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Author(s): McWilliams, D. F., Doherty, S. A., Jenkins, W. D., Maciewicz, R. A., Muir, K. W., Zhang, W. and Doherty, M.

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Mild acetabular dysplasia and risk of osteoarthritis of the hip: a case–control study

Daniel F McWilliams,[1] Sally A Doherty,[1] Wendy D Jenkins,[1] Rose A Maciewicz,[2] Kenneth R Muir,[3] Weiya Zhang,[1] Michael Doherty[1]

[1]Academic Rheumatology, University of Nottingham, Nottingham, UK
[2]AstraZeneca UK, Loughborough, UK
[3]Health Sciences Research Institute, University of Warwick, Coventry, UK

Correspondence to
Daniel McWilliams, Academic Rheumatology, Clinical Sciences Building, City Hospital, Nottingham NG5 1PB, UK; dan.mcwilliams@nottingham.ac.uk

ABSTRACT

Objective To determine whether mild variation in acetabular depth (AD) and shape is a risk factor for osteoarthritis (OA) of the hip.

Methods The unaffected contralateral hip of patients with unilateral hip OA was compared with hips of asymptomatic controls without hip OA, derived from the Nottingham Genetics Osteoarthritis and Lifestyle case–control study. Standardised anteroposterior x-rays of the pelvis were used to measure centre edge (CE) angle and AD. Cut-off points for narrow CE angle and shallow AD were calculated from the control group (mean $-1.96 \times SD$). The relative risk of hip OA associated with each feature was estimated using OR and 95% CI and adjusted risks were calculated by logistic regression.

Results In controls, both the CE angle and the AD were lower in the left hip than in the right hip. The CE angle related to age in both hips, and AD of the right hip was lower in men than in women. The contralateral unaffected hip in patients with unilateral hip OA had a decreased CE angle and AD compared with controls, irrespective of side. The lowest tertile of the CE angle in contralateral hips was associated with an eightfold risk of OA (aOR 8.06, 95% CI 4.87 to 13.35) and the lowest tertile of AD was associated with a 2.5-fold risk of OA (aOR 2.53, 95% CI 1.28 to 5.00). Significant increases in the risk of OA were also found as the CE angle and AD decreased.

Conclusion Constitutional mild acetabular dysplasia appears to increase the risk of hip OA.

INTRODUCTION

Osteoarthritis (OA) of the hip is a common cause of pain and disability in later life.[1] The causes of hip OA appear to be heterogeneous with constitutional, genetic and environmental risk factors influencing development of the disease. Heritability estimates for hip OA have been placed at 0.6 in women,[2] although the mechanisms that predispose to hip OA are unknown. Other known risk factors include severe developmental dysplasia of the hip that often presents in childhood,[3 4] occupational activities,[5] Legg–Calvé–Perthes disease,[6] obesity[5] and ageing.[5]

Mild hip dysplasia is a possible cause of adult-onset hip OA with a plausible biomechanical mechanism. Subtle differences in hip morphology between individuals may be asymptomatic, but could alter the biomechanical characteristics of the joint and contribute to OA. Geometric measures of hip morphology taken from x-rays, such as acetabular depth (AD) and centre edge (CE) angle, have been investigated previously in OA with varying results. Two systematic reviews by Lievense et al concluded that there was limited evidence for

dysplasia increasing incident hip OA,[7] mostly based upon findings of one study,[3 8] and limited evidence of no effect on progression,[9] mostly based on one other study.[10] Cross-sectional studies in Western European populations have supported an association between hip dysplasia and hip OA. Jacobsen found an association between hip OA and CE angle in the Osteoarthrosis substudy of the Copenhagen Heart Study.[11 12] In a 6-year follow-up study of 835 patients from the Netherlands, Reijman et al noted that the incidence of hip OA was related to both CE angle and AD.[13] Jessel et al used MRI to show that patients with acetabular dysplasia had less cartilage and were more likely to have hip OA.[14] Hip dysplasia has also been reported to cause new presentations of hip pain in one UK study,[15] but not in another study of adults in Copenhagen.[12] Some studies have also failed to link hip dysplasia and OA, including a radiographic assessment of 1315 men[16] and another of 393 women.[4] Geographical variations have been reported in hip OA and its relationship to mild dysplasia. Japanese,[17 18] African,[19 20] Turkish[21] and Sami[22] populations appear to have different prevalences of hip OA and acetabular dysplasia, and different associations than in Western Europeans. A caveat of many cross-sectional studies is that the measures of dysplasia are often undertaken on hips with established OA and could be secondary to OA and remodelling rather than being a pre-existing primary risk factor for its development.

Our group reported recently that non-spherical femoral head shape and shallow neck shaft angle were both more common in the contralateral unaffected hip of patients with unilateral hip OA than in non-OA controls in the Genetics of Osteoarthritis and Lifestyle (GOAL) study group.[23] The unaffected hip is likely to be representative of the individual's joint morphology before the onset of OA, and a study of the contralateral unaffected hip is a useful strategy for estimating risks of development of OA in cross-sectional studies. GOAL is a hospital-based case–control study primarily designed to investigate genetic and environment effects in patients with severe OA of the knee and/or hip sufficient to warrant consideration of joint replacement surgery.[23–26] Young patients (<45 years) and those with known hip disease or dysplasia identified in childhood were excluded from GOAL, making it suitable for analysis of morphological factors that may increase the risk of common adult-onset hip OA. The objective of this study was to determine in the GOAL cohort[23] whether acetabular dysplasia may be a risk factor for clinically severe adult-onset hip OA.

METHODS

Cases and controls

Participants were men and women aged >45 years originally recruited in Nottingham, UK, between 2002 and 2006 to participate in the GOAL case–control study. Recruitment details have been published previously.[23–26]

Patients with hip OA were recruited from hospital orthopaedic surgery lists (current and from the previous 5 years) and from a rheumatology OA clinic. All cases had been referred to the hospital with symptomatic clinically severe hip OA and most had undergone unilateral or bilateral total hip replacement within the previous 5 years. Subjects were excluded from the study if they had other major arthropathy (eg, rheumatoid arthritis or ankylosing spondylitis), Paget's disease of bone affecting the pelvis or femur, overt childhood hip disease (eg, Legg-Calvé-Perthes, slipped femoral epiphysis or severe acetabular dysplasia), polio, total hip replacement due to trauma or avascular necrosis of the femoral head or a terminal illness. All control subjects were recruited from lists of people who had been referred to hospital for intravenous urography (IVU) within the last 5 years and had responded to a written invitation to participate in GOAL. Individuals who had no radiographic evidence of hip OA on their screening IVU radiograph and who had no hip or knee symptoms or any of the exclusions listed above for cases were invited to take part in the study (and undergo further standardised radiography) in writing and with a telephone call which provided further information and screening for exclusions. Cases and controls were further characterised by interview at home and examination at Academic Rheumatology, Nottingham.

Height and weight were measured to calculate body mass index (BMI). The presence of interphalangeal nodes was determined clinically by established methods,[27 28] and a nodal phenotype was defined as Heberden and/or Bouchard nodes that affected at least two rays of each hand. A history of hip injury was defined as any self-reported significant hip injury (fracture, severe trauma requiring medical attention, immobilisation or crutches for ≥2 weeks) due to occupation, sport or leisure activities during the subject's lifetime. Previous hip pain on most days for at least 1 month was also reported.

Radiographic assessment of hips

For cases that had undergone total hip replacement, the preoperative x-ray was copied using Hipax Dicom digitising software (Hipax, Vorstetten, Germany) to enable scoring. In cases who had not undergone total hip replacement and in all controls, new x-rays were obtained unless the subject had undergone radiography of the pelvis (using the same standardised method) not more than 2 years previously.

Anteroposterior views of the pelvis were obtained using a standardised protocol[23] with the participant supine and feet internally rotated 10° (70 kV exposure, broad focus, 100 cm focus-to-film distance). A single observer (SAD) measured the minimum joint space width (JSW) in mm. A second observer (WDJ) measured the CE angle and AD according to the methods of Wiberg[29] and Murray[30] (figure 1). Each film was scanned and written to CD using the Hipax Dicom 4.2 x-ray image processor. CD images were read using the Hipax Private Health disc image viewer which enables straight line measurements between visually determined points to an accuracy of 0.01 mm and measurement of angles to an accuracy of 1° (manufacturer precision). Repeatability was estimated from 25 pelvic x-rays (7 non-OA and 18 hip OA cases) which were chosen
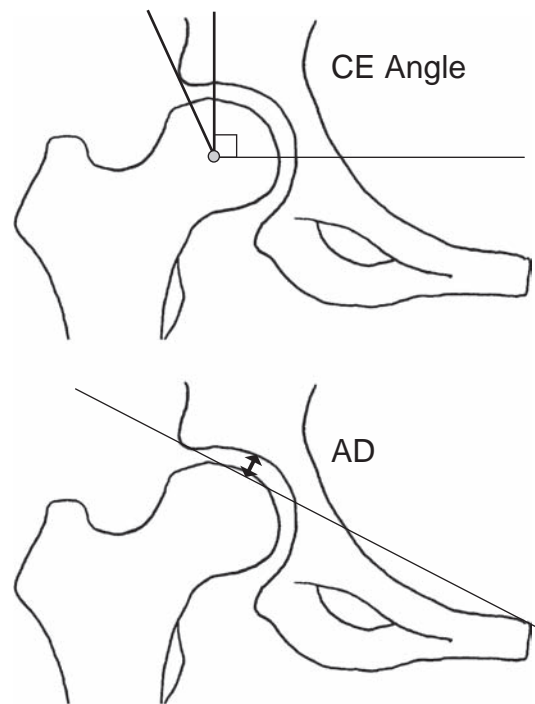

Figure 1 Measures of acetabular depth (AD) and centre edge (CE) angle. The diagram shows the guidelines used during measurement of AD and CE angle on hip x-rays.

from randomly assigned study numbers and assessed twice by each observer (WDJ/SAD). The 25 pelvic x-rays were comparable to the numbers that demonstrated repeatability in our previous studies, which were 24 pelvic[23] and 30 hand[25] x-rays. Repeat measurements were performed by each observer (JSW: SAD; CE angle and AD: WDJ) and the cases and controls were mixed together in the order of the randomly assigned study number. The x-rays were examined after the GOAL study sample collection had been completed and without a long time lag between each session of measurement. Radiographic hip OA was defined as a JSW ≤2.5 mm,[23] and subjects in the case group with JSW ≤2.5 mm in one hip only were classified as having unilateral hip OA. Subjects in the control group with JSW >2.5 mm in both hips were retained as non-OA controls for this study.

Statistical analysis

Repeatability of CE angle, AD and JSW were examined with intraclass coefficients (ICCs). Symmetry of these measures between left and right hips was examined in the control group. The prevalence of abnormal CE angle and AD was estimated using the cut-off values determined by mean–1.96×SD of each measure in the control population. In addition, we also used the standard thresholds found in the literature (CE angle 20°, AD 9 mm)[3 12 16] for comparison purposes. The comparison between cases and control was undertaken separately for right and left hips. Differences between continuous measures were examined using the Student t test or paired t test and correlations were examined with the Pearson correlation coefficient. Dichotomous data were analysed with a χ^2 test, or the McNemar test when comparing two dichotomous paired variables. Logistic regression analysis was used to adjust confounding factors, hips (right/left), age (tertiles), gender (female/male) and BMI (>25/≤25). Only one hip was used from controls on the basis of a random selection between right and left for each individual in the control group.

Extended report

RESULTS

Demographic characteristics

The demographic characteristics of the study group are shown in table 1. Cases of unilateral hip OA (n=566) from the GOAL case–control study hip OA group were selected using the above criteria (minimum JSW ≤2.5mm)[23] and non-OA controls (n=1108) had no radiographic OA in either hip and no current hip pain; 7% (78/1108) reported previous hip pain. The unilateral hip OA group comprised 232 (41%) right hips and 334 (59%) left hips and had a higher mean age and BMI as well as more reported hip injuries and Heberden/Bouchard nodes. Neither the AD nor the CE angle (on either side) differed significantly between control participants with right or left dominant hands (data not shown). The response rates from the eligible populations of cases and controls were 68% and 65%, respectively.

Repeatability of measurements

The ICC was used to assess the intraobserver variability of the CE angle and AD. Both measures were found to have good repeatability (CE angle: ICC 0.76, 95% CI 0.50 to 0.90; AD: ICC 0.79, 95% CI 0.55 to 0.91), as did minimum JSW (ICC 0.98, 95% CI 0.95 to 0.99).

Characterisation and asymmetry in the non-OA controls

The non-OA control group was used to examine the symmetry of normal hip morphology for CE angle and AD. Mean CE angle and AD were significantly lower in right hips than in left hips of the non-OA controls (table 2). The mean difference between right and left CE angles was 0.81° (p<0.01) and between ADs was 0.36 mm (p<0.01). Right and left hips were therefore analysed separately to control for the asymmetry. The CE angles were similar between genders while the right hips of men had lower AD than the right hips of women (13.25 mm vs 13.63 mm, p=0.038; table 2). The CE angle was strongly correlated with AD (right: r=0.71, p<0.001; left: r=0.70, p<0.001) and very weakly related to age (right: r=0.092, p=0.002; left: r=0.091, p=0.002). However, AD showed no relationship with age (right: r=−0.04, p=0.249; left: r =−0.03, p=0.381).

According to mean −1.96×SD in the control group, the thresholds for CE angle were 24.6° for the right hip and 25.7° for the left hip, while those for AD were 7.47 mm for the right hip and 7.83 mm for the left hip. The proportions of non-OA controls classified as 'dysplastic' by exceeding the limits of our criteria or standard criteria found in the literature (CE angle 20°, AD 9 mm) are shown in table 2.

Risk of hip OA

The mean CE angle was significantly lower in the unaffected contralateral hips of participants with unilateral hip OA than in controls (mean±SD 33.4±7.5 vs 37.1±6.4, p<0.001 for right hip; 33.4±8.1 vs 37.9±6.2, p<0.001 for left hip).The mean AD was also significantly lower in contralateral hips (mean±SD 11.7±3.0 vs 13.4± 3.0, p<0.001 for right hip; 12.1±3.4 vs 13.8±3.0, p<0.001 for left hip). Dysplastic CE angles were recorded in 11.2% (26/232) of right contralateral hips and 16.2% (54/334) of left contralateral hips and in 2.1% (23/1108) right control hips and 2.2% (24/1108) left control hips. Dysplastic ADs were found in 4.7% (11/232) of right contralateral hips and in 8.1% (27/334) of left contralateral hips. When compared with controls, univariate analysis indicated significantly higher risks of hip OA associated with dysplastic CE angle and AD in the contralateral hip in both women and men (table 3).

The results were supported by our adjusted analyses where both CE angle and AD were analysed together and remained as risk factors for hip OA after adjustment for right/left side, age, gender and BMI (figure 2). Compared with controls, an approximate eightfold risk was conferred by CE angle and a 2.5-fold risk by AD (table 4). Hip dysplasia was associated with an increased risk of hip OA (CE angle: aOR 1.76, 95% CI 1.48 to 2.08, p trend <0.001; AD: aOR 1.29, 95% CI 1.08 to 1.53, p trend =0.004).

Table 1 Demographic characteristics of the study groups

	Non-OA controls	Unilateral hip OA
N	1108	566
Age	64.2±8.4	67.4±7.2**
Women (%)	46.3	47.9
BMI	27.5±4.6	29.3±5.0**
Finger nodes (%)	12.0	26.1**
Hip injury (%)	2.0	7.8**

Mean±SD or prevalences are shown.
**p <0.01.
BMI, body mass index; OA, osteoarthritis.

Table 2 Characterisation of hips in the two study groups

	Non-OA controls				Unaffected contralateral hip			
	Right		Left		Right		Left	
	Women	Men	Women	Men	Women	Men	Women	Men
CE angle								
N	513	595	513	595	101	131	170	164
Mean±SD	37.4±6.2†	36.8±6.5††	38.1±6.2†	37.7±6.2††	33.96±7.85	33.03±7.18	34.22±8.44	32.51±7.71
1.96×SD threshold (%)	1.4	2.7	1.4	2.5	9.9	12.2	16.5	15.9
20° Threshold (%)	0.8	0.7	0.0	0.2	3.0	6.9	3.5	4.9
Acetabular depth								
Mean±SD	13.6±3.0*	13.3±3.1*	13.9±3.1	13.7±3.0	12.02±3.25	11.52±2.87	12.16±3.47	12.02±3.25
1.96×SD threshold (%)	1.4	2.0	1.2	1.5	4.1	5.3	6.5	9.8
9 mm Threshold (%)	5.3†	7.1†	3.3†	4.7†	16.8	19.1	15.3	17.7

The right and left hips of non-OA controls show a small amount of asymmetry of CE angle and AD. The hip morphologies of women and men were mostly similar. Mean and SDs of each measure are presented with the prevalence of 'dysplastic' hips according to our threshold and standard criteria from the literature. Comparisons of right versus left continuous measures were performed using the paired t test, while women were compared with men using the Student t test. Comparisons of right versus left 'dysplastic' hips were performed using the McNemar test and women were compared with men using the χ^2 test.
*p<0.05 women vs men.
†p<0.05, ††p<0.01 right vs left hips within the same sex.
OA, osteoarthritis.

Table 3 Mild constitutional dysplasia of the contralateral hip and risks of OA

| Measure | Threshold | Univariate analysis: OR (95% CI) | | | |
| | | Right | | Left | |
		Women	Men	Women	Men
CE angle	Mean−1.96×SD	6.90 (2.37 to 20.14)**	7.61 (3.29 to 17.61)**	24.65 (5.79 to 105.00)**	7.56 (3.20 to 17.86)**
	20°	3.96 (0.65 to 24.08)	NA	NA	14.72 (1.82 to 118.75)**
AD	Mean−1.96×SD	2.65 (0.65 to10.80)	3.19 (1.16 to 8.75)*	7.81 (1.69 to 36.12)**	12.27 (2.73 to 55.08)**
	9 mm	4.20 (1.93 to 9.15)**	3.42 (1.82 to 6.41)**	6.32 (2.68 to 14.92)**	4.92 (2.44 to 9.95)**

Univariate analysis of risks (OR (95% CI)) of hip OA determined using thresholds in unaffected contralateral hip acetabular morphology measures.
*p<0.05, **p<0.01.
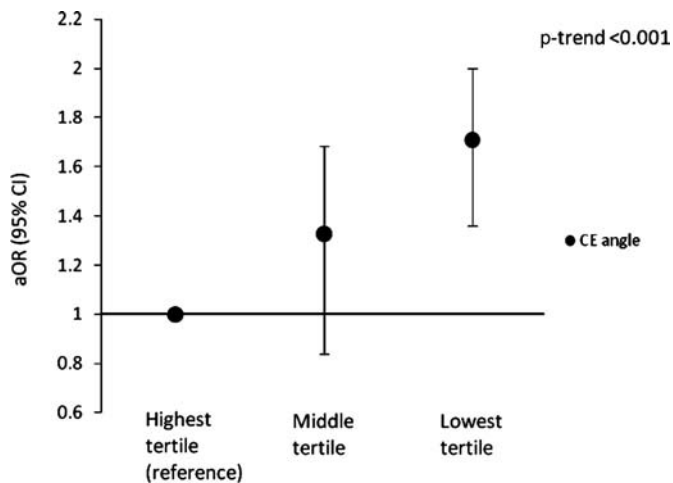AD, acetabular depth; CE, centre edge; OA, osteoarthritis.

Figure 2 Increasing risk of hip osteoarthritis (OA) with changes in the morphology of the unaffected contralateral hip. The risk of hip OA is increased as the tertiles for centre edge (CE) angle decrease. OR, 95% CI and p trend were adjusted for age, gender, body mass index, acetabular depth and right/left hip.

Table 4 Increased risk of hip OA with low AD and CE angle

	Threshold	aOR (95% CI)
CE angle	Mean−1.96×SD	8.06 (4.87 to 13.35)**
	20°	10.05 (2.89 to 35.01)**
AD	Mean−1.96×SD	2.53 (1.28 to 5.00)**
	9 mm	3.61 (2.47 to 5.27)**

Increased risk of hip OA, after adjustment for possible confounders, was associated with decreasing AD and CE angle. The risks for hip OA were associated with falling below the threshold for CE angle or AD in the contralateral unaffected hip. Logistic regression was adjusted for left/right hip, AD, CE angle, age, gender and body mass index.
**p<0.01
AD, acetabular depth; CE, centre edge; OA, osteoarthritis.

DISCUSSION

We found that CE angle and AD at the lowest extreme of the normal range in the unaffected contralateral hip were risk factors for OA. Also, as measures of CE angle and AD decreased, the risk of hip OA increased. Because hip disease presenting in childhood or young adulthood, including severe hip dysplasia, was excluded from the GOAL cohort, it appears that mild hip dysplasia may be a constitutional risk factor for some adults who develop clinically severe hip OA as adults. In the GOAL study population, other mild differences in morphology on the femoral side of the joint have also been found to be more common in the contralateral hips of those with unilateral hip OA,

suggesting that morphological variation may be a biomechanical risk factor for 'common' OA presenting in adults.[23]

Our study supports the finding that mild hip dysplasia is associated with hip OA in Western European populations.[3 11 13] Most literature relating to CE angle uses the thresholds 25° or 20° to define dysplasia, and a 9 mm threshold for AD is often used.[8 16] Owing to the small asymmetry of the right and left non-OA control hips, we calculated our own thresholds to define dysplasia from the non-OA controls, as previously described.[23] Right and left thresholds were numerically similar, implying that an approximation, or the mean of both, would have been adequate for our purposes. All hip x-rays were measured in a standardised way (as detailed earlier[23]), so this small asymmetry within the non-OA control group appears to be real, although it is possible that measurement error/variability could be responsible. The dominant side of participants is another plausible explanation, although hip dysplasia did not differ between those in the control group with right or left handedness. Our criteria for dysplastic CE angles were very close to the 25° threshold used in many studies, and we also included data using a 20° threshold for comparison purposes. Interestingly, the 20° threshold excluded almost all control hips, suggesting that it is either extremely specific for dysplasia or may exclude positives. This resulted in variable ORs during univariate analysis, which are unlikely to be reliable. Instead, the thresholds closer to 25° were superior for calculating risks. The criteria we used for acetabular dysplasia were considerably less than the 9 mm standard used in other studies, but both yielded similar findings. The use of the standard thresholds of 25° for CE angle and 9 mm for AD appear to be justified, and their use in future studies seems appropriate.

The correlation found between age and CE angle has also been reported in other studies where hip OA in younger patients was associated with lower CE angle.[16] The relationships between CE angle and AD with age appeared too weak to be clinically significant, despite their statistical significance. This was confirmed in our analyses where adjustments performed for age did not explain the association of hip morphology measurements with OA. The correlation between AD and CE angle has been reported in other radiographic studies of the hip,[16] and it is not surprising that they are related. Adjusting for CE angle and AD in the same logistic regression model showed that both are independent risk factors, despite their correlation. The risks conferred by AD were much closer to unity than those conferred by CE angle. Gender differences between hips were not especially prominent, in contrast to our previous study of hip morphology,[23] with only right AD differing in the non-OA control group (table 3). Our univariate analyses separated gender and our multivariate analysis adjusted for gender, but it did not explain the risks for OA in this study.

This case–control study of patients with severe hip OA recruited participants from hospital lists with clinically severe

OA. End stage cases increase the difficulty in investigating whether hip morphology is part of a developing OA or a preexisting constitutional risk factor. By focusing on the contralateral normal hip in people with unilateral hip OA, our findings provide evidence that CE angle and AD contribute towards causation. However, it is possible that these apparently normal asymptomatic hips might also be progressing towards OA themselves. Patients with unilateral hip OA may alter gait and the way they perform domestic/work activities to compensate for pain and limited function, meaning that the contralateral hips could be undergoing some degree of remodelling. Only a prospective study will be able to determine causes and effects of hip morphology on OA with certainty. However, hip OA may show some asymmetry with respect to JSW, which is narrower in left hips than in right hips of patients with hip OA,[31] so it is possible that morphological variants may favour one side over the other and be asymmetrical, although the determinants for this remain unclear. In this case–control hospital-based study, those invited to participate as controls might be more likely to decline. However, response rates from the eligible populations were good for both cases and controls, minimising potential biases. It is unknown whether any controls were referred for IVU because of hip pain. Previous pain in or around the hip region was reported in 7% of controls (who were aware that the study investigated hip OA), although those who had current hip or knee symptoms or radiographic hip OA were excluded. A single-view plain x-ray is relatively insensitive for detecting mild OA changes, so some controls may have had mild early OA. However, any minor changes in the controls due to hip OA would be expected to reduce rather than inflate the estimate of risk conferred by CE angle and AD.

In summary, this study supports the hypothesis that mild hip dysplasia is a constitutional risk factor for hip OA in adults. The measurement of changes in CE angle and AD may be useful to include as a potential risk factor in future prospective studies of hip OA.

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Mild acetabular dysplasia and risk of osteoarthritis of the hip: a case –control study

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