CHAPTER I
INTRODUCTION

Joint disease is a common cause of lameness in horses and a major cause of loss of function of horses engaged in athletic activities (1-2). In Great Britain, a study reported in 1985 found that the greatest cause of attrition of young Thoroughbreds from racing was lameness, and among these hoses joint disease figured prominently (2).

Trauma – related disease of joints is a very common in athletic horse. Traumatic joint injury may range from mild, to severe catastrophic injuries with complete loss of support for the joints. Joint disease includes a number of conditions resulting from trauma such as arthritis, synovitis, capsulitis, bone fracture, ligamentous tearing and osteoarthritis (3). From previous studies, they were found that thoroughbred racehorses had 81% forelimb injuries (4) and 90% of injuries caused by bone fracture in the forelimb which proximal sesamoid bones and 3rd metacarpus fractures were most common (5). In other study, it was found that 44.8% of warmblood sport horses died or were culled due to degenerative joint disease and 16.5% were from fractures (6).

In this thesis, the author emphasizes in three types of joint disease. The first, arthritis is a nonspecific term, may be defined simply as inflammation of a joint (7). The second, intraarticular fracture or osteochondral (chip) fragmentation (OC) is a common affliction of racehorses and can predispose the affected joints to arthritis (8). Chip fractures occur most commonly in the carpal (commonly called knee) and fetlock joints. Cases also occur in the pastern, coffin and hock joints but are much less common. Chip fractures involve the articular surface, which means that they pass through the articular cartilage and disrupt the smooth surface. The physical disruption of articular surface provides a physical defect in the joint. The fracture line also releases bone debris that causes synovial inflammation (synovitis). The chip fragments are usually still attached to synovial membrane and their movement within the joint causes direct tugging on the synovial membrane, which has been demonstrated as very painful. The horse therefore has pain directly from the site of the fracture (both synovial membrane tugging and nerve endings in the fractured bone), as well as due to inflammation of the synovial

membrane. The presence of a physically disrupted surface causes direct damage to the opposing articular surface as well as release of inflammatory mediators and catabolic enzymes to synovial fluid, which can contribute to the development of permanent osteoarthritis (9). The last, osteoarthritis (OA) is also called degenerative joint disease (DJD), which represents a group of disorders characterized by deterioration of the articular cartilage (10). Once damage, articular cartilage can not repair itself. The damage progresses and DJD has developed, which can not be corrected and often leads to premature retirement or humane destruction of the horse. Therefore, therapeutic objectives for OA are to limit inflammation and to prevent the release of destructive inflammatory mediators early in the course of the disease, before joint damage progresses to irreparable destruction of cartilage (11). So the success of therapy depends on early diagnosis. At present, Diagnosis of OA in horse can be performed by using several techniques such as physical examination, radiography, nuclear scintigraphy and magnetic resonance imaging (MRI). Although, MRI has also significant diagnostic potential in case of osteoarthritis in horse but it is expensive and not yet feasible for routine use in Thailand. For arthroscopy, this technique allows direct visualization of the cartilage and recently has become the gold standard for assessing cartilage lesion in vivo, but it is more invasive than radiography and MRI (12). In general, the radiography is used together with physical examination for diagnosis of OA; however, the early stage of cartilage degradation and osteoarthritis are difficult or impossible to define diagnostically. This coincide with the result from one study, there was no correlation between pathologic changes and clinical significance (13). And unfortunately, by the time of osteoarthritis are recognizable radiographically, the structural alterations in the articular cartilage are already irreversible (14).

Therefore, over the past decade, researchers have developed techniques to identify and quantify metabolic products of the articular cartilage. Attempts have been made to correlate elevated levels of synovial fluid and serum markers with the stage of joint disease and to elevate their usefulness in early diagnosis and in monitoring therapy. Monoclonal and polyclonal antibodies targeted to epitopes specifically located on

cartilage proteoglycan and collagen fragment present in the synovial fluid and serum have been tested as a more specific and sensitive tool for studying articular cartilage metabolism and pathology (15).

The purposes of this study reported here were 1) to compare the chondroitin sulfate epitope in serum between normal horses and horses with arthritis, osteochondral (chip) fracture or osteoarthritis. 2) to compare serum chondroitin sulfate epitope in horse with osteochondral fracture prior to and after treatment by arthroscopic surgery and 3) to compare this epitope in synovial fluid between abnormal and contralateral normal joints in horses with osteochondral fracture or osteoarthritis by using monoclonal antibody WF6, which produced and developed from Bone and Joint Research Laboratory, Faculty of Medicine, Chiang Mai University