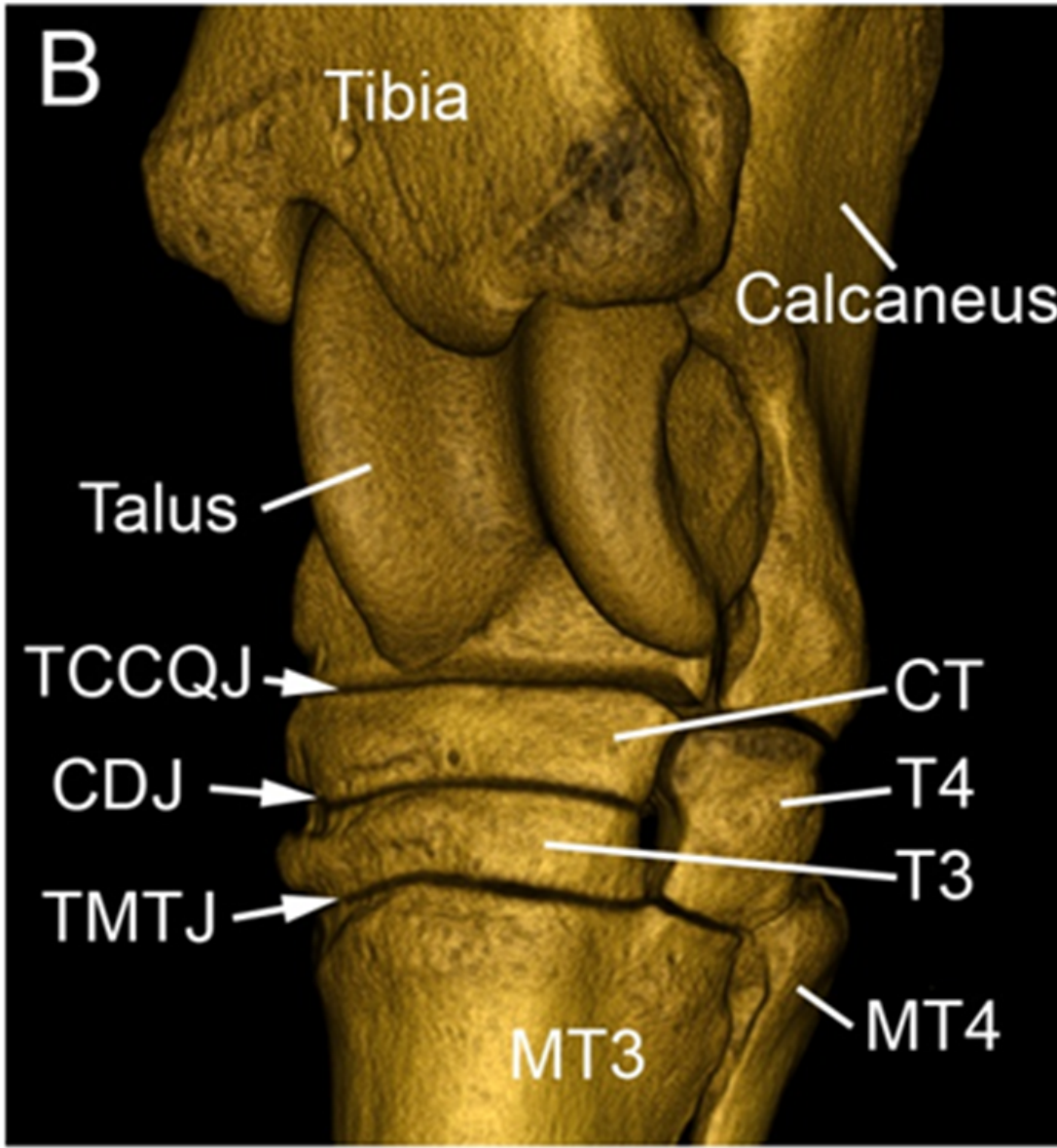
Bone spavin
dtOA

Old disease
New knowledge
New interpretations

Dr Sigríður Björnsdóttir

2025

Distal tarsal OA



„Old disease“

Spavin has been known as a cause of lameness ever since horses were first used by man (Schebitz 1965)

- First described and distinguished from other conditions of the tarsus by Busch in 1788
- Characterized by periosteal new bone formation medial to the dital hock
- In 1882, Havemann related the disease to the joint surfaces and ankylosis of the distal tarsal joints (Wamberg 1955)

Bone spavin has existed in the Icelandic horse since the origin of the breed, more than 1000 years ago

- Evidences found on tarsal and metatarsal bones preserved from heathen graves, i.e before year 1000
- Based on clinical signs, bone spavin was described to be a common disease in Icelandic horses at the end of the 18th century (Einarsson 1931)

Relics from heathen graves

Swedish study of Icelandic horses presented in 1994

Prevalence of radiographic signs (RS) 23%

- N=375
- 0-19 yr, mean age 8,1 yr
- One projection (dorsolateral- plantaromedial oblique)
- Horses younger than 5 years did not show any radiographic signs

Prevalence of +flexion test of the tarsus 25%

- Significant correlation between RS and lameness and +flexion test

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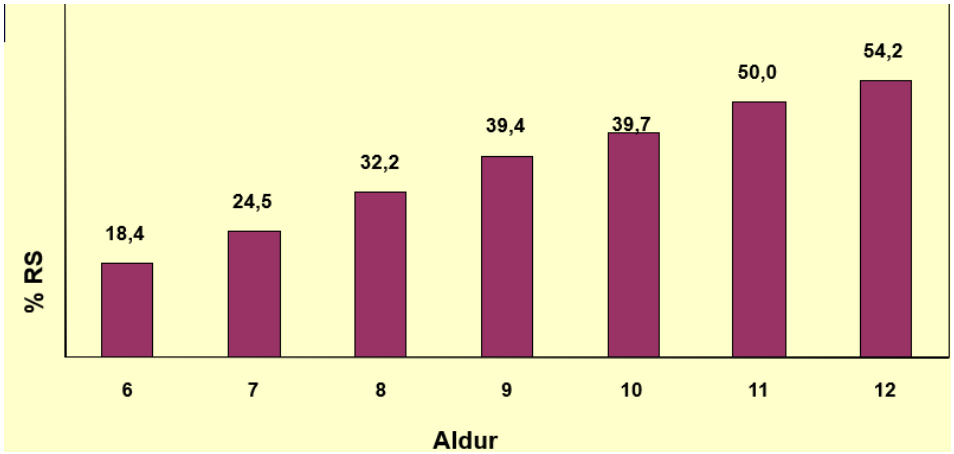
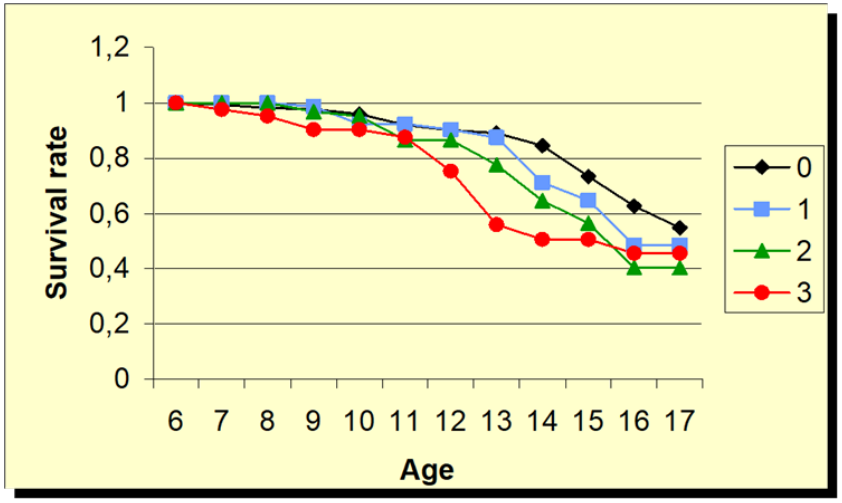
6-12-years old horses in use for riding

Prevalence and clinical relevance 1995-1996

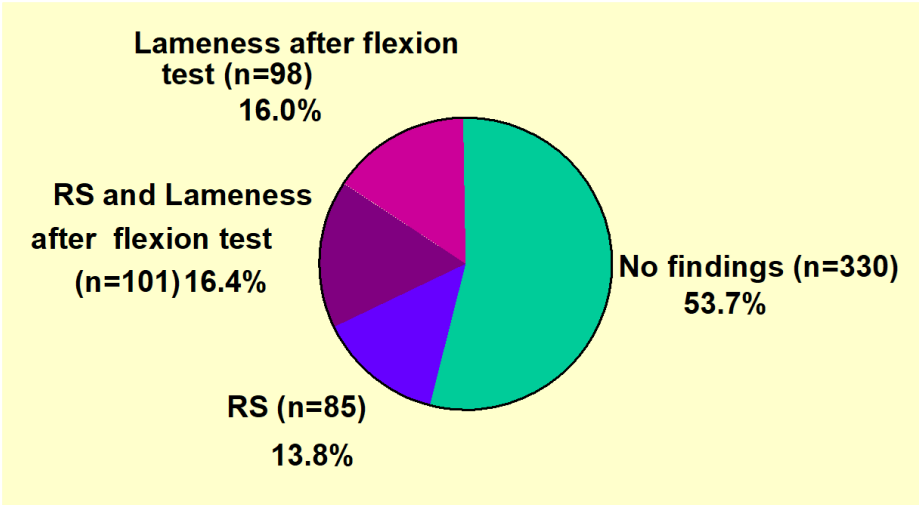
Precalence of radiographic signs 30,3%



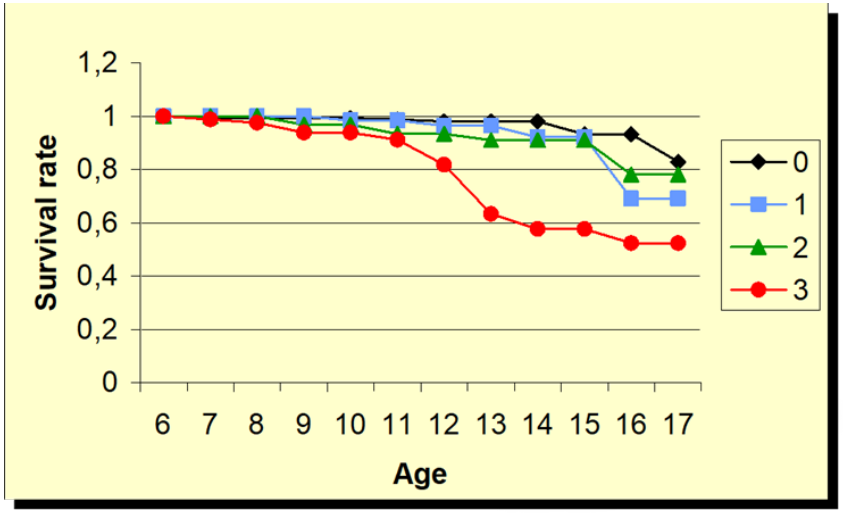
5 yr culling rate – all reasons



Relation to flexion test of the tarsi



5 yr culling rate- hind limb lameness



Risk factors – aethiological factors

Genetic predisposition

- **Estimated heritability h^2= 33%**
  - For age-at-onset of RS
    - A quantitative theshold trait
    - Underlying normal distribution of multigenetic effect
- Age
- Tarsal angle (small effect)

Not a training related disease

- No effect of workload and other environmental factors
  - Age when broken to saddle,
  - Breeding evaluation
  - Competing
  - Training intensity
  - Toelt ruled out as a risk factor

Screening for dtOA
Included in the breeding program from 2005

Stallions presented for breeding evaluation	• From the age of 5
The results registered in the data base WF	• **S** RS of dtOA not detected • **S** RS of dtOA
Breeders avoided to use stallions with **S** in breeding	• And they do

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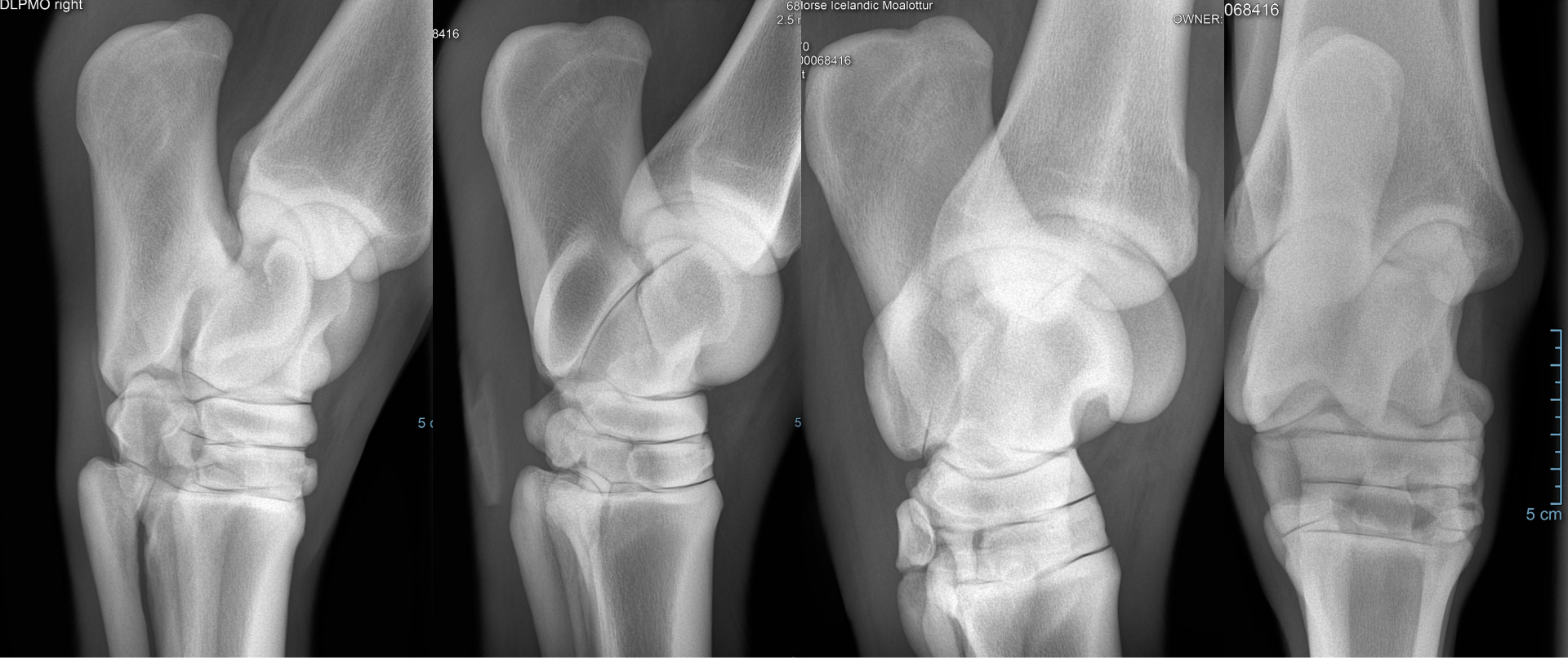
DLPMO right
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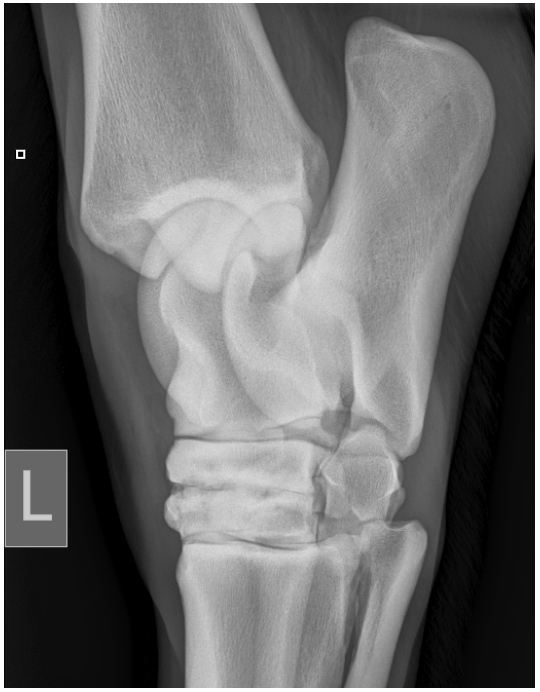
OWNER:
068416
5 cm


R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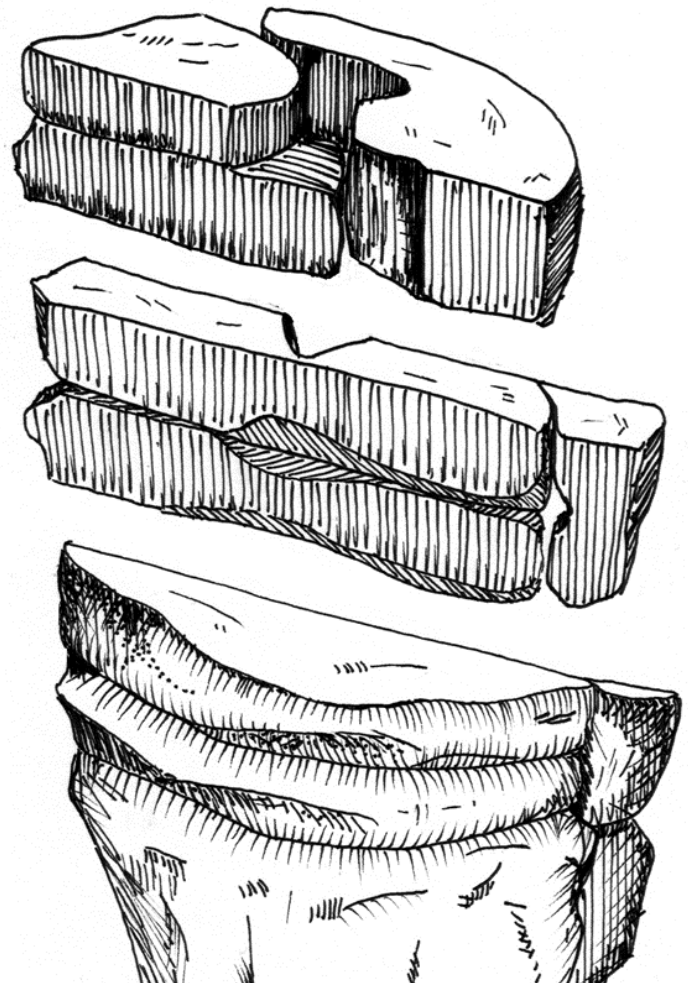
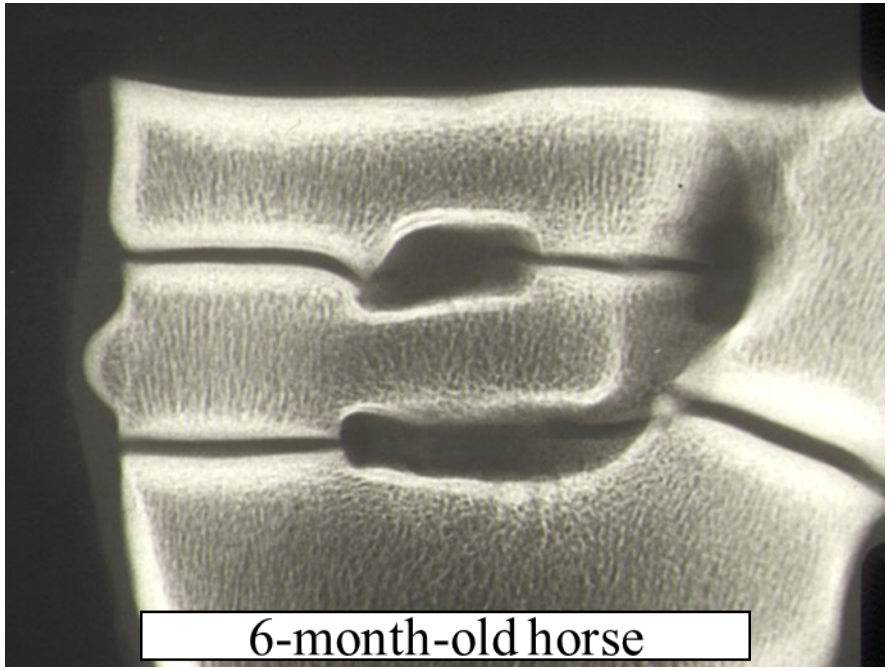
0,5 – 4,5-year-old

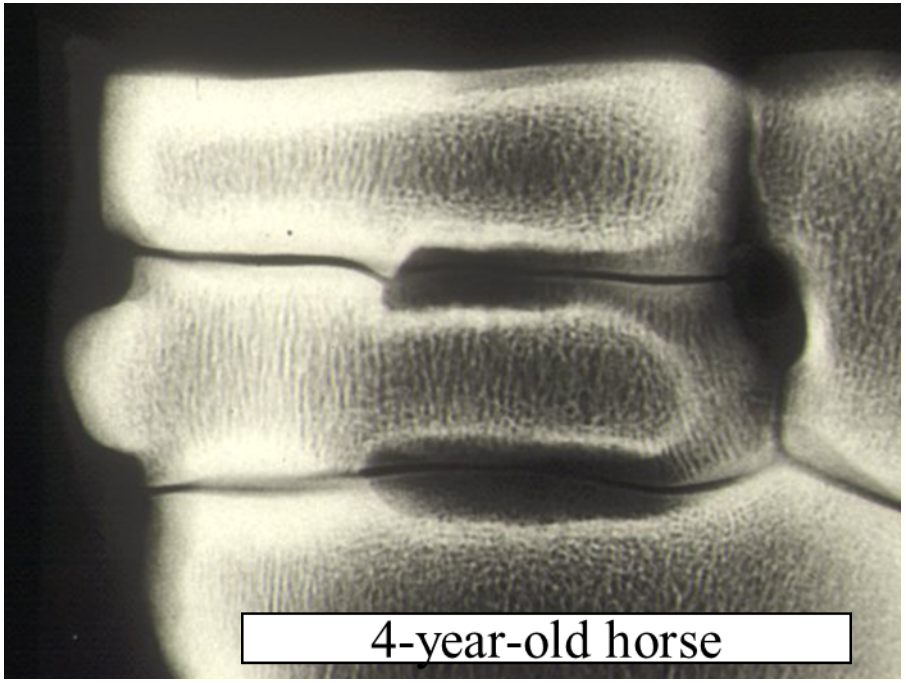
When does it start?

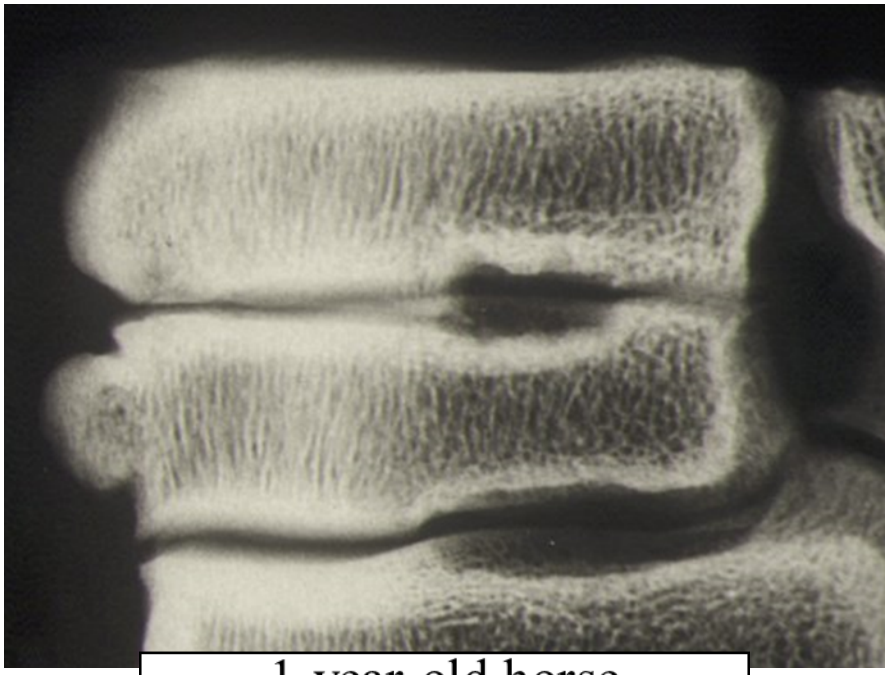
- Material from slaughterhouses
  - N=111
- Localization within the joint
- Cartilage/bone ?

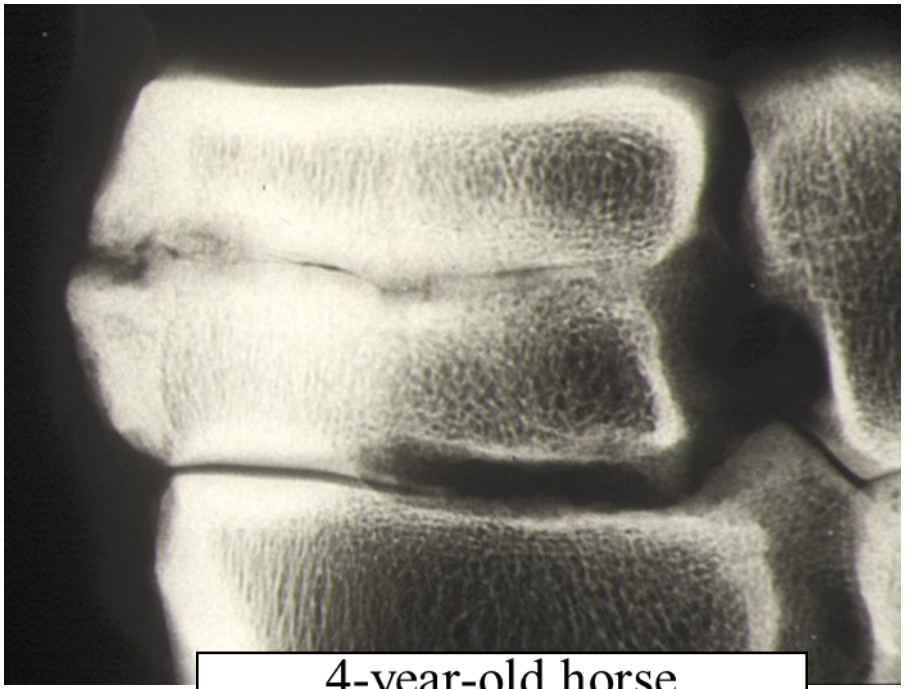
- High detail radiography
- Histology

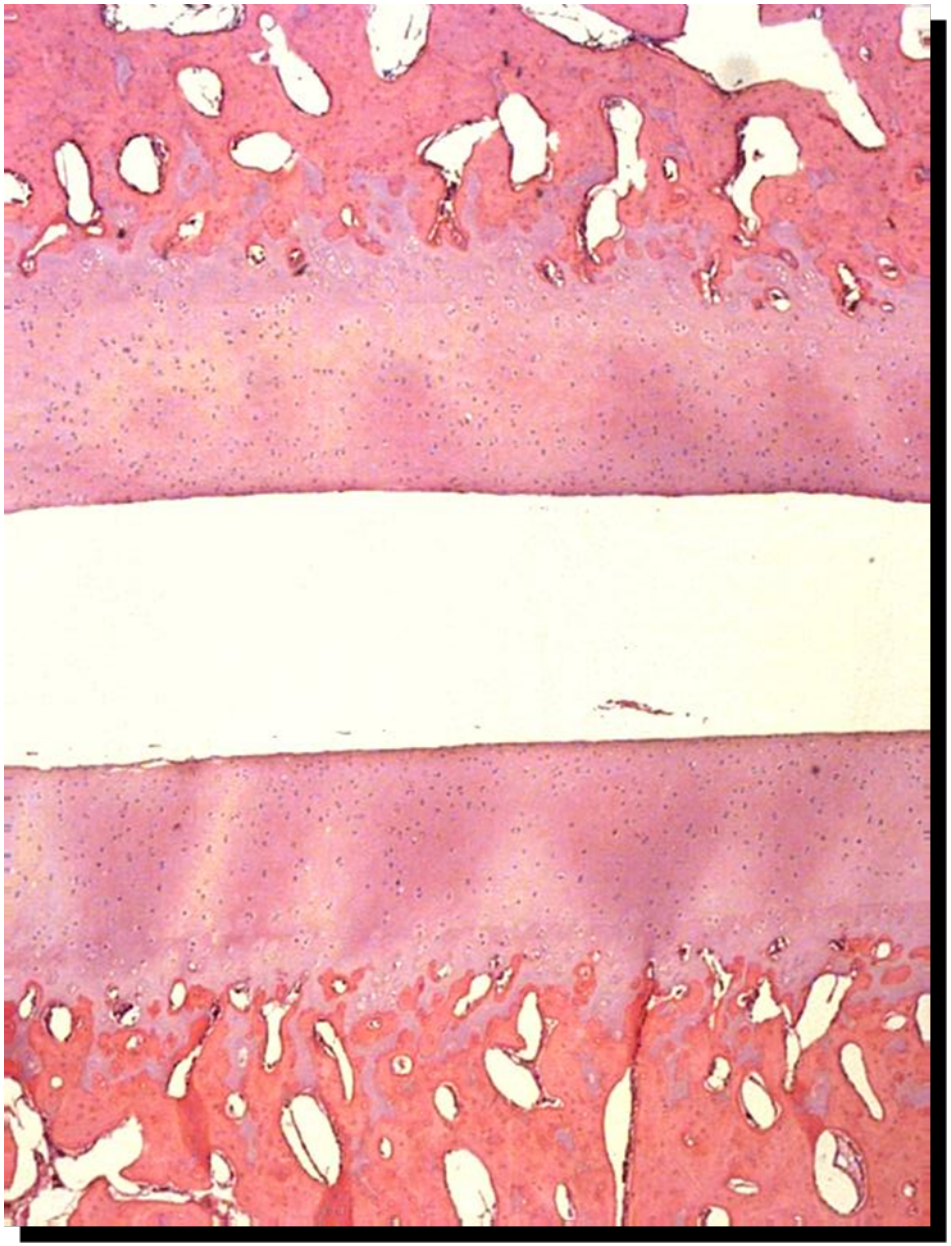


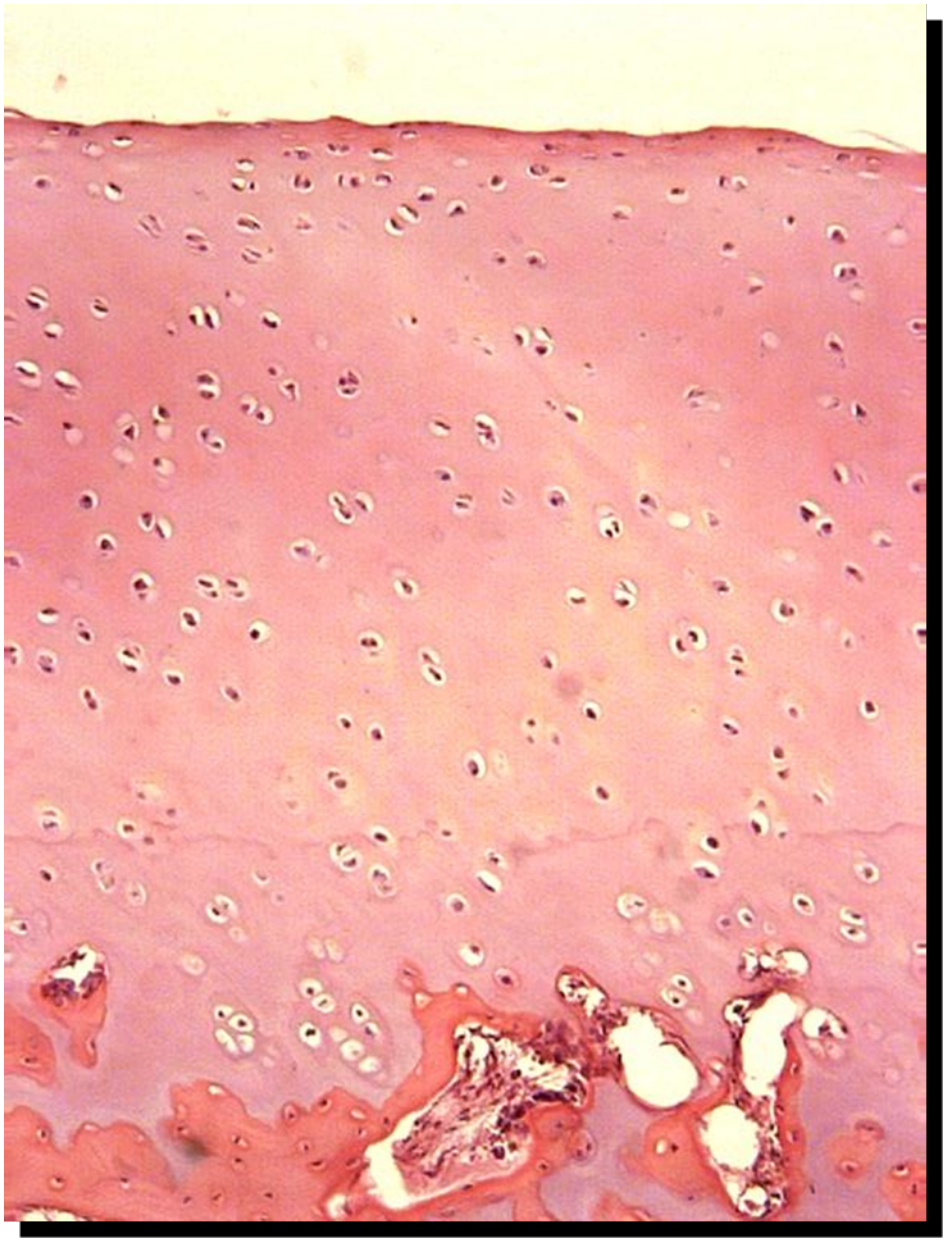

6-month-old horse


4-year-old horse

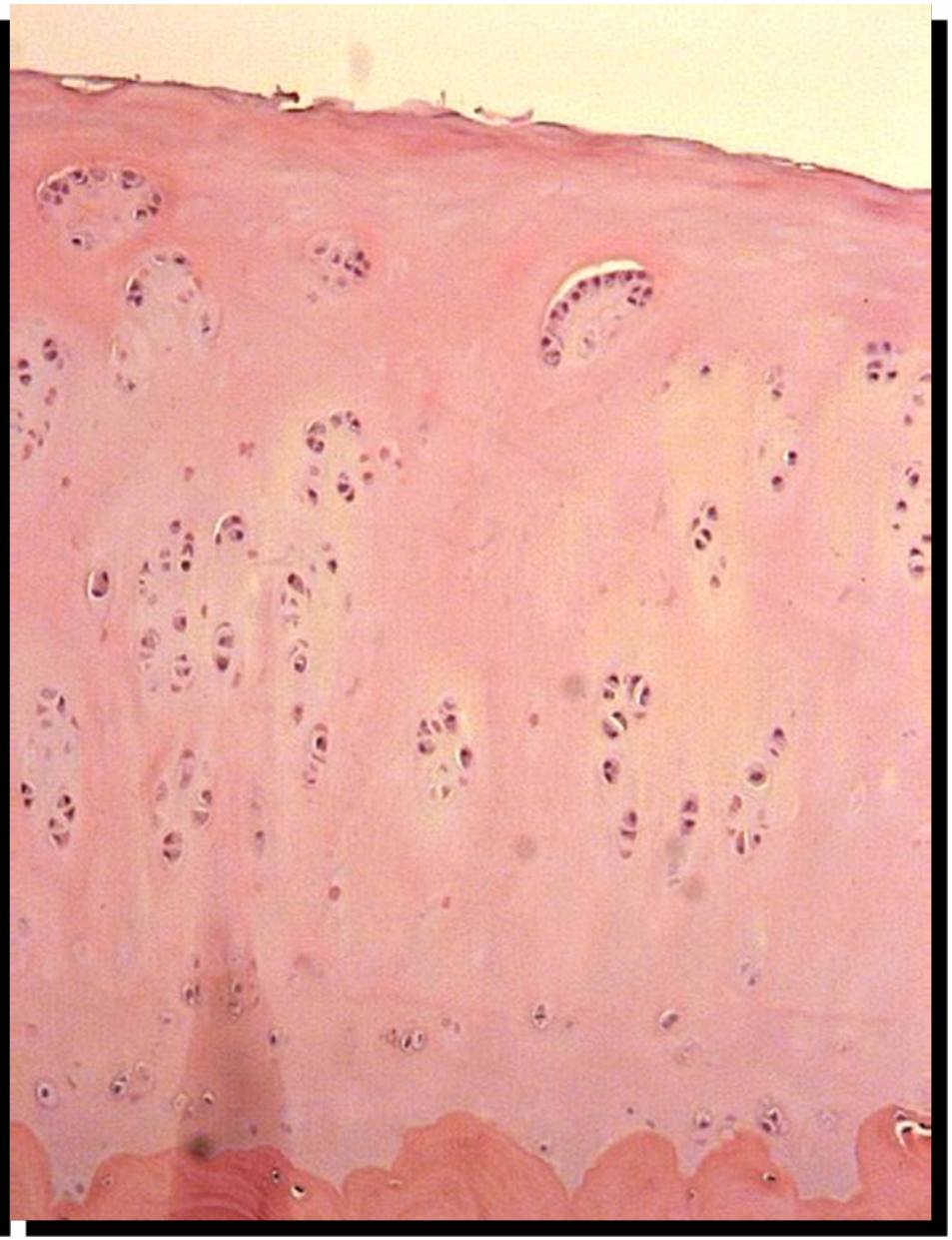

1-year-old horse


4-year-old horse



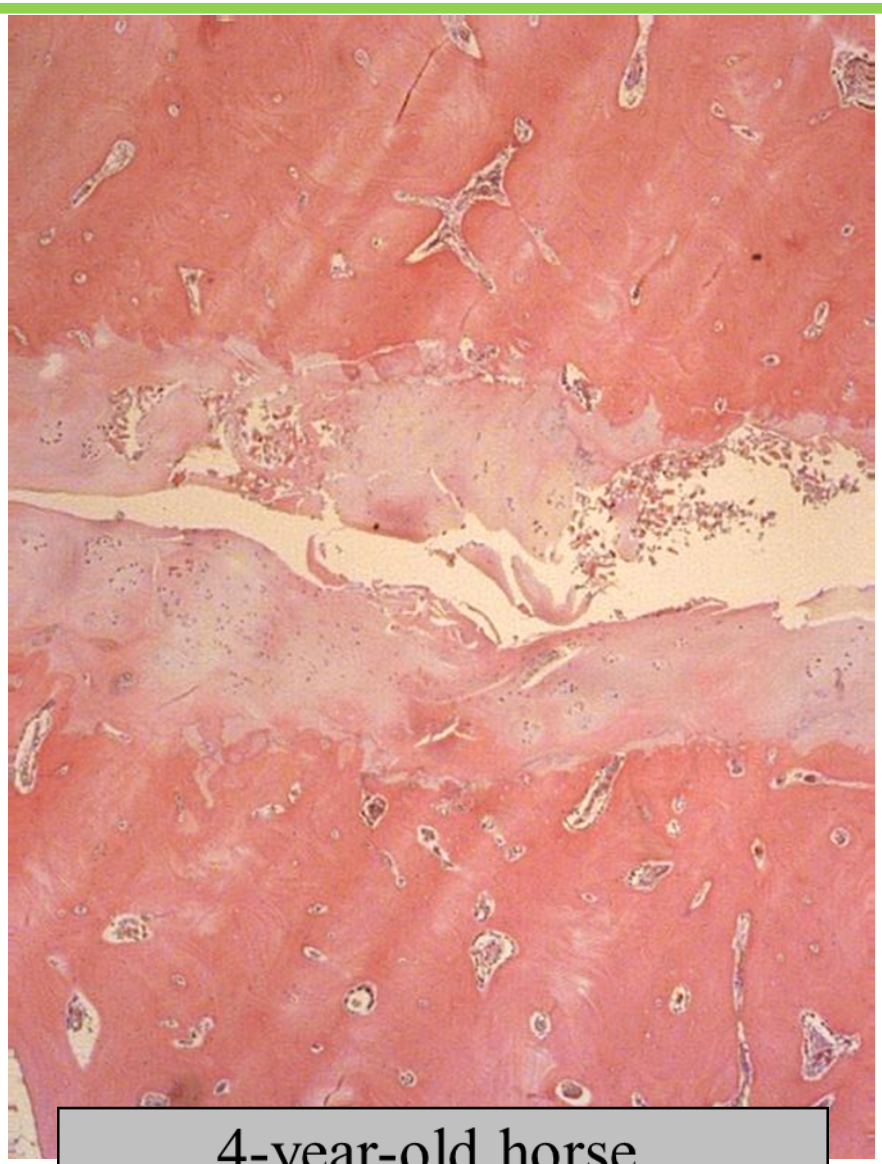


6-month-old horse



1-year-old horse

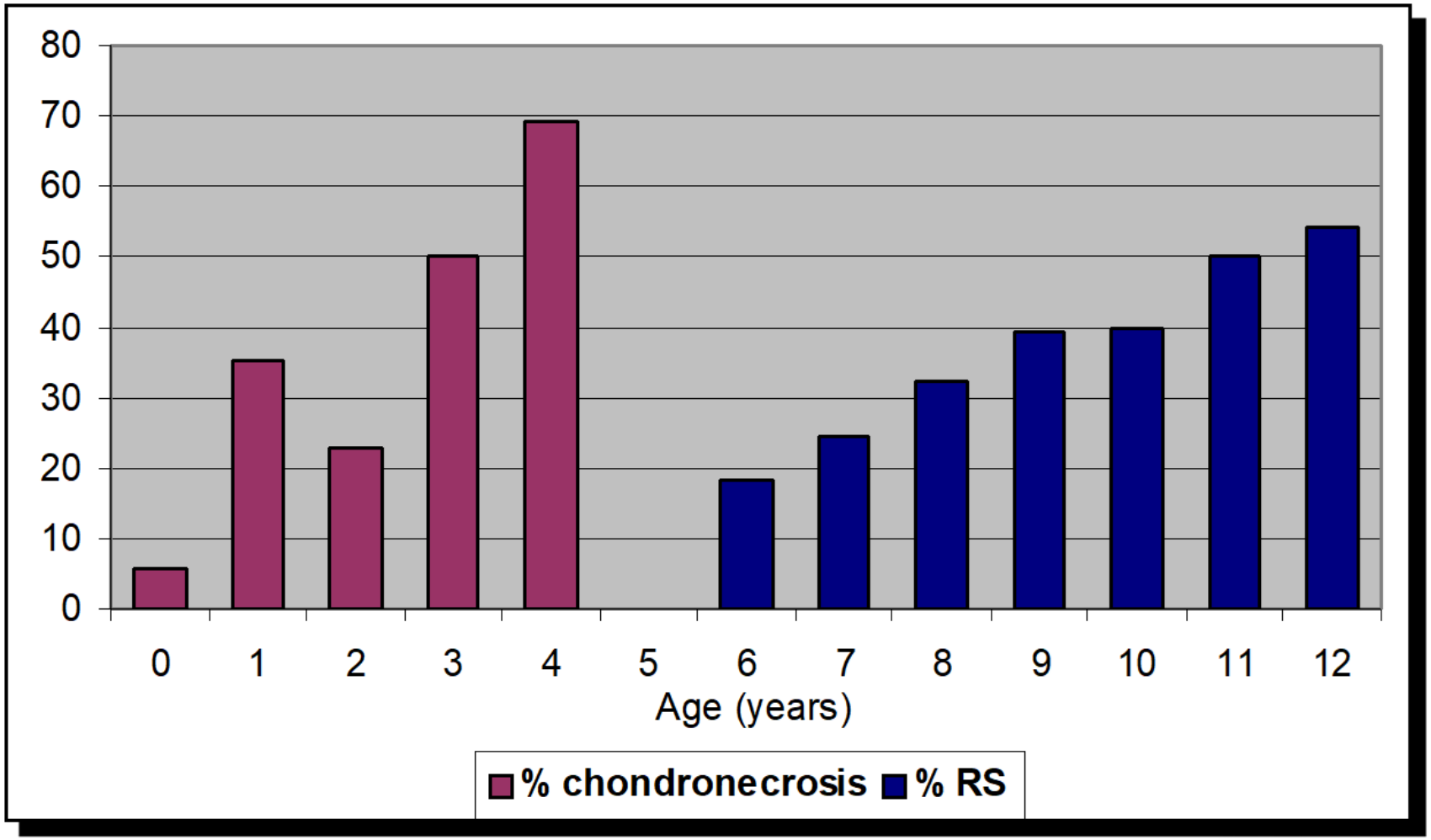
Histological findings strongly indicate the pathogenesis of OA



4-year-old horse

- Diffuse chondronecrosis
  - ↓ Proteoglycans
  - ↑ Collagen
- Cluster formation of chondrocytes
- Disruption of cartilage/bone interface
- Full thickness necrosis
  - -> dtOA

High prevalence of chondronecrosis in young horses



- Histological OA detected <1 year old
- Radiographic OA before 4 years old

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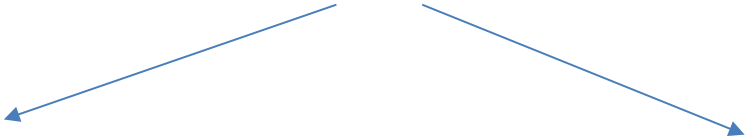
Pathogenesis?
Biomechanics, biochemical, **genetic**

Initial structure damage



- Early OA → Joint healing
  - Pre-radiographic
- Established OA
  - Radiographic OA

Where, within the joint does it start

- why and how –

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2,5-year-old horses

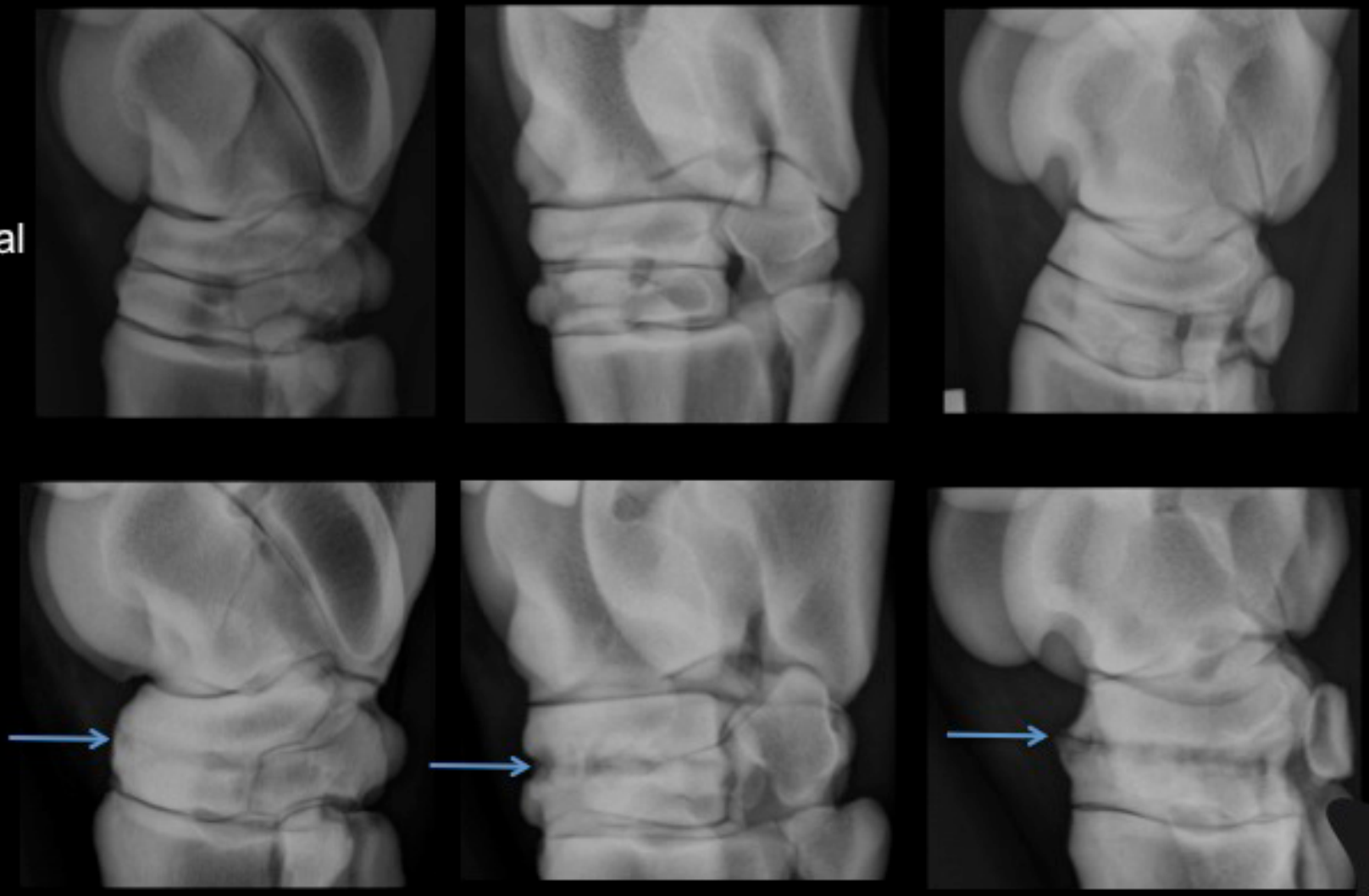
38 horses

- Approximately 50% with parents with or without OA
- Same environment
  - Lived together in Northen Iceland
  - No training
- Slaughtered for human consumption
  - At the age of 2,5 yr
- Tarsal joints in Uppsala within 50 hours

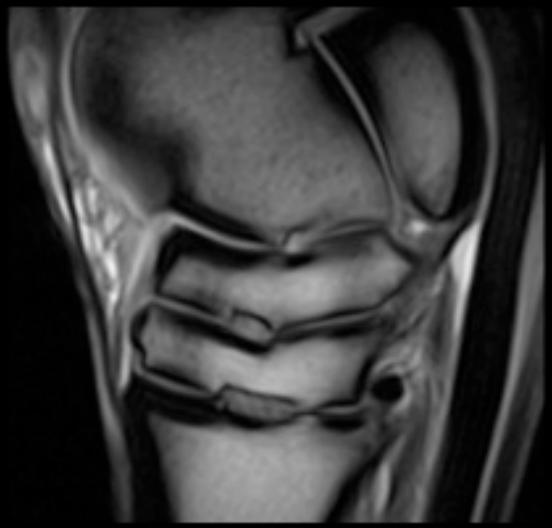
Methods

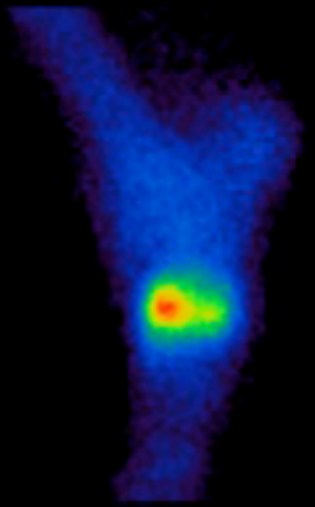
- 3D video morphometry
  - 3 times, one year intervals
- Regular blood samples
  - To identify serum markers
- Radiograph tarsal joints
  - 2.3 yrs
- CT, high and low field MRI
  - Tarsal specimens
- Microscopy-histology, scanning EM, confocal scanning light microscopy

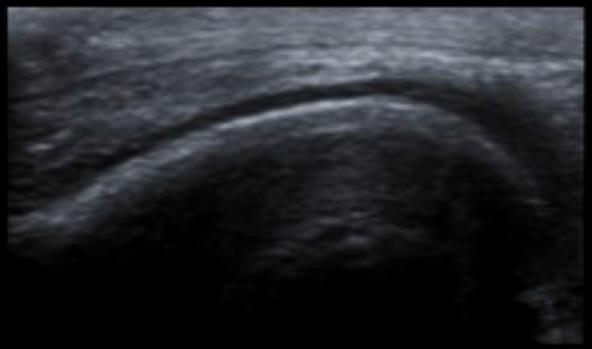
Radiographic distal tarsal OA

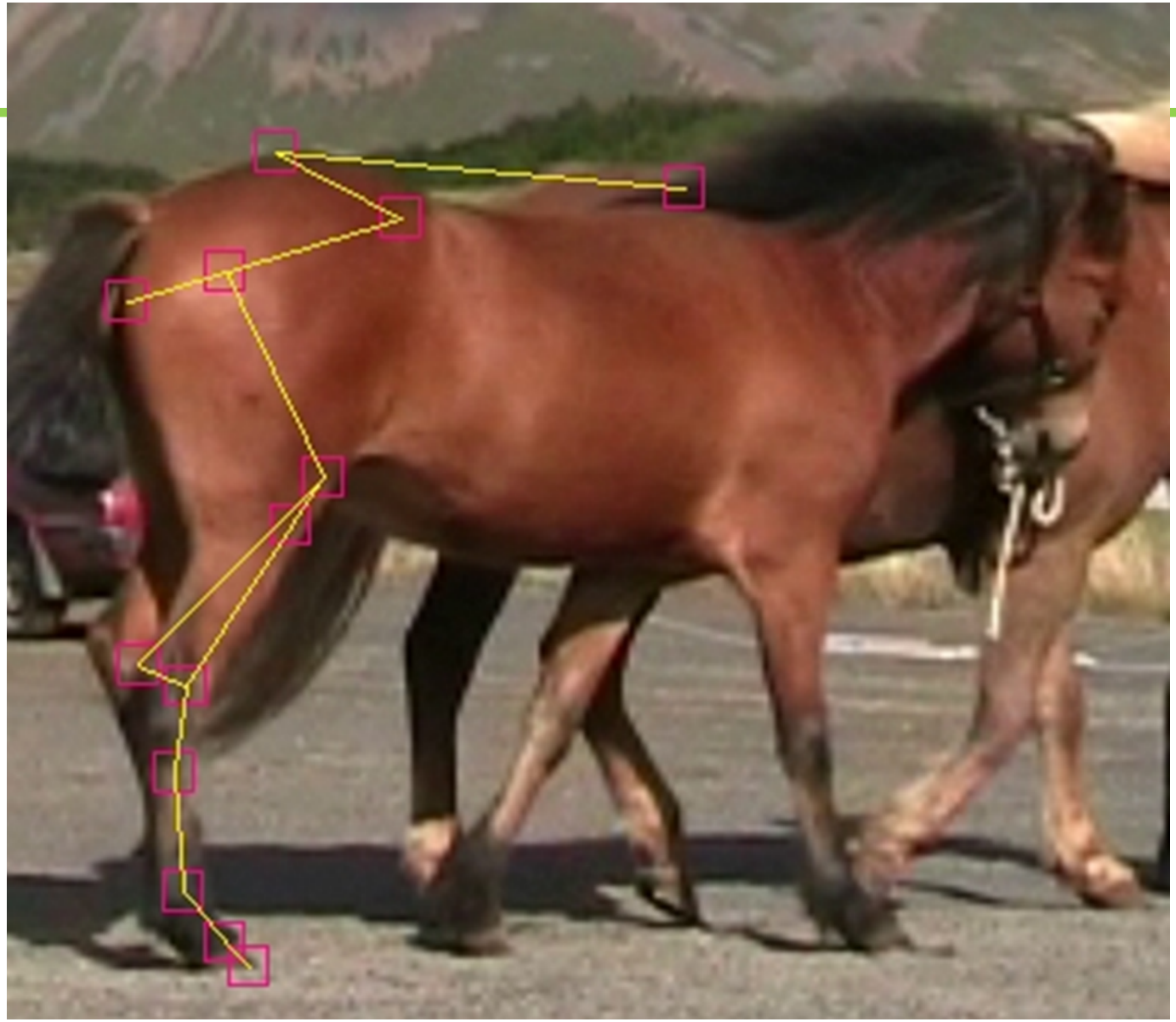


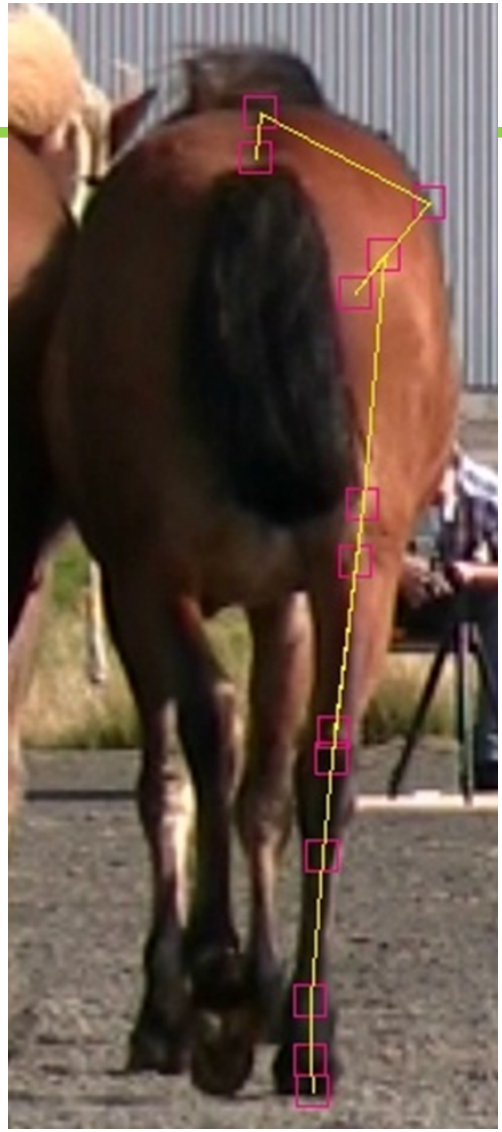
Imaging the joint organ











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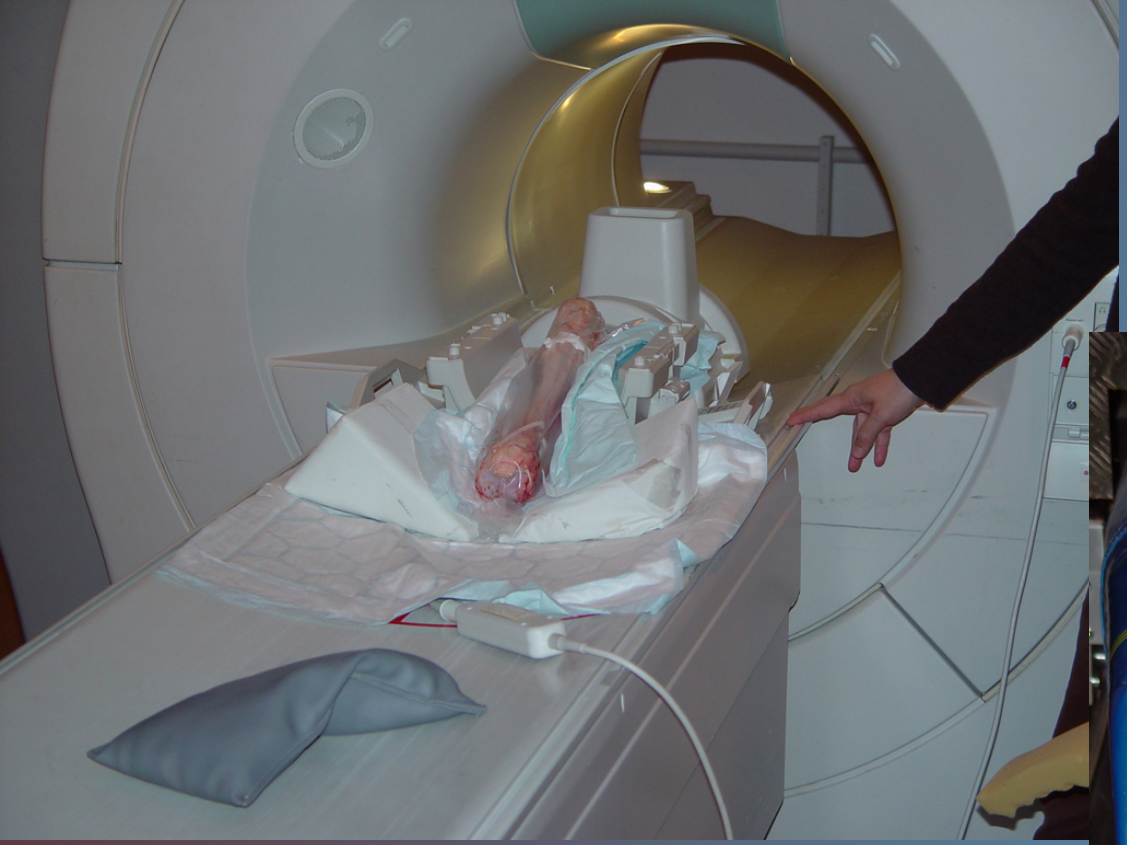


WL: 987 WW: 593
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WL: 987 WW: 593
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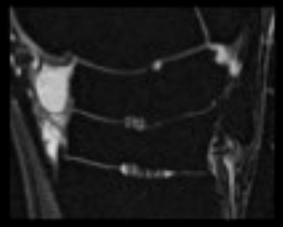
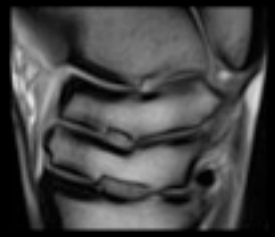
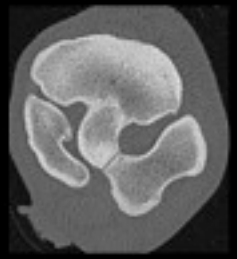
Methods

Iceland

Uppsala







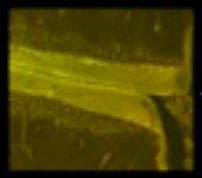
1.5T MRI 0.27T MRI CT

Image
co-registration

CT slab

Histology
Uppsala

Confocal scanning
Light microscopy



London

Scanning EM

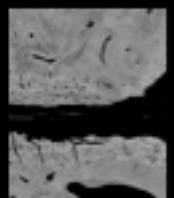


Image guided
sample selection

SLU

Early OA morphological changes

- First lesions found in the hyalin cartilage and then in the calified cartilage – not in the subchondral bone
- The lesions
  - articular mineralisation front defect,
  - central osteophytes and
  - hyperdense mineralisation front protrusions
- Radiography better or equal to low-field MRI
  - For diagnosting the early changes
- Articular mineralisation front defects were identified as a highly specific imaging feature in radiographs for early OA.

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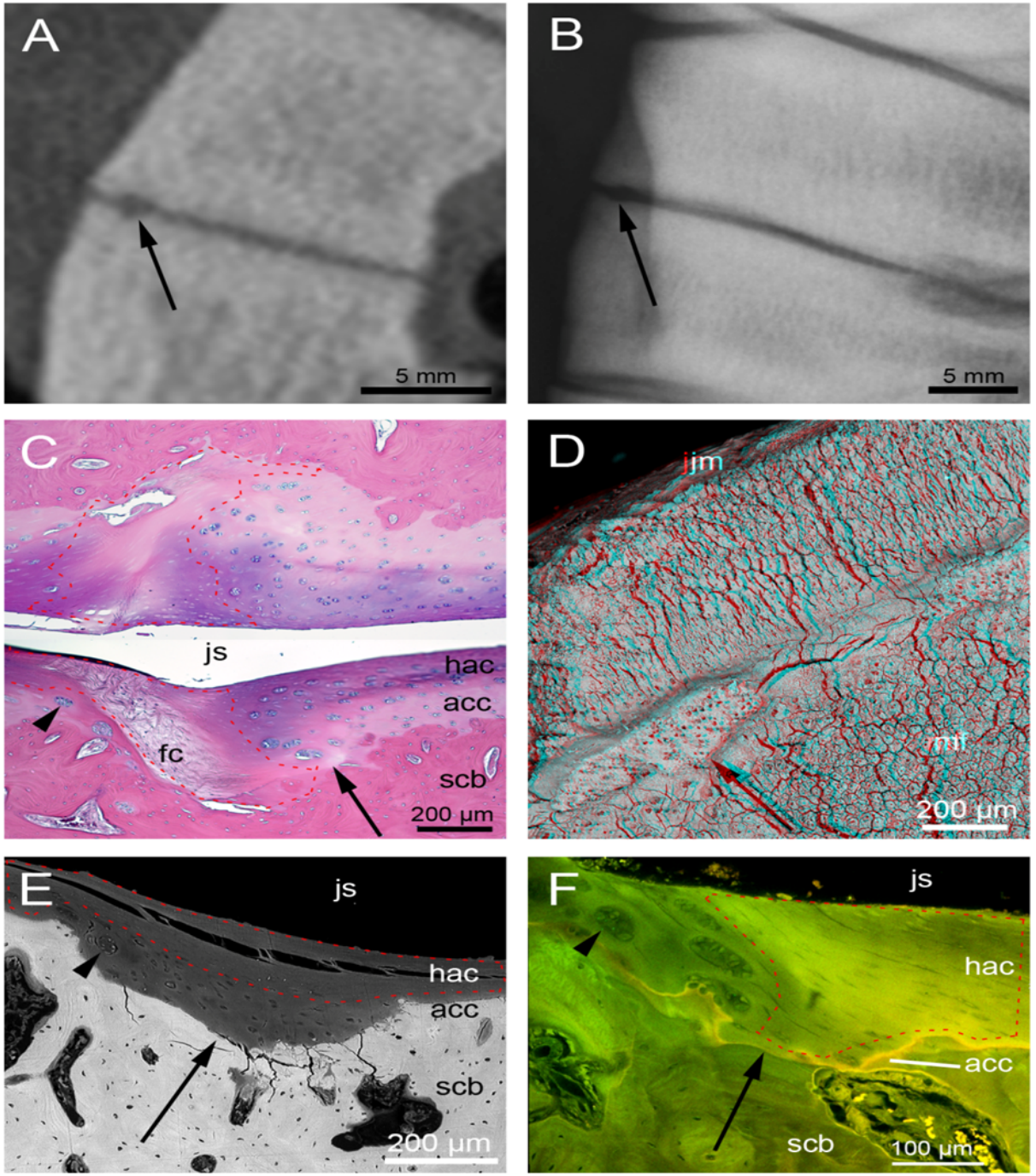
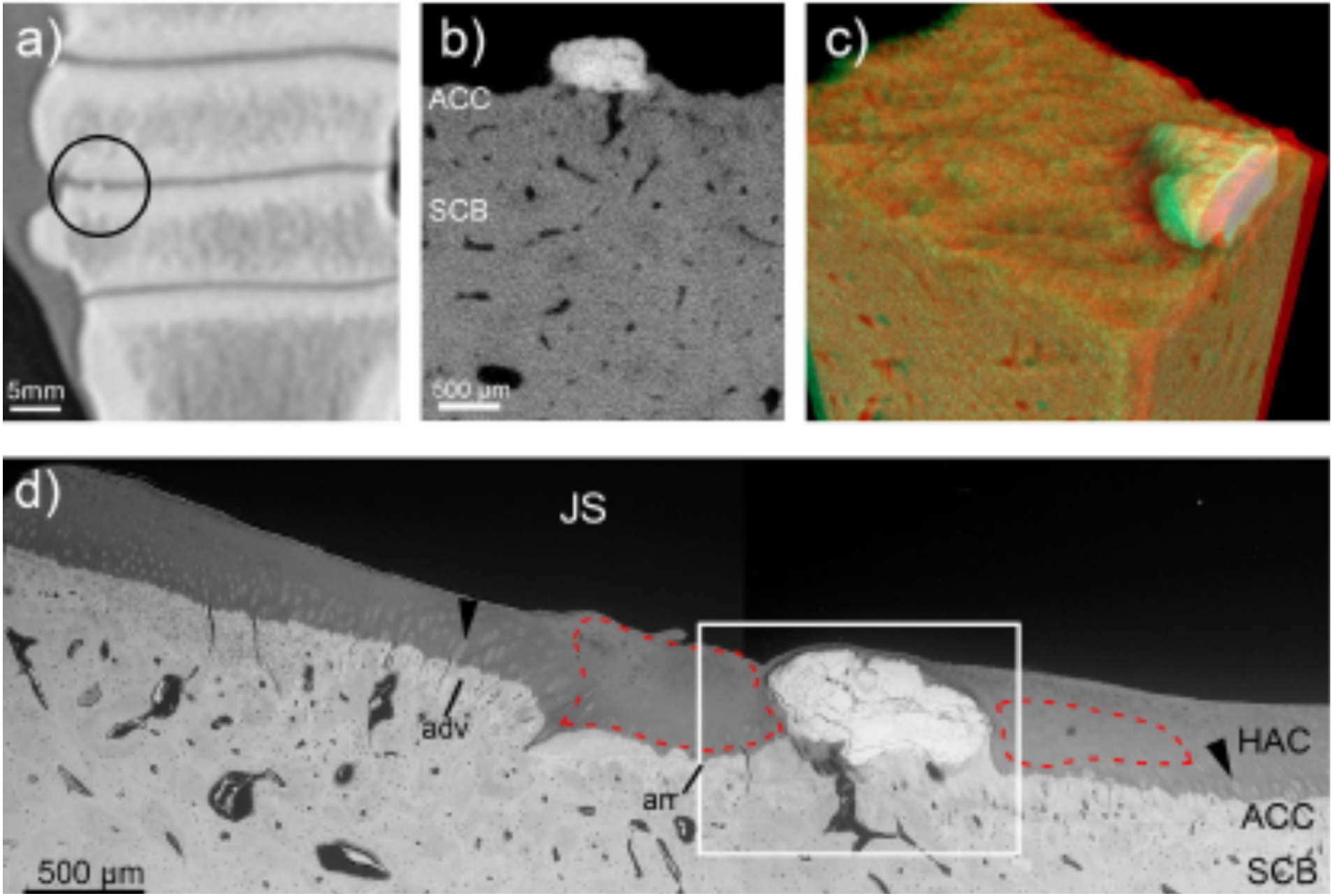
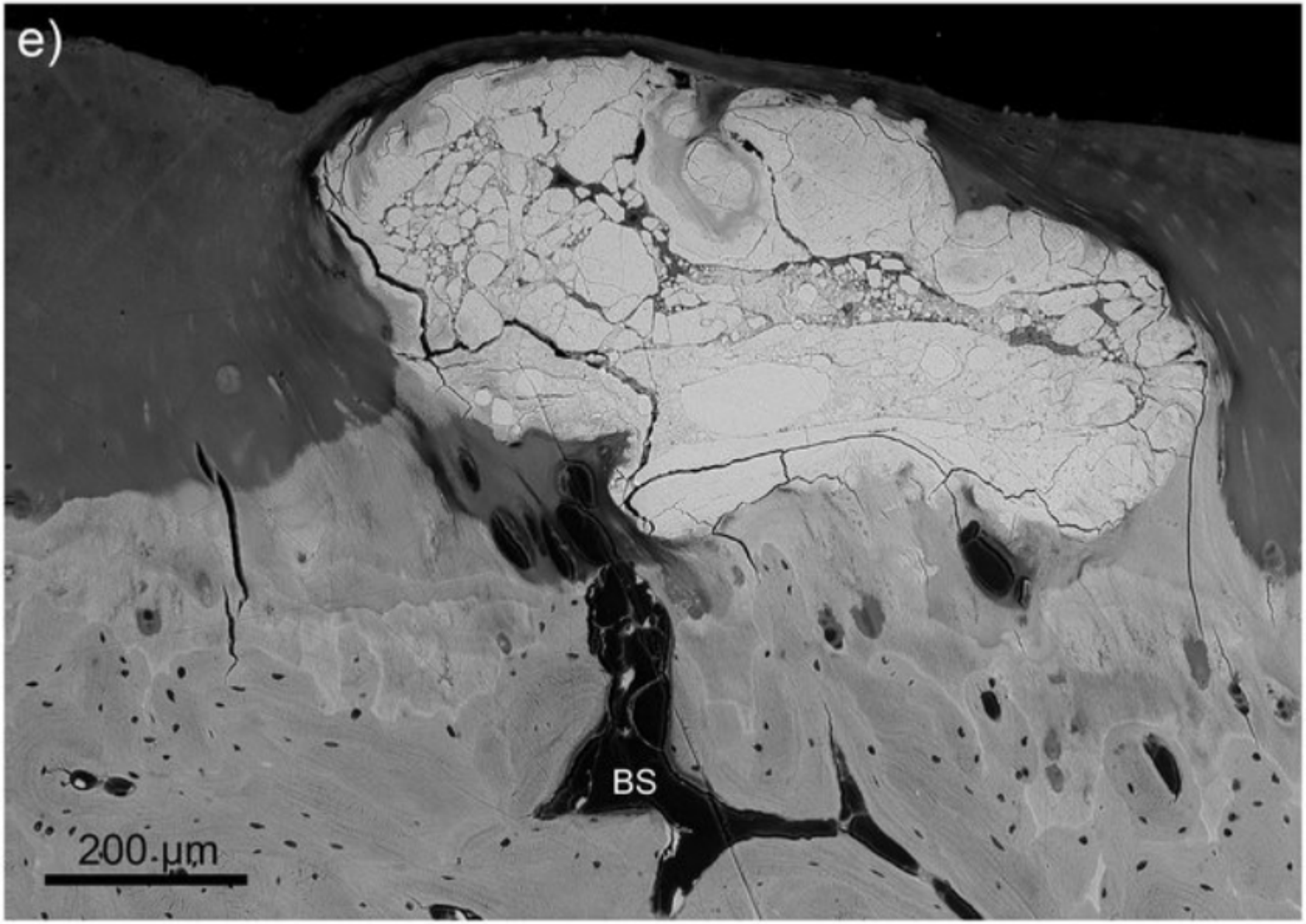
Articular mineralisation front defects

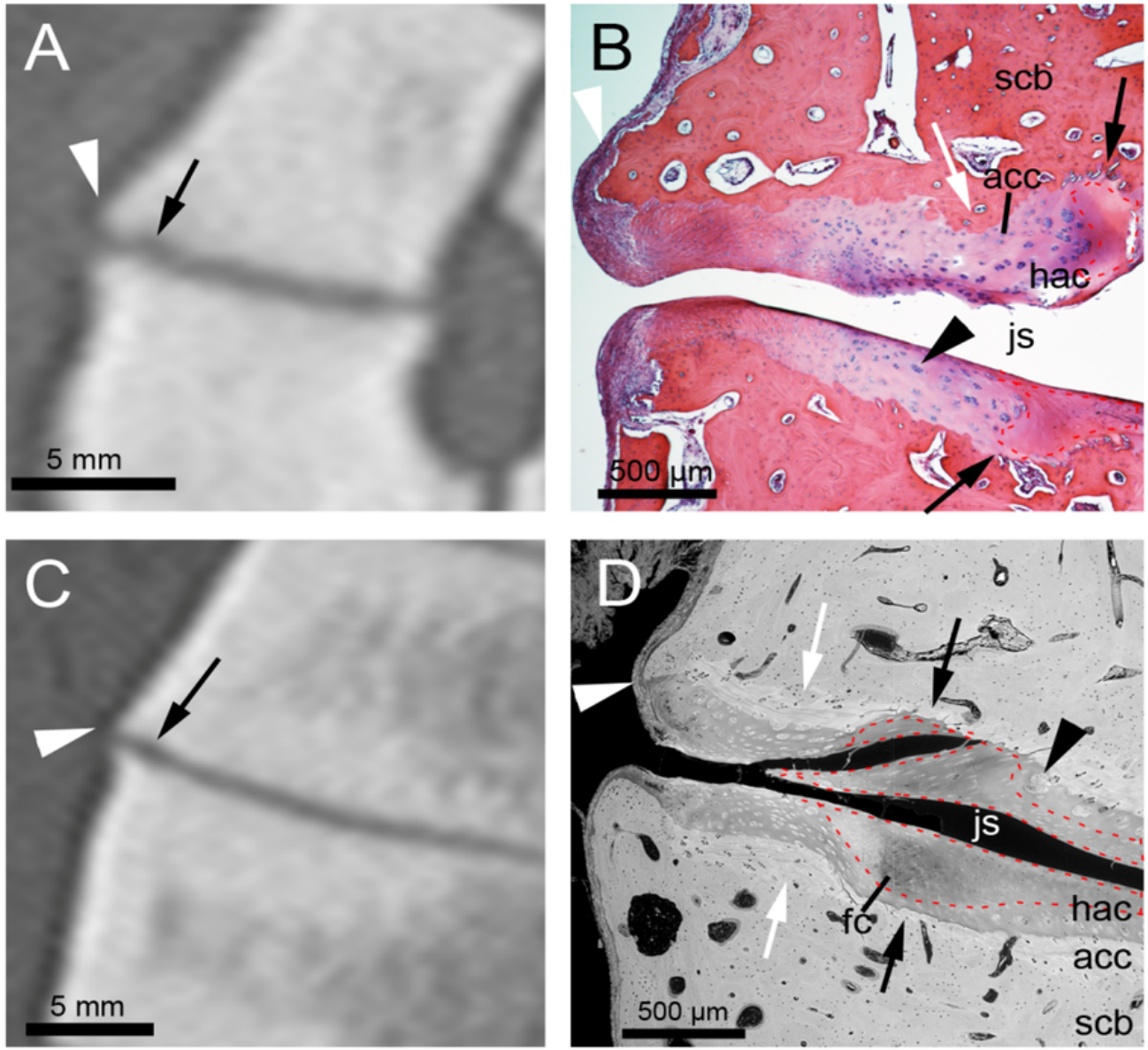


Central osteophytes

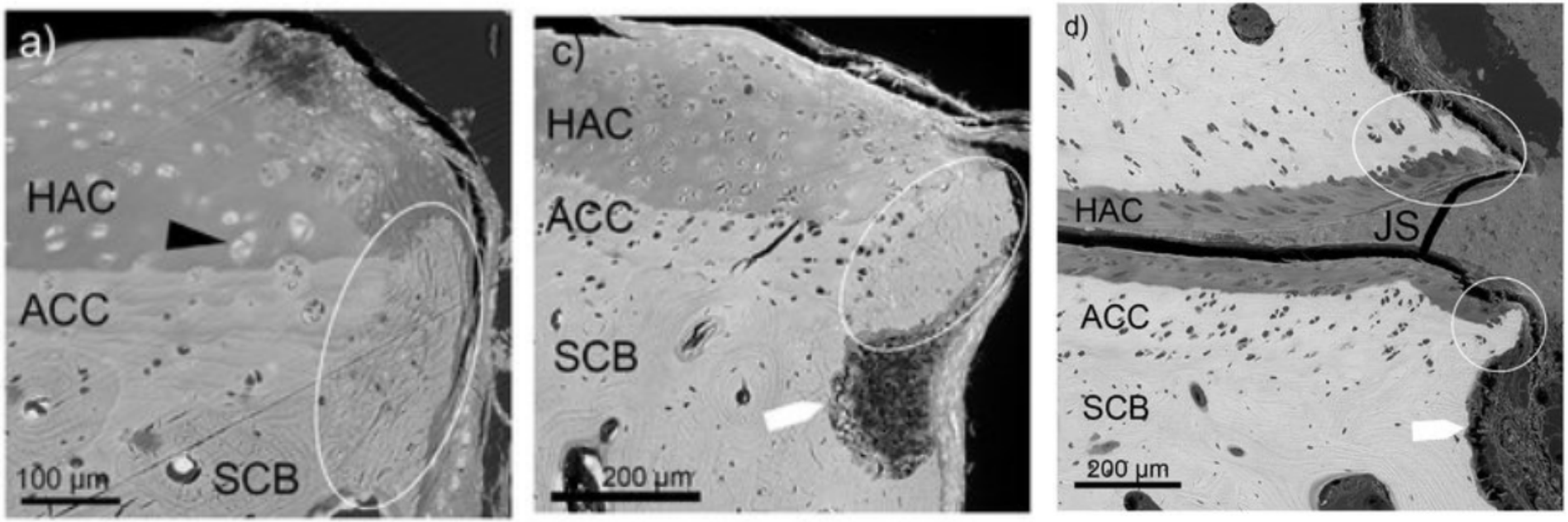



e)
BS
200 µm

- Marginal osteophytes (white arrowheads)
- Articular mineralisation front defects/articular calcified cartilage arrest (black arrows)
- Central osteophytes/articular calcified cartilage advancement (white arrows)
- chondrocyte clusters (black arrowheads)
- areas of chondrocyte necrosis (regions within the red dashed lines)
- fibrocartilage (fc) and a full thickness split of the hyaline articular cartilage



Joint margin lesion-types

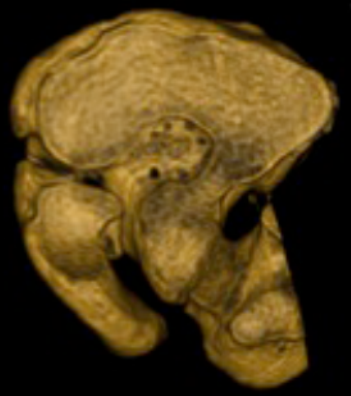


- (a,c,d) mineralisation of the periosteum/joint capsule
- (c,d) Joint margin extensions and adjacent joint margin erosions (compact arrow) that result in an elongated spur shape of the joint margin.

Paper 1 - Results

Dorsal

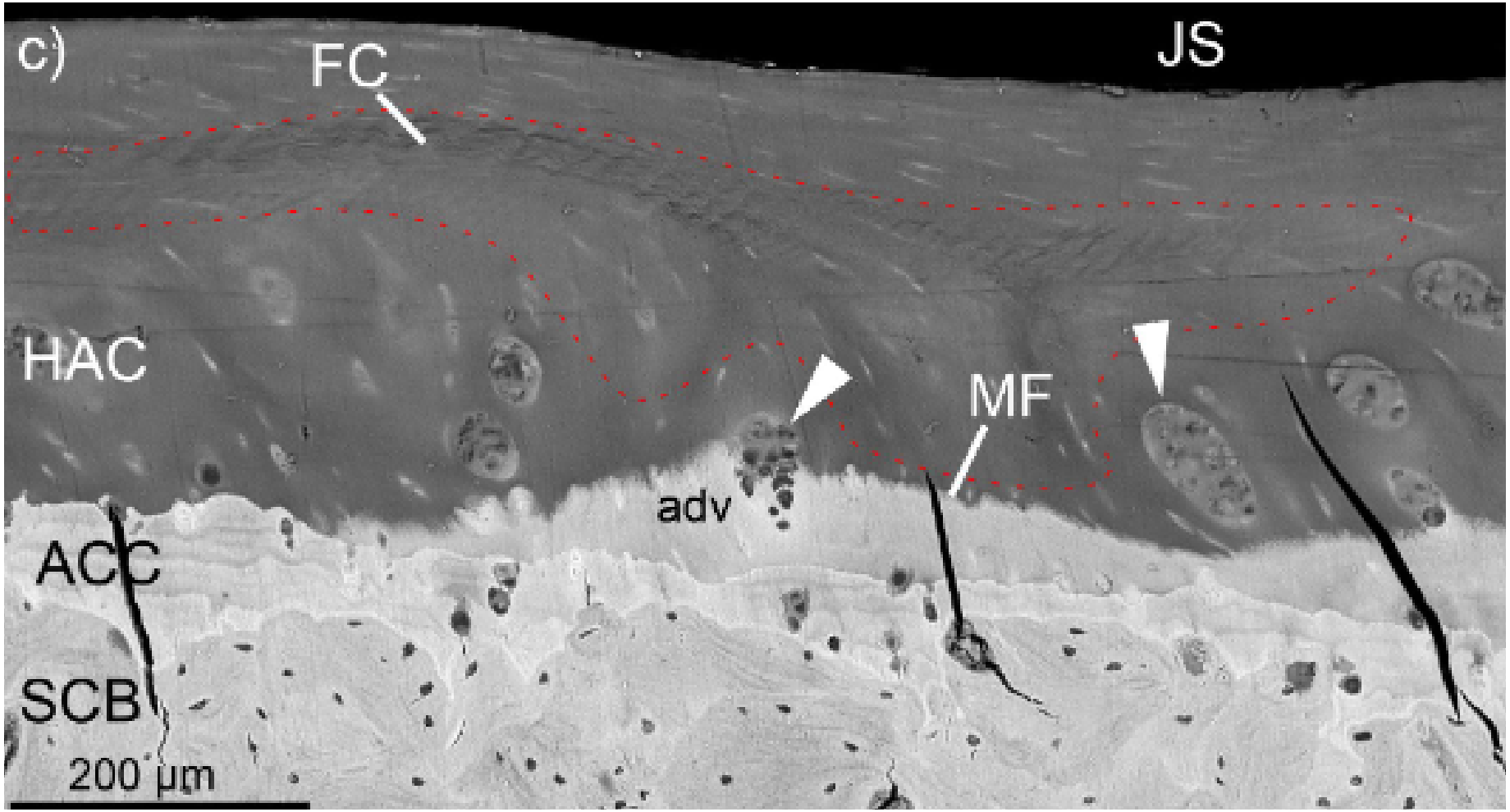
Histology sample sites
24 joints



Lateral

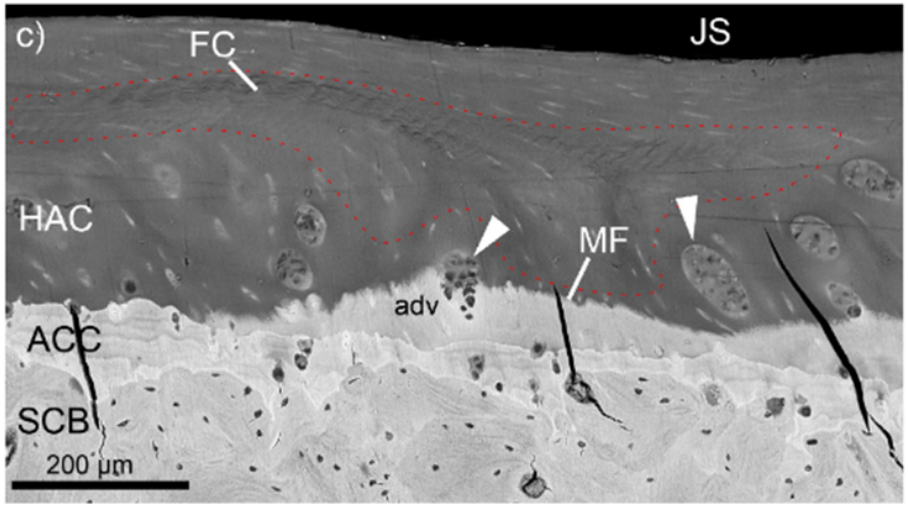
SLU

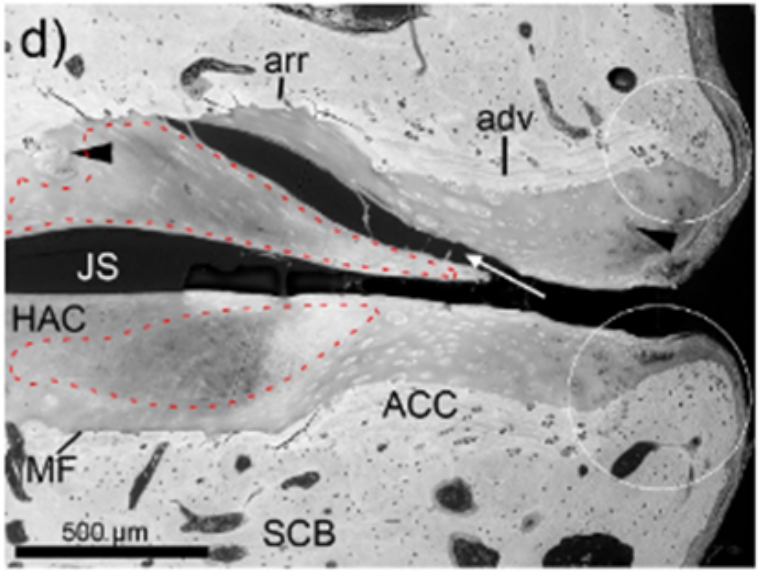
Close associations between HAC and ACC lesions in equine centrodistal joints



Osteochondral lesions

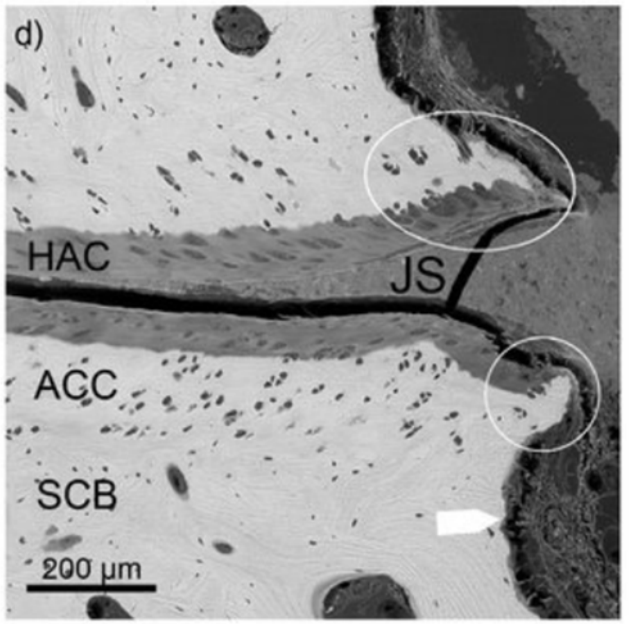
- Significant associations and strong correlations
  - HAC chondrocyte loss
  - HAC fibrillation
  - HAC central chondrocyte clusters
  - ACC arrest
  - ACC advance
- Moderate to high frequency in CD
  - Low frequency in TMT & TC
- The frequency of SCB lesion-types in all joints was low





Joint margin lesion-types

- No significant associations with other lesion-types in the centrodistal joints
- High frequency in both the centrodistal and tarsometatarsal joints



Development of OA in low-motion high-load (compression-loaded) equine joint

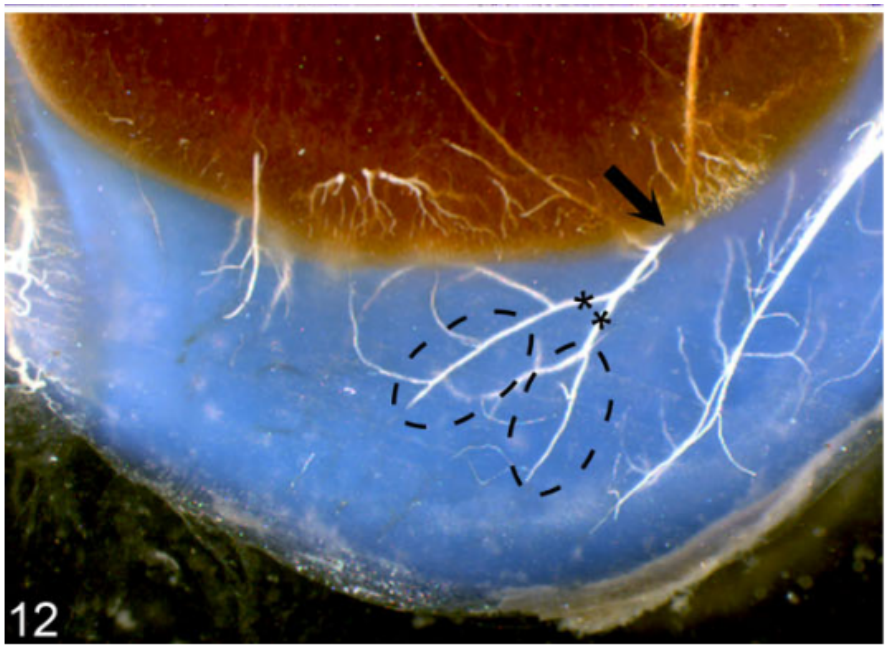
› Eur Cell Mater. 2014 Mar 25:27:213-36; discussion 234-6. doi: 10.22203/ecm.v027a16.

Osteochondral lesions in distal tarsal joints of Icelandic horses reveal strong associations between hyaline and calcified cartilage abnormalities

C J Ley [1], S Ekman, K Hansson, S Björnsdóttir, A Boyde

Osteochondrosis – state of art

- Disturbance in endochondral ossification
- Failure of the temporary, end arterial blood supply
  - -> ischaemic chondronecrosis at intermediate depth of growth cartilage



Ytrehus et al. 2004

Review

An Update on the Pathogenesis of Osteochondrosis

K. Olstad[1], S. Ekman[2], and C. S. Carlson[3]

Veterinary Pathology
2015, Vol. 52(5) 785-802

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Osteochondrosis in cuboidal bones ?

- Has been suggested previously as one of possible pathogenesis of juvenile spavin (Watrous 1991)

- Information needed:
  - Blood supply to growth cartilage of CTB and TIII
    - In foals younger than 6 months old

Cadavers of fetuses and foals <5 months

- Aims:
  - Describe the tarsal development
    - The central (CTB) and third tarsal bones (TIII)
  - Endochondral ossification and the growing cartilage
  - Describe any lesions detected
- Methods:
  - Post-mortem arterial perfusion (barium)
  - Micro CT
- Material:
  - 23 foals/fetuses, where off 12 Icelandic horses
  - Age: from day 228 of gestation to 5 months of age

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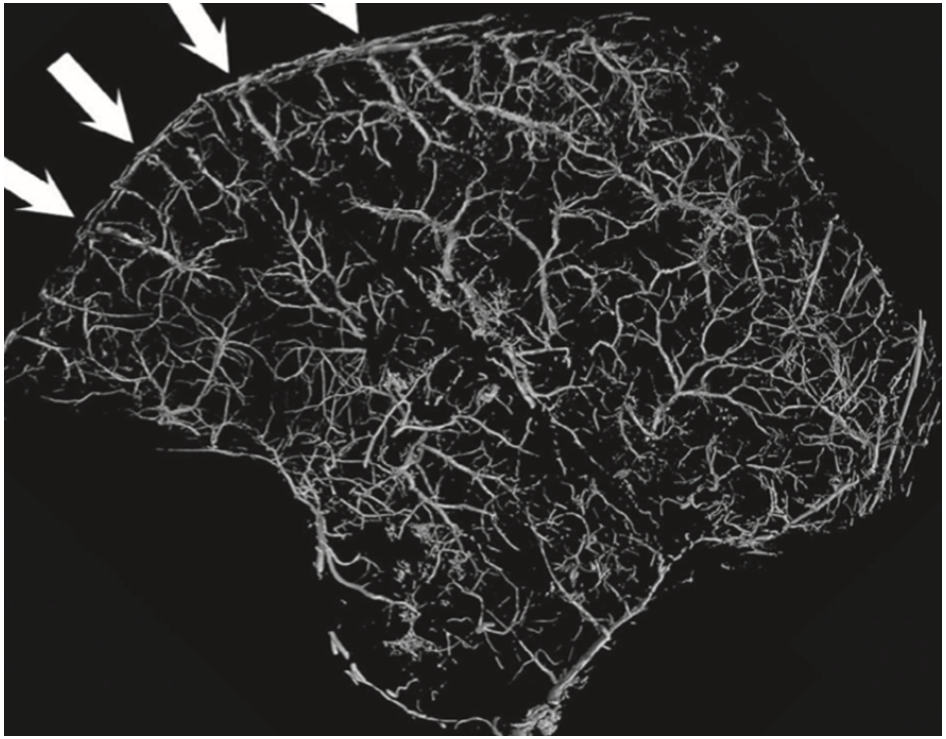
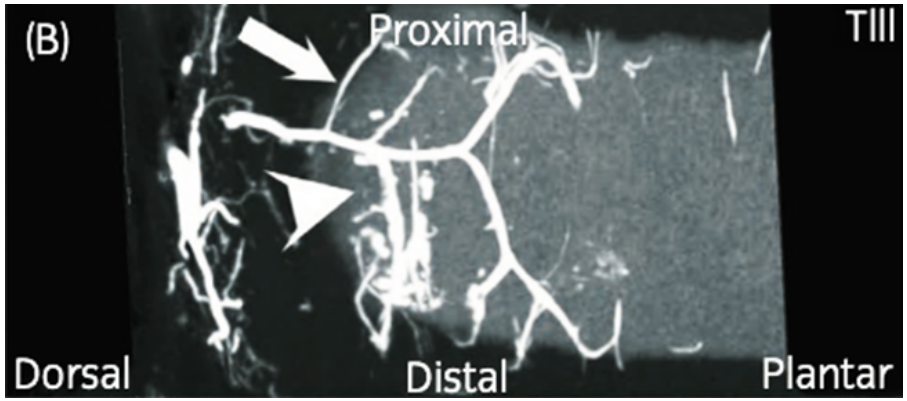
CTB Arterial perfusion with barium (pm)

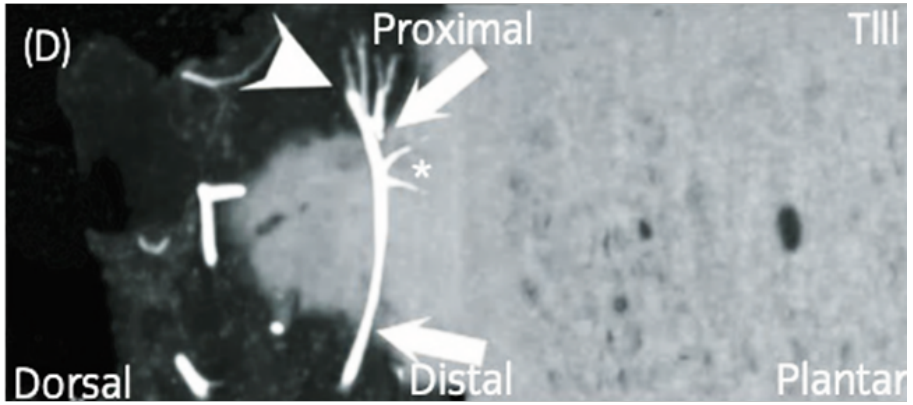


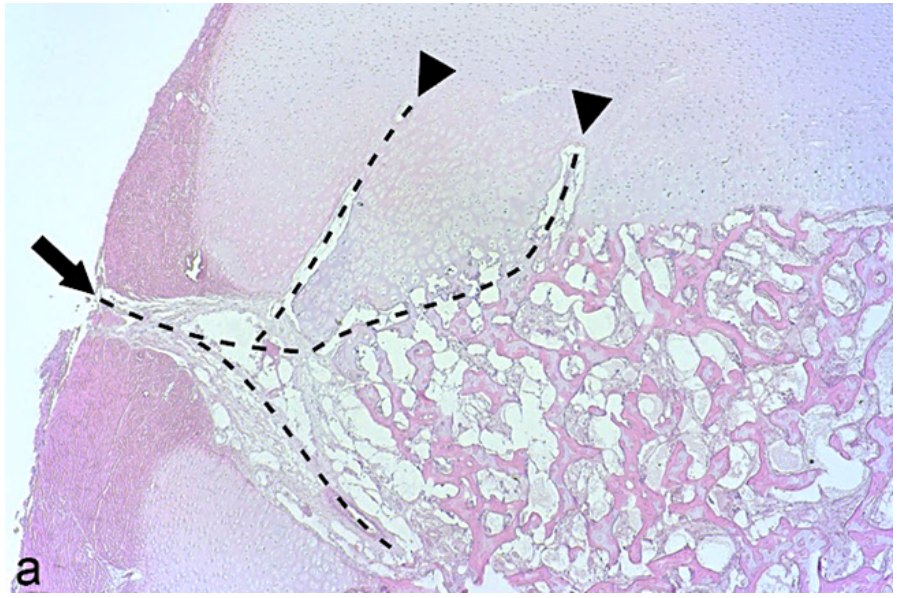
- Central nutrient arteries via ligament fossa
- Peripheral, perichondrial arterioles (white arrows)

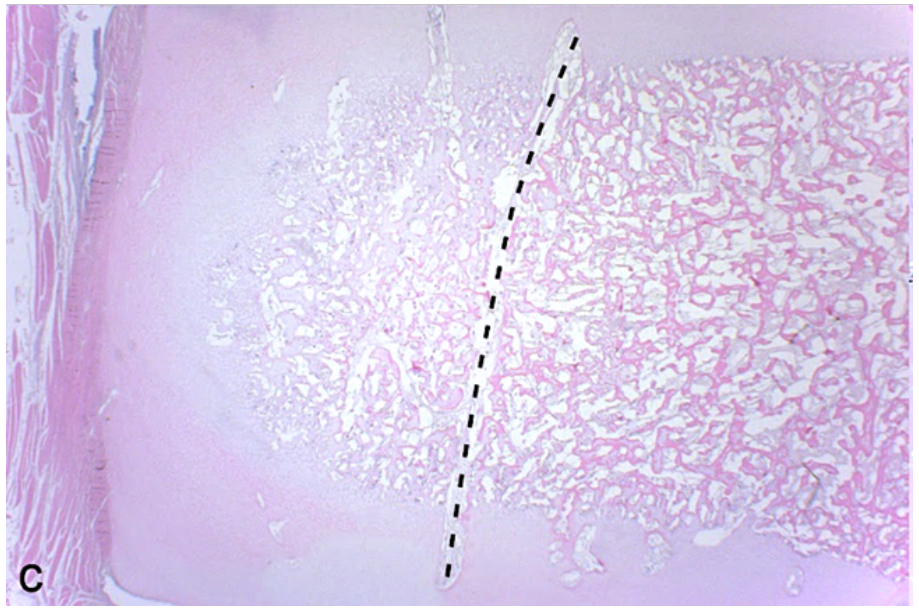
Vertically-turning vessels

Transverse vessels

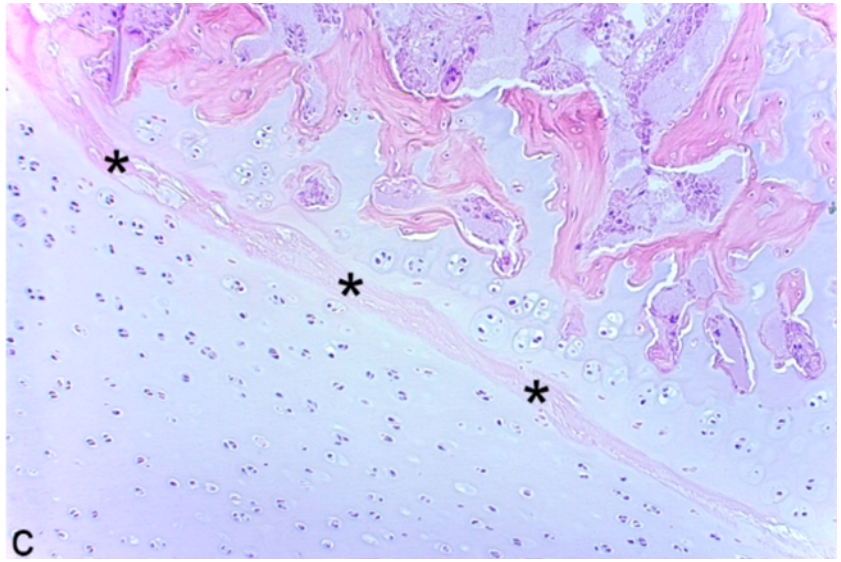


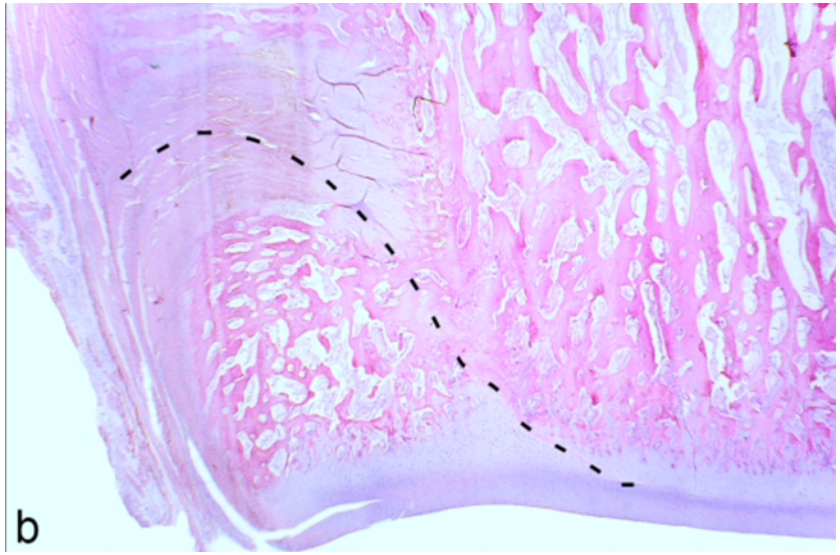




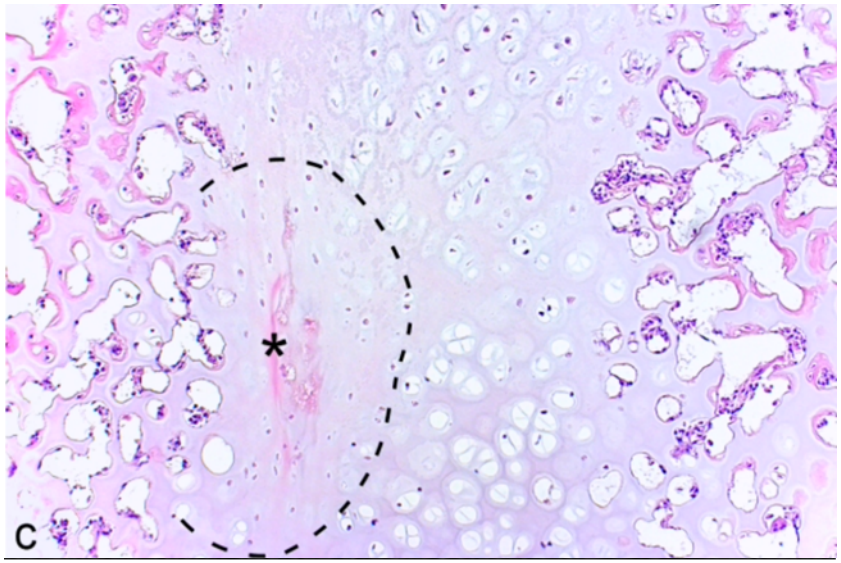


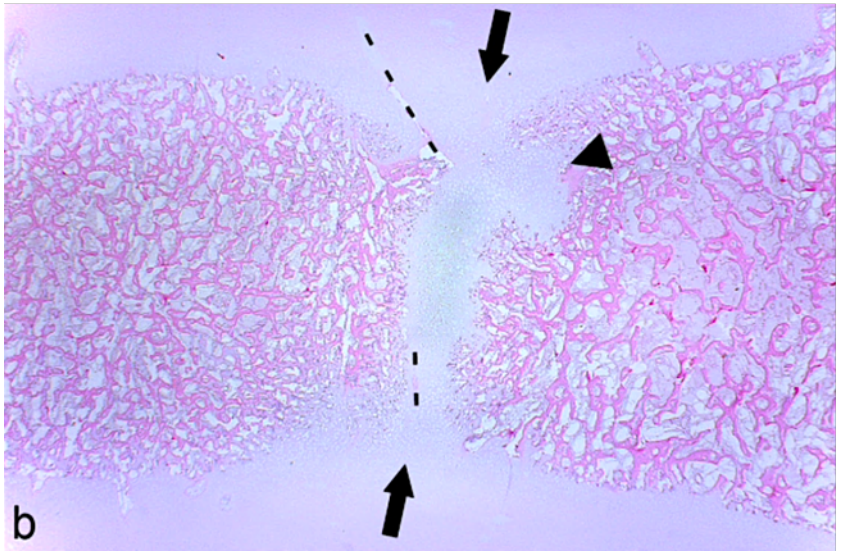
Vertically-turning defects





Transverse defects

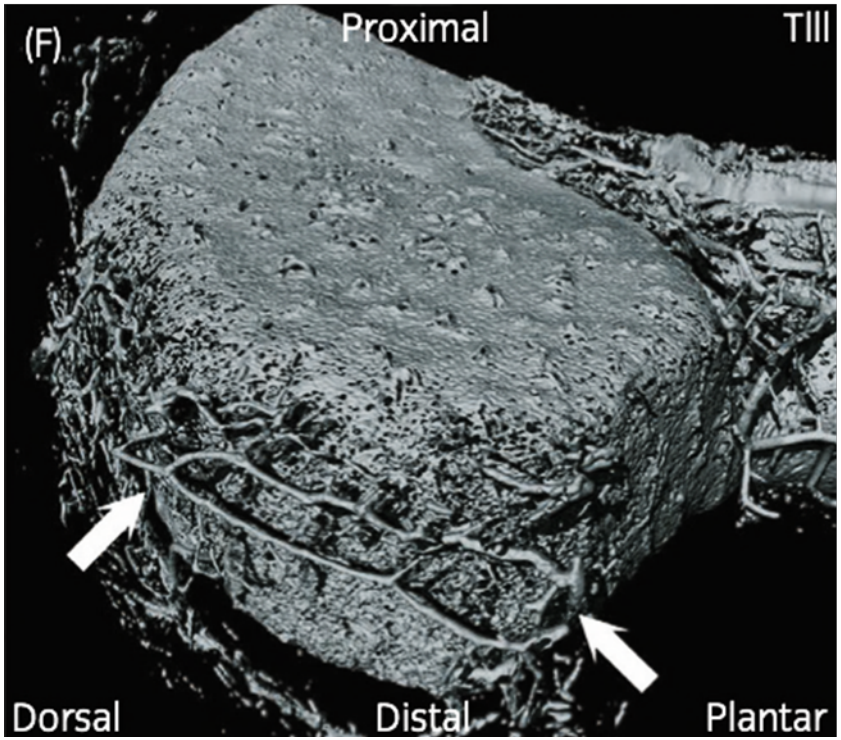




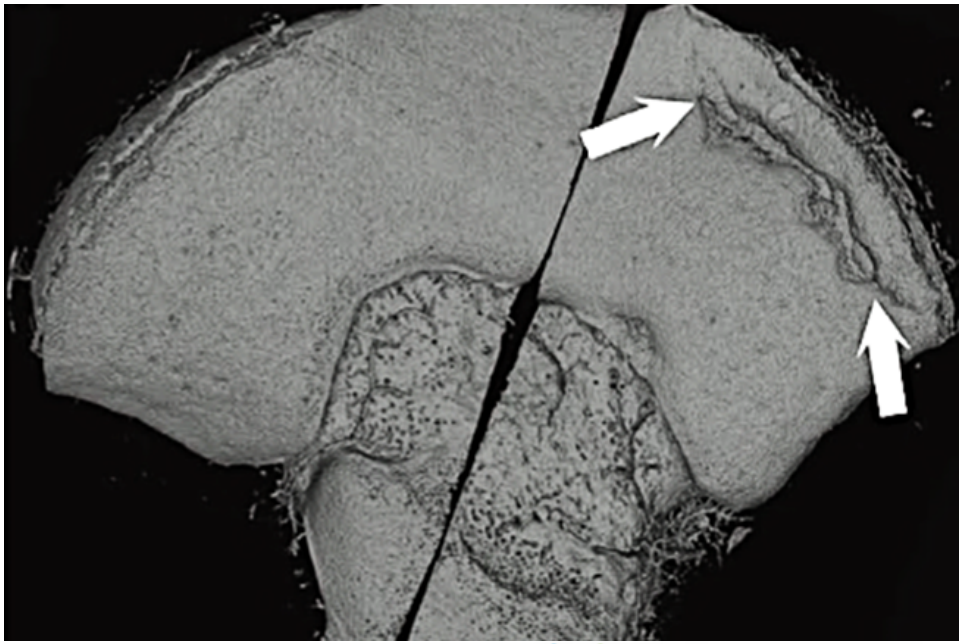
Radiological osteochondrosis lesions
Compatible with vascular failure

- Geometry

Circumferential vessels



Circumferential defects



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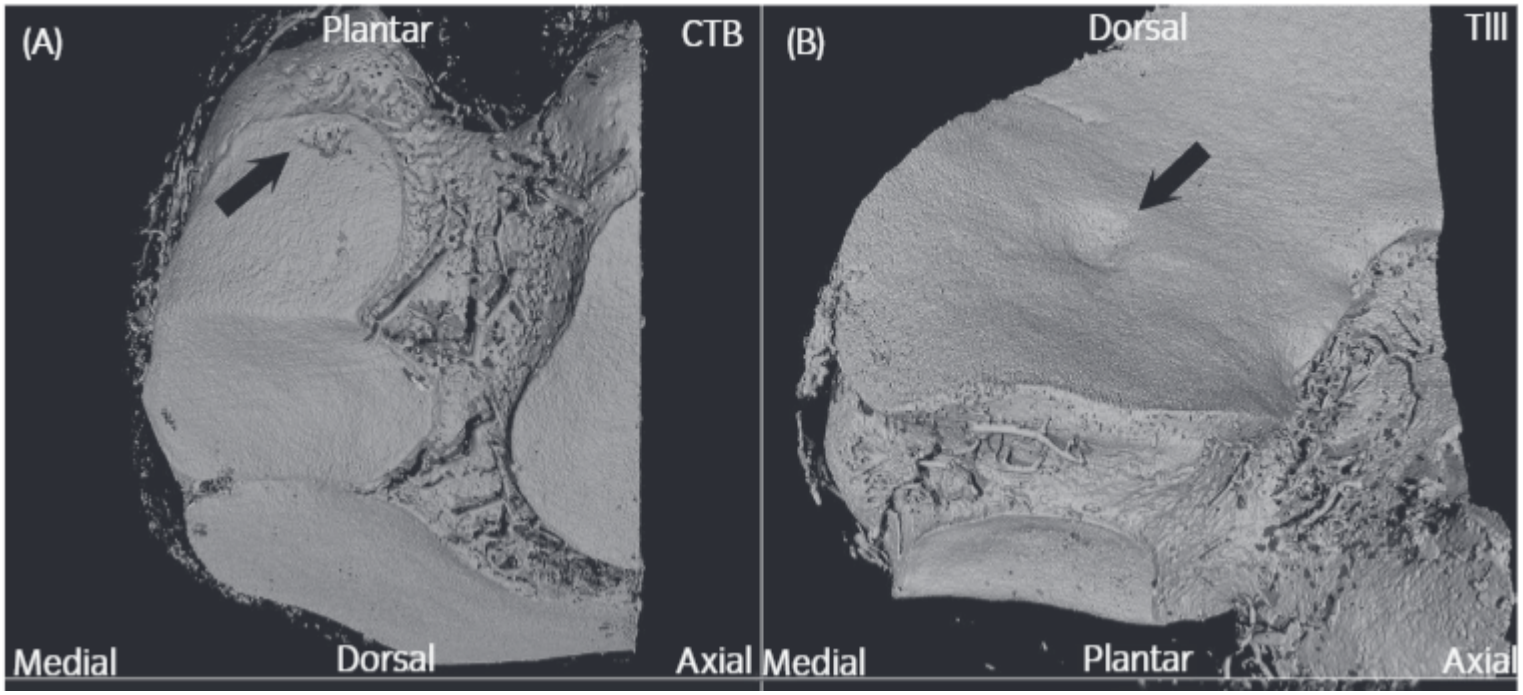


- Focal defects in the ossification front
  - = radiological osteochondrosis.
  - Fourteen of the 23 (61%) animals
    - 75% of the Icelandic foals (9/12)
- The majority of lesions matched the configuration and development of vertical vessels.

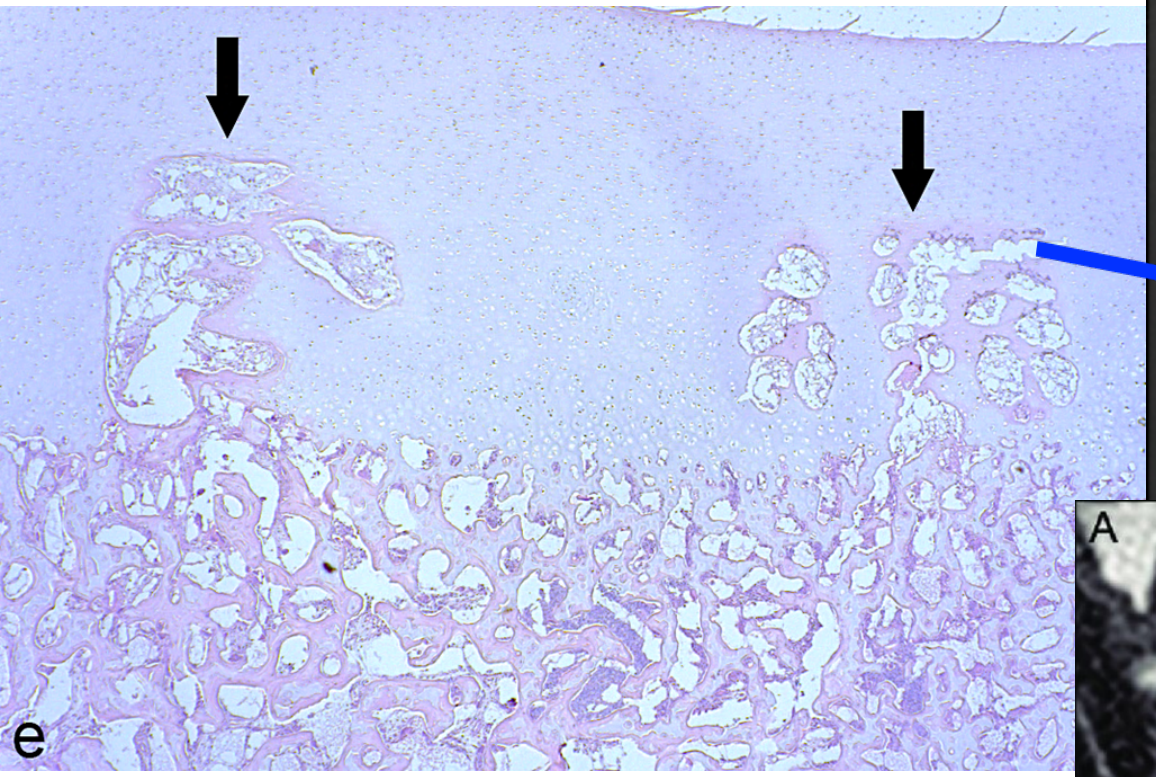
2°responses overlap with osteoarthritis

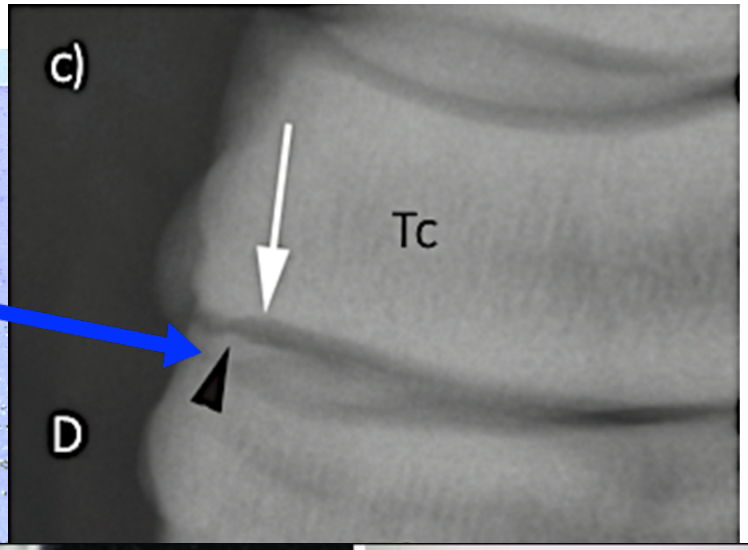
Adjacent vascular prolifertion

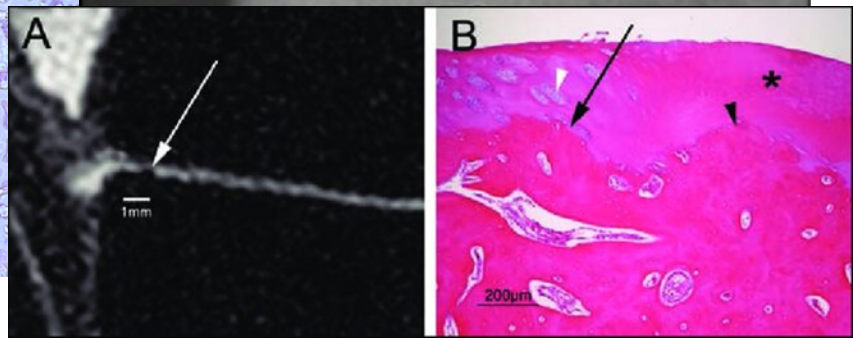
Centres of reparative ossification

Central osteophyte black arrowhead

Early osteoarthritis Ley et al, 2016





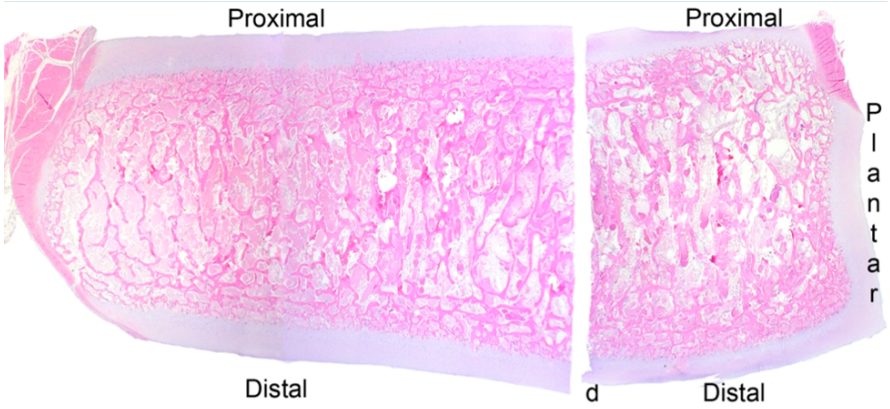


How does OC lead to dtOA

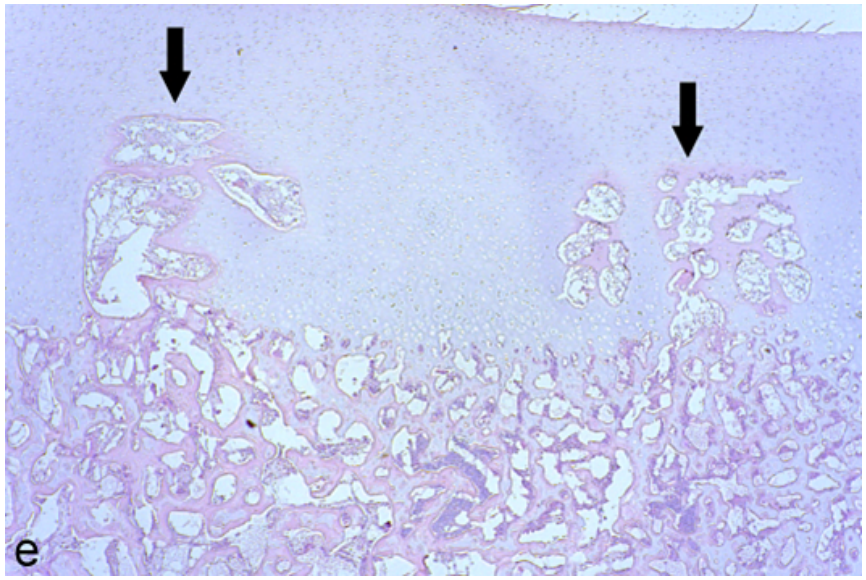
Normal TIII
Straight and smooth

TIII with osteochondrosis and repair

Even load dissipation



Uneven load dissipation



Peak forces + Superficial necrosis = early OA

Björnsdóttir et al. 2004

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Osteochondrosis in the CTB and TIII of young horses

- CTB and TIII grew by both
  - endochondral ossification and
  - intramembranous ossification
- The blood supply to the growth cartilage
  - regressed between 122 and 150 days
    - 4-5 mnd
- Radiological osteochondrosis defects represented
  - Vascular failure
    - Chondrocyte necrosis and retention

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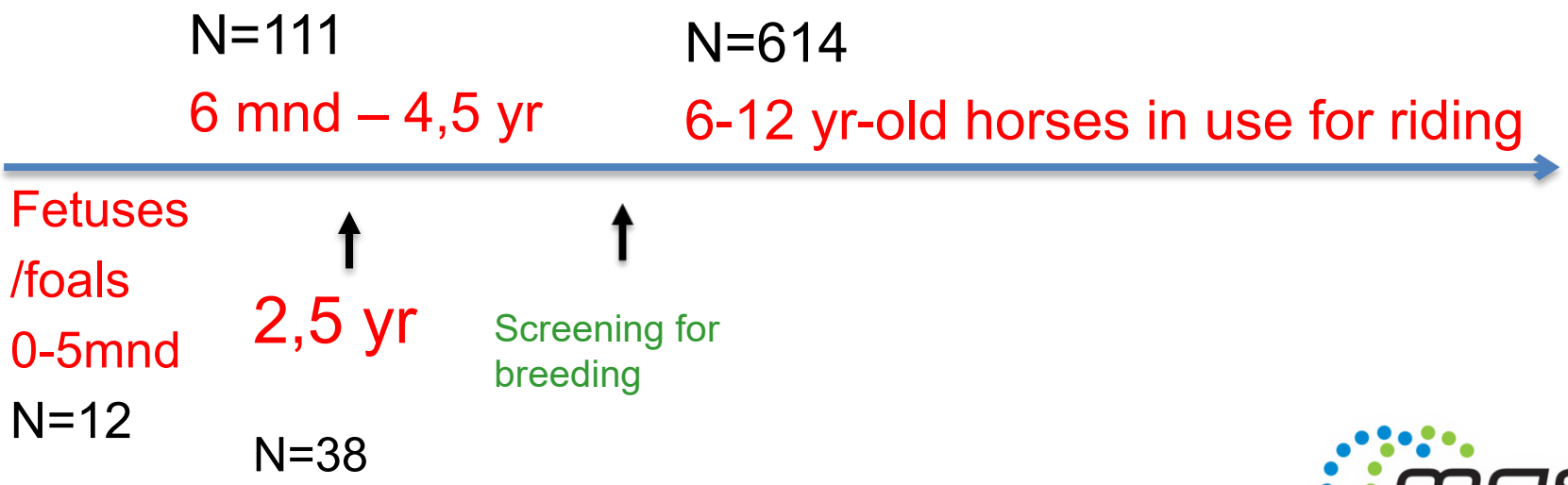
Conclusions

- Failure of the blood supply to growth cartilage causes OC in the CTB and TIII
  - Uneven cartilage – bone interface
- Hypothesis: OC causes uneven dissipation of load
  - leading to superficial chondronecrosis and dtOA



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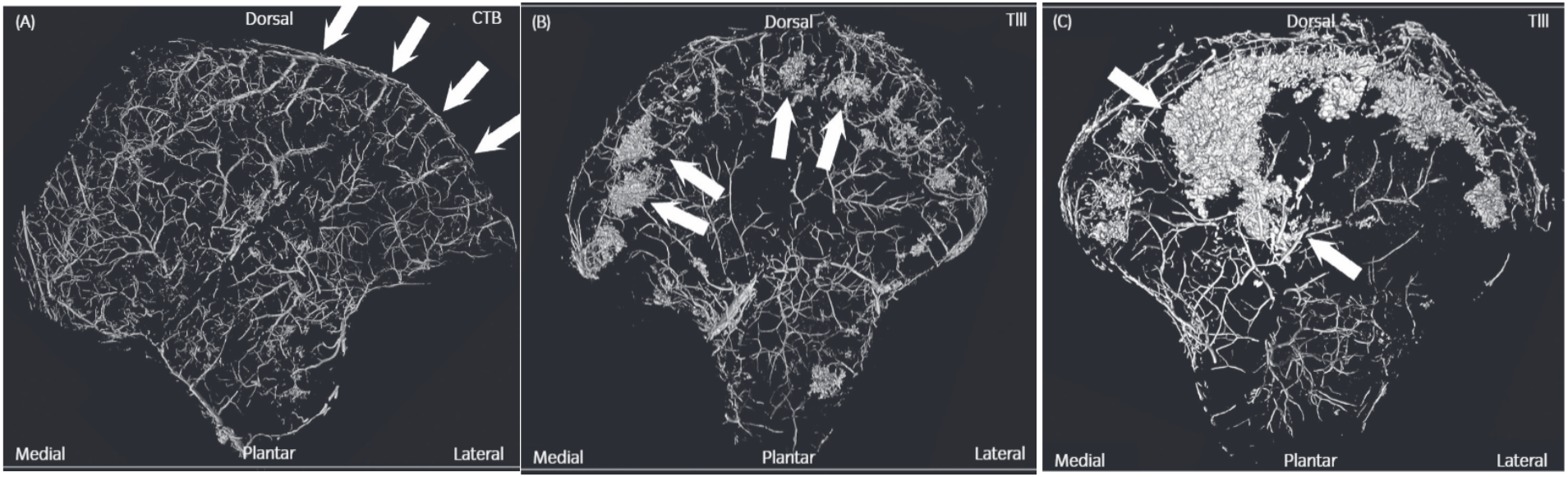
The CTB and TIII were supplied by nutrient arteries and perichondrial vessels with vertical, trans-verse and circumferential configurations.



- A, The perfusion of the central tarsal bone (CTB) of foal 21, judged to be the most complete. Vessels enter the growth cartilage at regular intervals (arrows) around the periphery.
- B, Multiple, small- to- medium foci of intensely dichotomously branching vessels (arrows) referred to as sinusoids are readily appreciable within the otherwise evenly perfused third tarsal bone (TIII) of foal 17.
- C, An untidy and irregular, extra- large sinusoid (between arrows) that includes large, coalescing barium spheres is visible in the TIII of foal 6.

GENERAL ARTICLE

Equine Veterinary Journal WILEY

Radiological, vascular osteochondrosis occurs in the distal tarsus, and may cause osteoarthritis

Sigurdur F. Sigurdsson[1] | Kristin Olstad[1] | Charles J. Ley[2] | Sigríður Björnsdóttir[3] | David J. Griffiths[4] | Cathrine T. Fjordbakk[1]

[1]Faculty of Veterinary Medicine, Department of Companion Animal Clinical Sciences, Equine Section, Norwegian University of Life Sciences, Oslo, Norway

[2]Department of Clinical Sciences, Swedish University of Agricultural Sciences, Uppsala, Sweden

[3]Agricultural University of Iceland, Hvanneyri, Iceland

[4]Faculty of Veterinary Medicine, Department of Basic Sciences and Aquatic Medicine, Anatomy Section, Norwegian University of Life Sciences, Oslo, Norway

Abstract

Background: Osteochondrosis occurs due to failure of the blood supply to growth cartilage. Osteochondrosis lesions have been identified in small tarsal bones and suggested to cause distal tarsal osteoarthritis; however, it has not been determined whether distal tarsal osteochondrosis lesions were the result of vascular failure.

Objectives: To perform post-mortem arterial perfusion and micro-computed tomography (CT) of the central (CTB) and third tarsal bones (TIII) of fetuses and foals up to 5 months old, to describe tarsal development and any lesions detected.

Study design: Descriptive, nonconsecutive case series.

Original Article

Osteochondrosis in the central and third tarsal bones of young horses

Veterinary Pathology
1–14

DOI: 10.1177/03009858231185108
journals.sagepub.com/home/vet
Sage

Kristin Olstad[1], Stina Ekman[2], Sigríður Björnsdóttir[3], Cathrine T. Fjordbakk[1], Kerstin Hansson[2], Sigurdur F. Sigurdsson[1], and Charles J. Ley[2]

Abstract

Recently, the central and third tarsal bones of 23 equine fetuses and foals were examined using micro-computed tomography. Radiological changes, including incomplete ossification and focal ossification defects interpreted as osteochondrosis, were detected in 16 of 23 cases. The geometry of the osteochondrosis defects suggested they were the result of vascular failure, but this requires histological confirmation. The study aim was to examine central and third tarsal bones from the 16 cases and to describe the tissues present, cartilage canals, and lesions, including suspected osteochondrosis lesions. Cases included 9 males and 7 females from 0 to 150 days of age, comprising 11 Icelandic horses, 2 standardbred horses, 2 warmblood riding horses, and 1 coldblooded trotting horse. Until 4 days of age, all aspects of the bones were covered by growth cartilage, but from 105 days, the dorsal and plantar aspects were covered by fibrous tissue undergoing intramembranous ossification. Cartilage canal vessels gradually decreased but were present in most cases up to 122 days and were absent in the next available case at 150 days. Radiological osteochondrosis defects were confirmed in histological sections from 3 cases and consisted of necrotic vessels surrounded by ischemic chondronecrosis (articular osteochondrosis) and areas of retained, morphologically viable hypertrophic chondrocytes (physeal osteochondrosis). The central and third tarsal bones formed by both endochondral and intramembranous ossification. The blood supply to the growth cartilage of the central and third tarsal bones regressed between 122 and 150 days of age. Radiological osteochondrosis defects represented vascular failure, with chondrocyte necrosis and retention, or a combination of articular and physeal osteochondrosis.

Keywords

Not training (work load) related disease

Developmental disease

- Chondronecroses before the age of 4 months
  - The temporary, end arterial blood supply of growth cartilage is fragile
- Extensive lesions (full deapth) more likely to develop to dtOA
- Milder ones will heal

Slow progression of OA

- Most often detected by radiology in grown up horses
  - From the age of 5
  - Lamenss probably later

Inherited disease

- Genetic progress faster than expected

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Osteochondrosis uncommon in Icelandic horses in contrary to dtOA

Thinner growing cartilage in Icelandic horses

- Explanes low prevalence of OC/OCD in general

High load/low motion

- Lack of dynamic load might reduce the possibility of oxygen diffusion
- Biomechanics might affect the repairing process

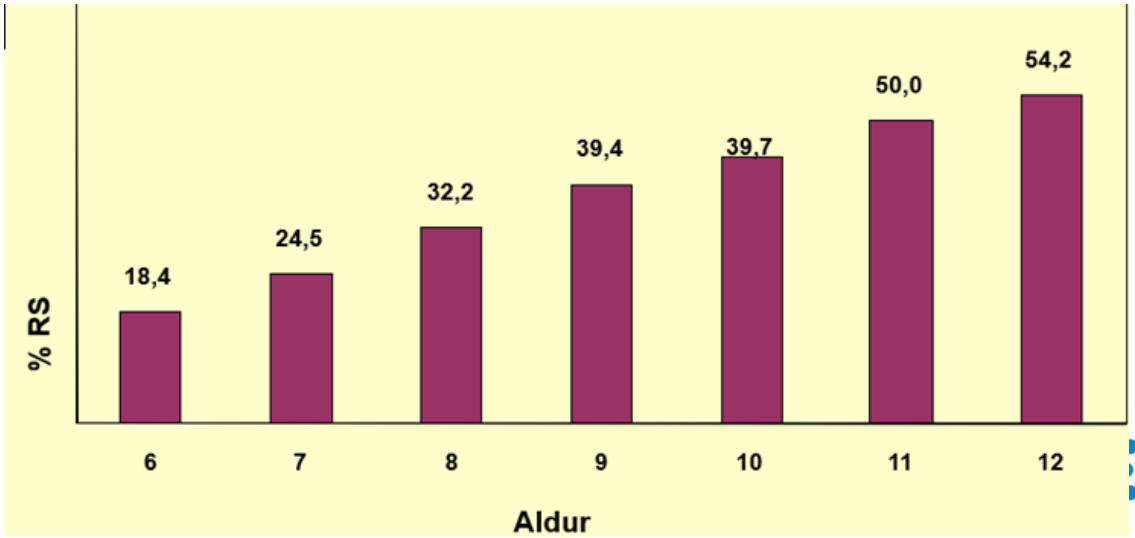
dtOA is an inherited disease

- Inherited weakness/defect in the temporary, end arterial blood supply of growth cartilage in the distal tarsal joints?
- Genetic progress faster than expected
- Suggests that the heritability was underestimated

Present prevalence of dtOA in Icelandic horses?

- In use for riding - age range 6-12 years

Pre-purchase examinations 2024								
	6 yr	7 yr	8 yr	9 yr	10 yr	11 yr	12 yr	Total
Total	91	86	58	44	26	20	15	340
No remarks	87	83	55	37	22	17	12	313
dtOA	4	3	3	7	4	3	3	27
	4%	3%	5%	16%	15%	15%	20%	8,00%



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We never see this anymore

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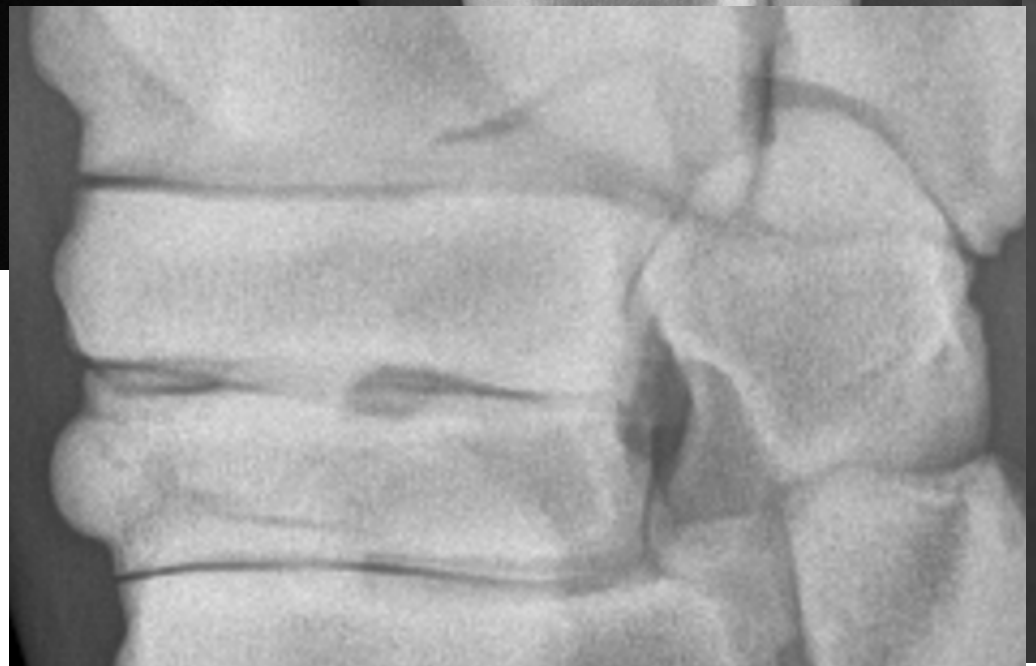

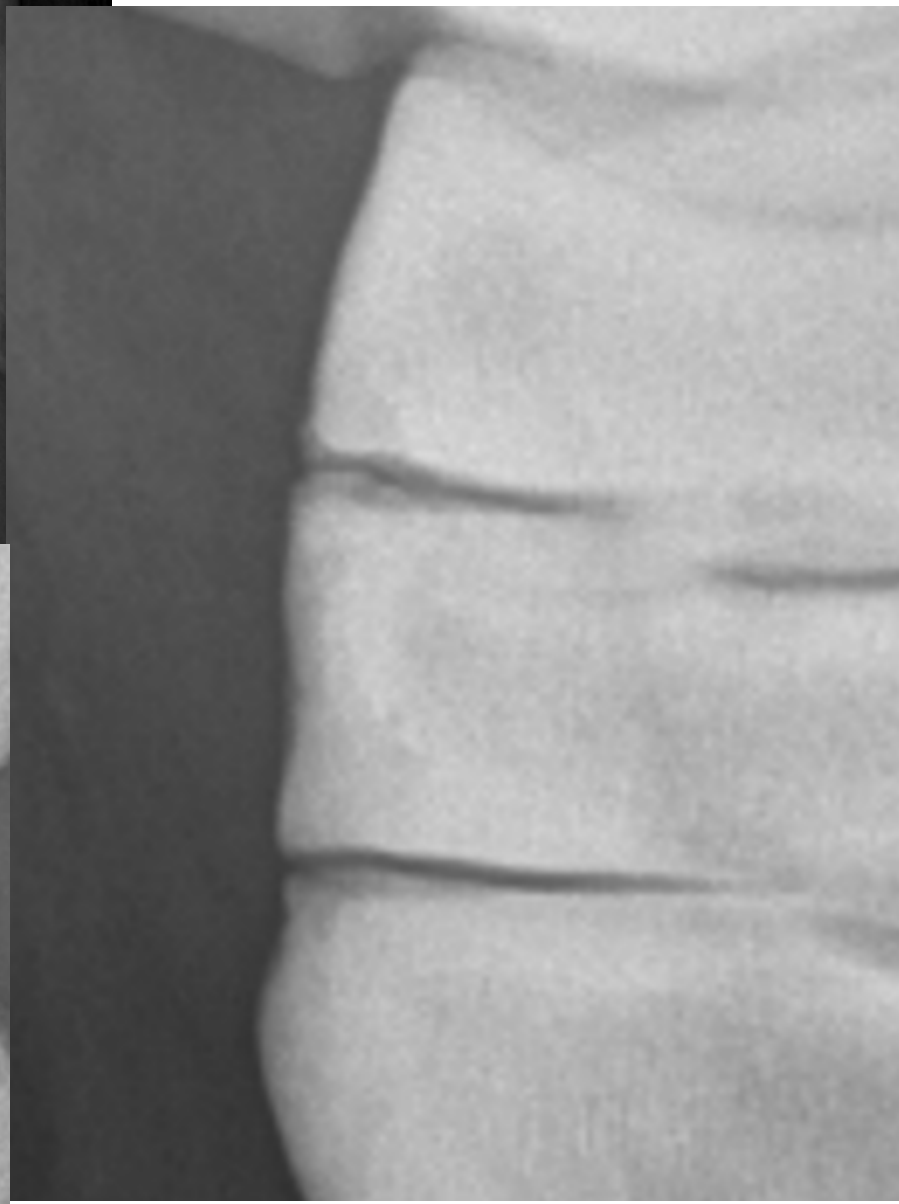

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Queen Mary University of London

Alan Boyde