Chapter I
INTRODUCTION

Osteoarthritis (OA) is the number one musculoskeletal disorder in the developed world and it ranks among the top problems of the health care systems in developed countries. Osteoarthritis is the most common disorder of the synovial joints in middle aged and older people (Buckwalter, J. A., Martin, J. A., & Reginster, J. Y., 2002). Up to 10% of the world population suffers from osteoarthritis, and it has been estimated that more than 50% of those 50 years of age and over are affected (Reginster, 2002).

Osteoarthritis (OA) is a common disorder of synovial joints (Dieppe and Lohmander, 2005). It is been increasingly discussed in the healthcare field due to rising incidence in our aging population. As reiterated by Buckwalter, J. A., Martin, J. A., & Reginster, J. Y. (2002), Osteoarthritis is the most common disorder of the synovial joints in middle aged and older people. This condition is strongly age-related, being less common before 40 years, but rising in frequency with age, such that most people older than 70 years have radiological evidence of Osteoarthritis in some joints.

Up to 10% of the world population suffers from Osteoarthritis, and it has been estimated that more than 50% of those aged over 50 years are affected (Reginster, 2002). It is now estimated that more one third of people over 45 years complain of Osteoarthritis related symptoms (Simanek, V., Kren, V., Ulrichova, J., & Gallo, J., 2005). It has also been noted to be a source of great morbidity, impaired quality of life
in affected individuals as well as significant burden to any health care system (Buckwalter, J. A., Martin, J. A., & Reginster, J. Y., 2002).

Osteoarthritis is a disease of the whole joint whose primary role is to provide stability and mobility to the skeleton. Synovial joints are most frequently affected by Osteoarthritis (Gerwin, N., Hops, C., & Lucke, A., 2006). This joint disease is characterized by localized degradation of the articular cartilage. The chondrocytes, which are the cartilage cells are known to play a key role, not only in the destructive process, but also in the repair response. It has become apparent that anabolic and catabolic mediators released from chondrocytes themselves or from other joint cells, drive both the degradation and repair process in the osteoarthritic joint (Van Der Kraan., Peter, M., Van Den Berg, & Wim, B., 2000).
In Osteoarthritis, maturing of the regenerated cells (chondrocytes) proceeds defectively. Therefore, the regenerated chondrocytes lose their ability to differentiate into hyaline cartilage (Chikladze and Chkhaidze 2005). Hyaline cartilage, the most abundant form of cartilage is a unique biological material that forms at the articular surface of joints and is to a large extent responsible for the almost frictionless movement of the articulating surfaces on each other (Gerwin, N., Hops, C., & Lucke, A., 2006).

Apoptotic chondrocyte death occurs more frequently in osteoarthritis compared to normal cartilage (Kim et al., 2000). Within the damaged region of osteoarthritis cartilage, most cells might first trigger a supposed recovery mechanisms. However, this is shortly followed by chondroptosis (apoptosis of chondrocytes) since repair mechanism is mostly unsuccessful (Perez, H. E., Luna, M. J., Rojas, M. L, & Kouri, J. B., 2005).

The cellular morphology of the different types of cartilages in conjunction with meniscal cartilage (in the knee for example) serves as shock absorbers at joints. Their degradation therefore leaves no cushioning or reduced cushioning at the joint of individuals with the osteoarthritic condition. Hence bare bones are left to grind against each other; which are believed to be the source of the mechanical pain. Nearly all people with knee osteoarthritis have meniscal tears and all these are not necessarily the cause of increased symptoms (Bhattacharyya, T., Gale, D., Dewire, P., Totterman, S., Gale, M. E., & Laughlin, S., 2003).

While osteoarthritis is most commonly initiated by host of factors such as abnormal biomechanical forces acting on the joint and the disease progression which
is mediated by dysregulated metabolic events. There is also an overwhelming influence of chemokines and pro-inflammatory cytokines (Pelletier, J. P., Martel-Pelletier, J., & Abramson, S. B., 2001). Pro-inflammatory cytokines, particularly interleukin 1b (IL-1b) and tumor necrosis factor alpha (TNF-), can auto-catalytically stimulate their own production as well as induce chondrocytes to produce additional inflammatory mediators, such as nitric oxide, and a variety of eicosanoids, such as prostaglandin E2 (PGE2) and leukotriene B4 (LTB4) (Kobayashi et al, 2005).

As the disease progresses, the subchondral bone undergoes profound changes which ultimately can affect the bone density. Bone remodeling and maintenance of normal synovial joint structure and function requires regular joint use and lifelong maintenance of weight-bearing activities. In Osteoarthritis, abnormal bone growth which are known as osteophytes become prominent. Osteophytes can lead to pockets of bone spur with the cartilage tissue. As a result, this causes thinning of the cartilage
and eventual exposure of smooth dense bone on the articular surface. As a result, hyaline articular cartilage is lost, and further causes capsular stretching and weakness of periarticular muscles surrounding the joints (Felson D.T., 2006).

Osteoarthritis is characterized pathologically by focusing on several areas of damage to the articular cartilage. This includes load-bearing areas, new bone formation at the joint margins (osteophytosis), changes in the subchondral bone, variable degrees of synovitis, and thickening of the joint capsule (Mainil-Varlet et al., 2003). Osteoarthritis is classified into two broad categories which include primary and secondary Osteoarthritis (Sharma L., 2001). Primary osteoarthritis also known as Idiopathic Osteoarthritis results from changes over time often linked to factors such as age, obesity, and a family history of osteoarthritis. The most common cause of osteoarthritis of the knee is age. Almost everyone will eventually develop some degree of osteoarthritis. However, several factors increase the risk of developing significant arthritis at an earlier age.
Causes of osteoarthritis:

**Weight**: Weight increases pressure on all the joints, especially the knees. Every pound of weight we gain adds 3 to 4 pounds of extra weight on our knees.

**Age**: The ability of cartilage to heal decreases as a person gets older.

**Heredity**: This includes genetic mutations that might make a person more likely to develop osteoarthritis of the knee. It may also be due to inherited abnormalities in the shape of the bones that surround the knee joint.

**Gender**: Women who are 55 and older are more likely than men to develop osteoarthritis of the knee.

**Repetitive stress injuries**: These are usually a result of the type of job a person has. People with certain occupations that include a lot of activity that can stress the joint, such as kneeling, squatting, or lifting heavy weights (55 pounds or more), are more likely to develop osteoarthritis of the knee because of the constant pressure on the joint.

**Athletics**: Athletes involved in soccer, tennis, or long-distance running may be at higher risk for developing osteoarthritis of the knee. That means athletes should take precautions to avoid injury. However, it's important to note that regular moderate exercise strengthens joints and can decrease the risk of osteoarthritis. In fact, weak muscles around the knee can lead to osteoarthritis.

**Other illnesses**: People with rheumatoid arthritis, the second most common type of arthritis, are also more likely to develop osteoarthritis. People with certain metabolic disorders, such as iron overload or excess growth hormone, also run a higher risk of osteoarthritis.
In primary Osteoarthritis, normal aging and wear and the tear of the tissue is responsible for the degeneration of articular cartilage and alterations in single or both joints. No specific inflammatory or metabolic condition known to be associated with arthritis is present in primary Osteoarthritis. There is also no history of specific injury or trauma related to the presence of primary Osteoarthritis. Primary osteoarthritis is mostly common with the joints of the hands, knees, hips, cervical and lumbar vertebrae and the metatarsophalangeal joint of the great toe.

In secondary osteoarthritis the condition is either injury related which occurs in isolated joints. Conditions that cause damage to cartilage may be present, such as: Inherited diseases of iron, calcium, or copper storage, such as hemochromatosis, hyperparathyroidism, or Wilson's disease all contribute to the development of secondary osteoarthritis. Other conditions include neurologic disorders that result in the loss of nerve function; congenital diseases that cause an imbalance in the joints; as well as bone disorders that affect joints. History of injury to joints, such as fractures and tears, or history of trauma to joints, such as repetitive heavy lifting or kneeling are also contributory factors of secondary osteoarthritis.

Osteoarthritis can affect any synovial joint. It also affects all the structures within a joint. Idiopathic osteoarthritis rarely occurs in the ankle, wrist, elbow and shoulder, but it is common in the hand, foot, knee, spine and hip joints (Cooper, 1998). Clinically, osteoarthritis manifests in the form of gradual development of joint pain, joint stiffness and crepitus with motion, joint effusions, and limitation of movement in the joints.
Generally the pain of knee osteoarthritis is usually related to activity (Felson, 2006). The source of pain is not particularly well understood and is best framed in a bio psycho social framework (Dieppe and Lohmander, 2005). It is caused by aberrant local mechanical factors in the joint with within the context of systemic susceptibility (Hunter and Felson, 2006).

Much as cartilage breakdown is one of the classical features of osteoarthritis; of the local events in the joint, loss of cartilage probably does not contribute directly to pain as it is a neural and does not have nerve ending to conduct the pain. On the other hand, the sub chondral bone, periosteum, synovium and joint capsule are all richly innervated and could be the source of nociceptive stimuli and pain in osteoarthritis (Hunter and Felson, 2006).

The natural history of knee osteoarthritis is highly variable, with the disease improving in some patients, remaining stable in others and gradually worsening in others (Felson, 2006). Cartilage loss in knee osteoarthritis is a multi-factorial process that is influenced by systemic risk factors such as age, sex, and obesity and by local mechanical factors such as misalignment in the joint and possible injury as a result.

The most common clinical complaint of individuals with knee osteoarthritis is joint stiffness and pain around the affected joints along with some limitation of function. This stiffness is believed to result from altered features of the components of the knee joint responsible for biomechanics of the joint. Although inflammation is a classical feature of osteoarthritis, it does not become pronounced until the advanced stages of the disease. It is characterized by night pain, which reflects either severe symptomatic disease or pain from causes other osteoarthritis such as inflammatory
arthritis, infection or crystal disease (Felson, 2006). A frequent sign of knee osteoarthritis is crepitus which is a grinding sensation within the joint. It is felt on passive range of motion due to irregularity of opposing cartilage surfaces.

Much as radiographs have been and still are useful for confirming the diagnosis of the disease, findings are found to be non specific and have been have highly questioned by researchers and clinicians. Some say radiographs may reveal clues of missed diagnosis (Felson, 2006) whereas others say that radiographic findings correlates poorly with the severity of the pain and that radiographs may even be normal in persons with disease (Hannan, M. T, Felson, D. T., & Pincus, T., 2000).

The two most common radiographic features of osteoarthritis include narrowing of the joint space and the presence or development of osteophytes or new bones and sometimes changes in subchondral bone. These common features only get visible in plain radiographs when the disease is in its advanced stage (Hunter and Felson, 2006).

Magnetic resonance imaging (MRI) is likely to reveal changes that indicate the presence of osteoarthritis, but it is not suggested in the workup of older persons with chronic knee pain. MRI findings of osteoarthritis, including meniscal tears, are
common in middleaged and older adults (Bhattacharyya, T., Gale, D., Dewire, P., Totterman, S., Gale, M. E., & Laughlin, S., 2003) with and without knee pain. A clinical review by Hunter and Felson, 2006 even stated that MRI may be used to facilitate other causes of knee pain that can be confused with knee osteoarthritis such as osteochondritis dissecans and avascular necrosis.

Like many other chronic diseases, osteoarthritis has risk factors that contribute to the development of osteoarthritis. These risk factors range from genetic to environmental factors. Risk factors for the development and progression of osteoarthritis include local biomechanical factors like obesity, joint injury, joint deformity, and extensive sport participation, as well as systemic factors including age, gender, ethnic characteristics, bone density, and estrogen deficiency (Gerwin, N., Hops, C., & Lucke, A., 2006; Svanborg, A., Bagge, E., & Brauner, D., 1993).

One of the strongest consistent risk factors associated with osteoarthritis is aging, but this may not be a key factor in development of osteoarthritis because of slowing down of process of osteoarthritis at ages above 75 (Svanborg, A., Bagge, E., & Brauner, D., 1993). Occupational kneeling and squatting and previous knee surgery is another set of risk factors for Knee osteoarthritis.

A finding from a Framingham Heart Study confirms that heavy physical occupation and leisure activity particularly in obese people predisposes them to subsequent osteoarthritis of the Knee (Madhok, R., Kerr, H., & Capell, H. A., 2000).

thickness chondral defects particularly femoral and patellar. This study concluded that osteophytes, high levels of type II collagen c-telopeptides (CTX-II) and the issue of body composition and body weight were found to have a role in knee osteoarthritis (Zhai, G., Cicuttini, F., Ding, C, Scott F, Garnero P, Jones G., 2006). Similar observations were made by Frankenburg and Zanarini, 2006 where obesity was a major factor in relation to the presence of osteoarthritis.

The management of osteoarthritis like many other chronic diseases cannot be achieved without considering general lifestyle changes. Numerous dietary factors have been noted in observational and laboratory studies to be linked with the cause of osteoarthritis. This includes vitamins A, C, E and D as well as boron (Hunter, D. J., & Felson, D. T., 2006).

These dietary factors have been to prevent cartilage degradation associated with osteoarthritis through four different mechanisms. This includes protection from oxidative damage, modulation of inflammatory response, facilitating cellular differentiation, and biological actions related with bone and collagen synthesis (Sowers M.F., 2001).

Antioxidants which are classically known to protect cells from lipid peroxidation could play an important role in preventing cartilage degradation in the joints as it is known to do in other tissues of the body. Chondrocytes can produce hydrogen peroxide and superoxide anions which can adversely affect the collagen structure and depolymerize synovial fluid hyaluronate (Hunter, D. J., & Felson, D. T., 2006; McAlindon, T. E., & Biggee, B. A., 2005).
The body’s defense against auto-catalytic peroxidation by these reactive oxygen species in the cells can be enhanced by dietary intake of antioxidants and some micronutrients. The concept of extracellular defense has led to the hypothesis that high dietary intakes of these micronutrients might protect age related disorders, including osteoarthritis (Hunter, D. J., & Felson, D. T., 2006).

Much as antioxidants and certain vitamins have been linked to preventing oxidative damage to the joint, very little is known about the tissue distribution and bioavailability of these factors especially vitamins within the joints (McAlindon, T. E., & Biggee, B. A., 2005). Another Felson et al., (2007) study, researchers singled out vitamin D and concluded that vitamin D status is unrelated to the risk of joint space or cartilage loss in knee osteoarthritis. Non-pharmocological options of treatment of osteoarthritis are gaining popularity (Singh G., 2003). In this light two most popular lifestyle approaches aimed at fighting osteoarthritis are losing weight in obese adults and or to incorporate exercise to improve health quality of life and positively impact body composition (Van Gool, C. H, et al., 2006).

Therapeutic exercises are known to reduce pain, increase muscle strength, increase range of motion, increase endurance and aerobic capacity and improve physical function and quality of life (Singh G., 2003; Balint and Szebenyi, 1997). At the joint level, exercise is known to be advantageous in increasing synovial fluid circulation, thereby providing nutrients to the articular cartilage which helps maintain periarticular muscle strength. A two-year clinical trial on home-based exercise in knee osteoarthritis patients showed the exercise arm of the trial had highly significant reduction in pain compared to the control group (Thomas et al., 2003).
Similar to treatment of other chronic diseases, complementary or alternative therapies have been commonly resorted to in the treatment of osteoarthritis. It is important that health-care providers and patients are aware of the evidence for or against these approaches (Schumacher H.R., 2004).

Evidence in support of other alternative therapies for osteoarthritis has been weak or contradictory such as in the case of homeopathy, magnet therapy, tai chi, leech therapy, music therapy, yoga, imagery and therapeutic touch (Ernst, 2006). Other approaches include intra-articular hyaluronate (injection) and the use of alternative therapies such as acupuncture or glucosamine have featured prominently in the treatment and management of osteoarthritis (Schumacher, 2004).

Complementary or alternative therapies have generated sufficiently promising results to warrant further investigation in large-scale, definitive, randomized clinical trials (Ernst E., 2006). In treating osteoarthritis, nutraceuticals such as glucosamine and chondroitin sulfate, two of the molecular building blocks found in articular cartilage, are the most commonly used alternative supplements. In randomized trials of variable quality, these compounds show efficacy in reducing symptoms, but neither has been shown to arrest progression of the disease or regenerate damaged cartilage (Ernst E., 2006). Several studies have found glucosamine and chondroitin sulfate efficacious in treating pain associated with osteoarthritis (Herrero-Beaumont, G., Rovati, L. C., Castaneda, S., Alvarez-Soria, M. A., & Largo, R., 2007; Clegg, D. O., Reda, D. J., Harris, C. L., Klein, M. A. et al., 2006).
A study involving obese older adults with knee osteoarthritis concluded that although dietary weight-loss-only and exercise-only interventions were effective in improving selected pain outcomes in osteoarthritis, it is clearly apparent that the combined intervention had the most consistent, positive, and longer lasting effect on the outcomes related to osteoarthritis (Rejeski, W. J., Focht, B. C., Messier, S. P., Morgan, T., Pahor, M., & Penninx, B., 2002). Hence the purpose of this investigation was to explore the combined benefit of diet and physical activity in the management of pain associated with osteoarthritis.

**Statement of the Problem**

Keeping the above concepts, the purpose of this study was to find out the effects of diet modification with and without home based exercise programme on self-reported knee pain osteoarthritis.

**Research Questions**

1. Would the diet modification with and without home based exercise programme improve the selected dependent variables (anthropometric measurements, knee pain and range of motion) while the presence of covariate (control)?

2. Would the diet modification with and without home based exercise programme differs each other and also with the control group while improving the selected dependent variables such as anthropometric measurements, knee pain and range of motion?

**Assumptions**

Validity of this study will rely on the following assumptions
1. The subjects made an honest effort to comply with the intervention protocol according to their specific training.

2. The subjects were performed the two different training sessions separately, for three alternative days per week.

3. The subjects complied with the best of their ability to the training and testing direction.

4. The subjects were tested accurately by standardized test items.

5. The selected tests were reliable and valuable for assess the selected variables.

6. Subjects were not performed any vigorous exercise during the course of study.

**Hypotheses**

It has been scientifically accepted that any systematic training over a continuous period of time would lead to produce changes on dependent variables. Based on this concept and the research questions the following hypotheses were formulated and tested at 0.05 level of confidence.

1. There would be significant improvement on reducing symptoms of self reported knee pain associated with osteoarthritis (weight, fat percentage, pain intensity and flexibility of knee joints) due to the effects of diet modification with and without home based exercise programmes.

2. There would be significant difference among the two experimental groups while improving the selected variables (weight, fat percentage, pain intensity
and flexibility of knee joints) due to the effect of diet modification with and without home based exercise programme.

**Delimitations**

The study was delimited to the following factors.

1. A total of forty subjects were participated voluntarily from Kanyakumari District, Tamil Nadu, India.

2. The participants were divided into two group namely diet-modification only and diet-modification with exercise only.

3. The following dependent variables were selected for this study such as weight, fat percentage, pain pattern, high pain intensity, current pain intensity, usual pain intensity, left and right leg extension, and left and right leg flexion.

4. The standardized tests were used to collect relevant data on the selected dependent variables.

5. The duration of training period was restricted to 12 weeks and the number of sessions per week was confined to three.

6. The level of significance was fixed at .05 levels, which was considered to be appropriate.

**Limitations**

Looking at the design of this study and the outcomes of the research, certain modifications to follow-up studies which can be suggested to address the limitations present in this study. These include:
1. Inclusion criteria and selection bias because the Osteoarthritis is more prevalent.

2. Short duration of the study.

3. The study was conducted over the course of 12 weeks and will not be able to control for variations in the environmental conditions and the level of acclimatization.

4. The background of the previous training was not taken into consideration.

5. Rest periods were not standardized for all subjects.

6. The researcher cannot control all outside activities, like social habits of the participants.

7. Though the participants were motivated verbally, no attempt was made to differentiate motivation level during the period of training and testing.

**Significance of the Study**

1. The ultimate aim of research in physical education is to help coaches and physical educators to treat their sports persons from knee injury.

2. A unique aspect of this work is that it includes recommendations for the practical use of research findings.

3. The study would add knowledge in the area of Sports training and Sports Medicine.

4. This study will add the quantum of knowledge in knee osteoarthritis for the players to take correct diet and physical activity.

5. The present study will help the fitness trainer and health professionals to treat the person with knee osteoarthritis.
Definition of the Operational Terms

Arthritis

Arthritis is a joint disorder causing pain, stiffness and inflammation of one or more joints (Brandt, K. D, 2000).

Osteoarthritis

Osteoarthritis, which is also known as degenerative joint disease (DJD), is a progressive disorder of the joints caused by gradual loss of cartilage and resulting in the development of bony spurs and cysts at the margins of the joints (Berger, R. G, 2001).

Diet

Diet is the sum of food consumed by a person or other organism. Dietary habits are the habitual decisions an individual or culture makes when choosing what foods to eat (“Diet is the”, 2009).

Weight

Body weight is described as the mass of an organism's body and it was measured in kilograms (Sutcliffe and Ganham, 1981).

Body Fat Percentage

Body Fat Percentage is a measure of the amount of subcutaneous fat, obtained by inserting a fold of skin into the jaws of a caliper. The skinfolds are usually measured on the upper arm, thigh, or upper abdomen, and the caliper measurements are later compared with precalibrated standard tables to assess an individual's body fat content indirectly (“Body Fat Percentage”, 2013).
**Pain Status**

McGill Pain Questionnaire is used for both assessment and monitoring of pain status. It provides quantitative measures of clinical pain that encompass its sensory, affective and other qualitative components and allows statistical analysis of data collected ("McGill Pain Questionnaire", 2011).

**Knee Flexion**

Knee flexion is defined as bending the joint resulting in a decrease of angle; moving the lower leg toward the back of the thigh ("Knee Flexion", 2013).

**Knee Extension**

Knee extension is defined as straightening the joint resulting in an increase of angle; moving the lower leg away from the back of the thigh ("Knee Extension", 2013).

**Summary of the Chapters**

This thesis consists of five chapters. The title was introduced in the first chapter with the statement of the problem, hypothesis, significance of the study and definitions of the terms are discussed. The next chapter describes the sources of review of related literature, Selection of subjects, variables, units, experimental design, statistical procedure and the effect of various training methods are given in the third chapter. Analysis of data and discussion on findings and discussion on hypothesis are highlighted in the fourth chapter. The thesis is concluded with summary, conclusions with suitable recommendations, which will be useful for further investigation.