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Higher Medially-directed Joint Reaction Forces are a Characteristic of Dysplastic Hips: A Comparative Study Using Subject-Specific Musculoskeletal Models

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Abstract

Acetabular dysplasia is a known cause of hip osteoarthritis. In addition to abnormal anatomy, changes in kinematics, joint reaction forces (JRFs), and muscle forces could cause tissue damage to the cartilage and labrum, and may contribute to pain and fatigue. The objective of this study was to compare lower extremity joint angles, moments, hip JRFs and muscle forces during gait between patients with symptomatic acetabular dysplasia and healthy controls. Marker trajectories and ground reaction forces were measured in 10 dysplasia patients and 10 typically developing control subjects. A musculoskeletal model was scaled in OpenSim to each subject and subject-specific hip joint centers were determined using reconstructions from CT images. Joint kinematics and moments were calculated using inverse kinematics and inverse dynamics, respectively. Muscle forces and hip JRFs were estimated with static optimization. Inter-group differences were tested for statistical significance ($p < 0.05$) and large effect sizes ($d > 0.8$). Results demonstrated that dysplasia patients had higher medially directed JRFs. Joint angles and moments were mostly similar between the groups, but large inter-group effect sizes suggested some restriction in range of motion by patients at the hip and ankle. Higher medially-directed JRFs and inter-group differences in hip muscle forces likely stem from lateralization of the hip joint center in dysplastic patients. Joint force differences, combined with reductions in range of motion at the hip and ankle may also indicate compensatory strategies by patients with dysplasia to maintain joint stability.

Key Terms

Biomechanics; hip; acetabular dysplasia; gait; musculoskeletal modeling

Introduction

Acetabular dysplasia is characterized by a shallow acetabulum that fails to cover and stabilize the femoral head (Cooperman et al., 1983). Poor coverage may lead to deleterious hip contact mechanics, including elevated load support by the acetabular labrum (Henak et al., 2011). Altered hip contact mechanics may, in turn, accelerate osteoarthritis (OA) development (Harris-Hayes and Royer, 2011). Surgeons seek to normalize contact mechanics by correcting hip anatomy (Sanchez-Sotelo et al., 2002). However, anatomy alone does not determine hip contact mechanics. Specifically, muscle forces, combined with joint kinematics and ground reaction forces (GRF), dictate the hip joint reaction force (JRF). The magnitude and direction of this JRF influences patterns of stress observed at the cartilage and labrum. Quantitative comparisons of joint kinematics, muscle forces, and JRFs between dysplastic hips and non-pathologic controls could therefore aid in understanding OA development in dysplastic hips, and may identify new treatment strategies.

Studies of gait in dysplasia patients include comparisons to the presumed healthy contralateral limb (Romano et al., 1996), a control group (Jacobsen et al., 2013; Pedersen et al., 2004; Romano et al., 1996; Skalshoi et al., 2015), or locomotion before and after corrective surgery (Endo et al., 2003; Pedersen et al., 2006). These studies have varied somewhat in their analysis techniques and the dependent variables of interest, but typically use marker-based motion capture to analyze joint kinematics and kinetics for young adults (mean ages 34–48). Results from these studies support the concept that dysplasia alters hip kinematics and kinetics during gait, but data have been contradictory. For example, Romano et al. (1996) and Jacobsen et al. (2013) found lower peak hip extension during stance in patients with dysplasia, whereas Pedersen et al. (2004) found no significant differences in hip extension during stance.

Quantifying joint and muscle forces, using patient specific kinematics and musculoskeletal models may help identify compensation strategies. Resulting data could serve as input to finite element models that estimate cartilage and labrum mechanics to study OA pathogenesis in dysplastic hips. Analysis of joint and muscle forces may also help explain pain, such as trochanteric or buttocks pain, which can present even in absence of articular tissue damage (Nunley et al., 2011). To date, one study has presented estimates of JRFs and muscle forces in patients with dysplasia during gait; results from this study support the concept that JRFs may be altered in dysplastic hips (Skalshoi et al., 2015). Specifically, hip joint forces in patients were estimated to be lower and more superiorly directed than those of controls, which was postulated to be a pain avoidance mechanism to reduce anterior joint loading. This prior study by Skalshoi et al. (2015) utilized generic geometry to calculate the location of the hip joint center (HJC) when predicting JRFs from the musculoskeletal model. Yet due to abnormally shallow acetabular shape, the HJC may be lateralized in patients with dysplasia (Leunig et al., 2001). Perturbations of the HJC may in turn influence calculations

of kinematics, muscle moment arms and subsequent muscle forces (Delp and Maloney, 1993; Maquet, 1999). Incorporating patient-specific HJCs into a musculoskeletal model could yield more physiological predictions.

In this study, we compared lower extremity joint angles, moments, hip JRFs and muscle forces between adults with symptomatic acetabular dysplasia and asymptomatic controls screened for dysplasia. To do so, we used in-vivo motion analysis and musculoskeletal models with HJCs based on 3D reconstructions from computed tomography (CT) images. Based on prior reports, and without a-priori knowledge of how incorporating subject-specific HJCs would affect JRFs for this population, we hypothesized that JRFs would be lower in the patients with dysplasia compared to controls (Skalshoi et al., 2015). Despite inconsistency in previous studies regarding hip range of motion (ROM) during gait, we hypothesized that if hip JRFs were lower in patients, they would be accompanied by less hip ROM.

Methods

Subject Recruitment and Motion Capture

With Institutional Review Board approval and informed consent, CT arthrograms and gait data (kinematics and GRFs) were collected from 10 patients diagnosed with acetabular dysplasia (7 female) and 10 control subjects (7 female). The 10 patients were screened from an original cohort of 38 patients (see supplementary material for details). Each patient had evidence of dysplasia, as determined by a musculoskeletal radiologist and indicated by a lateral center edge angle (LCEA) less than 20° (Delaunay et al., 1997; Werner et al., 2012; Wiberg, 1939). The LCEA indicates lateral coverage of the femur by the acetabulum and is defined in a coronal view as the angle between a line through the center of the femoral head, perpendicular to a line connecting the inferior boundaries of the ilioischial teardrops, and a line drawn from the center of the femoral head to the lateral extent of the acetabulum; LCEA values less than 20° are considered indicative of dysplasia (Wiberg, 1939). Five patients had radiographic evidence of bilateral dysplasia, but at the time of this study, all patients presented with unilateral symptoms and were recommended for hip preservation surgery of the single, symptomatic side. The control subjects had no history of hip dysfunction and were free of dysplasia and OA (e.g. LCEA >20°, no evidence of joint space narrowing, absence of visible cartilage or subchondral bone damage) upon inspection of CT images by the radiologist. CT scanner settings followed previous work, where the scan included the entire pelvis and proximal femurs (Harris et al., 2012).

Gait data were collected using twenty-one 14 mm retro-reflective spherical markers placed on the pelvis, lower limbs, C7 vertebra, and clavicles to define 8 segments, based on a modified Helen-Hayes marker set (Davis et al., 1991). Subjects walked barefoot at a self-selected speed across a 10 meter runway, with 3 meters given for acceleration before entering the capture volume. Marker trajectories were recorded at 100 Hz using 10 near-infrared cameras (Vicon; Oxford, UK). GRFs were recorded at 1000 Hz using 4 concealed force plates (AMTI, Watertown, MA).

Marker trajectories and analog data were captured and synchronized using Vicon Nexus (v1.8) and imported into Visual 3D (v 5.0; C-Motion Inc., Germantown, MD) for processing. Residual analysis was performed on marker and GRF data to determine filter cutoff frequencies that reduced noise without undue elimination of true signal (Winter, 2004). Accordingly, low-pass Butterworth filters were applied to marker and GRF data using cutoff frequencies of 6 Hz and 20 Hz, respectively. Filtered marker and GRF data were exported from Visual 3D and converted to a format compatible with OpenSim (Delp et al., 2007).

Musculoskeletal Modeling

In OpenSim (v3.3), a virtual marker set matching the experimental markers was placed on a 23 degree-of-freedom model of the lower limbs, pelvis, torso, and head. Eighty muscles of the torso and lower limbs were represented by 96 muscle-tendon-actuators; some muscles were represented by multiple actuators (e.g. gluteus medius). Muscles spanning the hip were modified according to Shelburne et al. (2010). Specifically, muscle geometry and maximum isometric forces were matched to experimental and imaging descriptions of muscle moment arms and isometric strength. The model used is available at https://simtk.org/home/hip_muscles/.

Subject-specific HJCs were determined from 3D reconstructions generated by segmenting the CT images using Amira software (v6.0, FEI, Hillsboro, OR) (Harris et al., 2012). Generic pelvis and femur geometries from OpenSim were imported to Amira and scaled using the marker-based scale factors determined with the OpenSim scaling tool. Next, the subject-specific reconstructions were aligned to the scaled generic geometry at the pelvic origin (midpoint between the left and right anterior superior iliac spines). Spheres were fit to the subject-specific femoral heads and the distance from each sphere centroid to the pelvic origin was determined. The resulting anteroposterior, superoinferior, and mediolateral coordinates of the centroids were used as the subject-specific HJC for the OpenSim model (Fig 1). To preserve symmetry in the OpenSim model, the left and right HJCs were taken as the average distance from the origin to the left and right centroids for control subjects and patients noted to have bilateral deformities. For unilateral patients, the left and right hip joint centers were established separately. Generic representation of the pelvis geometry within the OpenSim model was replaced by the subject-specific pelvis reconstruction for each subject. Moment arms for muscles spanning the hip were then updated for each subject by relocating the muscles' origin points onto the subject-specific pelvis based on the bony geometry and anatomical descriptions (Fig 2). The remaining model segments were scaled to each subject using spatial relationships between virtual and experimental markers in OpenSim.

Analyses were performed across a full gait cycle (foot strike to ipsilateral foot strike) of a representative trial for the symptomatic side of the patients and a randomly chosen side of the controls. Joint angles were calculated using inverse kinematics via weighted least squares minimization between experimental and virtual markers. Greater weights were assigned to markers least susceptible to soft-tissue motion and subject-to-subject positional variation. Net joint moments were calculated with inverse dynamics (Winter, 2004). A residual reduction algorithm (RRA) in OpenSim was used to minimize inconsistencies from

experimental factors (e.g. soft-tissue motion), joint angles, and modeling assumptions (e.g. mass distribution) (Delp et al., 2007). Next, static optimization, which has been found to be appropriate for gait (Anderson and Pandy, 2001), resolved net joint moments into individual muscle forces by minimizing the sum of squared muscle activations. Finally, resultant and anteroposterior, superoinferior, and mediolateral hip JRFs were calculated (Steele et al., 2012). Hip JRFs were presented in the pelvic coordinate frame to reflect the direction and magnitude relevant to the acetabulum.

Model Validation and Sensitivity

Model validation and sensitivity studies were conducted (see supplementary material). Model validation involved comparing electromyography (EMG) signals to model-estimated muscle activations for one representative female control subject. For all subjects, joint angles, moments, JRFs, and muscle forces were compared against literature values. Sensitivity studies examined the use of subject-specific HJCs with and without updates to the muscle moment arms.

Data and Statistical Analysis

Walking speeds, stride lengths, pelvis angles (tilt/list/rotation), hip angles (flexion/adduction/rotation), hip moments (flexion/adduction/rotation), knee and ankle angles (flexion), and knee and ankle moments (flexion) were calculated across the full gait cycle. Maximum and minimum angles and moments were identified for each subject as well as ROM at the hip, knee and ankle.

Hip JRFs and GRFs were normalized to bodyweight (xBW). Peak JRFs, during the loading response of early stance (~14% gait, termed 'JRF1') and in mid-to-late stance (~47% gait, termed 'JRF2'), were determined for each subject, as well as joint angles, moments, and GRFs at those time points. Finally, muscle forces at the time of JRF1 and JRF2 for the 21 muscles spanning the hip were calculated and normalized to bodyweight.

All variables were tested for normality using the Shapiro-Wilk test and homogeneity of variance using Levene's test ($\alpha=0.05$). Normally distributed variables with equivalent variances were compared between control and patient groups with a one-way ANOVA, while remaining variables were compared with Mann-Whitney U tests. Additionally, the effect sizes of intergroup differences were determined using Cohen's *d* (Cohen, 1988). For pelvic tilt, which has little variance and no obvious maximum or minimum during gait, angles were averaged across the gait cycle for each subject and statistically tested between groups. In light of modest sample sizes and a potentially large number of comparisons, primary analyses of muscles were performed by grouping muscles by function (flexors, extensors, abductors, adductors, internal rotators, external rotators) for testing between patients and controls. Secondary analyses with Mann-Whitney U tests were used to compare individual muscle forces. Significance for each test was set at $p \leq 0.05$. A definitive rubric of clinically meaningful effect sizes for biomechanical variables does not exist. Hence, the classification proposed by Cohen (1988) was used, where, $d=0.2$ indicated small effect, $d \geq 0.5$ medium effect, and $d \geq 0.8$ large effect.

Results

Subject Characteristics

Age, weight, height, and body-mass index were not statistically significantly different between controls and patients (Table 1). Walking speeds and stride lengths for patients were less than controls, with a large effect size in stride length. Differences in these variables were not statistically significant (Table 1).

Maximum and Minimum Joint Angles and Moments

For the full gait cycle, differences between patients and controls had large effect sizes only for hip flexion ROM, hip adduction ROM, maximum hip rotation moments, and maximum knee flexion moments (Table 2). Patients demonstrated lower values for each of these variables except knee flexion moments; differences were not statistically different.

Inter-Group Differences at JRF1 and JRF2

Patients showed less hip flexion at JRF1 (Table 3). Ankle flexion was significantly different at JRF1, with patients having neutral ankle flexion compared to plantar flexion in controls (Fig 3). At JRF2, no significant inter-group differences or large effect sizes for joint angles were found. Hip external rotation and ankle flexion moments were lower in patients at JRF1 while internal hip abduction moments (i.e. negative adduction moment) were lower in patients at JRF2 (Fig 4), but not to the point of statistical significance (Table 3).

Patients had significantly higher medially directed JRFs than controls at both JRF1 and JRF2, although resultant JRF magnitudes were not significantly different between the groups (Table 3). At JRF1, patients had slightly anteriorly directed JRFs while controls' JRFs were slightly posteriorly directed (Fig 5). Posterior (i.e. negative) GRFs were lower in patients at JRF1 and JRF 2 (Suppl. Fig. 3), with significance only at JRF1 (Table 3).

For muscle groups, large effect sizes were found at JRF1 for only the hip external rotators and at JRF2 for the hip internal rotators (Table 4). The external rotators, including the quadratus femoris, gemmelli, obturator externus, piriformis, and posterior portions of the gluteus medius and minimus, generated less force in the patients than controls at JRF1. The internal rotators, including anterior portions of gluteus medius and minimus, and the tensor fascia latae, generated more force in patients than in controls at JRF2. At JRF1, major force contributors, defined as muscles generating >10% BW, included the gluteus medius, minimus, and maximus; semimembranosus; and rectus femoris. At JRF2, major muscle contributors were the gluteus medius, gluteus minimus, biceps femoris short-head, sartorius, tensor fascia latae, iliacus, psoas, and rectus femoris. Significant differences in the piriformis were found at JRF1, with patients generating less force than controls (Table 4). No significant differences were found at JRF2, although there were large effect sizes for the gluteus minimus and the tensor fascia latae, with forces elevated for both muscles in the patients (Table 4).

Discussion

The objective of this study was to compare lower extremity joint angles, moments, hip JRFs, and muscle forces between young adult patients with acetabular dysplasia and healthy controls during walking. Most of the biomechanical variables analyzed were similar between patients and controls. Statistically significant differences were found for some variables, which should be cautiously considered, but may be meaningful when examining how acetabular dysplasia affects gait. Larger medially directed JRFs were found in the patients at the time of JRF peaks in early and mid-to-late stance. Patients had lower external rotator muscle forces in early stance, but elevated internal rotator muscle forces in mid-to-late stance. While joint angle and moment patterns were mostly similar between the groups, large inter-group effect sizes suggest some movement alterations by patients at the hip and ankle across the entire gait cycle and at the loading response in early stance.

We hypothesized that JRFs would be reduced in the dysplasia patients compared to controls. Instead, when using the subject-specific HJC in the musculoskeletal model, resultant JRFs were not different between groups. In fact, the medially directed JRF component was significantly greater in the patients during the loading response of early stance (i.e. at JRF1) and more so in later stance (i.e. at JRF2). The abnormal geometry of the dysplastic hip results in lateralization of the HJC compared to healthy hips. As a result, moment arms of lateral muscles spanning the hip are reduced compared to normal anatomy and higher medially directed JRFs are required to maintain the torques necessary to stabilize the hip joint (Delp and Maloney, 1993; Maquet, 1999). From a modeling and mechanics standpoint, reducing a muscle's moment arm may require increased force generation by that muscle, depending on the movement (Erdemir et al., 2007). This response can be seen in tensor fascia latae, gluteus minimus, and gluteus medius forces that were larger in patients than controls at JRF2. As a group, hip abductor muscle forces were larger in patients at JRF2, but these findings cannot be definitive because differences compared to controls were not statistically significant and only the tensor fascia latae had a large effect size. We speculate that larger differences in abductor muscle forces would be found between groups during lateral and multi-directional motions, during which the stabilization and movement requirements on the abductor muscles are greater.

For most adult patients with dysplasia who are able to freely ambulate, gait likely does not demand joint ROM that could put the hip in danger of subluxation. However, the results of this study suggest that patients develop movement strategies to protect the hip even during basic ambulation. Specifically, patients began gait with reduced hip flexion and had reduced hip adduction ROM throughout gait. Patients also had less ankle plantar flexion at foot strike and had neutral ankle flexion at JRF1, while controls were still plantarflexed. The lower hip and ankle flexion placed the limb in a more vertical position, closer to the patients' center of mass, which may have felt more stable during gait and contributed to lower posterior GRFs at JRF1. In turn, the patients' reduced posterior GRFs were associated with a slightly anterior JRF versus a JRF that was still posteriorly directed in the controls. At JRF2, anterior GRFs were again reduced in patients, but did not translate to a larger anterior JRF. This is perhaps due to muscles contributing more heavily than GRF at JRF2 for stabilization as the

torso passed over the stance leg and subjects transitioned to propulsion of the next step (e.g. the psoas, which had larger forces in patients than controls at JRF2).

Some prior studies reported a marked decrease in peak hip extension in patients, which was not a finding with our cohort (Jacobsen et al., 2013; Romano et al., 1996; Skalshoi et al., 2015). It has been postulated that reduced hip extension is a pain avoidance mechanism to reduce anterior joint loading (Skalshoi et al., 2015). Indeed, less terminal hip extension has been shown in modeling studies to reduce the anterior JRF (Lewis et al., 2010). However, decreasing walking speeds also reduces hip extension and overall sagittal hip ROM (Schwartz et al., 2008). Patients in the aforementioned studies walked slower than those in our study and slower than their respective control groups, which may explain the source of hip extension differences in previous reports compared to ours. By contrast, patients in the study by Pedersen et al. (2004) had walking speeds more similar to our cohort, and like ours, did not have significantly different hip extension than control subjects. Also, the average age of patients in prior studies was 8–20 years older than our cohort. Older patients with dysplasia have more time to develop compensatory patterns, which may explain why patients from these prior studies had more pronounced differences in gait compared to controls.

To our knowledge, only Skalshøi et al. (2015) have provided model-based estimates of JRFs in adults with dysplasia, where JRFs were less than those of controls. The contrast with our study may be related to our use of subject-specific HJC locations, which were generally lateralized compared to the generic models and likely resulted in higher overall JRF estimations and increased medial JRF components for patients. However, similar to Skalshøi et al. (2015) and other studies, we found that patients adjusted not only their movement patterns at the hip but markedly at the ankle as well, suggesting multi-joint effects of dysplasia during ambulation.

Additional, subtle compensatory mechanisms may be used by patients with dysplasia. For example, in this study, muscle forces of the hip external rotator group were lower in patients than controls at JRF1 and were accompanied by lower external rotation moments. The largest contributors to these forces were the piriformis and posterior portions of the gluteus medius and minimus for both groups. Likewise, the internal rotators generated more force in patients than controls at JRF2, largely from contributions of the anterior gluteus minimus and tensor fascia latae. However, the cause or effect of these differences is not clear. We believe this is motivation to examine higher demand activities that are common to daily life such as running or pivoting.

There were some limitations to this study that warrant discussion. First, the sample size was small and likely decreased the ability to detect significant differences between groups. Post-hoc power analyses indicated power ranging from 0.29–0.73 for the variables presented herein. Thus, we advocate caution when interpreting our results. Although, the number of patients was small, the group was homogeneous, with patients having previous surgery, femoroacetabular impingement, or acetabular retroversion being excluded. Thus, statistical differences detected for the current cohort may be more descriptive of this well-defined group than more generalized studies of hip dysplasia.

The musculoskeletal models herein considered the hip as an ideal ball and socket joint. Although we assigned subject-specific HJC locations, subtle translations of the hip joint, due to unstable anatomy and any muscular response to this instability, were not considered. Also, while the models incorporated subject-specific pelvic bony anatomy, subject-specific muscle reconstructions were not available. Thus, updates to muscle attachments relied on canonical anatomical descriptions. Muscle attachments could be refined with 3D reconstructions of the muscle and inclusion of the entire femur, but these were not available and the effects they would have on model estimates for this cohort are unknown. Future studies should address specific geometric differences in bone and muscle between controls and dysplastic cohorts.

Another limitation is the lack of experimental muscle strength and activation values from subjects in the current study, which along with muscle moment arms and other muscle parameters, affect estimations of muscle force (Erdemir et al., 2007; Herzog, 1992). However, the levels of agreement between model estimated muscle activations and the corresponding EMG signals of the control subject were within standards established for similar musculoskeletal models (Hicks et al., 2015). To our knowledge, experimental reports of activation differences between patients with dysplasia and controls during gait do not exist. Thus, we cannot know if the modeling methods used herein captured subtle inter-group muscle force differences. Instead, the models for all subjects relied on OpenSim's implementation of static optimization, which uses segmental kinematics and baseline muscle properties to minimize a muscle activation objective function. As such, model results reflect intergroup differences in movement and HJC location and neglect instances of muscle co-activation (which static optimization cannot predict for uni-joint muscles) and other alterations to muscle activity (Herzog and Binding, 1993). Despite the assumed similarity of muscle parameters between patients and controls, a strength of our study is that we adjusted not only the HJC, but also muscle attachments according to bony anatomy. Doing so ensured that the muscle moment arms were more subject-specific, which influenced when and how muscles were activated.

A final limitation is the inherent sensitivity of hip joint measurements to skin motion artifact (Leardini et al., 2005). Nevertheless, we believe skin marker artifact would be similar between groups, and thus our results provide good comparative estimates of muscle force distribution for both groups. In the future, applying more accurate kinematic measurements, as provided by dual fluoroscopy, and capturing activities that require greater hip ROM may delineate additional differences between groups.

In conclusion, our findings suggest that hip JRFs during gait are altered by acetabular dysplasia, primarily through an increase in medially directed forces. Even prior to the OA development, hips with dysplasia may compensate to stabilize the joint. Muscle forces and the JRF calculated in this study could be applied to patient-specific finite element models to analyze cartilage and labrum mechanics in dysplastic hips to better-understand the pathogenesis of OA in this population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

- Anderson FC, Pandy MG. Static and dynamic optimization solutions for gait are practically equivalent. *Journal of Biomechanics*. 2001; 34:153–161. [PubMed: 11165278]
- Cohen, J. Statistical power analysis for the behavioral sciences. 2. Lawrence Earlbaum Associates; Hillsdale, NJ: 1988.
- Cooperman DR, Wallensten R, Stulberg SD. Acetabular dysplasia in the adult. *Clinical Orthopaedics and Related Research*. 1983:79–85. [PubMed: 6839611]
- Davis RB, Ounpuu S, Tyburski D, Gage JR. A gait analysis data collection and reduction technique. *Human Movement Science*. 1991; 10:575–587.
- Delaunay S, Dussault RG, Kaplan PA, Alford BA. Radiographic measurements of dysplastic adult hips. *Skeletal Radiology*. 1997; 26:75–81. [PubMed: 9060097]
- Delp SL, Anderson FC, Arnold AS, Loan P, Habib A, John CT, Guendelman E, Thelen DG. Opensim: Open-source software to create and analyze dynamic simulations of movement. *IEEE Transactions on Biomedical Engineering*. 2007; 54:1940–1950. [PubMed: 18018689]
- Delp SL, Maloney W. Effects of hip center location on the moment-generating capacity of the muscles. *Journal of Biomechanics*. 1993; 26:485–499. [PubMed: 8478351]
- Endo H, Mitani S, Senda M, Kawai A, McCown C, Umeda M, Miyakawa T, Inoue H. Three-dimensional gait analysis of adults with hip dysplasia after rotational acetabular osteotomy. *Journal of Orthopaedic Science*. 2003; 8:762–771. [PubMed: 14648262]
- Erdemir A, McLean S, Herzog W, van den Bogert AJ. Model-based estimation of muscle forces exerted during movements. *Clinical Biomechanics (Bristol, Avon)*. 2007; 22:131–154.
- Harris-Hayes M, Royer NK. Relationship of acetabular dysplasia and femoroacetabular impingement to hip osteoarthritis: A focused review. *PM&R: the journal of injury, function, and rehabilitation*. 2011; 3:1055–1067. e1051.
- Harris MD, Anderson AE, Henak CR, Ellis BJ, Peters CL, Weiss JA. Finite element prediction of cartilage contact stresses in normal human hips. *Journal of Orthopaedic Research*. 2012; 30:1133–1139. [PubMed: 22213112]
- Henak CR, Ellis BJ, Harris MD, Anderson AE, Peters CL, Weiss JA. Role of the acetabular labrum in load support across the hip joint. *Journal of Biomechanics*. 2011; 44:2201–2206. [PubMed: 21757198]
- Herzog W. Sensitivity of muscle force estimations to changes in muscle input parameters using nonlinear optimization approaches. *Journal of Biomechanical Engineering*. 1992; 114:267–268. [PubMed: 1602772]
- Herzog W, Binding P. Cocontraction of pairs of antagonistic muscles: Analytical solution for planar static nonlinear optimization approaches. *Mathematical Biosciences*. 1993; 118:83–95. [PubMed: 8260761]
- Hicks JL, Uchida TK, Seth A, Rajagopal A, Delp SL. Is my model good enough? Best practices for verification and validation of musculoskeletal models and simulations of movement. *Journal of Biomechanical Engineering*. 2015; 137:020905. [PubMed: 25474098]
- Jacobsen JS, Nielsen DB, Sorensen H, Soballe K, Mechlenburg I. Changes in walking and running in patients with hip dysplasia. *Acta Orthopaedica*. 2013; 84:265–270. [PubMed: 23594221]
- Leardini A, Chiari L, Della Croce U, Cappozzo A. Human movement analysis using stereophotogrammetry. Part 3. Soft tissue artifact assessment and compensation. *Gait Posture*. 2005; 21:212–225. [PubMed: 15639400]

- Leunig M, Siebenrock KA, Ganz R. Rationale of periacetabular osteotomy and background work. *Instructional Course Lectures*. 2001; 50:229–238. [PubMed: 11372318]
- Lewis CL, Sahrman SA, Moran DW. Effect of hip angle on anterior hip joint force during gait. *Gait Posture*. 2010; 32:603–607. [PubMed: 20934338]
- Maquet P. Biomechanics of hip dysplasia. *Acta Orthopaedica Belgica*. 1999; 65:302–314. [PubMed: 10546353]
- Nunley RM, Prather H, Hunt D, Schoenecker PL, Clohisy JC. Clinical presentation of symptomatic acetabular dysplasia in skeletally mature patients. *Journal of Bone and Joint Surgery Am*. 2011; 93(Suppl 2):17–21.
- Pedersen EN, Alkjaer T, Soballe K, Simonsen EB. Walking pattern in 9 women with hip dysplasia 18 months after periacetabular osteotomy. *Acta Orthopaedica*. 2006; 77:203–208. [PubMed: 16752280]
- Pedersen EN, Simonsen EB, Alkjaer T, Soballe K. Walking pattern in adults with congenital hip dysplasia: 14 women examined by inverse dynamics. *Acta Orthopaedica Scandinavica*. 2004; 75:2–9.
- Romano CL, Frigo C, Randelli G, Pedotti A. Analysis of the gait of adults who had residua of congenital dysplasia of the hip. *Journal of Bone and Joint Surgery Am*. 1996; 78:1468–1479.
- Sanchez-Sotelo J, Trousdale RT, Berry DJ, Cabanela ME. Surgical treatment of developmental dysplasia of the hip in adults: I. Nonarthroplasty options. *Journal of the American Academy of Orthopaedic Surgeons*. 2002; 10:321–333. [PubMed: 12374483]
- Schwartz MH, Rozumalski A, Trost JP. The effect of walking speed on the gait of typically developing children. *Journal of Biomechanics*. 2008; 41:1639–1650. [PubMed: 18466909]
- Shelburne, KB., Decker, MJ., Krong, J., Torry, MR., Philippon, MJ. Muscle forces at the hip during squatting exercise. 56th Annual Meeting of the Orthopaedic Research Society; New Orleans, LA. 2010.
- Skalshoi O, Iversen CH, Nielsen DB, Jacobsen J, Mechlenburg I, Soballe K, Sorensen H. Walking patterns and hip contact forces in patients with hip dysplasia. *Gait Posture*. 2015; 42:529–533. [PubMed: 26365370]
- Steele KM, Demers MS, Schwartz MH, Delp SL. Compressive tibiofemoral force during crouch gait. *Gait Posture*. 2012; 35:556–560. [PubMed: 22206783]
- Werner CM, Ramseier LE, Ruckstuhl T, Stromberg J, Copeland CE, Turen CH, Rufibach K, Bouaicha S. Normal values of Wiberg's lateral center-edge angle and lequesne's acetabular index--a coxometric update. *Skeletal Radiology*. 2012; 41:1273–1278. [PubMed: 22584462]
- Wiberg G. Studies on dysplastic acetabula and congenital subluxation of the hip joint. *Acta Chirurgica Scandinavica*. 1939:5–135.
- Winter, D. Biomechanics and motor control of human movement. 3. Wiley; Hoboken: 2004.

Figure 1.

Determination of subject-specific hip joint center (HJC). *Left* – Baseline OpenSim pelvis and femurs (gold) scaled using marker-based scale factors from motion capture data; for visualization, red spheres were fit at the OpenSim-based HJC. *Middle* – Subject-specific 3D reconstructions from computed tomography (CT) images; green spheres were fit to each femoral head; the pelvis origin (midpoint between left and right anterosuperior iliac spines) was aligned with OpenSim origin (blue diamond). Subject-specific HJC was assigned as the location of the green sphere centroids relative to the pelvis origin. *Right* – Overlay of OpenSim and subject-specific geometry demonstrates differences in OpenSim-based vs subject-specific HJCs.

Figure 2. Musculoskeletal model with subject-specific pelvic anatomy. Hip muscle moment arms were updated for each subject by adjusting attachments at the pelvis to match the subject-specific pelvic anatomy.

Figure 3. Average joint angles. Shaded areas represent 95% confidence intervals. Vertical dashed lines indicate when during gait average JRF1 and JRF2 peaks occurred.

Figure 4. Average joint moments. Shaded areas represent 95% confidence intervals. Vertical dashed lines indicate when during gait average JRF1 and JRF2 peaks occurred.

Figure 5.

Average resultant JRFs and JRF components. Shaded areas represent 95% confidence intervals. Vertical dashed lines indicate when during gait average JRF1 and JRF2 peaks occurred.

Table 1

Demographic and spatiotemporal data for controls and patients with dysplasia. Demographic data are mean $\pm$ standard deviation, while spatiotemporal data are mean [95% CI].

	Control	Dysplastic	p value	Cohen's d
Age (years)	26 $\pm$ 3	26 $\pm$ 7	0.59	-
Weight (kg)	70.50 $\pm$ 19.32	65.3 $\pm$ 12.8	0.59	-
Height (m)	1.72 $\pm$ 0.09	1.69 $\pm$ 0.08	0.38	-
BMI (kg/m ²)	23.4 $\pm$ 4.5	22.7 $\pm$ 3.0	0.97	-
Walking speed (m/s)	1.21 [1.11, 1.31]	1.13 [1.03, 1.23]	0.38	0.5
Stride length (m)	1.30 [1.24, 1.36]	1.21 [1.13, 1.29]	0.12	0.9

Table 2

Maximum and minimum joint angles and moments across the entire gait cycle, which had large inter-group effect sizes. Values shown are mean [95% CI].

		Control	Dysplastic	p value	Cohen's <i>d</i>
Hip Flexion (°)	ROM	40.19 [37.97, 42.41]	36.49 [32.80, 40.18]	0.09	0.8
Hip Adduction (°)	ROM	16.87 [14.12, 19.62]	13.55 [11.47, 15.63]	0.08	0.9
Hip Rotation Moment (Nm/kg)	max	0.13 [0.11, 0.15]	0.08 [0.03, 0.13]	0.14	0.8
Knee Flexion Moment (Nm/kg)	max	0.26 [0.19, 0.33]	0.35 [0.24, 0.46]	0.09	0.8

Table 3

Joint reaction force (JRF), ground reaction force (GRF), joint angles, and moments, which had large inter-group effect sizes at either JRF1 or JRF2. Values shown are mean [95% CI]. Shaded values indicate statistically significant differences between controls and patients with dysplasia.

	At JRF1				At JRF2			
	Control	Dysplastic	P value	Cohen's d	Control	Dysplastic	P value	Cohen's d
JRF: ant-post (xBW)	-0.21 [-0.46, 0.04]	0.05 [-0.15, 0.25]	0.12	0.8	1.73 [1.33, 2.13]	1.68 [1.13, 2.23]	0.80	0.1
JRF: med-lat (xBW)	0.63 [0.39, 0.87]	0.78 [0.64, 0.92]	0.02	0.5	0.72 [0.52, 0.92]	1.09 [0.83, 1.35]	0.03	1.1
GRF: ant-post (xBW)	-0.14 [-0.16, -0.12]	-0.10 [-0.12, -0.08]	0.02	1.2	0.14 [0.12, 0.16]	0.11 [0.09, 0.13]	0.09	0.9
Hip Flexion (°)	19.75 [15.71, 23.79]	13.77 [8.12, 19.42]	0.11	0.8	-11.00 [-14.45, -7.55]	-10.00 [-16.25, -3.75]	0.68	0.1
Ankle Flexion (°)	-4.98 [-8.10, -1.86]	1.16 [-2.66, 4.98]	0.03	1.2	15.70 [12.72, 12.72]	15.61 [10.78, 20.44]	0.58	0.01
Hip Adduction Moment (Nm/kg)	-0.75 [-0.83, -0.67]	-0.67 [-0.79, -0.55]	0.26	0.6	-0.70 [-0.76, -0.64]	-0.64 [-0.69, -0.59]	0.14	0.8
Hip Rotation Moment (Nm/kg)	0.12 [0.10, 0.14]	0.08 [0.03, 0.13]	0.19	0.8	-0.03 [-0.05, -0.01]	-0.03 [-0.08, 0.02]	0.58	0.1
Ankle Flexion Moment (Nm/kg)	-0.34 [-0.40, -0.28]	-0.43 [-0.51, -0.35]	0.12	0.8	-1.47 [-1.55, -1.39]	-1.41 [-1.51, -1.31]	0.48	0.4

Table 4

Muscle force estimates at JRF1 and JRF2 for muscles generating at least 1% of BW (0.01 xBW). Values shown are mean [95% CI].

	Force (xBW) at JRF1				Force (xBW) at JRF2			
	Control	Dysplastic	p-value	Cohen's d	Control	Dysplastic	p-value	Cohen's d
Hip Flexors	0.16 [0.04, 0.28]	0.28 [0.02, 0.54]	0.74	0.4	2.86 [2.44, 3.29]	2.98 [2.21, 3.75]	0.80	0.1
Hip Extensors	1.10 [0.74, 1.46]	0.76 [0.52, 1.00]	0.15	0.7	0.13 [0.05, 0.21]	0.13 [0.04, 0.22]	0.80	0.01
Hip Abductors	2.23 [1.87, 2.59]	2.10 [1.80, 2.40]	0.60	0.3	1.69 [1.40, 1.98]	1.99 [1.72, 2.31]	0.18	0.7
Hip Adductors	0.01 [0.00, 0.02]	0.01 [0.00, 0.02]	0.28	0.4	<0.001 [0.000, 0.001]	<0.001 [0.000, 0.001]	0.58	0.04
Hip Internal Rotators	1.13 [0.98, 1.28]	1.22 [0.96, 1.49]	0.80	0.3	1.12 [0.92, 1.32]	1.40 [1.13, 1.67]	0.12	0.8
Hip External Rotators	0.38 [0.28, 0.47]	0.26 [0.20, 0.32]	0.06	1.0	0.10 [0.04, 0.16]	0.11 [0.03, 0.19]	0.91	0.1
Gluteus medius	1.62 [1.38, 1.86]	1.57 [1.32, 1.82]	0.82	0.1	1.16 [0.93, 1.39]	1.30 [1.06, 1.54]	0.44	0.4
Gluteus minimus	0.21 [0.23, 0.19]	0.21 [0.17, 0.25]	0.88	0.1	0.29 [0.25, 0.33]	0.39 [0.28, 0.50]	0.28	0.8
Gluteus maximus	0.51 [0.26, 0.76]	0.36 [0.22, 0.50]	0.53	0.5	0.04 [0.01, 0.07]	0.03 [0.00, 0.05]	0.68	0.2
Semimembranosus	0.14 [0.09, 0.19]	0.09 [0.03, 0.15]	0.29	0.5	<0.001 [0.000, 0.001]	<0.001 [0.000, 0.001]	0.28	0.6
Semitendinosus	0.03 [0.01, 0.05]	0.02 [0.00, 0.04]	0.22	0.3	<0.001 [0.000, <0.001]	<0.001 [0.000, <0.001]	0.97	0.2
Biceps femoris long-head	0.08 [0.04, 0.12]	0.05 [0.01, 0.09]	0.35	0.5	<0.001 [0.000, <0.001]	<0.001 [0.000, 0.001]	0.97	0.3
Biceps femoris short head	<0.001 [0.000, 0.001]	0.06 [0.00, 0.17]	0.04	0.5	0.38 [0.30, 0.48]	0.40 [0.16, 0.64]	0.35	0.1
Sartorius	0.004 [0.00, 0.01]	0.01 [0.00, 0.02]	0.99	0.4	0.09 [0.07, 0.11]	0.10 [0.03, 0.17]	0.39	0.2
Adductor magnus	0.01 [0.00, 0.02]	0.01 [0.00, 0.02]	0.25	0.4	<0.001 [0.000, <0.001]	<0.001 [0.000, <0.001]	0.97	0.4
Tensor fasciae latae	0.06 [0.03, 0.09]	0.08 [0.04, 0.12]	0.43	0.4	0.19 [0.16, 0.22]	0.27 [0.19, 0.35]	0.25	0.8
Iliacus	<0.001 [0.000, <0.001]	0.11 [0.00, 0.32]	0.28	0.5	1.59 [1.23, 1.95]	1.43 [1.02, 1.84]	0.58	0.3
Psoas	<0.001 [0.000, 0.001]	<0.001 [0.000, 0.001]	0.95	0.03	0.08 [0.00, 0.17]	0.22 [0.00, 0.59]	0.68	0.3
Piriformis	0.05 [0.03, 0.07]	0.02 [0.01, 0.03]	0.03	1.1	0.01 [0.00, 0.02]	0.01 [0.00, 0.03]	0.85	0.1
Rectus femoris	0.10 [0.00, 0.20]	0.08 [0.00, 0.16]	0.39	0.1	0.92 [0.76, 1.08]	0.92 [0.60, 1.24]	0.79	0.1