The American College of Veterinary Surgeons Foundation recently awarded Dr. Larry Bramlage the 2010 ACVS Foundation Legends Award for his dedication and contributions to the field of equine orthopedic surgery. The annual award recognizes ACVS Diplomates who have developed a surgical or diagnostic procedure of significant value, proven by becoming the treatment or test of choice for a given condition.

Over the span of his 30 year career, Dr. Bramlage has been influential in advancing the art and science of fracture repair. He is perhaps most well-known for developing the fetlock arthrodesis, a lifesaving procedure for severe fetlock injuries that fuses and stabilizes the joint. The technique is now the classic procedure used by most equine orthopedic surgeons for fetlock arthrodesis. Dr. Bramlage has also been a pioneer of equine arthroscopic surgery and a leader in equine lameness diagnostics.

In addition to clinical practice, Dr. Bramlage has authored or co-authored over 70 publications in referred journals, written numerous book chapters, and speaks at numerous professional meetings every year. He is a past president of the American Association of Equine Practitioners and the ACVS, served 12 years as chair of the Research Advisory Committee of the Grayson Jockey Club Research Foundation, and continues to serve on its Board of Directors. Dr. Bramlage also serves as the media liaison for the AAEP’s On Call program where experts are immediately available to the media during many televised races, including the Triple Crown and Breeders’ Cup.
- THE EFFECT OF TRAUMATIC AND DEVELOPMENTAL FRAGMENTATION ON THE JOINT -
DR. L. Bramlage, DVM MS

- THE PROGNOSIS FOR ATHLETIC ACTIVITY AFTER INJURY AND ARTHROSCOPIC SURGERY OF THE FETLOCK -
DR. L. Bramlage, DVM MS

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“An orthopedic surgeon must be able to think like a bone, and feel like a joint”
Anon.

The fetlock joint is arguably the joint that makes a horse a horse. Its unique anatomy and physiology allow the high-speed medium distance activity that has lead to the unique place for the horse in society, historically and currently. Its evolution allowed the horse to become a single digit quadruped. It is a joint, a shock absorber, an energy storage system, and a stabilizer of the distal limb. It is constructed like a suspensory bridge, with structural members incapable of supporting its loads until the appropriate ligament tenses and supports the bone. It is the most fascinating of the complex of joints that allow a horse to move at high speeds, over rough terrain, with little conscious concern. Due to its complexity it is vulnerable to a variety of traumatic and developmental problems that are the veterinarian’s purview.

This paper will discuss the traumatic and developmental diseases of the fetlock joint and concentrate on the physical causes of diseases of the fetlock joint and the surgical treatment. Space and time limitations will preclude details of surgical technique for most procedures. Concepts will be presented and surgical techniques referenced where possible.

Defining the problem
Short of a fracture needing the re-construction of an articular surface, the majority of clinical problems in the fetlock joint present a risk for secondary degenerative arthritis as their sequelae. To reach the point of a decision to treat the fetlock joint surgically we must be able to understand the concept of progressive degeneration, to assess the current status of the joint, and to understand when the surgeon can make a difference.

Primary degenerative arthritis is rare in the horse. What we see in the horse is degenerative arthritis secondary to an insult of traumatic or developmental origin. The overwhelming volume of literature in recent years has concentrated on the biology of the degenerative arthritis cycle. This paper will concentrate on debris in the joint, the resultant physical damage, and the biologic responses that it initiates.

The physical debris liberated into the joint as part of the original insult, and by joint’s attempt to heal, mediates most of the ongoing damage after a traumatic or developmental osteochondral fragment. If the cause is developmental or traumatic, the secondary reaction of the joint is remarkably similar. So, it follows, the treatment will be similar as well. When one examines the reaction of the joint to a traumatic disease such as an osteochondral fragment, or to a developmental abnormality such as an osteochondritis dessicans (OCD) the place where the two inciting causes are similar is in the shedding of debris into the joint.
Debris can be a physical or biologic stimulus and as the debris is liberated the joint responds with inflammation. Injection of cartilage particles experimentally can create synovitis and mechanical injury to cartilage but bone debris is likely more important and more damaging. Removal of the inciting lesion and its associated debris allows the joint to return normal if the secondary arthritis has not reached the critical threshold of self-perpetuating degeneration.

**Degenerative joint disease (DJD)** has sometimes erroneously become a “catch all” for any change seen on radiographs. Most joint inflammation seen in the equine athlete is not degenerative, especially in young horses; it is the response to traumatic or developmental insult. The response to trauma or a developmental OCD lesion is often reversible early in the course by removing the inciting cause and therefore it is not degenerative.

Degenerative arthritis certainly will occur if the inciting cause is left unattended and the destruction progresses to the point that it will proceed unabated even if the cause is removed. But fortunately, lameness and decreased performance usually occur in horses in strenuous activities before the joint reaches this state providing the opportunity for diagnosis and treatment of the primary disease before it becomes irreversible.

The clinical signs can be subtle, especially if the level of the horse’s activity is moderate. In the author’s experience the attending veterinarian for high level equine athletes has an advantage, in that horses in heavy work will show more significant signs than a horse in light activity even though the joint’s response is the same in total. High-level activity elevates the rate of debris shedding and magnifies the joint response, but hypothetically the amount of debris shedding appears to be a direct product of the severity of the problem and the activity level of the horse. Small problems or low-level activity may shed debris at a rate that causes a subtle joint response creating lameness that remains sub-clinical. But low-level clinical signs are indications of equal concern and eventually can cause acute significant lameness, because in the author’s opinion when debris shedding approaches a critical mass the clinical damage will be similar.

The need for treatment is obvious with acute damage such as a dorso-medial proximal phalanx (P-1) chip fracture of the fetlock joint in a racehorse, because the debris shedding is acute and the damage to the joint is rapid and causes lameness early in the course of the disease. But a show horse or a pleasure horse with a similar lesion may show only mild signs, such as effusion and pain on flexion, but no lameness. Though the debris shedding may be slower in less strenuous activity, clinical observation suggests slower shedding causes lameness and causes joint damage once a comparable amount of debris is shed.

The need for treatment in the racehorse is obvious when it cannot perform due to lameness. The need for treatment for a subtle injury in horses of less strenuous activity may be less clear when debris shedding causes only the synovial effusion, but the horse can still perform. The permanent damage accumulating in the joint is less obvious, because the rate of damage is slow, but the end effect is no less severe. Once the same amount of debris is liberated, even though it takes a much longer time, the end result is equally severe.8

The determination of the need for treatment in the pleasure or working horse with subclinical disease is more difficult. Not all horse’s careers are valuable enough to warrant surgical intervention for all conditions. Simply the fact that the horse can perform is not an indication that the fragment is innocuous, especially if the horse requires ongoing intra-articular therapy to mitigate the signs. Often a fragment will be tolerable for a prolonged period of time, especially with intra-articular medication. If the horse is easily replaceable, or its use can be easily changed, then the decision may be to tolerate the problem. But, if it is unacceptable to have the horse disabled in a few years and its career shortened, then
fragment removal is the best treatment. So, each diagnosis of potential damaging traumatic fragmentation or developmental bone malformation must be evaluated in light of the long-term effect on the horse’s career and performance level. The decision to remove or to tolerate the presence of a fragment needs to be an active one and not simply a matter of assuming that minimal clinical lameness means no harm.

Economic realities must figure into the evaluation. Early surgical removal requires an up front expense, but with the good prospect of permanently solving the disease.\textsuperscript{9-11} Forgoing the surgical treatment of the fragment and treating it medically, especially with \textit{symptom modifying medications}, will be progressively expensive over time, and will ameliorate the signs, but not the progression of the disease. Eventually the debris shedding will damage the hyaline cartilage to the degree that it will disable the joint. This will end or denigrate the horse’s career after a few years, necessitating replacement of the horse prior to the end of the horse’s natural career span. Medication may be able to relieve the clinical signs, or interrupt the biologic processes of joint inflammation by negating the biologic debris, but it will not stop the physical debris shedding, which continually takes its toll on the irreplaceable hyaline cartilage. Owners and some veterinarians mistakenly assume the mitigation of clinical signs is a cure, especially in high-class performance horses that can afford frequent intra-articular therapy. They assume that frequent joint injections are “simple maintenance”. Then when the “maintenance” no longer “maintains” the joint, they are confused about the lack of response, and consider surgery as a treatment. The worst choice that can be made is to treat horse with a surgically resolvable disease medically, allow the joint to suffer irreversible damage, and then attempt to surgically remove the cause. This guarantees the worst of both possibilities: the most possible expense, and the shortest possible career.

The \textit{irreversible change} in a joint is the loss of the structural aspect of the hyaline cartilage, primarily the collagen. The chondrocyte population is labile, but has some reproductive capability if the insult is not overwhelming.\textsuperscript{3,12} The physiologic component of the cartilage maintained by the chondrocytes, the proteoglycan, is replaceable; in fact it is in a constant state of flux, normally in equilibrium, with production equaling destruction.\textsuperscript{3} In inflammatory conditions the production may be slowed or the destruction accelerated, tilting the balance toward a deficit in proteoglycan balance but simple proteoglycan depletion is reversible.

The lubricating function of the proteoglycan protects the collagen in the normal joint. In the diseased joint the proteoglycan becomes depleted and the collagen becomes exposed and vulnerable. So, any measure that preserves proteoglycan function and its lubrication of the cartilage surface, and therefore its protection of the collagen, is likely worthwhile to consider for the prevention of collagen degeneration, and degenerative joint disease/degenerative arthritis. This is where the disease modifying medications have their greatest potential for benefit, but symptom-modifying medication has the greatest potential for harm.\textsuperscript{(Frisbie, Kawcak et al. 2009)} This is also where surgery can shine if the cause is removable, and the rest of the joint is still normal.

Extensive or prolonged loss of proteoglycan combined with use of the joint makes the cartilage collagen architecture so vulnerable to wear that it allows permanent damage in short order, in the form of destruction of the collagen “backbone” of cartilage.\textsuperscript{3,13} Medical treatment principally modifies the proteoglycan balance of production and destruction in the joint.\textsuperscript{14} No treatment, medical or surgical, has yet shown the ability to produce quality collagen replacement, of the type of collagen architecture that is present in hyaline cartilage.\textsuperscript{5,12,14}
The subchondral bone architecture is very important in its support of the collagen in the correct configuration for joint function.\textsuperscript{15,16} The bone determines the anatomy and keeps the cartilage in the correct location to articulate with its opposing joint surface. The calcified cartilage on the surface of the bone also serves as the anchoring point for the collagen of the cartilage. Bone can be replaced, but it is very difficult, nearly impossible, to replace the calcified cartilage/subchondral bone contour, and its perfectly adapted anatomy, of the normal joint.\textsuperscript{17} So the collagen’s role in normal joint function requires the anatomy of the bone be preserved.

Collagen can be created, and proteoglycan can be replaced, but the body is unable to re-create the arching configuration of the collagen architecture that anchors hyaline cartilage to the bone.\textsuperscript{15,17,18} It is this architecture that is required to reach the functionality of hyaline cartilage, and it is this architecture that is missing from fibro-cartilage that is formed after hyaline cartilage loss. “Hyaline like” replacement fibrocartilage has proteoglycan content and collagen, but not the collagen architecture that it takes to stand up to exercise in the horse. It is therefore, to this point in time, impossible to re-create normalcy in a damaged joint surface, in an adult horse, by any means. The lack of a solution for degeneration places a premium on prevention.

Figure 1: This diagrammatic representation of articular cartilage illustrates the collagen structure of hyaline articular cartilage. (Modified from Blue Histology of Skeletal Tissues School of Human Biology, U.West Aust.).
In degenerative arthritis the collagen is worn from the joint surface into the middle and deep layers of cartilage due to failure of the lubrication/protection mechanism and the physical effects of the debris. The superficial layer of the cartilage containing the tops of the arches of collagen is lost first. (Fig.1) This exposes the middle layer of collagen initiating the process we recognize as fibrillation of the cartilage. With fibrillation the low friction joint surface is replaced by the fibrillated, higher friction joint surface, lubrication becomes more difficult, proteoglycan loss accelerates and the physical wear overwhelms the cartilage’s resistance to wear resulting in progressive loss of joint function. This process can be accelerated or mitigated with exogenous joint therapy, systemic or local. Texts and manuscripts are voluminous on this subject in the literature.19

Hyaline cartilage cannot be repaired because it has no blood supply and must be replaced by fibro-cartilage; but fibro-cartilage does not have the collagen arches that provide the resistance to joint loading and wear unique to hyaline cartilage. Fibro cartilage has an irregular haphazard collagen arrangement with cross-linking of the fibrocartilage, but a relatively poor or non-existent anchorage to the bone. Fibro-cartilage is intended to cover joint surface defects with a scar, which it does effectively, but it is not constructed to participate in joint function. Since fibro-cartilage is relatively poorly anchored to the underlying sub-chondral bone and it functions well to cover a defect in the joint surface that penetrates the subchondral bone, but does not resist wear effectively and under heavy loading, especially in shear, fails and detaches from the bone it covers.17,18,20

Bone can heal, but has difficulty restoring joint surface architecture. Unless bone healing restores the innate joint anatomy perfectly, the repaired joint surface is suboptimal for weight bearing because the fibro-cartilage covering the injured bone does not reach perfect articulation with the opposing joint surface.

Taken together, the repair process for bone and cartilage of an injured joint surface is poor and achieves success as a scar, but not as a functional replacement.21 This can result in prevention of inflammation in the sedentary joint, but the scar cartilage and deranged bone anatomy cannot restore normal joint function to injured joint for athletic function.

What is surgically possible?
Surgical reconstruction of a joint surface whenever instability is present and preservation of as much articular surface as possible is a prime indication for surgical treatment. But, when a joint is injured, most treatments consist of removing the damaged area to negate the ongoing effects of the injury. The joint resumes function using the preserved normal hyaline cartilage and does not rely on the repaired injured surface. Millions of dollars are spent on researching improvements to this process of cartilage repair, but currently no clinically applicable techniques have resulted in a joint surface that can resist the loads created in the equine athlete.

The most theoretically promising approaches are the surgical techniques such as micro-fracture resurfacing which offers an opportunity for the fibro-cartilage to gain anchorage into the subchondral bone, but unfortunately this technique has proven to be unsuccessful in restoring athletic function after injuries commonly seen in the horse.22,23 Any injury over one centimeter in diameter, even in a person, cannot resist the loading asked of a repaired joint surface, even with the added benefit of micro-fracturing.24-26 (Fig. 2) In the author’s opinion micro fracturing injured joint surfaces on the dorsal condyles of the cannon bone, or on the articular surface of the sesamoid bones has not aided in recovery from the damage to those areas that badly need a treatment solution. These are such heavily loaded articular surfaces that asking fibro-cartilage to perform functionally is not likely feasible.
Injuries that violate the sub-chondral bone plate can re-create a superior fibro-cartilage because the fibrocartilage can anchor on the exposed cancellous bone effectively. However, a deficit in the bone rarely fills to the degree that the bone becomes congruent for weight bearing. So the cartilage is anchored well, and does not fail because it is in a protected environment below the normal articular surface and overlying cancellous bone. But, it does not participate in the function of the joint, and therefore is of a high quality histologically but is of little use functionally to the horse.

Injuries that remain weight bearing, even if micro-fractured, seldom maintain the fibrocartilage in the face of serious weight bearing exercise, as the collagen anchorage is insufficient to resist the forces of weight application and is quickly sheared from the subchondral bone. Removing the subchondral bone plate totally to expose cancellous bone helps the collagen anchor to the bone, but does not achieve function, because the normal architecture is destroyed to the point that joint function cannot be preserved. Forage of damaged subchondral bone with multiple drill holes improves anchorage, but does not produce enough functional cartilage to restore joint function, and is most likely impractical over a large area in the horse, because prolonged protection from loading while a functional fibrocartilage forms is not possible.

Grafting of cartilage, chondrocytes, or stem cells faces the same difficulty in establishing an attachment to the sub-chondral bone, as does granulation tissue trying to form fibrocartilage. Mosaic-plasty (taking small plugs of cartilage and bone from a remote site and inserting them in a denuded area) has been tried, but lack of equivalent donor sites, stabilization of the plugs, and re-construction of a functional anatomy without damage to the transplanted grafts have all limited use. Prosthetic replacements as in people have little chance to survive the biomechanics of the much larger horse and re-create an athlete in the horse, or in fact in people.

Biologic therapy such as stem cells and platelet rich plasma is being used and shows promise in aiding joint function, but is unlikely to be able to substantially replace articular cartilage during mechanical loading with the loads that are seen in the horse. The anchorage to the subchondral bone is still an unsolved problem.
So, surgical replacement or augmentation of an injured joint surface is to this point not a reality. If injured joint surfaces can be re-constructed using internal fixation to re-constitute the original anatomy with the original cartilage surface intact, or nearly so, surgery shines. But, surgery to repair, replace, or restore cartilage that has been lost due to the degenerative process is a clinical impossibility to this point.

**The Role of Surgery**
The primary role of surgery is to stop debris shedding and restore stability. This is not to disparage the role that surgery plays in the approach to arthritis. It emphasizes the role of **prevention of arthritis**, which is surgery’s strength, rather than the **treatment of arthritis**, which is its weakness. Surgery is primarily a bone treatment, with a few exceptions in the horse. Surgery stabilizes the reconstructable joint surfaces, and removes the damaged bone that would shed debris and further injure the joint. The joint must survive on whatever cartilage can be preserved by reconstruction, or by prevention of the degeneration mediated by debris shedding and the subsequent cascade of inflammation, which leads to degeneration. (Fig. 3)

**The Role of Medication in a Joint**
Symptom modifying medications can decrease or resolve the clinical signs that accompany arthritis and can be quite useful at buying time for the joint to heal itself, but can be abused if they are used to cover up a physical problem that continues to damage the joint. They should be used with a thorough understanding of their benefit as well as disadvantages for the joint. When used to simply cover up a physical injury, which results from shedding debris, obtunding the inflammatory response will eventually allow the physical injury to disable the joint. (Fig. 4) Disease modifying medications can promote proteoglycan anabolism and prevent destruction, and to some extent negate biologic debris that damages the joint, but they cannot stop physical debris shedding within the joint that results from unstable bone. It is the physical debris that is shed from traumatic and developmental lesions that does the ongoing permanent damage to the hyaline cartilage and therefore to the joint.5

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*Figure 3: Intra-operative picture of a dorsal P-I chip fracture removal shows the effect of debris shedding on the articular surface of the distal metacarpus (on the right of the picture). Note the score lines and cartilage fibrillation. The fragment has been removed from the left side, exposing healthy subchondral bone (P-I).*
To understand the reason for the ongoing damage a traumatic fragment of bone, or developmental fragment of cartilage and bone causes on normal joint surfaces one must review the way bone heals in the unstable situation and understand the concept of continual debris shedding. To conceptualize the unstable fragment of bone as a “stone in your shoe” far underestimates the pathology done by an unstable fragment.

**Gap-Strain Bone Healing and Its Effect on the Joint**

Bone has the capability to exactly reconstruct itself after fracture injury. It can do this via two primary mechanisms. If it is stable, bone can fill a gap with new bone (primary bone healing) and remodel to re-establish pre-injury anatomy. This will normally occur only in the case of a fracture that is non-displaced or has been surgically stabilized. In unstable bone healing the bone goes through a series of steps attempting to fill the gap between the bone ends with other tissue which eventually converts to bone (secondary bone healing).  

The relative motion of the bones in relation to each other dictates the kind of tissue that forms between the fracture ends in secondary bone healing. This “strain” between the bone ends is defined by the “change in length of the gap (motion) divided by the unit length (the gap)”.

Bone is a very stiff and strong structure, and as such has a very low strain tolerance until rupture, approximately 4% in cortical bone and approximately 5% in membranous bone. In stable situations motion is not a factor because the motion and therefore the strain is under 4%, and the gap can easily fill with replacement bone. In unstable situations the strain is greater than the bone can tolerate and therefore newly formed bone simply ruptures.

Cartilage has a 15% strain tolerance so it can tolerate 3-4 times more motion than bone; and granulation tissue, or immature fibrous tissue has a 100% strain tolerance. So with high motion between the two ends of a fractured bone the bone must start by forming granulation tissue because it will tolerate the highest strain. As more granulation tissue forms and the tissue holding the two ends of the bone together enlarge it becomes stiffer, thereby reducing the strain between the bone ends. The reduced strain allows less strain tolerant tissue such as cartilage to then form and the mass, known as callus, gradually.
creates a union by progressively enlarging and stiffening until it becomes stiff enough to form bone. We recognize this process in most fracture healing by the expanding callus that eventually stabilizes and unites a fractured bone, then re-organizes and replaces the callus with the stronger, smaller, more organized bone.

Because this process of secondary bone healing by callus is controlled biomechanically and dependent on the local biomechanics of the gap strain healing process, which is a ratio, the gap size and the motion are both important. The surgeon can facilitate healing by reducing the strain by fracture reduction and immobilization, and with restriction of exercise. Align the bone ends to a normal anatomical configuration because the ends of the bone must be in close proximity to allow favorable local biomechanical conditions to unite the bone. Counter-intuitively a very small gap will have a higher strain than will a large gap (motion divided by a small number results in a higher strain than the same motion divided by a larger number). To reduce strain the gap must enlarge before healing can occur. We recognize this process when unstable fractures get wider radiographically, increasing the gap, before they begin to heal. (Fig. 5)

Figure 5: This radiograph demonstrates a medial condylar fracture that was not stabilized surgically and has widened the fracture gap to reduce the strain. This is part of the healing process for an unstable fracture.

If the strain is over 100% it is too high to allow granulation tissue to unite the bone and the granulation tissue simply covers the raw bone ends as it does in any raw bone surface in a joint. Increasing the size of the gap, as long as the bone ends are still approximated and aligned and the strain can be reduced below 100% will aid fracture healing. Increasing the gap will reduce the strain, helping to get it below the 100% limit of granulation tissue, and allow the callus process to begin since fracture gap strain is a ratio, motion divided by the gap size. This is a locally mediated phenomenon stimulated by the local conditions surrounding the fracture. The radiographic widening results from softening of the bone by demineralization and disorganization of the bone, opening the space between the pieces of the fractured bone reducing the strain and allowing secondary bone healing to proceed. This is a novel and effective way to promote fracture healing if the bone is not loaded at the time.
This same process occurs in the joint when a fragment is present. Healing between the parent bone and fragment is attempted but this is a very high strain environment because the motion is high in a joint, and the gap is very small. Therefore the parent bone actively softens, and disorganizes to try to open the gap and allow healing to occur. But, if the bone is loaded during this process it breaks up and is shed as it is demineralizing. Marked reduction of motion will sometimes accomplish healing and a chip fracture will heal. But even cast immobilization is not enough to heal most fragments and the secondary bone healing process persists, and accelerates with resumption of exercise. Because of the loading with motion this progressive destruction of the parent bone at the interface with the fragment, or OCD, results in tremendous amounts of bone matrix and mineral continually shedding into the joint. This constant debris shedding is the primary cause of the degeneration of a joint rather than the presence of the fragment and the “stone in your shoe” phenomenon. In most joints fragment stability is not possible to achieve.

Continued attempts at healing unstable fragments occur if raw bone is exposed, with resultant debris shedding progressively joint damaging the joint. In instances of strain in excess of what the bone healing process can tolerate, neutralization of raw bone interfaces will eventually occur by covering the bone surfaces with granulation tissue, which if it cannot reduce the strain to under 100%, then matures into fibro-cartilage via the cartilage healing process; this is the same scar cartilage that covers all raw bone surfaces in the joint. But, after fibro-cartilage forms, high-level use can erode this cartilage as it does in all high load situations, exposing bone again, stimulating secondary bone healing, and re-igniting the debris shedding. So clinically the degree of success of fragment neutralization by fibrocartilage parallels the degree of success of joint surface healing by fibro-cartilage. In the author’s experience for less strenuous uses fibrocartilage can be functional, but for increasing athletic activity the probability of erosion of the fibrocartilage and re-initiation of debris shedding increases with the level of athletic activity.

When exercise resumes, if the fibrocartilage fails, the parent bone again responds by trying to increase the gap size to reduce the strain and heal the bone. The fragment can also respond in the same way if it retains sufficient blood supply, but this is not usually the case. The reaction can be identified radiographically as demineralization at the interface of a mobile fragment and can be seen with the arthroscope as the softening of the parent bone, which is making an attempt to heal the fragment. The most striking evidence however, is on the normal cartilage surface where the debris gets interposed between the cartilage surfaces and physically scores the normal cartilage. These “score lines” destroy the superficial layer of articular cartilage where the pinnacles of the arcades of collagen normally form the tough gliding surface of the articular cartilage. This damage is permanent, and tolerated to a point, after which enough loss of superficial layer of cartilage has occurred that the cartilage can no longer protect itself and it erodes to bone.

The fact that chip fractures are not created by a single event, and are the result of repetitive trauma, amplifies this response. So by the time the fragment separates radiographically the bone demineralization and softening is well underway from attempted healing, debris has already been shed into the joint. This process is an active response, which is minimally influenced by the size of the fragment. Even small fragments stimulate the softening by the parent bone over a large area, shedding debris and doing permanent damage to the joint. The size of the parent bone/fragment interface influences the amount of debris shed at one time and the rate of damage to the normal joint. Mobile fragments stimulate the debris shedding into the joint and is the same response to traumatic and developmental fragments becomes indistinguishable and ends with the same result.5

Many interpret the cause of pain and lameness from a chip fracture as direct physical injury to the joint surface. This is rarely the case. Cartilage has no nerve endings with which to
generate pain. The healing process results in trauma from small particles of bone being shed as well as creating biologic debris, such as interleukin-I beta, which promotes the synovial inflammation and cartilage destruction that are the subject of much joint research and the target of all joint therapy.\textsuperscript{19}

The primary traumatic injury with secondary debris shedding, if left unchecked, causes secondary degenerative arthritis via a two-pronged process. It is a biologic process, which attacks the physiologic function of the proteoglycans and eventually the collagen; and it is a physical process that directly abrades the hyaline cartilage surface causing irreversible physical damage to the cartilage structure, especially the collagen, that becomes the degenerative arthritis that disables the athlete. The biologic process alone can eventually reach the degenerative threshold, once the collagen has been degraded enzymatically or eroded physically due to lack of adequate lubrication. But the physical debris fragments stimulated by the healing response of the parent bone is the most direct path to degeneration via the physical damage and the secondary biologic response they create. Debris is the primary enemy of the athletic horse’s joint.

**Debris management**
Natural joint management of debris causes prioritization of joint resources. In the normal situation, the synovial fluid is low in protein, contains almost no fibrin and is viscous due to the large amount of hyaluronan within the joint.\textsuperscript{4} The purpose of normal synovial fluid is to lubricate all joint functions and promote free flowing joint motion. It would be counterproductive, however to lubricate debris within the joint and encourage it to repeatedly pass through the articulation. So the lubricating capability of the joint is sacrificed temporarily for increases in fibrin and reduction of joint motion to give the joint an opportunity to clear the offending debris. When the insult is neutralized in the joint, the synovial fluid then returns to its normal highly lubricating capabilities. The need to remove debris is a common occurrence in a normal joint.

It is clear therefore that allowing persistent debris shedding and low grade inflammation within the joint is counterproductive in the long-term as it compromises the joint’s ability to lubricate itself and maintain the articular cartilage in a wear free state. The critical nature of maintaining the hyaline cartilage, since it cannot be reconstructed or replaced, points out the high priority that should be placed on removal of even low grade chronic insults and normalizing the homeostatic lubricating mechanism that preserves the hyaline cartilage.

When fragments of cartilage or bone are shed within the joint, the joint responds by producing fibrin. In the margins of the joint this fibrin attaches to the synovial villi and catches the debris as it circulates in the joint. The fibrin facilitates the attachment of the debris to the synovial villi for elimination. The debris is therefore removed from bearing surfaces of the joint, preventing it from abrading the articular cartilage. The debris is sequestered in the cul-de-sacs of the joint. The tiny granulomatous nodules encasing the debris can often be seen arthroscopically. (Fig. 6) The fibrin and small nodules stabilize the debris so the cellular components, chiefly the neutrophils and macrophages, can remove it.
Debris removal necessarily requires release of potentially destructive enzymes and free radical molecules as part of the process. When it occurs in short bursts with acute insults the process rapidly clears the joint of debris and has little ill effect on the articular cartilage and joint environment. There is not enough wear during a transient period of inflammation to do significant harm, but if the balance of production and degradation is shifted toward depletion of proteoglycan long enough to affect cartilage lubrication destruction of the cartilage surface is initiated.

This rapid efficient process of debris removal makes it possible for the joint to respond to an acute insult such as surgery. If the joint were not able to tolerate an acute insult, surgery would not be possible. If the debris shedding is continuous, however, then the continual enzyme liberation within the articular cavity affects not only the debris, but also the lubrication mechanisms of the joint and the normal articular cartilage by progressive attrition of the proteoglycan content and loss of the normal lubrication mechanism. This makes the articular cartilage vulnerable because it shifts the proteoglycan equation of production equaling destruction in the toward destruction, and therefore proteoglycan loss. This compromises the articular cartilage lubrication mechanism, allowing surface wear and fibrillation to occur. This causes normally physiologic loads to gradually erode the layers of the normal hyaline cartilage, a permanent, progressive and irreversible, degenerative change.

**Surgical Treatment of Joint Injury**

The role of surgery in the fetlock joint then is simple. Restore stability to **facilitate primary bone healing** or remove unstable fragments to **prevent secondary bone healing**. With few exceptions surgery treats bone disease whereas surgery cannot “treat” arthritis that is underway. If the arthritis is not beyond resolution, surgery can delay or mitigate the progression by removing the inciting cause. Removal of fragments of traumatic or developmental origin keeps the parent bone normal, and prevents the debris that does most of the damage. The horse performs on the remaining normal joint surface. The restoration of damaged arthritic joint surfaces by surgical techniques remains a strongly pursued goal, but the “state of the art” at this time is that it does not work very well. The best chance for athletic function in a joint with a fragment is to prevent the primary injury.
from stimulating the process that degenerates the rest of the joint by removing the fragmentation and associated debris. Joints with major cartilage loss in critical areas remain career damaging or career ending challenges. So surgery is a better prevention than a cure for arthritis with joint injuries in the horse.

**Surgical Treatment, do we use it enough?**
Surgical treatment of joint fragmentation is rapid and effective in stopping debris being shed into the joint. The removal of unstable bone and its associated debris preserves normal joint surface that will be lost if the parent bone attempts to heal an unstable fragment. The process of the parent bone trying to reattach and heal an unstable fragment is often worse than the primary disease. The extreme range of motion of the fetlock joint makes it even more vulnerable to this type of damage than most other joints. Physical debris tends to disseminate throughout the articular surface scoring the cartilage surface until it is sequesters in the cul-de-sacs of the joint. (Fig. 7)

Any opportunity to interrupt this debris shedding process has a positive effect on a horse’s joint. Surgery should be used more, and earlier than we often elect, to treat articular injury before it does irreversible damage.

The strategy in treating traumatic and developmental joint injury is to negate the inciting and perpetuating cause, usually an unstable or malformed area of joint surface, and preserve every bit of normal joint architecture and hyaline cartilage possible. Surgical stabilization or surgical removal of the problem, whenever possible, is the most definitive treatment and results in the fastest, best resolution of the problem. In most instances, modifying the clinical signs medically, without eliminating the perpetuating physical cause, risks permanent damage.
Literature


What Causes Joint Pain after an Injury?

- Bone inflammation
  - Bone Hypertension
  - Invasion of sensitive structures
- Stone in your shoe effect (only free fragment)
- Biologic effect on the joint cavity-stretch
- Avascular necrosis?????

Our recent discussions of Joint diseases have cemented the concept of DJD

- But, in some instances has
  - Erroneously led to a catch all diagnosis
  - Fosters cookbook treatment plans
  - Discourages the tailoring of treatment plans to the individual patient
Clinical Presentation
*primary insult with secondary response*

- Usually singular or paired joints
- Acute insult or chronic accumulated damage followed by regression and plateau then resolution or accumulation
- Complaint is often reduced performance

Review
*Collagen is critical*

- Hyaline Articular Cartilage is not replaceable
- What is replaceable
  - Proteoglycan
  - Chondrocytes (to some degree)
  - Calcified cartilage

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Collagen arches
Surgery, in my opinion, is primarily a bone treatment

- In general, surgically, what we can do for bone has a much bigger effect than what we can do for the rest of the joint, in the horse
- Stabilize Bone so That Primary Bone Healing Can Occur
- Control Secondary Bone Healing and therefore Prevent Debris

How does the Joint deal with Debris

- The fibrin attaches the debris to the villi which then encases it in fibrous tissue with time and digests it or encapsulates it
- This results in vascular hypertrophy and low grade inflammation until the debris is gone
- Low grade inflammation results in decreases in
  - Joint Hyaluronan
  - Cartilage proteoglycan
- Which results in decreased joint lubrication
Ms J. Rattray studied in several countries resulting in a PhD. graduation in 2008 at the University of Utrecht. From 2009 until now she has worked with the GD – Animal Health Service on innovation of laboratory diagnostics, setting up and implementing research plans, maintaining high technical level of laboratory analysis and advising laboratory management on technical/scientific issues. PhD. J Rattray has published several articles.

**Current Employment**

2009-11  
Research Scientist Veterinary Chemistry, GD - Animal Health Service, Deventer, the Netherlands.  
Core tasks: innovation of laboratory diagnostics, setting up and implementing research plans, maintaining high technical level of laboratory analysis, advising laboratory management on technical/scientific issues.

**Education**

2008  
PhD in marine organic biogeochemistry, University of Utrecht.  
Thesis: Ladderane Lipids In Anammox Bacteria: Occurrence, Biosynthesis and Application as Environmental Markers.

2003  
MSc Environmental Science, University of Wageningen.  
Thesis: Denitrification losses from agricultural ditches.

2000  
BSc Environmental Science (Environmental Chemistry), University of Plymouth, England.

1998  
HND Environmental Protection and Management, Scottish Agricultural College, Ayr, Scotland.
Osteochondrosis (OC) is a problem in the bone ageing process which occurs in many animal species, including the horse. In horses, changes in OC are important for both breeding selection, purchase and sale inspection purposes. Changes in OC can be observed radiographically and are defined by changes in subchondral bone contours which are not accompanied by fragmentation. If the X-ray examination reveals bone fragmentation then the condition is called osteochondrosis dissecans (OCD). We speak of OCD when in addition to contour changes one or more fragments are visible. These fragments are usually not separate, but are linked to the underlying bone cartilage. Sometimes these pieces separate and become abandoned secondary ossification cartilage pieces, called secondary chondrometalasie. In the formation of cartilage into bone, various factors play a role. OC begins with the newborn foal and possibly at an earlier stage. At the age of five months the incidence is highest, when most foals still drink their mother’s milk.

In addition to a good genetic background, proper bone development requires several minerals (calcium, phosphorus, magnesium), trace elements (copper, zinc, manganese) and vitamins (vitamin D, K). Two experiments were therefore carried out to investigate the effect of giving a supplement to foals aged between 0 and 12 months old, and to observe the incidence of osteochondrosis. A second goal of the study was to measure the relationship between bone metabolism and osteochondrosis in foals in order to find biomarkers for osteochondrosis. Experiment 1 was conducted on 63 KWPN (Studbook of the Royal Dutch Sport Horse) warmblood foals between 0 and 5 months of age. The experiment showed that administering extra supplement significantly reduces the risk of OC. It was also shown that a high bone turnover (high levels of CTx and / or osteocalcin) is more common in animals with OC. During experiment 1 the composition of the mother’s milk was also analysed for variations in the natural mineral content, and the composition of the milk showed little variation.
Conclusions from the trial on foals aged 0-5 months (Experiment 1) are that the supplement halves the incidence of osteochondrosis in foals at 5 months old. An active bone metabolism increases the risk of osteochondrosis and that movement reduces the risk of osteochondrosis.

Determining changes in OC in horses is currently done through x-ray examination with the most important joints being the horse’s fetlock, knee joint and tarsus (hock). This screening can be done from the age of 12 months, when the radiographic image is almost stable. In Experiment 1, the foals averaged five months old when the x-rays were taken and were evaluated for the presence of radiologically visible changes relating to OC. There is a relationship between radiographic changes visible at five months and twelve months, but this relationship is not always clear. Five month old foals with no radiographically visible changes relating to OC can still go on to develop OC at 12 months and vice versa. To translate the results of Experiment 1 into practice, it was necessary to repeat the research over the period of 5 to 12 months. Only these results can actually be used in practice.

In experiment 2, 52 KWPN warmblood foals were use between 5 and 12 months of age. Again half of the foals received a supplement containing highly digestible magnesium and phosphorus and in all foals the incidence of OC was investigated at both ages. The bone parameters CTx (C-telopeptide) and osteocalcin were also analysed.

The findings from Experiment 2 showed there was no direct link between feeding supplement to foals from 5 months to 12 months and the incidence of osteochondrosis. However, it did appear that the supplement provides a greater chance of improving osteochondrosis at 12 months. There was also no significant relationship demonstrated between bone metabolism and the risk of osteochondrosis. However, there is a tendency that increased bone cleavage increases the incidence of osteochondrosis and increased bone turnover reduces the incidence of osteochondrosis.

The combined conclusions of Experiments 1 and 2 are therefore:
1. Extra addition of supplement significantly reduces the risk of osteochondrosis.
2. There is a tendency that more active bone (especially increased bone cleavage) leads to an increased risk of osteochondrosis. However, the differences are not such that it yields a useful test as an alternative to X-rays.
3. The best results are seen when the supplement is provided as early as possible.
Effect of supplement on osteochondrosis and its relationship with bone parameters in foals

Jayne E. Rattray

Motivation

- Biomarkers for bone metabolism now available
- Study the role of supplements in bone development
- Osteochondrosis is a significant problem in foals
Content of the supplement

- During two separate experiments a supplement was given to the foals and it contained:
  - Extra Magnesium with a high digestibility
  - Extra Phosphate with a high digestibility
  - WITHOUT giving extra CALCIUM!

Blood analysis
Conclusions

1. Giving the supplement significantly reduces the risk of osteochondrosis.

2. There is a tendency that a more active bone metabolism (especially increased bone cleavage) leads to an increased risk of osteochondrosis. However, the differences are not such that it yields a useful test as an alternative to X-rays.

3. The best results are obtained when the supplement is provided as early as possible.
In 1998, Elizabeth van Grevenhof started her study animal health at the HAS-Van Hall Larenstein in Deventer. She has completed her graduation at the University of Liverpool, and in 2002 received her degree.

In 2002 she began a Masters program of Animal Sciences at Wageningen University. She graduated in two specializations, quantitative genetics and breeding and veterinary epidemiology and thereby obtained her MSc degree. For her first specialization, she used data from Nutreco indicators to identify the sensitivity of ascites in broilers. For the second specialization, she has worked with University College Dublin in Ireland to determine the efficiency of detecting tuberculosis in cattle at abattoirs.

In 2004 Ilse received her MSc degree, after which she began in 2005 as a research assistant at the Department of Animal Breeding and Genetics at Wageningen University. In 2006 she started her PhD research "Breeding against Osteochondrosis: phenotypic and genetic analysis in horses and pigs".

In May 2011 she successfully defended her study and this resulted in getting her Dr. degree. Ilse is currently working on this project as a postdoctoral researcher in Animal Breeding and Genetics in Wageningen.
In 1998, Danielle Arts began her studies with Zootechnology at Wageningen University. In January 2004 she obtained her degree and received the Ir. / MSc title. She has worked two specializations on during her studies and graduated first in the main direction of quantitative genetics and breeding, and second in the direction of Livestock.

For her first specialization, she made use of data from slaughter pigs, which she herself collected during her internship in the United States (Iowa State University and Premium Standard Farms, Missouri). The aim was to calculate genetic parameters for various carcass parts in the pig. During her second specialization, she worked on embryo transplantation in pigs in association with the Institute for Pig Genetics in Beuningen.

After graduation, she worked for Nutreco from 2004 to 2008, the IT department Breeding, as part of the R & D department. Here, she has 5 different types of breeding programs (pork, chicken, turkey, salmon and layers), a new design for a breeding database for storage of the breeding dates. Nutreco’s breeding business in 2007 sold to Hendrix Genetics, where Danielle followed. Besides its IT operations, she was partially effective as a geneticist in the breeding of broilers and breeding, she has worked on various issues and was responsible for running part of the breeding program.

Since October 2008 she worked as assistant Breeding for the Royal Dutch Sport Horse federation (KWPN) where she is responsible for the genetic evaluation and all the scientific research within the pedigree. For this she works closely with scientists from Wageningen University.
For decennia, osteochondrosis has been the most important orthopaedic developmental disorder in horses. In this species, OC is mostly found in the femoropatellar and tarsocrural joints. Given the supposed or perceived impact on locomotion, performance and commercial value, much research has been carried out in the horse. Selection against OC has not been very effective in the most practical breeding programs, possibly because of the incomplete phenotypic definition. In studies designed to fill this gap, prerequisites are a sufficiently large population sample that is well defined for age, gender, and parentage, and a detailed and consistent radiographic scoring system.

Wageningen University and KWPN have carried out a study recently to assess the prevalences of OC in various joints, estimate genetic parameters for OC-traits and to evaluate the prospects of various breeding strategies against OC, including genomic selection.

This study used radiographic data from 811 KWPN-yearlings which descended from 32 representative breeding stallions. Animals were scored for OC based on radiographs from eight joints: the femoropatellar (FP), tarsocrural (TC), metacarpophalangeal (MCP) and metatarsophalangeal (MTP) joints. A total of 28 predilection sites per animal were scrutinized for the presence of OC lesions: 5 in the FP joint, 7 in the TC joint and one in each of the MCP and MTP joints. At each site OC was scored on a categorical scale from A through E, adapted from Dik et al.(1999). This study resulted in implementing a progeny testing structure for newly approved stallions since 2007 within the breeding program of KWPN.

**Prevalence of OC in horses**
In the FP-joint, the percentage of animals showing normal joint contours in all sites was 60.7%. For the TC joint and the combined MCP/MTP joints, these figures were 68.8% and 64.6% respectively. For all joints combined, the percentage dropped to 30.5%. Sedation improved detection of OC lesions in the FP joint. There was a high correlation between the right and left joints. From this study we learned that scoring on a detailed scale is necessary to achieve good insight into the prevalence of OC.

**Genetic parameters of OC in horses**
For an effective breeding program and to predict the selection response it is necessary to estimate the genetic parameters between joints and between manifestations (flattened bone contours or fragments) of OC.

At the animal level, the overall heritability of OC was 0.23, the heritability of flattened bone contours was 0.08 and the heritability of fragments was 0.22. At joint level, the heritability was highest in the TC-joints, intermediate in the MCP/MTP joints and lowest in the FP-joints. The genetic correlation between TC and FP joints was high, whereas the genetic correlation between the MCP/MTP and other joints were moderate. The genetic correlation between the flattened bone contours and fragments at the animal level was 0.80.

The results suggest that selection against OC could be performed by taking into account the OC status of all 4 joints and discerning between flattened bone contours and fragments.

**Breeding strategies against OC**
Three selection strategies were compared with regard to the effectiveness in optimizing the long-term response to selection against OC. In these scenarios, selection was on one trait only. The number of selection candidates and the total number of animals selected were fixed.
Three different basic scenarios:

1) phenotypic selection using pedigree and own performance
2) phenotypic selection using own performance, pedigree and progeny information in a two stage selection
3) Genomic selection using pedigree and marker information

It can be concluded that genomic selection is a realistic option in the KWPN horse population for selection against OC. Even though dealing with a difficult trait such as OC, the size of the reference population that is needed to reach equal response to selection using genomic selection compared to phenotypic selection on own performance is not more than 2,225, which is certainly feasible. When genomic selection is compared with phenotypic selection on progeny testing, this figure is larger \( n_p = 9,175 \), but when taking into account the reduction of the generation interval with two years, the number falls to 5,590, which must be considered feasible as well. When systematic phenotyping progeny from newly approved stallions is used to build a reference population, annually approximately 400 individuals can be genotyped and phenotyped and the reference population can be built in 14 years time. Therefore, it can be concluded that progeny testing will lead to the highest accuracy, until the required reference population (5,590) has been built for genomic selection, when henceforth genomic selection will lead to the highest gain.

**Implementation results in KWPN breeding program**

The Royal Warmblood Studbook of the Netherlands (KWPN) is the Dutch breeding organization of warmblood sporthorses specialized in dressage and showjumping (together representing 90% of population). Besides this, the studbook comprises some typical Dutch-origin breeds which are harness and Gelder horses (10% of population). With 30,000 members and approximately 12,000 foals born each year, the KWPN is one of the largest sport horse studbook in the world. For years, the KWPN has held a top position in dressage and showjumping in the studbook ranking of the international breeding organization, the World Breeding Federation for Sport Horses (WBFSH). The goal of the KWPN is to breed a performance horse that can compete at the highest level of the sport. To achieve this goal, a horse obviously must have a healthy constitution, correct and functional conformation, and correct to superb gaits. Performance is by far the most important trait while health and conformation/movement are important traits as well to support the main breeding goal. Breeding values are estimated based on sport and test results using an animal model. Since 1989 the KWPN estimates breeding values for performance (jumping, dressage and harness sport) and conformation/movement. Selection on health includes locomotion, respiration and fertility. For these traits no breeding values are estimated yet, but only phenotypic selection on own performance of the horse is done.

**Selection on OC**

For OC, the selection strategy moved from selection on own performance to selection on progeny information and breeding values for OC are calculated. Since 2009 the progeny testing system is implemented in the breeding program for newly approved stallions. For every stallion, 20 randomly chosen yearlings are selected for screening on OC by taking radiographs. The same scoring system A through E is used as described in the study above. These scores, of 28 locations per horse, are the input for the breeding value for OC. Breeding values are published for stallions only and were published for the first time in January 2012 for 36 stallions. The KWPN is the first studbook worldwide in publishing breeding values related to OC, with the aim of significant improvement of the orthopaedic health of our horses and to support the main breeding goal trait.
Defining osteochondrosis and its genetic background

Ilse van Grevenhof
Animal Breeding and Genomics Centre

What is OC?

- Health problem in horses
- Disturbance of ossification
- In joints of young growing animals
- Previous studies show prevalences vary 7% – 64%
  - Definition OC
  - Number of joints, radiographs, locations
  - Preselected data sample
Effects influencing OC?

- Heritability
- Exterior
- Growth
- Exercise
- Feeding
- Housing

Results

- Animal level
  - Worst animal had 11 out of 28 ‘ideal’ locations
  - Best animal had 28 out of 28 ‘ideal’ locations

<table>
<thead>
<tr>
<th>Joint</th>
<th>Percentage of animals with fully ‘ideal’ joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stifle</td>
<td>61%</td>
</tr>
<tr>
<td>Hock</td>
<td>69%</td>
</tr>
<tr>
<td>Fetlock</td>
<td>65%</td>
</tr>
<tr>
<td>Total</td>
<td>30%</td>
</tr>
</tbody>
</table>
Housing and feeding effects

- Housing systems
  - 50% concrete/slatted
  - 50% deep litter

- Feeding strategy
  - Ad libitum
  - Restricted (80% of ad libitum)

- OC scored at age of 6 months, after slaughter

2-by-2 factorial design

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Are there prospects for breeding?

Standardized EBV of sires (All joints combined)

~12%

Phenotypic SD

-0.47 0.33

~58%

Sires (n=32)
Three ways of selection against OC

Conclusions

- OC in horses
  - 70% prevalence
  - 23% heritable

- OC in pigs
  - Reduced prevalence OC in deep litter
  - Reduced prevalence OC with restricted feeding

- Selection strategies
  - Selection on progeny
  - Future for selection on DNA
Implementation OC-breeding value in breeding program

Daniëlle Arts
KWPN – Royal Dutch Sporthorse Studbook

History – OC selection within KWPN

- 1987
  - selection on hock-joint, own performance stallions
  - OC is a binary trait (0/1)
- 1994
  - selection also on knee, own performance stallions
  - OC is a binary trait (0/1)
- 2007
  - results population scan
  - Development new selection strategy
    - progeny testing
    - all joints (stifle, hock and fetlock)
    - detailed scoring system for OC-trait (classes A-E)
- 2009
  - implementation progeny-testing
- 2012
  - first OC-health breeding values published
**Implementation progeny testing**

- All approved and recognized stallions since 2007
- 20 yearlings per sire are radiographed and judged in 28 locations in stifle, hock and fetlock joints
- Yearlings are chosen randomly per sire
- Calculation of breeding values, publication per stallion

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**Publication breeding values**

- Breeding value OC is presented in “genetic profile”, together with all major traits in KWPN-breeding goal

<table>
<thead>
<tr>
<th>Trait</th>
<th>Genetic profile</th>
<th>relb:</th>
<th>* * *</th>
<th>average</th>
<th>* *</th>
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<tr>
<td>Dressage</td>
<td>low</td>
<td>55%</td>
<td></td>
<td>161</td>
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<tr>
<td>Conformation</td>
<td>low</td>
<td>58%</td>
<td></td>
<td>106</td>
<td>high</td>
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<tr>
<td>Free movement</td>
<td>low</td>
<td>58%</td>
<td></td>
<td>110</td>
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</tr>
<tr>
<td>OC-Health</td>
<td>low</td>
<td>60%</td>
<td></td>
<td>106</td>
<td>high</td>
</tr>
<tr>
<td>Height</td>
<td>small</td>
<td>63%</td>
<td></td>
<td>101</td>
<td>large</td>
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</tbody>
</table>

- **Goal:** Balancing important traits against each other and no focus on one single trait