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# Hip Joint Restoration

## Worldwide Advances in Arthroscopy, Arthroplasty, Osteotomy and Joint Preservation Surgery

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# Preface

Those of us who treat patients with hip pain know that the surgical treatment of hip disease has undergone tremendous growth during the last decade. Prior books on the hip have either addressed arthroplasty, in providing prosthetic solutions for end-stage hip arthritis, or focused on minimally invasive arthroscopy of the hip. Many of them were technical treatises. Yet hip surgery has moved on greatly from the days when all that might be offered was fracture fixation or arthroplasty and, for the younger patient, the instruction to wait until they had reached sufficient age to justify a prosthesis. The thrust of most surgeons in the twenty-first century is to achieve as much benefit as possible for the patient while keeping surgical trauma to a minimum. Fulfillment of this ambition requires an understanding of new concepts and new procedures, as well as new training to accompany them.

The authors of this book believe that hip disease presents as a spectrum of symptoms and pathology, and so any comprehensive text must include the accurate diagnosis and treatment of both the biologic and the prosthetic hip. With the explosion of information on hip disease in the literature, particularly in the treatment of younger patients, the authors felt that it was time for a comprehensive treatise on this subject. Arthroscopy of the Hip, according to many, is the fastest growing specialty area within orthopedics. Accordingly, an extensive amount of this book is devoted to determining proper surgical indications as well as knowledge of surgical techniques and outcomes for the expanding number of surgical procedures in this area.

This book is divided into 16 parts. Pathology within the hip is best understood in contradistinction to normal growth and development. Early chapters also focus on discerning extra-articular from intra-articular etiologies of hip pain. Digital imaging, including CT, MRI, and ultrasound, has immensely increased our diagnostic understanding of the joint and the periarticular soft tissues. At times, MR imaging may disclose combined issues in pathology such as intra-articular loose bodies in combination with osteonecrosis of the hip or, similarly, an acetabular labral tear in combination with abductor muscle attenuation.

The spectrum of treatment of hip disease importantly includes hip osteotomies, whether of the femur or the acetabulum, or in combination. Knowledge of these procedures and their indications is a critical prerequisite for successful outcomes, especially in young patients. However, some young patients do require total hip arthroplasty, typically secondary to osteonecrosis, tumors, trauma, or collagen disease. Several chapters are devoted to the latest evidence-based information on bearing surfaces, and implant selection as well as surgical techniques.

A critically important area for increased understanding is patient outcomes following hip arthroscopy, osteotomy, or total joint replacement. Importantly, world experts in validated outcome measures and quality of life indicators are authors of chapters in this book. Another unique feature of this volume is a section describing the growth and development of hip arthroscopic surgery in each of the world's continents, authored by experts in each of these geographical areas. Finally, there is an entire section devoted to research and future developments. The robustness of the information as well as the development in these areas adds significantly to the depth of knowledge contained within this book.

There has never been such an exciting time to be a specialist in hip surgery, nor such a time to feel so proud. This book brings together a large number of specialists in the field, each of whom has given up valuable hours to prepare their text. As editors we are enormously grateful to them. Our authors are excellent clinicians, respected practitioners, but, more than anything, good personal friends. So join us on the tidal wave of surgical development shown on these pages, the tidal wave in the surgical treatment of hip disease.

In conclusion, this book has been a truly collaborative effort but would never have been possible without the tireless efforts of Connie Walsh, Miranda Finch and Kristopher Spring at Springer whose expertise, patience, and attention to detail have been vital. We also profusely thank our colleagues and fellow members of ISHA (The International Society of Hip Arthroscopy) who have pitched in as authors and section editors to share their knowledge and understanding of hip disease in making this work an important treatise. And finally, we thank our spouses and families for their support and understanding during this extensive endeavor.

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# Abbreviations

| | |
|---|---|
| ADL | Activities of daily living |
| AIIS | Anterior inferior iliac spine |
| ASIS | Anterior superior iliac spine |
| AVN | Avascular necrosis |
| BMP | Bone morphogenic proteins |
| BW | Body weight |
| CMI | Core muscle injury |
| COPD | Chronic obstructive pulmonary disease |
| CT | Computerized tomography |
| DEXRIT | Dynamic external rotatory impingement test |
| DGS | Deep gluteal syndrome |
| DHS | Dynamic hip screw |
| DIRI | Dynamic internal rotatory impingement test |
| EMG | Electromyography |
| FABER | Flexion, abduction, external rotation |
| FADDIR | Flexion adduction internal rotation test |
| FAI | Femoroacetabular impingement |
| GRF | Ground reaction force(s) |
| HHS | Harris hip score |
| HHSm | Modified Harris hip score |
| HPI | History of present illness |
| iHOT | international hip outcome tool |
| IPI | Iliopsoas impingement |
| ITB | Iliotibial band |
| L | Left |
| MAHORN | Multicenter arthroscopy of the hip outcomes research network |
| MFCA | Medial femoral circumflex artery |
| MRI | Magnetic resonance imaging |
| NAHS | Nonarthritic hip score |
| NSAIDS | Nonsteroidal anti-inflammatory drugs |
| OA | Osteoarthritis |
| ON | Osteonecrosis |
| ONFH | Osteonecrosis of the femoral head |
| PRP | Platelet rich plasma |
| R | Right |
| ROM | Range of motion |
| SCFE | Slipped capital femoral epiphysis |
| SI | Sacroiliac |

| | |
|---|---|
| TFL | Tensor fasciae latae |
| THA | Total hip arthroplasty |
| VAS | Visual Analog Pain Scale |
| WOMAC | Western Ontario and McMaster University |

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**Joseph C. McCarthy, MD** is the chief of Reconstructive Hip and Knee Joint Surgery at Newton Wellesley Hospital, a member of the joint arthroplasty staff at Massachusetts General Hospital, and Associate professor of Orthopedic Surgery at Harvard University, part-time. He is also a clinical professor of Orthopedic Surgery at Tufts University for the past 20 years. He has had a long-standing interest in hip joint preservation and, along with Dr. James Glick, pioneered hip arthroscopy in the United States. This focus included published work on hip anatomy and morphology as well as minimally invasive surgical approaches, development of a dedicated hip distractor and instruments solely for use in hip arthroscopic procedures.

Dr. McCarthy completed his undergraduate education at the University of Notre Dame, during which time he played on the varsity hockey and baseball teams. He then matriculated to Georgetown University medical school. Following a medical internship at Georgetown University Hospital, he completed surgical and orthopedic training at Tufts University in Boston. After residency, he completed a fellowship in hip and knee reconstructive joint surgery at Massachusetts General Hospital under the direction of Dr. William Harris and Hugh Chandler. Subsequently his entire practice career has been in Boston.

During his career he has served the American Academy of Orthopedic Surgeons as a member of the Committee on the Hip, program chair for the Hip Society, and has been on the board of directors for both the AAOS and the orthopedic research and education foundation (OREF). He has also been the chair of the board of specialty societies of the AAOS and chairman of the Shands Society of OREF. He is a founding member of the International Society of Hip Arthroscopy. He is also past president of the American Association of Hip and Knee Surgeons and the International Society of Hip Arthroscopy.

For his research he has received both the Otto Aufranc Award and the Frank Stinchfield Award from the Hip Society and the AAOS. Both of these awards were for work on understanding the acetabular labrum and, when injured, its relationship to degenerative arthritis of the hip joint. Dr. McCarthy has been Director of the biologic hip portion of the Harvard hip/knee course for the past 8 years and has been co-chairman of the AAOS Learning Center hip arthroscopy course for the past 12 years.

Dr. McCarthy has been committed to scholarship in the field of hip preservation, publishing the first validated hip outcomes scoring system for the native hip, as well as been an active member of MAHORN (Multi-center Arthroscopic Hip Outcomes Research Network). Most recently he has authored work on hip joint lubrication and EMG muscle effects associated with acetabular labral tears. Dr. McCarthy has authored over 175 peer-reviewed articles, posters, book chapters, and books.

**Philip C. Noble, BE MEngSci PhD** was educated in Australia in diverse subjects including Engineering, the Physical Sciences, and Classical Philosophy. After being awarded a Winston Churchill Fellowship in 1979, he traveled throughout Europe, the United Kingdom, and North America and eventually returned to Houston to work in the Texas Medical Center. He was awarded his PhD from the University of Strathclyde in Glasgow, Scotland, where he studied the biomechanics of the hip and hip replacement under the direction of Professor John Paul. He now serves as the Director of Research in the Institute of Orthopedic Research and Education and holds the rank of Professor in the Department of Orthopaedic Surgery at Baylor College of Medicine.

Phil Noble's research interests include the biomechanics of the hip and knee joints and their replacements, the morphometry of human bones, the biology and biomechanics of cartilage, tendon, and ligaments, and the quantitative assessment of clinical outcomes. He has numerous publications related to the hip and is a frequent speaker at national and international conferences. He is also a member of The Hip Society, The Knee Society, The International Hip Society, and The American Academy of Orthopedic Surgeons. Dr. Noble is internationally renowned as a designer and innovator and is the holder of numerous patents. He has participated in the development of more than 20 designs of total hip and knee replacements and associated instrumentation.

Dr. Noble is active in philanthropic activities and is particularly interested in issues related to diversity and underrepresented minorities. He is also committed to mentorship and has been instrumental in the training and development of over 150 postgraduates interested in careers in orthopedic surgery and related research. His recreational interests include foreign travel, gardening, and ornamental glass.

**Richard N. Villar, BSc (Hons) MA MS FRCS** qualified in medicine at St. Thomas's Hospital in London (UK) before joining the military as the Regimental Medical Officer to the SAS. After leaving the Army he completed his surgical studies in Southampton and Cambridge, joining the consultant staff of Addenbrooke's Hospital (Cambridge) in 1988. It was there that he developed his interest in conservative hip surgery and now has one of the largest experiences of hip arthroscopic (keyhole) procedures in the world.

Now in independent practice, Richard is a Founding Member, and first President, of the International Society for Hip Arthroscopy. He has served as a member of Council of the British Orthopaedic Association, is an assessor for the General Medical Council, and is a member of the United Kingdom's Disaster Emergency Response Team.

In this latter capacity he was operational in the aftermaths of the Kashmir, Java, and Haiti earthquakes. Richard regards orthopedic surgery as both his profession and hobby.

When not dealing with problematic hips and knees, he can be found either playing his classical guitar or on a mountainside, maintaining his skills as an International Mountain Leader.

# Part I

# Structure and Function of the Tissues of the Hip (Normal and Diseased)

Richard E. Field

# Development of the Hip: Phylogeny and Ontogeny

1

Tom Hogervorst, Karl-Philipp Kienle, and Moritz Tannast

## Introduction

The human hip is a conceptually simple ball and socket joint, but functions as part of a complex anatomic unit consisting of the femur, the pelvis and the lumbosacral spine. This unit is highly variable between different species of animals. Human hip evolution is characterized by obligate bipedal gait and encephalization (development of a disproportionately enlarged brain). This makes the female pelvis the only skeletal element that conveys information about these two most peculiar traits of human evolution. It shows both the adaptations that occurred to facilitate a permanent bipedal gait and, at the same time, the adaptations to accommodate the birth of a large-brained baby.

Human hip morphogenesis can deviate from its normal pathway by developmental hip disorders. Common developmental hip disorders such as developmental dysplasia of the hip, slipped capital femoral epiphysis (SCFE) but also femoroacetabular impingement can be explained from an evolutionary perspective. Below, we review relevant aspects of evolution (phylogeny) and human hip morphogenesis (ontogeny) that are relevant to the understanding of hip morphotypes and related hip disorders.

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## Evolution of Bone and Locomotion

Mineralized tissues (enamel, dentine and bone) were a major breakthrough in evolution. Calcium carbonate ($CaCo_3$), the common constituent of rock, was always present in ocean water and started being used as reinforcement in organisms about half a billion years ago in the Cambrian era [1]. Since then, fossils demonstrate the calcified remains of life's evolution. But the fossil record will, by definition, always remain incomplete, and it is genetic studies that have revolutionized our understanding of the early stages in evolution of mineralized tissues. For example, a related family of genes that likely arose from a common ancestor produces the mineralized tissues for teeth (enamel, dentine) and skeletons (bone extracellular matrix) [2]. Teeth-like structures probably evolved first, allowing new forms of predation, followed by a dermal exoskeleton of dentine, enamel and bone [3]. Teeth produced big changes in feeding and predation while development of endo- and exoskeletons allowed radical changes in locomotion. Bone likely appeared as an attachment to dentine in scales [2] in exoskeletons. The stunning fossils from the Cambrian era document an explosion in the possible basic structures of bodies (body plans) [4, 5]. The vast majority of these have long gone extinct and the remaining body plans (i.e. at phylum and subphylum level) now show striking *invariability*, for which genetic explanations have been suggested recently [6]. In contrast, within phyla, a spectacular variety in animal form has developed. On the phylum level, the existence of an endo- versus an exoskeleton (e.g. Arthropodae) imparts major differences in function. An exoskeleton affords strength and allows limbs to be longer which enhances both protection and locomotion. An endoskeleton has the advantage over exoskeletons that it frees the skin to function as sensory and thermoregulatory organ.

Already in lobe-finned fishes such as the *Eusthenopteron* (Devonian period [415–375 million years ago]), we find the primordial tetrapod body structure consisting of a longitudinal body axis with four perpendicular appendages. Indeed, their paired breast and pelvic fins have the pattern of our



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limbs today: one proximal (femur and humerus) and two distal bones (tibia/fibula and radius/ulna, Fig. 1.1). Their pelvic fins had what can be interpreted as a proto-femur. Once "on land" a suite of further developments improved locomotion, both in endurance and speed.

## Evolution of Tetrapod Gait

The development of terrestrial life forms hinged on the evolution of limbs from paired fins, limbs that, eventually, could bear the animal's weight against gravity (Fig. 1.2). Molecular genetic studies now show the fin to limb transformation can be made by subtle changes in a relatively small number of genetic switches [7], i.e. without the need for extraordinary processes or genetic mechanisms. Amphibians started walking with a sprawling gait, with the limbs still perpendicular to the long axis of the body—as with the paired fins in fish. But on land this requires permanent elevation of the body above the plane of the appendages to prevent contact between the trunk and the ground (Fig. 1.3).

Much heavier loads can be carried by limbs that are vertical than those that are horizontal, and so, vertical limb alignment allowed dinosaurs to grow to a huge size. The emergence of vertical limb positions and rounding of the hip joint also enabled increased stride length, while adoption of an erect posture decoupled walking from breathing. This increased stamina, as running no longer counteracted breathing [8].

## Mammalian Hip Types

Mammals display large variation in hip morphology. Conceptually, two types of hip can be distinguished, c*oxa recta* and *coxa rotunda* [9, 10], based on differences in *proximal femoral concavity* [11]. Concavity is a compound measure, influenced by the relative dimensions of the femoral head and neck (*head-neck ratio*), the roundness of the femoral head (*sphericity*) and the position of the femoral head relative to the neck [12]. Concavity thus determines the potential for femoral impingement (the acetabular parameters are depth and sphericity). Concavity can be quantified by angular measurements, e.g. alpha [13], beta, gamma and delta angles [12] and linear measurements (offset) or ratios. Coxa recta and rotunda relate to the ossification pattern of the proximal femur [14] and locomotor categories. Specifically, a single coalescence of the proximal femur is seen in coxa recta, whereas separation of the trochanteric and capital epiphysis is seen in coxa rotunda. Typically, coxa recta is seen in runners and jumpers, rotunda in climbers, amphibians and swimmers (Fig. 1.4. horse/walrus). In humans (and in the nonhuman apes), the two epiphyses of the proximal femur separate, i.e. a coxa rotunda ossification pattern. However, some morphotypes of the human hip appear to mimic the normal morphology of species with "coalesced" epiphyses [14], i.e. a coxa recta or "cam-type" hip [15].

## Locomotion in the Nonhuman Apes

The nonhuman apes have a varied repertoire of locomotion including arm slinging, climbing, quadrupedal knuckle walking and bipedal walking. The nonhuman apes (chimpanzee, bonobo, gorilla, gibbon, orangutan) do not run bipedally [16], and their bipedal walking is not the true upright walking seen in modern humans. Due to a stiff spine [17], bipedal walking in the nonhuman apes requires flexion in both hips and knees to position the trunk over the feet (Fig. 1.5). Bipedal walking in the nonhuman apes therefore requires constant activity of hip extensors (hamstrings) and

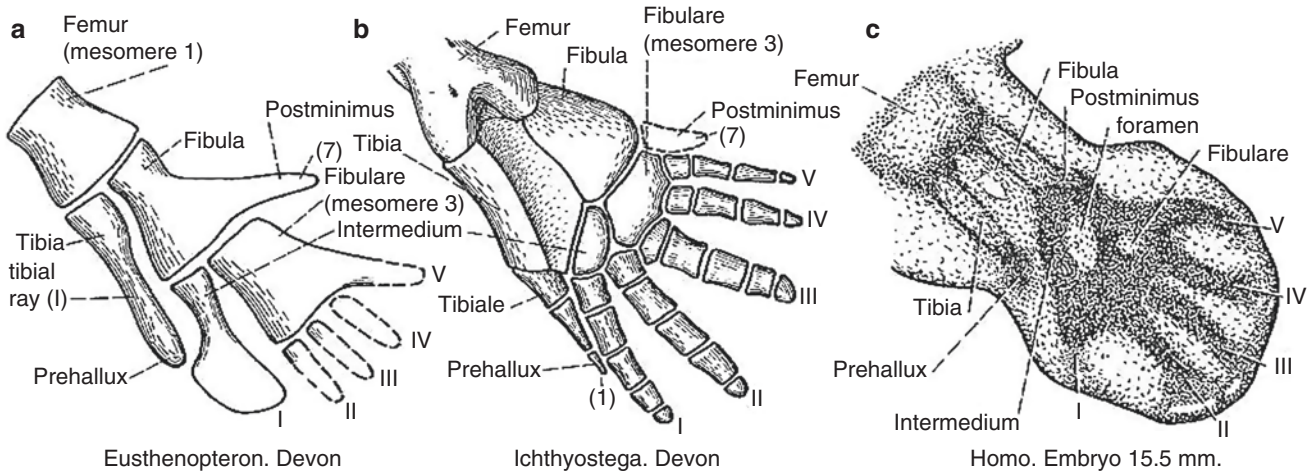


**Fig. 1.1** The ancient building plan of (hind) limbs. Eusthenopteron is a lobe-finned fish, Ichtyostega is a *fishapod,* comparable to Acanthostega (Fig. 1.2) From [81] and Hogervorst T, Bouma HW, de Vos J. Evolution of the hip and pelvis. Acta Orthop Suppl. 2009 Aug;80(336):1–39. Reprinted with permission from Informa Healthcare

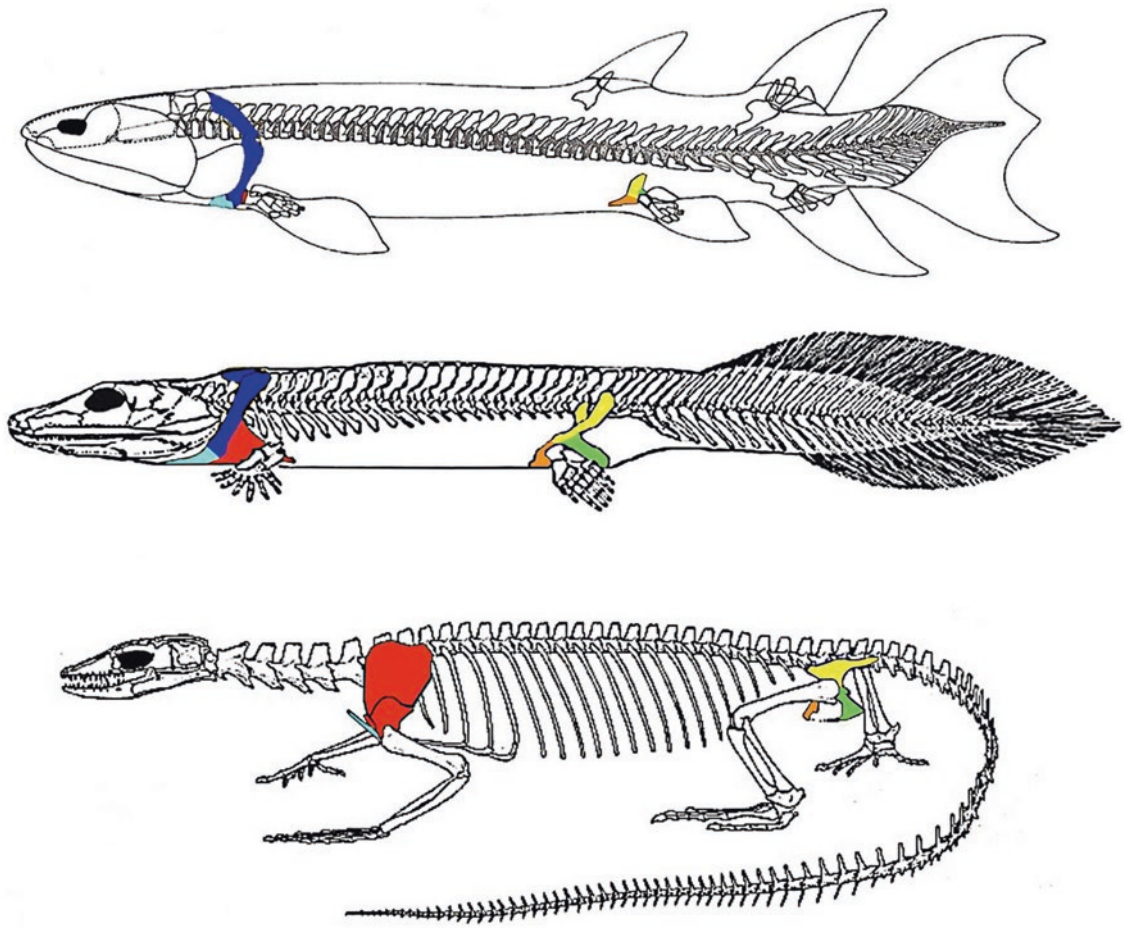

**Fig. 1.2** *Eusthenopteron* (*top*), a fossil lobe-finned fish of approx. 380 million years ago, is thought to represent the beginning of the transition to land-life. It has no neck and a bony connection of the pectoral girdle (with its pectoral fins) to the skull. The pelvic bone (*yellow*, *green*, *orange*), however, is not attached to the spine. *Acanthostega* (a fossil *fishapod*, *middle*) can be seen as the other end of the transition to land-life, and the time elapsed between these two fossils is about 15 million years. The scapulocoracoid (*red*) has increased in size, and the pectoral girdle is no longer attached to the cranium. The pelvis has enlarged markedly and is now attached to the spine by ligaments and muscles. *Varanus* (*bottom*), an extant lizard, has its clavicle (*light blue*) not attached to the spine, allowing movement between the scapulocoracoid and the spine to increase stride length. The pelvis has a bony connection to the spine through the sacroiliac joints. From Hogervorst T, Bouma HW, de Vos J. Evolution of the hip and pelvis. Acta Orthop Suppl. 2009 Aug;80(336):1–39. Reprinted with permission from Informa Healthcare

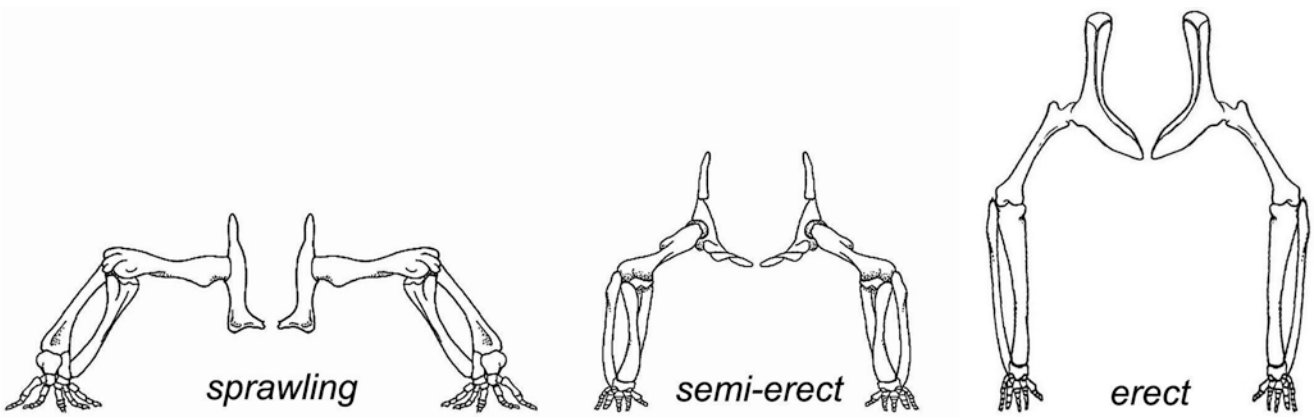


**Fig. 1.3** Posture types: sprawling (e.g. reptiles), semi-erect and erect (e.g. cursorial mammals)

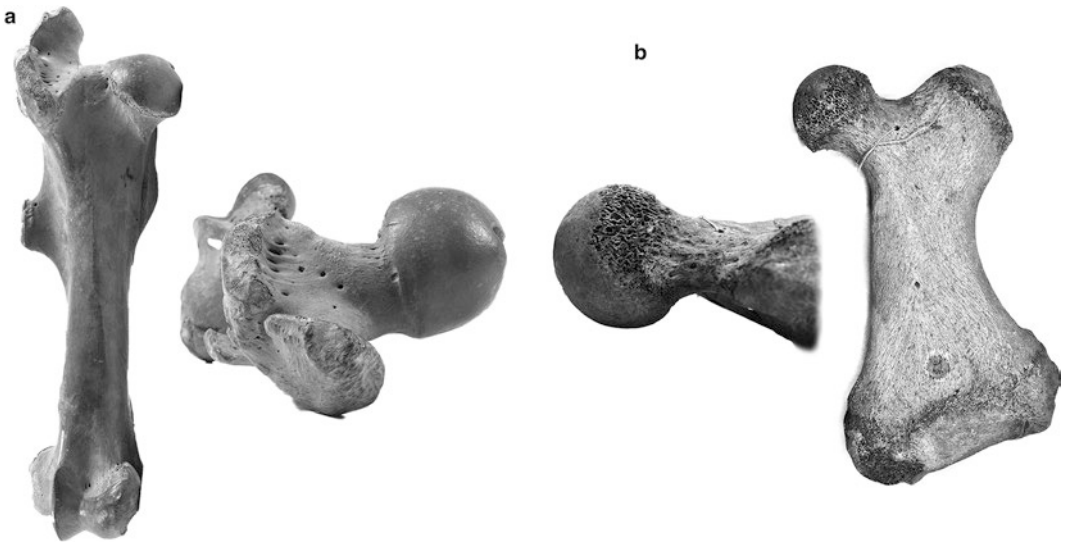


**Fig 1.4** Femora of a horse (**a**), a cursorial (an animal adapted for running) with coxa recta (*left*) and a walrus (**b**), a swimmer with coxa rotunda (*right*)

knee extensors (quadriceps). Further, in the nonhuman apes, the orientation of the pelvic ala coincides with the coronal plane. Consequently, the gluteus medius and minimus continue to function as hip extensors rather than abductors (Fig. 1.6), and so nonhuman apes tend to walk with less lateral balance control [18].

## Early Human Hips: Ancestors

DNA studies date the shared ancestor of chimpanzees and humans to approx. six to seven million years [19], which makes this a recent event, in evolutionary terms. Many quadrupedal mammals can also walk upright (e.g. bears, apes), but for modern humans, upright walking quickly became the *only* gait type. Many adaptations in lumbosacro-pelvic morphology occurred within a relatively short time span to enable this (for review see [17]). Spectacular fossil finds of the last 50 years [10] show that obligate and exclusive bipedal gait was established within 2–3 million years of early human evolution [20]. Human bipedal gait is highly efficient compared to that of the other large apes. Little muscle activity is required to maintain human upright posture [21]. Compared to the extensive changes in lumbosacral spine and pelvis, the changes in the early human hip joint can be considered minor. When comparing with the nonhuman apes, the changes in the proximal femur reflect the increased loading of bipedal gait and running (nonhuman apes do not run bipedally). The human hip has a thicker femoral neck (decreased head-neck ratio, Fig. 1.7) and reduced concavity compared to the nonhuman apes (Figs. 1.8 and 1.9).

## Comparison of Human and Ape Hip Morphology

A comprehensive study of cadaveric femora of 375 North Americans (divided equally between males and females and blacks and whites) found considerable variability in the concavity of the femoral head-neck junction, both superiorly (gamma angle) and anteriorly (alpha angle) [12]. Epidemiology studies in predominantly white populations concur, with a prevalence of approx. 8–20% of the coxa recta morphotype [13, 22–27]. Coxa profunda (defined as a lateral CE angle >39° or a posterior wall sign) appears to have comparable prevalence although this has been less extensively studied, and gender differences are conflicting [23, 27].

However, it appears that considerable differences in human hip morphology exist between different ethnic populations. Although studies comparable to Toogood et al. are as yet lacking, low concavity of the proximal femur appears to be rare in many [28–32], if not all [33, 34], Asian populations. Thus, it has been speculated that the lower incidence of osteoarthritis in the "Asian hip" [35, 36] is related to this higher concavity and decreased FAI [28, 37]. In contrast, the nonhuman apes share a much more uniform hip morphotype, with no important differences in concavity, either between species or between the sexes. In 210 cadaveric great ape femora (chimpanzee/bonobo and gorilla) examined using the exact methods of Toogood et al., much lower variability in concavity was found [38]. The nonhuman apes have coxa rotunda (Figs. 1.7 and 1.9), allowing a large range of motion, advantageous for a locomotion generalist and particularly so for climbing. Perhaps, increasing the loading history during

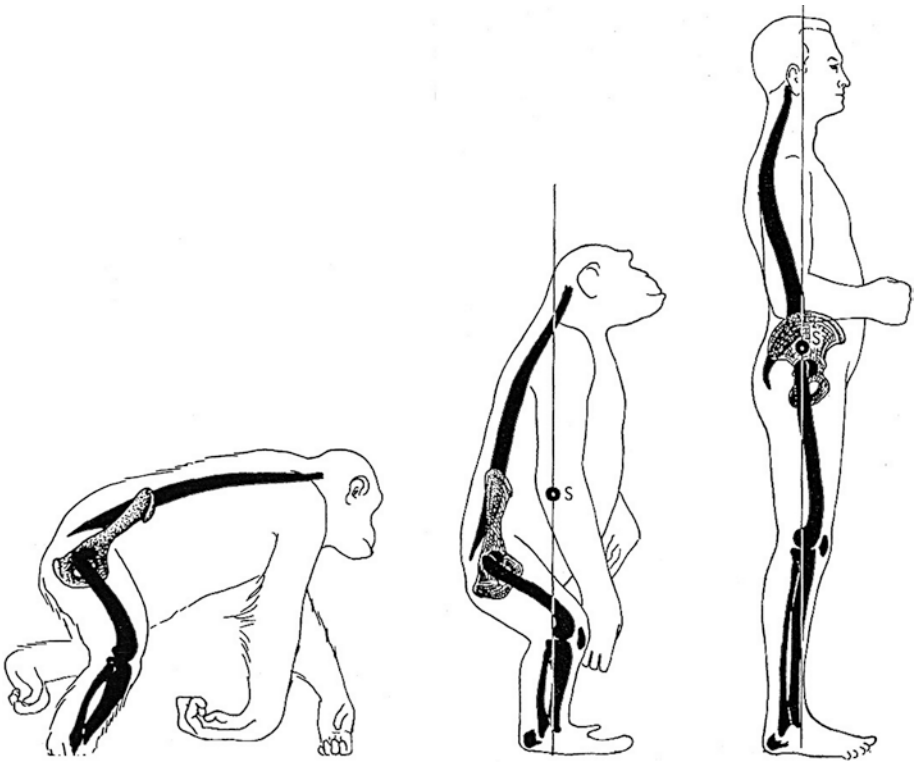


**Fig. 1.5** Chimpanzee bent hip—bent knee gait has about the same position of the femur in the acetabulum as when knuckle walking, which is in its mid-range. From [82] Kummer B. Biomechanik: Form und Funktion des Bewegungsapparates. Dt. Ärzte-Verlag 2004. Reprinted with permission from German Ärzte-Verlag GmbH

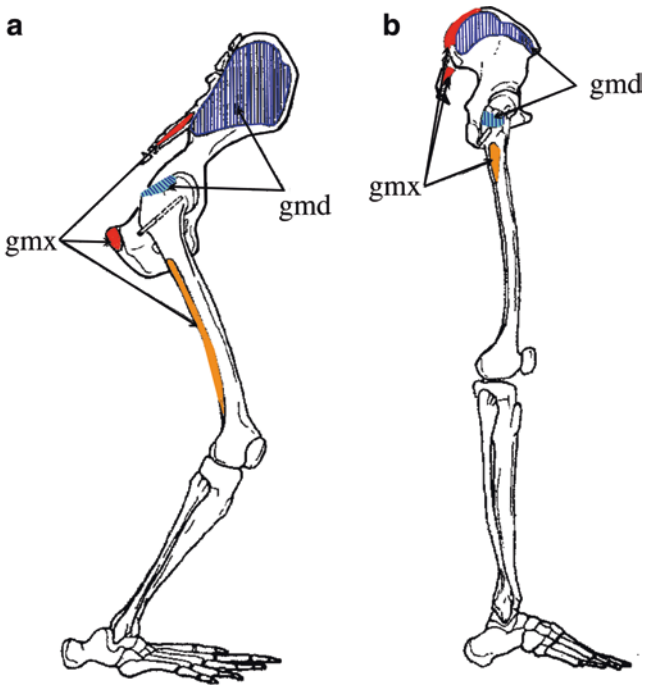


**Fig. 1.6** Rearrangement of gluteal origins and insertions. A gorilla, B human. (*Red*) gluteus maximus origin, orange insertion. (*Blue*) gluteus medius origin, cyan insertion. *Gmx* gluteus maximus, *gmd* gluteus medius. From [83] Hogervorst T, Vereecke E. Evolution of the human hip. Part 2: muscling the double extension. J Hip Preserv Surg. 2015;2:3–14. Reprinted with permission from Oxford Journals

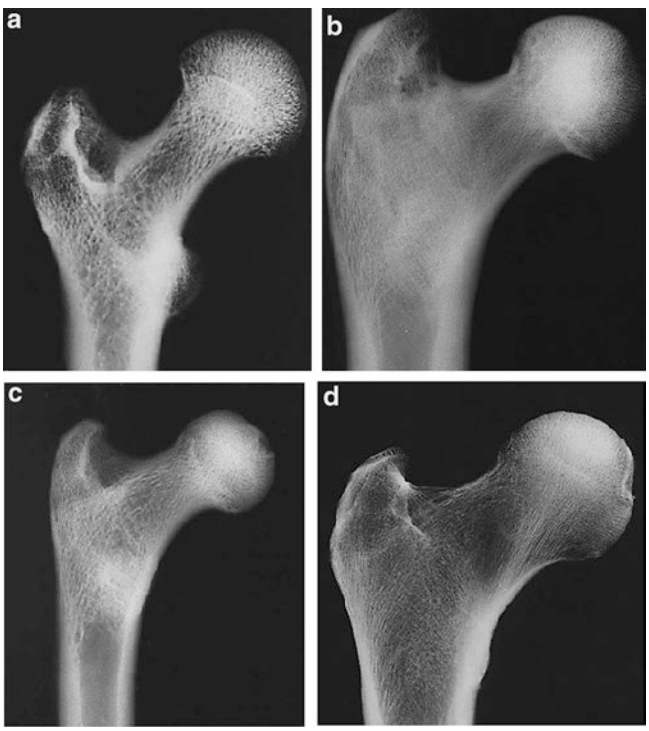


**Fig. 1.7** Radiographs of hominid hips with head-neck ratios (head largest diameter divided by smallest neck width). The human hip has the thickest neck, decreasing offset and impingement-free motion. Also note thicker cortical bone in the superior femoral neck of the apes (from Lovejoy 2005 [15], with permission). (**a**) Orangutan (1.8) (**b**) Gorilla (1.5) (**c**) Chimpanzee (1.5) (**d**) Homo (1.35). AP view, not to scale

growth induces morphotype changes in macaques [39], as it may in humans (see below).

Thus, modern human hip morphology appears more variable than in the nonhuman apes. This variability currently has no full explanation, yet is important, as certain hip morphotypes are associated with development of OA. Both genetics and biomechanics (loading history) play a role in the development of hip morphotypes (ontogeny) [40].


**Fig. 1.8** Concavity in nonhuman apes and humans. (**a**) Gorilla, (**b**) chimpanzee, (**c**, **d**) human. The nonhuman apes uniformly have large concavity and more so anteriorly than posteriorly. Some humans (**c**) have only small concavity anteriorly, and others have larger anterior concavity (**d**), but virtually all humans have large concavity posteriorly (**c**, **d**). View is perpendicular to the superior femoral neck. From Fikkers JT, et al. What Ape Proximal Femora Tell Us About Femoroacetabular Impingement: A Comparison. Clin Orthop Relat Res. 2014 Jul 1. Reprinted with permission from Springer

## Ontogeny: Growth and Development of the Human Hip

The evolutionary perspective outlined above can serve to better understand several phenomena of human hip growth and development. Quadrupedal mammals have a horizontal spine and the abdomen and uterus hang under it like a hammock. But the human foetus is positioned in an upright mother. It has a very large head and long legs, and, in the last trimester, the uterus wall tends to hyperflex the human hip, levering the long femur against the prominent anterosuperior iliac spine [41, 42]. (The nonhuman apes have no prominent anterior iliac spines, smaller heads and shorter legs; dysplasia is all but unknown.) The human hyperflexed position may be a mechanical factor to explain the decrease in relative acetabular depth [43, 44] and increase in femoral anteversion [44–47] with increasing gestation, producing a neonatal/infant hip dysplasia [41].

After birth, the human hip extends and more varied hip positions may explain the consistent finding in normal growth and development of postnatal femoral detorsion and relative deepening of the acetabulum [48]. Remarkably, for the rest of the hip's lifespan, most of its loading will occur near the limit of hip extension. Walking humans extend their hip approximately 5° at toe-off, and active prone extension is only 10°–20° [49, 50]. Active hip flexion is 120°, 35° when walking and 50° when running [51]. Thus, the human weight-bearing range of hip motion shifts close to its extension limit during bipedal gait development. Quadrupeds bear weight closer to

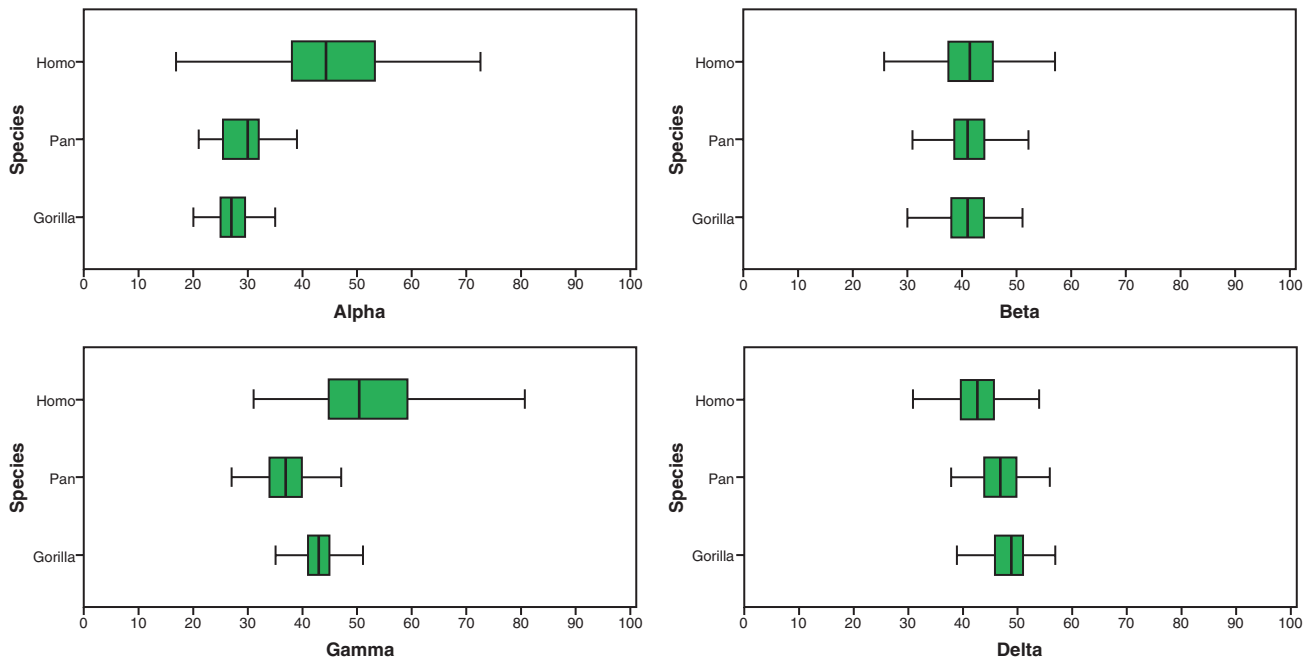


**Fig. 1.9** Variable concavity measurements in humans, uniform in the nonhuman apes. From Fikkers JT, et al. What Ape Proximal Femora Tell Us About Femoroacetabular Impingement: A Comparison. Clin Orthop Relat Res. 2014 Jul 1. Reprinted with permission from Springer

mid-range hip flexion [52]. In this position femoral neck anteversion aligns the capital physis more perpendicular to the vertical gait forces [53]. The human (extended) hip position increases shear forces on the capital physis. During adolescence, the peripheral epiphysis extends towards the femoral neck (Fig. 1.10). Prospective MRI studies of children and adolescents (9–17 years) now show that in normal development, this extension is almost similar at each position of the femoral head, which means the epiphyseal torsion angle does not change (Figs. 1.10 and 1.11 [54, 55]).

Due to their specialized bipedal gait, humans have much higher peak hip forces than quadrupeds of similar weight, and these forces increase markedly with running or sports [56, 57]. These factors can summate to a loading history of high, perhaps excessive, loads on the growing hip, particularly because human growth and development is 5–6 years longer compared with chimpanzees [58, 59].

Thus, human hip ontogeny is characterized by specific mechanical factors that can be interpreted to explain neonatal/infant hip dysplasia, to increase shear forces on the capital epiphysis that may induce SCFE or subclinical slippage and to summate to a loading history that may induce morphologic changes in the growing hip [40].

## Developmental Hip Disorders

During normal growth and development, the human femoral head is spherical in childhood [48], but aspherical femoral head morphology (coxa recta with alpha angle >60°) has already been shown in boys 12 years of age (Figs. 1.12 and 1.13) [60]. Asymmetric changes in physeal extension can reduce concavity at head-neck junction, creating a coxa recta. Siebenrock et al. found increased extension of the physeal scar in the anterosuperior head quadrant of FAI patients and suggested a growth abnormality of the physis can explain the differences in femoral head-neck offset between FAI patients and controls [61]. Alternatively, "subclinical" slippage of the epiphysis during adolescence has been proposed to explain the proximal femoral morphology of FAI, based on observations of epiphyseal tilt in cadaver femora, radiographs and recently MRI [11, 62, 63].

There are no longitudinal studies demonstrating the effect of loading on hip morphology in children or adolescents. But two cross-sectional studies in European populations found reduced concavity may develop with exposure of the hip to repetitive exercise through intense sports training during adolescence [60, 64]. Intriguingly, these differences were more pronounced in athletes with a closed capital physis, indicating ongoing effect of load history after physeal closure. Thus, high-intensity sports during adolescence may be associated with a higher prevalence of coxa recta morphotype, at least in European populations. As indicated above, comparable studies have not been undertaken in Asian populations. However, since coxa recta may be rare in Asian populations, the explanation that coxa recta exclusively occurs as an adaptive response to a high loading history appears not entirely sufficient [40]. Perhaps genetic differences explain varying responses to a given loading history. Research linking genes to hip morphotypes has begun to unravel the genetic basis of different hip morphotypes [65, 66] and has revealed that the majority of genes imparting OA susceptibility appear to be involved in skeletogenesis and/or homeostasis of bone and cartilage [40].

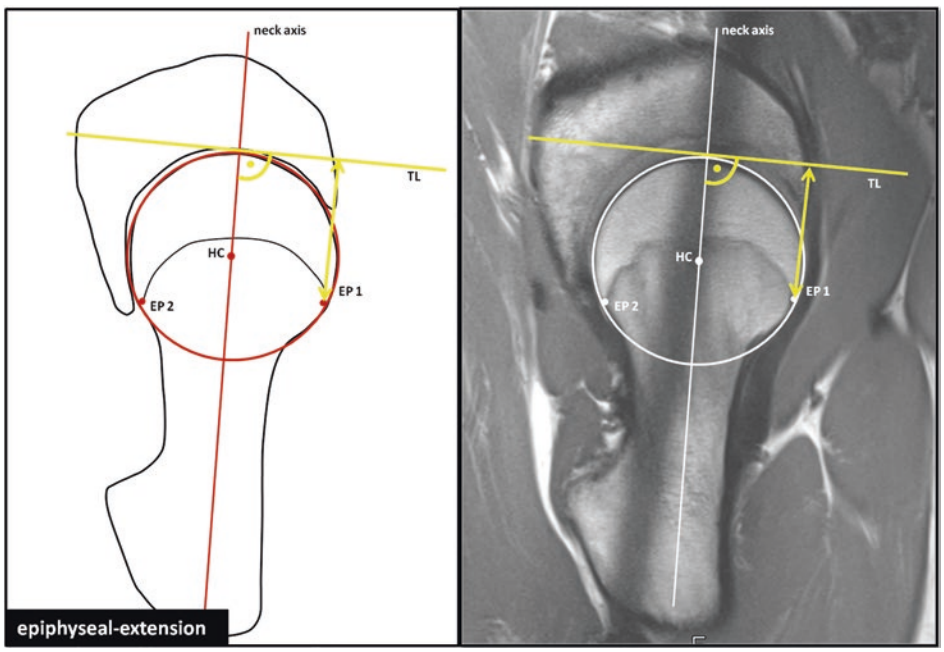


**Fig. 1.10** Epiphyseal extension, defined as the distance from a orthogonal straight line on the femoral neck axis to EP1 (point with largest epiphyseal extension)

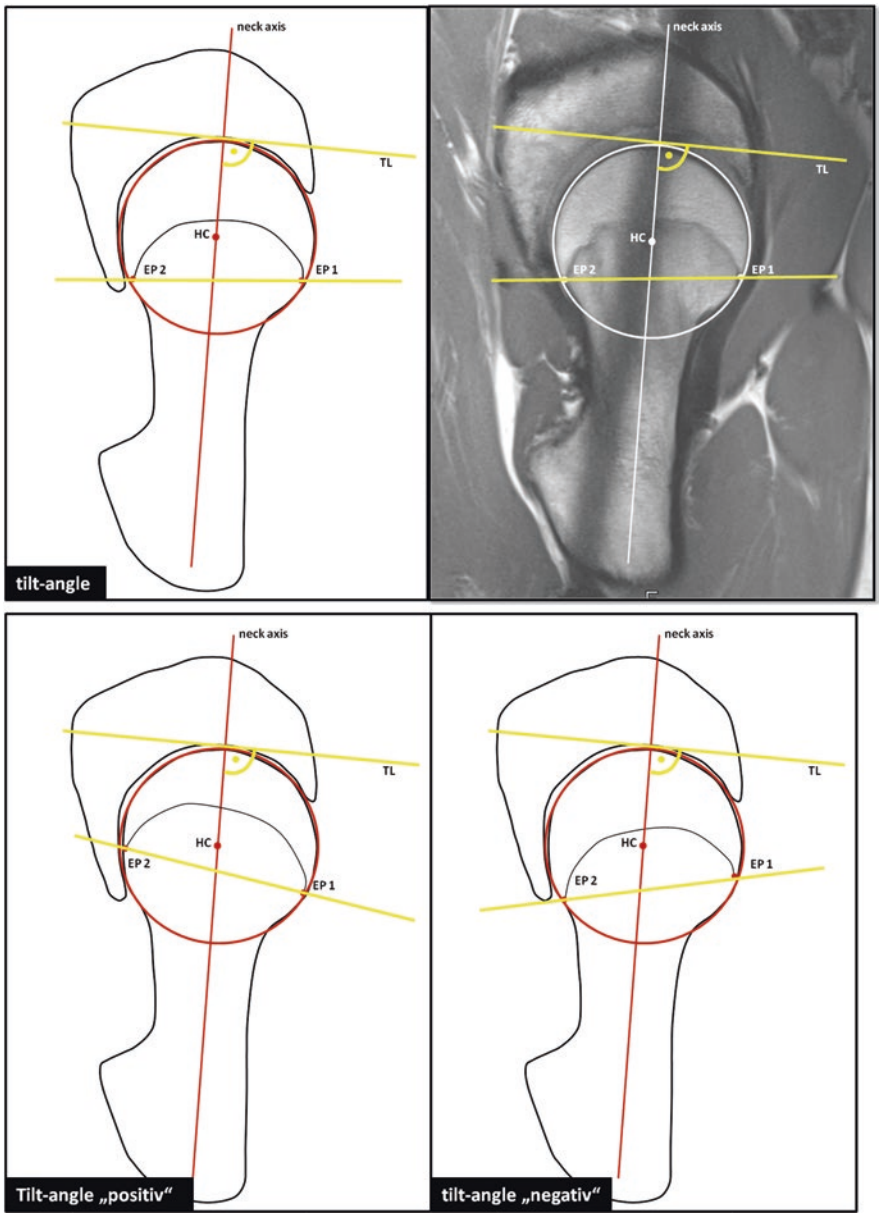


**Fig. 1.11** The epiphyseal torsion angle is defined by two lines: one line is orthogonal to the femoral neck axis, and the other line connects EP1 and EP2, the points at the largest epiphyseal extension

In normal growth and development, the apical parts of the human acetabulum are retroverted in childhood, but become progressively anteverted during maturation, according to prospective MRI studies (Fig. 1.14). CT studies in normal adults report anteversion at the mid-level of the acetabulum of about 22°–24° in male and 19°–25° in females [67, 68]. The prevalence of retroversion is approx. 5 % in the general population, but 20 % in patients with osteoarthritis [69, 70].

Acetabular depth and anteversion determine the femoral head coverage and range of hip motion (Fig. 1.13). Reduced coverage in developmental dysplasia and overcoverage, either locally (retroversion) or globally (coxa profunda), can both produce local cumulative mechanical overload that can damage joint structures and lead to OA. In all, five parameters influence whether femoroacetabular impingement will occur: acetabular coverage and version, femoral head sphericity and version and neck shaft angle. Each of these five parameters has a reciprocal interaction with the others; for example, a shallow acetabulum delays impingement of the femoral head with the acetabular rim. New parameters are being developed that visualize and quantify this interaction of the proximal femur and acetabulum [71].

Population studies show small or no differences in the prevalence of both undercoverage (DDH) [23, 72] and overcoverage (coxa profunda) in males and females [23, 27]. In contrast, such differences do exist between different ethnic populations, with Asian populations having higher prevalence of DDH [37, 73, 74] and European or North American populations having more overcoverage [37].

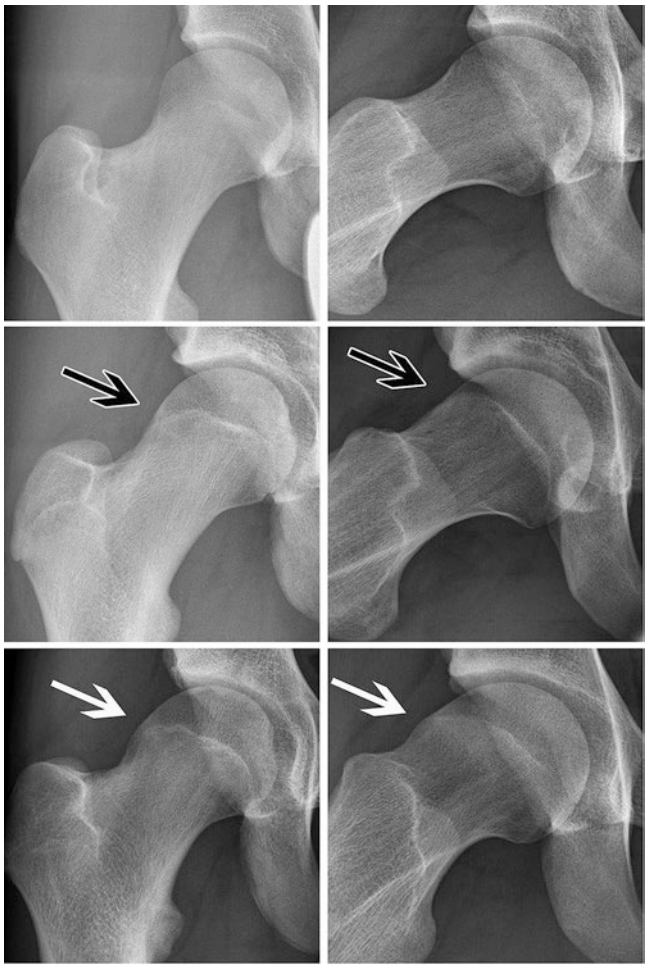

**Fig. 1.12** Anteroposterior pelvic and frog leg lateral radiographs (*left*) in human adolescents illustrating normal (*top row*) and coxa recta (*middle* and *bottom rows*). From Agricola R, Bessems JH, Ginai AZ, Heijboer MP, van der Heijden RA, Verhaar JA, et al. The Development of Cam-Type Deformity in Adolescent and Young Male Soccer Players. Am J Sports Med. 2012 Mar 13;40(5):1099–106. Reprinted with permission from SAGE Publications

Perhaps the intrauterine mechanical factors outlined above help explain neonatal DDH (but clearly not its ethnic disparities). After birth, mechanical factors, such as prolonged extended hip positions used with the swaddling of babies, influence whether neonatal hip dysplasia corrects or not [75]. For DDH of later onset however, we lack prospective MRI studies documenting the causative changes. Ontogeny is characterized by reciprocal development of the femur and acetabulum and indeed extends to the entire innominate bone [76]. Cultures that keep the hips of infants extended during most of the time (i.e. through swaddling) have a much higher incidence of hip dysplasia than cultures in which the hips are held apart (and therefore in a centred position) during carrying (i.e. in a back sling) [73].

The origin of coxa profunda is unproven due to the same lack of prospective MRI studies. This limits explanations to speculation, with factors cited including the efficiency of the abductor muscles [77], obstetric factors [78] and sex hormones [79, 80] or a combination of these.

## Summary

Reviewing human hip phylogeny, and comparing our hip to other mammals and the great apes, helps us recognize the peculiar features of the human hip. The hip joint sees a marked shift in the default loading position from a hyperflexed position in utero, to close to its extension limit in later life. The hip also has a remarkably long period of growth and development and is often exposed to large loads with its physes still open.

Prospective MRI studies that are now appearing may confirm earlier suggestions that a high loading history may determine a hip's eventual morphotype. Conversely, an emerging body of evidence attests to an interplay between

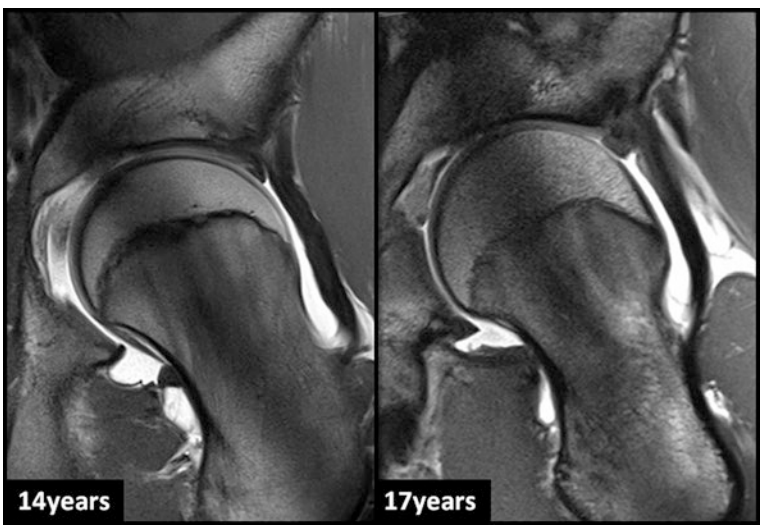


**Fig. 1.13** Development of coxa recta in a highly active adolescent basketball player within 3 years

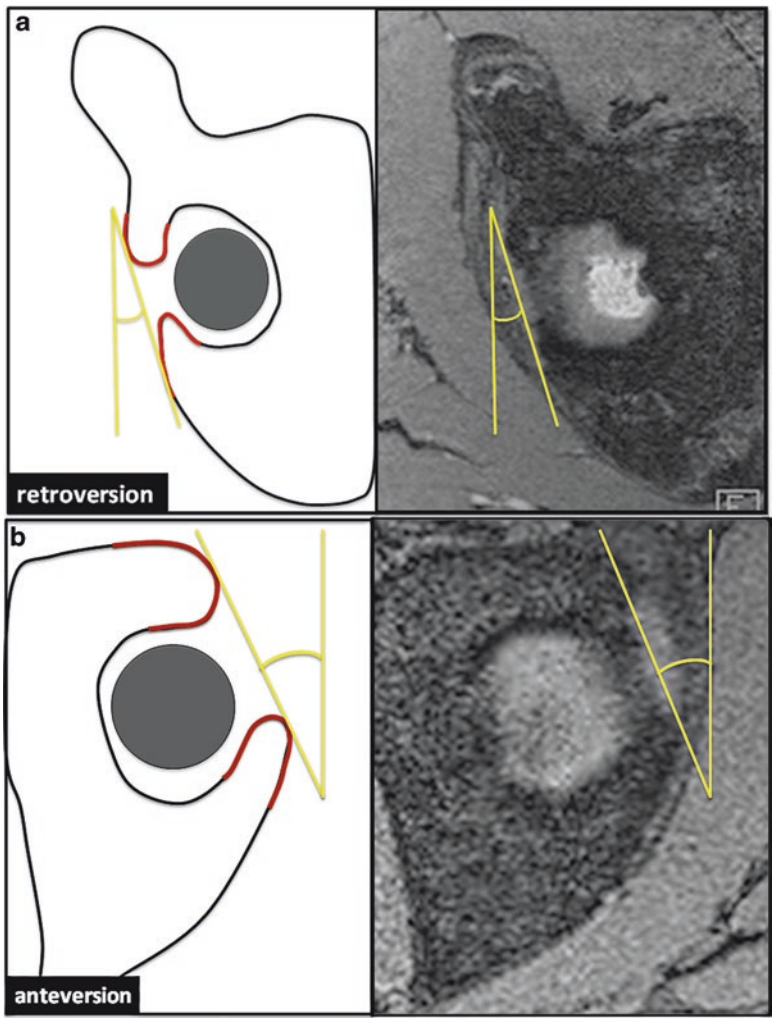


**Fig. 1.14** Acetabular version in the apical part of the joint; measured between a straight sagittal line and a line connecting the anterior and posterior acetabular edge. *Top diagram* (**a**) and MR image depict the hip of a 7-year-old; the *bottom* (**b**) diagram and MR image depict the hip of a 15-year-old

genetic and mechanical factors in the development of the hip morphotype.

Genes orchestrate hip morphogenesis, but their influence most likely acts through several or numerous genes, each with modest effect sizes. Presently therefore, we do not know whether hip morphogenesis is primarily determined by genetic or mechanical factors.

Whether a given hip morphotype will lead to progressive OA is, again, influenced by mechanical factors, such as its loading history, but appears influenced also by an intrinsic ability of its cartilage to withstand mechanical stress.

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# Anatomy: Cartilage

2

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## Introduction

The articular cartilage is a highly hydrated, avascular, aneural, and alymphatic tissue that provides a gliding surface for smooth articular motion and allows load distribution and reversible deformation in response to mechanical stimulation. Its complex organization and composition, including specific orientation of collagen fibers and chondrocytes within the different cartilage layers, contribute to these unique properties. Chondrocytes dynamically remodel their surrounding extracellular matrix and receive signaling cues from the matrix, communication that is essential for cartilage homeostasis. This unique cell type has multiple functions, from cartilage development to the mature functions of the articular cartilage. Chondrocytes are involved in growth plate development during the growth of the long bones, in the formation of articulations during joint development, and in the maintenance of the mature articular cartilage throughout life. During joint development it is believed that articular chondrocytes are derived from the interzone's outer layer; however, this is still a controversial topic, and more recent studies suggest that these cells may originate from an early chondrocyte subpopulation that arises at the same time as interzone formation. Due to its avascular nature, cartilage nutrition and waste product removal are dependent on processes such as diffusion and fluid flow. This complex, unique tissue is regulated by multiple mechanisms from mechanical to growth factor stimulation. Functional and structural dysregulations occurring early in development may lead to diseases such as chondrodysplasia, while later in life dysregulation during cartilage maintenance may lead to osteoarthritis (OA). In this chapter, we will discuss the macroscopic and microscopic features of cartilage and will describe the developmental mechanisms that contribute to its final form. In addition, we will briefly present a number of structural and biological changes in the cartilage that occur in joint diseases, such as OA.

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## Gross Anatomy of the Hip Cartilage

While the bony anatomy of the hip joint consists of the femoral head and the acetabulum, the soft tissue components of the hip are comprised of cartilage, ligaments, capsule, and synovium. The two main types of cartilage within the hip are articular hyaline cartilage on the femoral head and acetabulum and fibrocartilage that comprises the labrum.

The hyaline cartilage is the smooth, white tissue that covers 60–70 % of the spherical femoral head. All areas of the femoral head that articulate with the acetabulum are covered in hyaline cartilage, which extends past the equator of the femoral head. There is only one small, uncovered central area on the femoral head known as the fovea capitis, where the ligamentum teres inserts (Fig. 2.1).

The thickness of the articular cartilage varies throughout the femoral head (range 0.8–3.8 mm [1–3]) and acetabulum (range 1.2–4.8 mm [2, 3]). As expected, the thickness of the articular cartilage has been shown to be highest in the superolateral quadrant for both the femoral head and acetabulum. The thinnest region for the femoral head is medial to the fovea, whereas the area closest to the acetabular fossa is the thinnest for the acetabulum. The cartilage thickness decreases concentrically as one goes from the thickest to the thinnest cartilage point [4].

The function of the hyaline cartilage is to provide a nearly frictionless surface (coefficient of friction = 0.001) where the femoral head can move within the acetabular socket [5] and



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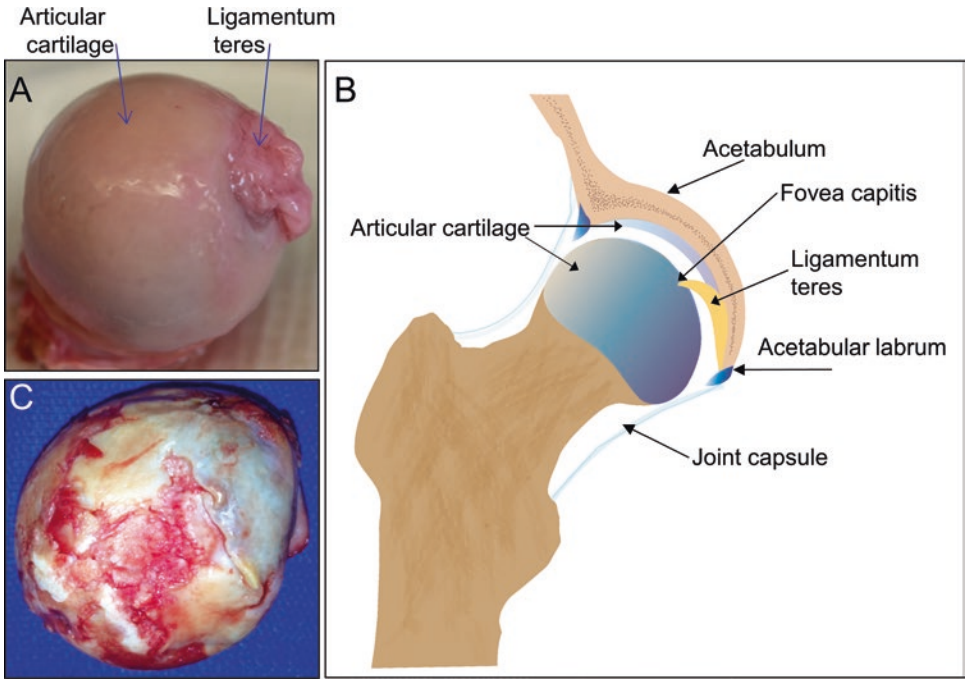


**Fig. 2.1** The hip joint. (**a**) Appearance of the normal healthy cartilage surface of the spherical femoral head. (**b**) Hip joint schematic depicting the main components of the joint: femoral head and acetabulum covered by the articular cartilage, ligamentum teres, fovea capitis, acetabular labrum, and the joint capsule. (**c**) Cartilage degeneration manifests as fibrillations, cartilage loss, and deformations in the smooth cartilage structure

to support and distribute mechanical load [6]. Due to its viscoelastic properties, healthy articular cartilage can restore its original shape after deformation in response to mechanical stimuli [7, 8].

In the presence of osteoarthritis (OA), the frictional coefficient of the articulating surfaces increases in direct proportion to the severity of cartilage degeneration due to the increase in surface roughness of the tissue [9]. Grossly, this manifests as fibrillations and deformations in the smooth cartilage structure, which may lead to femoral head collapse (Fig. 2.1c). This loss of the cartilage also reduces the shock-absorbing capacity of the hyaline cartilage and leads to further cartilage degeneration.

## Synovial Joint Formation

### Developmental Skeletogenesis and Synovial Joint Formation

The process of limb development begins when mesenchymal cells from the lateral plate mesoderm condense at the future place of skeletal elements and form the skeletal blastema [10–13] (Fig. 2.2a). The cells in the center of these condensations then differentiate into chondrocytes (Fig. 2.2b.1), which deposit an extracellular matrix rich in collagen type II and proteoglycans (PG) [10, 14]. Members of the SOX family of transcription factors are required for this chondrogenic differentiation [15, 16]. At the periphery of the condensations, cells flatten and elongate to form the perichondrium and later differentiate into preosteoblasts (Fig. 2.2b.2) [17].

In the hind limb, these mesenchymal condensations start as uninterrupted Y-shaped structures with the proximal arm of the Y corresponding to the future femur and the two arms of the Y corresponding to the tibia and fibula [10, 13]. Once the shape of the future bones is defined, joint formation is initiated at the site of the future articulation, with the establishment of a region of high mesenchymal cell density called the interzone (Fig. 2.2b.3) [18]. This area can be differentiated from the neighboring cells histologically and also by the expression of specific markers such as growth differentiation factor 5, GDF5, a member of the transforming growth factor β (TGF-β) superfamily [19, 20]. The interzone is subdivided into three layers (Fig. 2.2c.1, 3): a central layer, the intermediate lamina (which will break down (Fig. 2.2d.1) and eventually form the joint cavity), and two layers of high cell density on either side of the intermediate lamina that eventually become two regions of articular chondrocytes separated from each other by the fluid-filled joint cavity (Fig. 2.2e.1, 2) [21]. The origin of the articular chondrocytes is still a controversial issue, and while a number of studies suggest that they are derived from interzone cells that redifferentiate into chondrocytes, more recent cell lineage tracing studies suggest that they may also originate from a subpopulation of early chondrocytes [19].

### Human Hip Joint Development

In this section we will present a few selected stages of hip joint development as a guideline; however, the time lines of human embryonic joint development are quite variable, as described by many authors [22–24]. The interzone of the hip in a 13 mm

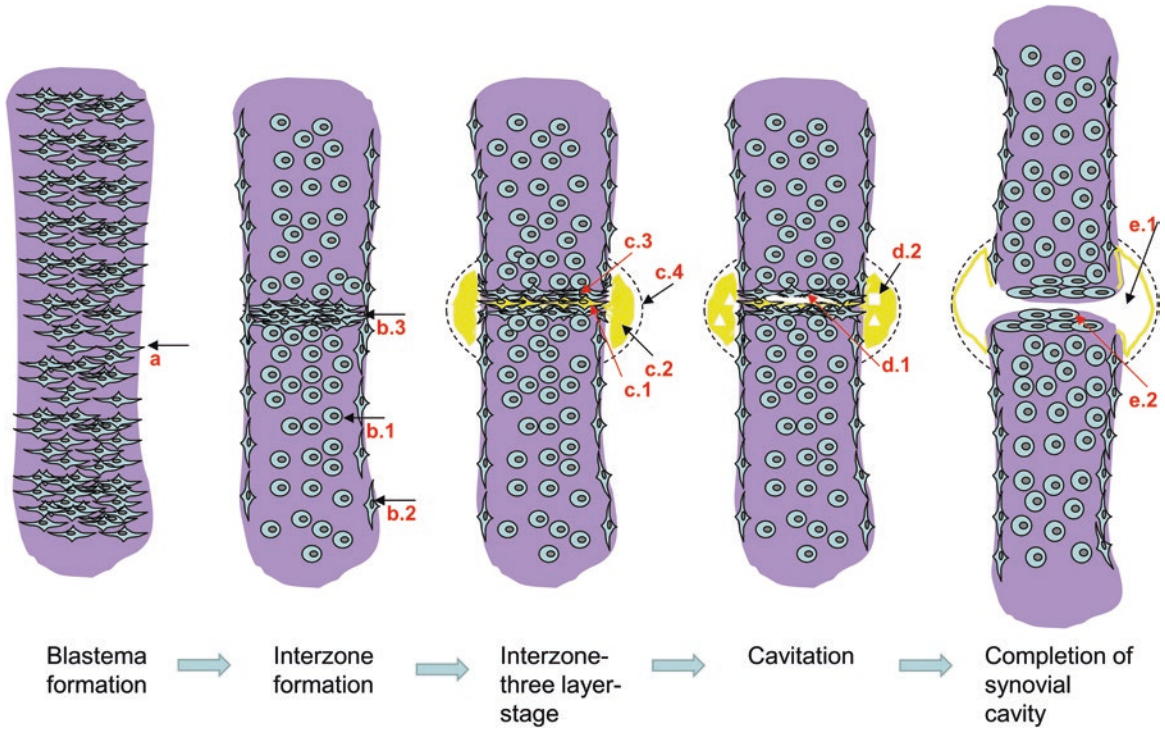


**Fig. 2.2** Synovial joint formation. A schematic of a general synovial joint. (**a**) Condensation of mesenchymal cells at the location of future skeletal elements; (**b.1**) differentiation of mesenchymal cells into chondrocytes; (**b.2**) formation of the perichondrium; (**b.3**) establishment of the interzone. With further development, three layers of the interzone form: a central layer (**c.1**) and two outer layers of high cell density (**c.3**) that will eventually become two regions of articular chondrocytes (**e.2**). Within the general mesenchyme, the future fibrous capsule can be observed as condensations (**c.4**). In the next stages the central interzone layer (**c.1**) and the synovial mesenchyme (**c.2**) will start to break down (**d.1**, **d.2**) in order to form the joint cavity. After the cavitation process, the two future articulating bones (e.g., femur and acetabulum) will be separated from each other by a fluid-filled cavity (**e.1**)

embryo is continuous with the cartilage on either side of the joint and with the perichondrium peripherally. By 14 mm the shape of articular surfaces resembles their future final appearance. Both the joint fibrous capsule (Fig. 2.2c.4) and the synovial mesenchyme (Fig. 2.2c.2) (which lies between the capsule and the extracapsular perichondrium) are derived from the general mesenchyme in the vicinity of the joint [22]. It is believed that the intra-capsular structures, such as the ligamentum teres, are formed from condensations of the synovial mesenchyme. At ~16 mm, interzones are still continuous with the perichondrium, and no capsular structures can be yet identified at the hip [22]. Gardner and Gray [23] noticed a cellular condensation between the coxal bone and the femur as the first indication of the future capsule. By 30 mm the three layers of the interzone can be observed with the middle zone being differentiated from the other two zones by a difference in density of histological staining (Fig. 2.2c.1) [22–24].

The labrum and the ligamentum teres appear as cellular condensations when the human embryo is around 22–25 mm [23]. The central interzone layer is connected in the joint periphery with the synovial mesenchyme, while the two interzone chondrogenic layers are continuous with the intra-capsular perichondrium [22]. At ~30 mm the middle layer of the interzone and the interior part of the synovial mesenchyme are starting to break down (Fig. 2.2d.1, 2), in order to give rise to joint cavities by the process of liquefaction [22, 23]. Some of the cells from these regions will be destroyed, but the majority of them will actually attach to the wall of the joint cavity and become part of the joint lining [22–24]. At 34 mm the first appearance of the hip joint cavity has been reported by some investigators [23], though more advanced stages have been reported by others, e.g., Haines described a cavity that already surrounds the head of the femur, and ligamentum teres could also be identified within the synovium mesenchyme at this stage [22]. Haines [22] also observed the complete separation (dehiscence) (Fig. 2.2e.1, 2) of the two hip articular surfaces before the embryo reached 45 mm.

## Structure and Composition of the Articular Cartilage

### Cartilage Constituents

The articular cartilage is a hypocellular, avascular, aneural, and alymphatic tissue [25]. The mature articular cartilage is maintained by chondrocytes that are the only resident cell type (with the exception of a stem cell progenitor population [26, 27]) and represent only ~5 % of the wet weight of the tissue [28].

Fluid within this highly hydrated tissue is responsible for 60–80 % of its wet weight and contains water and dissolved gases, small proteins, metabolites, and ions [8, 11, 29]. Structural macromolecules make up the remaining 20–40 % of the wet weight, represented by collagens (15–22 %; mainly collagen type II, but also types IX and XI), proteoglycans (4–7 %), and other non-collagenous proteins [11, 30].

The collagen network is principally responsible for the tensile strength of the cartilage tissue, while the proteoglycans provide the cartilage with elastic resistance to compressive forces [7, 31]. The collagen network interacts intimately with proteoglycan aggregates which are highly sulfated and consist of a protein core (i.e., aggrecan), hyaluronic acid, and sulfated glycosaminoglycan (GAG) side chains made up of chondroitin sulfate, heparan sulfate, and dermatan sulfate [7, 28, 30].

### Zonal Structure

The composition, organization, mechanical properties, cell morphology, and cell function of the articular cartilage vary with the depth from the joint surface. Four different zones have been identified, located from the articular surface to subchondral bone: superficial zone, transitional (middle) zone, deep zone, and the calcified cartilage zone [29].

The superficial zone is the thinnest cartilage zone (10–20 % of the articular cartilage thickness) [32, 33] and consists of two layers: the first layer, closest to the articular surface, is acellular and is comprised of a sheet of fine fibrous material; the second layer consists of flat chondrocytes with their long axis parallel to the cartilage surface. Their matrix is rich in collagen, fibronectin, and water, but is poor in proteoglycans [29], which can be seen with decreased staining with Safranin O (Fig. 2.3). The collagen fibers are also aligned parallel to the joint surface.

The transitional (middle) zone (40–60 %) [32, 33] has spheroidal-shaped chondrocytes and a matrix consisting of larger and less organized collagen fibers, with increased proteoglycan concentration and decreased collagen and water concentration relative to the superficial zone [29]. The deep zone (30–40 %) [32, 33] also has round chondrocytes that tend to organize in columns perpendicular to the articular surface (Fig. 2.3). Among all zones, the deep zone is the least hydrated and has the largest collagen fibers, which are oriented perpendicular to the articular surface, and the highest concentration of proteoglycan [29].

A wavy tidemark of basophilic matrix separates the deep zone from the final zone: the calcified cartilage zone. Collagen fibers lengthen from the middle zone to the calcified cartilage passing through the tidemark (Fig. 2.3). Mechanically, this region transfers joint forces from the cartilage to the underlying subchondral bone via vertically oriented collagen fibrils [34]. Chondrocytes in this zone are surrounded by a calcified matrix and have a small volume and a small amount of intracellular organelles. Overall, the calcified zone marks the transition from the soft cartilage to stiff subchondral bone and is important for connecting non-calcified cartilage to bone [29, 35].

The subchondral bone is interdigitated with calcified cartilage, except that the fibers do not extend from the calcified zone to the bone. The cortical portion of the subchondral bone is localized underneath the calcified cartilage and exhibits low porosity and vascularity, while the subchondral trabecular bone is positioned distally from the cortical bone (Fig. 2.3) and contains trabeculae oriented perpendicular to the cortical subchondral plate [36]. This physical linkage between the cartilage and bone is a critical component in the pathogenesis of degenerative diseases such as OA [34].

### Extracellular Structure

The composition and organization of the extracellular matrix also change with the distance from the cell and can be divided in three regions: the pericellular, territorial, and interterritorial matrices [29]. The chondrocyte and its pericellular matrix form the chondron that is the basic cellular structural unit of the cartilage [37, 38]. The chondrocyte cell membrane appears to attach to the surrounding pericellular matrix, which is rich in proteoglycans (derived from aggrecan, perlecan, and biglycan), collagen (a non-fibrillar collagen type VI, which is specific to the pericellular matrix), and small amounts of collagen types II and IX), hyaluronan, and fibronectin [39, 40]. It is well established that the interactions between the extracellular matrix and chondrocytes are vital for normal cartilage function. The close contact of the pericellular matrix with the chondrocyte membrane suggests that cellular activities are critically regulated by these cell-matrix interactions [29, 40, 41].

The next region is the territorial matrix that surrounds the pericellular matrix of individual chondrocytes or a group of chondrocytes in some areas (e.g., the chondrocyte column in the deep zone) [29, 32]. This region contains thin collagen fibers that appear to adhere to the pericellular

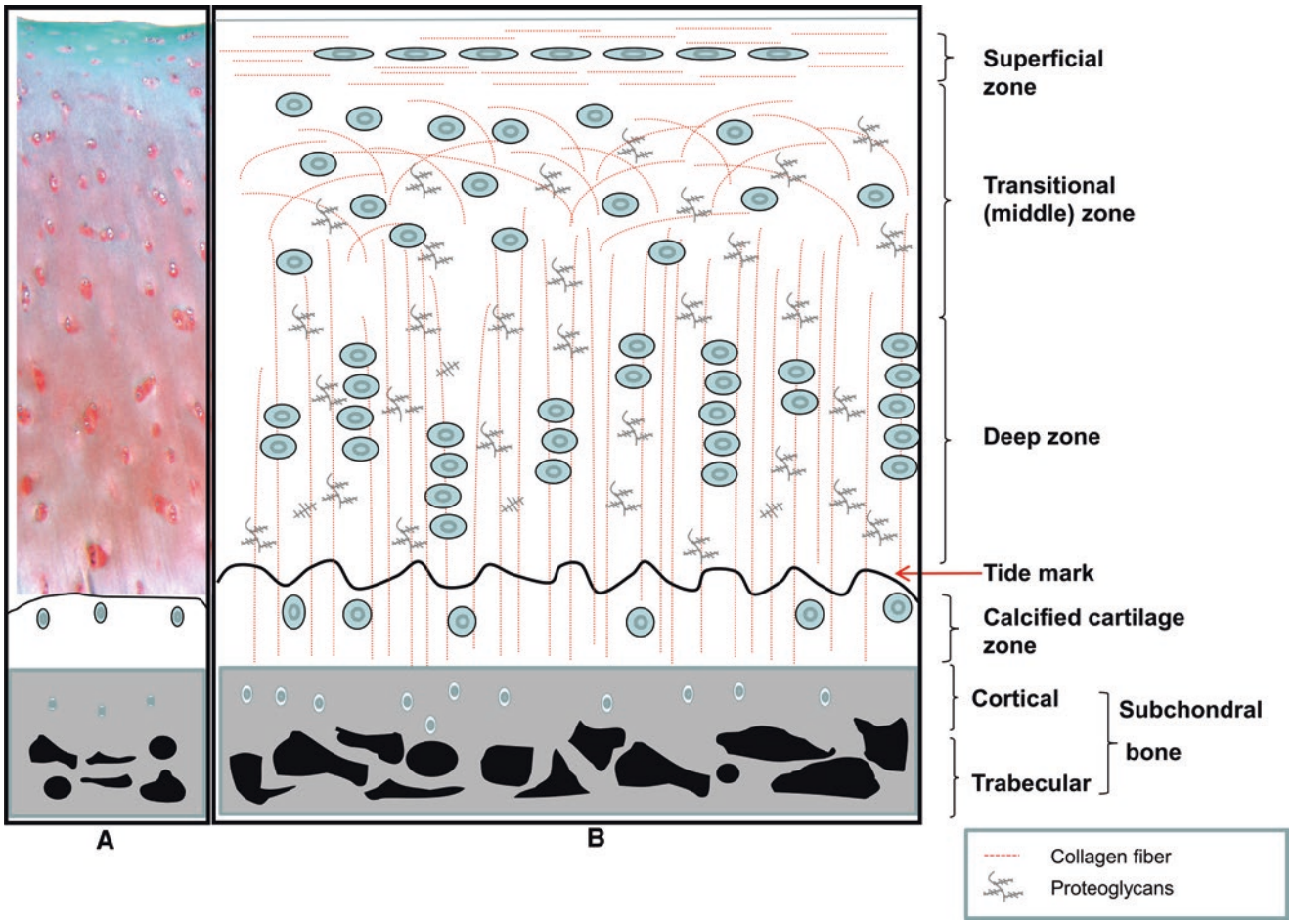


**Fig. 2.3** The articular cartilage structure. Safranin O staining of femoral head cartilage from a 34-year-old woman (**a**); schematic of the articular cartilage (**b**). The articular cartilage consists of four different zones from articular surface to subchondral bone: superficial zone, transitional (middle) zone, deep zone, and the calcified cartilage zone. The superficial zone is rich in collagen, fibronectin, and water, but is poor in proteoglycans [29] which can be seen with decreased Safranin O stain (**a**). A wavy tidemark separates the deep zone from the calcified cartilage zone. Collagen fibers lengthen from the middle zone to calcified cartilage passing through the tidemark, but do not extend to the subchondral bone

matrix. These fibers intersect at different angles, at some distance from the cell, to form a fibrillar network around each chondrocyte [29].

The majority of the chondrocyte matrix is represented by the interterritorial matrix, which is the furthest away from the cell and contains thick collagen fibers. The orientation of these fibers is different than that in the territorial matrix, as they do not surround the chondrocytes but are angled differently in the distinct cartilage zones, changing their orientation from parallel to the articular surface in the superficial zone to perpendicular in the deep zone [29].

## Chondrocyte-Matrix Interaction

Dynamic cell-matrix interactions are essential for the biological functions of the cartilage. Chondrocytes constantly remodel their matrix in response to different stimuli, such as physical and chemical changes in the neighboring environment [42]. The stimuli that can trigger anabolic or catabolic responses from chondrocytes include mechanical forces (e.g., exercise), biomolecules (e.g., cytokines, local growth factors, hormones), and matrix composition [28].

The matrix not only protects the chondrocytes from mechanical insults but also provides the cells with signaling cues that regulate gene expression. There is a direct interaction between the extracellular matrix molecules and chondrocytes through cell surface receptors such as the integrins, a family of dimeric transmembrane adhesion receptors consisting of α and β subunits [43, 44].

Integrins play an essential role in the attachment of cells to their extracellular matrix, but also in chondrocyte differentiation, survival and matrix regulation, and in mediating the response of chondrocytes to numerous signals, including mechanical loading. These receptors are known to interact with pericellular matrix proteins, such as collagen type VI