Adult Hip Dysplasia - Prevalence and Consequence

Vinge, Rebecka

2025

Document Version:
Publisher's PDF, also known as Version of record

Adult Hip Dysplasia

Prevalence and Consequence

REBECKA VINGE
DEPARTMENT OF CLINICAL SCIENCES LUND | FACULTY OF MEDICINE | LUND UNIVERSITY

Adult Hip Dysplasia – Prevalence and Consequence

Adult Hip Dysplasia

Prevalence and Consequence

Rebecka Vinge

LUND
UNIVERSITY

DOCTORAL DISSERTATION

Doctoral dissertation for the degree of Doctor of Philosophy (PhD) at the Faculty of Medicine at Lund University to be publicly defended on the 28th of May 2025, at 09.00 in Segerfalksalen, BMC, Lund

Faculty opponent
Professor Young-Jo Kim
Harvard Medical School, Boston Children´s Hospital, Boston, USA

Organization: LUND UNIVERSITY **Date of issue:** 28th of May 2025

Document name: Doctoral dissertation **Author:** Rebecka Vinge

Title and subtitle: Adult Hip Dysplasia - Prevalence and Consequence

Abstract:

Background: Adult hip dysplasia is one of the few potentially modifiable risk factors for hip osteoarthritis (OA). However, the prevalence of hip dysplasia and the strength of its association with OA are not fully understood.

Aims and methods:

I. To determine the prevalence of hip dysplasia in a Swedish population and investigate whether the condition was mentioned in radiology reports. The center edge angle was measured, and the radiology reports were reviewed for 1870 individuals aged 20–70 years who had undergone an anteroposterior pelvic radiograph in Malmö, Sweden, between 2007–2008 and had no other hip pathology.

II. To explore the development of radiographic hip OA in individuals with unilateral hip dysplasia identified in Study I and compare OA outcomes between dysplastic and contralateral non-dysplastic hips. A longitudinal review of the radiographic records of 50 individuals with unilateral hip dysplasia and available follow-up imaging was conducted to assess OA incidence, time to detection of OA, and joint space width.

III. To examine the relationship between hip dysplasia and the risk of developing radiographic hip OA at several time points using different definitions of hip dysplasia based on lateral and/or anterior undercoverage. The association was explored at the 2-, 5-, 8- and 10-year follow-ups of the CHECK study, with between 1169 to 1262 included hips depending on the follow-up time point.

IV. To examine the long-term association between hip dysplasia and clinically relevant hip OA. The outcome was defined by an expert diagnosis incorporating both clinical and radiographic data from the 5- to 10-year follow-ups of the CHECK study, and the analysis included 468 hips.

Results and conclusions:

In our Swedish study population, 5.2% had hip dysplasia, with most cases overlooked by radiologists, emphasizing the need for increased awareness of the condition. We found no evidence that radiographic OA developed earlier or more frequently in dysplastic hips compared to contralateral non-dysplastic hips of individuals with unilateral hip dysplasia, suggesting similar radiographic monitoring for both. The strongest and most consistent associations with radiographic OA development were observed when both anterior and lateral projections were used to define hip dysplasia. The highest increased risk (2.5-fold) was found at the 2-year follow-up. All associations weakened over time and disappeared by the 10-year follow-up. These findings highlight the importance of early detection of hip dysplasia, and incorporation of anterior undercoverage assessment, to identify individuals at higher risk of radiographic OA development. Lastly, individuals with hip dysplasia were found to have a nearly three times increased risk of clinically relevant hip OA after 10 years of follow-up, underscoring the need for a clinically relevant definition of OA in future research on hip dysplasia.

Key words: Hip dysplasia, Center edge angle, Osteoarthritis, Total hip replacement.

Language: English **Number of pages**: 76

ISSN:1652-8220 **ISBN:** 978-91-8021-716-3

Adult Hip Dysplasia

Prevalence and Consequence

Rebecka Vinge

LUND
UNIVERSITY

Supervisors

Professor Carl Johan Tiderius, MD, PhD

Professor Cecilia Rogmark, MD, PhD

Assistant Professor Jos Runhaar, PhD

Lunds University, Faculty of Medicine, Department of Clinical Sciences

ISBN 978-91-8021-716-3
ISSN 1652-8220

Printed in Sweden by Media-Tryck, Lund University
Lund 2025

To Fredrik and Vendela

Table of Contents

List of publications

Paper I

Leide R, Bohman A, Wenger D, Overgaard S, Tiderius CJ, Rogmark C. Hip dysplasia is not uncommon but frequently overlooked: a cross-sectional study based on radiographic examination of 1,870 adults. Acta Orthopaedica. 2021; 92 (5): 575-580.

Paper II

Vinge R, Rogmark C, Wenger D, Overgaard S, Tiderius CJ. Osteoarthritis development in untreated individuals with hip dysplasia – a longitudinal radiographic study. Manuscript.

Paper III

Riedstra NS, **Vinge R,** Herfkens J, Eygendaal D, Bierma-Zeinstra SMA, Runhaar J, van Buuren MMA, Agricola R. Acetabular dysplasia and the risk of developing hip osteoarthritis at 2,5,8 and 10 years follow-up in a prospective nationwide cohort study (CHECK). Seminars in Arthritis and Rheumatism. 2023;60: 152194.

Paper IV

Vinge R, Riedstra N, Tiderius CJ, Bierma-Zeinstra S, Agricola R, Runhaar J. Hip dysplasia as risk factor for clinically relevant and radiographic hip osteoarthritis: 10-year results from the CHECK cohort. Rheumatology (Oxford). 2025;64(1):149-155.

Abbreviations

ACR	American College of Rheumatology
ACEA	Anterior center edge angle
AIA	Acetabular index angle
AP	Anteroposterior
ASM	Active shape model
CHECK	Cohort Hip and Cohort Knee
CT	Computed tomography
DDH	Developmental dysplasia of the hip
FAI	Femoroacetabular impingement
FADIR	Flexion-adduction-internal-rotation
FOI	Foramen obturator index
FP	False-profile
GEE	Generalized estimating equations
GP	General practitioner
ICC	Intraclass correlation coefficient
JSN	Joint space narrowing
JSW	Joint space width
K&L	Kellgren and Lawrence
LCEA	Lateral center edge angle
MRI	Magnetic resonance imaging
NIH	Neonatal instability of the hip
OA	Osteoarthritis
PAO	Periacetabular osteotomy
ROM	Range of motion
SSM	Statistical shape model
THR	Total hip replacement

Abstract

Background

Adult hip dysplasia is one of the few potentially modifiable risk factors for hip osteoarthritis (OA). However, the prevalence of hip dysplasia and the strength of its association with OA are not fully understood.

Aims and methods

Studies I and II are based on a retrospective review of the radiographic records from the regional healthcare system in Skåne, Sweden. Studies III and IV are based on the CHECK study, a prospective cohort study with 10 years of follow-up involving 1002 Dutch participants aged 45–65 years, all of whom had recently developed hip and/or knee pain.

I. To determine the prevalence of hip dysplasia in a Swedish population and investigate whether hip dysplasia was mentioned in radiology reports. The center edge angle was measured bilaterally in individuals aged 20–70 years who had undergone an anteroposterior pelvic radiograph in Malmö, Sweden, between 2007–2008 and had no other hip pathology. Radiology reports were then reviewed. The study included 1870 participants.

II. To explore the development of radiographic hip OA in participants with unilateral hip dysplasia identified in Study I and compare OA outcomes between dysplastic and contralateral non-dysplastic hips. A longitudinal review of the radiographic records of 50 individuals with unilateral hip dysplasia and available follow-up imaging was conducted assessing OA incidence, time to detection of OA and minimum joint space width.

III. To examine the relationship between hip dysplasia and the risk of developing radiographic hip OA at various time points and assess how the strength of the association changes over time. We also aimed to investigate the relationship between hip dysplasia and radiographic hip OA using different definitions of hip dysplasia, based on measurements of the center edge angle on lateral and/or anterior projections. Hip dysplasia was defined in three ways: lateral undercoverage, anterior undercoverage, and a combination of both. The association was explored at 2-, 5-, 8- and 10-year follow-ups, with 1169 to 1262 included hips depending on the follow-up time point.

IV. To examine the long-term association between hip dysplasia and clinically relevant hip OA. The outcome was defined by an expert diagnosis incorporating both clinical and radiographic data from the 5- to 10-year follow-up, and the study included 468 hips.

Results and conclusions

I. The prevalence of hip dysplasia was 5.2% in our Swedish study population, with only 7% of cases mentioned in the radiology reports. These findings underscore the need for increased awareness of hip dysplasia within Swedish healthcare.

II. There was no evidence that radiographic OA developed earlier or more frequently in dysplastic hips compared to contralateral non-dysplastic hips of individuals with unilateral hip dysplasia, suggesting that similar radiographic monitoring for OA development is appropriate for both.

III. Not all definitions of hip dysplasia were associated with development of radiographic hip OA at all time points. The strongest and most consistent associations were observed when hip dysplasia was defined by both anterior and lateral undercoverage. The highest increased risk (2.5-fold) was observed at the 2-year follow-up. Statistically significant associations at the 2- and 5-year follow-ups weakened by the 8-year follow-up and eventually disappeared by the 10-year follow-up. These findings highlight the importance of early detection of hip dysplasia, and incorporating assessment of anterior undercoverage, to identify individuals at higher risk of radiographic OA development.

IV. Individuals with hip dysplasia had nearly a threefold increased risk for clinically relevant hip OA, as assessed by experts using both radiographic and clinical data from the 5- to 10-year follow-ups. These findings emphasize the importance of using clinically relevant OA definitions in future hip dysplasia research.

Populärvetenskaplig sammanfattning

Höftdysplasi är ett tillstånd där höftledens ledskål inte täcker lårbenshuvudet tillräckligt, vilket leder till instabilitet och minskad belastningsyta i leden.

Hos nyfödda baseras diagnosen på höftledens stabilitet och kallas developmental dysplasia of the hip (DDH). Alla nyfödda barn i Sverige undersöks av barnläkare för att kontrollera om det finns någon instabilitet i höftlederna. Vid misstanke om instabilitet genomgår barnet ytterligare en undersökning av ortopedläkare, kompletterad med ultraljudsundersökning. Om DDH diagnostiseras, inleds behandling med en skena som håller höfterna i en ”grodposition”, vilket främjar normal utveckling av ledskålen. Om DDH upptäcks och behandlas tidigt får nästan alla drabbade barn normala höfter i vuxen ålder.

Höftdysplasi kan även förekomma hos vuxna som inte haft någon känd DDH i barndomen. Diagnosen ställs då via röntgenbilder av bäckenet, där center edge-vinkeln mäts för att bedöma i vilken utsträckning som ledskålen täcker ledhuvudet. Det är fortfarande oklart om höftdysplasi hos vuxna är en form av DDH som missades vid nyföddhetsscreeningen eller om det är ett tillstånd som utvecklats i en senare fas av skelettmognaden. I Norge och Danmark har man funnit att höftdysplasi förekommer hos 3% respektive 5% av den vuxna befolkningen. I Sverige får höftdysplasi relativt sett begränsad uppmärksamhet inom sjukvården, och förekomsten har inte studerats.

Flera studier har visat att höftdysplasi hos vuxna ökar risken för utveckling av höftartros, men kunskapen om sambandet är fortfarande begränsad. Tidigare forskning har enbart studerat sambandet med radiologisk artros, trots att artros inom sjukvården diagnostiseras både utifrån röntgenfynd, symptom och fynd vid klinisk undersökning. Eftersom höftdysplasi är en av få potentiellt påverkbara riskfaktorer för höftartros, är det av stort intresse att förstå sambandet bättre.

Syftet med denna avhandling är att undersöka förekomsten och konsekvenserna av höftdysplasi hos vuxna. Studie I och II baseras på retrospektiv granskning av röntgenarkivet i Region Skåne, medan Studie III och IV bygger på en stor prospektiv kohortstudie av holländska individer i åldern 45–65 år som nyligen drabbats av höft- och/eller knäsmärta.

Studie I

I Studie I granskade vi bäckenröntgenbilder tagna i Malmö under åren 2007–2008 av individer i åldrarna 20–70 år för att undersöka förekomsten av höftdysplasi. Vi mätte center edge vinkeln för att identifiera höftdysplasi och granskade röntgenutlåtandet för att se om tillståndet hade påpekats av röntgenläkaren. Höftdysplasi förekom i en eller

båda höfterna hos 5% av studiedeltagarna, och bara 7% av dessa fall hade noterats i röntgenutlåtandet.

Studie II

I Studie II undersökte vi individer med ensidig höftdysplasi som hade identifierats i Studie I. Vi inkluderade de som hade genomgått ny bilddiagnostik av bäckenet sedan 2007–2008 och granskade om radiologisk höftartros hade utvecklats. Vi fann inga bevis för att individer med ensidigt höftdysplasi utvecklade radiologisk artros tidigare eller oftare i sin dysplastiska höft jämfört med sin icke-dysplastiska höft.

Studie III

I Studie III undersökte vi sambandet mellan höftdysplasi och radiologisk höftartros vid fyra olika tidpunkter, baserat på tre olika definitioner av höftdysplasi. Traditionellt definieras höftdysplasi av att ledskålen inte ger tillräcklig täckning av lårbenshuvudet i sidled, vilket mäts på en röntgenbild tagen framifrån. Men även den främre delen av ledskålen kan ge otillräcklig täckning, vilket mäts på en röntgenbild tagen från sidan. Vi definierade höftdysplasi baserat på nedsatt täckning i följande riktningar: i sidled, i den främre delen av ledskålen, samt i båda riktningar. Det starkaste och mest konsekventa sambandet mellan höftdysplasi och radiologisk artros fanns när höftdysplasi definierades av nedsatt täckning i båda riktningar. Risken för radiologisk artrosutveckling var som högst vid 2-årsuppföljningen, då risken var 2,5 gånger högre. Sambandet försvagades över tid och var inte längre statistiskt signifikant vid 10-årsuppföljningen för någon av definitionerna av höftdysplasi. Våra resultat stämmer överens med tidigare studier som tyder på att höftdysplasi framför allt ökar risken för radiologisk artrosutveckling i ung vuxen ålder.

Studie IV

I Studie IV granskade vi sambandet mellan höftdysplasi och kliniskt relevant artros. I stället för att enbart bedöma artros utifrån röntgenbilder, fick artrosexperter även tillgång till information om symptom och kliniska undersökningsfynd från flera års upprepade uppföljningar, fem till tio år från studiestart. Baserat på denna omfattande information ombads experterna att avgöra om det förelåg klinisk relevant artros. Vi fann en trefaldig ökning av risken för kliniskt relevant artros hos individer med höftdysplasi.

Slutsatser

Sammanfattningsvis visar våra resultat att höftdysplasi förekommer hos drygt 5% av den vuxna befolkningen i Sverige och att det ofta missas av röntgenläkare, vilket understryker behovet av ökad medvetenhet om tillståndet inom svensk sjukvård.

Sambandet mellan höftdysplasi och radiologisk höftartros verkar inte lika starkt som tidigare rapporterat. Vi fann inga bevis för att radiologisk artros utvecklas tidigare eller oftare i dysplastiska höfter jämfört med normala höfter hos individer med ensidig höftdysplasi.

För medelålders individer med höftdysplasi kan risken för att utveckla radiologisk artros vara mer än dubbelt så stor på kort sikt, men sambandet försvagas och försvinner på lång sikt. Sambandet förstärks på kort sikt om höftdysplasi förekommer i den främre delen av ledskålen, utöver den traditionella definitionen av nedsatt täckning i sidled. Dessa resultat belyser att tidig identifiering av höftdysplasi, samt kartläggning av höftdysplasins utbredning i den främre delen av ledskålen, kan hjälpa till att identifiera individer med högre risk för artrosutveckling.

Trots att vi inte såg någon ökad risk för radiologisk artrosutveckling vid långtidsuppföljning, fann vi en trefaldigt ökad risk för kliniskt relevant artros. Detta belyser vikten av att använda en kliniskt relevant definition av höftartros när sambandet mellan höftdysplasi och artros studeras, eftersom det annars kan underskattas eller förbises.

Introduction

Hip dysplasia

Hip dysplasia is a condition characterized by a shallow acetabulum, which, compared to a normal acetabulum is globally deficient in both shape and orientation (1). This results in acetabular undercoverage of the femoral head (2) and affects the stability and biomechanics of the hip joint (3). The condition ranges from mild dysplasia, where only the acetabulum is affected, to severe dysplasia, where the deficient acetabulum is accompanied by deformities of the proximal femur (4). In cases of severe instability, the dysplastic hip joint can become subluxated or dislocated.

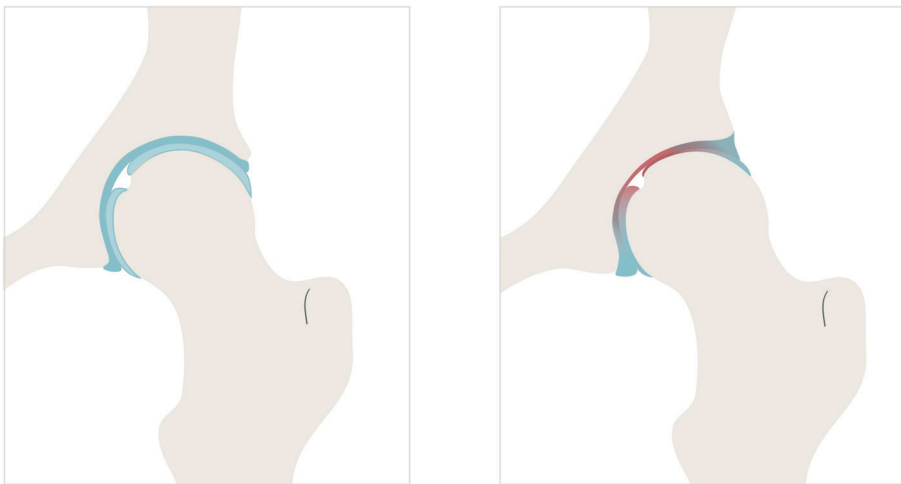

Figure 1: Visualization of hip dysplasia.
The left illustration depicts a hip joint with normal acetabular coverage of the femoral head, while the right illustration shows a dysplastic hip joint with insufficient coverage of the femoral head. Illustrations by Katarina Jandér, created under the author's instructions.

Terminology

There are various terms used to describe hip dysplasia, and the diversity of these terms reflects the evolving understanding of the condition's origin and characteristics. Initially, hip dysplasia was considered a congenital condition and referred to as

congenital dislocation or subluxation of the hip (2). In the late 20th century, it was recognized that the condition was not entirely congenital, and the term developmental dysplasia of the hip (DDH) was introduced (5). When DDH is diagnosed in newborns due to unstable or dislocated hips, it can also be called neonatal instability of the hip (NIH) (6). More recently, it has been recognized that hip dysplasia can be diagnosed in adulthood without a history of infant hip dysplasia (7). As the link between infant and adult hip dysplasia remains unclear, there is no general term for the adult condition. Some authors use the term DDH, while others use terms such as acetabular dysplasia or adult hip dysplasia. However, acetabular dysplasia can also be used to describe hip dysplasia in infants with dysplastic acetabula without accompanied deformities of the proximal femur. Consequently, the terms alone do not always provide a complete description of the condition's characteristics.

The focus of my research is adult hip dysplasia in individuals with no known history of infant hip disease, and the condition will be referred to as "hip dysplasia" throughout this book. The term "DDH" will be used specifically to describe hip dysplasia diagnosed in infants.

Developmental dysplasia of the hip

The etiology of DDH is not fully understood but risk factors include breech position, family history, female sex, oligohydramnios and high birth weight (8, 9).

In countries with well-developed healthcare systems, all newborns are screened for DDH. Screening methods vary between and within countries (10), as there are different opinions on whether neonatal DDH should be defined based on *stability* or *morphology*.

In Sweden, all newborns undergo clinical examination that includes a dislocation provocation test known as the Barlow test (11), followed by a reduction maneuver called the Ortolani test (12). Cases with suspected instability are referred to an orthopedic department for further clinical examination, supported by either dynamic ultrasound that evaluates *stability* (13) or static ultrasound that evaluates *morphology* (14).

At our department we use dynamic ultrasound and find approximately seven newborns with unstable or dislocated hips per 1000 births (6). Thanks to the nationwide screening program, only 0.12 dislocated hips per 1000 births are diagnosed more than two weeks after birth in Sweden (15). While it is possible that these late-diagnosed dislocations were missed during neonatal screening, there is also evidence suggesting that DDH can present later in hip development, even when clinical examination and morphology appeared normal at birth (16). As a result, Swedish children are routinely examined for DDH by a general practitioner (GP) at one month, six months and twelve months of age, in addition

to the neonatal screening. These secondary screenings detect approximately half of all cases of dislocated hips that present after the neonatal screening (17).

Without treatment, dislocated or subluxated DDH can lead to abduction contracture, leg shortening, limping and early development of OA (2, 18). Treatment is initiated as soon as DDH is diagnosed and typically involves various types of abduction splints. Regardless of how the diagnosis is defined at the neonatal screening, follow-up evaluation is based on the *morphology* seen in radiographs. Even when treatment is started early, hips that are unstable at the neonatal screening tend to have a more dysplastic morphology on 1-year radiographs compared to reference hips that are stable at the neonatal screening (6). However, these dysplastic hips appear to remodel as skeletal development progresses. Long-term follow-up of individuals with unstable hips at neonatal screening and dysplastic morphology at the 1-year follow-up showed that only 1 in 21 subjects had residual hip dysplasia at skeletal maturation. Furthermore, these individuals were reported to have normal clinical outcomes and good cartilage quality at follow-up (19).

In conclusion, our experience from clinical practice and research conducted at our department suggest that individuals with DDH have a favorable prognosis if the condition is detected and treated early.

Adult hip dysplasia

The etiology of hip dysplasia in adults without a history of infant hip disease remains unknown. It could arise from a neonatally unstable hip that was missed during the neonatal screening or develop later during the skeletal development. Increasing evidence suggests that adult hip dysplasia may be a distinct condition. For instance, female sex and left hip predominance are not as common in adult hip dysplasia as in DDH (7). Until more is known about the relationship between DDH and adult hip dysplasia, I prefer to treat them as two separate conditions.

Symptoms and clinical examination

The cause of symptoms in dysplastic hips without OA is not always clear. First, not all individuals with hip dysplasia experience symptoms. For those who do, common symptoms include pain in the hip area, limping and a sensation of locking, clicking or popping in the hip. Hip dysplasia can also reduce walking distance and impair the ability to perform sport activities (20-22). The pain may be localized in either the groin

or the lateral part of the hip. Groin pain could be caused by concurrent retroversion of the acetabulum, leading to anterior impingement similar to femoroacetabular impingement (FAI) (23). Up to 40% of dysplastic hips have been reported to have acetabular retroversion (24). Lateral hip pain is more commonly associated with instability in the hip joint, which can cause overloading of soft tissue structures, such as the abductor muscles (23).

Clinical examination should include assessment of gait, range of motion (ROM), muscle strength, instability, impingement and hypermobility. ROM may be increased in different directions, depending on the pattern of the acetabular deficiency (25). Studies have found that hip flexion and abductor muscle strength are weaker in individuals with hip dysplasia compared to controls (26), and the Trendelenburg sign is positive in approximately 40% of symptomatic individuals with hip dysplasia (22). Instability can be tested in several ways, one of which is simultaneous hyperextension, abduction and external rotation of the hip (27). The flexion-adduction-internal rotation (FADIR) test, which is typically known as an impingement test, is often positive in symptomatic dysplastic hips (22). Hypermobility is traditionally evaluated using the Beighton score (28).

Symptoms of hip dysplasia can fluctuate, and during pain-free periods, there may be no indication of pathology during clinical examination (20). In the presence of pain, intra-articular injection with local anesthesia can help determine whether the pain is intra- or extra-articular (29). If intra-articular pain is confirmed, it is often difficult to distinguish FAI and hip dysplasia solely on clinical presentation and patient history (30). Due to the nonspecific nature of hip dysplasia's presentation, delayed diagnosis is common (22).

Radiographic examination

Center edge angle

Radiographic examination is essential for diagnosing hip dysplasia. The most commonly used radiographic measurement to define hip dysplasia is the center edge angle, which is measured on anteroposterior radiograph (AP) of the pelvis. The angle reflects the acetabular coverage of the femoral head. It is defined as the angle between two lines drawn through the center of the femoral head: the first line is perpendicular to the horizontal line, and the second line extends to the lateral subchondral zone of the acetabular roof, known as the sourcil (2).

The lateral margin

The center edge angle was first described by Wiberg in 1939. In his thesis, Wiberg describes the lateral margin of the center edge angle as "*the point where the curving of the acetabular border laterosuperiorly begins, i.e. where the bony support may be considered to end. The dense shadow in the roentgen picture ends there*" (2). The dense shadow that Wiberg describes refers to the sourcil, which means "eyebrow" in French.

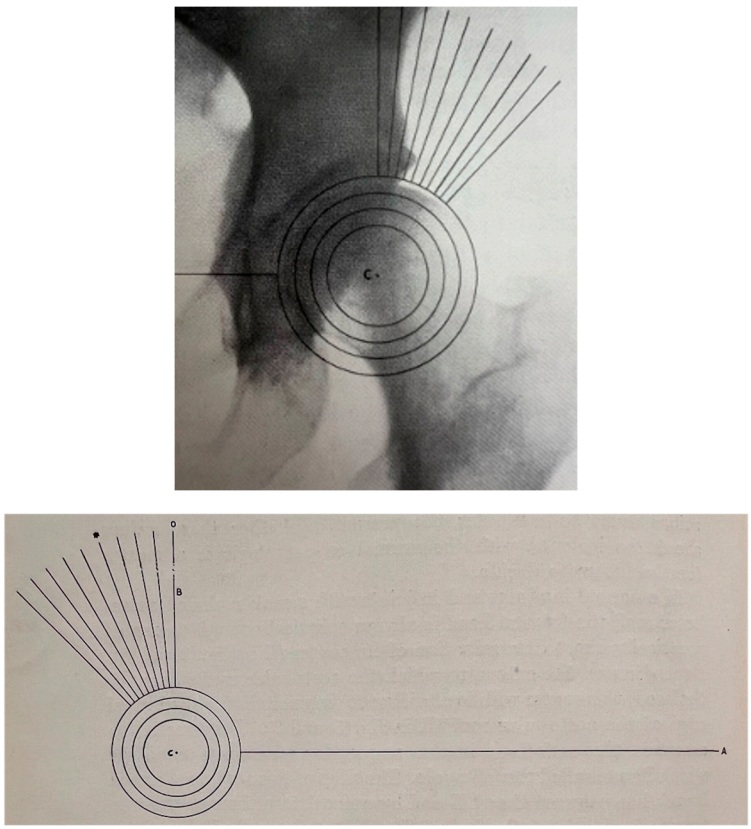

Figure 2: Wiberg's center edge angle.
Pictures from Wiberg's thesis, illustrating how he used a pattern laid over the radiographs to measure the center edge angle. Points C and A are positioned at the centers of the femoral heads, and the line between these two points forms the horizontal line of the pelvis. Line B is perpendicular to the horizontal line, representing a center edge angle of 0°. To the lateral side of line B are lines with 5° increments that are used to measure the center edge angle. In this example, the angle was measured as 14° (2).

Unfortunately, several authors have misunderstood Wiberg's description of the lateral margin, mistaking it for the lateral edge of the bony acetabulum instead of the sourcil (31-36). In 1990, Ogata proposed a "refined" center edge angle, that was identical to Wiberg's original version (35). Several authors have since compared these angles and

found that the angle according to Ogata is significantly lower than the misinterpreted angle of Wiberg (31, 32, 35, 37). It is important to be aware of this controversy when reviewing research about hip dysplasia. Even if a study claims that the center edge angle was measured according to Wiberg, it is crucial to carefully read the text and examine any images provided in the methods section to verify where the lateral margin has been placed. In Figure 3, I have demonstrated measurements using the two alternative lateral margins.

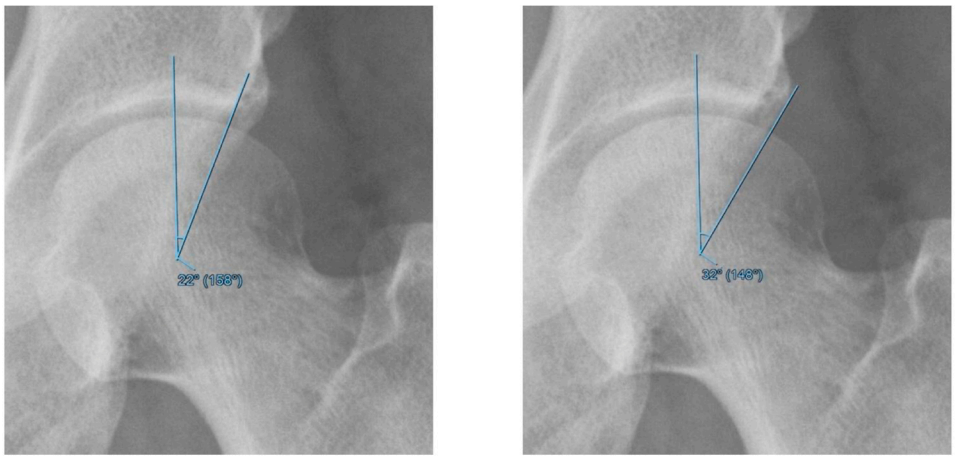

Figure 3: The lateral margin of the center edge angle.
The center edge angle measured to the lateral edge of the sourcil (left) and the lateral bony edge of the acetabulum (right).

Measurements on different projections

The original center edge angle is measured on AP pelvic radiographs and reflects the lateral coverage of the femoral head, known as the lateral center edge angle (LCEA). As previously mentioned, the shape of the dysplastic acetabulum is globally deficient (1), which means that the LCEA may not identify all cases of hip dysplasia. One way to improve the three-dimensional understanding of the dysplastic hip is by adding a false-profile (FP) radiograph (38), which allows for the quantification of the anterior coverage of the femoral head. The so-called anterior center edge angle (ACEA) has been reported to identify a significant number of dysplastic hips that would be missed if only the LCEA was measured (39).

Cutoff

According to Wiberg's original description, hips with an LCEA <20° are considered dysplastic, hips with an LCEA between 20–25° are classified as borderline hip dysplasia, and hips with an LCEA >25° are deemed non-dysplastic (2). Some studies, however, use broader spans for borderline hip dysplasia, such as 18–25° (40).

Automated measurement

Statistical shape models (SSM) identify and quantify the shape of objects within a study population. The key element of a SSM is the shape model, which is composed of a set of landmark points that outline the object of interest. When measuring the center edge angle, the object of interest is the proximal femur and pelvis. The number of landmark points vary between studies and can be positioned either manually by an observer or automatically through trained algorithms such as an Active Shape Model (ASM). Once the landmarks are defined, distances and angles between them can be calculated to capture the geometric properties of the shape (e.g. the center edge angle) (41).

Foramen obturator index

The foramen obturator index (FOI) is used to determine the degree of pelvic rotation in the axial plane. The FOI is calculated by dividing the widest horizontal diameter of the right obturator foramen by the widest horizontal diameter of the left obturator foramen (42). A FOI between 0.7 and 1.8 is recommended when assessing the LCEA, as greater pelvic rotation may affect LCEA measurements more than 2° (43).

Pelvic tilt

Pelvic tilt has been reported to affect the LCEA measurement (2, 43), although not to a clinically relevant extent (43). On AP pelvic radiographs, pelvic tilt can be assessed by measuring the distance between the coccyx and the symphysis, however, this distance has been considered difficult to identify on radiographs (44).

Additional radiographic assessment

Symptomatic patients with hip dysplasia should be further assessed with additional radiographic measurements and imaging modalities to fully evaluate the morphology and pathology of the hip joint.

Acetabular index angle

The acetabular index angle (AIA) describes the slope of the acetabular sourcil (42), with a normal range suggested to be between 3° and 13° (45).

Acetabular retroversion

The normal acetabulum is anteverted, with the acetabular socket directed anteriorly. In contrast, the socket is directed more posteriorly in a retroverted acetabulum. As previously mentioned, the prevalence of acetabular retroversion is high in dysplastic hips (24), and several measurements can be used to assess retroversion on AP pelvic

radiographs. The posterior wall sign is positive when the posterior wall is medial to the center of the femoral head. The cross-over sign is positive when the anterior wall is more lateral than the posterior wall or when they cross over. The ischial spine sign is positive when the ischial spine projects medial to the pelvic brim (46).

Alpha angle

The alpha angle is used to identify cam morphology, a possible cause of FAI that can coexist with hip dysplasia.

Osteoarthritis assessment

The presence of OA in dysplastic hips has a significant impact on treatment options and is discussed in a separate section further ahead in the introduction.

MRI

MRI can be performed to assess degenerative changes in the cartilage and labrum. Dysplastic hips typically suffer from labral injury in the anterolateral region, which coincides with the area where insufficient coverage is most common (24). MRI can also provide a more comprehensive three-dimensional understanding of acetabular coverage and acetabular version compared to plain radiography.

Prevalence of hip dysplasia based on the center edge angle

Previous studies have reported prevalence rates of hip dysplasia ranging from 1 to 15% when defined by an LCEA <20° (Table 1–2). The highest prevalence rates are typically observed in Asian populations (47, 48). The most relevant studies for our purposes are those by Jacobsen (49) and Engesæter (50), as they provide individual-level prevalence rates for Caucasian participants who are representative of the general population. Jacobsen found a prevalence of 5.5% in men and 5.4% in women in a Danish population with a mean age of 60 years. Engesæter's reported a slightly lower prevalence of 3.3% in Norwegian 19-year-olds. Prior to this thesis, no studies had investigated the prevalence of hip dysplasia in Sweden. We perceive that adult hip dysplasia has received limited attention in Swedish healthcare, particularly when compared to other countries.

Detection of hip dysplasia

Patients with symptomatic hip dysplasia often experience a delay in diagnosis (22). One possible explanation is low awareness among clinicians and radiologists. A delayed diagnosis can impair treatment outcomes and limit treatment options (51, 52).

Table 1: Prevalence on individual level.

Previous prevalence studies that have defined hip dysplasia on individual level based on the lateral center edge angle (LCEA).

Author	Country	Imaging	Hips (n)	Age (years)	Female	Type of population	Mean LCEA	Margin LCEA	Prevalence
Jacobsen 2005 (49)	Denmark	AP pelvic	3568	20–91 Mean: M 61.0 F 61.8	63%	Population-based	34.6°	Sourcil	Either hip: LCEA ≤ 20° M 5.5% LCEA ≤ 20° F 5.4%
Engesæter 2013 (50)	Norway	AP pelvic	2072	Mean: 18.6 (range 17.3–20.2)	58%	Population-based	Not stated	Bone	Either hip: LCEA <20° 3.3% (95% CI 2.5–4.1) LCEA <25° 20% (95% CI 18–21)
Raveendran 2018 (53)	USA	AP pelvic	5192	Mean 63 (SD 10)	57%	Population-based	Not stated	Sourcil	LCEA ≤20°: Unilateral: F: 5.8% (95% CI 4.8–7.0) M: 8.5% (95% CI 7.0–10.2) Bilateral: F: 3.1% (95% CI 2.4–4.0) M: 1.6% (95% CI 1.0–2.5)

AP = anteroposterior, LCEA = lateral center edge angle, F = females, M = males.

Table 2: Prevalence on hip level.

Previous prevalence studies that have defined hip dysplasia on hip level based on the lateral center edge angle (LCEA).

Author	Country	Imaging	Hips (n)	Age (years)	Female	Type of population	Mean LCEA	Margin LCEA	Prevalence
Croft 1991 (54)	England	Urograms	2604	60–75	0%	Referred to urogram	36.2° (SD 6.9)	Not clear	LCEA <25° 3.6% LCEA ≤20° 1.0%
Smith 1995 (55)	England	Urograms	393	60–75	100%	Referred to urogram	38° (SD 6.5)	Sourcil	LCEA <25° 3.8%
Inoue 2000 (48)	France	Urograms	783	20–79	30%	Referred to urogram	M 37.8° F 36.9°	Bone	LCEA <25° M 1.8% LCEA <25° F 5.6%
	Japan	Urograms	1538	20–79	46%	Referred to urogram	M 35.1° F 32.8°	Bone	LCEA <25° M 5.1% LCEA <25° F 11.6%
Umer 2009 (56)	Pakistan	AP pelvic	500	Mean: 38 (range 15–78)	46%	Referred to urogram, asymptomatic	35.5° (SD 6.6)	Not clear	LCEA <25° 1.4% LCEA <20° 0.8%
Anderson 2016 (57)	USA	AP pelvic	1081	Mean age: 67 (SD 8)	63%	Senior athletes, asymptomatic	Not stated	Sourcil	LCEA <20° 2.8%
Kim 2019 (47)	South Korea	AP pelvic	400	18–50	64%	Asymptomatic	26.2° (SD 6.0)	Sourcil	LCE <20° 15%

AP = anteroposterior, LCEA = lateral center edge angle, F = females, M = males .

Treatment

All treatment options discussed below apply to symptomatic individuals with hip dysplasia. Based on current knowledge, I have found no evidence to justify prophylactic treatment for asymptomatic adults with hip dysplasia.

Periacetabular osteotomy

Periacetabular osteotomy (PAO) is the most common surgical treatment for hip dysplasia in adults without hip osteoarthritis. Through a series of osteotomies, the acetabular portion of the pelvis is mobilized and reoriented to increase the acetabular coverage of the femoral head (58). The goal of a PAO is primarily to reduce pain and improve function. It is also hypothesized that a PAO may decrease the risk of OA development (59). However, this hypothesis remains unanswered until the natural history of adult hip dysplasia is better understood. Dysplastic hips with OA should not be treated with a PAO, as it has been shown that these hips have a high risk of early conversion to total hip replacement (THR) (52).

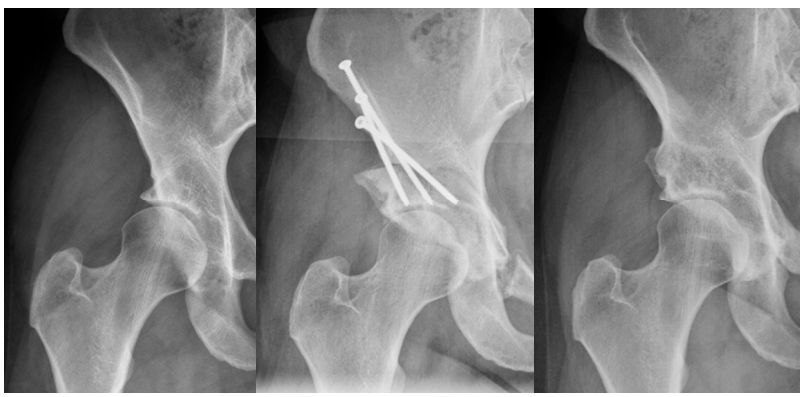

Figure 4: Periacetabular osteotomy.
From left to right: preoperative radiograph, radiograph six weeks postoperatively, and radiograph two years postoperatively with scews removed.

Physiotherapy

Given the increasing attention that hip dysplasia has received over the past decades, it is surprising that so few studies have investigated physiotherapy as a treatment option. The few existing studies primarily focus on training programs designed to increase abduction muscle strength (21). In clinical practice, physiotherapy can serve as the sole

treatment for individuals with borderline hip dysplasia or those with hip dysplasia who, for any reason, are not candidates for surgery. It can also be used as preoperative treatment for individuals scheduled for surgery. To date, no studies have compared conservative treatment with surgical treatment in patients with hip dysplasia.

Total hip replacement

Individuals with hip dysplasia who are not candidates for PAO due to OA, or who experience unsatisfactory results from prior conservative or surgical treatment, can be treated with a THR. A study that compared results from the hip registers of the Nordic countries reported that hip dysplasia accounted for 8% of THRs in Norway, 2% in Denmark, and 2% in Sweden (60).

Hip osteoarthritis

Up to one in four individuals will develop symptomatic hip OA during their lifetime (61) and the prevalence continues to rise (62). Unfortunately, the degenerative process often remains silently active for many years before symptoms emerge, and by the time of diagnosis, the damage is likely irreversible (63). Current treatment focuses on symptom management, as no curative treatments are available. Identifying modifiable risk factors is key to finding preventive treatments (64). Person-level risk factors include age, genetic factors and high-impact occupational activities. Female sex and overweight may also be risk factors, but the association with hip OA is not as strong as it is with knee OA. Joint-level risk factors include hip dysplasia, FAI and traumatic labral tears (65).

OA can be defined based on clinical information or radiography, but there is considerable discord between the two (66). Most importantly, symptomatic hip OA may not be detectable on plain radiographs in early stages of the disease. In clinical practice, it is therefore recommended that early OA should be diagnosed based on symptoms and clinical findings (67). According to the Swedish national program for the management of hip OA, patients should be referred for radiographic examination when the diagnosis is unclear or when there is a need for orthopedic consultation. In the absence of better alternatives, radiographic OA definitions are generally used for research purposes (66).

Radiographic hip osteoarthritis

Radiographic hip OA is traditionally assessed on AP radiographs using joint space measurements or grading systems (68).

Osteoarthritis grading

There are three grading systems for radiographic hip OA: the Kellgren and Lawrence (K&L) classification (69), the Tönnis classification (42) and the Croft classification (70). Although the number of grades differs between the systems, they all assess the presence of osteophytes, joint space narrowing and subchondral sclerosis. Evidence suggest that the K&L classification system has the highest inter-observer reliability, making it widely used in both research and clinical practice. Definite OA is typically classified as a K&L grade ≥2 (71). The K&L classification is presented in Table 3.

Table 3: The Kellgren and Lawrence classification for grading of hip osteoartritis (OA).

Grade	OA severity	Criteria
0	None	Definite absence of radiographic changes of OA
1	Doubtful	Doubtful joint space narrowing and possible osteophytic lipping
2	Minimal	Definite osteophytes and possible joint space narrowing
3	Moderate	Moderate multiple osteophytes, definite joint space narrowing and possible deformity of bone ends
4	Severe	Large osteophytes, marked joint space narrowing, severe sclerosis, and definite deformity of bone ends

Joint space width

The joint space width (JSW) can be used to define hip OA in various ways. Some studies use an absolute cutoff (e.g., ≤2 mm) (54, 72-75), while others measure a decrease in JSW (76) or use a grading system (77, 78).

The minimum JSW refers to the smallest value obtained when measuring the JSW in three different locations in the joint: the lateral margin of the sourcil, the center of the weigh bearing surface, and the medial margin of the weight-bearing surface (79).

Alternative modalities for osteoarthritis assessment

Computed tomography (CT) and magnetic resonance imaging (MRI) are comparable to, and in some respects superior to, plain radiography for the evaluation of OA. However, these modalities are more expensive and less widely available than plain radiography, making them less commonly used for the assessment of OA in clinical practice.

Clinically relevant hip osteoarthritis

There is no generally accepted definition of hip OA for use in clinical practice (67). The American College of Rheumatology (ACR) criteria is an established classification system that combines clinical assessment (hip pain) and radiographic assessment specifically for hip OA. However, the value of the ACR-criteria has been found to be limited in both research and clinical practice (67).

Hip dysplasia and hip osteoarthritis

Hip dysplasia as a risk factor for osteoarthritis

Hip dysplasia has been reported to decrease the weight-bearing surface between the acetabulum and the femoral head, leading to increased contact stress and damage to the articular cartilage (80). Dysplastic hips often have labral hypertrophy (81), which may be a compensatory response to the insufficient weight-bearing surface. Over time, the suboptimal loading pattern could result in OA.

There is growing evidence that adult hip dysplasia is a risk factor for hip OA (68, 82). However, the magnitude of the association remains unclear. I have summarized relevant prospective studies in Table 4 and Table 5. As shown in the tables, adjusted odds ratios (OR) range from 1.7 (95% CI 1.2–2.5) to 5.5 (95% CI 2.4–12.3). This variation might be explained by differences in population characteristics, definitions of dysplasia and OA, or follow-up time. Reijman et al. demonstrated that a lower LCEA-threshold resulted in a stronger association between hip dysplasia and OA (76). Agricola et al. demonstrated that the association became stronger when hip dysplasia was defined by an ACEA <25° in addition to an LCEA <25° (83). The influence of follow-up time remains unknown, but there are indications that the association is stronger in younger populations (84). Additionally, several cross-sectional studies and a few prospective studies have not found any statistically significant associations between hip dysplasia and OA (82). Finally, all previous studies that I have found in this field have used radiographic definitions for OA, without taking symptoms or clinical findings into account.

The natural history of osteoarthritis development in dysplastic hips

Although hip dysplasia appears to be a moderately strong risk factor for hip OA, all dysplastic hips do not develop OA. Longitudinal studies tracking adults with hip dysplasia have yielded conflicting results regarding the development of OA (as summarized in Table 6–8). Moreover, many of these studies fail to include information

about the participants' history of DDH, making it difficult to interpret the results in clinical context.

A deeper understanding of the natural history of hip dysplasia is essential for accurately informing patients about the prognosis of the condition, and for assessing whether interventions can alter the course of hip dysplasia and prevent or delay the onset of OA. More research in this area is needed to provide clearer answers and guide clinical decision-making.

Table 4: Prospective cohort studies.

Previous prospective cohort studies that have investigated the association between hip dysplasia and osteoarthritis (OA) by calculating adjusted odds ratios (aOR) or adjusted hazard ratios (aHR).

Author	Participants (hips), cohort, country	Follow up (years)	Age (years)	Female	Type of population	Margin LCEA	Dysplasia definition	OA definition	Association (95% CI)
Reijman 2005 (76)	835 (1670) Rotterdam Study, the Netherlands	6.6	66 (SD 6)	57%	Population-based, K&L 0–1, hip pain 6%	Not clear	LCEA <25°	Decrease JSW ≥1 mm	aOR 4.3 (2.2–8.7)
								K&L ≥2	aOR 2.4 (1.2–4.7)
							LCEA <30°	Decrease JSW ≥1 mm	aOR 2.8 (1.9–4.2)
								K&L ≥2	aOR 1.7 (1.2–2.5)
Agricola 2013 (83)	720 (1391) CHECK study, the Netherlands	5	56 (SD 5)	79%	New consulters for hip pain (18%), knee pain (40%) or boh (42%). K&L 0–1	Sourcil	LCEA <25°	K&L ≥2 or THR	aOR 2.8 (1.5–5.2)
							ACEA <25°	K&L ≥2 or THR	aOR 2.6 (1.4–4.8)
							LCEA + ACEA <25°	K&L ≥2 or THR	aOR 5.5 (2.4–12.3)
Thomas 2014 (85)	358 (670) Chingford study, UK	18	52 (IQR 48–56)	100%	Population-based	Not clear	N/A	K&L ≥2	13% increased risk per degree LCEA-decrease below 28 aOR 0.87 (0.8–0.98)
Saberi Hosnijeh 2017 (84)	4438 Rotterdam study extended, the Netherlands	9	63 (SD 6)	56%	Population-based K&L 0–1 baseline, hip pain 12%	Sourcil	LCEA <20°	K&L ≥2	aOR 2.2 (1.5–3.2)
Iidaka 2020 (86)	2975 ROAD study, Japan	7	70 (range 23–94)	65%	Population-based K&L 0–1	Not clear	LCEA <20°	K&L ≥2	aHR 2.1 (1.3–3.2)
								Increase of ≥1 grade	aHR 14.8 (3.7–56.0)

LCEA/ACEA = lateral/anterior center edge angle, K&L = Kellgren and Lawrence, THR = total hip replacement, JSN = joint space narrowing, N/A = not applicable.

Table 5: Prospective nested case control studies.

Previous prospective nested case control studies that have investigated the association between hip dysplasia and osteoarthritis (OA).

Author	Participants (hips), cohort, country	Follow up (years)	Age (years)	Female	Type of population	Margin LCEA	Dysplasia definition	OA definition	Association (95% CI)
Lane 2000 (87)	176 (352) Cases 58 (116) (OA at follow-up) Controls 118 (236) (no OA at follow-up) Study of Osteoporotic Fractures USA	8	70.3 (SD 4.7)	100%	Population-based	Bone as default, sourcil when bony edge was difficult to locate	LCEA <30°	Original summary grade, similar to K&L ≥2	aOR 3.3 (1.1–10.1)
Nicholls 2011(88)	135 (268) Cases 25 hips (THR year 20) Controls 243 hips (native hips year 20) Chingford study UK	19	55 (IQR 50-60)	100%	Population-based	Not clear	N/A	THR	10.5% increased risk of THR per 1° LCEA-decrease (2.0–18.2)

LCEA = lateral center edge angle, K&L = Kellgren and Lawrence, THR = total hip replacement, aOR = adjusted odds ratio, N/A = not applicable.

Table 6: Retrospective dysplasia cohorts, without control groups.

Previous longitudinal retrospective studies that have studied osteoarthritis (OA) development in adult cohorts with hip dysplasia, without control groups.

Author	Study design Country	Participants	DDH-cases included	Follow up (years)	Baseline characteristics	Dysplasia definition	OA definition	Results
Cooperman 1983 (89)	Retrospective Sweden	32 dysplastic hips (20 adults)	Unknown	22	Mean age 43 years (27–57) Mean LCEA 7.4° (-5°–20°) No hips subluxated No OA	LCEA ≤20° Margin not clear	JSW expressed as % of normal JSW	30/32 hips developed OA. No dysplasia parameter could predict the rate of the OA process
Hasegawa 1992 (90)	Retrospective/ prospective not stated Japan	86 dysplastic hips (59 adults)	Yes	9.2	Mean age 30 years (range 13–61), all with closed epiphysis F 92% Some hips subluxated 55% incipient OA	Unclear definition	Original definition based on sklerosis and JSW	33% of hips without OA at baseline, and 66% of hips with incipent OA at baseline had OA progress. LCEA lower in group that developed advanced OA
Amagami 2024 (91)	Retrospective Japan	92 dysplastic hips. Contralateral hip had undergone rotational acetabular osteotomy	Unknown	24 (SD 3.7)	Mean age 43 years (9.7) F 98% K&L 0 Asymptomatic	LCEA ≤25° Sourcil	K&L >0	29% developed K&L ≥0, of which 63% underwent THR. LCEA was significantly lower in group with OA onset

DDH = developmental dysplasia of the hip, LCEA = lateral center edge angle, JSW = joint space width, F = female, K&L = Kellgren and Lawrence, THR = total hip replacement.

Table 7: Prospective dysplasia cohorts, without control groups.

Previous longitudinal prospective studies that have studied osteoarthritis (OA) development in adult cohorts with hip dysplasia, without control groups.

Author	Study design Country	Participants	DDH-cases included	Follow up (years)	Baseline characteristics	Dysplasia definition	OA definition	Results
Wiberg 1939 (2)	Prospective Sweden	Cases: 17 dysplastic hips, 9/17 subluxated Controls: missing	Likely	4–29	Median age 35 years (range 13–60) Mean LCEA 9° (range 2–18), excluding 4 cases with negative values	LCEA <20° Sourcil	Original definition	OA developed in all cases. Median time to OA 15 (4–28) years
Hisatome 2005 (92)	Prospective Japan	Cases: 61 dysplastic hips that had undergone rotational acetabular osteotomy on contralateral hip due to dysplasia. Controls: missing	Likely	10 (range 7–16)	Mean age 38 years (range 20–58) F 92% No dislocated hips 57% no OA, 43% early OA Minimally symptomatic: 83.5% Asymptomatic: 16.5%	LCEA <20° Margin not clear	Early OA: slight JSN + subchondral sklerosis)	1/35 cases without OA at baseline developed early OA, 6/26 cases with early OA at baseline had OA progression. 10-year survival comparable for hips with LCEA <10° and LCEA ≥10°

DDH = developmental dysplasia of the hip, LCEA = lateral center edge angle, F = female, JSN = joint space narrowing.

Table 8: Dysplasia cohorts with control groups.

Previous longitudinal studies that have studied osteoarthritis (OA) development in adult cohorts with hip dysplasia, with control groups.

Author	Study design Country	Participants	DDH-cases included	Follow up (years)	Baseline characteristics	Dysplasia definition	OA definition	Results
Murphy 1995 (93)	Retrospective Switzerland	117 hips (both dysplastic and non-dysplastic) of which the contralateral hip had undergone THR secondary to hip dysplasia. 2 groups defined by severity of OA outcome	Likely	Followed until age 65 or end stage OA, duration unclear	No completely dislocated hips. Many hips had OA at baseline. Other baseline characteristics not stated	N/A Margin not clear	K&L	Dysplasia parameters signifiantly different between group 1 (n=74, K&L 3–4 or THR) and group 2 (n=43, K&L 0–2)
Jacobsen 2005 (79)	Prospective Denmark	Cases: 81 individuals with uni- or bilateral dysplasia Controls: 136, matched	No	9–12	Mean age (years): F 53 (range 25–70) M 49 (range 24–79) No OA. Cases: 67% F, mean LCEA range 6–20°, no subluxated hips. Controls: F 51%	Cases: LCEA ≤20° Controls: LCEA ≥25° Sourcil	Change in minimum JSW between baseline and follow-up	No difference between cases and controls in change in minimum JSW
Wyles 2017 (94)	Retrospective USA	Cases: 48 dysplastic hips. Controls: 40, not matched. Contralateral hip had undergone THR due to OA	Unknown	20 (range 10–35)	Mean age 47 (18–55) years F 56% Tönnis grade 0	Based on several dysplastic parameters, including LCEA <25° Margin not clear	Tönnis	40% of dysplastic hips and 15% of control hips progressed from grade 0 – grade 3/THR (HR 2.8 (95% CI 1.1–7.0)

DDH = developmental dysplasia of the hip, THR = total hip replacement, N/A = not applicable, K&L = Kellgren and Lawrence, M = men, F = female, LCEA = lateral center edge angle, JSW = joint space width, HR = hazard ratio.

Summary of thesis aims

The overall aim of this thesis is to gain a better understanding of the prevalence and consequences of adult hip dysplasia. The specific aims are:

- To determine the prevalence of adult hip dysplasia in a Swedish population.
- To investigate whether hip dysplasia is mentioned in radiology reports.
- To study the development of radiographic hip OA in participants with unilateral hip dysplasia identified in the abovementioned prevalence study, and to compare OA outcomes between dysplastic and contralateral non-dysplastic hips.
- To examine the relationship between hip dysplasia and the risk of developing radiographic OA at several time points, and to evaluate whether the magnitude of the association changes over time.
- To explore the relationship between hip dysplasia and radiographic hip OA using different definitions of hip dysplasia, based on lateral and/or anterior undercoverage.
- To investigate the long-term association between hip dysplasia and clinically relevant hip OA, as defined by an expert diagnosis incorporating both clinical and radiographic data.

Methods

Study designs

Table 9: Study designs of Studies I-IV.

	Study I	Study II	Study III	Study IV
Design	Cross-sectional	Longitudinal	Longitudinal	Longitudinal
Data collection	Retrospective review of radiographic records	Retrospective review of radiographic records	Prospective collection of radiographs, questionnaires and clinical examination	Prospective collection of radiographs, questionnaires and clinical examination
Follow-up time (years)	N/A	Median 13 (IQR 15–8)	2, 5, 8 and 10	10
Study site	Sweden	Sweden	The Netherlands	The Netherlands
Cohort, sample size	AP pelvic radiographs from Malmö 2007–2008 1870 participants (3740 hips)	Individuals with unilateral hip dysplasia, identified in Study I 50 participants (100 hips)	CHECK 1169–1262 hips depending on follow-up time point	CHECK 468 hips
Outcomes	Prevalence of hip dysplasia Mention of hip dysplasia in radiology reports	Radiographic OA outcome in individuals with unilateral hip dysplasia with comparison between dysplastic and non-dysplastic hips	Association with radiographic OA at year 2, 5, 8 and 10 Association with radiographic OA when hip dysplasia was defined by the LCEA and/or the ACEA	Association with clinically relevant OA at year 5–10 Association with radiographic OA at year 10

Study populations

Studies I and II

Studies I and II were based on participants identified in the radiographic records of the regional healthcare system in Skåne, Sweden, which serves a population of 1.4 million people across ten public health hospitals. The inclusion process is outlined in the flowchart in Figure 5.

In Study I, radiographic records from Skåne University Hospital in Malmö were reviewed to identify all AP pelvic radiographs performed during 2007–2008. Participants aged 20–70 years with a Swedish personal identity number were included to ensure full skeletal maturity and to minimize age-related degenerative changes that could affect measurement quality. If multiple radiographs were available, only the first one was included. The radiographs, referrals and radiology reports were reviewed to determine participant eligibility. Exclusion criteria included: FOI outside 0.7–1.8, OA, hip implant, hip fracture, childhood hip disorder, inflammatory hip disease, avascular necrosis of the femoral head, skeletal deformity of the hip joint due to neurological disease, and poor imaging quality.

In Study II, the study population consisted of individuals with unilateral hip dysplasia identified in Study I. Participants with unilateral hip dysplasia (LCEA ≤20° for the dysplastic hip and LCEA >20° for the non-dysplastic hip) and K&L grade <2 at baseline were included. Follow-up imaging (plain radiographs, CT or MRI) from all hospitals in Skåne was reviewed to assess the presence of radiographic OA, including THR, provided both hips were visualized, and OA could be assessed with certainty according to clinical expertise.

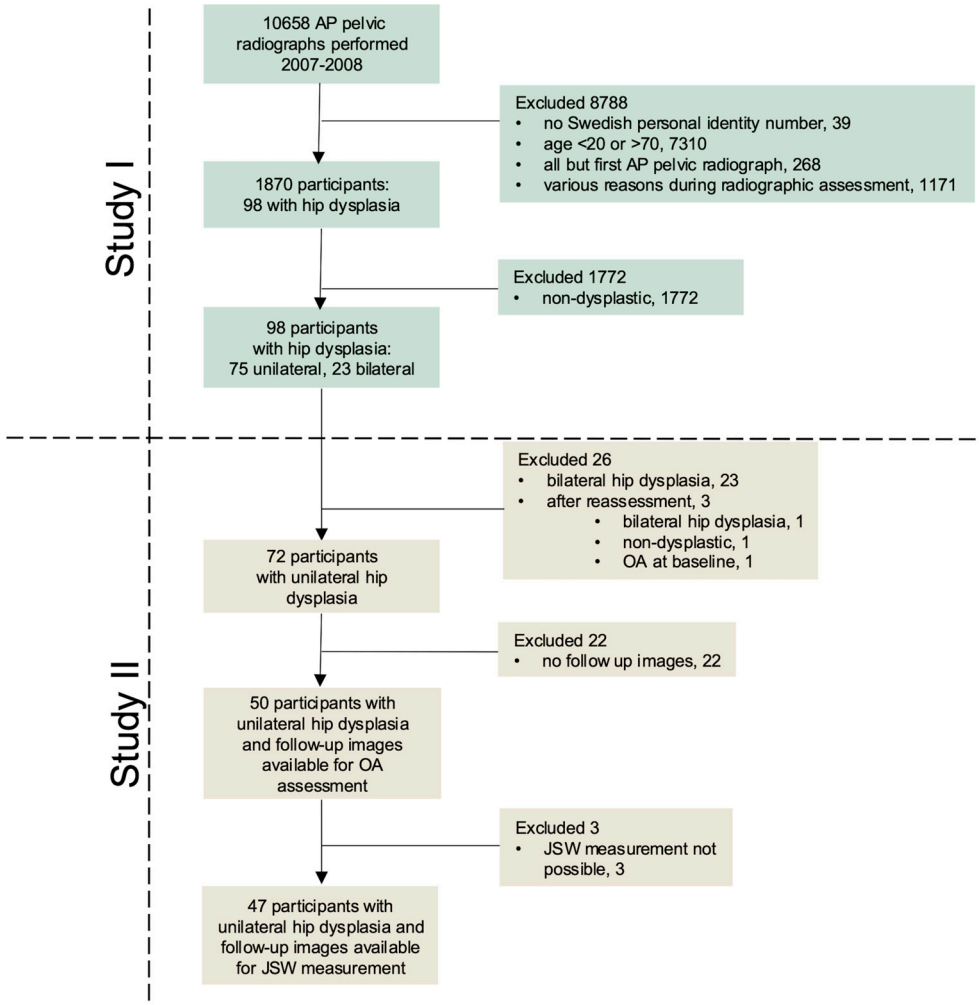

Figure 5: Flowchart of inclusion in Studies I and II.
Participants were included based on radiographic records from the regional healthcare system in Skåne, Sweden.

Studies III and IV

Studies III and IV are based on the Cohort Hip and Cohort Knee (CHECK) study, a prospective multicenter cohort study conducted in the Netherlands. Upon entry, all 1002 CHECK participants, aged 45–65 years, had pain or stiffness in the knee and/or hip, and had either not consulted a GP for these symptoms or had done so within the

last six months. Participants were recruited through GPs, local newspaper advertisement, the Dutch Arthritis Foundation's website, and via flyers between October 2002 and September 2005.

Exclusion criteria included comorbidities that would prevent 10-year follow-up, malignancy in the last five years, language barriers, and conditions (other than possible OA) that could explain their musculoskeletal symptoms. For hip-related issues, exclusion criteria included: trauma, rheumatoid arthritis, congenital hip dysplasia, Perthes disease, subluxation, osteochondritis dissecans, fracture, septic arthritis, K&L grade 4, THR, previous hip surgery, or isolated symptoms of bursitis or tendinitis. Ten medical centers participated, collecting questionnaires, radiographs, and conducting clinical examinations at baseline, and 2, 5, 8 and 10-year follow-ups. Only 145 participants (14%) were lost to follow-up.

In Study III, all hips with an available center edge angle and without radiographic OA (K&L <2) at baseline, were included. For each follow-up, hips with available K&L gradings were included (Figure 6).

In Study IV, the same inclusion criteria were used as in Study III, with the additional requirement of hip pain at baseline, available K&L grade at the 10-year follow-up, and an expert diagnosis for clinically relevant hip OA (Figure 6).

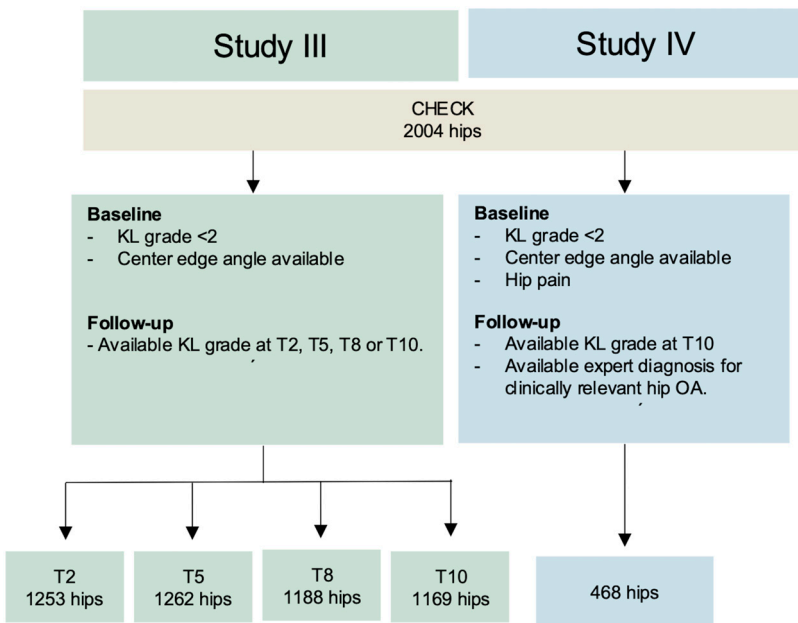

Figure 6: Flowchart of inclusion in Studies III and IV.
Inclusion of participants from the CHECK study. T2–T10 = 2–10-year follow-up.

Radiographic measurements of hip dysplasia

Center edge angle

Throughout Studies I–IV, the center edge angle was defined according to Wiberg's description, with the sourcil as the lateral margin (2).

In Study I, the LCEA was manually measured on 1870 supine AP pelvic radiographs using the dysplasia guide of the Sectra 2D Planning System. The measurements were repeated on 50 randomly selected radiographs two months after completing the initial assessment to evaluate intra-observer reliability. Inter-observer reliability was not assessed. Hip dysplasia was defined as an LCEA ≤20°, and borderline hip dysplasia was defined as an LCEA ≤25°.

In Study II, the LCEA measurements from Study I were reassessed due to the experience that some landmarks were not always clearly visible, and minor adjustments could significantly alter the angle. While reassessing all 3740 measurements from Study I was considered unmanageable, it was manageable for the 100 measurements included in Study II.

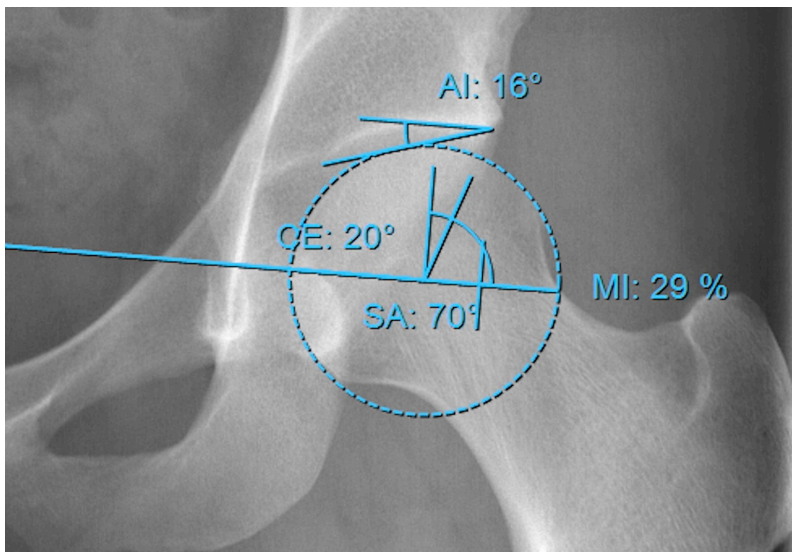

Figure 7: Measurement of the LCEA (marked CE) and AIA (marked AI) in Study I.

In Studies III and IV, observers outlined the bony contours of the proximal femur and pelvis by manually positioning 75 landmark points using a SSM software (ASM toolkit). Three observers positioned points for the LCEA, and two observers positioned points for the ACEA. The observers were blinded to both clinical and radiographic outcomes. The angles were automatically calculated using a MATLAB script. For the ACEA, the horizontal line was defined by the horizontal line of the radiographic film.

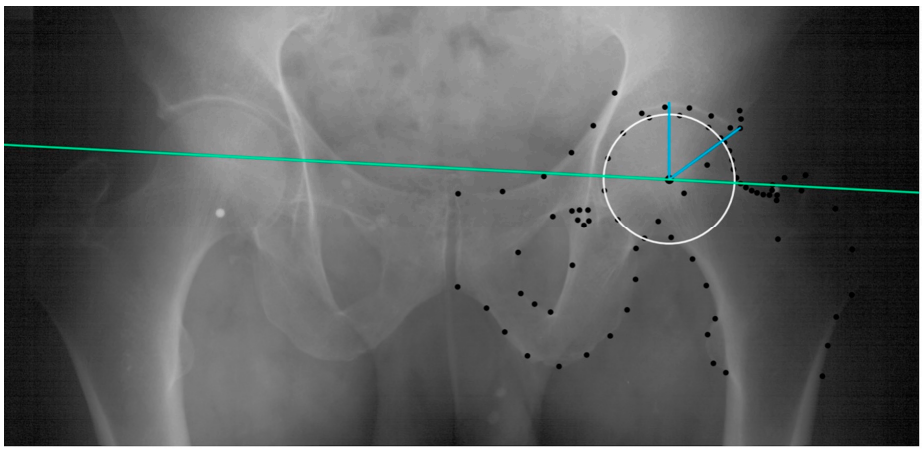

Figure 8: Measurement of the LCEA in Studies III and IV.

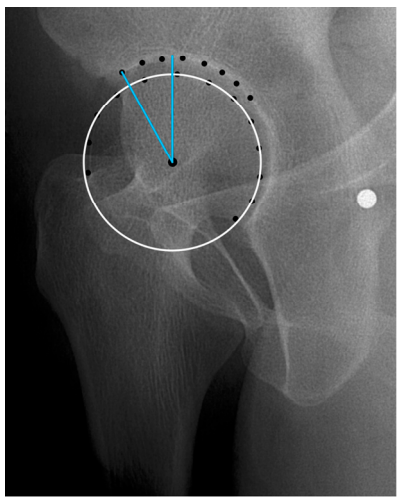

Figure 9: Measurement of the ACEA in Study III.

In Study III, hip dysplasia was defined in three different ways: LCEA <25°, ACEA <25°, and a combination of both. In Study IV hip dysplasia was defined as an LCEA ≤20°. The LCEA was measured on weight-bearing AP radiographs of the pelvis obtained with the feet in 15° internal rotation and with a symmetrical pelvis (95). The ACEA was measured on weight-bearing FP radiographs, with the pelvis rotated 65° in relation to the radiographic table (38). The set of landmark points was positioned twice by each observer on 25 randomly selected radiographs, with a two-months interval, to assess inter- and intra-observer reliability (83).

Acetabular index angle

In Study I, the AIA was measured alongside the LCEA to complement the anatomical description of the studied hips, but it was not used to define the prevalence of hip dysplasia. The AIA was defined as the angle between the horizontal line and the line connecting the lateral and medial margin of the sourcil (Figure 7) (42). The measurements were repeated on 50 randomly selected radiographs two months after completing the initial assessment to evaluate intra-observer reliability. The inter-observer variability was not assessed.

Table 10: Overview of radiographic measurements performed to assess hip dysplasia.

	Observer	Method	Software	Measurements	Cutoff
Study I	R Vinge	Manual	Sectra	LCEA AIA	≤20°, ≤25° N/A
Study II	R Vinge	Manual	Sectra	Reassessment of LCEA-measurement from Study I	≤20°
Study III	Observers from CHECK (LCEA – 3 observers, ACEA – 2 observers)	Manual placement of 75 landmark points, automated calculation	ASM tool kit, MATLAB script	LCEA ACEA	<25° <25°
Study IV	Observers from CHECK	Manual placement of 75 landmark points, automated calculation	ASM tool kit, MATLAB script	LCEA	≤20°

LCEA/ACEA= lateral/anterior center edge angle, AIA = acetabular index angle, N/A = not applicable, CHECK = Cohort Hip and Cohort Knee, ASM = active shape model.

Outcome variables

Prevalence of hip dysplasia

In Study I, the prevalence of hip dysplasia was defined as the proportion of the study population with hip dysplasia in either one or both hips. The prevalence was calculated for both hip dysplasia (LCEA ≤20°) and borderline hip dysplasia (≤25°).

Mention of hip dysplasia in radiology reports

In Study I, radiology reports of the included radiographs were reviewed to identify mention of hip dysplasia, either explicitly or through typical features. The digital radiographic reports were directly linked to the radiographs.

Minimum joint space width

In Study II, the minimum JSW was measured bilaterally at baseline and on the last available radiograph in which the participant had native hips. Measurements were made in three locations: the lateral margin of the sourcil, the center of the weigh bearing surface, and the medial margin of the weight-bearing surface. The minimum JSW was registered as the lowest of the three values (79).

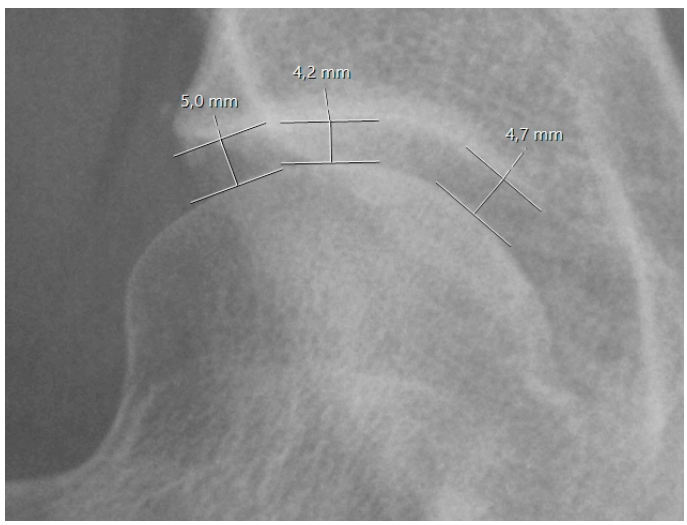

Figure 10: Measurement of the minimum joint space width (JSW) in Study II.
In this example the minimum JSW was 4.2 mm.

General assessment of osteoarthritis

In Study II, OA was assessed using plain radiography, CT, and MRI according to the K&L classification in a dichotomized manner: <2 = no OA and ≥2 or THR = OA. This outcome was not referred to as K&L since the definition of K&L grades is based on assessment of AP pelvic radiographs only. THR was included in the OA outcome and was also reported separately.

Kellgren and Lawrence classification

In Studies III and IV, baseline AP pelvic radiographs were scored according to the K&L classification by the CHECK steering committee, consisting of senior researchers with expertise in radiographic OA. K&L grading was performed after each follow-up by trained observers who had access to radiographs and grades from previous time points, including their chronological order. Four trained observers scored the radiographs at the 2- and 5-year follow-ups, while five trained observers scored the radiographs at the 8- and 10-year follow-ups. These trained observers were medical students who had received extensive training from a musculoskeletal radiologist and a GP with expertise in early OA. The GP supervised the trained observers throughout the study.

After the final follow-up, the observers reviewed all grades in chronological order, reassessed where needed and checked for missing data.

Inter-observer reliability between the trained observers and the GP was tested using 38 radiographs from the 5-year follow-up, scored by all five observers. Hip OA was defined as a K&L grade ≥2 or a THR.

Expert diagnosis for clinically relevant hip osteoarthritis

In Study IV, clinically relevant hip OA was determined through an expert-based diagnosis. The 24 experts – GPs, rheumatologists and orthopedic surgeons with extensive OA experience – evaluated data from questionnaires, physical examination and radiographs collected 5-, 8- and 10-years from baseline.

Questionnaires included WOMAC scores for pain, stiffness and function along with questions on physical activity, comorbidities, current hip pain, and the history of any hip pathology. Physical examination assessed pain and ROM during passive flexion, internal rotation, external rotation, and abduction. Radiographic data included AP pelvic and FP hip radiographs, K&L grades, and separate grades for the presence of osteophytes and JSN.

Each hip was assessed by a pair of experts (one GP and one secondary care clinician), who individually reviewed the data and determined the presence of clinically relevant OA based on their expertise. They also rated their certainty of the assessment from 1 (definitely not clinically relevant OA) to 100 (definitely clinically relevant OA).

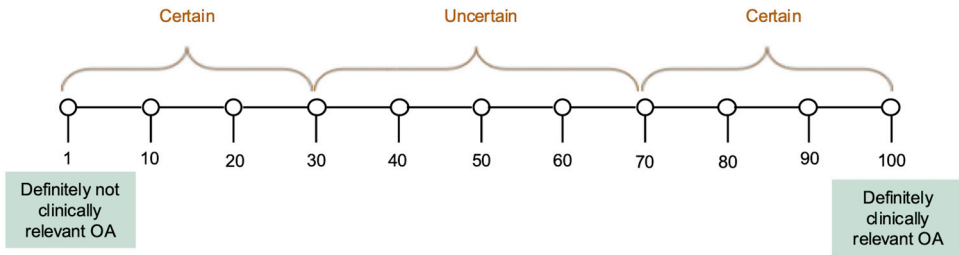

Figure 11: Certainty scale for expert diagnosis of clinically relevant OA in Study IV.

The agreement within the expert pair was assessed to determine the outcome. If the experts agreed, the matter was confirmed (either clinically relevant OA or not). If they disagreed and both had rated uncertainty (>30 to <70), the outcome was reported as uncertain. In other cases of disagreement, the assessment was revisited during a consensus meeting. If consensus could not be reached, the outcome was reported as uncertain.

The diagnosis of clinically relevant OA was not limited to a single follow-up but was based on data collected from year 5 to 10 after baseline. Further details on the expert diagnosis can be found elsewhere (96).

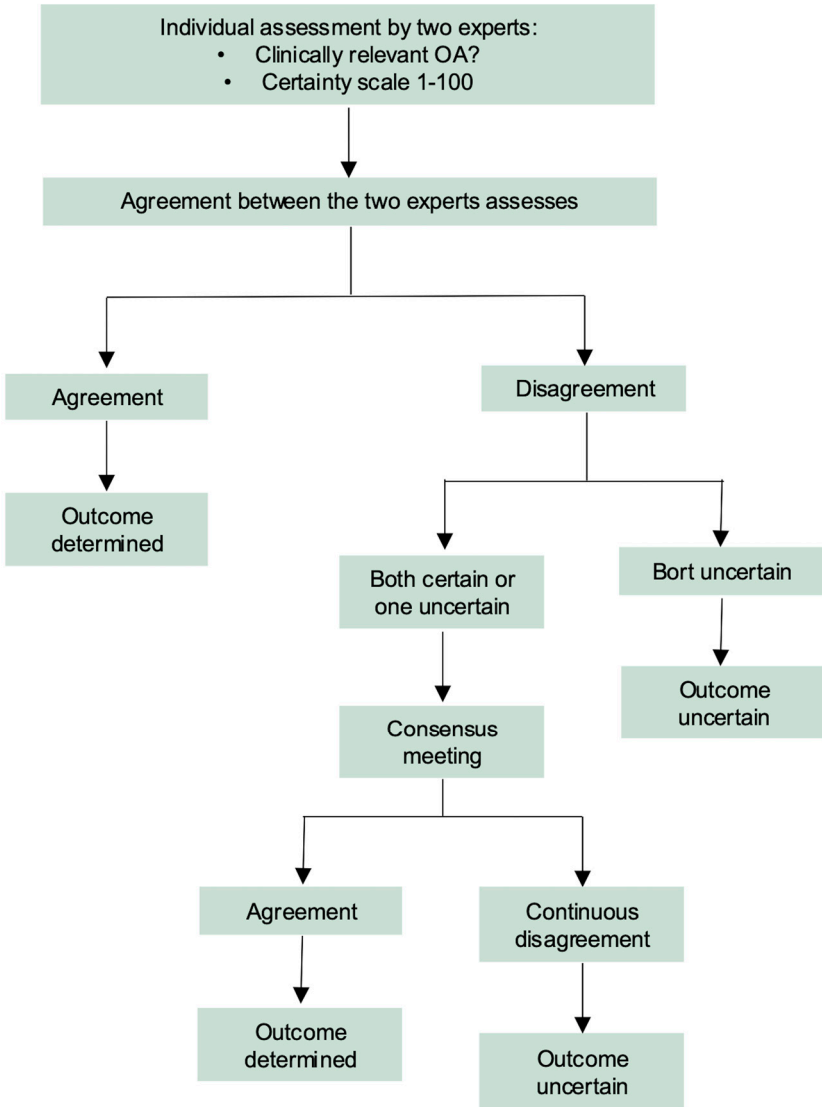

Figure 12: Flowchart for obtaining the expert diagnosis of clinically relevant hip OA in Study IV.

Table 11: Overview of the methods and definitions used for hip osteoarthritis (OA).

	Observer	Data source	Method	Outcome variable
Study I	R Vinge	AP pelvic radiographs	JSN on either side	OA = JSN No OA = no JSN
Study II	R Vinge	Plain radiographs	K&L	OA = K&L ≥2/THR No OA = K&L <2
		CT MRI	Same rationale as K&L	
Study III	Baseline: senior researchers with OA expertise	AP pelvic radiographs	K&L	OA = ≥2/THR No OA = K&L <2
	Follow-up: trained observers supervised by GP with OA expertise			
Study IV	Expert diagnosis: pair of one GP and one rheumatologist or ortopaedic surgeon. All with extensive OA experience.	AP pelvic radiographs Questionnaires Physical examination	Expert diagnosis for clinically relevant OA	OA = yes/uncertain No OA = no
	Radiographic hip OA: same methods and definition as in Study III.			

AP = anteroposterior, JSN = joint space narrowing, K&L = Kellgren and Lawrence, THR = total hip replacement.

Statistical analysis

Power analysis

In Study I, a power analysis showed that 1400 subjects were required to obtain a dysplasia prevalence with a precision of ±1 percent unit.

Descriptive statistics

Means with standard deviations (SD) were used for normally distributed variables, and medians with ranges or interquartile ranges (IQR) were used for non-normally distributed variables. Categorical variables were presented with absolute numbers and percentages.

Interferential statistics

Normally distributed variables were compared between independent groups using Student's t-test for independent samples and between dependent groups using the

paired samples t-test. Non-normally distributed variables were compared between independent groups using the Mann-Whitney U-test. Categorical variables were compared between independent groups with the chi-square test, and between dependent groups using McNemar's test. The Clopper-Pearson method was used to calculate 95% CIs for proportions. Correlation was assessed with Pearson's correlation coefficient.

Associations between a predictor (hip dysplasia) and binary outcome (different definitions for hip OA) were analyzed using a generalized estimating equations (GEE) logistic regression model. The strength of association was expressed as odds ratios (OR) with 95% CI, adjusted for age, sex and BMI. Throughout, p-values below 0.05 were considered statistically significant.

GEE is a method that accounts for correlation between observations, such as the correlation between the right and left hip of the same participant in Studies III and IV.

Inter- and intra-observer reliability

In Study I, intra-observer reliability for LCEA and AIA-measurements was assessed by calculating systematic error, random error, and the intraclass correlation coefficient (ICC). In Studies III and IV, ICC was used to evaluate both inter- and intra-observer reliability for LCEA and ACEA measurements. All ICCs were calculated with 95% CIs and interpreted according to Koo and Li (97).

The inter-observer reliability of the 5-year K&L scoring from the CHECK-cohort (Studies III and IV) was evaluated using prevalence-adjusted bias-adjusted kappa (PABAK) with 95% CIs. K&L grades were dichotomized (0 and ≥1), and PABAK was calculated for each of the four trained observers compared to the experienced observer.

Systematic error = (mean of measurement 1 – mean of measurement 2)/2

Random error = SD ((measurement 1 – measurement 2)/ √2)

ICC is a ratio of true variance divided by total variance (true + error variance). It ranges from 0 to 1, with values closer to 1 indicating stronger reliability. Interpretation according to Koo and Li: <0.5 = poor, 0.5–0.75 = moderate, 0.75–0.9 = good and >0.9 = excellent (97). ICC is suited for analysing agreement in continous variables.

The kappa coefficient quantifies agreement beyond chance, ranging from -1 to 1. -1 indicates less agreement than expected by chance, 0 indicates agreement by chance, and 1 indicates perfect agreement. Interpretation: 0–0.2 = poor, 0.21–0.4 = fair, 0.41–0.6 = moderate, 0.61–0.8 = substantial, 0.81–1 = almost perfect. A key limitation of kappa is its sensitivity to observer bias and outcome prevalence. A prevalence-adjusted and bias-adjusted kappa can account for this (98). Kappa is suited for analyzing agreement in categorical variables.

Sensitivity analyses

Detailed results from the sensitivity analyses in Study IV can be found in the Supplementary data of the study (99).

Confounders

In Study IV, sensitivity analyses of the regression model were performed both without any adjustment and with the addition of K&L grade 1 at baseline as a confounder, alongside the standard confounders (age, sex and BMI). The ORs derived from these sensitivity analyses were comparable to those in the main analysis.

Dichotomization of the variable clinically relevant hip osteoarthritis

To fit the clinically relevant hip OA outcome (Study IV) into the GEE logistic regression model, the variable needed to be dichotomized. This required determining whether to exclude the "uncertain" value or combine it with "yes = definitely clinically relevant hip OA" or "no = definitely not clinically relevant hip OA".

In a previous CHECK study, "uncertain" was categorized as "yes" for knee OA (96). To evaluate whether this approach was appropriate for our study of hip OA, we conducted a sensitivity analysis. First, we excluded "uncertain" to assess the association between hip dysplasia and "yes". Then, we excluded "yes" to examine the association between hip dysplasia and "uncertain". The association between hip dysplasia and "uncertain" was strong and consistent with the direction of the association between hip dysplasia and "yes", supporting the decision to categorize "uncertain" as "yes".

Table 12: Overview of who performed the statistical anayses in Studies I–IV.

	Main person to perform statistical analysis	Exception
Study I	R Vinge	Power analysis performed by statistician
Study II	R Vinge	–
Study III	N Riedstra	Inter- and intraobserver reliability for the center edge angle and K&L grades was performed and published by other CHECK-researchers (83, 98).
Study IV	R Vinge	

Ethical approval

Study I was approved by the Region Ethics Review Board (2015/910) with an opt-out consent from participants. Study II was approved by the Swedish Ethical Review Authority (2023-04683-01) allowing an extended review of radiographic records without additional participant consent.

Studies III and IV, conducted in the Netherlands, did not require approval from the Swedish Ethical Review Authority. Written informed consent was obtained from all participants, and the CHECK study was approved by the Medical Ethics Committee of the University Medical Center Utrecht (02/017-E).

Results

Study populations

The baseline characteristics of the participants in Studies I–IV are presented in Table 13. Distribution of the LCEA and AIA measurements of Study I are presented as histograms in Figures 13 and 14. There was a strong negative correlation between the LCEA and AIA; Pearson's correlation coefficient was -0.77 ($p<0.001$) for right hips and -0.76 ($p<0.001$) for left hips.

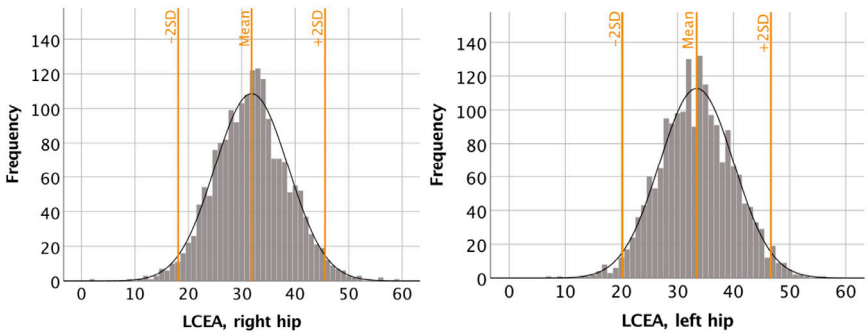

Figure 13: Distribution of the measurements of the lateral center edge angle (LCEA) in Study I. The mean LCEA was 32°(SD 6.9) and 33°(SD 5.5) in right and left hips, respectively.

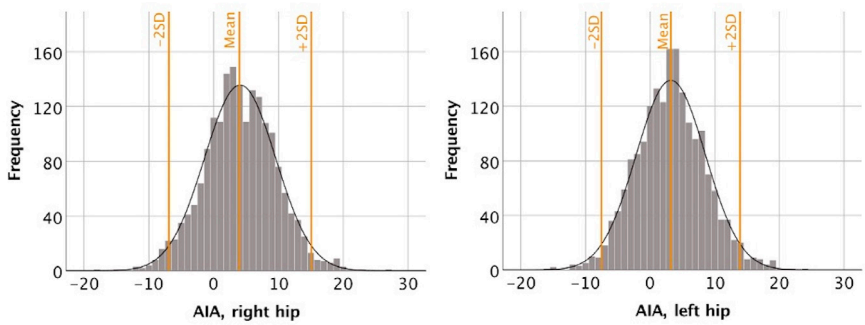

Figure 14: Distribution of the measurements of the acetabular index angle (AIA) in Study I. The mean AIA was 4.1°(SD 5.5) and 3.2°(SD 5.4) in right and left hips, respectively.

Table 13: Baseline characteristics in Studies I–IV.

	Study I	Study II	Study III	Study IV
Number of participants	1870 individuals, 3740 hips	50 individuals, 100 hips	1265 hips	251 individuals, 468 hips
Age, years	Median 53 (range 20–70)	Median 47 (IQR 62–39)	Mean 56 (SD 5)	Mean 55.5 (SD 5.4)
Female, %	63% (n=1171)	72% (n=36)	82% (n=1038)	88% (n=221)
BMI, kg/m2	Not studied	Not studied	Mean 26 (SD 4)	Mean 26 (SD 4)
Pain present	Not studied	Not studied	Hip and/or knee pain	Hip pain
Trauma as reason for referral, %	28% (n=530)	36% (n=18)	N/A	N/A
K&L 1 at baseline, % (hip level)	Not studied	Not studied	26% (n=322)	31% (n=143)
Prevalence of hip dysplasia, % (hip level)	LCEA ≤20°: 3.2% (n=121) LCEA ≤25°: 14.7% (n=550)	LCEA ≤20°: 50% (n=50)	LCEA <25°: 11.4% (n=144) ACEA <25°: 9.0% (n=112) LCEA & ACEA <25°: 3.7% (n=47)	LCEA ≤20°: 3.6% (n=17) LCEA ≤25°: 13.2% (n=62)
LCEA, °	Mean: M: 32° (SD 5.8), F: 33° (SD 6.6°)	Median: Dysplastic: 18.5° (IQR 20–17) Non-dysplastic: 25° (IQR 28–23)	Not reported	Not reported
AIA, °	Mean: M: 4.2° (SD 4.7) F: 3.3° (SD 5.1)	Not studied	Not studied	Not studied

N/A = not applicable, K&L = Kellgren and Lawrence, LCEA/ACEA = lateral/anterior center edge angle, M=males, F=females.

Prevalence of hip dysplasia

In Study I, we identified 98 adults with hip dysplasia in either the right and/or left hip, resulting in a prevalence of 5.2% (95% CI 4.3–6.3). Of these, 23% (n=23) had bilateral hip dysplasia. There was no statistically significant difference in prevalence between women (5.6%, 95% CI 4.4–7.1) and men (4.6%, 95% CI 3.2–6.4). Similarly, no statistically significant difference was found in prevalence between individuals examined due to trauma (more likely to be representative of the general population) and those examined for other causes (more likely to have hip symptoms): 6.4% (95% CI 4.5–8.8) vs. 4.8% (95% CI 3.7–6.1), respectively. The prevalence of borderline hip dysplasia was 21% (n=400). The ICC for intra-observer reliability was excellent (>0.9) for the LCEA and good (0.75–0.9) for the AIA.

Mention of hip dysplasia in radiology reports

In Study I, the condition was not mentioned in the radiology report for 91 of the 98 cases with hip dysplasia.

Hip dysplasia and hip osteoarthritis

Osteoarthritis development in a hip dysplasia cohort

In Study II, 50 individuals with unilateral hip dysplasia were included from the 98 individuals with hip dysplasia that were identified in Study I. Twelve participants had passed away during the study period, with a mean time to death of 9.3 years (SD 4.4). No participants had moved out of the catchment area. The most common imaging modality for OA assessment was plain radiographs, followed by CT (Table 14).

Table 14: Modalities used for osteoarthritis assessment in Study II.

Modality	Frequency
AP pelvic radiograph	21
Abdominal CT	15
Spine MRI	6
CT urography	3
Urography	2
Abdominal radiograph	2
CT angiography of aorta	1
Total	50

OA was detected in 19/50 dysplastic hips and 16/50 non-dysplastic hips, with no statistically significant difference in incidence rates (p=0.55) (Table 15). Five dysplastic hips and four non-dysplastic hips had undergone THR (p=1.0).

Table 15: Osteoarthritis (OA) incidence in Study II.

	Dysplastic hips, LCEA ≤20° (n=50)	Non-dysplastic hips, LCEA >20° (n=50)	
OA incidence	38% (n=19)	32% (n=16)	
		LCEA 21–25° (n=28)	**LCEA >25° (n=22)**
		29% (n=8)	36% (n=8)

LCEA = lateral center edge angle.

The minimum JSW was measured bilaterally in the last available imaging of 47 participants. The mean minimum JSW was 3.3 mm (SD 1.3) for dysplastic hips and 3.5 mm (SD 0.9) for non-dysplastic hips (mean difference -0.2 mm, 95% CI -0.6–0.2, p=0.308). The measurements are visualized in the univariate scatterplot shown in Figure 15.

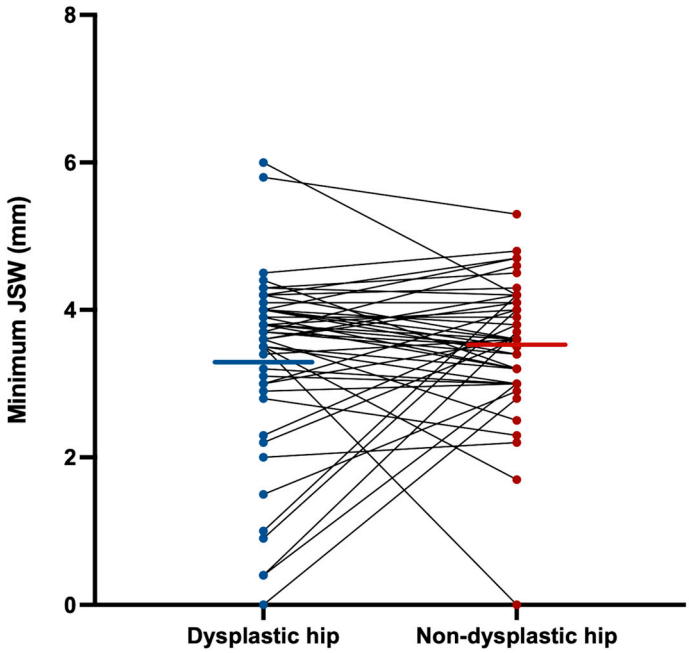

Figure 15: Univariate scatterplot of minimum joint space width (JSW) at follow-up in Study II. Measurements for each subject's dysplastic and non-dysplastic hip are connected by a black line. Mean values are indicated by horizontal lines.

There was no indication that hip dysplasia led to earlier development of OA in dysplastic hips compared to non-dysplastic hips. The mean time to OA for the 19 dysplastic hips that developed OA was 8.2 years (SD 5.2), compared to 9.2 years (SD 5.1) for the 16 non-dysplastic hips that developed OA (Figure 16). A paired-samples t-test, limited to the 12 subjects who developed bilateral OA, showed a mean difference of -1.2 years, 95% CI -4.8–2.4, p=0.47. Student's t-test for independent samples comparing all dysplastic and non-dysplastic hips that developed OA showed a mean difference of -1.5 years, 95% CI -5.0–2.1, p=0.41. The median time to THR was 5.8 years (IQR 4.4–8.6) for dysplastic hips and 10.7 years (IQR 4.5–15.2) for non-dysplastic hips. A comparison analysis was not considered relevant, as only three subjects had undergone bilateral THR.

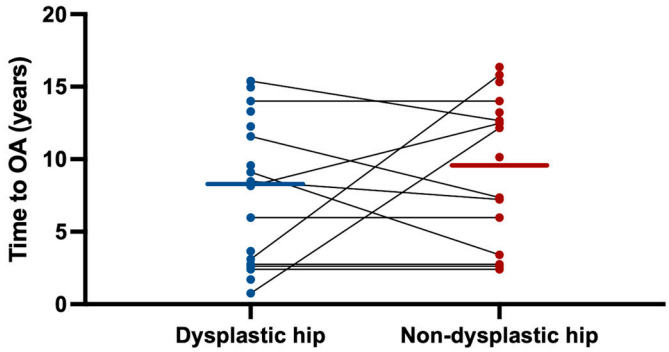

Figure 16: Univariate scatterplot for time to osteoarthritis (OA) in Study II.
The values of each subject's dysplastic and non-dysplastic hip are connected by a black line. Mean values are indicated by horizontal lines.

Hip dysplasia as a risk factor for hip osteoarthritis

The magnitude of association between hip dysplasia and development of hip OA was investigated in Studies III and IV. The inter- and intra-observer reliability analyses for the radiographic measurements in these studies have been previously published, with ICCs showing excellent (>0.9) reliability for both inter- and intra-observer measurements of the LCEA and ACEA (83). Additionally, an average PABAK of 0.8 (0.71–0.91) indicated substantial inter-observer reliability for the K&L grades (98). The OA incidence rates are presented in Table 16.

Table 16: Osteoarthritis (OA) incidence in Studies III and IV.

		OA incidence			
		Year 2	**Year 5**	**Year 8**	**Year 10**
Study III	**Radiographic hip OA**	5.5% (69/1253)	14% (178/1262)	24% (279/1188)	42% (495/1169)
Study IV	**Radiographic hip OA**				45.9% (215/468)
	Clinically relevant hip OA				31% (145/468)

Association between hip dysplasia and radiographic OA at different time points

In Study III, we analyzed the association between hip dysplasia and incident radiographic hip OA at four time points, using three different definitions of hip OA based on lateral and/or anterior coverage. Not all definitions of hip dysplasia were associated with incident radiographic hip OA at every time point, as shown in Table

17. In cases where a statistically significant association was observed at the 2- or 5-year follow-up, the association weakened at the 8-year follow-up and eventually disappeared by the 10-year follow-up. This pattern of diminishing association over time was most pronounced for the definition of hip dysplasia that combined LCEA and ACEA <25°.

Table 17: Association between hip dysplasia and radiographic hip osteoarthritis (OA) in Study III. Hip dysplasia was defined in three different ways and the association was studied at four time points. Statistically significant associations are shown in bold.

	Incident radiographic hip OA Adjusted OR (95% CI)			
Study III	**Year 2**	**Year 5**	**Year 8**	**Year 10**
LCEA <25°	1.69 (0.90–3.16)	1.44 (0.90–2.30)	**1.56 (1.08–2.26)**	1.21 (0.86–1.69)
ACEA <25°	1.93 (0.93–4.01)	**2.07 (1.28–3.34)**	**1.86 (1.22–2.84)**	1.11 (0.75–1.66)
LCEA & ACEA <25°	**2.46 (1.00–6.04)**	**2.43 (1.25–4.76)**	**1.88 (1.03–4.42)**	1.29 (0.71–2.35)

Association between different definitions of hip dysplasia and radiographic OA

In Study III, we found that the combination of an LCEA and ACEA <25° showed the strongest and most consistent association with radiographic hip OA at all time points compared to either an LCEA <25° or an ACEA <25° alone (Table 17).

Association between hip dysplasia and clinically relevant hip OA

In Study IV, we found that hip dysplasia was associated with clinically relevant hip OA (adjusted OR 2.8, 95% CI 1.15–6.79), but not with incident radiographic OA (adjusted OR 0.78, 95% CI 0.26–2.30) after 10 years follow-up.

Discussion

Main findings

The overall aim of this thesis was to improve the understanding of the prevalence and consequences of adult hip dysplasia. In a retrospective, cross-sectional review of radiographic records, we found hip dysplasia to be present in 5.2% of our Swedish study population, with only 7% of cases commented upon in the radiology reports (Study I). This prevalence study provided a cohort of individuals with hip dysplasia, from which we selected unilateral cases for a longitudinal review of their radiographic records. We found no evidence suggesting that radiographic OA developed earlier or more frequently in dysplastic hips compared to contralateral non-dysplastic hips (Study II).

Next, we prospectively examined the association between hip dysplasia and incident radiographic hip OA at four time points, using three different definitions of hip dysplasia, in a multicenter cohort study in the Netherlands. The highest increased risk (2.5-fold) was observed at the 2-year follow-up. Statistically significant associations at the 2- and 5-year follow-ups weakened by the 8-year follow-up and eventually disappeared by the 10-year follow-up. The combination of an LCEA and an ACEA <25° showed the strongest and most consistent association with incident radiographic hip OA, compared to either an LCEA <25° or an ACEA <25° alone (Study III).

Although no statistically significant association between hip dysplasia and radiographic hip OA was found at the 10-year follow-up, we observed an almost threefold increased risk for clinically relevant hip OA, as assessed by experts using both radiographic and clinical data (Study IV).

Interpretations and implications

Prevalence of hip dysplasia

A long-standing tradition of clinical screening of DDH, combined with the assumption that adult hip dysplasia is a consequence of undiagnosed DDH, may have led to the misconception that adult hip dysplasia is not a concern in Sweden. However, Study I suggests that adult dysplasia may affect as much as 5% of the Swedish population.

We calculated this prevalence based on an LCEA ≤20°, measured to the sourcil, consistent with the definition used in Jacobsen's Danish study (49). Jacobsen reported a prevalence of 5% in both men and women with baseline characteristics similar to those in our population. In contrast, Engesaeter's study of 19-year-old Norwegian participants reported a slightly lower prevalence of 3% (50). This discrepancy may be attributed to methodological differences, including measuring the LCEA to the bony edge of the acetabulum rather than to the sourcil, as well as using a slightly stricter cutoff of <20°. Interestingly, we would expect a higher prevalence in a younger population, as older individuals with hip dysplasia are more likely to be excluded from prevalence studies due to the development of OA.

Other previous studies have reported varying prevalence results, but direct comparisons with our study are complicated by differences in methodology. Some studies include populations with different ethnic backgrounds (47, 48, 53, 56), while others focus on specific subgroups, such as senior athletes (57). Additionally, some studies measured the LCEA on urograms rather than AP pelvic radiographs, which may contribute to variability in the reported prevalence (48, 54, 55). Despite these differences, our results appear to be representative. A recent meta-analysis, which pooled data from 11 studies and 12000 individuals, found that 6% of the general population had an LCEA <20° (100).

We also calculated the prevalence of an LCEA ≤25° in our cohort, which resulted in a prevalence of 21%, consistent with previous findings (100). While there is no universally accepted cutoff for dysplasia, defining it to include 20% of the population raises questions about its clinical relevance. Based on my experience in measuring the center-edge angle, I recommend not relying too strictly on these cutoffs in clinical practice, as small adjustments can significantly alter the angle. In borderline cases with hip symptoms, it is likely more important to further evaluate the hip morphology and rule out other potential causes of the symptoms, rather than focusing on whether the LCEA is 21° or 19°. For research purposes, I appreciate studies that report multiple cutoffs and provide detailed measurement protocols.

Mention of hip dysplasia in radiology report

Our results from Study I suggest that hip dysplasia is often overlooked by radiologists in Swedish healthcare. Underreading is a common radiological error, particularly in the musculoskeletal section (101). These findings indicate that many adults with hip dysplasia likely face an unmet need for proper diagnosis and information in today's healthcare system, highlighting the need for increased awareness of hip dysplasia among both clinicians and radiologists.

A recent Swedish study presented a screening project aimed at detecting hip dysplasia in teenagers and young adults. Radiographs of patients aged 12–44, referred for elective hip radiography, were screened by radiologists in clinical practice using a pre-specified algorithm. After the screening project was implemented, three times as many patients were diagnosed with hip dysplasia. This study demonstrates that greater awareness and knowledge of hip dysplasia can significantly improve its identification (102).

Hip dysplasia and hip osteoarthritis

Development of radiographic hip OA in a hip dysplasia cohort

In Study II, we found no evidence suggesting that radiographic OA developed earlier or more frequently in dysplastic hips compared to contralateral non-dysplastic hips in individuals with unilateral hip dysplasia.

Our findings are in line with results from a prospective case-control study that reported no difference in change in minimum JSW between dysplastic hips (LCEA ≤20°) and control hips (≥25°). The study's 81 dysplastic participants had comparable baseline characteristics and follow-up time as our cohort and had no history of DDH (79).

In contrast, a retrospective study reported that dysplastic hips (LCEA <25°) had a higher risk of OA progression compared with hips with normal morphology (≥25°). The study population consisted of individuals under 55 years age who had undergone unilateral THR due to OA in the contralateral hip. The age and sex distribution of their participants were comparable to ours, but the mean follow-up was longer, and it was not stated if their participants had a history of DDH. Compared to our cohort, their study population likely had an increased risk of development and progression of OA as they had already undergone THR due to OA in the contralateral hip (94).

Other longitudinal studies exploring OA development in adult cohorts with hip dysplasia have methodological limitations. They lack control groups, and some include subjects with a history of childhood DDH or pre-existing OA at baseline, which may have confounded their results (89, 91-93).

In our dysplasia cohort in Study II, approximately one third of both dysplastic and non-dysplastic hips developed incident radiographic hip OA. It is difficult to place these incidence estimates into a broader context, as study populations and OA definitions vary across previous studies on OA incidence (66). For instance, in a cohort representative of the general population aged 55 years and older, with a mean baseline age of 66 years (SD 6.5), one fifth of participants developed radiographic OA after seven years of follow-up (76). In contrast, nearly half of the hips in middle-aged

participants seeking consultation for recent onset of hip pain developed radiographic hip OA 10 years from baseline in Study IV.

In perspective, our results indicate that there is no need to monitor dysplastic hips for radiographic OA more extensively than the non-dysplastic hips in adults with unilateral hip dysplasia.

Association with radiographic hip OA

To the best of our knowledge, Study III is the first to investigate the association between hip dysplasia and radiographic hip OA at multiple time points. Our findings suggest that hip dysplasia is a significant risk factor in the short term; however, this association diminishes and eventually disappears after longer follow-up. These results are consistent with previous studies that have reported stronger associations in younger populations (84), and may also explain why some studies have failed to identify statistically significant associations (82). Nevertheless, hip dysplasia remains a moderately strong risk factor for radiographic hip OA in studies with shorter follow-up durations. This was recently confirmed in a meta-analysis of 19000 hips followed for 4–8 years, which found an adjusted OR of 2.0 (95% CI for 1.3–3.0) for an LCEA ≤20° and 1.8 (95% CI 1.4–2.3) for an LCEA ≤25°. Our findings emphasize the importance of early detection of hip dysplasia, particularly if treatments are to be implemented before OA develops. This is particularly relevant for participants being considered for a PAO, as preoperative early OA has been shown to significantly predict early postoperative failure (103).

In Study III, we also demonstrated that the association between hip dysplasia and radiographic hip OA strengthened when the ACEA was included in the definition of hip dysplasia. This finding, previously reported in the 5-year results from the CHECK-study (83), has now been confirmed across 2-, 5-, and 8-year follow-ups as well. These results suggest that using both FP and AP pelvic radiographs to assess acetabular coverage of the femoral head can help identify dysplastic individuals who are at especially high risk for OA development.

Association with clinically relevant hip OA

Study IV is unique in its investigation of the association between hip dysplasia and hip OA, using both radiographic and clinical data to define OA. The study found that middle-aged participants with hip dysplasia, who sought consultation for newly developed hip pain, had an increased long-term risk of clinically relevant hip OA, but not of incident radiographic hip OA.

When studying the association between hip dysplasia and OA, it is reasonable to include a radiographic definition of OA as it is more objective and enables comparison with previous studies. However, symptoms and clinical findings play a substantial role in diagnosing OA in clinical practice and should therefore be considered in research definitions as well. Furthermore, our results demonstrate that the long-term association between hip dysplasia and hip OA may be overlooked unless a clinically relevant definition is used to define hip OA.

Limitations

Study I

The primary limitation of Study I is that the study population consists of patients who actively sought medical care, rather than a random sample from the general population. However, we believe that the similar prevalences observed between individuals who underwent radiographic examination due to trauma and those examined for non-trauma reasons suggest that our sample reflects the general population. Another limitation is that OA was not assessed using a standardized scoring system. In Study II, we reassessed the subgroup of individuals with unilateral hip dysplasia using the K&L classification and found that only one hip in 98 individuals was misclassified as not having OA, despite having OA (K&L ≥2) at baseline.

Study II

The assessment of hip OA in Study II is limited to findings available in the radiographic records. This approach may have missed clinically relevant hip OA without obvious radiographic findings, as well as radiographic hip OA for which radiographs were unavailable. However, we only included imaging where the hips were bilaterally visualized, ensuring that the availability of imaging affected dysplastic and non-dysplastic hips equally.

Additionally, the non-dysplastic hips had a median LCEA of 25° (IQR 28–23) which is lower than normative values reported in previous population-based studies (31, 104) and falls within the range that some authors classify as borderline hip dysplasia. Therefore, the findings may not be generalizable to populations with unilateral hip dysplasia in which the non-dysplastic hip has higher acetabular coverage. However, our separate analysis of OA incidence for hips with an LCEA between 21° and 25° versus hips with an LCEA above 25° revealed no evidence of different outcomes between the two groups.

Study III

An important limitation of Study III is the inability to construct a horizontal reference line based on anatomical landmarks when measuring the ACEA on FP radiographs. Instead, the horizontal line is defined by the horizontal axis of the radiographic film. Despite this limitation, the obtained ACEA appears to accurately reflect acetabular undercoverage, as it strengthens the association with hip OA development.

Study IV

In Study IV, we applied strict inclusion criteria and a strict cutoff for the LCEA to ensure high clinical relevance. As a result, the prevalence and absolute number of hips with hip dysplasia were relatively low (3.6%, n=17). Studying an exposure with low prevalence leads to a loss of precision in risk estimates (105), which likely explains the relatively wide confidence interval for the risk estimates of hip dysplasia in clinically relevant hip OA (adjusted OR 2.8, 95% CI 1.15–6.79).

Lastly, the expert diagnosis of clinically relevant hip OA may be difficult to reproduce, and one could argue that the well-established ACR criteria should have been used instead. However, as discussed in the introduction, the value of the ACR criteria has been found to be limited in both research and clinical practice (67). This is evident in the CHECK cohort, where the ACR criteria were met by 16% of participants at both baseline and the 10-year follow-up, but only 4% fulfilled the criteria at all time points (106). Our expert-based definition of clinically relevant OA relied on extensive clinical and radiographic data over time, closely resembling real-life assessments. Ultimately, the aim of Study IV was to obtain clinically relevant results, rather than reproducible ones.

Conclusions

Our results indicate that the prevalence of adult hip dysplasia in the Swedish population is 5%, and that it is often overlooked by radiologists, highlighting the need for greater awareness of the condition within Swedish healthcare (Study I).

The association between adult hip dysplasia and the development of radiographic hip OA may not be as strong as previously reported. We found no evidence that radiographic OA develops earlier or more frequently in dysplastic hips compared to contralateral non-dysplastic hips, suggesting that dysplastic hips in individuals with unilateral hip dysplasia can be radiographically monitored in the same way as the contralateral non-dysplastic hips (Study II). Furthermore, our results indicate that the

association between hip dysplasia and radiographic hip OA weakens and eventually diminishes with increasing age. However, in the short-term, the association becomes more pronounced when the acetabular undercoverage is present both anteriorly and laterally. These findings suggest that early detection of hip dysplasia and the incorporation of FP hip radiographs into the diagnostic process could help identify individuals at higher risk of developing radiographic OA (Study III).

Although we found no long-term association between hip dysplasia and development of radiographic hip OA, we observed a threefold increased risk for clinically relevant hip OA. These findings highlight the importance of using clinically relevant definitions for hip OA in future research on hip dysplasia to avoid underestimating or overlooking the association (Study IV).

Future directions

First, it is essential to investigate when during skeletal maturation adult hip dysplasia develops and whether there is a link between DDH and adult hip dysplasia. These questions are being explored by the "HIPSTAR" project, led by Erasmus Medical Center in Rotterdam. The cohort includes 8000 children followed from fetal age to adulthood, with serial radiographic examinations performed throughout their growth.

Hip dysplasia is a complex, three-dimensional condition that we attempt to define using two-dimensional measurements. As a result, the research community may never universally agree on a single definition of hip dysplasia that can be applied to plain radiographs. In recent years, the LCEA measured to the sourcil has emerged as the most commonly used method. However, the choice of cutoff values remains variable, as seen in this thesis, where we used both 20° and 25°. The role of anterior undercoverage warrants further investigation, as much of the existing dysplasia research focuses on lateral undercoverage. Regardless of how hip dysplasia is defined, it is crucial to clearly state the selected definition, and, ideally, perform sensitivity analyses with alternative definitions.

This thesis highlights the need for increased awareness of adult hip dysplasia. Healthcare systems should adopt systematic screening programs, such as the one described by Møse et al (102). Increased radiographic identification also requires well-functioning clinics capable of further evaluation and management of these individuals. The assessment of young adults with hip pain and radiographic signs of hip dysplasia is complex and should ideally be managed by orthopedic surgeons who specialize in hip conditions, as exemplified by the "Youth Hip Clinic" in the study by Møse et al.

Next, the question arises regarding how symptomatic adults with hip dysplasia should be treated. No previous studies have compared conservative treatment with surgical intervention for this patient group. This knowledge gap may soon be addressed by an ongoing randomized controlled trial comparing PAO combined with physiotherapy to physiotherapy alone, focusing on changes in patient-reported symptoms (21).

Much research remains to be done to understand the natural history of adult hip dysplasia. Long-term prospective observational studies are required to track the development of OA in individuals diagnosed with hip dysplasia at a young adult age. Preferably, these studies should include both symptomatic and asymptomatic participants, as well as control groups. Finally, I suggest that future studies incorporate clinically relevant definitions of OA when investigating hip dysplasia.

Acknowledgements

Carl Johan Tiderius, thank you for your unwavering belief in me and for being so generous with your time, guidance and thoughts. Both my research and clinical career have been shaped with you by my side, and I am truly grateful for the support you have given me. You have consistently challenged me – though it was sometimes difficult, it has been incredibly rewarding. Most importantly, you have helped me recognize the small victories along the way and reminded me to celebrate them. I look forward to continuing to work with you, both as a colleague and a friend.

Jos Runhaar, thank you for the patience and support you have shown me ever since I first visited you in Rotterdam as a curious medical student taking my first steps as a researcher. I have learned so much from you, and it has been a truly rewarding experience to collaborate with you and your impressive department.

Cecilia Rogmark, thank you for trusting me to carry out the prevalence study and for having the patience to allow me to make my own mark on it. I appreciate that you have helped me see new perspectives and that you have always been available to offer guidance on research questions beyond our collaborations.

Daniel Wenger, thank you for contributing with your wisdom and attention to detail. Your genuine interest and curiosity for research is contagious, and collaborating with you has been a privilege.

A big thank you to the remaining members of my research group – **Adam Sand**, **Amanda Maripuu**, **Jens Nilsson,** and **Jakob Örtegren**. I am grateful for our teamwork on grant applications and research discussions, and I look forward to future collaborations. It's an honor to work alongside each of you.

Thank you to my co-authors, **Sita Bierma-Zeinstra, Rintje Agricola, Noortje Riedstra** and **Søren Overgaard,** for everything you have contributed with.

Thank you to **Jan-Åke Nilsson** and **Aleksandra Turkiewicz** for your invaluable statistical advice.

Mom and Dad, thank you for always being so positive and supportive, and for all the time you have spent with Vendela so that I could focus on my thesis.

Vendela, thank you for being the light of my life and a never-ending source of joy. I feel incredibly lucky to be your mom.

Fredrik, you have carried our family and our home throughout this process with such grace, all while being my constant source of support. My gratitude for everything you have done for me knows no bounds. I love you.

References

1. van Bosse H, Wedge JH, Babyn P. How are dysplastic hips different? A three-dimensional CT study. Clin Orthop Relat Res. 2015;473(5):1712-23.
2. Wiberg G. Studies on dysplastic acetabula and congenital subluxation of the hip. With special reference to the complication of osteoarthritis. Acta Chir Scand. 1939;83(58):5-135.
3. Henak CR, Abraham CL, Anderson AE, Maas SA, Ellis BJ, Peters CL, et al. Patient-specific analysis of cartilage and labrum mechanics in human hips with acetabular dysplasia. Osteoarthritis Cartilage. 2014;22(2):210-7.
4. Clohisy JC, Nunley RM, Carlisle JC, Schoenecker PL. Incidence and characteristics of femoral deformities in the dysplastic hip. Clin Orthop Relat Res. 2009;467(1):128-34.
5. Klisic PJ. Congenital dislocation of the hip--a misleading term: brief report. The Journal of bone and joint surgery British volume. 1989;71(1):136.
6. Wenger D, Düppe H, Tiderius CJ. Acetabular dysplasia at the age of 1 year in children with neonatal instability of the hip. Acta Orthop. 2013;84(5):483-8.
7. Lee CB, Mata-Fink A, Millis MB, Kim YJ. Demographic differences in adolescent-diagnosed and adult-diagnosed acetabular dysplasia compared with infantile developmental dysplasia of the hip. J Pediatr Orthop. 2013;33(2):107-11.
8. de Hundt M, Vlemmix F, Bais JM, Hutton EK, de Groot CJ, Mol BW, et al. Risk factors for developmental dysplasia of the hip: a meta-analysis. Eur J Obstet Gynecol Reprod Biol. 2012;165(1):8-17.
9. Tirta M, Rahbek O, Kold S, Husum HC. Risk Factors for Developmental Dysplasia of the Hip Before 3 Months of Age: A Meta-Analysis. JAMA Netw Open. 2025;8(1):e2456153.
10. Krysta W, Dudek P, Pulik Ł, Łęgosz P. Screening of Developmental Dysplasia of the Hip in Europe: A Systematic Review. Children (Basel). 2024;11(1).
11. Barlow TG. EARLY DIAGNOSIS AND TREATMENT OF CONGENITAL DISLOCATION OF THE HIP. The Journal of Bone & Joint Surgery British Volume. 1962;44-B(2):292-301.
12. Ortolani M. Congenital hip dysplasia in the light of early and very early diagnosis. Clin Orthop Relat Res. 1976(119):6-10.
13. Dahlström H, Oberg L, Friberg S. Sonography in congenital dislocation of the hip. Acta orthopaedica Scandinavica. 1986;57(5):402-6.
14. Graf R. Classification of hip joint dysplasia by means of sonography. Arch Orthop Trauma Surg (1978). 1984;102(4):248-55.

15. Wenger D, Düppe H, Nilsson J, Tiderius CJ. Incidence of Late-Diagnosed Hip Dislocation After Universal Clinical Screening in Sweden. JAMA Netw Open. 2019;2(11):e1914779.
16. Raimann A, Baar A, Raimann R, Morcuende JA. Late developmental dislocation of the hip after initial normal evaluation: a report of five cases. J Pediatr Orthop. 2007;27(1):32-6.
17. Wenger D, Tiderius CJ, Düppe H. Estimated effect of secondary screening for hip dislocation. Arch Dis Child. 2020;105(12):1175-9.
18. Wedge JH, Wasylenko MJ. The natural history of congenital dislocation of the hip: a critical review. Clin Orthop Relat Res. 1978(137):154-62.
19. Wenger D, Siversson C, Dahlberg LE, Tiderius CJ. Residual hip dysplasia at 1 year after treatment for neonatal hip instability is not related to degenerative joint disease in young adulthood: a 21-year follow-up study including dGEMRIC. Osteoarthritis Cartilage. 2016;24(3):436-42.
20. Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome. A clinical presentation of dysplasia of the hip. The Journal of bone and joint surgery British volume. 1991;73(3):423-9.
21. Reimer LCU, Jakobsen SS, Mortensen L, Dalgas U, Jacobsen JS, Soballe K, et al. Efficacy of periacetabular osteotomy followed by progressive resistance training compared to progressive resistance training as non-surgical treatment in patients with hip dysplasia (PreserveHip) - a protocol for a randomised controlled trial. BMJ Open. 2019;9(12):e032782.
22. Nunley RM, Prather H, Hunt D, Schoenecker PL, Clohisy JC. Clinical presentation of symptomatic acetabular dysplasia in skeletally mature patients. J Bone Joint Surg Am. 2011;93 Suppl 2:17-21.
23. Willey M, Holland T, Thomas-Aitken H, Goetz JE. Diagnosis and Management of Borderline Hip Dysplasia and Acetabular Retroversion. J Hip Surg. 2018;2(4):156-66.
24. Troelsen A. Assessment of adult hip dysplasia and the outcome of surgical treatment. Dan Med J. 2012;59(6):B4450.
25. Nepple JJ, Wells J, Ross JR, Bedi A, Schoenecker PL, Clohisy JC. Three Patterns of Acetabular Deficiency Are Common in Young Adult Patients With Acetabular Dysplasia. Clin Orthop Relat Res. 2017;475(4):1037-44.
26. Sørensen H, Nielsen DB, Jacobsen JS, Søballe K, Mechlenburg I. Isokinetic dynamometry and gait analysis reveal different hip joint status in patients with hip dysplasia. Hip Int. 2019;29(2):215-21.
27. Hoppe DJ, Truntzer JN, Shapiro LM, Abrams GD, Safran MR. Diagnostic Accuracy of 3 Physical Examination Tests in the Assessment of Hip Microinstability. Orthopaedic journal of sports medicine. 2017;5(11):2325967117740121.
28. Engelbert RH, Rombaut L. Clinimetrics: Assessment of generalised joint hypermobility: the Beighton score. J Physiother. 2022;68(3):208.

29. Byrd JW, Jones KS. Diagnostic accuracy of clinical assessment, magnetic resonance imaging, magnetic resonance arthrography, and intra-articular injection in hip arthroscopy patients. The American journal of sports medicine. 2004;32(7):1668-74.
30. Kappe T, Kocak T, Reichel H, Fraitzl CR. Can femoroacetabular impingement and hip dysplasia be distinguished by clinical presentation and patient history? Knee surgery, sports traumatology, arthroscopy : official journal of the ESSKA. 2012;20(2):387-92.
31. Laborie LB, Engesaeter IO, Lehmann TG, Sera F, Dezateux C, Engesaeter LB, et al. Radiographic measurements of hip dysplasia at skeletal maturity--new reference intervals based on 2,038 19-year-old Norwegians. Skeletal radiology. 2013;42(7):925-35.
32. Mittal A, Bomar JD, Jeffords ME, Huang MT, Wenger DR, Upasani VV. Defining the lateral edge of the femoroacetabular articulation: correlation analysis between radiographs and computed tomography. Journal of children's orthopaedics. 2016;10(5):365-70.
33. Werner CM, Ramseier LE, Ruckstuhl T, Stromberg J, Copeland CE, Turen CH, et al. Normal values of Wiberg's lateral center-edge angle and Lequesne's acetabular index--a coxometric update. Skeletal radiology. 2012;41(10):1273-8.
34. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis--what the radiologist should know. AJR American journal of roentgenology. 2007;188(6):1540-52.
35. Ogata S, Moriya H, Tsuchiya K, Akita T, Kamegaya M, Someya M. Acetabular cover in congenital dislocation of the hip. The Journal of bone and joint surgery British volume. 1990;72(2):190-6.
36. Chadayammuri V, Garabekyan T, Jesse MK, Pascual-Garrido C, Strickland C, Milligan K, et al. Measurement of lateral acetabular coverage: a comparison between CT and plain radiography. Journal of hip preservation surgery. 2015;2(4):392-400.
37. Omeroglu H, Bicimoglu A, Agus H, Tumer Y. Measurement of center-edge angle in developmental dysplasia of the hip: a comparison of two methods in patients under 20 years of age. Skeletal radiology. 2002;31(1):25-9.
38. Lequesne MG, Laredo JD. The faux profil (oblique view) of the hip in the standing position. Contribution to the evaluation of osteoarthritis of the adult hip. Ann Rheum Dis. 1998;57(11):676-81.
39. Herfkens J, van Buuren MMA, Riedstra NS, Verhaar JAN, Mascarenhas VV, Agricola R. Adding false-profile radiographs improves detection of developmental dysplasia of the hip, data from the CHECK cohort. Journal of hip preservation surgery. 2022.
40. McClincy MP, Wylie JD, Yen YM, Novais EN. Mild or Borderline Hip Dysplasia: Are We Characterizing Hips With a Lateral Center-Edge Angle Between 18° and 25° Appropriately? The American journal of sports medicine. 2019;47(1):112-22.
41. Boel F, de Vos-Jakobs S, Riedstra NS, Lindner C, Runhaar J, Bierma-Zeinstra SMA, et al. Automated radiographic hip morphology measurements: An open-access method. Osteoarthr Imaging. 2024;4(2):100181.

42. Tonnis D. Normal values of the hip joint for the evaluation of X-rays in children and adults. Clin Orthop Relat Res. 1976(119):39-47.
43. Jacobsen S, Sonne-Holm S, Lund B, Soballe K, Kiaer T, Rovsing H, et al. Pelvic orientation and assessment of hip dysplasia in adults. Acta orthopaedica Scandinavica. 2004;75(6):721-9.
44. Laborie LB, Lehmann TG, Engesaeter IO, Eastwood DM, Engesaeter LB, Rosendahl K. Prevalence of radiographic findings thought to be associated with femoroacetabular impingement in a population-based cohort of 2081 healthy young adults. Radiology. 2011;260(2):494-502.
45. Tannast M, Hanke MS, Zheng G, Steppacher SD, Siebenrock KA. What are the radiographic reference values for acetabular under- and overcoverage? Clin Orthop Relat Res. 2015;473(4):1234-46.
46. Tannast M, Fritsch S, Zheng G, Siebenrock KA, Steppacher SD. Which radiographic hip parameters do not have to be corrected for pelvic rotation and tilt? Clin Orthop Relat Res. 2015;473(4):1255-66.
47. Kim CH, Park JI, Shin DJ, Oh SH, Jeong MY, Yoon PW. Prevalence of radiologic acetabular dysplasia in asymptomatic Asian volunteers. Journal of hip preservation surgery. 2019;6(1):55-9.
48. Inoue K, Wicart P, Kawasaki T, Huang J, Ushiyama T, Hukuda S, et al. Prevalence of hip osteoarthritis and acetabular dysplasia in french and japanese adults. Rheumatology (Oxford). 2000;39(7):745-8.
49. Jacobsen S, Sonne-Holm S. Hip dysplasia: a significant risk factor for the development of hip osteoarthritis. A cross-sectional survey. Rheumatology (Oxford). 2005;44(2):211-8.
50. Engesaeter IO, Laborie LB, Lehmann TG, Fevang JM, Lie SA, Engesaeter LB, et al. Prevalence of radiographic findings associated with hip dysplasia in a population-based cohort of 2081 19-year-old Norwegians. The bone & joint journal. 2013;95-b(2):279-85.
51. Kennedy JW, Brydone AS, Meek DR, Patil SR. Delays in diagnosis are associated with poorer outcomes in adult hip dysplasia. Scott Med J. 2017;62(3):96-100.
52. Lerch TD, Steppacher SD, Liechti EF, Tannast M, Siebenrock KA. One-third of Hips After Periacetabular Osteotomy Survive 30 Years With Good Clinical Results, No Progression of Arthritis, or Conversion to THA. Clin Orthop Relat Res. 2017;475(4):1154-68.
53. Raveendran R, Stiller JL, Alvarez C, Renner JB, Schwartz TA, Arden NK, et al. Population-based prevalence of multiple radiographically-defined hip morphologies: the Johnston County Osteoarthritis Project. Osteoarthritis Cartilage. 2018;26(1):54-61.
54. Croft P, Cooper C, Wickham C, Coggon D. Osteoarthritis of the hip and acetabular dysplasia. Ann Rheum Dis. 1991;50(5):308-10.

55. Smith RW, Egger P, Coggon D, Cawley MI, Cooper C. Osteoarthritis of the hip joint and acetabular dysplasia in women. Ann Rheum Dis. 1995;54(3):179-81.
56. Umer M, Sepah YJ, Asif S, Azam I, Jawad MU. Acetabular morphometry and prevalence of hip dysplasia in the South Asian population. Orthop Rev (Pavia). 2009;1(1):e10.
57. Anderson LA, Anderson MB, Kapron A, Aoki SK, Erickson JA, Chrastil J, et al. The 2015 Frank Stinchfield Award: Radiographic Abnormalities Common in Senior Athletes With Well-functioning Hips but Not Associated With Osteoarthritis. Clin Orthop Relat Res. 2016;474(2):342-52.
58. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. Clin Orthop Relat Res. 1988(232):26-36.
59. Wyles CC, Vargas JS, Heidenreich MJ, Mara KC, Peters CL, Clohisy JC, et al. Natural History of the Dysplastic Hip Following Modern Periacetabular Osteotomy. J Bone Joint Surg Am. 2019;101(10):932-8.
60. Lohmander LS, Engesaeter LB, Herberts P, Ingvarsson T, Lucht U, Puolakka TJ. Standardized incidence rates of total hip replacement for primary hip osteoarthritis in the 5 Nordic countries: similarities and differences. Acta Orthop. 2006;77(5):733-40.
61. Murphy LB, Helmick CG, Schwartz TA, Renner JB, Tudor G, Koch GG, et al. One in four people may develop symptomatic hip osteoarthritis in his or her lifetime. Osteoarthritis Cartilage. 2010;18(11):1372-9.
62. Turkiewicz A, Petersson IF, Björk J, Hawker G, Dahlberg LE, Lohmander LS, et al. Current and future impact of osteoarthritis on health care: a population-based study with projections to year 2032. Osteoarthritis Cartilage. 2014;22(11):1826-32.
63. Glyn-Jones S, Palmer AJR, Agricola R, Price AJ, Vincent TL, Weinans H, et al. Osteoarthritis. The Lancet. 2015;386(9991):376-87.
64. Hunter DJ, Bierma-Zeinstra S. Osteoarthritis. Lancet (London, England). 2019;393(10182):1745-59.
65. Murphy NJ, Eyles JP, Hunter DJ. Hip Osteoarthritis: Etiopathogenesis and Implications for Management. Advances in therapy. 2016;33(11):1921-46.
66. Pereira D, Peleteiro B, Araujo J, Branco J, Santos RA, Ramos E. The effect of osteoarthritis definition on prevalence and incidence estimates: a systematic review. Osteoarthritis Cartilage. 2011;19(11):1270-85.
67. Cibere J. Do we need radiographs to diagnose osteoarthritis? Best practice & research Clinical rheumatology. 2006;20(1):27-38.
68. Casartelli NC, Maffiuletti NA, Valenzuela PL, Grassi A, Ferrari E, van Buuren MMA, et al. Is hip morphology a risk factor for developing hip osteoarthritis? A systematic review with meta-analysis. Osteoarthritis Cartilage. 2021;29(9):1252-64.

69. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957;16(4):494-502.
70. Croft P, Cooper C, Wickham C, Coggon D. Defining osteoarthritis of the hip for epidemiologic studies. Am J Epidemiol. 1990;132(3):514-22.
71. Kohn MD, Sassoon AA, Fernando ND. Classifications in Brief: Kellgren-Lawrence Classification of Osteoarthritis. Clin Orthop Relat Res. 2016;474(8):1886-93.
72. Doherty M, Courtney P, Doherty S, Jenkins W, Maciewicz RA, Muir K, et al. Nonspherical femoral head shape (pistol grip deformity), neck shaft angle, and risk of hip osteoarthritis: a case-control study. Arthritis Rheum. 2008;58(10):3172-82.
73. Chung CY, Park MS, Lee KM, Lee SH, Kim TK, Kim KW, et al. Hip osteoarthritis and risk factors in elderly Korean population. Osteoarthritis Cartilage. 2010;18(3):312-6.
74. Gosvig KK, Jacobsen S, Sonne-Holm S, Palm H, Troelsen A. Prevalence of malformations of the hip joint and their relationship to sex, groin pain, and risk of osteoarthritis: a population-based survey. J Bone Joint Surg Am. 2010;92(5):1162-9.
75. Jacobsen S, Sonne-Holm S, Soballe K, Gebuhr P, Lund B. Hip dysplasia and osteoarthrosis: a survey of 4151 subjects from the Osteoarthrosis Substudy of the Copenhagen City Heart Study. Acta Orthop. 2005;76(2):149-58.
76. Reijman M, Hazes JM, Pols HA, Koes BW, Bierma-Zeinstra SM. Acetabular dysplasia predicts incident osteoarthritis of the hip: the Rotterdam study. Arthritis Rheum. 2005;52(3):787-93.
77. Agricola R, Heijboer MP, Bierma-Zeinstra SM, Verhaar JA, Weinans H, Waarsing JH. Cam impingement causes osteoarthritis of the hip: a nationwide prospective cohort study (CHECK). Ann Rheum Dis. 2013;72(6):918-23.
78. Bouyer B, Mazieres B, Guillemin F, Bouttier R, Fautrel B, Morvan J, et al. Association between hip morphology and prevalence, clinical severity and progression of hip osteoarthritis over 3 years: The knee and hip osteoarthritis long-term assessment cohort results. Joint, bone, spine : revue du rhumatisme. 2016;83(4):432-8.
79. Jacobsen S, Sonne-Holm S, Søballe K, Gebuhr P, Lund B. Joint space width in dysplasia of the hip: a case-control study of 81 adults followed for ten years. The Journal of bone and joint surgery British volume. 2005;87(4):471-7.
80. Mavcic B, Iglic A, Kralj-Iglic V, Brand RA, Vengust R. Cumulative hip contact stress predicts osteoarthritis in DDH. Clin Orthop Relat Res. 2008;466(4):884-91.
81. Sankar WN, Beaulé PE, Clohisy JC, Kim YJ, Millis MB, Peters CL, et al. Labral morphologic characteristics in patients with symptomatic acetabular dysplasia. The American journal of sports medicine. 2015;43(9):2152-6.
82. Harris-Hayes M, Royer NK. Relationship of acetabular dysplasia and femoroacetabular impingement to hip osteoarthritis: a focused review. PM R. 2011;3(11):1055-67 e1.

83. Agricola R, Heijboer MP, Roze RH, Reijman M, Bierma-Zeinstra SM, Verhaar JA, et al. Pincer deformity does not lead to osteoarthritis of the hip whereas acetabular dysplasia does: acetabular coverage and development of osteoarthritis in a nationwide prospective cohort study (CHECK). Osteoarthritis Cartilage. 2013;21(10):1514-21.
84. Saberi Hosnijeh F, Zuiderwijk ME, Versteeg M, Smeele HT, Hofman A, Uitterlinden AG, et al. Cam Deformity and Acetabular Dysplasia as Risk Factors for Hip Osteoarthritis. Arthritis & rheumatology (Hoboken, NJ). 2017;69(1):86-93.
85. Thomas GE, Palmer AJ, Batra RN, Kiran A, Hart D, Spector T, et al. Subclinical deformities of the hip are significant predictors of radiographic osteoarthritis and joint replacement in women. A 20 year longitudinal cohort study. Osteoarthritis Cartilage. 2014;22(10):1504-10.
86. Iidaka T, Muraki S, Oka H, Horii C, Kawaguchi H, Nakamura K, et al. Incidence rate and risk factors for radiographic hip osteoarthritis in Japanese men and women: a 10-year follow-up of the ROAD study. Osteoarthritis Cartilage. 2020;28(2):182-8.
87. Lane NE, Lin P, Christiansen L, Gore LR, Williams EN, Hochberg MC, et al. Association of mild acetabular dysplasia with an increased risk of incident hip osteoarthritis in elderly white women: the study of osteoporotic fractures. Arthritis Rheum. 2000;43(2):400-4.
88. Nicholls AS, Kiran A, Pollard TC, Hart DJ, Arden CP, Spector T, et al. The association between hip morphology parameters and nineteen-year risk of end-stage osteoarthritis of the hip: a nested case-control study. Arthritis Rheum. 2011;63(11):3392-400.
89. Cooperman DR, Wallensten R, Stulberg SD. Acetabular dysplasia in the adult. Clin Orthop Relat Res. 1983(175):79-85.
90. Hasegawa Y, Iwata H, Mizuno M, Genda E, Sato S, Miura T. The natural course of osteoarthritis of the hip due to subluxation or acetabular dysplasia. Archives of orthopaedic and trauma surgery. 1992;111(4):187-91.
91. Amagami A, Sugiyama H, Tonotsuka H, Saito M. Long-term course of developmental dysplasia of the hip: follow-up of the non-operated hips of patients undergoing unilateral rotational acetabular osteotomy for twenty-four years. Archives of orthopaedic and trauma surgery. 2024;144(3):997-1004.
92. Hisatome T, Yasunaga Y, Tanaka R, Yamasaki T, Ishida O, Ochi M. Natural course of the minimally symptomatic nonoperated hip in patients with bilateral hip dysplasia treated with contralateral rotational acetabular osteotomy. J Orthop Sci. 2005;10(6):574-80.
93. Murphy SB, Ganz R, Müller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict the outcome. J Bone Joint Surg Am. 1995;77(7):985-9.
94. Wyles CC, Heidenreich MJ, Jeng J, Larson DR, Trousdale RT, Sierra RJ. The John Charnley Award: Redefining the Natural History of Osteoarthritis in Patients With Hip Dysplasia and Impingement. Clin Orthop Relat Res. 2017;475(2):336-50.

95. Agricola R, Reijman M, Bierma-Zeinstra SM, Verhaar JA, Weinans H, Waarsing JH. Total hip replacement but not clinical osteoarthritis can be predicted by the shape of the hip: a prospective cohort study (CHECK). Osteoarthritis Cartilage. 2013;21(4):559-64.
96. Runhaar J, Kloppenburg M, Boers M, Bijlsma JWJ, Bierma-Zeinstra SMA. Towards developing diagnostic criteria for early knee osteoarthritis: data from the CHECK study. Rheumatology (Oxford). 2021;60(5):2448-55.
97. Koo TK, Li MY. A Guideline of Selecting and Reporting Intraclass Correlation Coefficients for Reliability Research. J Chiropr Med. 2016;15(2):155-63.
98. Damen J, Schiphof D, Wolde ST, Cats HA, Bierma-Zeinstra SM, Oei EH. Inter-observer reliability for radiographic assessment of early osteoarthritis features: the CHECK (cohort hip and cohort knee) study. Osteoarthritis Cartilage. 2014;22(7):969-74.
99. Vinge R, Riedstra N, Tiderius CJ, Bierma-Zeinstra S, Agricola R, Runhaar J. Hip dysplasia as risk factor for clinically relevant and radiographic hip osteoarthritis: 10-year results from the CHECK cohort. Rheumatology (Oxford). 2025;64(1):149-55.
100. Freiman SM, Schwabe MT, Fowler L, Clohisy JC, Nepple JJ. Prevalence of Borderline Acetabular Dysplasia in Symptomatic and Asymptomatic Populations: A Systematic Review and Meta-analysis. Orthopaedic journal of sports medicine. 2022;10(2):23259671211040455.
101. Kim YW, Mansfield LT. Fool me twice: delayed diagnoses in radiology with emphasis on perpetuated errors. AJR American journal of roentgenology. 2014;202(3):465-70.
102. Møse FB, Mohseni S, Borg T. A pilot screening project for the detection of hip dysplasia in young patients. Journal of hip preservation surgery. 2024;11(3):176-81.
103. Cunningham T, Jessel R, Zurakowski D, Millis MB, Kim YJ. Delayed gadolinium-enhanced magnetic resonance imaging of cartilage to predict early failure of Bernese periacetabular osteotomy for hip dysplasia. J Bone Joint Surg Am. 2006;88(7):1540-8.
104. Leide R, Bohman A, Wenger D, Overgaard S, Tiderius CJ, Rogmark C. Hip dysplasia is not uncommon but frequently overlooked: a cross-sectional study based on radiographic examination of 1,870 adults. Acta Orthop. 2021;92(5):575-80.
105. Doerken S, Avalos M, Lagarde E, Schumacher M. Penalized logistic regression with low prevalence exposures beyond high dimensional settings. PLoS One. 2019;14(5):e0217057.
106. Schiphof D, Runhaar J, Waarsing JH, van Spil WE, van Middelkoop M, Bierma-Zeinstra SMA. The clinical and radiographic course of early knee and hip osteoarthritis over 10 years in CHECK (Cohort Hip and Cohort Knee). Osteoarthritis Cartilage. 2019;27(10):1491-500.

Printed by Media-Tryck, Lund 2025 NORDIC SWAN ECOLABEL 3041 0903

About the author

REBECKA VINGE, MD, is a resident in orthopedic surgery at Skåne University Hospital in Sweden. Her research focuses on the epidemiology and clinical significance of adult hip dysplasia, a condition characterized by insufficient acetabular coverage of the femoral head, which predisposes individuals to hip pain, functional impairment, and the development of osteoarthritis. Dr. Vinge's work aims to raise awareness of adult hip dysplasia and enhance understanding of its association with osteoarthritis, with the ultimate goal of contributing to earlier detection and improved long-term patient outcomes.

LUND UNIVERSITY

FACULTY OF MEDICINE

Department of Clinical Sciences

Lund University, Faculty of Medicine
Doctoral Dissertation Series 2025:63
ISBN 978-91-8021-716-3
ISSN 1652-8220