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March, 2007


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Abstract

Gender-Based Effects of Diet Modification in Combination with an At-Home Exercise Regimen on Pain and Joint Mobility in Knee Osteoarthritis

By
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Background: 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 estimated to cost western countries such as Australia, Canada, United Kingdom and France as well as Western Europe as high as 1-2.5% of the gross national product (GNP) of their economies. This figure is much higher in the United States where the CDC puts the total direct and indirect costs of arthritis and other rheumatic conditions in the US during 1997 at $86.2 billion, or 1% of the US gross domestic product. It is projected that by the year 2020; approximately 18.2% of Americans will be affected by arthritic disorders equaling 60 million people. The current price related to OA to the US economy is sad to say even higher and growing by the minute.

Methods: A total of 63 subjects were randomly assigned to either the dietary modification only group or the dietary modification and exercise regimen group for a period of three months. Subjects made two visits to the assessment laboratory where anthropometric measurements, range of motion, and pain assessment were made. Additionally, subjects completed dietary, supplement use, and physical activity questionnaires. Subjects received relevant counseling on diet modification and incorporation of an at-home exercise regimen.

Results: Baseline body weight and fat mass was higher in the nutrition alone group compared to nutrition and exercise group in both genders. No changes were seen in either of the two values after treatment in the two treatment groups. There was a significant reduction (p-value > 0.029) in current pain intensity from baseline to final. There were also reductions in all pain parameters assessed across both genders. After three months of intervention, nutrition and exercise intervention had more favorable effect (an improvement in knee range of motion) on extension than nutrition alone intervention group and that the effect of the combined intervention was significant (P= 0.041).

Conclusion: Our study findings indicate that improvement in range of motion along with reduction in the level of pain can be achieved by individuals with knee pain associated with OA who modify their lifestyles by incorporating exercise coupled with dietary behavioral changes.
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**Table of Contents**

**Introduction** 1

**Literature Review** 4
- Epidemiology and Prevalence 4
- Classification of Osteoarthritis 6
- Composition of Cartilage 7
- Changes in the Diseased Joint 8
- Osteoarthritis of the Knee 11
- Estrogen and Osteoarthritis 14
- Biochemical Markers in Osteoarthritis 16
- Nutrition, Exercise and Osteoarthritis 18
- Surgical Treatments for OA 20
- Pharmacological Treatments for Osteoarthritis 22
- Alternative therapies for Osteoarthritis 25

**Methods** 28
- Subject recruitment and selection 28
- Screening 28
- Data collection methods 29
- Treatment protocol 31
- Statistical analysis 32

**Tables** 34
**Results** 46
**Discussions** 49
**Limitations** 52
**Recommendations and Future Directions** 53
**References** 54

**Appendix** 65
- Medical history questionnaire 66
- Physical activity questionnaire 70
- Pain assessment questionnaire 73
- Food frequency questionnaire 81
- Supplement use questionnaire 89

**Tables** 34
- Table 1: Characteristics of Study Participants (General) 34
- Table 2: Gender Classification of Anthropometric Measurements (Males) 35
- Table 3: Gender Classification of Anthropometric Measurements (Females) 36
- Table 4: Characteristics of Study Participants (Gender Specific) 37
- Table 5: Correlations among Anthropometric Measurements 38
- Table 6: Correlations among Anthropometric Measurements –Intent to 39
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. OA is the most common disorder of the synovial joints in middle aged and older people (Buckwalter et al; 2004). Up to 10% of the world population suffers from OA, and it has been estimated that more than 50% of those 50 years of age and over are affected (Reginster, 2002). It is projected that by the year 2020; approximately 18.2% of Americans will be affected by arthritic disorders equaling 60 million people (CDC, MMWR, 1994; Lawrence et al.; 1998).

Osteoarthritis is estimated to cost western countries such as Australia, Canada, United Kingdom and France as well as Western Europe as high as 1-2.5% of the gross national product (GNP) of their economies (March et al; 1997). This figure is much higher in the United States where the CDC puts the total direct and indirect costs of arthritis and other rheumatic conditions in the US during 1997 at $86.2 billion, or 1% of the US gross domestic product (CDC, MMWR 2004). The current price related to OA to the US economy is sad to say even higher and swelling by the minute.

Obesity is a well recognized and modifiable risk factor associated with knee OA. The risk factor for OA increases by 15% for each additional unit of body mass index above 27 (Sowers, 2001). There are numerous biologically plausible mechanisms through which nutritional factors might favorably influence pathophysiological processes in OA (McAlindon and Biggee 2005). In general, nutrition and exercise have long been touted
as the first line of treatment in the management of OA (O’Reilly et al., 2001). Among obese women, functional improvement correlated with weight loss and walking, encouraging continued emphasis on weight loss for managing knee OA (Martin et al., 2001) in another study. A study involving obese older adults with knee OA concluded that although dietary weight-loss-only and exercise-only interventions were effective in improving selected pain outcomes in OA, it is clearly apparent that the combined intervention had the most consistent, positive, and longer lasting effect on the outcomes related to OA (Rejeski et al., 2002). Hence, we conducted a short-term study in subjects with self-reported knee pain associated with OA. The purpose of this investigation was to explore the combined benefit of diet and physical activity in the management of pain associated with OA.

**Study Objectives and Hypothesis**

The central hypothesis of this thesis is to show that the effect of dietary modification in combination with an at-home exercise regimen on functional status of individuals with self-reported knee pain OA is gender based.

**Specific Objectives**

Objective 1: To examine the gender-based differences in pain outcomes when comparing individuals on the combined dietary and exercise treatment in comparison to diet alone.

Objective 2: To examine the gender-based differences in pain outcomes in relation the changes in range of motion in individuals on the combined dietary and exercise treatment in comparison to diet alone.
Objective 3: To access how gender-based differences in pain outcomes at baseline differ in completers and dropouts in individuals on the combined dietary and exercise treatment in comparison to diet alone.
Osteoarthritis (OA) is a common disorder of synovial joints (Dieppe and Lohmander, 2005). The current definition of OA by the American College of Rheumatology “is a heterogeneous group of conditions that leads to joint symptoms and signs which are associated with defective integrity of articular cartilage, in addition to related changes in the underlying bone at the joint margins”.

Osteoarthritis is been increasingly discussed in the healthcare field due to rising incidence in our aging population. Its severity and prevalence is becoming pronounced during earlier decades of lives in the US population. As reiterated by Buckwalter et al; in a 2004, OA 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 OA in some joints (Peterson, 1996). Up to 10% of the world population suffers from OA, 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 OA related symptoms (Simanek, et al, 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, 2004).
Osteoarthritis is the most prevalent form of musculoskeletal disorder in western countries (Reginster, 2002). Much as statistics of its incidence from western countries such as Australia, Canada, United Kingdom and France as well as Western Europe in general are on the rise, the current data of OA incidence in the United States is even more alarming (CDC MMWR 2001). For example, approximately 8% (6 million) of the French population was diagnosed as having OA in the 1990s (Reginster, 2002). Whereas in the United States, a CDC MMWR report states that an estimated 16% of the population or 43 million people had some form of Arthritis specifically OA (CDC 2001). The prevalence of OA is going to continue to soar in the coming decade as the elderly ‘the baby boomers’ generation becomes a larger proportion in the population. With this aging phenomenon, experts have projected that by the year 2020; approximately 18.2% of Americans will be affected by arthritic disorders equaling 60 million people (CDC, MMWR, 1994; Lawrence et al.; 1998). A more recent finding by the CDC states that as the US population ages, the number of Americans ages over 65 years with arthritis is projected to increase from approximately 21.4 million in 2005 to 41.4 million by the year 2030 (CDC, MMWR, 2003).

The toll of OA on the economies of western countries such as Canada, Europe and the US can not be overemphasized. The enormous economic burden of this disease comprises of direct costs such as the cost associated with drugs, medical care, hospitals, research, pensions and benefits and indirect cost such as premature mortality and chronic and short-term disability (Reginster, 2002). The ‘COART’ study in France found that functional impairment often associated with osteoarthritis, indicates that the
The societal and economic burden is huge, similar to that created by coronary heart disease (Le Pen et al.; 2005).

The burden of the disease relates not only to its prevalence, but also to the cost of the disease to the health care system of a country. The annual cost of musculoskeletal disorders has been estimated as up to 1-2.5% of the GNP of the countries studied, including Australia, Canada, France, and the UK (March et al; 1997). In the States for example, the Centers for Disease Control and Prevention (CDC), puts the total direct and indirect costs of arthritis and other rheumatic conditions in the US during 1997 at $86.2 billion, or 1% of the US gross domestic product (CDC, MMWR 2004). These current costs which are bound to increase enormously as the incidence of OA increases with a growing aging population may be grossly be understating the economic burden in the future.

**Classification of Osteoarthritis:**

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). OA is classified into two broad categories which include primary and secondary OA (Sharma, 2001).

Primary osteoarthritis also known as Idiopathic OA results from changes over time often linked to factors such as age, obesity, and a family history of osteoarthritis (Wise, 2004).
In primary OA, 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 OA (Wise, 2004). There is also no history of specific injury or trauma related to the presence of primary OA. Primary OA 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 OA 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 OA. 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 (Wise, 2004). 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 OA.

**Composition of Cartilage:**
Cartilage is a dense connective tissue which is composed of chondrocytes that are dispersed in a firm gel-like matrix. Chondrocytes and the precursor forms of chondrocytes known as chondroblasts arises from the mesenchyma cells are found in cartilage (AHSMD, 2004). Chondroblasts are responsible for the secretion and
maintenance of the matrix. The matrix is mainly composed of proteoglycans, a special type of glycosaminoglycans (Poole, 1999). The most common types are chondroitin sulfate and keratin sulfate. Cartilage is found in the joints, the rib cage, ear, nose, in the throat and between the intervertebral discs. There are three main types of cartilages: hyaline, elastic and fibrocartilage. Morphologically, the articular cartilage has a highly intricate and ordered structure and the integrity of the tissue is maintained by a variety of complex interactions between the chondrocytes and the protein matrix (Poole, 2001). Proteoglycans, which consist of a large number of highly negatively charged polysulphated glycosaminoglycans, are responsible for drawing water molecules into the extra cellular matrix and providing compressive stiffness (Goodrich and Nixon, 2006). Likewise, the anatomical arrangement of collagen fibrils at various depths of cartilage provides structural support, which accounts for tensile stiffness. The collagen fibrils and their arrangement also limit water content within the cartilage. Together, proteoglycans and collagen fibrils are mainly responsible for the resiliency of hyaline cartilage and its inherent biomechanical properties (Goodrich and Nixon, 2006).

Changes in the Diseased Joint:

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 OA (Gerwin et al., 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 (van der Kraan et al., 2000). 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 (Fukui et al., 2001; van der Kraan et al., 2000). In Osteoarthritis, maturing of the regenerated cells (chondrocytes) proceeds defectively. Therefore, the regenerated chondrocytes loss 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 et al., 2006).

Apoptotic chondrocyte death occurs more frequently in OA compared to normal cartilage (Kim et al., 2000). Within the damaged region of OA 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 et al., 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 OA have meniscal tears and all these are not necessarily the cause of increased symptoms (Bhattacharyya et al.; 2003).
The intricate mechanism of progressive cartilage degeneration remains largely unknown. The process however is categorized into three overlapping steps. In step I: Injury, genetics or unknown stimulus disrupts or alter the cartilage matrix. Step II, the chondrocytes in response to the alteration try to repair the damaged cartilage. The final step is the declined ability of the chondrocytes' response to restoring damaged cartilage thus leading to loss of articular cartilage (Fukui, et al., 2001). The new or regenerated cartilage/chondrocytes that is formed is therefore much more fibrous and less able to withstand mechanical stress.

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 proinflammatory cytokines (Pelletier et al., 2001). Pro-inflammatory cytokines, particularly interleukin 1b (IL-1b) and tumor necrosis factor alpha (TNF-α), 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; Abramson and Yazici, 2006).

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 OA, 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, 2006).

**Osteoarthritis of the Knee:**

Osteoarthritis can affect any synovial joint. It also affects all the structures within a joint. Idiopathic OA rarely occurs in the ankle, wrist, elbow and shoulder, but it is common in the hand, foot, knee, spine and hip joints (Copper, 1998). Clinically, OA 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 (Decision resources, 2006). Generally the pain of knee OA is usually related to activity (Felson, 2006). The source of pain is not particularly well understood and is best framed in a biopsychosoical 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 OA; of the local events in the joint, loss of cartilage probably does not contribute directly to pain as it is aneural and does not have nerve ending to conduct the pain. On the other hand, the subchondral bone, periosteum, synovium and joint capsule are all richly innervated and could be the source of nociceptive stimuli and pain in OA (Hunter and Felson, 2006).

The natural history of knee OA 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 OA 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 disalignment in the joint and possible injury as a result (Hunter et al., 2006). The most common clinical complaint of individuals with knee OA 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 OA, 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 OA such as inflammatory arthritis, infection or crystal disease (Felson, 2006). A frequent sign of knee OA 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.

**Diagnosis of Osteoarthritis**

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 et al, 2000). The two most common radiographic features of OA 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 middle-aged and older adults (Bhattacharyya et al., 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 OA such as osteochondritis dissecans and avascular necrosis.

Risk Factors for Osteoarthritis

Like many other chronic diseases, OA has risk factors that contribute to the development of OA. These risk factors range from genetic to environmental factors. Risk factors for the development and progression of OA 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 et al., 2006; Svanborg et al., 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 OA at ages above 75 (Svanborg et al., 1993). Occupational kneeling and squatting and previous knee surgery is another set of risk factors for Knee OA. A finding from a Framingham Heart Study confirms that heavy physical occupation and leisure activity
particularly in obese people predisposes them to subsequent OA of the Knee (Madhok et al., 2000).

An Australian study by Zhai et al., 2006 concluded that knee pain is significantly associated with non-full 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 OA (Zhai et al., 2006). Similar observations were made by Frankenburg and Zanarini, 2006 where obesity was a major factor in relation to the presence of OA. Other research studies on the burden of musculoskeletal defects in as it contributes to increasing the risk of OA have recommended efforts aimed at reducing the burden of musculoskeletal diseases by encouraging exercise and obesity prevention campaigns (Brooks, 2006) related to decreasing the incidence of OA.

**Estrogen and Osteoarthritis:**

Sex hormones have been thought to affect the occurrence and the progression of OA. Several clinical observations have suggested a relationship between OA and a changed estrogen metabolism in menopausal women (Claasen et al., 2006). The issue of the effect of estrogen on OA has however mixed findings to date. While some findings indicate the protective effects of estrogen on OA, others have found no effect of estrogen on OA and with some others propounding even an increased risk (Richette et al., 2003; Parazzini et al., 2003, Von Mullen et al., 2002; Erb et al., 2000). Studies have concluded that, estrogen deprivation at the menopause seems to be associated with
increases in the frequency of knee, hip, and finger osteoarthritis and in the severity of hip osteoarthritis (Richette et al., 2003; Parazzini et al., 2003).

On the other hand, after adjusting for potential confounding factors the odds ratios among hormone therapy (HT) or estrogen therapy (ET) users compared with nonusers was similar (Erb et al., 2000) in a study which concluded that sex hormones were protective. The authors of the Framingham osteoarthritis study concluded that estrogen use in women is not associated with an increased risk of radiographic knee osteoarthritis (Hannan et al., 1990).

On the other hand, estrogen use in postmenopausal women for a period greater than 1 year was significantly associated with an increase in hand osteoarthritis (OR 1.6; 95% CI 1.05–2.3) and hip osteoarthritis (OR 5.0; 95% CI 1.7–4.8) (Von Mullen et al., 2002). Limitations such as detection bias and estrogen users being more likely to visit physicians, age as a confounding factor, and dose response effects in the various studies have been cited (Wluka et al., 2000; Parazzini, 2003) to cloud the conclusion on the issue of the effect of estrogen use in women and the relationship to OA.

In a summary report on estrogen and its link to OA, the American College of Obstetricians and Gynecologists Women’s Health Care Physicians (Obstet Gynecol. 2004) concluded that studies with estrogen or hormone therapy and its relationship to OA are relatively poor in their design in addressing the issue. Most studies have been observational, evaluated osteoarthritis as a secondary outcome, have lacked statistical
power and the use of well-standardized measurement tools and had small sample sizes. The only randomized controlled trial showing no relationship of HT to knee osteoarthritis was limited only to the knee and only to women with known coronary disease, and the study was not powered for the osteoarthritis comparison. While studies are limited, the summary report went on to conclude that there is no compelling evidence that ET has any beneficial or harmful effects on osteoarthritis. From a biological sense, the presence of estrogens receptor in the synovium, of both animals and humans (Nasatzky et al., 1994, Tsai and Liu 1993) may explain that a link between estrogens and OA needs to be further investigated.

**Biochemical Markers in Osteoarthritis:**

Much as physical evidence remains the most paramount diagnostic feature for every human disease, it can not be certainly due to the invasive nature of a full diagnosis of certain conditions. This phenomenon even becomes of a concern due to chronic condition such as OA. Currently, plain weight bearing radiograph evaluation remains the technique used to clinically diagnose OA. This shows a joint space narrowing in the tibiofemoral joint (Felson et al., 2007; Kijowski et al., 2006). Like many other disease diagnostic methods, these radiographic changes are not detectable till the disease process has reached advanced and possibly irreversible tissue alteration stage (Merle-Vincent et al., 2007) thereby defeating important purpose for preventing the condition.

Detection, treatment or management of OA is highly dependent on advances in imaging and biomarkers. Although radiographs are heavily relied on in OA detection, changes
are slow and not uniform among individuals followed in clinical studies, where there is
evidence that patients segregate into slow versus rapid progressors (Kijowski et al.,
2006; Raynauld, et al., 2004). Using the premise of irreparable damage of the tissues of
the joint in OA, a very appropriate substitute for radiography can be the use of and
applicable biochemical. Blood-based proteomic analyses suggest that biochemical
alterations can be observed well before radiologic damage is evidenced in OA
(Garnero, 2006). New cartilage-specific markers, including assays for type II collagen
synthesis and degradation, have been developed (Lorenz et al., 2005; Garnero et al.,
2004 and 2005). Several biochemical markers have been identified that are elevated in
populations of patients with hip or knee OA, including cartilage oligomeric matrix protein
(COMP), hyaluronic acid, c-reactive proteins (CRP), glycoprotein, GP- 39 (serum YKL-
40) and c-terminal cross linking telopeptides of collagen type II (CTXII), (Garnero et al.,
2004 and 2005). Recent prospective studies indicate that blood and urine levels of
these new markers are highly correlated with the presence and progression of joint
damage (Garnero, 2006).

A study by (Bruyere et al., 2006) also found that a single measurement of serum
hyaluronic acid or short term changes in urine CTX-II could identify patients with the
highest risk of osteoarthritis progression. In another study, urinary CTXII was reduced
significantly and in a dose-dependent manner by treatment with risedronate, suggesting
a positive effect of the treatment on OA (Bingham et al., 2004). Since biological markers
respond rapidly to treatment, they will also certainly play an important role in the
development and the monitoring of disease-modifying therapies in OA.
Nutrition, Exercise and Osteoarthritis:

The management of OA like many other chronic diseases can not 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 OA. This includes vitamins A, C, E and D as well as boron (Hunter et al., 2002). These dietary factors have been to prevent cartilage degradation associated with OA 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, 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 collage structure and depolymerize synovial fluid hyaluronate (Hunter et al., 2002; McAlindon and Felson, 1997). 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 OA (Hunter et al., 2002). 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 and Felson, 1997). Another Felson et al., 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 OA (Felson et al., 2007). Non-pharmocological options of treatment of OA are gaining popularity (Singh, 2003). In this light two most popular lifestyle approaches aimed at fighting OA are losing weight in obese adults and or to incorporate exercise to improve health quality of life and positively impact body composition (C.H. van Gool et al., 2006).

The American College of Rheumatology recommends exercise to be a vital component of therapy. 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, 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 (Lane and Buckwalter 1999). A two-year clinical trial on home-based exercise in knee OA patients showed the exercise arm of the trial had highly significant reduction in pain compared to the control group (Thomas et al., 2002).

Obesity is major risk factor of OA, especially for the knee and hip (Andrianokos et al., 2006). People with BMI greater than 30 are four times more likely to have knee OA than people in the normal BMI range. The body exerts three to six times its weight on the knee during a single leg stance in walking. Any increase in weight multiplied by this factor therefore indicates how much excess force is exerted across the knee when an overweight individual walks (Hunter et al., 2002). At the metabolic level, TNF-a, the
interleukins, or leptin produced from the adipose tissues affect the cartilage and the bone thereby predisposing them to OA (Miller et al., 2003).

Weight control therefore has both anecdotally and scientifically been linked to reduction of OA. In a study by Felson and colleagues (Felson et al., 2002), weight loss was shown to reduce the risk of subsequent symptomatic knee. Additionally findings from the Framingham study suggest that a weight loss of 5kg decreases the risk of developing knee OA by 50% (Hunter et al., 2002).

Several studies have also demonstrated positive correlation of BMI and Knee OA severity and incidence (Hansen et al., 2007, Schneider et al., 2007, Focht et al., 2005). With obesity being the strongest modifiable risk factor for the incidence and progression of OA, findings from several researchers have concluded that a combination of exercise and weight loss being the ideal intervention for OA (Hansen et al., 2007, Schneider et al., 2007). A study by Focht and colleagues, stated that physical activity and weight loss should indeed be the intervention of choice in older, overweight, and obese adults with knee OA (Focht et al., 2005)

**Surgical Treatments for OA:**

The purpose of surgical treatment for knee OA is to reduce pain, increase function and improve symptoms overall. Patient satisfaction is a fundamental goal in treating OA (American Academy of Orthopedic Surgeons -AAOS, 2003). There are four main surgical treatments used for OA. These include osteotomy, debridement, arthrodesis and arthroplasty or total knee replacement. Although these surgical procedures remain largely plausible, the choice of a particular procedure depends on the severity and
pattern of pain, functional impairment, anatomical abnormalities, individual patient ability to cooperate with the individual plan, general health of the patient, age, and risk/benefit and cost/benefit ratio (Goldberg, 2001).

An osteotomy may be recommended if damage to the knee cartilage is primarily in one section (compartment) of the knee. An osteotomy also may be recommended if a broken knee does not heal properly. This procedure involves reshaping the bones to improve knee alignment. The joint is repositioned to move the mechanical axis of weight bearing for the limb away from the damaged area. This shifts the weight bearing stresses from the damaged section to a healthier part of the knee. An osteotomy can restore knee function and diminish osteoarthritis pain (AAOS, 2003). It may even stimulate the growth of new cartilage. Although an osteotomy can decrease pain and improve function, the results often deteriorate over the long term. Many people who have an osteotomy will eventually need a total knee replacement [arthroplasty] (AAOS, 2003).

An arthroplasty is a joint replacement procedure. The replacement parts are made of cobalt-chrome or titanium metals and smooth, wear-resistant plastic (polyethylene). The results of total joint replacement are generally excellent. Patients are believed to experience significant pain relief and improved physical functioning. There are some risks to the surgery, and full rehabilitation may take three to six months. In addition, the prosthesis (artificial joint) may eventually loosen or wear out so that a second surgery is needed. However, at the 10-year mark, the success rate with most prostheses today is about 90 percent (Romanwoski, and Recippi, 2002, AAOS, 2003).
Arthroscopy is a surgical procedure that uses small incisions and miniature instruments. A tiny telescope (arthroscope) is inserted into the joint space, which is then filled with fluids to facilitate clear visibility of the components of the joint. Using tiny instruments, damaged cartilage is trimmed to remove any loose particles or debris from the joint (a procedure called debridement) and clean the joint ‘[a process called "lavage" or "irrigation"](AAOS, 2003). Arthroscopy can be helpful if the joint pain results from a tear in the cartilage or meniscus, or if bits of debris are causing problems in bending or straightening the joint. In people under age 55, arthroscopic surgery may help delay the need for more serious surgery such as a joint replacement.

**Pharmacological Treatments for Osteoarthritis:**

Pharmacological interventions remain a plausible option in OA treatment and management and in most case the first line of action (Bjordal et al., 2006). Although non-pharmacological interventions are the cornerstone of OA management, analgesics play a central role during the painful periods of the condition (Zhang et al., 2005, American College of Rheumatology ACR, 2000). The European League Against Rheumatism (EULAR) guidelines state that both pharmacological and non-pharmacological interventions are needed for optimal treatment of OA (Bjordal et al., 2006). The ACR guidelines (1995) suggest that using simple analgesics up to a full dose as first-line therapy, after failure of non-pharmacological interventions (ACR; 1995).

The choice of an effective pharmacological intervention for OA treatment with minimal risk or adverse reactions comes with raving debate in the scientific arena. Current
practice guidelines advocate the use of a simple analgesic, acetaminophen (paracetamol), or a non-steroidal anti-inflammatory drug (NSAID), given either systemically or topically as first-line or second-line therapies in patients with symptomatic OA (Zhang et al., 2005; ACR, 2000). Several studies have highlighted the benefits as well as the side-effects of the various pharmacological treatments of OA. In a meta-analysis of randomized controlled trials it has been shown that acetaminophen has a similar safety profile to that of placebo, whereas conventional NSAIDs—unlike coxibs—cause more GI discomfort (i.e. abdominal pain, GI distress, nausea, vomiting, dyspepsia, or diarrhea) than acetaminophen (Zhang et al., 2004). These findings were questioned by researchers as to lacking in number of subjects, shorter duration and the statistical power to produce a generalizable result even in an OA population (Glass, 2006; Bjordal et al., 2006; Lee et al., 2004).

Although much has not been done to address the full the safety profile of acetaminophen in OA treatment and management, it is widely accepted as safer regimen in the treatment of pain due to OA compared to NSAIDs which is believed to have a legion of side effects (Towheed et al., 2004; Barlow et al., 2000). As a result, acetaminophen should still be regarded as free of major GI toxicity (Bannwarth 2004; Graham et al., 2005).

A class of pharmacological therapies, NSAIDs has been widely supported to be utilized as second line therapy, starting with lowest effective dose for the shortest possible duration of treatment and then raising it to high doses if needed (Bjordal et al., 2006; Bannwarth, 2006, ACR, 2000). This clinical advice stems from fact that NSAIDs
have a greater risk of serious adverse events. These include lower gastrointestinal, renal or hepatic toxicity as well as increased risk of cardiovascular thrombotic events (Bannwarth, 2006). The primary mechanism of action of NSAIDs is the inhibition of the enzyme cyclooxygenase, which catalyzes arachidonic acid to prostaglandins and leukotrienes both of which are pro-inflammatory. Arachidonic acid is released from membrane phospholipids as a response to inflammatory stimuli. Prostaglandins establish the inflammatory response. NSAIDs interfere with prostaglandin production by inhibiting cyclooxygenase. The enzyme cyclooxygenase, also known as COX, has two forms, known as COX-1 and COX-2. NSAIDs affect both forms of cyclooxygenase. COX-1 is involved in maintaining healthy tissue, while COX-2 is involved in the inflammation pathway (Bannwarth, 2006; Pareek et al., 2006). The main purpose of this class of NSAIDs is therefore to inhibit COX-2 in the inflammation process related to conditions such as OA. COX-2 selective inhibitors also known as ‘coxibs’ is a subset of NSAIDs required to perform this function in relation to controlling the pain and inflammation associated with OA.

Side Effects of the Cox-Inhibitors

Cyclooxygenase inhibitors that block all the factors that contribute to the inflammatory process of the joint remains the ultimate goal in the management and treatment of pain associated with OA. Cyclooxygenase-2 (COX-2) selective inhibitors exhibit better GI tolerability than conventional NSAIDs, but their cardiovascular safety is controversial (Pareek et al., 2006). Even amongst the Cox-2 inhibitors; there exists a formidable debate about how ‘side-effect free’ particular drug agents in this class are. Some have
even been found to be superior by way of WOMAC scores, epigastric discomfort, dyspepsia and abdominal pain (Glass 2006). In a study on the safety and efficacy of NSAIDs in OA treatment, a contrasting observation was found between two NSAIDs: Aceclofenac and diclofenac. Aceclofenac is an effective and well-tolerated drug in osteoarthritis in the Indian setting. An NSAID with high efficacy, high GI tolerability and devoid of adverse cardiovascular effects is therefore a profile preferred by physicians (Pareek et al., 2006).

**Alternative therapies for Osteoarthritis:**

Similar to treatment of other chronic diseases, complementary or alternative therapies have been commonly resorted to in the treatment of OA (Glass 2006). It is important that health-care providers and patients are aware of the evidence for or against these approaches (Schumacher, 2004). Evidence in support of other alternative therapies for OA 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 OA (Schumacher, 2004).

Complementary or alternative therapies have generated sufficiently promising results to warrant further investigation in large-scale, definitive, randomized clinical trials (Ernst, 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, 2006, Morelli et al., 2003). Several studies have found glucosamine and chondroitin sulfate efficacious in treating pain associated with OA (Herrero-Beaumont, et al., 2007; Bennett et al., 2007; Clegg et al., 2006).

Glucosamine (an amino sugar) sulfate works by down regulating the catabolic effects of pro-inflammatory molecules, such as IL-1, which are present in osteoarthritic cartilage (Herrero-Beaumont et al., 2007). On the other hand, Chondroitin sulfate is a complex carbohydrate that helps cartilage to retain water and is believed to inhibit enzymes that break down cartilage (Hamby, 2006). It is also thought to stimulate proteoglycan synthesis in articular cartilage (Glass, 2006). Although there is no evidence from human studies to support this theoretical role in cartilage repair (Glass, 2006), a meta-analysis of studies on its effectiveness have concluded that glucosamine and chondroitin sulfate may be beneficial in the treatment of OA (Herrero-Beaumont et al., 2007, Glass 2006, Leeb et al., 2000). This debate however continues to date.

A number of nutraceuticals and functional foods capable of curbing the pro-inflammatory factors in the joints responsible for pain and inflammation of the joint have also seen much attention in the research arena. Phytochemicals; a typical one of which is flaxseed (which decreases some markers of inflammation (Bloedon and Szapary, 2004) has been highly touted to be possibly effective in the treatment of OA. Although the use of flaxseed in the treatment of OA still remains very popular; there is no
published scientific research in support its current anecdotal usage. Physical activity and exercising have also been recommended in the pain management of OA like any other musculoskeletal condition.

Dietary supplements and single joint treatments are gaining increasing popularity, with evidence of efficacy in randomized controlled trials compared with placebo, and minimal side-effects compared with some more traditional therapies (Derrett-Smith and Beynon, 2006) is currently scarce. Again non-pharmacologic modalities, such as rest and cold applications, are useful for certain types and lifestyle modification in the form of diet can also play a role in chronic disease management (Schumacher, 2004).
Methods

Subject Recruitment and Selection:
A total of 63 subjects, between the ages of 45-69, with a mild to moderate degree of self-reported knee pain associated with joint osteoarthritis, regardless of sex, ethnicity, and race, were recruited. The screening criteria for the subjects included appropriate age, no history of severe liver and kidney disease, agreement not to start on any new prescribed medication for knee pain during the three months of intervention, willingness to modify diet and/or incorporate an at-home exercise program, and commitment to make two visits to the Human Assessment Laboratory in the Department of Nutritional Sciences.

At the first visit, subjects were provided with a verbal and written description of the project and with answers to any questions regarding their participation in this study. The potential subject was assured that his/her participation is completely voluntary and was then asked to sign an informed consent form. A copy of the signed consent form was also made available to the subject to take home. The subjects were randomly assigned to either the dietary modification only group or the dietary modification/exercise regimen group for a period of three months. They were followed up by two monthly calls and then they made a final visit in the third month.

Screening:
A trained graduate student screened potential subjects over the phone by conducting a short medical history questionnaire. The questionnaire helped to identify subjects who were interested and met the inclusion criteria of age, health status, and knee pain
status. The subjects were then asked to come to the DC medical center. All measurements and questionnaires were conducted in the Human Assessment laboratory located in the first floor of the French east building.

Data Collection Methods:

**Anthropometric data:**
Anthropometric data including height, weight, and body composition were determined at both the initial and the final visit. Body weight was measured using an electronic scale (Tanita Best Weight; Cincinnati, OH) with an accuracy of +/- 0.5 lbs. Height was obtained at baseline and at the end of study using a stadiometer (Health-O-Meter; Cincinnati, OH). The stadiometer measures height in inches with an accuracy of +/- 0.1 inch. Bioelectrical impedance (RJL Systems; Clinton Twp, MI) which is a widely used and an acceptable method of measuring body composition was used at baseline and at the end of the three months. It provides correlations in the range of 0.75-0.92 for total body weight and 0.46-0.79 for extra cellular water. The results were then analyzed using analysis software available at the manufacturer's web site http://interactive.rjlsystems.com.

**Questionnaires:**
A medical history questionnaire was completed at the first visit for each of the study participants. This questionnaire was used to further support our prescreening of individuals who were excluded due to certain chronic and acute conditions. The physical
activity questionnaire, pain assessment questionnaire, food frequency questionnaire, and the vitamin/mineral supplement use questionnaire were conducted at both the initial and the final visit.

All the questionnaires have been validated for using with osteoarthritis patients. Physical activity was evaluated using the Five-City Project Physical Activity Recall. The pain assessment questionnaire used in this study was a modification of the McGill Pain Questionnaire and SF-36 health survey questionnaire. 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 (http://www.qolid.org/public/MPQ.html). The SF-36 health survey questionnaire can detect the subtle changes in health that follow an intervention and produces reliable and valid results. It is easy to understand and relevant to most people's lives (http://www.bupa.co.uk/healthsurvey/html/why/sf36.html).

The food frequency questionnaire and a vitamin and mineral supplement questionnaire adapted for studies on osteoarthritis patients were conducted to assess the nutritional status of the subjects and to find any relationship between intake of specific food groups as well as fluid intake and the severity and progression of osteoarthritis.
**Treatment Protocol:**

Subjects in both groups received individual counseling to modify their dietary intake of fat, cholesterol, and sodium, and to reduce the overall intake of calories. Additionally, study participants were provided with dietary guidelines for Americans, established by the United States Department of Agriculture (USDA), to increase their fruit and vegetable intake to increase the intake of antioxidant vitamins and minerals.

Research suggests that increased antioxidants in the diet help to decrease the inflammatory process associated with cartilage degeneration. Subjects in the dietary modification/exercise regimen group were also provided with counseling to incorporate an at-home exercise program to their daily routine. This exercise program was designed by a Physical Therapist and is a modification of an exercise program recommended by the orthopedic surgeons/rheumatologists focused on knee OA. The exercise program was designed to improve the strength of muscles acting around the knee, the range of motion at the knee joint, and the locomotor function. The participants were encouraged to do the exercise regimen daily with both legs for 20-30 minutes. The exercise program was self paced and the participants were advised to make it more challenging by increasing the number of repetitions of each exercise. The exercise program was taught to the participants in the assessment laboratory by trained graduate students. To help subjects follow the exercise program at home, they were provided with a theraband device, a video tape of the exercise regimen, and a schematic presentation of the individual exercises. The theraband was used to increase the resistance against which the muscles worked. Study participants were also provided with a three month
calendar to assess their compliance to the exercise protocol at the end of the study.

All the subjects were followed up by two monthly phone calls to address issues or problems related to the study treatments. These follow-up phone calls were also utilized to evaluate the compliance of study participants. The subjects returned for their final visit approximately 90 days after the initial visit. During the final visit, all of the measures obtained at baseline were repeated to evaluate the effect of the treatment.

**Statistical Analysis:**

Descriptive statistics including means, standard deviations, standard errors, minima and maxima, were determined for all variables. Distributions of the response variables were examined to determine if statistical tests of hypothesis based on the assumption of normality are appropriate, or whether transformed data or non-parametric tests should be used. An unpaired t-test was performed on all the data to compare the improvement following three months of both the interventions. After satisfying the assumption of normality, a student t-test was used to compare the baseline values of body weight, body mass, and anthropometric measurements and relevant covariates such as age were compared for the two groups. The baseline values of pain and stiffness assessments were compared to the after treatment values among the two groups using the test for ordinal data. Spearman correlation test was performed to determine if any relationship exists between the four pain parameters for each individual.

Logistic regression analysis was used to compare the effect of treatments on the four pain parameters for the subjects in both the intervention groups. The logistic regression
analysis included adjustment for body weight, fat and fat free mass because the baseline values of weight, fat and fat-free mass differed between the two intervention groups. Dropouts of the study were analyzed by an intent-to-treat analysis to see how they affect the general result. Tests of fixed effects were performed on the data for range of motion (extension and flexion), which provided with the p-values and 95% confidence intervals for the effect of treatment on flexion and abduction. It was tested using a random effects model as well. All the data are reported as mean +/- standard deviation, with p<0.05 regarded as significant.
## Table 1:

### Characteristics of Study Participants (General)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Nutrition</th>
<th>Nutrition &amp; Exercise</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Baseline</td>
<td>Final</td>
<td>diff</td>
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<td></td>
<td>n=31</td>
<td>n=29</td>
<td></td>
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<td>Age (yr)</td>
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<td>42.2 ± 10.8</td>
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<tr>
<td>Fat Free Mass</td>
<td>56.5 ± 11.2</td>
<td>57.8 ± 10.8</td>
<td>-0.86 ± 1.8</td>
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</tbody>
</table>

Data shown are mean +/- standard deviation.  
N=21-32  
Diff, difference  
Level of significance is P-value of ≤ 0.05
Table 2:
Gender Classification of Anthropometric Measurements (Males)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± Standard Deviation</th>
<th>Mean Differences</th>
<th>T-Test</th>
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<th>t</th>
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<td>Nutrition and Nutrition &amp; Exercise</td>
<td>Variances</td>
<td>t-Value</td>
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<td>Final</td>
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<td>Final</td>
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<tr>
<td>Fat Free Mass</td>
<td>Baseline</td>
<td>77.95±6.00</td>
<td>77.98±5.05</td>
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</tr>
<tr>
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<td>Final</td>
<td>77.85±5.92</td>
<td>78.30±5.77</td>
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</table>
Table 3:
Gender Classification of Anthropometric Measurements (Females)

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<th>T-Test</th>
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<td>202.78±46.38</td>
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<td>45.44±7.25</td>
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<td>54.67±9.49</td>
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Table 4:
Characteristics of Study Participants (Gender Specific)

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<th>Females (Total Number = 54)</th>
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<td>Final</td>
<td>78.10±5.47</td>
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Table 5:
Correlations among Anthropometric Measurements

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<th>PARAMETER</th>
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<th>Final</th>
<th>Baseline</th>
<th>Final</th>
<th>Baseline</th>
<th>Final</th>
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<td><strong>Body Weight (lb)</strong></td>
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<tr>
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<tr>
<td>FWT</td>
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<tr>
<td>BFAT</td>
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</table>

P-value: >0.0001

Legend: **BWT**: baseline body weight; **FW**: final weight; **BFAT**: base line fat mass; **FFAT**: final fat mass; **FFM**: baseline fat-free mass; **FFFM**: final fat-free mass
Table 6:
Correlations among Anthropometric Measurements –Intent to Treat (General)

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<tr>
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<th>P-Value</th>
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<td>FWT</td>
<td>BFAT</td>
<td>FFAT</td>
<td>FFM</td>
<td>FFFM</td>
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<td></td>
<td></td>
<td>1.00</td>
<td></td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Legend:
**BWT**: baseline body weight; **FW**: final weight; **BFAT**: base line fat mass; **FFAT**: final fat mass; **FFM**: baseline fat-free mass; **FFFM**: final fat-free mass
### TABLE 7:
Levels of Pain of Study Participants

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Nutrition</th>
<th></th>
<th></th>
<th>Nutrition &amp; Exercise</th>
<th></th>
<th></th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Final</td>
<td>diff</td>
<td>Baseline</td>
<td>Final</td>
<td>diff</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=31</td>
<td>n=29</td>
<td></td>
<td>n=32</td>
<td>n=21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain Pattern</td>
<td>1.76 ± 0.52</td>
<td>1.98 ± 0.51</td>
<td>0.21 ± 0.81</td>
<td>1.89 ± 0.57</td>
<td>2.24 ± 0.61</td>
<td>0.20 ± 0.64</td>
<td>0.980</td>
</tr>
<tr>
<td>Current Pain Intensity</td>
<td>1.31 ± 0.90</td>
<td>1.45 ± 0.77</td>
<td>0.09 ± 0.79</td>
<td>1.58 ± 0.87</td>
<td>1.00 ± 0.86</td>
<td>-0.68 ± 0.89</td>
<td>0.029</td>
</tr>
<tr>
<td>Highest Pain Intensity</td>
<td>3.53 ± 0.97</td>
<td>2.84 ± 0.73</td>
<td>-0.72 ±0.91</td>
<td>3.55 ± 0.82</td>
<td>2.65 ± 0.99</td>
<td>-0.90 ± 0.85</td>
<td>0.499</td>
</tr>
<tr>
<td>Usual Pain Intensity</td>
<td>1.65 ± 0.89</td>
<td>1.65 ± 0.51</td>
<td>-0.01 ±0.84</td>
<td>1.72 ± 0.71</td>
<td>1.33 ± 0.66</td>
<td>-0.35 ± 0.69</td>
<td>0.137</td>
</tr>
</tbody>
</table>

Data shown are mean ± standard deviation (SD)
N=20-32
Diff, difference
Level of significance is P-value of ≤ 0.05
### Table 8:
Levels of Pain of Study Participants (Gender Specific)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Minimum</td>
</tr>
<tr>
<td>Pain Pattern</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1.97±0.66</td>
<td>1.00</td>
</tr>
<tr>
<td>Final</td>
<td>1.86±0.71</td>
<td>1.00</td>
</tr>
<tr>
<td>Pain Intensity Current</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1.05±0.52</td>
<td>0.00</td>
</tr>
<tr>
<td>Final</td>
<td>1.00±1.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Pain Intensity Highest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>3.44±1.01</td>
<td>0.00</td>
</tr>
<tr>
<td>Final</td>
<td>2.55±1.23</td>
<td>2.00</td>
</tr>
<tr>
<td>Pain Intensity Least</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>0.77±0.66</td>
<td>0.00</td>
</tr>
<tr>
<td>Final</td>
<td>1.05±0.80</td>
<td>0.00</td>
</tr>
<tr>
<td>Pain Intensity lowest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>0.33±0.71</td>
<td>0.00</td>
</tr>
<tr>
<td>Final</td>
<td>0.33±0.71</td>
<td>0.00</td>
</tr>
<tr>
<td>Pain Intensity Usual</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1.57±0.81</td>
<td>0.20</td>
</tr>
<tr>
<td>Final</td>
<td>1.51±0.77</td>
<td>1.00</td>
</tr>
<tr>
<td>Parameter</td>
<td>Mean ± Standard Deviation</td>
<td>Mean Differences</td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------------</td>
<td>------------------</td>
</tr>
<tr>
<td></td>
<td>Nutrition Only</td>
<td>Nutrition &amp; Exercise</td>
</tr>
<tr>
<td>Left Flexion</td>
<td>-1.16±4.32</td>
<td>-5.27±4.01</td>
</tr>
<tr>
<td>Right Flexion</td>
<td>0.33±2.35</td>
<td>-5.46±7.17</td>
</tr>
<tr>
<td>Left abduction</td>
<td>-0.08±29.12</td>
<td>-5.86±9.32</td>
</tr>
<tr>
<td>Right abduction</td>
<td>9.0±22.01</td>
<td>11.93±14.95</td>
</tr>
</tbody>
</table>

Table 9:
Gender Classification of Range of Motion of Left and Right Knee (Males)
Table 10:
Gender Classification of Range of Motion of Left and Right Knee (Females)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± Standard Deviation</th>
<th>Mean Differences</th>
<th>T-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nutrition Only</td>
<td>Nutrition &amp; Exercise</td>
<td>Nutrition and Nutrition &amp; Exercise</td>
</tr>
<tr>
<td>Left Flexion</td>
<td>-1.97±3.72</td>
<td>-4.69±6.35</td>
<td>2.71</td>
</tr>
<tr>
<td>Right Flexion</td>
<td>-2.18±5.41</td>
<td>-5.33±6.67</td>
<td>3.14</td>
</tr>
<tr>
<td>Left abduction</td>
<td>1.22±125.39</td>
<td>-5.86±9.32</td>
<td>1.71</td>
</tr>
<tr>
<td>Right abduction</td>
<td>-3.54±12.59</td>
<td>-1.44±13.63</td>
<td>-2.10</td>
</tr>
</tbody>
</table>
## Table 11:
Gender Classification of Range of Motion of Left and Right Knee (Males) by Intent-to-Treat

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± Standard Deviation</th>
<th>Mean Differences</th>
<th>T-Test</th>
<th>Variances</th>
<th>t-Value</th>
<th>Pr &gt;</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nutrition Only</td>
<td>Nutrition &amp; Exercise</td>
<td>Nutrition and Nutrition &amp; Exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Flexion</td>
<td>-1.16±4.32</td>
<td>-5.27±4.01</td>
<td>4.10</td>
<td>Unequal</td>
<td>1.46</td>
<td>0.19</td>
<td></td>
</tr>
<tr>
<td>Right Flexion</td>
<td>0.33±2.35</td>
<td>-5.46±7.17</td>
<td>5.80</td>
<td>Unequal</td>
<td>1.70</td>
<td>0.15</td>
<td></td>
</tr>
<tr>
<td>Left abduction</td>
<td>-0.08±29.12</td>
<td>-5.86±9.32</td>
<td>5.78</td>
<td>Unequal</td>
<td>0.38</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>Right abduction</td>
<td>9.0±22.01</td>
<td>11.93±14.95</td>
<td>20.93</td>
<td>Unequal</td>
<td>1.63</td>
<td>0.16</td>
<td></td>
</tr>
</tbody>
</table>
Table 12:

Gender Classification of Range of Motion of Left and Right Knee (Females) by Intent –to-Treat

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± Standard Deviation</th>
<th>Mean Differences</th>
<th>T-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nutrition Only</td>
<td>Nutrition &amp; Exercise</td>
<td>Nutrition and Nutrition&amp; Exercise</td>
</tr>
<tr>
<td>Left Flexion</td>
<td>-1.82±3.61</td>
<td>-2.61±5.23</td>
<td>0.77</td>
</tr>
<tr>
<td>Right Flexion</td>
<td>-2.05±5.23</td>
<td>-2.96±5.59</td>
<td>0.94</td>
</tr>
<tr>
<td>Left abduction</td>
<td>1.13±14.79</td>
<td>-0.27±9.07</td>
<td>1.41</td>
</tr>
<tr>
<td>Right abduction</td>
<td>-3.28±12.14</td>
<td>-0.80±10.03</td>
<td>-2.48</td>
</tr>
</tbody>
</table>
Results

A total 63 subjects consisting of 9 males and 54 females were recruited for this study. Subjects were randomized into either the nutrition intervention or the combined nutrition and exercise intervention. The nutrition intervention had 31 subjects, while the nutrition and exercise intervention had 32 subjects. With an attrition of 21%, the overall retention rate was 79%. The retention for nutrition intervention was 94% with the nutrition and exercise group having retention of 63%. The nutrition and exercise intervention had a higher attrition rate.

Baseline and final values including age, weight, fat mass and fat-free mass across the two treatment groups are show in Table 1. Whereas Tables 2 and 3 presents this data by gender respectively. Although baseline body weight and fat mass was higher in the nutrition alone group compared to nutrition and exercise group (not statistically significant) in both genders, no change were seen in either of the two values after treatment in the two treatment groups (Table 2 & 3). The above characteristic of the study subjects in relation to anthropometric parameters by gender showing minima and maxima values are shown in Table 4.

Although the correlations among the anthropometric parameters were statistically significant (Table 5); correlations between all the anthropometric parameters at baseline and end of study, most of them were fairly weak and negatively with a few such as final weight, final fat mass and final fat-free mass being strongly and positively correlated with their respective baseline values. There was no
difference in correlation amongst all anthropometric parameters when they were analyzed by intent-to-treat analysis (Table 6).

Parameters related to pain assessment are shown in Table 7. In particular, there was a significant reduction (p-value > 0.029) in current pain intensity from baseline to final (Table 7). There were also reductions in all pain parameters assessed across both genders (Table 8). After three months of intervention, the nutrition and exercise treatment in comparison to nutrition alone was associated with a greater reduction in pain from baseline to follow up (p= 0.02). These results were unchanged when adjusted for baseline values of body weight, fat and fat free mass. To determine if any relationship exists between the different pain parameters, a spearman correlation analysis was performed. The findings suggest that when the frequency of pain decreases (pain pattern), the level of pain intensity that an individual experiences also decreases. This correlation was found with subjects on the nutrition and exercise treatment and not with subjects in the nutrition treatment alone.

The range of motion was detected by measuring abduction and flexion of both knees with a stadiometer. There was no difference in the baseline values of abduction and flexion in either knee between the two treatment groups (Tables 9 &10). However, after three months of intervention, nutrition and exercise intervention had more favorable effect on extension than nutrition alone intervention group and that the effect of the combined intervention was significant (P= 0.041). There was no significant difference in flexion from
baseline to final in either of the treatments groups or between the treatment groups. There were also no significant differences in detected range of motion between males and females. Similarly, there were no significant changes in range of motion values in males by intent-to-treat analysis (Table 11). On the other hand, the females had slightly changes in detected range of motion when they were analyzed by the intent-to-treat analysis, these were however found to be not significant (Table 12).
Discussion

The overall findings of our three-month intervention study suggests that a dietary modification in combination with an at-home exercise regimen can be beneficial in improving self reported knee pain associated with osteoarthritis. The at-home exercise program was self paced however it was designed to become progressively more challenging over time. It was designed to improve the strength of the muscles in the knee joint, as well as positive impact the range of motion and the motor function. This improvement in self-reported knee pain of our study subjects could be attributed to the combination of diet and therapeutic exercise. This finding is comparable to the conclusions of a Cochrane review in which land-based therapeutic exercise seemed to reduce pain and improve function in symptomatic OA of the knee (Fransen et al., 2004). The generalizability of our findings, particularly in relation to the exercise intervention is limited. This is due the study criteria being self-reported knee pain. The observed improvement in the different pain parameters were not all found to be significant. However, it was in concordance with findings of other researchers (Scott et al., 2004, Fransen et al., 2004) which had either insufficient data or insignificant findings on comparable pain parameters.

As our study indicated, that slight changes in anthropometric parameters was seen in all the subjects at the end of the study. More specifically female subjects in the study had reductions in body weight, fat mass and fat free mass in both treatment groups of the study. Whereas, male subjects did not have any marked
reduction in any of these parameters except for fat free mass. These findings are comparable to a study by Hansen and colleagues where nutrition and exercise resulted in weight loss and reduced fat mass in obese individuals with knee OA (Hansen et al., 2007). In relation to our findings, the gender specific reductions in anthropometric parameters could be attributed to higher compliance in the females who may be more likely to have focused on weight reduction. A study by Schneider and colleagues had indicated similarly the improvements being more attributed to weight loss commitment that differs in male versus female (Schneider et al., 2007). Furthermore, female subjects in our study which were assigned to the nutrition and exercise intervention may have great motivation and a sense of control related to diet restriction and participating in the exercise regimen (C.H. van Gool et al., 2006). Since our study subjects were predominantly obese, patients with the highest fat mass are most likely to lose the largest amount of fat mass in such lifestyle intervention programs (Hansen et al., 2007) which was the focus of our study.

On the issue of weight loss in relation to pain reduction there have been little data regarding the efficacy of weight loss for treatment of OA (Glass 2006). Unlike the findings of an 18-month controlled trial with a diet-plus-exercise group, which showed statistically significant improvement in pain and self-reported physical function (Messier et al., 2004), our study did not show significant changes when relating pain parameters to changes in range of motion. This may be due to the duration of treatment being much longer than the 3-months in our
Improvement in range of motion of the affected knee was further supported by reduction in the level of pain experienced by the individuals in the combine nutrition and exercise group. This observation may stem from the fact that moderate regular exercise restores articular surface on osteoarthritis joint by decreasing contact pressures of the articular surfaces combined with the movement of the joint, which improves joint mobility (Buckwalter and Mow, 2001).

Compliance to the study protocol was monitored by monthly phone call follow-up as well as completing an exercise performance calendar. We did not have any problem with compliance regardless of the fact that the exercise regimen was a home-based one as compared to other facility based ones. The issue of compliance could not affect the results of our finding which is similar to one observed by McCarthy and colleagues in a study where home based exercising subjects and facility based ones were found to be equally compliant (McCarthy et al., 2004). Although recruitment was quite successful, there was a high attrition rate of 21%. There were more dropouts in the nutrition and exercise group than the nutrition alone. The generally high attrition rate of our study did not however affect the results of our study as statistically assessed by the intent-to-treat analysis.

Our study findings have resulted in some interesting effects on the issue of nutrition/exercise versus nutrition alone in OA pain management. Whereas other studies (Hansen et al., 2007; Messier et al., 2004) involved older obese or over
weight adults, our study involved a relatively younger population from middle age to younger old adults. Much as studies have been geared towards older adults who more likely to have OA, studies targeting the middle age of the population will provide useful information on the study and research of osteoarthritis in general. Especially, since the condition of OA is evident more so in the US population at an early age. Therefore, the interventions that focus on diet and exercise are primary candidate for the approach in this population.

Study Limitations

Looking at the design of our study and the outcomes of our research, certain modifications to follow-up studies which can be suggested to address the limitations present in our study. These include:

- More females than males limiting the generalizability of findings to only female populations.
- Inclusion criteria and Selection bias because the OA is more prevalent in women aged 45 years and over.
- Short duration of the study
- No systematic analysis of dropouts of the study
- Community based subject recruitment has compliance challenges as opposed to other methods such as facility or medical practitioner recruitment.
Recommendations and Future Directions

In relation to some of the limitations of our study and some of the general considerations with regards to studies of this nature, the design of a future study of this kind could be more potent in examining similar objectives of our study when the following considerations are looked at:

- Longer duration of Study
- Assessment times at least three to enable assessment of changes between completers and drop-outs of the study at each phase of the study.
- Increased sample size
- Biomarker-based assessment of improvement outcomes coupled with radiographic measurements
References:


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space narrowing, especially in the lateral femorotibial compartment, in early knee osteoarthritis. Ann Rheum Dis. 2007


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89. Poole AR. An Introduction to the Pathophysiology of Osteoarthritis Front Biosci 1999; 4:D662–70.


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Appendix
HEALTH AND MEDICAL HISTORY QUESTIONNAIRE

I. Medical History

A. Skeletal Health

Personal history of skeletal disorders:
1. Not known
2. Yes: uncontrolled
3. Yes: Medications
4. Yes: Exercise program
5. Yes: Modified diet
6. Yes: Surgery
7. Yes: Combined program

Give details

Type of Medication(s)
Current dosage
Years taken

How does this condition affect your activity?

Family history of skeletal disorders:
1. None
2. One parent
3. Both parents
4. One close relative
5. More than one close relative

Relative(s)
Comments:

B. Cardiovascular Function

Personal history of cardiovascular disease:
1. Not known
2. Yes: uncontrolled
3. Yes: Medications
4. Yes: Exercise program ___________________
5. Yes: Modified diet _________________
6. Yes: Surgery ________________
7. Yes: Combined program ___________

Give details _______________________________________________________

Type of Medication(s) _____________________________________________
Current dosage ________________ Years taken _________________________

How does this condition affect your activity?

**Family history of cardiovascular disease:**

1. None _____
2. One parent _____
3. Both parents _____
4. One close relative _____
5. More than one close relative _____

Relative(s) ______________________________________________
Comments: ______________________________________________

C. Hypertension

1. None known __________________
2. Yes: uncontrolled ________________
3. Yes: Medications ______________
4. Yes: Exercise program ________________
5. Yes: Modified diet ________________
6. Yes: Yes: combined program__________
7. Most recent blood pressure __________

Explain _______________________________________________________

Type of Medication(s) ____________________________________________
Current dosage ________________ Years taken _______________________

Ever taken thiazide diuretics? ____________

D. Diabetes

1. No record or indication _____
2. In past, but not now _____
3. Yes, well controlled _____
4. Yes, not controlled _____

Explain _______________________________________________________

Type of Medication(s)_________________________________________
Current dosage _____________________ Years taken________________

How does this condition affect your activity? _______________________

67
E. Gastrointestinal/Digestive Problems

1. No record or indication ____
2. In past, but not now ____
3. Yes, well controlled ____
4. Yes, not controlled ____
Explain

Type of Medication(s)
Current dosage _____________________ Years taken ____________________
Ever taken steroids (i.e., prednisone)? ______________________
Currently taking antacids? ______________________
How does this condition affect your activity? _________________

F. Liver Disease/Problems

1. No record or indication ____
2. In past, but not now ____
3. Yes, well controlled ____
4. Yes, not controlled ____
Explain

Type of Medication(s)
Current dosage _____________________ Years taken ____________________
How does this condition affect your activity? _________________

G. Respiratory Problems

1. No record or indication ____
2. In past, but not now ____
3. Yes, well controlled ____
4. Yes, not controlled ____
Explain

Type of Medication(s)
Current dosage _____________________ Years taken ____________________
How does this condition affect your activity? _________________

H. Thyroid Disorder

1. No record or indication ____
2. In past, but not now ____
Hyper? _____  Hypo? ____
Explain

Type of Medication(s)
II. Medication or Drug Use

A. Previous or Present Use of Any of the Following (Specify):

1. Anabolic steroids
2. Corticosteroids or glucocorticoids
3. Thiazide diuretics
4. Vitamin D

B. Previous or Present Use of Alcoholic Beverages (Beer, wine, hard liquor)

Please indicate:
Frequency of intake (Times/week or times/month): ____
Number of servings at a sitting: ____
Number of years of use: ____

C. Currently /previously a smoker? ____
If yes, number of cigarettes per day________________

D. Currently using any laxatives (fibercon, colace., metamucil, Ex lax, including fiber supplements):
Indicate type__________________________ and amount per day________________
How often per week____________________

III. Physical Activity

A. Occupational Intensity (respond to 1, 2, 3, or 4):
1. Majority of time: Sitting ____  Standing ____  Walking ___
2. Equal amount of time:
   Sitting and Standing ____
   Walking and Sitting ____
   Standing and Walking ____
3. Combination: Sitting, Standing, and Walking ___
4. Much of time: Lifting & Carrying ___
PHYSICAL ACTIVITY QUESTIONNAIRE

First we would like to know about your physical activity during the past 7 days. But first, let me ask you about your sleep habits.

1. On the average, how many hours did you sleep each night during the last five weekday nights (Sunday-Thursday)? ______hours

2. On the average, how many hours did you sleep each night last Friday and Saturday nights? ______hours

Now I am going to ask you about your physical activity during the past 7 days, that is, the last 5 weekdays and last weekend, Saturday and Sunday.

We are not going to talk about light activities such as slow walking, light housework, or non-strenuous sports such as bowling, archery, or softball.

Please look at this list which shows some examples of what we consider moderate, hard, and very hard activities. (interviewer: hand subject the following list and allow time for the subject to read it over.)

People engage in many other types of activities, and if you are not sure where one of your activities fits, please ask me about it.

3. First, let’s consider moderate activities. What activities did you do and how many total hours did you spend during the last 5 weekdays doing these moderate activities or others like them?
4. Last Saturday and Sunday, how many hours did you spend on moderate activities and what did you do? (Probe: Can you think of any other sports, job, or household activities that would fit into this category?)

_____ hours

5. Now, let’s look at hard activities. What activities did you do and how many total hours did you spend during the last 5 weekdays doing these hard activities or others like them? Please tell me to the nearest half-hour. _____ hours

6. Last Saturday and Sunday, how many hours did you spend on hard activities and what did you do? (Probe: Can you think of any other sports, job, or household activities that would fit into this category?)

_______ hours

7. Now, let’s look at very hard activities. What activities did you do and how many total hours did you spend during the last 5 weekdays doing these very hard activities or others like them? Please tell me to the nearest half-hour. (Probe: Can you think of any other sports, job, or household activities that would fit into this category?)

_______ hours

8. Last Saturday and Sunday, how many hours did you spend on very hard activities and what did you do? (Probe: Can you think of any other sports, job, or household activities that would fit into this category?)

_______ hours

9. Compared with your physical activity over the past month, was last week’s physical activity more, or less, or about the same?

_____ 1. More
_____ 2. Less
_____ 3. About the same

Interviewer: Please list below any activities reported by the subject, which you don’t know how to classify. Flag this record for review and completion.

Activity (brief description) Hours: workday Hours: weekend day
10. Are you engaged in any regular exercise?

Walk_______________, Minutes per day________, Days per week________________
Power Walk__________, Minutes per day________, Days per week________________
Treadmill____________, Minutes per day________, Days per week________________
Aerobics_____________, Minutes per day________, Days per week________________
Weight aerobics_______, Minutes per day________, Days per week________________
Bicycling/Stationary bike_______, Minutes per day________, Days per week________
Tennis_______________, Minutes per day________, Days per week________________
Other, please specify:
____________________, Minutes per day________, Days per week________________
____________________, Minutes per day________, Days per week________________
____________________, Minutes per day________, Days per week________________
____________________, Minutes per day________, Days per week________________

EXAMPLES OF ACTIVITIES IN EACH CATEGORY

**Moderate activity (3-5 mets)**

**Occupational Tasks:**
1) Delivering mail or patrolling on foot
2) House painting
3) Truck driving (making deliveries, lifting/carrying light objects)

**Household Activities:**
1) Sweeping, mopping, cleaning windows
2) Mowing the lawn with a power mower
3) Raking the lawn and yardwork
4) Light carpentry

**Sports:**
1) Table tennis or Ping-Pong
2) Softball, baseball
3) Volleyball
4) Dancing: folk, square, aerobics (low impact & intensity)
5) Brisk walking (3 to 4 mile/hr; 15-20 min/mile)
6) Bicycling on level ground (10-15 mile/hr)
7) Golfing (walking and pulling/carrying own clubs)
Calisthenics exercise and weight lifting

HARD ACTIVITY (5.1 – 6.9 METS)

Occupational Tasks:
1) Heavy carpentry
2) Construction work

Household Tasks:
1) Scrubbing floors
2) Shoveling snow
3) Moving (lifting furniture and boxes)

Sports:
1) Racket Sports: badminton, paddleball, tennis (double)
2) Basketball
3) Rowing or canoeing leisurely
4) Dancing: disco, jazz, aerobics (medium impact & intensity)
5) Power walking (> mile/hr; <15 min/mile) or hiking
6) Vigorous bicycling (16 – 20 mile/hr)
7) Jogging (∆5 mile/hr)
8) Swimming
9) Roller or ice skating
10) Stationary bicycling

VERY HARD ACTIVITY (≥7.0 METS)

Occupational Tasks:
1) Digging or chopping with heavy tools
2) Carrying heavy loads, such as bricks or lumber

Sports
1) Racket Sports: handball, racketball, squash, tennis (singles)
2) Soccer
3) Snow skiing (down hill and cross country)
4) Dancing: aerobics (high impact & intensity)
5) Jumping rope
6) Vigorous bicycling on hills
7) Jogging or running (∆8 mile/hr)
Knee Pain Study

Subject ID
Date of Birth
Interviewer:

PAIN ASSESSMENT QUESTIONNAIRE

1. Where is your Pain?

Please mark, on the drawings below, the areas where you feel pain. Put E if external, or I if internal, near the areas which you mark. Put EI if both external and internal.
2. How Does Your Pain Change With Time?

A. Which word or words would you use to describe the pattern of your pain?

1. Continuous
   Steady
   Constant

2. Rhythmic
   Periodic
   Intermittent

3. Brief
   Momentary
   Transient

B. What kind of things relieve your pain?

C. What kind of things increase your pain?
3. How Strong Is Your Pain?
The following 5 word ratings describe pain of increasing intensity.

**Rating 1= Mild:** Actual pain which can be defined and described. It does not interfere with normal activity. Distraction or concentration on something else allows the pain to be unnoticed at least for short periods. The person is able to sleep and relief measures may or may not be used.

**Rating 2= Discomforting:** Pain is more obvious. Awareness is constant and the individual is more aware of its presence, e.g. irritable, weary. This rating can be tolerated, but may interfere with functional abilities; however, usually does not interfere with sleep. Relief measures will probably be used.

**Rating 3= Distressing:** Pain becomes the main focus as is immediate relief. Ability to perform daily activities is severely affected, e.g. will not cook, drive, groom, etc. unless there is absolutely no choice. Unable to sleep; emotional responses often accompany this rating. More specific, stronger, or multiple pain relief measures are used.

**Rating 4= Horrible:** Pain consumes every moment; the person is nonfunctional due to pain. Secondary physiologic responses e.g. nausea, sweating are often present. The person requires care, e.g. food will not be prepared and will be eaten only if specifically brought. Stronger emotional responses, e.g. feeling depressed and powerless, occur. Sleep rarely comes. Usual pain relief measures are not often effective.

**Rating 5= Excruciating:** The person cannot think clearly; may be irrational. As the body undergoes severe physiologic responses, health professionals may be alarmed and must rule out a medical emergency. Reality, e.g. time, is distorted; pain is the only focus; the person is hopeless and despairing.

Please refer to the above information in order to answer each question below, write the number of the most appropriate rating in the space beside the question.

1. Which rating describes your pain right now?

2. Which rating describes it at its worst?

3. Which rating describes it when it is least?
4. What is your baseline or lowest rating? (you can use a rating of 0 to recall times of being symptom-free)

_______________________________

5. What is your usual rating?

_______________________________

6. If more than one rating, what percent of the time do you have each intensity?

___________

4. How Does Pain Affect Your Ability to Function?
(To answer this question, indicate the painful part, or joint, in your body which you are referring to) Check the most appropriate answer.

My ________________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)

Degree of Bend: Forward ___________; Backward ___________

My ________________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)

Degree of Bend: Forward ___________; Backward ___________

My ________________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)
Degree of Bend: Forward ___________; Backward ____________

My ___________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)

Degree of Bend: Forward ___________; Backward ____________

My ___________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)

Degree of Bend: Forward ___________; Backward ____________

My ___________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)

Degree of Bend: Forward ___________; Backward ____________

My ___________________________ (body part or joint)
( ) I am capable of moving (or bending) this part of my body freely (1)
( ) I am capable of moving (or bending) this part of my body, but with some difficulty (2)
( ) I am capable of some very limited movement of this part of my body, because of pain (3)
( ) I am unable to move this part of my body, because of pain (4)
Does your pain affect your ability to lift heavy objects?
(Check the most appropriate answer)

With my__________________________ (body part, or joint)
( ) I can lift a 10 lb bag with no difficulty for at least a continuous 10 minutes (1)
( ) I can lift a 10 lb bag for less than 10 minutes (2)
( ) I cannot lift a 10 lb bag because of pain (3)

With my__________________________ (body part, or joint)
( ) I can lift a 10 lb bag with no difficulty for at least a continuous 10 minutes (1)
( ) I can lift a 10 lb bag for less than 10 minutes (2)
( ) I cannot lift a 10 lb bag because of pain (3)

With my__________________________ (body part, or joint)
( ) I can lift a 10 lb bag with no difficulty for at least a continuous 10 minutes (1)
( ) I can lift a 10 lb bag for less than 10 minutes (2)
( ) I cannot lift a 10 lb bag because of pain (3)

With my__________________________ (body part, or joint)
( ) I can lift a 10 lb bag with no difficulty for at least a continuous 10 minutes (1)
( ) I can lift a 10 lb bag for less than 10 minutes (2)
( ) I cannot lift a 10 lb bag because of pain (3)
( ) I can lift a 10 lb bag for less than 10 minutes (2)

( ) I cannot lift a 10 lb bag because of pain (3)
Knee Pain Study

Subject ID
Date of Birth
Interviewer:

SEVEN DAY FOOD FREQUENCY QUESTIONNAIRE

This questionnaire asks you about your consumption of foods and beverages over the past week, which includes the time from exactly one week ago until the last meal you had before you fill out this questionnaire. The “How Often” columns are for day, week, or rarely/never. We want you to think back over the past week and tell us how many times (per day, if you consume the item every day, or per week) you consumed each item. A medium serving is in parentheses.

EXAMPLES:

Ate 1/2 grapefruit about twice last week.
Ate 1 large hamburger four times last week.
Drank 2 cups of whole milk each day.

<table>
<thead>
<tr>
<th>Type of Food</th>
<th>How Often</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Medium Serving)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grapefruit (1/2)</td>
<td>Day 2</td>
<td>X</td>
</tr>
<tr>
<td>Hamburger, regular (1 patty, 3 oz)</td>
<td>Week 4</td>
<td>X</td>
</tr>
<tr>
<td>Whole milk (1 cup, 8 oz)</td>
<td>Day 2</td>
<td>X</td>
</tr>
<tr>
<td>Type of Food</td>
<td>How Often</td>
<td>Size</td>
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<tr>
<td>-------------------------------</td>
<td>-----------</td>
<td>------</td>
</tr>
<tr>
<td></td>
<td>Day</td>
<td>Week</td>
</tr>
<tr>
<td>DAIRY FOODS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole milk (1 cup, 8 oz)</td>
<td></td>
<td></td>
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<tr>
<td>2% milk (1 cup, 8 oz)</td>
<td></td>
<td></td>
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<tr>
<td>Skim milk (1 cup, 8 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cream, whipped (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sour cream (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coffee cream (1 Tbsp)</td>
<td></td>
<td></td>
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<tr>
<td>Ice cream (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low fat ice cream (½ cup)</td>
<td></td>
<td></td>
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<tr>
<td>Frozen yogurt (½ cup)</td>
<td></td>
<td></td>
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<tr>
<td>Yogurt (1 cup)</td>
<td></td>
<td></td>
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<tr>
<td>Low fat yogurt (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cottage cheese (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cream cheese (1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low fat cream cheese (1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other cheese (1 slice or 1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low fat cheese (1 slice or 1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Margarine (1 tsp)</td>
<td></td>
<td></td>
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<tr>
<td>Butter (1 tsp)</td>
<td></td>
<td></td>
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<tr>
<td>Reduced fat margarine (1 tsp)</td>
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<td></td>
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<tr>
<td>FRUITS, FRUIT JUICES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raisins (1 oz or 1 sm box)</td>
<td></td>
<td></td>
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<tr>
<td>Grapes (20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prunes (½ cup)</td>
<td></td>
<td></td>
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<tr>
<td>Bananas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cantaloupe (¼ melon)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Watermelon (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apples, applesauce or pears</td>
<td></td>
<td></td>
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<tr>
<td>(1 fresh, ½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apple juice (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oranges</td>
<td></td>
<td></td>
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<tr>
<td>Orange juice (½ cup)</td>
<td></td>
<td></td>
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<tr>
<td>Grapefruit (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grapefruit juice (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other fruit juices (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strawberries—fresh, frozen, or canned (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blueberries—fresh, frozen, or canned (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of Food</td>
<td>How Often</td>
<td>Size</td>
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<tr>
<td>--------------------------------------------------</td>
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</tr>
<tr>
<td>(Medium Serving)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peaches (1 fresh, ½ cup canned)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apricots (1 fresh, ½ cup canned)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plums (1 fresh, ½ cup canned)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Honeydew melon (¼ melon)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>VEGETABLES.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>VEGETABLE JUICE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tomatoes (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tomato juice (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tomato sauce (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spaghetti sauce (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red chili sauce, taco sauce, or salsa (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tofu or soybeans (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>String beans, green beans (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Broccoli (½ cup)</td>
<td></td>
<td></td>
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<tr>
<td>Cabbage (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cole slaw (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cauliflower (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brussels sprouts (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carrots, raw (½ carrot or 2-4 sticks)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carrots, cooked (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corn (1 ear or ½ cup frozen or canned)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peas (½ cup fresh, frozen or canned)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lima beans (½ cup frozen, or canned)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed vegetables (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beans or lentils, baked or dried (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Summer or yellow squash (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Winter squash (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zucchini (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yam or sweet potato (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinach, (cooked ½ cup, raw 1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iceberg lettuce, romaine or leaf (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Celery (4&quot; stick)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Beets (½ cup)  
Alfalfa sprouts (½ cup)  
Kale, mustard, or chard greens (½ cup)  
Vegetable, vegetable beef, minestrone or tomato soup (1 cup)  

<table>
<thead>
<tr>
<th>Type of Food</th>
<th>How Often</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Medium Serving)</td>
<td>Day</td>
<td>Week</td>
</tr>
<tr>
<td><strong>EGGS, MEAT, ETC.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eggs (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chicken or turkey, roasted or broiled with skin (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chicken or turkey, roasted or broiled skinless (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chicken, fried with skin (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacon (2 slices)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hot dogs (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low fat hot dogs (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sausage (2 patties or 2 links)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bologna (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other processed luncheon meat (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver, chicken or beef (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamburger, regular (1 patty, 3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamburger, lean (1 patty, 3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meat loaf (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pork, chops, roasts (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lamb (3-4 oz)</td>
<td></td>
<td></td>
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<tr>
<td>Beef, roast, steak (3-4 oz)</td>
<td></td>
<td></td>
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<tr>
<td>Beef stew with vegetables (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ham (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuna fish (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuna salad (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fish, baked or broiled (3-4 oz)</td>
<td></td>
<td></td>
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<tr>
<td>Fish, fried or fish sandwich (3-4 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shrimp, Lobster, Scallops</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pizza (2 slices)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed dishes with cheese (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lasagna or meat pasta dishes (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of Food</td>
<td>How Often</td>
<td>Size</td>
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<td>--------------------------------------------------</td>
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<td>------</td>
</tr>
<tr>
<td>(Medium Serving)</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Day</td>
<td>Week</td>
</tr>
<tr>
<td><strong>BREADS, CEREALS, STARCHES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cold breakfast cereal (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cold breakfast cereal—fortified (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cooked oatmeal (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other cooked breakfast cereal (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White bread (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pita bread (1 piece)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dark bread (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>English muffin (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bagel (1)</td>
<td></td>
<td></td>
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<tr>
<td>Dinner roll (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamburger or hotdog bun (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muffin (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biscuit (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corn bread, corn muffin (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brown rice (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White rice (1cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spaghetti noodles (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macaroni noodles (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other pasta noodles (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulgar, kasha, couscous (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancakes or waffles (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potatoes, french fries or fried (½ cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potatoes, baked or boiled (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mashed potatoes (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potato chips or corn chips (small bag or 1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saltine crackers (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saltine crackers, low sodium (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saltine crackers, fat free (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other crackers (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other crackers, low fat (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of Food</td>
<td>How Often</td>
<td>Size</td>
</tr>
<tr>
<td>-------------------------------------------------</td>
<td>-----------</td>
<td>------</td>
</tr>
<tr>
<td>(Medium Serving)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BEVERAGES</strong></td>
<td></td>
<td>S M</td>
</tr>
<tr>
<td>Regular soft drink (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet soft drink (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caffeine free soft drink (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caffeine free, Diet soft drink (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lemonade or other non-carbonated drink (1 glass, bottle, or can)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coffee (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decaffeinated coffee (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tea (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herbal tea (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beer (1 glass, bottle, or can)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red wine (4 oz glass)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White wine (4 oz glass)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whiskey, gin, or other liquor (1 drink or shot)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>SWEETS, BAKED GOODS, MISC.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chocolate (1 small bar or 1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Candy bar (1 small bar)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Candy without chocolate (1 oz)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cookies, home baked (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cookies, ready made (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brownies (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doughnuts (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cake, home baked (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cake, ready made (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweet roll, coffee cake, or other pastry ready made (1 serving)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of Food</td>
<td>How Often</td>
<td>Size</td>
</tr>
<tr>
<td>-----------------------------------------------------------------------------</td>
<td>-----------</td>
<td>------</td>
</tr>
<tr>
<td>(Medium Serving)</td>
<td>Day</td>
<td>Week</td>
</tr>
<tr>
<td>Sweet roll, coffee cake, or other pastry home baked (1 serving)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pie, homemade (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pie, ready made (1 slice)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jam, jelly, preserves, syrup, or Honey (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peanut butter (1 Tbsp)</td>
<td></td>
<td></td>
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<tr>
<td>Popcorn (1 cup)</td>
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<td></td>
</tr>
<tr>
<td>Popcorn, air popped (1 cup)</td>
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<td></td>
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<tr>
<td>Nuts (small packet or 1 oz)</td>
<td></td>
<td></td>
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<tr>
<td>Bran, added to food (1 Tbsp)</td>
<td></td>
<td></td>
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<tr>
<td>Wheat germ (1 Tbsp)</td>
<td></td>
<td></td>
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<tr>
<td>Chowder or cream soup (1 cup)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oil and vinegar dressing (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mayonnaise or other creamy salad dressing, Regular (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mayonnaise or other creamy salad dressing, Low Fat or Reduced Calorie, Lite (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mayonnaise or other creamy salad dressing, Fat Free (1 Tbsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mustard, dry or prepared (1 tsp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salt (1 shake)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pepper (1 shake)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Can you think of any other food or drink that you had in the past week that was not on this form? If so, what was it? What was the amount? How many times did you have it this past week?

Food__________________________________________________________

Amount__________________, How often per day______________, per week__________

Food__________________________________________________________

Amount__________________, How often per day______________, per week__________

Food__________________________________________________________
Amount_________________, How often per day_____________, per week__________

Food___________________________________________________________________________

Amount_________________, How often per day_____________, per week__________
Knee Pain Study

Subject ID_________
Date of Birth_______
Interviewer:____________________

VITAMIN AND MINERAL SUPPLEMENT USE QUESTIONNAIRE

1. Do you take any vitamin or mineral supplement(s)? Yes ___ No _____
2. If Yes, please, list all names of vitamin or mineral supplements, and how often do you take the supplement(s)?

Name ______________________ How often ____ per day Or ____ per week
Name ______________________ How often ____ per day Or ____ per week
Name ______________________ How often ____ per day Or ____ per week
Name ______________________ How often ____ per day Or ____ per week
Name ______________________ How often ____ per day Or ____ per week