthritis, Sims et al.\textsuperscript{56} found an increased GM muscle activation amplitude bilaterally as compared to the control group; while, hip abductor strength was not significantly different between the two groups. These findings indicate GM muscle dysfunction related to onset or amplitude, not necessarily strength. If there is a change in the muscle’s activation pattern in an injured runner, then addressing GM strength alone may not be sufficient to return the runner to premorbid status.

Additionally, GM muscle fatigue could contribute to frontal plane pelvic drop (Specific Aim #3). Previous research has shown that hip abduction peak torque and fatigability were not significantly correlated, suggesting that strength and fatigability should be evaluated separately.\textsuperscript{59} Meanwhile, Eggen et al.\textsuperscript{60} found that knee valgus movement increased following isometric hip abductor fatigue. Considering the stance phase of running is a closed kinetic chain activity, the LE might compensate for frontal
plane pelvic drop via increased genu valgum. These studies may point toward a relationship between frontal plane pelvic drop and GM muscle fatigue.

**Frontal Plane Pelvic Drop, Kinetics, and Running Performance**

Previous research has demonstrated a vGRF of 2 to 5 times body weight during running.\(^6^1,\,^6^2\) Altered gait mechanics, however, can create a mechanically disadvantaged system, and lead to an increased vGRF. Ferber et al.\(^6^3\) found that female runners with a history of LE stress fracture had a 36% greater vGRF (3.87 versus 2.48 times BW (body weight)) compared to controls. Without compensation, an increased frontal plane pelvic drop could create a mechanically unstable system, which could result in an increased vGRF. This scenario might not only increase the risk of injury, but also lead to a decreased running performance.

Previous research has shown a relationship between vGRF and metabolic cost during steady-state level running.\(^6^4,\,^6^5\) Running economy (RE), or the steady-state volume of oxygen (VO\(_2\)) consumed at a given running velocity, has been strongly correlated with distance running performance.\(^6^6-7^2\) Factors that increase the energy required while running will increase VO\(_2\), leading to a cascade of events that increase RE and hinder running performance.

Inefficient running gait mechanics, to include increased vGRF and vertical oscillation of the body’s center of mass, have been associated with an increased RE, secondary to increased energy costs.\(^7^3-7^5\) Heise and Martin\(^7^3\) found two GRF variables, the total vertical impulse and net vertical impulse, demonstrated statistically significant positive correlations (\(r = 0.62, r = 0.60\), respectively) with RE. Based on this
relationship, the authors concluded that 40% of RE variability could be physiologically attributed to greater overall muscle support required during ground contact. This relationship can be further substantiated by Abe et al., who identified a significant correlation between the eccentric to concentric ratio of the vastus lateralis muscle and metabolic energy costs in seven male novice distance runners during a ninety minute run.

As described previously, an increased frontal plane pelvic drop might in theory contribute to increased vGRF or muscle activity requirements. Therefore, research is needed to quantify the relationship frontal plane pelvic drop and RE (Specific Aim #4). If a direct relationship exists between frontal plane pelvic drop and RE, then a runner with increased frontal plane pelvic drop may reach a point where he is metabolically unable to perform at the same level as a healthy runner. A better understanding of this relationship is therefore needed when working with athletes in order to optimize their running performance.

**PURPOSE, SPECIFIC AIMS, AND HYPOTHESES**

To begin to fill this research void, the purpose of this investigation was to quantify the relationship between frontal plane pelvic drop in runners and isometric GM muscle strength (Specific Aim #1), GM muscle timing (Specific Aim #2), GM muscle fatigue (Specific Aim #3), and metabolic energy demands (Specific Aim #4) during running.

**Specific Aim #1**

To examine the relationship between isometric GM muscle torque and the magnitude of frontal plane pelvic drop seen during the stance phase of running.
**Hypothesis 1** - It is hypothesized that subjects with decreased isometric GM muscle strength will have more frontal plane pelvic drop of the ipsilateral limb during stance phase.

**Specific Aim #2**

To investigate whether GM muscle activation patterns are linked to frontal plane pelvic drop.

**Hypothesis 2** – There will be a delay in GM peak amplitude timing in runners with an increased frontal plane pelvic drop and the magnitude of the delay will be correlated with the amount of pelvic drop.

**Hypothesis 3** – There will be a delay in GM muscle activation following initial contact in runners with an increased frontal plane pelvic drop and the magnitude of this delay will be correlated with the amount of pelvic drop.

**Specific Aim #3**

To assess the relationship between GM muscle fatigue rates and the degree of frontal plane pelvic drop.

**Hypothesis 4** - It is hypothesized that runners presenting with increased frontal plane pelvic drop will show a higher rate of GM muscle fatigue while running.

**Specific Aim #4**

To study whether the degree of frontal plane pelvic drop is related to changes in metabolic energy demands.
Hypothesis 5 - Increased frontal plane pelvic drop will result in increased energy demands, as indicated by RE, to compensate for an inefficient running gait.
CHAPTER 2 - METHODS

SUBJECTS

Subjects were recruited by posted advertisement from the student body at Virginia Commonwealth University to establish a convenience sample. All subjects provided informed consent in accordance with the Virginia Commonwealth University institutional review board and completed a self-report running questionnaire regarding their general health and running (see Appendix 1). The questionnaire was used as a screening tool to establish whether the subject fit within the guidelines of the inclusion and exclusion criteria. Subjects were included if they ran five or more miles per week in order to obtain an active, recreational runner sample. Exclusion criteria included: a history of cardiopulmonary problems, LE neuromuscular impairment preventing the subject from running safely, or physician’s orders prohibiting running.

MEASURES

Gluteus Medius Muscle Isometric Torque

Subjects’ GM isometric force production was tested with a hand-held dynamometer using a method that was previously reported as reliable. This method positions the subject in side lying on a treatment table with a pillow placed between the knees to approximate 10° hip abduction for the leg to be tested. A strap was secured around the plinth table and subject just proximal to the iliac crest to stabilize the trunk. A
second strap secured the center of the hand-held dynamometer (Lafayette Instruments, Lafayette, IN) force pad just proximal to the lateral knee joint line. The dynamometer uses a load cell-based system to measure static forces from 0.0 to 199.9 kg with an accuracy of ± 0.5% of the full scale and a sensitivity of 0.1 kg.

With the subject in a relaxed position, the dynamometer was tarred and the baseline value recorded. The subject was then instructed to raise his leg upward with maximal effort and hold for 5 seconds. As the subject rested for 15 seconds, the maximum isometric force produced was recorded, and the dynamometer zeroed. This procedure was repeated for a total of 4 trials (1 practice and 3 trials). The subject was repositioned and the procedure repeated for the opposite leg.

**Gluteus Medius Muscle Activity**

With the subject positioned prone on a plinth table, rubbing alcohol was used to abrade the skin and reduce skin impedance over the GM prior to application of the sEMG (surface electromyography) electrodes (Ambu® Blue Sensor SP, Denmark). To collect bilateral GM activity, two 38 mm diameter sEMG electrodes were attached on each side parallel to the GM muscle fiber. The proximal electrode was approximately one-inch below the midpoint of the iliac crest, and the second electrode one inch distal from the center of the first electrode. The sEMG ground electrode was placed over the left fibular head. sEMG data was sampled at 1000 Hz. The sEMG unit (MyoSystem 1200™, Noraxon, Scottsdale, AZ) has a sensitivity of ±1 µV, a bandwidth of 10 to 500 Hz, and a common mode rejection ratio of 130 dB.
Kinematics

Kinematic three-dimensional data on pelvic motion were collected at a sampling rate of 60 Hz using a 6 degree-of-freedom electromagnetic kinematic tracking system (MotionMonitor™ version 7.0, Innovative Sports Training, Chicago, IL). The system has a spatial resolution of 0.5 mm of translation and 0.1° rotation. To reduce noise associated with the treadmill (Cateye EC-T220, Boulder, CO) motor, a Faraday shield made of copper mesh was fitted over the motor. The transmitter was placed on a wooden support that straddled the back of the treadmill to center the transmitter approximately 79 cm behind the subject and 84.5 cm above the treadmill. The transmitter was oriented with the subject facing the +x direction, +y to the subject’s right, and +z towards the ceiling. With the subject standing on the treadmill Tuff-Skin® was applied over bilateral PSIS (posterior superior iliac spine) followed by double-sided tape to hold the kinematic sensors (Polhemus Fastrak®, Colchester, VT). A harness belt further secured the PSIS markers, while a Velcro trunk harness tethered the sEMG and kinematic sensor leads to reduce noise secondary to motion artifact. Both the waist and trunk harnesses were further secured with Co-Flex® to prevent slipping. The difference between the right and left PSIS sensor readings served as the comparative standard for quantification of frontal plane pelvic motion. Using a difference signal removes the common signal associated with skin/sensor movement. Subtracting an initial baseline position also eliminates measurement errors associated with sensor placement.
**Metabolic Energy**

The rate of metabolic energy consumption (VO\(_2\) consumption and volume of carbon dioxide (VCO\(_2\)) production) was measured using ventilatory expired gas analysis (SensorMedics, Yorba Linda, CA). Ventilatory expired gas analysis was obtained using a metabolic cart (Vmax Spectra29, SensorMedics, Inc., Yorba Linda, CA). The metabolic measurement system and flow sensor were calibrated prior to each test. The oxygen and carbon dioxide sensors were calibrated using gases of known oxygen, nitrogen, and carbon dioxide concentrations prior to each session. Open spirometry methods were used. VO\(_2\) consumption and VCO\(_2\) production were measured as a function of time. The volume was then normalized to the subject’s BW for comparison. Ambient air with known oxygen concentration was inhaled through a mask that covered the subject’s mouth and nose, and measurement of the volume and percentage of O\(_2\) expired was used to calculate the amount of O\(_2\) consumption.

**Foot-strike**

Ground reaction force data were used to identify FS during the treadmill run. A single load cell was attached to the rear of the treadmill, in place of the treadmill’s right supporting foot. The signal from the load cell was amplified and low pass filtered at 500 Hz (Model SGA Interface, Inc., Scottsdale, AZ). The amplifier / load cell combination was found to be linear with a sensitivity of 0.11 kg/v. The load cell recorded both left and right foot contacts. To distinguish left and right FS, a trigger was depressed just prior to a right FS once during each 10-second data collection period. A positive voltage change in both the load cell and trigger data were associated with
increased loads on the treadmill secondary to FS or trigger depression, respectively. FS was delineated by the rising edge of this signal from a –5v baseline; whereas trigger onset corresponded to a positive voltage change from a 0v baseline.

Load cell, trigger, and sEMG data were collected via a 16 channel differential input 12 bit A/D card (Measurement Computing™, Norton, MA) (see Appendix 2).

**EXPERIMENTAL SET-UP**

With the consent form signed and the questionnaire completed, the subject was placed in supine and the distance from the greater trochanter to knee joint line measured. The subject’s isometric GM force was then tested. The subject was then positioned in prone on the plinth table and sEMG electrodes placed over the subject’s GM. The subject was then asked to stand on the treadmill while kinematic markers and sEMG cables were attached and secured in place. Once cables were tethered to the subject’s midback and suspended from the ceiling, the subject was asked to march in place to ensure the cables did not hinder movement (see Figure 2). After calibration of the metabolic measurement system and flow sensor, the mask was secured over the subject’s mouth and nose.
TREADMILL PROTOCOL

Following set-up and calibration of the sEMG, electromagnetic, and metabolic systems, subjects were asked to stand statically in anatomical position on the treadmill while baseline data were collected. Baseline sEMG and kinematic data were collected for 10 seconds, whereas, metabolic data were collected for two minutes. Subjects were then asked to begin a five-minute warm-up on the treadmill. Subjects were instructed to increase their running speed over the five-minute warm-up to reach a self-selected, comfortable, maintenance pace, up to 12.0 mph, which could be maintained for 30 minutes. A time zero sEMG and kinematic data collection were made following the five-minute warm-up. The subject continued to run at this pace for 30 minutes. Kinematic and sEMG data were collected synchronously in 10-second increments every two minutes; while metabolic data were collected continuously for the entire 30 minutes.
After the data collection period, the speed was reduced to 3.0 mph, and the subject was instructed to cool-down over a five-minute period.

**DATA PROCESSING AND ANALYSIS**

Kinematic and sEMG data processing were performed using custom software written in the MATLAB™ (MathWorks Inc, version 7.1) programming language (see Appendix 3). GM isometric torque and metabolic energy data analyses were performed in Microsoft Excel.

**Gluteus Medius Muscle Isometric Torque**

For **Specific Aim #1**, average maximum GM muscle isometric force was calculated for each leg using the recorded forces minus baseline. To normalize for BW, average maximum GM muscle isometric force in kilograms was converted to Newtons to obtain a unit of force using the following equation:

\[ \text{GMF} = ((BW \times 0.161) + F_{kg}) \times (9.807 \text{m/s}^2) \]

where:

- GM F = gluteus medius muscle isometric force (Newtons)
- BW = body weight (kilograms)
- F_{kg} = recorded gluteus medius muscle isometric force (kilograms).

Newtons were then converted to torque using the thigh length as the moment arm:

\[ \text{GM T} = \text{GM F} \times ma \]

where:

- GM T = gluteus medius muscle isometric torque (Nm)
GM F = gluteus medius muscle isometric force (Newtons)
ma = thigh length (meters).

**Foot-strike**

Trigger data were first analyzed to distinguish left and right FS. Trigger onset was defined as the frame where the voltage was greater than 1v. Meanwhile, FS onset occurred when voltage increased from a –5v baseline. Using the trigger onset frame as a marker, a right FS would correspond to the first FS following this trigger. Once this right FS was identified, foot contact before and after would be classified as alternating left and right FS. The frames for left and right FS were retained for later use.

**Kinematics**

Standing baseline x, y, and z data for the left and right sides were first averaged over the 10-seconds of data. Then, baseline pelvic angle was calculated using the averaged 3-dimensional data to find the angle between the left and right PSIS relative to a horizontal plane as shown in Figure 3.

![Figure 3](image-url)  
**Figure 3** – Calculation of the angle (θ) between a line and a plane.
Baseline pelvic angle was thus obtained using the following equation:

\[
BL = \left( \sin^{-1} \left( \frac{a}{\sqrt{((c)^2 + (b)^2 + (a)^2)}} \right) \right), \text{ or}
\]

\[
BL = (\sin^{-1} \left( \frac{(aveLz - aveRz)}{\sqrt{(aveLx - aveRx)^2 + (aveLy - aveRy)^2 + (aveLz - aveRz)^2}} \right))
\]

where:

BL = baseline, relative to the left side (degrees)
aveLz, aveLx, aveLy = average of the left side z, x, and y direction data (mm)
aveRz, aveRx, aveRy = average of the right side z, x, and y direction data (mm).

For each of the sixteen data collection points during the run, pelvic angle was then calculated relative to the left side for the entire 10-second data collection using the following equation:

\[
L \deg = (\sin^{-1} \left( \frac{(Lz - Rz)}{\sqrt{(Lx - Rx)^2 + (Ly - Ry)^2 + (Lz - Rz)^2}} \right)) - BL
\]

where:

Ldeg = left pelvic angle (degrees)
Lz, Lx, Ly = left side z, x, and y direction data (mm)
Rz, Rx, Ry = right side z, x, and y direction data (mm).

The pelvic kinematic data were then divided into gait cycles for the left side using the first left FS to one frame prior to the subsequent left FS. In order to represent the data relative to the gait cycle, the data were first fit using a cubic spline to produce a piecewise polynomial. The polynomial was then evaluated and expanded to 1000 points to
normalize the data to represent 100% of a gait cycle. Each gait cycle then had the same number of points to allow comparisons between gait cycles and point-by-point averaging.

The minimum and maximum pelvic angles occurring during foot contact were identified for each gait cycle (see Figure 4). The minimum (negative angle relative to horizontal) represented left frontal pelvic drop; while, the maximum (positive angle relative to horizontal) represented right frontal plane pelvic drop. Left and right pelvic drop for all gait cycles were ensemble averaged to obtain a left and right pelvic angle for the ten second data collection period (see Figure 5). The absolute value of the left pelvic angle was used in the analyses.

![Figure 4](image-url)  
**Figure 4** – Identification of left and right pelvic drop.
Figure 5 – Pelvic angle over a 10-second trial, showing 13 gait cycles in this case. Left and right pelvic drop for each gait cycle were ensemble averaged to obtain the average left and right pelvic drop for the 10-second period.

**Gluteus Medius Muscle Activity**

For **Specific Aim #2**, identification of GM peak EMG amplitude and onset activation were determined for each gait cycle. sEMG data were processed with a root mean squared time constant of 25ms. The load cell FS data served as the temporal event marker from which the time of GM peak EMG amplitude and onset were linked.

**Gluteus Medius Muscle Peak Amplitude**

For each gait cycle, left and right maximum amplitude of GM activity was identified for each gait cycle, and the mean peak amplitude calculated for the 10-second block. Average peak amplitude was then calculated across all blocks for later use in
determining threshold for left and right GM onset. Time of peak GM amplitude was found, and the median time of peak amplitude for each 10-second block calculated.

**Gluteus Medius Muscle Onset**

Fifty percent of the left and right average peak amplitude was calculated and used as the threshold from which left and right GM onset was identified, respectively. The cubic spline of bilateral sEMG data for each gait cycle was performed, followed by evaluation of the polynomial and expansion of the data to 1000 points to normalize to the data to 100% of the gait cycle. Thus, sEMG data could be compared across gait cycles and each onset demarcated as a percent of that cycle. The first frame where sEMG amplitude was greater than this threshold was marked as GM onset (see Figure 6). Since the GM has been shown to turn on at approximately 85% of the gait cycle (terminal swing) and continue to 25% (midstance), identification of GM onset began at 51% of the gait cycle (when the GM should be off) and continued to 100%. If onset was not located in this interval, identification then resumed to the start of the data at 0%, and continued to 50% if necessary. This process was repeated for each gait cycle, and the median GM onset identified for the left and right sides for the 10-second block.
Figure 6 – Identification of GM sEMG threshold and onset. One 10-second data collection is represented, with 13 gait cycles in this case.

To create a timeline (see Figure 7) for both the time of peak amplitude and onset of sEMG, median values found above were transformed. A value of zero represented FS, and corresponded to 0% of the gait cycle. If the peak amplitude and onset times were greater than 500, 1001 was subtracted, and the absolute value of this number taken. If the value was less than or equal to 500, it was simply made negative. Thereby, amplitudes and onset times occurring prior to foot contact (>500) were positive; while, times after foot contact (<=500) were negative. A tenth of these values then represented a percentage of the gait cycle.
Gluteus Medius Muscle Fatigue

For Specific Aim #3, sEMG raw data were filtered from 20 to 200 Hz. A Fast Fourier Transform (FFT) was performed for the left and right sides for the entire 10-second running block. The area under the FFT curve was calculated, and the median frequency corresponded to the frequency at 50% of this area. This process was repeated for each block, and the median frequency plotted against time. A first order regression line was fit to these data and the slopes recorded (see Figure 8).
Figure 8 – Example of one subject’s gluteus medius median frequency plotted versus time. Based on the first order regression line, the slope was found to be –2.07. A negative slope would indicate fatigue.

Metabolic Energy

For Specific Aim #4, VO₂ data were averaged between minutes 0 to 5 and 25 to 30 of the run, to obtain start and end VO₂. To normalize for the subject’s self-selected running speed, VO₂ was then converted to RE using the following equation:

\[
RE(\text{ml}^{-1} \text{kg}^{-1} \text{km}^{-1}) = \frac{VO_2(\text{ml}^{-1} \text{kg}^{-1} \text{min}^{-1})}{\text{speed}(\text{km} \text{hr}^{-1})} / 60(\text{min} \text{hr}^{-1})
\]

STATISTICAL ANALYSIS

Average pelvic drop for each block were averaged over the 30-minute run for use in statistical analyses for Specific Aim #1 and 3. Average pelvic drop between minutes 0 to 4 and 26 to 30 were calculated and a difference obtained for use with the metabolic energy data for Specific Aim #4. Analyses were performed using SPSS® version 14.0.
For all tests, $\alpha = 0.05$ was used as the significance level. For the purpose of evaluating Specific Aims #1, 2, and 3, a Pearson’s Correlation was used to analyze the relationship between GM torque production, muscle onset, peak amplitude timing, and fatigue; and the degree of frontal plane pelvic drop. Here, frontal plane pelvic drop was the dependent variable; while, GM torque production, muscle onset, peak amplitude timing, and fatigue were the independent variables. For Specific Aim #4, a Pearson’s Correlation was run to assess the relationship between $\Delta$RE and $\Delta$pelvic drop. $\Delta$RE was the dependent variable, and $\Delta$pelvic drop the independent variable. Following analysis of the first ten subjects, a power analysis was conducted using nQuery Advisor® 6.0.
CHAPTER 3 - RESULTS

Data from the first 10 subjects (5 males, 5 females) (mean 25.9 years, 172.97 cm height, 70.08 kg weight, 23.42 miles run per week, see Appendix 4) served as pilot data. Pearson’s Correlations (R) between the average pelvic drop of the left (Table 1) and right (Table 2) sides and the independent variables, as well as resulting effect size based on 80% power for **Specific Aims #1, 2, and 3** are summarized below.

Table 1 – Summary Statistics for Left Pelvic Drop and the Independent Variables.

<table>
<thead>
<tr>
<th></th>
<th>Torque (Aim #1)</th>
<th>Time of Peak (Aim #2)</th>
<th>Time of Onset (Aim #2)</th>
<th>Fatigue (Aim #3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation (R)</td>
<td>-0.620</td>
<td>-0.017</td>
<td>-0.314</td>
<td>0.583</td>
</tr>
<tr>
<td>p-value</td>
<td>0.056</td>
<td>0.962</td>
<td>0.377</td>
<td>0.077</td>
</tr>
<tr>
<td>Subjects needed for 80% power</td>
<td>18</td>
<td>27157</td>
<td>78</td>
<td>21</td>
</tr>
</tbody>
</table>

Table 2 – Summary Statistics for Right Pelvic Drop and the Independent Variables.

<table>
<thead>
<tr>
<th></th>
<th>Torque (Aim #1)</th>
<th>Time of Peak (Aim #2)</th>
<th>Time of Onset (Aim #2)</th>
<th>Fatigue (Aim #3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation (R)</td>
<td>-0.210</td>
<td>-0.669</td>
<td>-0.369</td>
<td>-0.077</td>
</tr>
<tr>
<td>p-value</td>
<td>0.561</td>
<td>0.034</td>
<td>0.294</td>
<td>0.833</td>
</tr>
<tr>
<td>Subjects needed for 80% power</td>
<td>176</td>
<td>15</td>
<td>56</td>
<td>72</td>
</tr>
</tbody>
</table>
Pearson’s Correlation for **Specific Aim #4**, the relationship between $\Delta \text{RE}$ and $\Delta \text{frontal plane pelvic drop}$, yielded an $R = 0.327$, $p = 0.356$, with a calculated sample size of 72 to achieve 80% power.

Based on the power analyses, 11 additional subjects (4 males, 7 females) were recruited for Specific Aims #1, 2, and 3 (mean age 24.55 years, 173.76 cm height, 71.13 kg weight, and 19.82 miles run per week). In these additional subjects, metabolic data were not collected, only kinematic, GM torque, and GM sEMG data were gathered. Demographics and history of running-related injuries in the past 6 months for the 21 subjects are summarized in Appendix 4. Mean demographics and ranges for the 21 subjects were: age 25.19 years (19 - 34 years), height 173.39 cm (152.4 - 193.04 cm), weight 70.63 kg (52.16 - 102.06 kg), and 21.53 average miles run per week (5 - 50 miles). None of the subjects had an injury causing a decrement in running performance at the time of data collection. A dependent t–test showed there was no significant difference between the first and last group of subjects for age ($p = 0.600$), height ($p = 0.889$), weight ($p = 0.842$), average number of miles run per week ($p = 0.212$), or self-selected running speed during data collection ($p = 0.288$).

**KINEMATICS**

Left and right side pelvic drop angles for each 2-minute data collection period over the 30 minute run, along with the resulting average and standard deviations are presented in Appendix 5. An ICC was performed to assess the test/retest reliability of the pelvic drop measure in five subjects and found to be 0.80, demonstrating good reliability. The standard error of the mean (SEM) was $0.36^\circ$. Average pelvic angle and standard
deviations across all subjects (n = 21) are depicted in Figures 9 and 10 for the left and right sides, respectively.

![Graph of Left Side Pelvic Drop](image)

**Figure 9** – Left side average maximum frontal plane pelvic drop across the 30-minute run. Error bars indicate standard deviations. An increasing angle of pelvic drop from minute 0 to 30 implies pelvic drop increased.

For the left side, the average frontal plane pelvic drop was 10.94° ± 1.67°. When looking at the left side average pelvic drop depicted in Figure 9, the observed trend was one of slight increased pelvic drop, from 9.53° ± 3.26° at minute zero to 11.87° ± 4.39° at minute 30. From the start and end means, effect size was calculated as Cohen’s d = 0.61.
On the right side, the average pelvic drop was $7.57^\circ \pm 1.18^\circ$. The overall trend of the right side demonstrates a fairly constant average pelvic drop across the 30 minute run (see Figure 10). The average starting pelvic drop was $7.48^\circ \pm 2.83^\circ$, and the average pelvic drop at minute 30 was $8.07^\circ \pm 3.56^\circ$. Using the start and end pelvic drop means,
the effect size was calculated as Cohen’s $d = 0.18$. When comparing minute 30 means for the left and right sides, the effect size was Cohen’s $d = 0.95$.

Standard deviations and SEM were calculated for subjects 1-10 and 11-21 for both the left and right sides (see Table 3). These values demonstrated small variations in the pelvic drop measure, with little variation due to randomness. A dependent t-test verified no statistically significant difference in pelvic drop between the first ten and last eleven subjects ($p = 0.101$, left and $p = 0.792$, right). The 95% confidence interval of the difference was between -5.48 and 0.58 (left), and -2.51 and 3.20 (right). Data from the first ten and last eleven subjects could therefore be combined.

Table 3 – Summary of standard deviations and SEM for pelvic drop data.

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>Standard Deviation</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left 1-10</td>
<td>3.65</td>
<td>1.15</td>
</tr>
<tr>
<td>Left 11-21</td>
<td>3.14</td>
<td>0.99</td>
</tr>
<tr>
<td>Right 1-10</td>
<td>2.77</td>
<td>0.88</td>
</tr>
<tr>
<td>Right 11-21</td>
<td>3.65</td>
<td>1.15</td>
</tr>
</tbody>
</table>

GLUTEUS MEDIUS MUSCLE ISOMETRIC TORQUE (Specific Aim #1)

Appendix 6 contains a summary table of GM average maximum isometric force and resulting torque data for the left and right sides of all subjects. To assess the reliability of the GM isometric force measurement, an ICC was performed on the three repetitions for each leg in the 21 subjects and shown to be 0.90, demonstrating good reliability. Figures 11 and 12 depict the relationship between each subject’s average
Frontal plane pelvic drop and average maximum static torque for the left and right sides, respectively.

**Figure 11** – Left side average maximum frontal plane pelvic drop versus gluteus medius muscle average maximum isometric torque.

The scatter plot of left side average frontal plane pelvic drop versus average isometric torque indicates a random relationship between these variables in this sample (Figure 11). In turn, the Pearson’s Correlation for the left side was found to be
$R = -0.212 \ (p = 0.356)$, indicating a poor negative correlation between frontal plane pelvic drop and isometric torque for the left side.

**Figure 12** – Right side average maximum frontal plane pelvic drop versus gluteus medius muscle average maximum isometric torque.

The right side scatter plot demonstrating the relationship between average frontal plane pelvic drop and average maximum isometric torque again shows a fairly random
association between these two variables (Figure 12). The right side Pearson’s Correlation was poor, R = 0.022 (p = 0.925).

The relationship between frontal plane pelvic drop for each data collection period and torque was also investigated. Pearson’s Correlations showed no significant correlation on either the left or right side between frontal plane pelvic drop and torque for any of the data collection periods during the 30-minute run.

**GLUTEUS MEDIUS MUSCLE PEAK AMPLITUDE TIMING (Specific Aim #2)**

Median left and right side time of GM sEMG peak amplitude timing data and standard deviations for each 2-minute increment of the 30 minute run for all subjects, as well as each subject’s average sEMG peak timing, are presented in Appendix 7. The Pearson’s Correlations between frontal plane pelvic drop and timing of GM peak amplitude for the left (R = -0.039, p = 0.479) and right (R = 0.029, p = 0.601) sides were both poor. Figures 13 and 14 depict the relationship between these variables for the left and right sides, respectively. A value of zero GM peak amplitude timing on the x axes would indicate that peak amplitude occurred at foot contact (0% gait cycle); whereas, positive values occurred prior to FS and negative values after FS. A value of 10 would occur during terminal swing (10% of the gait cycle prior to FS).
Figure 13 – Left side average maximum frontal plane pelvic drop versus gluteus medius peak amplitude timing. Average data for every 10-second data collection period for each subject is represented. 0% gait cycle represents foot contact, while positive values occur prior to FS and negative values after FS.

As indicated in Figure 13, left side GM peak amplitude occurred between 48.30% and −29.60%, with an average of 6.52% ± 12.33%. Peak amplitude therefore occurred sometime between left initial swing and left midstance, with the majority of cases clustered between left terminal swing and foot contact.
Figure 14 – Right side average maximum frontal plane pelvic drop versus gluteus medius peak amplitude timing. Average data for every 10-second data collection period for each subject is represented. 0% gait cycle represents foot contact, while positive values occur prior to FS and negative values after FS.

Figure 14 shows that right side GM peak amplitude occurred between 44.60% and −47.90% of the gait cycle, with an average time of peak at 1.82% ± 7.47% of the gait cycle. Similar to the left side, time of peak amplitude was clustered just before the time
of right FS. There were outliers, however, around the time of right initial and midswing, as well as right midstance.

To assess for changes across the 30-minute run, correlations were run for each of the data collection periods between frontal plane pelvic drop and GM peak timing. Pearson’s Correlations revealed no significant correlations for the left side. Significant correlations were seen on the right at minutes 6 and 20. At minute 6, however, one subject’s GM peak timing occurred much earlier than the rest of the group (46.3% versus a mean of 3.89% of the gait cycle for the remaining 20 subjects). When this subject was removed from the analysis, the correlation was no longer significant (R = -0.246, p = 0.296). The scatter plot for minute 20 revealed a random relationship between right pelvic drop and GM peak timing. Therefore, there was no relationship for either the left or right sides between pelvic drop and GM peak timing as a function of time.

**GLUTEUS MEDIUS MUSCLE ONSET (Specific Aim #2)**

Appendix 8 contains summary tables for each subject’s median GM onset timing across the 30 minute run, as well as standard deviations. Pearson’s Correlations between frontal plane pelvic drop and GM onset were R = -0.173 (p = 0.001) and R = -0.057 (p = 0.296) for the left and right sides, respectively. In both cases the correlations were poor. Left and right scatter plots for the relationship between these variables are presented in Figures 15 and 16. Interpretation of GM onset timing is the same as described above for GM peak amplitude timing; positive values occur prior to foot contact with zero representing FS.
As seen in Figure 15, there was considerable variability in the timing of GM onset, with a range of 49.7% to 4.75%, representing the early stages of left swing phase to just prior to left FS. Timing of left side GM onset averaged 21.62% ± 15.74% of the gait cycle prior to foot contact. This would be during left terminal swing phase.
Figure 16 – Right side average maximum frontal plane pelvic drop versus gluteus medius muscle onset timing. Average data for every 10-second data collection period for each subject is represented. 0% gait cycle represents foot contact, while positive values occur during the swing phase prior to right FS.

Right side GM onset timing ranges were similar to the left side, with a range from 49.7% to 0.6%, representing gait cycle times from early right swing phase to just prior to right FS. The average right side GM onset was 18.53% ± 17.13%, corresponding to right terminal swing phase.
The effect of time during the 30 minute run on the relationship between frontal plane pelvic drop and GM onset was investigated. Pearson’s Correlations for the left and right sides for each of the data collection periods showed no significant correlations between these variables at any time point for either side.

**GLUTEUS MEDIUS MUSCLE FATIGUE (Specific Aim #3)**

Appendix 9 contains tables summarizing GM fatigue rates for each subject during the 30 minute run. Pearson’s Correlations between frontal plane pelvic drop and GM fatigue rate yielded poor correlations of \( R = 0.030 \) (\( p = 0.896 \)) for the left and \( R = -0.001 \) (\( p = 0.996 \)) for the right. These relationships are presented in Figures 17 and 18 as scatter plots for the left and right sides, respectively.

Although not included within Specific Aim #3, the relationship between the rate of pelvic drop and the rate of GM fatigue was investigated (see Figures 19 and 20, respectively). For the left side, the Pearson’s Correlation was \( R = -0.123 \), \( p = 0.594 \); while, the right side was \( R = 0.103 \), \( p = 0.657 \).
Figure 17 – Left side average maximum frontal plane pelvic drop versus gluteus medius fatigue rate. Each fatigue rate represents a subject’s slope of median frequency across the 30-minute run. Negative values (to the left of the dashed line) would be indicative of fatigue.

As shown in Figure 17, left GM fatigue rates clustered around zero, with the exception of one outlier. The average GM fatigue rate was -0.015 ± 0.55, with a range of 0.61 to -2.07. Based on the data presented in Appendix 9 the subjects’ average median frequency at minute zero was 82.60 Hz ± 18.79 Hz, with a range from 45.70 Hz to 114.80 Hz. The average median frequency at 30 minutes was 82.69 Hz ± 21.06 Hz, with a range from 46.50 Hz to 113.90 Hz.
Figure 18 – Right side average maximum frontal plane pelvic drop versus gluteus medius fatigue rate. Each fatigue rate represents a subject’s slope of median frequency across the 30-minute run. Negative values would be indicative of fatigue.

Right side GM fatigue rates ranged from -1.64 to 1.48 with an average of 0.22 ± 0.68. The majority of subjects’ right side GM fatigue rates clustered between -1 to 1, as shown in Figure 18. The average median frequency at minute zero was 75.27 Hz ± 25.78 Hz, with a range from 29.40 Hz to 116.40 Hz. At minute 30 the average median frequency was 82.82 Hz ± 21.71 Hz, ranging from 37.50 Hz to 112.60 Hz.
Figure 19 – Left side rate of maximum frontal plane pelvic drop versus gluteus medius fatigue rate.

As shown in Figure 19 and as indicated by the poor Pearson’s Correlation, GM fatigue rate was not correlated with the rate of pelvic drop. In the presence of fatigue (rate of GM fatigue < 0), subjects did not exhibit an increased rate of pelvic drop as would be hypothesized.
As shown in Figure 20, few subjects experience GM fatigue (rate of fatigue < 0), and those who did experience fatigue did not have an increased rate of pelvic drop as would be expected. In those subjects who did not have GM fatigue (rate of fatigue > 0), five experience an increased rate of pelvic drop, which was counterintuitive.
METABOLIC ENERGY (Specific Aim #4)

Table 4 summaries the average start (minute 0 through 5) and end (minute 25 through 30) VO₂, trial running speed, and resulting RE and ΔRE for the first 10 subjects.

**Table 4** – Summary of metabolic energy data. Start data represents an average of minutes 0 to 5; while, end data represents the average of minutes 25 to 30. ΔRE was calculated as the difference between RE end and RE start.

<table>
<thead>
<tr>
<th>Subject</th>
<th>VO₂ Start (ml/kg/min)</th>
<th>VO₂ End (ml/kg/min)</th>
<th>Speed (km/h)</th>
<th>RE Start (ml/kg/km)</th>
<th>RE End (ml/kg/km)</th>
<th>ΔRE (ml/kg/km)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>32.09</td>
<td>31.91</td>
<td>10.78</td>
<td>0.050</td>
<td>0.049</td>
<td>-0.001</td>
</tr>
<tr>
<td>2</td>
<td>32.97</td>
<td>36.65</td>
<td>10.46</td>
<td>0.053</td>
<td>0.058</td>
<td>0.005</td>
</tr>
<tr>
<td>3</td>
<td>37.11</td>
<td>38.30</td>
<td>10.78</td>
<td>0.057</td>
<td>0.059</td>
<td>0.002</td>
</tr>
<tr>
<td>4</td>
<td>38.81</td>
<td>42.48</td>
<td>10.3</td>
<td>0.063</td>
<td>0.069</td>
<td>0.006</td>
</tr>
<tr>
<td>5</td>
<td>35.04</td>
<td>35.95</td>
<td>10.14</td>
<td>0.058</td>
<td>0.059</td>
<td>0.001</td>
</tr>
<tr>
<td>6</td>
<td>32.78</td>
<td>43.91</td>
<td>10.46</td>
<td>0.052</td>
<td>0.070</td>
<td>0.018</td>
</tr>
<tr>
<td>7</td>
<td>42.59</td>
<td>46.79</td>
<td>11.59</td>
<td>0.061</td>
<td>0.067</td>
<td>0.006</td>
</tr>
<tr>
<td>8</td>
<td>41.57</td>
<td>44.13</td>
<td>12.07</td>
<td>0.057</td>
<td>0.061</td>
<td>0.004</td>
</tr>
<tr>
<td>9</td>
<td>36.70</td>
<td>27.46</td>
<td>12.87</td>
<td>0.048</td>
<td>0.036</td>
<td>-0.012</td>
</tr>
<tr>
<td>10</td>
<td>49.99</td>
<td>52.30</td>
<td>12.07</td>
<td>0.069</td>
<td>0.072</td>
<td>0.003</td>
</tr>
</tbody>
</table>

The Pearson’s Correlation for the relationship between ΔRE and Δfrontal plane pelvic drop was R = 0.327, p = 0.356, suggesting there was a poor association between these two variables as shown in Figure 21. Based on this correlation, the calculated sample size needed to attain 80% power was 72. Therefore, metabolic data collection was not continued on the last eleven subjects, as described previously.
Figure 21 – Change in running economy versus change in average maximum frontal plane pelvic drop. The difference between the averages of minutes 25 to 30 (end) and minutes 0 to 5 (start) for RE and pelvic drop were calculated to determine change over the 30 minute trial.
CHAPTER 4 - DISCUSSION

The purpose of this investigation was to quantify the relationship between frontal plane pelvic drop in runners and isometric GM muscle strength (Specific Aim #1), GM muscle timing (Specific Aim #2), GM muscle fatigue (Specific Aim #3), and metabolic energy demands (Specific Aim #4). A better understanding of the connection between frontal plane pelvic drop and these variables would provide insight into the treatment of running-related injuries and the optimization of running performance.

FRONTAL PLANE PELVIC DROP

The test/retest reliability of the frontal plane pelvic drop measure demonstrated good reliability. The SEM calculated from the test-retest trials showed that differences in this measure greater than 0.36° could be attributed to factors other than random error. Additionally, standard deviations and SEM presented in Table 3 demonstrated that this kinematic measure had little variation accounted for by random error. Average standard deviations for each 10-second data collection across all subjects were small for both the left (1.67°) and right (1.18°) sides, indicating small intra-subject/intra-trial variance. The data presented in Figures 9 and 10 show a slight increased in average maximum pelvic drop for the left side, while the right side remained fairly stable throughout the 30-minute run. This is further supported by the effect size of 0.61 for the left side, indicating a detectable change from minute 0 to minute 30; while, the right side had an effect size of
0.18 that would be considered small and difficult to detect. Additionally, a comparison of minute 30 pelvic drop means for the left and right side resulted in an effect size of 0.95, indicating a detectable difference between the two sides at minute 30.

Although, previous studies have not investigated frontal plane pelvic motion over extended periods of running, Schache et al.\textsuperscript{82} did study pelvic motion over 5-second increments during treadmill running. Schache et al. reported the average pelvic drop over three 5-second data collections in 10 subjects (9 males, 1 female) who ran at a speed consistent with their previously determined self-selected overground running speeds (8.9 mph average) to be $7.6^\circ \pm 1.6^\circ$ (range $5.9^\circ$ to $10.6^\circ$) on the left and $6.9^\circ \pm 2.6^\circ$ (range $2.9^\circ$ to $10.6^\circ$) on the right. Average maximum pelvic drop values in the current study were consistent with those reported by Schache et al.

\textbf{Gluteus Medius Muscle Performance}

\textit{Gluteus Medius Muscle Isometric Torque (Specific Aim #1)}

The purpose of \textbf{Specific Aim #1} was to examine the relationship between isometric GM muscle torque and the magnitude of frontal plane pelvic drop seen during the stance phase of running. It was hypothesized that subjects with decreased isometric GM muscle strength would have more frontal plane pelvic drop. However, a random relationship and poor correlations were demonstrated between the average maximum pelvic drop and GM average maximum isometric torque, as shown in Figures 10 and 11. Thus, the hypothesis was not supported.

The use of hand-held dynamometry and a make test have limitations, to include tester strength and subject participation. The use of a strap to secure the dynamometer
eliminated the effect of tester strength, which has previously been shown to be a limitation of hand-held dynamometry. A practice trial and recovery time between trials avoided error due to subject effort. Additionally, the tester used consistent directions during testing to limit the potential influence of verbal feedback on subject motivation. The conversion of force measurements to torque allowed comparison across subjects by normalizing for BW and thigh length. Lastly, only one tester performed the measurement and demonstrated good intratester, intrasession reliability (ICC = 0.90).

Prospective, case-control, and case-series studies have established a link between hip abduction isometric strength deficits and LE injuries. Additionally, Ferber et al. demonstrated an association between increased vGRF and LE stress fractures in female runners. Although both decreased hip abduction isometric strength and increased vGRF are linked to LE injuries, the relationship between decreased hip abduction static strength, increased vGRF, and/or frontal plane pelvic drop has not been established.

This research aimed to investigate the link between GM isometric strength and frontal plan pelvic drop; however, it showed that static GM strength was a poor predictor of frontal plane pelvic drop. One question should therefore be posed: Is a static measure of GM strength appropriate to relate to dynamic measures?

Based on the research findings, one should question whether a dynamic rather than static measure of GM strength would be more appropriate. Clinically, qualitative observations during running gait analysis are typically linked to quantitative static strength assessments secondary to a lack of costly evaluative equipment or time. These
findings suggest this strategy is not appropriate for hip abduction. Future research is therefore needed to identify dynamic strength measures that would better predict biomechanical components of running gait.

**Gluteus Medius Muscle Activation Patterns (Specific Aim #2)** The purpose of **Specific Aim #2** was to investigate whether GM muscle activation patterns were linked to frontal plane pelvic drop. It was hypothesized that there would be a correlation between increased pelvic drop and a delay in peak amplitude of GM muscle activation. As shown in Figures 12 and 13, bilateral GM peak amplitude timing tended to cluster between terminal swing and initial contact of the ipsilateral LE, regardless of the degree of pelvic drop. Thus, the time of peak amplitude did not influence frontal plane pelvic drop, and the correlation between these variables was poor bilaterally.

The finding that the time of peak amplitude clustered around the time of FS is consistent with that reported by Cappellini et al. They showed that GM muscle activity ramped up during terminal swing, peaked around the time of FS, began a return to baseline just after FS, and reached baseline at approximately mid stance. They displayed this pattern for running at 5, 7, 9, and 12 km/h. Although GM intensity increased with increasing running speed, at each running speed the GM pattern of activity and time of peak onset persisted.

It was also hypothesized that there would be a delay in GM muscle activation following initial contact in runners with an increased frontal plane pelvic drop and the magnitude of this delay would be correlated with the amount of pelvic drop. For both the left and right sides GM activation occurred between ipsilateral initial swing and terminal
swing, as shown in Figures 14 and 15. There was no instance of activation following FS during the ipsilateral stance phase. Bilaterally, timing of GM onset was variable during the swing phase. The right side Pearson’s Correlation showed a significantly small relationship between frontal plane pelvic drop and timing of GM onset; however, the association was random as depicted in Figure 16. Therefore, for both the left and right sides there was no relationship demonstrated between timing of GM activation and frontal plane pelvic drop. The hypothesis was therefore rejected.
Figure 22 – A comparison of gluteus medius muscle activity during the gait cycle, as shown by Mann et al.\textsuperscript{26} and the current research. Positive values occur prior to foot contact, and negative values occur after foot contact.

As seen in the bottom bar of Figure 22, GM time of onset averaged 20.08\% ± 16.51\% of the gait cycle, and activity ceased at an average of -11.22\% ± 14.18\% of the gait cycle (where positive values indicate activity prior to FS, 0\% represents FS, and negative values occurred after FS). In comparison, Mann et al.\textsuperscript{26} reported GM onset to occur at 15\%, and terminate at -20\% (top bar). Figure 22 shows that the GM onset of the subjects in the current study fired 5\% of the gait cycle sooner than that reported by Mann et al., and terminated 10\% earlier. This change in GM activity may be due in part to
running speed. In the current study, the average running speed was 10.74 km/hr; whereas, the speed in the study by Mann et al. was 12.07 km/hr. Although Mann et al. did not present GM activity at a slower speed, they did show a 5% later onset and 10% earlier termination (overall 15% decrease in time of GM activity as a function of the gait cycle) from a running speed of 12.07 km/hr to 16.09 km/hr.

**Gluteus Medius Muscle Fatigue (Specific Aim #3)**

The purpose of Specific Aim #3 was to assess the relationship between GM muscle fatigue rates and the degree of frontal plane pelvic drop. It was hypothesized that runners presenting with increased frontal plane pelvic drop would show a higher rate of GM muscle fatigue while running as compared to controls. For both the left and right sides, the Pearson’s Correlations between average frontal plane pelvic drop and the GM fatigue rate were poor. The hypothesis was therefore rejected.

As shown in Figures 16 and 17, the GM fatigue rates for the left and right sides clustered around zero, regardless of the degree of frontal plane pelvic drop. There were five instances on the left side and six on the right where the fatigue rate was less than zero, suggesting fatigue. In all but one of these for each side, however, the fatigue rate was still close to zero. A GM fatigue rate that remained close to or greater than zero suggests that those subjects did not experience significant fatigue over the 30-minute run. The lack of GM fatigue in the majority of subjects leads to the following questions: (1) Did some of the subjects adapt their running gait or muscle recruitment pattern? (2) Was there an effect of muscle temperature on the fatigue measure? (3) Did the subjects self-select a speed at which they would minimize GM fatigue during the 30-minute run?
It is possible that the varying rates of GM fatigue across subjects could be secondary to attempts to optimize or adapt their running gait, and in turn muscle recruitment strategy. Three possible scenarios exist. (1) Subjects could have adapted their technique prior to fatigue, in an attempt to prevent fatigue onset. (2) Some of the subjects could have modified their performance after fatigue onset, in order to adjust and complete the run. (3) Subjects did not experience fatigue, and there was no need to change their running strategy. If subjects modified their running technique they, in turn, might be able to prevent the start of or further progression of GM fatigue. Previous research has shown strategic changes in movement execution or postural control.84, 85 Galganski et al.84 showed differences in movement execution with age, which the authors hypothesized was due to a “play-it-safe” strategy in older adults to compensate for decreased force generation accuracy and consistency. Madigan et al.85 noted a postural change of slight forward lean in subjects following lumbar extensor fatigue, to control postural sway. In the current study, it is plausible that subjects consciously or unconsciously made attempts to modify their running gait or muscle recruitment while running. Unfortunately, it is not possible to tease out which subjects fell into the above three categories. But attempts to optimize performance, regardless of the timing, might explain why an overall fatigue was not seen consistently across the 30-minute run.

When looking at GM median frequency across the run, there were instances where there appeared to be either an initial downward fatigue rate, prior to a ramping, or simply a ramping during the first half of the data collection. After this point, subjects’ median frequency tended to stabilize. An effect of an insufficient warm-up period prior
to data collection, causing differences between subjects’ muscle temperature, could explain the changes in fatigue rates and low median frequencies observed initially. As muscle temperature increases, median frequency also increases. In turn the use of median frequency to determine fatigue rates is confounded by a temperature effect.

Using a 40% maximum isometric voluntary contraction of the vastus lateralis and recording median frequencies between 39° and 34° C, Madigan and Pidcoe found a linear relationship between EMG mean power frequency and muscle temperature. The mean slope normalized to the initial EMG mean power frequency was 3.26%/°C.

Unfortunately, there is no previous research investigating a median frequency and GM muscle temperature relationship, or this relationship during running. Although subjects were given a five-minute warm-up prior to the start of data collection, they were able to increase their speed as desired. Therefore, variability was introduced to the rate of warm-up. Even if warm-up speed was uniform across subjects, their rate of muscle temperature change would not be consistent across subjects. To compensate for the effects of GM muscle temperature on median frequency, temperature measurements would need to be made at the start and end, as well as periodically throughout the run to establish the relationship between these variables and correct for temperature effects on EMG median frequency data within a subject.

Based on the lack of muscle fatigue seen in the subjects, it is possible that the subjects self-selected a running speed that was not challenging enough to elicit fatigue. Therefore, future research should implement an incremental speed increase such that the subjects would eventually be challenged to the point of GM fatigue.
The 30-minute treadmill protocol was long enough that many of the subjects had considerable perspiration. Although effort was made to create a comfortable running environment and sEMG electrodes were firmly secured during application, moisture still accumulated underneath the electrode as the subject perspired. The presence of perspiration would improve conductivity, thus confounding sEMG data. This, in addition to a muscle temperature effect, could explain changes in GM median frequency described above. An incremental protocol would likely take less than 30-minutes of treadmill running to induce fatigue, thus reducing the amount of perspiration.

Because GM isometric torque, timing of peak amplitude and onset, and fatigue rate were not correlated with pelvic drop, the question arises as to whether muscles other than the GM are key contributors to pelvic stability while running. Future studies are needed to evaluate the influence of other proximal muscles on pelvic kinematics and running biomechanics. In addition to hip abduction strength, these studies could include strength of other core stabilizing and proximal muscle groups to include abdominals, quadratus lumborum, and back extensors. This would provide insight into whether there is an interaction between core muscles that provides proximal stability during running, as well as the degree to which each muscle contributes to this control. These results may be of significance in the rehabilitation and athletic training settings as they imply that a rehabilitation program focused solely on GM strengthening will not decrease frontal plane pelvic drop, possibly resulting in suboptimal running performance.

Since neither GM isometric torque, peak amplitude timing, activation onset, nor fatigue rate correlated with frontal plane pelvic drop, the question of whether frontal
plane pelvic drop is a suitable dynamic measure within the kinetic chain arises. Unfortunately, there are no previous studies investigating frontal plane pelvic drop and its relationship to other dynamic measures. Typically, when observations of frontal plane pelvic drop are performed in a clinical setting, it is impossible to isolate this frontal plane motion at the pelvis. Clinicians may think they are observing pelvic drop, but are influenced by changes elsewhere in the kinetic chain. For instance, an increased frontal plane pelvic drop in a closed kinetic chain would also present more distally as thigh adduction and/or genu valgum in the absence of compensation.

Therefore, is there a more robust measure that would encompass increased pelvic motion and its distal effects? The use of an instrumented treadmill to provide vGRF data would provide more insight into the big picture. Changes throughout the kinetic chain could be captured by vGRF. Since vGRF has previously been connected with an increased injury rate, it would be helpful to link vGRF with changes in LE kinematics that could be more easily identified clinically.63

To address these limitations and remaining questions, future studies would therefore include the use of an instrumented treadmill, LE kinematics, sEMG data on the core muscles, muscle temperature readings, and an incremented treadmill protocol. Using an instrumented treadmill and acquiring LE kinematics would provide data that could establish links between kinetics and joint movement. LE kinematic data would allow calculation of joint torques while running, providing answers as to whether dynamic measures of strength correlate with pelvic drop. sEMG data of the core muscles would help identify which muscles or interaction of muscles have the greatest effect on
kinetic and kinematic changes in running. Lastly, muscle temperature data and an incremental protocol would address limitations with the GM median frequency in this research.

**Running Performance (Specific Aim #4)**

The purpose of **Specific Aim #4** was to study whether the degree of frontal plane pelvic drop was related to changes in metabolic energy demands. It was hypothesized that increased frontal plane pelvic drop would result in increased energy demands, as indicated by RE, to compensate for an inefficient running gait. The Pearson’s Correlation between the change in frontal plane pelvic and RE was poor. An increase in RE was not dependent on an increase in pelvic drop, as seen in Figure 21. Therefore, the hypothesis was rejected.

Allowing subjects to self-select their running speed seemed to result in study limitations. Given that little change in RE was observed over the 30-minute run, the question of whether or not the self-selected speed was challenging for each subject seems appropriate. It is plausible that subjects self-selected a speed that allowed them to maximally utilize their physiologic capabilities. Thus, the subjects choose a speed, which would impose the least metabolic requirements while allowing them to complete the 30-minute run without taxing physiological components. Future studies would therefore first establish the subject’s maximal VO₂. Previous research has shown that when RE is expressed as a percentage of maximal VO₂, the difference in RE between subjects is magnified.88 Trial speed for biomechanical data collection would then be selected based on a percent of the subject’s previously determined maximal VO₂. This would eliminate
the effect of subjects’ self-selecting speeds that may prove to be metabolically less
demanding.

Another possible limitation of this study was inclusion/exclusion criteria for
average distance run per week. A minimum of 8.05 km per week (5 miles per week)
requirement may prove to have been too broad. In turn, the sample included subjects
who ran up to 80.5 km per week (50 miles per week) in preparation for a marathon.
Although post-hoc testing for the correlation between ΔRE and miles run per week
showed no interaction (R = -0.119, p = 0.743), more specific criteria, such as running an
average of 5 to 10 miles per week, would create a more homogenous sample.

Future studies collecting additional kinetic and kinematic variables, as described
previously, would also allow investigation into whether interaction of these variables best
correlates to RE. Williams and Cavanagh\textsuperscript{75} studied the relationship between sagittal
plane running kinematics and submaximal VO\textsubscript{2} in 31 subjects who had been training at
least 25 miles per week for the past several months. They concluded that no single
variable, but rather a weighted sum of variables explained variation in submaximal VO\textsubscript{2}.

In conclusion, GM static strength, peak activity and onset timings, and fatigue did
not predict frontal plane pelvic drop. Pelvic drop also did not have an effect on running
performance, as indicated by RE. One should therefore be weary of a GM centered
treatment approach when addressing frontal plane pelvic instability in runners. Future
research into other core stabilizing muscles and their interactions could provide guidance
into which muscles should be the focus of treatment in this patient population.
Additional research should also include kinetic as well as LE kinematic analysis of
running gait to investigate the link between these variables, their relationship to muscle performance, as well as to running performance.
References


(77) Bohannon RW. Reference values for extremity muscle strength obtained by hand-held dynamometry from adults aged 20 to 79 years. *Archives of Physical Medicine and Rehabilitation* 1997;78(1):26-32.


APPENDIX 1

Self-report questionnaire
RUNNING QUESTIONNAIRE

Name:___________________________________________

Age:_______ Height:_______ Weight:_______

Average miles run per week:________

Longest run per week:________ Minutes _______ Miles

Number of years running:________

Running shoe brand/type:__________________________________________

How long have you worn your present shoes:_____________

Have you had a running-related injury in the past 6 months?  If yes, explain.
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Do you now or have you ever experienced any of the following?

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased running speed/distance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Running-related pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leg weakness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tingling in your legs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sharp pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cramping</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX 2

Pin-out for the ribbon cable going to the A/D card
APPENDIX 3

MATLAB™ programs

The following pages contain the MATLAB™ programming language used to identify FS; frontal plane pelvic drop; GM onset, peak amplitude, and median frequency.
This program identifies the frames of foot strike and average left and right maximum pelvic drop for the specified trial.

```matlab
clear all
clf;
%---------------------------------------------------------------------------
%Input sensor, LC, and trigger data
%---------------------------------------------------------------------------
trial=input ('Input minute number ');
G=input('Select File for Data ', 's');
g=xlsread (G, 'a10:i10009');
%identify frame #, z, x, and y coordinates for the frame
F=g(:,1);
Lz=g(:,2);
Rz=g(:,3);
Lx=g(:,4);
Rx=g(:,5);
Ly=g(:,6);
Ry=g(:,7);
frame=g(:,1);
LC=g(:,8);
trigger=g(:,9);
len=length(frame);
%---------------------------------------------------------------------------
%finds frame when trigger is turned on
%---------------------------------------------------------------------------
for i=1:len
    if trigger(i)>1
        frametrigger=frame(i);
        break;
    end
end
%---------------------------------------------------------------------------
%finds frame after trigger on corresponding to the Right FS(flagR)
%---------------------------------------------------------------------------
for j=(frametrigger-1):len
    if LC(j-1)=-5 & LC(j)>-5
        flagR=frame(j);
        break;
    end
end
%---------------------------------------------------------------------------
%finds all frames of FS prior to flagR
%---------------------------------------------------------------------------
for k=2:flagR
    if LC(k-1)=-5 & LC(k)>-5
        LConA(k)=frame(k);
    end
end
A=LConA';
[row, col, v]=find (A);%erases zeros out of column
```
A=v; %column of FS prior to flagR
%---------------------------------------------------------------------
%finds all frames of FS after flagR
%---------------------------------------------------------------------
for m=flagR:(len-1)
    if LC(m)==-5 && LC(m+1)>-5
        LConB(m+1)=frame(m+1);
    end
end
B=LConB';
[row, col, w]=find(B); %erases zeros out of column
B=w; %column of FS after flagR
%---------------------------------------------------------------------
%first element in B=flagR so R contact, L after...
%---------------------------------------------------------------------
Blen=length(B);
o=1;
if Blen==1
    RightB(o)=B;
else
    for n=2:2:Blen
        RightB(o)=B(n-1);
        LeftB(o)=B(n);
        o=o+1;
    end
end
%---------------------------------------------------------------------
%if length of A(prior to flagR) is even, first element is Right FS
%---------------------------------------------------------------------
Alen=length(A);
q=1;
e=Alen/2;
f=iswhole (e);
for p=1:2:Alen
    if f==1
        LeftA(q)=A(p+1);
        RightA(q)=A(p);
    elseif p==Alen
        LeftA(q)=A(p);
    else
        LeftA(q)=A(p);
        RightA(q)=A(p+1);
    end
q=q+1;
end
%---------------------------------------------------------------------
%add A+B columns to become 1 column
%---------------------------------------------------------------------
if Blen==1
    LLC=LeftA';
    RLC=vertcat (RightA', RightB');
else
    LLC=vertcat (LeftA', LeftB');
    RLC=vertcat (RightA', RightB');
end
llc=LLC;
rlc=RLC;
lenLLC=length(LLC);
lenRLC=length(RLC);
if lenLLC>lenRLC
    rlc(lenLLC)=0;
elseif lenRLC>lenLLC
    llc(lenRLC)=0;
end
lc=[llc rlc];
Contact=['Contact', num2str(trial)];
xlswrite (Contact, lc);

%---------------------------------------------------------
%load in files
%---------------------------------------------------------
B=input('Select File for Baseline Angles ', 's');
b=xlsread (B,'a10:g10009');
aveLz=mean(b(:,2));
aveRz=mean(b(:,3));
aveLx=mean(b(:,4));
aveRx=mean(b(:,5));
aveLy=mean(b(:,6));
aveRy=mean(b(:,7));
BL=(asind((aveLz-aveRz)/(sqrt((aveLx-aveRx)^2+(aveLy-aveRy)^2+(aveLz-
    aveRz)^2))));
BR=(asind((aveRz-aveLz)/(sqrt((aveLx-aveRx)^2+(aveLy-aveRy)^2+(aveLz-
    aveRz)^2))));

%--------------------------------------------------------
%identify L angles from LIC to LIC to find L PSIS mvt
%--------------------------------------------------------
flen=length(F);
LEFT=zeros(flen,1);
for i=1:flen
    Ldeg=((asind((Lz(i)-Rz(i))/(sqrt((Lx(i)-Rx(i))^2+(Ly(i)-
    Ry(i)))^2+(Lv(i)-Rv(i))^2)))-BL);%in degrees
    LEFT(i)=Ldeg;
end
l=length(llc);

%--------------------------------------------------------------------
%Find Left and Right Min during each gait cycle, polynomial fit,
average curve
%--------------------------------------------------------------------
for i=1:l-1
    a=i;
    b=i+1;
    clear ft;
    ft=llc(a):llc(b)-1;%identifies each gait cycle data
    fttlen=length(ft); %length of gait cycle
la=LEFT(ft); % pelvic motion for corresponding gait cycle
cLC=LC(ft);
for j=2:1:ftlen
    x1(1)=0;
    x1(j)=x1(j-1)+(1000/(ftlen-1));
end
for jj=2:1:ftlen
    x2(1)=0;
    x2(jj)=x2(jj-1)+(1000/(ftlen-1));
end
x1=x1';
x2=x2';
xx=spline(x1,la); % cubic spline
yLC=spline(x2,cLC);
t=ppval(xx,x1);
tLLC=ppval(yLC,x2);
norm=(0:1:1000);
s=(ppval(xx,norm));
sLLC=(ppval(yLC,norm));
qq(:,i)=s';
LMin(i)=min(s(1:250));
RMin(i)=max(s(400:1000));
qLC(:,i)=sLLC';
clear x1;
clear x2;
end
aveqq=mean(qq'); % ensemble average of kinematic data
figure(2)
plot(aveqq);
hold on
aveqLC=mean(qLC'); % ensemble average of LC data
plot(aveqLC, 'k');
title('Left Ensemble Average');
hold off

% Find Left and Right Average pelvic drop
AveLMin=mean(LMin);
AveRMin=mean(RMin);
ave=[AveLMin AveRMin];

% Save pelvic ROM for all cycles, and average left and right pelvic drop
Degree=['Degree', num2str(trial)];
xlswrite(Degree, LEFT);
AveMin=['AveMin', num2str(trial)];
xlswrite(AveMin, ave);
clear all;
trial=input ('Input minute number ');
%---------------------------------------------------------
%load in files
%---------------------------------------------------------
L=input ('Select File for foot contact data ', 's');
l=xlsread (L);
LLC=l(:,1);
RLC=l(:,2);
R=input('Select file for EMG data ', 's');
RMS=xlsread (R, 'a10:m10009');
LRMS=RMS(:,12);
RRMS=RMS(:,13);
R=length(RLC);
for i=1:R-1
   a=i;
b=i+1;
   EMGl=LRMS(LLC(a):(LLC(b)-1));
   EMGr=RRMS(RLC(a):(RLC(b)-1));
   peak_EMGl(i)=max(EMGl);%identify L peak EMG for current gait cycle
   peak_EMGr(i)=max(EMGr);%identify R peak EMG for current gait cycle
   min_EMGl(i)=min(EMGl);%identify L minimum EMG for current gait cycle
   min_EMGr(i)=min(EMGr);%identify R minimum EMG for current gait cycle
end
LpeakEMG=mean(peak_EMGl);%Find average L peak EMG
SDLpeakEMG=std(peak_EMGl);%Find SD of L peak EMG
RpeakEMG=mean(peak_EMGr);
SDRpeakEMG=std(peak_EMGr);
LminEMG=mean(min_EMGl);
SDLminEMG=std(min_EMGl);
RminEMG=mean(min_EMGr);
SDRminEMG=std(min_EMGr);
maxminEMG=[LpeakEMG SDLpeakEMG RpeakEMG SDRpeakEMG LminEMG SDLminEMG RminEMG SDRminEMG];
EMGdata=['EMGdata',num2str(trial)];
xlswrite (EMGdata, maxminEMG);
clear all;
cif;
trial=input ('Input minute number ');
%load in files
%-------------------------------------------------------------
L=input ('Select File for foot contact data ', 's');
l=xlsread (L);
LLC=l(:,1);
RLC=l(:,2);
R=input('Select file for EMG data ', 's');
RMS=xlsread (R, 'a10:m10009');
LRMS=RMS(:,12);
RRMS=RMS(:,13);
R=length(RLC);
P=input('Select file for average peak EMG data ', 's');
Peak=xlsread (P);
LPeak=Peak(1,1);
RPeak=Peak(1,2);
LMin=Peak(1,3);
RMin=Peak(1,4);
%---------------------------------------------------------------------
%identify threshold based on 50% of average peak over 30 min run
%---------------------------------------------------------------------
l_on=.50*LPeak;
r_on=.50*RPeak;
%----------------------------------------------------------------------
%Cubic spline data to find ave EMG for each gait cycle
%----------------------------------------------------------------------
for i=1:R-1
a=i;
b=i+1;
gt=RLC(a):(RLC(b)-1);
gtl=LLC(a):(LLC(b)-1);
gtlend(i)=length(gtl);
Lgtlen=length(gtl);
EMGl=LRMS(LLC(a):(LLC(b)-1));
EMGr=RRMS(RLC(a):(RLC(b)-1));

for k=2:1:Lgtlen
Le1(1)=0;
Le1(k)=Le1(k-1)+(1000/(Lgtlen-1));
end
for j=2:1:gtlen(i)
Re1(1)=0;
Re1(j)=Re1(j-1)+(1000/(gtlen(i)-1));
end
Le1=Le1';
Re1=Re1';
ee=spline(Le1,EMGl);%cubic spline
ff=spline(Re1,EMGr);
t=ppval(ee,Le1);
tt=ppval(ff,Re1);
figure(1);
subplot(1,2,1);
plot(Le1,EMGl);
hold on;
plot(Le1,t,'c-');
title('Left RMS EMG');
subplot(1,2,2);
plot(Re1,EMGr,'b');
title('Right RMS EMG');
hold on;
plot(Re1,tt,'g-');
norm=(0:1:1000);
s=(ppval(ee,norm));
qq(:,i)=s';
rr(:,i)=tt';
[tL,I]=max(qq);%identify % gait cycle where peak occurs
if I(i)>500
   III(i)=-(I(i)-1001);
else
   III(i)=-I(i);
end
[tR,II]=max(rr);
if II(i)>500
   IIII(i)=-(II(i)-1001);
else
   IIII(i)=-II(i);
end
%---------------------------------------------
%Find onset for gait cycle based on 50% peak
%---------------------------------------------
lcount=1;
for ii=501:1001
   if qq(ii,i)>l_on && qq(ii-1,i)<=l_on
      Lflag(i,lcount)=-(ii-1001);
      lcount=lcount+1;
   end
   if qq(ii,i)<=l_on && qq(ii-1,i)>l_on
      Lflag(i,lcount)=-(ii-1001);
      lcount=lcount+1;
   end
end
for ii=1
   if qq(ii,i)>l_on && qq(1001,i)<=l_on
      Lflag(i,lcount)=-ii;
   end
end
lcount=lcount+1;
end
if qq(ii,i)<=l_on && qq(1001,i)>l_on
    Lflag(i,lcount)=-ii;
    lcount=lcount+1;
end
end
for ii=2:500
    if qq(ii,i)>l_on && qq(ii-1,i)<=l_on
        Lflag(i,lcount)=-ii;
        lcount=lcount+1;
    end
    if qq(ii,i)<=l_on && qq(ii-1,i)>l_on
        Lflag(i,lcount)=-ii;
        lcount=lcount+1;
    end
end
rcount=1;
for jj=501:1001
    if rr(jj,i)>r_on && rr(jj-1,i)<=r_on
        Rflag(i,rcount)=-(jj-1001);
        rcount=rcount+1;
    end
    if rr(jj,i)<=r_on && rr(jj-1,i)>r_on
        Rflag(i,rcount)=-(jj-1001);
        rcount=rcount+1;
    end
end
for jj=1
    if rr(jj,i)>r_on && rr(1001,i)<=r_on
        Rflag(i,rcount)=-jj;
        rcount=rcount+1;
    end
    if rr(jj,i)<=r_on && rr(1001,i)>r_on
        Rflag(i,rcount)=-jj;
        rcount=rcount+1;
    end
end
for jj=2:500
    if rr(jj,i)>r_on && rr(jj-1,i)<=r_on
        Rflag(i,rcount)=-jj;
        rcount=rcount+1;
    end
    if rr(jj,i)<=r_on && rr(jj-1,i)>r_on
        Rflag(i,rcount)=-jj;
        rcount=rcount+1;
    end
end
clear Le1;
clear Re1;
clear lcount;
clear rcount;
end

for z=1:R-1
    for col=2:50
        if Lflag(z,col)==0 && Lflag(z,col+1)==0
            even=col/2;
            if iswhole(even)==1
                Lflagoff(z)=Lflag(z,col);
                break;
            elseif iswhole(even)~=1
                Lflagoff(z)=Lflag(z,col-1);
                break;
            end
        end
    end
end

for zz=1:R-1
    for colR=2:50
        if Rflag(zz,colR)==0 && Rflag(zz,colR+1)==0
            evenR=colR/2;
            if iswhole(evenR)==1
                Rflagoff(zz)=Rflag(zz,colR);
                break;
            elseif iswhole(evenR)~=1
                Rflagoff(zz)=Rflag(zz,colR-1);
                break;
            end
        end
    end
end

%------------------------------------------------------------------
%Save data
%------------------------------------------------------------------
tpeakEMGL=mean(III);
medtpeakEMGL=median(III);
SDLtpeakEMG=std(III);
tpeakEMGR=mean(IIII);
medtpeakEMGR=median(IIII);
SDRtpeakEMG=std(IIII);

Lonset=mean(Lflag(1:(R-1),1));
medLonset=median(Lflag(1:(R-1),1));
SDLonset=std(Lflag(1:(R-1),1));
Ronset=mean(Rflag(1:(R-1),1));
medRonset=median(Rflag(1:(R-1),1));
SDRonset=std(Rflag(1:(R-1),1));

Loff=mean(Lflagoff);
medLoff=median(Lflagoff);
SDLoff=std(Lflagoff);
Roff=mean(Rflagoff);
medRoff=median(Rflagoff);
SDRoff=std(Rflagoff);

%--------------------------------------------------------
%Plot sEMG for each gait cycle
%--------------------------------------------------------
qq_a=qq';
rr_a=rr';
EMGlave=mean(qq_a);
EMGrave=mean(rr_a);
subplot(1,2,1);
plot(norm,EMGlave, 'k');
hold on;
subplot (1,2,2);
plot(norm, EMGrave, 'k');
hold on;
subplot(1,2,1);
plot(norm, l_on, 'r--');
hold off;
subplot(1,2,2);
plot(norm, r_on, 'r--');
hold off;

peakinfo=[tpeakEMGL medtpeakEMGL SDLtpeakEMGL tpeakEMGR medtpeakEMGR
SDRtpeakEMG Lonset medLonset SDLonset Ronset medRonset SDLoff
medoff SDloff Roff medRoff SDRoff];
Peak=['Peak',num2str(trial)];
xlswrite (Peak, peakinfo);

hold off;
clear all;
cif;

% Program to compute median frequency for L and R EMG data

trial=input ('Input minute number ');

% load in data files

F=input('Select file for Raw EMG ', 's');
fat=xlsread (F, 'a10:k10009');
Lraw=fat(:,10);
Rraw=fat(:,11);

file_len=length(Lraw);
sampling_rate = 1000;                         % set to 1000Hz
interval = 1 / sampling_rate;

% REMOVE OFFSET FROM DATA
Lraw = Lraw - mean(Lraw);
Rraw = Rraw - mean(Rraw);

% CREATE TIME ARRAY FOR PLOTTING
cnt=1;
while cnt <= file_len
    xtime(cnt) = (cnt-1)*interval;
    cnt = cnt + 1;
end

% PLOT RAW DATA
subplot(4,1,1)
plot(xtime,Lraw,'b')
str = sprintf('Raw Data (L)');
title(str)
xlabel('time')
ylabel('volts')
axis([0 10 -1 1]);

% FFT

Y=fft(Lraw);
YY=Y.*conj(Y)/length(Y);
yy=YY(1:5000);

f=1000*(0:length(yy)/2)/length(yy);
subplot(4,1,2)
plot(f(1:int16(length(yy)/2)),yy(1:int16(length(yy)/2)),'b')
hold on;

% COMPUTE MEDIAN FREQUENCY
cnt = length(Y)/2+1;
sum = 0;
for i=1:cnt
    sum = sum + (f(i) * YY(i));
end
half_sum = sum/2; sum = 0;

i=1;
while sum <= half_sum
    sum = sum + (f(i) * YY(i));
    median_freqL = f(i);
    i = i + 1;
end

median_freqL;
x = [median_freqL median_freqL]; plot(x,ylim,'r'); % ylim = axis limits
str = sprintf('FFT (L) median freq = %.2f',median_freqL);
title(str)
xlabel('freq')
ylabel('amplitude')
hold off;

% PLOT RAW DATA
subplot(4,1,3)
plot(xtime,Rraw,'b')
str = sprintf('Raw Data (R)');
title(str)
xlabel('time')
ylabel('volts')
axis([0 10 -1 1]);

Z=fft(Rraw);
ZZ=Z.*conj(Z)/length(Z);
zz=ZZ(1:5000);
fz=1000*(0:length(Z)/2)/length(Z);
subplot(4,1,4)
plot(fz(1:int16(length(Z)/2)),zz(1:int16(length(Z)/2)),'b')
hold on;

% COMPUTE MEDIAN FREQUENCY
cntz = length(Z)/2+1;
sum = 0;
for i=1:cntz
    sum = sum + (fz(i) * ZZ(i));
end
half_sum = sum/2; sum = 0;

i=1;
while sum <= half_sum
    sum = sum + (fz(i) * ZZ(i));
    median_freqR = fz(i);
i = i + 1;
end

median_freqR;
x = [median_freqR median_freqR]; plot(x,ylim,'r'); % ylim = axis limits
str = sprintf('FFT (R) median freq = %.2f',median_freqR);
title(str)
xlabel('freq')
ylabel('amplitude')
hold off;

mf=[median_freqL median_freqR];
Freq=['Freq',num2str(trial)];
xlswrite (Freq, mf);
APPENDIX 4

Summary of subjects’ demographics and injury history
<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Height(cm)</th>
<th>Weight(kg)</th>
<th># km Run/Week</th>
<th>km/hr</th>
<th>History of Running-Related Injuries in Past 6 Months</th>
</tr>
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<td>L ankle sprain</td>
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</table>
APPENDIX 5

Frontal plane pelvic drop data

This appendix contains pelvic drop angles (degrees) at each 2-minute increment during the 30-minute run and average values for the left and right sides. Following each is its corresponding standard deviation table.
<table>
<thead>
<tr>
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<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
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<th>18</th>
<th>20</th>
<th>22</th>
<th>24</th>
<th>26</th>
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<th>30</th>
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<td>8.31</td>
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</table>

Average data for left side pelvic drop (degrees) for each 10-second data collection. Each subject’s average pelvic drop across each 2-minute increment is also presented.
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<th>AVE</th>
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Average data for right side pelvic drop (degrees) for each 10-second data collection. Each subject’s average pelvic drop across each 2-minute increment is also presented.
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Standard deviations (SD) in degrees for the right side pelvic drop for each 10-second data collection period. Each subject’s average standard deviation is presented in the last column.
APPENDIX 6

Gluteus medius muscle isometric force data

This appendix presents a table summarizing GM isometric force (F) for the 3 trials, as well as resulting averages (Ave), standard deviations (SD), and isometric torque (T) data for the left (L) and right (R) sides of all subjects.
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APPENDIX 7

Time of gluteus medius peak amplitude data

This appendix contains left and right side data on time of peak sEMG amplitude including accompanying standard deviation tables for each 2-minute increment over the 30-minute run. A value of zero GM peak amplitude timing would indicate that peak amplitude occurred at foot contact (0% gait cycle); whereas, positive values occurred prior to FS and negative values after FS.
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Median time of right side peak GM activity (\(\%\text{gaitcycle} \times 10^{-1}\)) for each 10-second data collection. The average across all trials is shown in the last column.
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Standard deviations (SD) of right side time of peak GM amplitude (%gaitcycle*10^-1) for each 10-second data collection period. Average SD for each subject across all trials is shown in the last column.
APPENDIX 8

Time of gluteus medius muscle onset data

The following appendix contains tables for left and right GM sEMG time of onset for each 2-minute increment for the 30-minute run and the resulting average. Standard deviation data follow the corresponding table.
**Median time of left GM sEMG onset (\(\%\) gait cycle \(\times 10^{-1}\)) for each 10-second data collection, as well as average GM sEMG onset across all trials.**
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**Standard deviations (SD) of the time of left side GM activity onset (%gaitcycle*10^-1) for each 10-second data collection window. The average SD across all data collections for each subject is presented in the last column.**
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### Standard deviations (SD) of the time of right side GM activity onset (%gaitcycle * 10^-1) for each 10-second data collection window. The average SD across all data collections for each subject is presented in the last column.

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APPENDIX 9

Gluteus medius muscle median frequency data

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**RIGHT**

Right side median frequency (Hz) for each 10-second data collection period. The slope of median frequency across the 30-minute run is presented in the last column.
APPENDIX 10

Gluteus medius muscle peak amplitude data

Left and right side GM peak amplitude are summarized in the following tables. Pearson’s Correlations between average maximum pelvic drop and GM peak amplitude were $R = 0.036$ ($p = 0.0513$) and $R = -0.027$ ($p = 0.621$), for the left and right sides respectively, indicating poor correlations between pelvic drop and GM peak amplitude.
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Left side average peak amplitude (v) for each 10-second data collection period. Average peak amplitude across all trials is shown in the last column.
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Right side average peak amplitude (v) for each 10-second data collection period. Average peak amplitude across all trials is shown in the last column.
APPENDIX 11

Observed power following data collection for Specific Aims #1, 2, and 3
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APPENDIX 12

Pearson’s Correlations (R) between frontal plane pelvic drop and gluteus medius muscle torque as a function of time
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APPENDIX 13

Pearson’s Correlations (R) between frontal plane pelvic drop and gluteus medius muscle peak timing as a function of time
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APPENDIX 14

Pearson’s Correlations (R) between frontal plane pelvic drop and gluteus medius muscle onset as a function of time
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APPENDIX 15

Virginia Commonwealth University Institutional Review Board approval letter
DATE: September 6, 2007

TO: Peter E. Pelcuz, PT, DPT, PhD
    Physical Therapy
    Box 980224

FROM: William E. Smith, PharmD, MPH, PhD
      Chairperson, VCU IRB Panel A
      Box 980568

RE: VCU IRB #: HM10526
    Title: Validity of a Clinical Assessment Tool for Evaluating Hip Drop in Runners

On August 30, 2008, this research study was approved for continuation by expedited review according to 45 CFR 46.108(b) and 45 CFR 46.109(c) and 45 CFR 46.110 Category 1 and 4.

VCU IRB APPROVED CONSENT/ASSENT FORM (attached):
- Revised Subject Information and Consent Form (dated 10/18/06; received 8/20/07; 5 pages)

This approval expires on July 31, 2008. Federal Regulations/VCU Policy and Procedures require continuing review prior to continuation of approval past that date. Continuing Review report forms will be mailed to you prior to the scheduled review.

The Primary Reviewer assigned to your research study is Traci Wakefield, RN, MSN, PCCN. If you have any questions, please contact Ms. Wakefield at tswakefi@vcu.edu or 828-4083 or 827-0035; or you may contact Stephan Hicks, IRB Coordinator, VCU Office of Research Subjects Protection, at hicks32@vcu.edu or 828-9876.

Attachment – Conditions of Approval
VITA

Evie N. Burnet was born on February 26, 1978 in Newport News, VA. She graduated from Homer L. Ferguson High School, Newport News, VA in 1996. She received her Bachelor of Science degree in Biology from The University of North Carolina at Chapel Hill in 2000. She received her Doctor of Physical Therapy degree from Virginia Commonwealth University, Richmond, VA in 2003. She subsequently worked as a physical therapist in Williamsburg, VA before pursuing her Ph.D.