



Fifty years of osteochondrosis

"One should make complicated things as simple as possible.
but not simpler"

Albert Einstein

Although the term 'osteochondritis dissecans' was coined in the late 19th century in human medicine [1], the real history of osteochondrosis (OC) in the horse started in the same year that *Equine Veterinary Journal* was launched. In 1968 Birkeland and Haakenstad published their now classic study on 'intracapsular bony fragments' of the distal tibia of the horse [2], creating awareness about a then emerging topic that would not disappear anymore from the equine veterinary literature and has remained actual to the present day. Osteochondrosis is a fascinating disorder, principally because of its complexity. It is this complexity and multi-facet character that makes the disease into such a good metaphor of (equine) medicine in general and, related to its again complex aetiological background, also of the content of *Equine Veterinary Journal* in the past 50 years.

An infusion of calcium borogluconate may produce a miraculous effect in a cow with milk fever, but few medical disorders have such relatively uncomplicated aetiology and even less such a simple and effective therapy. Osteochondrosis seems to be on the other end of the spectrum. To start with, there was uncertainty about which articular fragments are the result of OC and which not [3,4]. It took a while before the dynamic character of the disorder became well-recognised, and it is now known that lesions present at a young age can resolve without leaving any trace [5,6]. This feature makes the phenotype of the disorder age-dependent, and in turn this has huge implications for selective breeding [7].

There has been debate about the relative influence of environmental and genetic factors on the manifestation of the disease with reported heritability estimates ranging from 0.0 [8] to 0.52 [9]. Despite earlier hopes that 'the OC gene(s)' would be found, there is now consensus that the genetic basis of OC is polygenic and highly complex. The review by Naccache *et al.* [10] in this issue of *EVJ* describes quantitative trait loci (QTLs) shown to be significantly linked to OC in genome-wide studies on not less than 20 of the 33 chromosomes of the horse. A further complicating and most likely related issue is that the genetic contributors to OC may vary with OC at different locations and even in different manifestations of OC at the same location [11]. Nevertheless, some of the QTLs are more prominent and more frequently found than others and many of the genes involved are related to function and pathways relevant for cartilage or bone metabolism. Therefore, this research is highly relevant for better understanding the disorder and may also help in genomic selection.

A similar variety exists in the environmental factors that have been implicated in the aetiology of OC. These include nutritional factors that can grossly be divided into mineral imbalances and excessive energy intake. Copper has been investigated most extensively [12], it was once believed to be the decisive element in OC but is now regarded as more important in the repair process rather than lesion pathogenesis [13]. Excessive energy intake, especially in the form of easily digestible carbohydrates, has been related to osteochondrosis [14,15] and thought to act via disturbance of hormonal balance, or possibly through the accelerating effect of high energy intake on the animals' growth rate [16]. It is now recognised that rather than growth rate per se, the occurrence of growth spurts during certain windows of vulnerability in the development of joints is important. These vulnerability windows vary for each joint and most likely, also by site within a given joint.

Overall, there has been considerable progress in the understanding of the background of those seemingly simple intra-articular fragments over the last 50 years. It has become clear that OC is comparable in its highly complex character with many other disorders in veterinary medicine that have a large impact on health and welfare, but that appear to be sometimes excessively multifactorial and often heterogeneous in nature, such as (equine) metabolic syndrome, osteoarthritis and many forms of cancer. These disorders will remain challenging for a long time to come and no 'one size fits all' solutions can be expected.

There is another reason to consider OC when we reflect on the achievements of fifty years *Equine Veterinary Journal*. Whereas OC is inherent to the horse (and some other species including humans), its clinical relevance is man-made. In a study conducted in feral horses, which had not known other than natural selection for a long time, OC was present indeed. However, prevalence in the talocrural joint did not exceed 2.5% and was nil in the femoropatellar joint [17]. In the general equine population, the prevalence of osteochondrosis differs per breed of horse, but is much higher in virtually all populations [18]. In pony breeds, which have never been selected for similar traits as racehorses and sport horses, OC was and still is virtually absent [19]. These facts strongly suggest that OC has been inadvertently introduced by selecting for other, desired traits. One of these traits is without doubt height at the withers. Of the Dutch Warmblood, it is known that it has become taller at a rate of approximately 1 mm per year over the past decades. Although this seems very little, it is still an inch in 25 years. If two-hands tall Eohippus or Hyracotherium, the browsing forest-dweller with which equine evolution started some 65 million years ago, had evolved at this speed, the horse would now have stood a staggering 40 miles at the withers. *Equine Veterinary Journal* has published over 60 papers on osteochondrosis in the past 50 years. It has also published scores of papers on exercise-induced pulmonary haemorrhage (EIPH), pathogenesis and epidemiological features of distal metacarpal fractures and carpal chip fractures in racehorses, equine metabolic syndrome, laminitis and colic. These are all disorders to which the equine species is susceptible, but which have become clinical problems with substantial impact on welfare of substantial numbers of horses because of either the use humans make of these animals, or the way they keep them. From this viewpoint it can be stated that it is less the horse itself, but the man-made drivers in the equine industry that create the bulk of work for the equine veterinarian. We now live in an era in which the right of the human being to deal at will with animals is heavily challenged. In the veterinary world this is very clear in the increasing public opposition against certain dog breeds where excessive selection for certain desired traits has led to animals in which physiological frontiers have been crossed. We are not that far in the equine industry, but that industry is under increasing scrutiny too and some of the drivers in the industry will not stand the test of public opinion. Although it is natural for equine practitioners to take the side of the industry that employs them, in the longer term it may be more rewarding to opt for a more independent, balanced and open-minded attitude that acknowledges changes in society and does not a priori just follow where the industry goes. In the end such attitude will even benefit the horse, the equine practitioner and the equestrian industry.

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