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SYMPOSIUM: DEVELOPMENTAL DYSPLASIA OF THE HIP

Cumulative Hip Contact Stress Predicts Osteoarthritis in DDH

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Abstract Hip stresses are generally believed to influence whether a hip develops osteoarthritis (OA); similarly, various osteotomies have been proposed to reduce contact stresses and the risk of OA. We asked whether elevated hip contact stress predicted osteoarthritis in initially asymptomatic human hips. We identified 58 nonoperatively treated nonsubluxated hips with developmental dysplasia (DDH) without symptoms at skeletal maturity; the control group included 48 adult hips without hip disease. The minimum followup was 20 years (mean, 29 years; range, 20–41 years). Peak contact stress was computed with the HIPSTRESS method using anteroposterior pelvic radiographs at skeletal maturity. The cumulative contact stress was determined by multiplying the peak contact stress by age at followup. We compared WOMAC scores and radiographic indices of OA. Dysplastic hips had higher mean peak contact and higher mean cumulative contact stress than normal hips. Mean WOMAC scores and percentage of asymptomatic hips in the study group (mean age 51 years) were similar to those in the control group (mean age 68 years). After adjusting for gender and age, the cumulative contact stress, Wiberg center-edge angle, body mass index, but not the peak contact stress, independently predicted the final WOMAC score in dysplastic hips but not in normal hips. Cumulative contact stress predicted early hip OA better than the Wiberg center-edge angle.
Level of Evidence: Level II, prognostic study. See the Guidelines for Authors for a complete description of levels of evidence.

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Introduction

Contact stresses are generally believed to influence whether a hip develops OA and using similar reasoning, various osteotomies have been performed to reduce contact stresses and the risk of OA. Hip dysplasia (developmental dysplasia of the hip or DDH) has traditionally been evaluated by morphologic parameters (the Wiberg center-edge angle, the vertical center-anterior angle, acetabular depth, and acetabular index) [7] from pelvic radiographs. With advances in knowledge, it has been established that DDH (previously called "congenital hip dislocation") is not a uniform clinical entity but rather a broad continuous spectrum ranging from asymptomatic modestly shallow acetabula as the mildest form to frankly dislocated hips as the most severe form. Because insufficient acetabular coverage implies the usual hip loads are distributed on a

smaller weight-bearing surface compared with normal hips, biomechanical research has focused on estimation of the contact stress in the hip rather than simply morphologic evaluation [3]. Peak contact stress in the hip has been compared between different groups of patients with developmental dysplasia of the hip to evaluate outcomes of surgical procedures [12, 37]. These and other experimental studies [29, 36] and clinical reports [13, 25] confirm patients with hip dysplasia have higher contact stress than healthy subjects.

In hips with more severe hip dysplasia, several epidemiologic cross-sectional surveys suggest increased incidence of hip osteoarthritis (OA) [16, 17, 23, 33]. Furthermore, hip dysplasia is one of the independent risk factors for hip OA in addition to age and body mass index [17]. A higher incidence of hip OA together with higher average values of contact stress in dysplastic hips have led to the hypothesis that contact stress may be one of the key parameters involved in cartilage degeneration [4, 5]. Clinical confirmation of the predictive value of contact stress was reported in two clinical studies of patients with hip dysplasia treated with closed reduction and followed up to the average age of 31 years. These authors concluded increased cumulative stress exposure bears higher risk for an unfavorable clinical outcome or osteonecrosis [9, 27]. Although the more severe cases of hip dysplasia are clearly associated with early degeneration [3, 24] (with onset of symptoms in nonsubluxated dysplastic hips at the average age of 35 years [11]), reports on patients with borderline dysplastic hips have been more controversial. One study with 10-year followup of age-matched patients with residual dysplasia without subluxation and normal hips, reported no differences in the reduction of the joint space width or in self-reported hip pain [18]. A recent systematic review found little evidence for a relationship between hip dysplasia and late hip OA discovered in patients older than 50 years of age [23]. However, the authors recognized the relationship for the subsequent risk of OA in persons diagnosed with dysplasia at a young age compared with the subsequent risk of young patients with OA without dysplasia. Also, the role of smaller variations of contact stress in the population with normal hips has not been clearly established. Some authors have speculated most of the cases of "idiopathic" hip OA, in fact, arise as a result of subtle abnormalities in the anatomic structure of the hip that remained unrecognized during childhood and adolescence and only began to cause clinical symptoms in old age [10, 31, 35]. The question, therefore, arises whether contact stress could predict OA in patients with only subtle abnormalities [26].

We asked whether initially asymptomatic nonsubluxated dysplastic hips treated nonoperatively differed from a control population of normal hips with respect to (1) higher long-term prevalence of hip OA than a control population of normal hips, (2) higher peak contact stress, (3) higher cumulative contact stress, and (4) higher CE angles. We further ascertained whether high cumulative contact stress predicted late hip OA in initially asymptomatic nonsubluxated dysplastic hips treated nonoperatively and in the control population. Finally, we compared the predictive value of cumulative contact stress for late OA with the predictive value of earlier reported risk factors, including the Wiberg center-edge angle, peak contact stress, and body mass index.

Materials and Methods

We systematically screened the archives of the Department of Orthopaedic Surgery, University Medical Center, Ljubljana, Slovenia, for anteroposterior pelvic radiographs of patients who made their first outpatient visit between 1965 and 1967. We then reviewed 7750 medical records of those so identified. We included patients (1) with at least one anteroposterior pelvic radiograph taken before January 1, 1985, and (2) who were at least 15 years of age with skeletal maturity at the time the radiograph was taken. We excluded patients with (1) any hip pathology except asymptomatic hip dysplasia; (2) degenerative radiographic changes of hips or clinical complaints in the hip at the time the initial radiograph was taken; (3) insufficient technical quality of the radiograph or incomplete presentation of the pelvis on the radiograph; and (4) all hips surgically operated at any time except cases with THA resulting from OA. If both hips of a subject met the inclusion and exclusion criteria, the hips were analyzed as two individual cases. Of 174 subjects eligible for participation, 60 were either dead or lost to followup; from the remaining 114 patients, 61 (54%) agreed to participate in the study. The study group and the control group were matched in proportions of female hips and right-sided hips, race, average body mass index, and the duration of followup, but not age (Table 1).

These 61 patients were further subdivided into the study group and the control group based on the diagnosis in the medical records. The study group of 34 patients (29 women, five men) had 58 nonoperatively treated hips diagnosed as "congenital dislocation of the hip" or "developmental dysplasia of the hip" and had no clinical complaints or radiographic degenerative changes (Kellgren-Lawrence Grade 0) [19] at the time the first radiograph after skeletal maturity was taken. This group also included patients who were successfully treated for hip dysplasia during childhood and had no residual acetabular dysplasia present at skeletal maturity. The control group of 27 patients (24 women, three men) consisted of 48 skeletally mature hips with diagnoses not related to the hip

Table 1. Comparison of matching parameters, ϑ_{CE}, resultant hip force, p_{max}, p_{cml}, and clinical outcome measures between the study group and the control group

Parameters	Study group	Control group	p Value
Total number of hips	58	48	—
Number of female hips	48	42	0.591
Number of right-sided hips	27	26	0.558
Race	White	White	—
Body mass index (kg/m^2)	26.0 ± 3.6 (19.0–34.5)	24.7 ± 4.3 (17.8–39.3)	0.076
Followup period (years)	29 ± 6 (20–41)	29 ± 7 (20–39)	0.634
Age at initial radiograph (years)	22 ± 8 (15–51)	38 ± 13 (16–63)	< 0.001
Age at followup (years)	51 ± 11 (38–80)	68 ± 10 (43–84)	< 0.001
Wiberg center-edge angle (ϑ_{CE}) (degrees)	26 ± 10 (8–39)	36 ± 7 (24–56)	< 0.001
Resultant hip force (kN)	1.91 ± 0.43 (1.27–3.00)	1.81 ± 0.26 (1.11–2.62)	0.134
Peak contact hip stress (p_{max}) (MPa)	2.90 ± 1.36 (1.38–7.88)	1.66 ± 0.37 (0.92–2.96)	< 0.001
Cumulative contact hip stress (p_{cml}) (MPa-years)	147 ± 85 (57–498)	111 ± 26 (61–181)	0.003
WOMAC score at followup (mm)	580 ± 700 (0–2400)	540 ± 760 (0–2100)	0.736
Kellgren-Lawrence grade at followup (number of hips with Grades 0, 1, 2, 3, 4)	10, 2, 4, 1, 6	3, 1, 2, 0, 0	—
Number of hips without any clinical complaints at followup	24	22	0.696
Number of hips with THA at followup	3	0	0.250

The differences between proportions of female hips, right-sided hips, and asymptomatic hips at followup and hips with THA at followup were tested with Fisher's exact test; values of continuous numeric variables are reported as mean ± standard deviation followed by range in the parentheses and significance with the two-sided unpaired Student's t-test; the Kellgren-Lawrence grade at followup is reported for 23 hips in the study group and six hips in the control group with available radiographs.

(in most cases lumbalgia) and had no clinical complaints in the hip or radiographic signs of any hip abnormality at the time the initial radiograph was taken. None of the participants in the control group had a family history of congenital hip dislocation or hip dysplasia.

The officially evaluated translation of the WOMAC 3.1-VAS questionnaire in the Slovenian language (©Prof. Nicholas Bellamy) [2] was sent to all participants who were instructed to complete the forms at home and return them by mail in a prepaid envelope. The participants also completed an accompanying form with questions on body height, present body weight, body weight at the time the initial radiograph was taken, and any surgical procedures in the hip not recorded in the medical records. The time interval between the initial radiograph and clinical assessment with the WOMAC questionnaire was at least 20 years in all patients. In the WOMAC 3.1-VAS questionnaire, the best total score without any clinical complaints corresponds to 0 mm on the visual analog scale and the worst total score with maximal hip pain, stiffness, and functional disability corresponds to 2400 mm on the visual analog scale. Because we considered THA the worst clinical outcome of OA, WOMAC score of these hips was set to 2400 mm for the purpose of analysis.

Anteroposterior pelvic radiographs taken at followup were available for 23 hips from the study group and six hips from the control group; these were analyzed to complement the self-reported assessment of hip OA. All radiographs were analyzed by a single observer (BM) using the Kellgren-Lawrence radiographic scale [19]; the minimum joint space width was also measured, because these two methods are the most reliable measures of radiographic OA in epidemiologic studies [32]. Joint space width was measured in each hip at the lateral margin of the subchondral sclerosis, at the transection of the weight-bearing surface by a vertical line through the femoral head center, and at the medial margin of the weight-bearing surface bordering the fovea. Minimum joint space width was selected as the smallest of these three measurements and values 2 mm or less were defined as the presence of OA according to Lanyon et al. [22]. Hips that had undergone THA were assigned Kellgren-Lawrence Grade 4 and joint space width less than 2 mm for the purpose of analysis (ie, worst case scenarios).

The peak contact stress was determined with the HIPSTRESS method based on a 3-D mathematical model of the resultant hip joint force in the one-legged stance [14] and of the contact stress [15, 25]. The HIPSTRESS method can be applied by using data from anteroposterior pelvic radiographs as an input. We considered one-legged stance a representative position for slow gait as the most frequent activity in everyday life [28]. The model accounts for the fact that in one-legged stance, the pelvis is slightly inclined and the hip centers are not level. The error in estimating the peak contact stress due to estimated error in determination of geometrical parameters amounts to approximately 10%

[25]. We manually measured the following radiographic parameters from anteroposterior pelvic radiographs [25]: the interhip distance (*l*), the pelvic height (*H*), the pelvic width laterally from the femoral head center (*C*), the coordinates of the insertion point of abductors on the greater trochanter (point coordinates T_x and T_z in the frontal plane), the radius of the femoral head (r), and the Wiberg center-edge angle (ϑ_{CE}). The 3-D reference coordinates of the muscle attachment points were taken from Dostal and Andrews [8] and adjusted by linear scaling with regard to the radiographic pelvic parameters (l, C, H, T_x, T_z) for each individual hip. The solution of the vector equations for the equilibrium of forces and torques yielded the three components of the resultant hip force and the tensions in the abductor muscles. From known values of body weight, the femoral head radius (r), the Wiberg center-edge angle (ϑ_{CE}), the magnitude of the resultant hip force, and the inclination of the resultant hip force with respect to the vertical, the peak contact stress (p_{max}), was computed for every individual hip [15]. The cumulative contact stress (p_{cml}) was computed by multiplying p_{max} of each hip with the patient's age at followup. Body weight (W_B) was used for computation of the peak contact stress and the body mass index. In subjects whose body weight had changed over the years, we used the average between the body weight at the time the initial radiograph was taken and the body weight at followup.

The differences between the study group and the control group in proportions of female hips, right-sided hips, asymptomatic hips at followup, and hips with THA at followup were tested with Fisher's exact test [1]. The study group of dysplastic hips and the control group of normal hips were matched on gender, number of right-sided hips, body mass index, race, and followup period (Table 1). Continuous numeric variables in the two groups were compared with the two-sided unpaired Student's t-test. Correlation of the Kellgren-Lawrence grade [19] and the minimum joint width with the WOMAC score was assessed with the nonparametric Spearman's rank correlation coefficient rho. Bivariate correlations of p_{cml}, p_{max}, and ϑ_{CE} with WOMAC score were evaluated with the Pearson's correlation coefficient. The predictive value of the cumulative contact stress (p_{cml}) in the development of hip OA was analyzed by using multiple linear regression with WOMAC score as the numeric dependent variable (clinical end point) and gender, body mass index, and p_{cml} normalized to the body weight (p_{cml}/W_B) as independent variables. Normalized p_{cml}/W_B was used to avoid collinearity with the body mass index. The predictive value of the peak contact stress (p_{max}) in the development of hip OA was analyzed by using multiple linear regression with WOMAC score as the numeric dependent variable (clinical end point) and gender, body mass index, age, and p_{max} normalized to the body weight (p_{max}/W_B) as independent variables. We normalized p_{max}/W_B to avoid collinearity with the body mass index. The predictive value of the Wiberg center-edge angle (ϑ_{CE}) in the development of hip OA was analyzed by using multiple linear regression with the WOMAC score as the numeric dependent variable (clinical end point) and gender, body mass index, age, and ϑ_{CE} as independent variables. All multiple linear regression models with predictive values met the assumptions of linearity, homoscedasticity, and normal distribution of residuals. The results were presented in the form of unstandardized coefficients β (ie, regression coefficients) and standardized coefficients β (to compare the relative strength of the various predictors within the model). We considered two-sided p values 0.05 or less as significant in all analyses. Post hoc power analysis was performed for all correlations, and multiple linear regression models with values of power 0.80 or more were considered satisfactory. We pooled the participants from both the dysplastic and normal groups together (106 hips) and analyzed the optimal values for sensitivity and specificity of biomechanical predictors for the prediction of WOMAC score greater than 1200 mm. Analyses were performed with SPSS® 13.0 for Windows (SPSS Inc, Chicago, IL), Microsoft Office Excel 2003 (Microsoft Inc, Redmond, WA), and GPower 2.0 (Faul F and Erdfelder E, Bonn, Germany).

Results

At last followup we observed no difference between the study group and the control group in the proportion of asymptomatic hips, the proportion of THAs, the values of WOMAC score, or the distribution of radiographic Kellgren-Lawrence scores (Table 1). WOMAC scores were normally distributed both in the dysplastic group and the normal group.

Dysplastic hips with higher p_{cml} exhibited the same prevalence of OA at the average age of 51 years as normal hips at the average age of 68 years. In 29 hips with available radiographs (23 hips from the study group, six hips from the control group), the Kellgren-Lawrence grade [19] correlated with ($\rho = 0.810$; $p < 0.001$; power = 1.00) the WOMAC score and the minimum joint space width 2 mm or less correlated with ($\rho = 0.580$; $p = 0.001$; power = 0.96) the WOMAC score.

Compared to patients with normal hips, the patients with dysplasia had higher average values of peak contact stress (p_{max}) ($p < 0.001$), higher average values of cumulative contact stress (p_{cml}) ($p = 0.003$), and lower average values of Wiberg center-edge angle (ϑ_{CE}) ($p < 0.001$). We found no difference in the magnitude of the resultant hip force.

After adjusting for gender and age, the cumulative contact stress, Wiberg center-edge angle, body mass index, but not the peak contact stress, independently predicted the final WOMAC score in dysplastic hips but not in normal hips. In the study group, we found a bivariate correlation of p_{cml} ($r = 0.482$; $p < 0.001$; power = 0.98), p_{max} ($r = 0.376$; $p = 0.004$; power = 0.86), and ϑ_{CE} ($r = -0.402$; $p = 0.002$; power = 0.91) with WOMAC score at followup. We also found p_{cml} normalized to the body weight ($\beta = 0.381$; $p = 0.004$), ϑ_{CE} ($\beta = -0.306$; $p = 0.012$), and body mass index ($\beta \geq 0.239$; $p \leq 0.051$) independently predicted the WOMAC score, whereas p_{max} normalized to the body weight did not (Tables 2, 3, 4). In the control group, none of the variables p_{cml}, p_{max}, and ϑ_{CE} had any correlation with WOMAC score at followup and none of the multiple linear models (Tables 5, 6) predicted the WOMAC. In addition to whole WOMAC score, multiple linear regression models were also used to analyze subgroups of specific questions (five questions for pain, two questions for stiffness, 17 questions for functional disability) as separate clinical endpoints. In the dysplastic group the adjusted R-squared values for whole WOMAC score, separate pain score, separate stiffness score, and separate functional disability score did not differ. In the normal group none of the multiple linear models predicted subgroups of WOMAC questions.

After pooling the subjects in the two groups the optimal values for predicting the WOMAC were 28° for the center-edge angle (48% sensitivity and 73% specificity),

Table 2. Normalized cumulative contact stress of study group and BMI predicted WOMAC

Independent variable	Unstandardized coefficient β	Standard error	Standardized coefficient β	p Value
Constant	−940.582	515.877	—	0.074
Gender (male = 1)	−236.540	249.484	−0.119	0.347
Body mass index	42.410	21.206	0.239	0.051
p_{cml}/W_B	2.492	0.834	0.381	0.004

Study group (n = 58): multiple linear regression coefficients of the normalized cumulative contact hip stress (p_{cml}/W_B) (kPa·years/N), body mass index (kg/m^2), and gender (male = 1) with WOMAC score at followup (mm) as the numeric dependent variable (model summary: $r = 0.531$; adjusted $r^2 = 0.242$; $p < 0.001$; power = 0.95).

Table 3. Normalized peak contact stress of study group did not predict WOMAC

Independent variable	Unstandardized coefficient β	Standard error	Standardized coefficient β	p Value
Constant	−1736.100	622.761	—	0.007
Gender	−325.440	259.190	−0.163	0.215
Body mass index	43.179	21.388	0.244	0.049
Age	18.657	8.173	0.272	0.026
p_{max}/W_B	91.786	50.089	0.238	0.073

Study group (n = 58): multiple linear regression coefficients of the normalized peak contact hip stress (p_{max}/W_B) (kPa/N), age (years), body mass index (kg/m^2), and gender (male = 1) with WOMAC score at followup (mm) as the numeric dependent variable (model summary: $r = 0.529$; adjusted $r^2 = 0.225$; $p = 0.001$; power = 0.90).

Table 4. Wiberg CE angle of study group predicted WOMAC

Independent variable	Unstandardized coefficient β	Standard error	Standardized coefficient β	p Value
Constant	−438.376	700.001	—	0.534
Gender	−397.767	232.562	−0.199	0.093
Body mass index	46.492	20.568	0.262	0.028
Age	15.049	8.058	0.219	0.067
ϑ_{CE}	−29.997	11.545	−0.306	0.012

Study group (n = 58): multiple linear regression coefficients of the Wiberg center-edge angle (ϑ_{CE}) (degrees), age (years), body mass index (kg/m^2), and gender (male = 1) with WOMAC score at followup (mm) as the numeric dependent variable (model summary: $r = 0.566$; adjusted $r^2 = 0.269$; $p < 0.001$; power = 0.96).

Table 5. Normalized cumulative contact stress of control group did not predict WOMAC

Independent variable	Unstandardized coefficient β	Standard error	Standardized coefficient β	p Value
Constant	337.244	1010.251	—	0.740
Gender	−559.766	379.247	−0.246	0.147
Body mass index	10.862	31.383	0.051	0.731
p_{cml}/W_B	−0.099	3.112	−0.005	0.975

Control group (n = 48): multiple linear regression coefficients of the normalized cumulative contact hip stress (p_{cml}/W_B) (kPa·years/N), body mass index (kg/m^2), and gender (male = 1) with WOMAC score at followup (mm) as the numeric dependent variable (model summary: $r = 0.246$; adjusted $r^2 < 0.001$; $p = 0.428$; power < 0.80).

Table 6. Normalized peak contact stress of control group did not predict WOMAC

Independent variable	Unstandardized coefficient β	Standard error	Standardized coefficient β	p Value
Constant	500.986	1383.114	—	0.719
Gender	−694.071	396.996	−0.305	0.088
Body mass index	8.943	31.382	0.042	0.777
Age	5.575	10.894	0.077	0.611
p_{max}/W_B	−198.124	253.819	−0.137	0.439

Control group (n = 48): multiple linear regression coefficients of the normalized peak contact hip stress (p_{max}/W_B) (kPa/N), age (years), body mass index (kg/m^2), and gender (male = 1) with WOMAC score at followup (mm) as the numeric dependent variable (model summary: $r = 0.288$; adjusted $r^2 < 0.001$; $p = 0.431$; power < 0.80).

2.85 MPa for the peak contact stress (36% sensitivity and 86% specificity), and 140 MPa-years for the cumulative contact stress (40% sensitivity and 84% specificity).

Discussion

Contact stresses are generally believed to influence whether a hip develops OA and using similar reasoning, various osteotomies have been proposed to reduce contact stresses and the risk of OA. To address these presumptions, we asked whether initially asymptomatic nonsubluxated dysplastic hips treated nonoperatively had higher long-term prevalence of hip OA, higher peak contact stress, and higher cumulative contact stress than a control population of normal hips. We then explored the predictive value of cumulative contact stress for late OA with the predictive value of earlier reported risk factors, including the Wiberg center-edge angle, peak contact stress, and body mass index.

We note several important limitations. The peak contact stress computed from anteroposterior radiographs corresponds to static one-legged stance only; it does not account for different body positions or dynamic activities. The cumulative contact stress computed as the product of age and the peak contact stress is therefore only a static approximation of the lifelong hip loading whereby stress is computed for the same body position in all patients. Conventional 2-D radiographic indices reflect only to a limited extent the true acetabular coverage of the femoral head, and methods have been developed that enable estimation of 3-D femoral head coverage (MRI, CT). However, modern 3-D imaging technology has not been in use long enough to enable prospective or retrospective studies with 20-year followup, and such long-term estimations will only be possible in the future. The small sample power in the group of normal hips could be attributed to small size, and a considerably larger sample would be required to confirm whether the predictive value of contact stress in this population is indeed so low. A small percentage of patients willing to participate in the study can cause potential selection bias, and we suspect the symptomatic patients would respond more likely than asymptomatic ones. However, because nearly half of the responding participants were asymptomatic at followup in both the study group and the control group, we presume the potential bias similarly affected both groups. As a result of small numbers of available radiographs at followup (40% of patients in the study group and 13% of cases in the control group), it was not possible to analyze the predictive value of p_{cml} and p_{max} by setting radiographic scores as a separate clinical end point for hip OA, and hip OA assessment was primarily based on WOMAC score. Radiographic appearance at followup with self-reported hip symptoms nevertheless correlated with the WOMAC score, the Kellgren-Lawrence score, and the joint space width measurements. We did not estimate an important issue—the patients' physical activity levels throughout their lifetime. However, we think higher OA incidence in dysplastic hips is not the consequence of any higher activity levels since patients with hip dysplasia were commonly advised in their teens not to engage in professions and activities that require constant or frequent standing, walking, or lifting. It is therefore reasonable to assume physical activity levels in the study group were even lower than in the control group. Besides, there is no clear evidence of lifelong standing, walking, or lifting in the population with normal hips is a risk factor for OA [17, 34]. Therefore, in this regard, there is no indication of any bias between the study and the control group.

Although the control group was older than the study group, we believe this strengthens rather than diminishes our conclusions, because one would generally anticipate a higher risk of OA in older patients, but we rather found the opposite. It could be speculated the long-term exposure to high contact stress only becomes detrimental after a certain threshold is reached. To examine such a possibility, one would have to compare dysplastic and normal hips with similar levels of cumulative stress. Although our control subjects were on average 17 years older, cumulative stress in the control group was still lower than the study group. Linear extrapolation shows the control hips would achieve the same cumulative stress level as dysplastic hips only at an average age of 90 years. Similar extrapolation suggested the dysplastic groups at a mean age of 68 (average age of the control group) would have a cumulative contact stress of 196 MPa-years and a mean WOMAC score 770 mm. By that time, the number of hips with WOMAC greater than 1200 mm (moderate to severe osteoarthritis) would increase from 12 (21%) to 17 (29%). Our data therefore suggest high cumulative stress had a positive predictive value for early hip OA, but low cumulative pressure does not guarantee the absence of hip OA in the sixth or seventh decade.

In previous studies, [17, 20, 23] hip dysplasia and OA have typically been analyzed as binary variables. However, the definitions of the two clinical entities have differed considerably among different authors. In two epidemiologic studies, the cutoff points for CE angle in assessment of hip dysplasia ranged from 20° to 30° [17, 20]; and the definition of hip OA in epidemiologic studies was based on self-reported assessment, clinically measured range of motion, radiographic scales, joint width measurements, or the risk of progression to THA. However, the choice of the particular outcome end point of hip OA substantially alters the results and conclusions [17, 23, 32]. We avoided this problem by analyzing the degree of increased contact stress

and the symptoms of hip OA as continuous variables. Our results are consistent with the findings of authors who have observed high positive predictive value of severe hip dysplasia for OA [16, 30]. We confirmed the CE angle does not predict the long-term occurrence of OA in the general population of normal hips [6], but it does predict the long-term clinical status of patients with mild hip dysplasia [21, 33]. We also confirmed another study [17] suggesting body mass index predicted hip OA; however, we found this effect limited to patients with hip dysplasia. Furthermore, our data on these mild dysplastic hips with followup to the average age of 51 years confirms two previous studies on dysplastic hips with followup to the average age of 31 years suggesting a correlation between cumulative contact stress and long-term hip status and no predictive value of the absolute values of contact stress [9, 27].

High cumulative contact stress predicted early hip OA in our study, and did so better than the Wiberg center-edge angle. The data also confirm spatial and temporal aspects of contact stress should be taken into account in OA risk assessment [3]. A relatively low negative predictive value of cumulative contact stress suggests that in addition to the spatial and temporal aspects of stress one should consider other factors influencing the development of OA.

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