Osteochondrosis in the Horse: Review and Research Update

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Terminology and History

- **Osteochondrosis (OC)**
- Osteochondritis vs **osteochondrosis dessicans (OCD)**
  - Not primary inflammatory etiology
- In humans, different disease location = different disease name
  - Theimann's disease (fingers/toes)
  - Panner's disease (elbow)
  - Freiberg's disease (metatarsophalangeal joint)
  - OCD (elbow/knee/ankle)
- Juvenile osteochondral condition (JOCC)

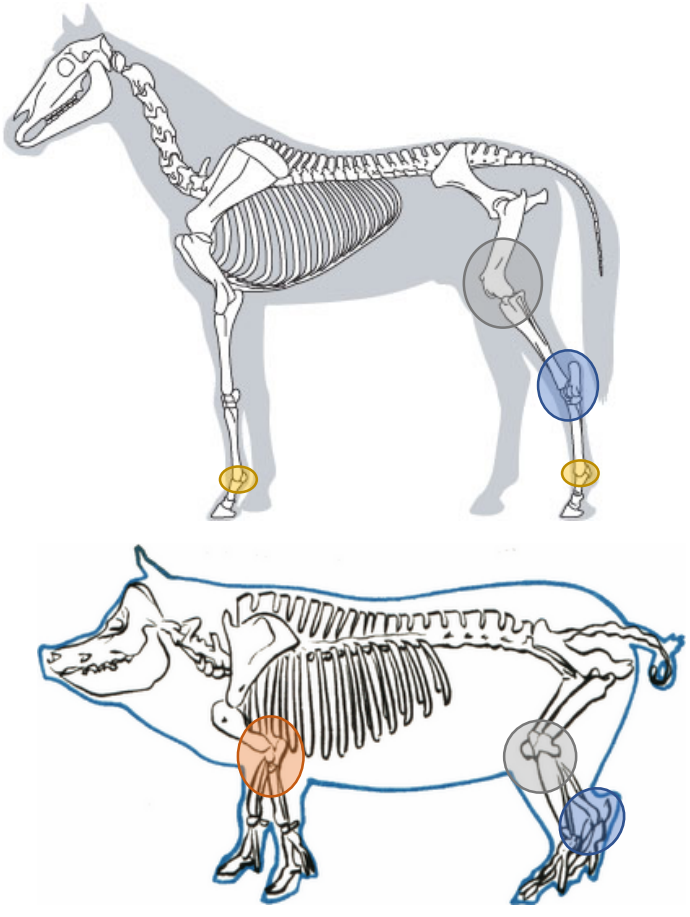
Franz König 1887

Osteochondrosis is a **developmental orthopedic disease (DOD)**

- Also included under this umbrella term: physitis, flexural limb deformity, incomplete ossification of cuboidal bones, angular limb deformity, subchondral bone cysts
- Most simply defined as a failure of normal endochondral ossification
- Affects individuals across species: horse, pig, dog, chicken, cow, human

Predilection sites

- Known predilection sites in the horse include fetlock, hock, stifle; less commonly neck, shoulder, hip
- Predilection sites are shared across species: stifle, elbow, tarsocrural, metacarpal(tarsal)phalangeal



- Predilection sites within joints (in order of frequency)
    - Hock: Distal intermediate ridge of the tibia (DIRT), lateral trochlear ridge of the talus (LTR), medial malleolus of the tibia (MM), medial trochlear ridge of the talus (MTR)
    - Fetlock: Dorsal distal mid-sagittal ridge MC/MTIII, dorsal margin P1
        - Controversial: palmar/plantar margin P1
    - Stifle: Lateral trochlear ridge of the femur (LTR), medial trochlear ridge of the femur (MTR), distal patella, intertrochlear groove
- Multiple OC lesions in the same horse are not uncommon
    - Bilateral lesions:
        - Stifle: 17.5-20.5% of affected animals
        - Hock: 6-10% of affected animals (probably underreported)
        - Fetlock: can be bilateral or all 4 affected
    - Multiple lesions within the same joint
        - Especially seen in the hock (i.e. DIRT + LTR)
    - Less common to have multiple different joints affected (i.e. hock + stifle)

Prevalence

- From **7% to >80%** prevalence of OC lesions in any location based on radiographic surveys of yearling horses of various breeds
- Prevalence varies by site and breed
    - E.g. average reported prevalence of hock lesions in Standardbreds 14.7%, compared to 5.3% in Thoroughbreds

Proposed pathogenesis (how does it happen?)

- Trauma
    - Key precipitating factor for clinical signs; cannot account for histologic changes or non-weightbearing predilection sites
- Inflammation
    - Not supported by histologic studies
- Osteonecrosis
    - Likely secondary to detachment of fragment
- Cartilage extracellular matrix abnormalities
    - Likely secondary to chondronecrosis
- **Vascular abnormalities**
    - Supported by work in animal models

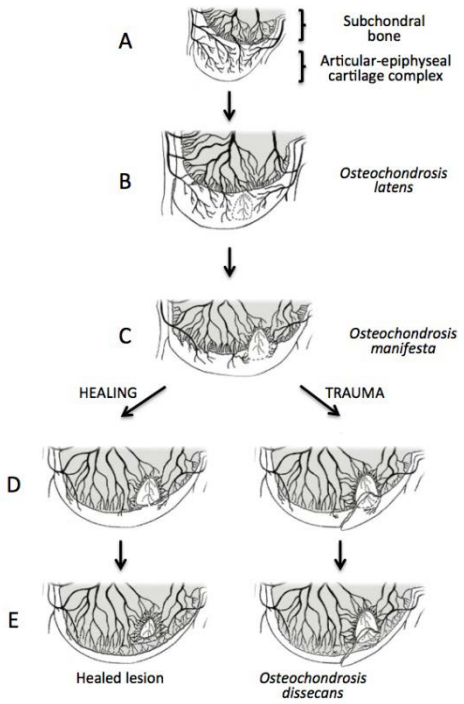


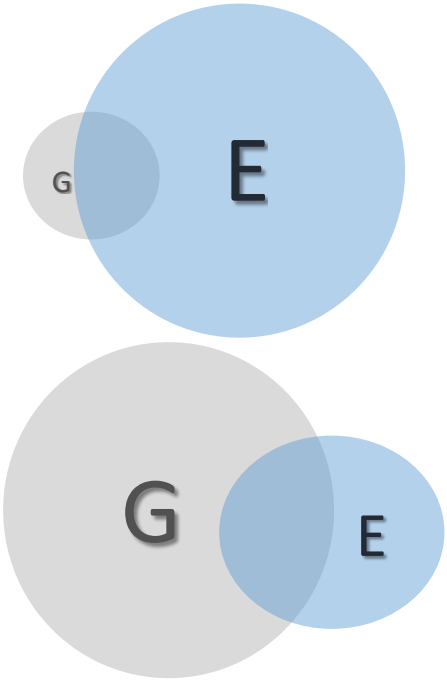
Fig 5, McCoy et al., *Osteoarthritis Cart* 21:1638-47, 2013

Suggested risk factors for OC (why does it happen?)

- **Genetics**
- **Nutrition**
- **Exercise**
- Conformation
- Other biomechanical forces
- Trauma
- Stress response
- *In utero* environment
- Hormonal interactions

OC is a **complex** disease: influenced by the interaction between genes and the environment

- We can think about this in a couple of different ways. For example, on a farm with moderate disease prevalence, may have one of 2 situations (shown a bit "extreme" here):
  1. Low genetic risk with lots of environmental risk factors (diet, etc.) - will see improvement in farm prevalence with alterations in management factors alone; this is what has been addressed through 25 years of previous research.
  2. High genetic risk with low to moderate environmental risk factors – if we can identify early the horses that fall into this category, then we can proactively alter management to reduce this component of risk as much as possible and consider making changes in breeding practices.



Role of genetics

- Certain lines seem to be predisposed
  - Up to 70% of foals from a single sire affected
- Offspring of affected sires twice as likely to be affected as offspring of unaffected sires
- Heritability estimates (tarsal OC) suggest that **up to 50% of disease risk is inherited**
  - Standardbred: 0.24-0.52
  - Warmblood: 0.25-0.41
- Many regions of the genome reportedly associated with OC – area of ongoing research

Role of nutrition

- High dietary energy/"overfeeding"
  - Feeding for rapid growth has been anecdotally associated with increased risk of OC for decades
  - In foals experimentally fed 100% or 130-150% recommended amounts of balanced ration:

        - Higher feed resulted in higher ADG
        - Rapid growth was positively correlated with cervical, but not limb, OC lesions
        - Limb OC lesions were correlated more with height at withers than body weight, regardless of feeding group
    - In foals experimentally fed 129% dietary energy:
        - No difference in body weight or height between groups
        - High DE group had significantly more OC lesions
    - In large surveys of foals being fed various diets at their home farms:
        - No statistical association between level of energy and nutrient intake and incidence of OC
        - No difference globally in body weight/withers height between OC-affected and OC-unaffected, but OC of stifle/hock associated with higher body weight after weaning
        - Larger girth associated with increased severity of lesions, especially in foals with limited/irregular exercise
- Protein
    - Excess dietary protein has been suggested to increase risk of OC, but little evidence
    - In a small number of foals experimentally fed 126% of recommended crude protein, incidence of OC lesions was not different from controls
- Minerals/micronutrients
    - **Copper** is important for normal cross-linking of collagen and deficiency has been linked to defective cartilage formation
        - 7 of 8 TB foals with severe, multifocal OC lesions had low serum Cu levels, 3 also had high serum and liver Zn levels
        - In foals experimentally fed low Cu diets, cartilage abnormalities of varying severity (including OC in multiple joints) developed over 6 months
    - **Calcium** and **phosphorus** levels play an important role in skeletal development
        - Ratio important (i.e. 1.3:1)
        - In foals experimentally fed 388% of recommended P or 342% Ca:
            - Foals on high Ca diet did not develop more OC lesions than control
            - High Ca did not rescue formation of OC lesions associated with high dietary energy ration
            - Foals on high P diet did develop more OC lesions than high Ca or control despite lower feed intake
- On 19 farms in OH and KY:
    - [Ca], [P], [Cu], and [Zn] were higher in the diets of yearlings with lower incidence of DOD, but were not deficient in any diet
    - After adjustment of rations on 17 farms:
    - Average DOD score in yearlings improved across farms; largest improvements seen in farms with the worst problem before ration changes
    - Only feed intake (↓) and copper intake (↑) changed significantly overall
- Overall conclusion: **nutrition can play an important role in disease development, but it interacts with genetics and other factors; "windows of susceptibility" may be important**

Role of exercise

- In OC-predisposed foals:
  - Number of lesions at 5 and 11mo not different between stall-rested and forced- or pasture-exercised
  - Trend towards greater severity of lesions in stall-rested foals
- In a large random survey of foals:
  - Irregular exercise was associated with a higher risk of severe OC when compared to regular moderate exercise (e.g. daily turnout)
- Overall conclusion: **possible modifying role in disease**

Typical signalment

- Common scenarios for diagnosis of OC
  - Yearling racehorse going to/at sale
  - Young sport horse entering training (2-4yr)
  - Any horse undergoing a comprehensive prepurchase exam that includes radiographs
- Common presenting complaints
  - Joint effusion
    - Most commonly the only presenting complaint in yearlings
  - Lameness
    - Generally mild, but may be severe depending on location and size of lesion
    - Usually accompanied by effusion
    - More common in horses that have just been put into work
  - ***No clinical signs***

Diagnosis of OC

- Clinical Signs
  - High index of suspicion with appropriate signalment
  - Unreliable
- Radiographs/fluoroscopy
  - Most common
  - False negatives possible
- Ultrasound
  - May be more sensitive than radiographs for certain lesions
- Arthroscopy
  - Gold standard

Radiographic diagnosis

- Standard radiographic views are often sufficient for diagnosis
  - Helpful to see fragment(s) on >1 view
- Special views
  - 30 degree DLPMO better to image medial malleolus lesions (hock) than standard 45 degree
  - Flexed and/or oblique lateral view in stifle may be useful; skyline view for patella in certain cases
- **Take bilateral films even if unilateral effusion**
- When should you radiograph?
  - If concerned about OC because of genetics, clinical signs, etc. not a bad idea to screen early (i.e. 6 months of age)

- BUT, OC lesions can heal spontaneously
- Radiographic studies suggest lesions become permanent after:
  - 5 months of age in the fetlock and hock
  - 8 months of age in the stifle
- "Windows of susceptibility" related to timing of epiphyseal cartilage ossification
- **Any healing that will occur typically complete by 11 months of age**

Ultrasound

- U/S found to be more sensitive than rads for diagnosis of MTR lesions in the stifle and MM and DIRT lesions in the hock
- Extremely useful for diagnosing OC in cervical vertebrae, shoulder, hip

When evaluating diagnostic imaging findings and determining treatment approach, ask:

- OC vs traumatic fragment?
- Clinical significance of lesion(s)?
  - Location?
  - Severity?
  - How long has it been there?
  - Age of horse?
  - Clinical signs?
- Intended use/owner expectations for horse?

Conservative treatment

- Rest
- Diet modification (i.e. reduce dietary energy)
- Systemic NSAIDs
- Intraarticular (or systemic) HA, PSGAGs
- Oral joint supplements (i.e. chondroitin sulfate)
- **Most likely to be successful in mild disease with minimal effusion and no lameness and/or disease in very young horses**

Surgical treatment

- Arthroscopic removal of fragments, curettage of fragment bed/abnormal tissue
  - Generally recommended if a fragment/flap is present
- Reattachment of selected large osteochondral fragments with PDS pins has been reported
- Allows for complete evaluation of the joint
- May recommend bilateral arthroscopy even if radiographic evidence for disease is equivocal in the 2nd joint
- Frequently chosen for horses being prepped for sale even if no clinical signs
  - **Presence of an OC lesion can decrease sale price**
- For resolution of effusion, early intervention is preferred
  - **Chronic effusion may not resolve even with surgical correction of lesion**
  - Important for many show horses

Treatment outcomes

- Conservative treatment
  - **Mixed reports** – outcome likely depends on severity and location of lesions
  - Less likely to do well:
    - Stifle lesions (compared to hock/fetlock)

        - Large lesions
        - Lesions with osteochondral fragmentation
    - In two different studies, racehorses performed poorly with conservative treatment compared to non-racehorses
        - Good outcomes in 37.5% of racing horses (all breeds) compared to 50% of pleasure horses and 60% of show horses
        - Good outcomes in 23% of Standardbreds compared to 80% of Warmbloods
    - Race performance has been reported equivalent in conservatively-treated Standardbreds compared to unaffected individuals
        - BUT horses with more severe lesions tended to be tx surgically
- Surgical treatment
    - **In general, excellent outcomes reported**
    - Hock:
        - 83.1% of non-racehorses, 72.6% of racehorses returned to intended use
        - 76% of racehorses started at least once after sx
    - Stifle:
        - 67% of non-racehorses, 62% of racehorses with successful outcomes
    - Larger lesions or joints with other pathology less likely to do well even with sx intervention
    - Affected racehorses reportedly less likely to race as 2-year-olds than unaffected siblings
        - May be due to training days lost with sx
    - Standardbred racehorses undergoing surgical intervention prior to yearling sales performed equivalently to related unaffected horses, both as 2-year-olds and over several race seasons
    - Resolution of effusion and lameness:
        - 79 horses with arthroscopically-removed OC lesions of the hock:
            - 25/79 horses lame prior to sx; 3/79 lame after sx
            - 38/112 limbs flexion positive prior to sx; 11/112 flexion positive after surgery
            - 15/113 joints had no effusion prior to sx; 41/113 had no effusion after sx
        - Effusion scores significantly decreased in horses with continued effusion
        - Resolution of lameness/flexion within 5 wks; effusion can continue to improve over a longer period

Published general treatment guidelines (McIlwraith CW. *Vet J* 197:19-28, 2013)

- Conservative
    - Stifle: if no fragmentation or if *in situ* fragment is <2cm long and <5mm deep
    - Fetlock: sagittal ridge flattening w/o fragments
    - Shoulder: lesions restricted to glenoid
- Surgical
    - Stifle: fragments >2cm long and/or >5mm deep
    - Hock: any lesion if clinical signs present
    - Fetlock: any lesion with fragmentation
    - Shoulder: any lesion not localized to glenoid

Should you breed horses with OC?

- Very contentious question – subject of much debate. Some points to ponder:
  1. OC does have a strong genetic component
     - BUT, difficult to make breeding decisions when specific major risk genes are unknown
  2. OC does have major environmental risk factors
     - BUT, optimizing management does not eliminate all disease
  3. Horses with OC can go on to perform at elite levels if treated
     - BUT, severely affected or untreated horses can have career- or life-limiting consequences to disease

Research in OC is ongoing

- Pathogenesis: experimentally-induced models of disease
  - Cartilage canal transection in ponies – recapitulates lateral trochlear ridge lesions in the stifle
- Diagnosis: MRI
  - MRI of developing joints in foals demonstrates differences in cartilage thickness at predilection sites
  - Irregularities of ossification can be visualized – this may be a new tool for early diagnosis
- Nutrition: metabolomics
  - Metabolomics is the study of the metabolic profile of a biological system
  - Differences in metabolite levels between OC-affected and unaffected individuals may identify key nutrients that play a role in disease development
- Genetics: identifying genes underlying disease risk
  - Identification of specific genes/variants underlying disease risk is the first step towards developing a genetic risk model that can be applied to individual horses to quantify risk; this will 1) help to identify at-risk individuals for management changes and possibly early medical/surgical intervention; and 2) help to make informed breeding decisions in at-risk pedigrees
  - Whole-genome approaches are being taken to identify chromosomal regions associated with OC, as well as to identify specific variants within those regions

Original references for reported data available upon request.