Priority Setting in Musculoskeletal Disorders: A Focus on Osteoarthritis

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Priority Setting in Musculoskeletal Disorders: A focus on Osteoarthritis

1 Introduction

This report is part of a series that covers the methodology and role of priority setting within the Australian health care system. An earlier report discussed the relative strengths of the different priority setting techniques (Segal, L. and Y. Chen, Priority setting models for health. The role of priority setting and a critique of alternative models - full report, 2001, Centre for Health Program Evaluation. Research Report 22: Melbourne). The purpose of the present report, using musculoskeletal disorders as an example, is to identify a specific musculoskeletal disorder to focus on, and then to explicate the disorder in terms of epidemiology, progression, and impact on mortality and morbidity. Finally we broadly describe the current strategies and/or opportunities for the prevention and treatment of the disease in Australia.

1.1 Rationale for focusing on Osteoarthritis (OA)

An important purpose of this Priority Setting project is to test the Priority Setting Model in a single disorder where the majority of the disease burden results from morbidity rather than mortality. Further, it is desirable to select a condition which has the following three features:

- The disease is common and has a substantial impact on the health of the community and the health system;
- There are opportunities for intervention from across the disease spectrum, including at least primary prevention (requiring the existence of modifiable risk factors), and/or early diagnosis and management of those with identified disease;
- A range of disease management strategies are available which might include secondary prevention options and possible strategies to address iatrogenic complications or side effects, i.e. persons with established disease can be identified and alternative treatments evaluated.

Osteoarthritis is the most prevalent arthritic disorder and most commonly affects the knee and hip joints. It is the most common cause of musculoskeletal disability in elderly people. There is no clear clinical definition of OA, and marked changes can be present on X-ray in the absence of symptoms, or symptoms can be present with little evidence of deterioration on X-ray (see later). The condition is identified by the insidious onset of joint pain and stiffness. The main initial symptom is pain made worse by movement and eased by rest. As the joint deteriorates there is a loss of movement and joint instability occurs, pain becomes more pronounced and may be present at rest and at night.
Osteoarthritis is the most common type of arthritis, especially among older people. Sometimes it is called degenerative joint disease. Osteoarthritis is a joint disease that mostly affects the cartilage, the tissue that covers the ends of bones in a joint (see Figure 1a). Healthy cartilage allows bones to glide over one another. It also absorbs the shock of physical movement. In osteoarthritis, the surface layer of cartilage breaks down and wears away. This allows bones under the cartilage to rub together, causing pain, swelling and loss of motion of the joint. Over time, the joint may lose its normal shape. Also, osteophytes (small growths of bone or spurs) may grow on the edges of the joint. Bits of bone or cartilage can break off and float inside the joint space. This causes more pain and damage (see Figure 1b).

People with osteoarthritis usually have joint pain and limited movement. Unlike some other forms of arthritis, osteoarthritis only affects joints, and not internal organs. For example, rheumatoid arthritis—the second most common form of arthritis—affects other parts of the body besides the joints. It begins earlier than osteoarthritis, causes inflammation, and in addition to pain and loss of mobility may make people feel sick, tired, and feverish.
2 Musculoskeletal disorders: overview

Musculoskeletal disorders comprise a wide range of disorders of the muscles and bones but the most prevalent and well known are the arthritic disorders. The most common within this group are:

- osteoarthritis
- rheumatoid arthritis
- fibromyalgia
- systemic lupus erythematosus
- gout
- osteoporosis
- back problems

Each disorder has a different clinical presentation, a different set of causal factors, and the approaches to management vary. An important similarity is how musculoskeletal disorders interfere with mobility and therefore activities of daily living. Approximately one third of older adults report that they suffer from arthritis. In children, juvenile arthritis affects an estimated 0.3 to 4.0 per 1000 children and involves mild to profound disability, often diagnosed many years after onset [1, 2].

The primary health burden of musculoskeletal disorders is through a loss of quality of life associated with disability and pain. There is in addition a small but poorly defined mortality impact. Musculoskeletal impairments ranked number one in chronic impairments in the United States [3] and chronic musculoskeletal pain is reported in surveys by about 1 in 4 people in both less and more developed countries [4, 5]. Musculoskeletal conditions were the most expensive disease category in a Swedish cost of illness study, representing 22.6% of the total cost of illness [6]. Measured in terms of disability adjusted life years (DALYs), osteoarthritis is the fourth most frequent predicted cause of health problem worldwide in women and the eighth in men [7]. The Victorian burden of disease studies indicate that for women aged between 35 and 64, osteoarthritis alone is the third leading cause of DALY whereas it is the tenth for men of that age group [8].

During the development of this research project, osteoarthritis was identified as an appropriate condition within the musculoskeletal group for use in this priority setting exercise. The epidemiology and personal impact are discussed in detail in the next sections. A brief review the nature of other common musculoskeletal disorders is presented in Appendix I.
Table 1  Burden of musculoskeletal diseases: DALYs, cost of management and number affected

<table>
<thead>
<tr>
<th>Disease</th>
<th>Prevalence (per 1,000)(^a)</th>
<th>DALYs (1996)(^a)</th>
<th>Cost 1993-4 ($million)(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatoid arthritis</td>
<td>55.1</td>
<td>11,989</td>
<td>129</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>625.1</td>
<td>625.1</td>
<td>700</td>
</tr>
<tr>
<td>Chronic back pain</td>
<td>565.9</td>
<td>4,016</td>
<td>294</td>
</tr>
<tr>
<td>Slipped disc</td>
<td>340.1</td>
<td>3,875</td>
<td>N/A</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>155.2</td>
<td>2,555</td>
<td>60</td>
</tr>
<tr>
<td>Other musculoskeletal disorders</td>
<td>618.6</td>
<td>7,727</td>
<td>1,561</td>
</tr>
<tr>
<td>All musculoskeletal disorders</td>
<td>2,380.0</td>
<td>89,916</td>
<td>3,002</td>
</tr>
<tr>
<td>Asthma</td>
<td>1206.1</td>
<td>36,240</td>
<td>480</td>
</tr>
<tr>
<td>Diabetes</td>
<td>543.0</td>
<td>74,930</td>
<td>370</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>407.0</td>
<td>548,585</td>
<td>3,720</td>
</tr>
<tr>
<td>Cancers</td>
<td>N/A</td>
<td>478,580</td>
<td>1,900</td>
</tr>
</tbody>
</table>


3  Diagnosis, disease stages and prevalence

The diagnosis of OA is usually based on clinical and radiographic (X-ray) features. In the early stages, the radiograph may be normal although joint space narrowing may (or may not) become evident over several years as articular cartilage is lost. Other radiographic findings include bone thickening, cysts and osteophytosis (bony protrusions). A change in the contour of the joint, due to bony remodelling, and subluxation (partial dislocation) may be seen. Although joint space narrowing has been considered to be a radiographic indicator of articular cartilage thinning, in patients with early OA who do not have radiographic evidence of bony changes (e.g. bone thickening, cysts or osteophytes), joint space narrowing alone does not accurately indicate the condition of the articular cartilage. Similarly, osteophytes alone, in the absence of other radiographic features of OA, may be due to natural aging rather than to OA.

3.1  Primary versus Secondary Osteoarthritis

Primary, or idiopathic, is the most common type of OA and has no identifiable underlying etiology or predisposing cause. Secondary OA is a broad term given to cases where an underlying cause is known. Pathologically it is indistinguishable from primary OA. In some cases, the distinction between primary and secondary disease is unclear because the clinical presentation and symptoms of both classifications are often very similar. Clinically, it is generally not important to make a distinction between the two forms.

3.2  Clinical stages of OA

There is often great disparity between the severity of radiographic findings, the severity of symptoms, and function in OA. This was very clearly demonstrated in an analysis of the United States National Health and Nutrition Examination Survey I where 6,880 persons ages 25-74 were
Radiographic stage 2-4 knee OA was found in 319 subjects (3.7%) but only 47% of these individuals reported knee pain, and only 61% reported that a physician had told them that they had arthritis. Knee pain was reported by 1,004 subjects (14.6%), only 15% of whom had radiographic stage 2-4 changes of OA, and 59% of whom reported having a diagnosis of arthritis by a physician. A report of arthritis diagnosed by a physician was given by 1,762 subjects (25.6%), of whom only 11% had stage 2-4 radiographic knee OA and 34% reported knee pain. Substantial discordance exists between radiographic OA of the knee versus knee pain, versus a diagnosis of arthritis by a physician [10].

Given that there is no clear disease staging of OA and no way to detect early disease, magnetic resonance imaging (MRI) is being investigated as a method for identifying early stage disease [11]. It is uncertain how such technologically advanced and expensive imaging might change our understanding of disease staging and influence OA management practice [11]. Population-based methods are probably necessary, with regular follow-up scans over 10 to 15 years to validate MRI as a screening and management tool in OA.

Most epidemiologic studies have utilised the Empire Rheumatism Council system to classify stages of OA. It was first described over 30 years ago by Kellgren and Lawrence [9]. The system assigns one of five grades to OA at various joints; knee, hip, hand and spine (see table 2).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Classification</th>
<th>Description</th>
<th>Disability weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>normal</td>
<td>No features of OA</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>doubtful</td>
<td>Minute osteophytes, doubtful significance</td>
<td>N/A</td>
</tr>
<tr>
<td>2</td>
<td>minimal</td>
<td>Definite osteophyte with unimpaired joint space</td>
<td>asymptomatic   0.14  symptomatic 0.14</td>
</tr>
<tr>
<td>3</td>
<td>moderate</td>
<td>Moderate diminution of joint space</td>
<td>asymptomatic   0.14  symptomatic 0.42</td>
</tr>
<tr>
<td>4</td>
<td>severe</td>
<td>Joint space greatly impaired with sclerosis of subchondral bone</td>
<td>asymptomatic   0.14  symptomatic 0.42</td>
</tr>
</tbody>
</table>

### 3.3 Prevalence

Precise estimates of the prevalence of OA are not available for Australia, as the population-based studies using clinical investigations have not been undertaken. Furthermore, the lack of a clear definition of the disease and the insidious onset confound efforts to ascertain prevalence. In the 1995 ABS National Health Survey, respondents were asked to indicate whether they suffered from a number of chronic diseases, including osteo- and rheumatoid arthritis. In this survey, 6.4% of respondents reported that they had OA (or about 1.2 million, based on the 1995 Australian population of 18 million). A further 2.6% reported they had rheumatoid arthritis and 5.7% reported they had arthritis but could not specify whether it was osteo- or rheumatoid arthritis. Overall these data suggest that up to 14.7% of Australians suffer from arthritis, most of which is osteoarthritis. This may well be an overestimate due to the methodological limitations of self-report as some individuals without OA may report having the disease [12].
The prevalence of OA increases substantially with age [13, 14]. For those older than 50 years, 19% report that they have OA, 7% report that they have RA and a further 15% report that they have arthritis but do not distinguish between the two conditions. A total of 41% of Australians over the age of 50 report that they have arthritis (source: [12]).

In a community based survey in Northern Sydney, “arthritis or rheumatism” was the leading self-reported chronic condition reported by 59.5%, 55.8% and 59.7% of women and 40.5%, 47.0% and 43.6% of men in the three age groups (65-74, 75-84 and 85 years and over), respectively [15].

The most recent major report from the USA National Health Interview Surveys (NHIS) indicates that arthritis and other rheumatic conditions are among the most prevalent diseases in the United States and the most frequent cause of disability [14, 16, 17]. Using a system where respondents reported various musculoskeletal conditions, ICD-9 disease classification codes were generated via recommendations from the National Arthritis Data Workgroup [19]. The results indicated that 12.5% of males and 19.5% of female had arthritis (an overall rate of 16.1%) and the rates increase with age. Interpretation of these data are somewhat problematic as the data are self-report and include a wide variety of musculoskeletal disorders, although most would be due to OA.

A recent population-based survey of 39,240 Canadians used a definition of arthritis of; a long-term health condition of “arthritis or rheumatism” being diagnosed by a health professional [18]. The crude prevalence for those 20 years and over was 14.2%, however this varied substantially by gender and place of birth. The prevalence for women was 17.6% and 10.5% for men. The age-sex adjusted prevalence was 6.9% for those born in Asia, 14.2% in Europe/Australia, and 14.5% for those born in North America.

Overall, large population-based surveys indicate that the prevalence of OA in Australia or similarly industrialised countries is between 14% and 16% of all persons and that the disease is more frequent in older age groups and in women.

4 Aetiology and risk factors

Akin to most chronic diseases, OA appears to have a long induction period with several years between exposure to a risk factor and manifestation of the disease. The relative contributions of biological, lifestyle and socio-economic factors in OA is unclear. Genetic factors may play a substantial role, however observed ethnic differences in prevalence may in part be explained by lifestyle factors (activity and diet), or socio-economic factors. Classic twin studies indicate that more than 50% of OA may be due to genetic factors.

4.1 Physical activity, occupational and sports injury

There is some evidence that too much physical activity may lead to OA. The continuous stress that physical activity places on the joints can result in microtrauma and degeneration of the articular cartilage. The onset of osteoarthritis appears to depend on the frequency, intensity and duration of physical activity. Research has shown that individuals of all ages can tolerate moderate amounts of exercise without adverse consequences or accelerated development of osteoarthritis.

Recent influential reports have emanated from the Framingham Heart Study. McAlindon et al recently reported a 10-year follow-up of 473 elderly individuals who were radiographically asymptomatic for OA of the knee in 1983-85, completed a physical activity exam in 1988-1989,
and were then followed-up with knee radiographs and clinical exam in 1992-1993 [19]. The investigators reported an increased risk of radiographic OA for heavy physical activity (OR 1.3 per hour of exercise, 95% CI 1.1-1.6, p for trend 0.0006). A similar trend was also found for symptomatic OA. No increased risk of OA was found for medium and light physical activity. The risk was also considerably higher for those with both high physical activity and high BMI.

Jobs that involve physical activity that is repetitious, overworking the joints and fatiguing muscles that protect joints tend to increase the risk of OA in those joints [20]. Occupations that involve physical labour are associated with a higher incidence of OA [21-23]. In Norway, manual workers have nearly twice the probability of becoming a disability pensioner with osteoarthritis compared to professionals [24]. Occupational activities such as climbing stairs, walking on uneven ground, standing and sitting have been inconsistently linked to OA risk [22-24].

For overweight people in occupations that involve high physical activity the risk of OA is increased substantially. In theory therefore, prevalence of OA may be reduced through targeting this population. However further research is required to properly identify which particular activities lead to this increased risk [20].

Of interest is the role of recreational physical activity. A recent case-control study involving 55 men and 226 women aged 55-75 years receiving knee arthroplasty for primary osteoarthritis in a Finish university hospital, and 524 controls selected randomly from the population, found that the risk of knee osteoarthritis requiring arthroplasty decreased with increasing cumulative hours of recreational physical exercise. The adjusted odds ratios (95% CI) of knee arthroplasty were 0.91 (0.31-2.63) in men with a low number of cumulative exercise hours and 0.35 (0.12-0.95) in those with a high number of cumulative exercise hours, with a history of no regular physical exercise as the reference. For the women, the corresponding odds ratios were 0.56 (0.3-0.93) and 0.56 (0.32-0.98). This study demonstrated that moderate recreational physical activity is associated with a substantial decrease in the knee osteoarthritis requiring arthroplasty [25].

4.2 Obesity

Case-control studies have demonstrated a strong association between knee osteoarthritis and obesity [26]. Longitudinal studies, such as the Framingham study, also show that high BMI predicts the development of OA later in life [27]. Consistent trends of increasing risk of knee OA with increasing BMI have been observed in several additional cohort studies and are summarised below.

The US National Health and Nutrition survey (1971-1975), a population health survey, revealed strong trends for increasing risk of OA with increasing BMI. Women with a BMI between 30 and 35 kg/m$^2$ had almost four times the risk of knee OA when compared with women with a BMI under 25 kg/m$^2$.

Coggon et al found a surprising high risk of OA in obese people presenting for knee surgery when compared with population-based controls. When compared with people in the acceptable weight range (BMI 24 to 24.9 kg/m$^2$), those who were severely obese (BMI >35 kg/m$^2$) had more than a ten-fold risk of OA [29]. This study suggested that if all overweight and obese people reduced their weight by 5kg or until their BMI was within the recommended normal range, 24% of surgical cases of knee OA [95% CI 19 to 27] might be avoided.
Substantial evidence exists that indicates that BMI asserts the increased risk through physical mechanisms. The arthrogenic effect is primarily on the knee through structural mechanisms and it also increases the risk of hand and hip OA [29]. While the increased joint stress accompanying obesity may explain the strong linkage between obesity and knee OA risk, it does not necessarily explain why obese people have a high risk of OA in the hand nor why obese women are at higher comparative risk of knee disease than obese men [31, 32, 33]. Obesity is not only a risk factor for the onset of OA, but also a risk factor for radiological progression [32-34]. A striking finding has been that women who are obese but who subsequently lose weight have lower incidence of OA later in life [34]. Recent increases in weight are also associated with additional risk of OA [35].

Quadriceps strength is thought to be a predictor of future development of OA [36, 37, 38] however the determinants of quadriceps strength have not been well studied. It is likely that physical inactivity is the primary determinant. This is supported by studies that suggest physical activity reduces symptoms associated with OA [39].

4.3 Hormonal factors and bone density

A higher incidence of OA just after menopause has been noted which suggests that estrogen deficiency may play a role in the disease. There seems to be a complex relationship between bone mineral density (related to estrogen and physical activity), osteoporosis (i.e. weakening of the bones, particularly in elderly women) and OA. People with OA are less likely to have osteoporosis. There have been insufficient longitudinal studies to clarify these relationships, particularly in a way that may inform public health policy and lifestyle intervention programs.

4.4 Nutrition

There are very few studies that have systematically examined the role of diet as a risk factor in OA. Possible mechanisms involve the role of antioxidants, which when consumed at suboptimal level over many years (10 or more), lead to increased risk of many common diseases such as cancer and heart disease. Since antioxidants provide defence against tissue injury, high dietary intake is thought to protect against OA.

In the Framingham study, a 10-year follow-up revealed that antioxidants were not associated with incident OA, but a 3-fold reduction in risk of progression was found for both the middle tertile and highest tertile of vitamin C intake. This related predominantly to a reduced risk of cartilage loss. A lower risk of progression was also seen for those with higher beta-carotene intake [40].

Vitamin D, an important micronutrient in bone mineralisation, has also been studied in the Framingham study. The risk of radiological progression was increased threefold for persons in the middle and lower tertiles of vitamin D intake, but it was not associated with incident OA [41]. In another longitudinal study, low serum levels of vitamin D were associated with incident changes of radiographic hip OA characterized by joint space narrowing [42].

Dietary nutrients have often been regarded as useful by patients for self-treatment, the studies so far on aetiology have not produced conclusive evidence. The few rigorous longitudinal studies provide equivocal evidence that requires confirmation.
4.5 Genetics

Despite OA being a heterogeneous disease, studies of twins show that overall genetic factors account for at least 50% of hand and hip OA and a smaller percent of knee OA [45]. An additional study in Finland suggested that the effect may be stronger in women rather than in men [46, 47].

There have been several studies attempting to isolate which genes play the major role in the development of OA (reviewed by Loughlin, 2001 [46]), but to date these have only revealed several candidate regions on several chromosomes. These studies suggest that the genes that control the manufacture of some types of collagen and the vitamin D receptor may be involved in OA susceptibility. These studies suffer from the same problems of most that attempt to isolate the genetic causes of chronic disease, namely that there is likely to be several genes acting in concert, the disease is heterogeneous, and environmental factors are likely to play a substantial role in gene expression [48, 49].

4.6 Socioeconomic determinants

An association between socio-economic status (SES) and musculoskeletal disorders has been well documented and is a consistent finding with other chronic diseases – people of low SES tend to carry a larger burden of the disease [50-53].

Whether socio-economic factors are determinants of OA is difficult to confirm, due in part to the long induction period of the disease. Further, the disability associated with the disease leads to reduced opportunity for employment and lower education and may be associated with less effective management [54]. The evidence suggests that being poor is more likely to be a consequence rather than a cause of OA. Australian data demonstrate only modest association between OA and lower SES.

In the 1995 South Australian Health Omnibus survey, 3000 respondents aged 15 or over were asked “Have you ever been told by a doctor that you have arthritis?” and “What type?” Medically confirmed arthritis was self-reported in 666 (22.1%) as osteoarthritis (OA) (8.6%), rheumatoid arthritis (RA) (4.0%), and other, or unspecified arthritis (9.6%). People with arthritis were more likely to be female, aged, and of lower socioeconomic status [55].

In the 1995 ABS National Health Survey, 6.4% of the Australian population reported that they had OA. When the socio-economic status (in terms of SIEFA scores) was divided into quintiles, the prevalence in each of the quintile from lowest to highest was 7.5%, 6.8%, 6.1%, 5.8% and 6.0% suggesting a modest association with SES. After adjustment for age and sex, those in the lowest quintile are about 20% more likely to report having OA than those in the highest quintile (OR 1.18)\(^1\).

\(^1\) Source: Australian Bureau of Statistics. National Health Survey: Cat 4399.0; 1995.
### 4.7 Summary of risk factors for OA: Which are amenable to modification in the Australian Health Care setting?

Not all risk factors for OA are amenable to modification through general public health intervention, community interventions or one-on-one interventions with health professionals. Table 3 lists modifiable and non-modifiable risk factors. The most prevalent and strongest modifiable risk factors for OA are obesity and occupation. Quadriceps strength may also an important predictor of future development of OA, with physical activity the most likely determinant of quadriceps strength.

Thus interventions that focus on reducing obesity and increasing moderate physical activity as well as specific programs addressed at occupational risk and management of injury are likely to produce the highest reduction in the prevalence in OA. However there has not been any population or community studies that have tested this assertion.

Age, gender and genetics, while non-modifiable, identify potential target groups. For instance older women have an elevated risk of developing OA so programs might target them, particularly when other risk factors are evident such as obesity and physical inactivity. Similarly, primary prevention programs might target certain occupations involving weight bearing activities. While socio-economic factors may not be directly amenable to change, the underlying behaviours that are often the cause of socio-economic gradients may be manipulable, through for instance targeted education (particularly around healthy lifestyle behaviours) to reduce OA incidence.

### Table 3 Risk factors for Osteoarthritis

<table>
<thead>
<tr>
<th>Potentially modifiable</th>
<th>Not readily amenable to modification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity</td>
<td>Age</td>
</tr>
<tr>
<td>Physical inactivity</td>
<td>Gender</td>
</tr>
<tr>
<td>Quadriceps strength</td>
<td>Genes</td>
</tr>
<tr>
<td>Occupational and recreational injury</td>
<td>Socio-economic status</td>
</tr>
<tr>
<td>Diet</td>
<td></td>
</tr>
</tbody>
</table>

### 5 Mortality and morbidity burden due to Osteoarthritis

#### 5.2 Mortality

While OA is a chronic progressive disease for which a substantial impact on quality of life is well documented [53, 54], there has been only a limited number of studies of whether people with OA are at increased risk of early death. Early estimates of increased risk of death of those with OA compared with the general population have ranged from 11% to 45% in women [55]. While studies suggest no significant increase in mortality associated with OA in men [56]. However, these studies have serious methodological flaws making interpretation difficult.

A more recent study based on a cohort of 296 women aged 42-76 years who were employed in the US radium dial-painting industry between 1915 and 1945 and followed up for between 3 and 28 years found that those with full-body radiographically defined OA had moderately decreased survival times. The hazards ratio per 3 additional joint groups involved was 1.56 (1.24-1.97) [57].
This corresponds to a small absolute relative risk of death (0.02 to 0.5 percent) but the exposed population is large. The authors proposed that the mechanism may involve reduced physical activity and the effects of treatments.

A contributor to additional mortality in persons with OA is iatrogenic disease, notably resulting from perforated stomach ulcers. In the United States, gastrointestinal complications induced by nonsteroidal anti-inflammatory drugs (NSAIDs) cause more than 100,000 hospitalisations and about 16,500 deaths annually [58]. A similar proportion of deaths are thought to occur in Australia (Personal communications; L March, N Bogduk 2001).

In summary, those with OA appear to have increased mortality but the currently available evidence make estimates of the magnitude unreliable.

5.2 Morbidity

The morbidity impact or loss of quality of life from OA derives from two main sources; pain and reduced capacity to undertake various activities. The joint pain of OA is often described as a deep ache and is localised to the involved joint. Generally the pain of OA is present during joint use and relieved by rest, but as the disease progresses, it may become persistent. Pain at night tends to interfere with sleep, particularly in advanced OA of the hip. Stiffness of the involved joint upon arising in the morning or after a period of inactivity may be prominent but usually lasts less than 20 minutes. As the disease progresses, decreased quality of life occurs through more severe pain, limitations to activities, deformation and, for some confinement to wheelchairs. A hypothetical disease pathway is shown in Figure 2.

**Figure 2 Proposed disease pathway of osteoarthritis**

| Onset of Osteoarthritis | Symptoms (eg pain, stiffness) | Disability (eg difficulty walking) | Handicap (eg difficulties performing activities of daily living) |

The assessment of the morbidity impact of OA has involved four types of instruments; i) disease specific instruments (eg the Western Ontario and McMaster Universities osteoarthritis index; WOMAC), ii) instruments focused on a particular dimension of quality of life most commonly pain (eg the McGill Pain Questionnaire) iii) generic health status instruments (eg the SF-36) and iv) utility instruments (EQ5D) [59-61].

Evidence of loss of quality of life with OA is widely documented in population surveys, cohort studies and intervention studies. These suggest loss in quality of life is both substantial and significant. Table 2 shows disability weights associated with the various stages of the disease – the severest symptomatic stages (3 and 4) are associated with a weigh of 0.42, ie about 42% of health the of someone at full health without disease. The morbidity impact of OA will be documented in detail in subsequent reports.
5.3 Natural course of Osteoarthritis

The natural course of OA can only be properly studied in large cohort studies where full OA-specific clinical details are collected at baseline and participants are followed for many years. There are, however, very few studies that have examined this aspect of OA and fundamental difficulties with the definition and measurement of OA limit the value of the findings. The main difficulties are that the definition(s) of OA is(are) imprecise and the relationship between radiographic definitions (e.g. presence of osteophytes or joint space narrowing) have a poor concordance with patient-related outcomes such as pain, stiffness or limitations to activities of daily living.

To aid the understanding of OA progression, Klippel and Dieppe [63] tabulated studies of the natural history of knee and hip OA published between 1970 and 1995. Eight studies of knee OA were identified and only four studies of hip OA were identified. Follow-up ranged from 1 year to 15 years, there was a mix of clinical and radiographic measures and the number of subjects ranged from 31 to 353. For knee OA, the percent of subjects whose OA deteriorated over the follow-up ranged from 22% (52 subjects followed up radiographically for 2 years [64]) to 72% (350 knees followed up radiographically for 2 years [65]). Some of these studies, as well as more recent studies, are briefly described below.

The US Framingham study provides some of the clearest evidence regarding the development of OA in those initially without any disease [35]. In this study 869 people without OA at baseline participated in an 8-year follow-up study (mean age 70.8 years at baseline). About 16% developed OA in any joint. Rates of incident knee disease were 1.7 [1.0-2.7] times higher in women than in men. Progression of the disease was unrelated to gender and rates did not vary by age. Among women, approximately 2% per year developed incident radiographic disease, 1% per year developed symptomatic knee OA, and about 4% per year experienced progressive knee OA. No data were presented on the progression of the disease in people with established disease [35].

Evidence of OA incidence and progression in a general population study has also been obtained from the UK Chingford study [31]. In a 4-year follow-up, 715 paired radiographs were graded for osteophytes and 644 for joint space narrowing. Incident knee osteophytes developed in 95 women (13%), equating to an incidence of about 3% per year. Incident knee osteophytes increased by 20% per 5-year age increase. The study provided poor data on progression; of the 115 women with osteophytes at baseline only 1 appeared to have improved, and among the 186 women who had joint space narrowing, 11 appeared to improve. No data was reported on the degree or progression of the disease.

In a one-year follow-up study of patients with OA of the knee a small deterioration of the joint space narrowing was determined radiologically and evaluated on a 6 grade scale. In 360 patients, between 8% and 16% were found to have an improvement in radiologic features whereas between 27% and 38% of patients had worsening OA [33]. Changes in the joint space narrowing were more closely correlated with treatments received by the patients for OA (NSAID intake, synovial fluid aspiration) than with changes in the recorded clinical variables (pain on a visual analog scale, Lequesne algofunctional index). Moreover, some factors such as obesity, generalized OA and flares of OA appeared to be correlated with the deterioration of joint space narrowing.
In a 6 to 8 year retrospective longitudinal trial of 61 patients who underwent total hip arthroplasty, Conrozier et al found that the yearly mean joint narrowing ranged from 0.03- 2.5 mm/yr (median 0.29). The most rapid progression of joint space narrowing was associated with older age and absence of osteophytes [66].

In a 10-year follow-up of 169 consecutive patients who initially presented with OA of the hands or knees, Harris et al 1994 [67] found that the rate of progression was highly variable, depending on the site of the disease and classification system. For knee OA, disease progression defined radiographically was evident in 36% of patients (20 from a total of 55).

Current evidence produces a very unclear pattern of the rate of disease progression in OA and only general statements can be made. The majority of people with OA appear to have a disease that is progressive. Those with incident (and therefore early) disease progress slowly over the first few years (approximately 4% in women [35]), but radiographic progression appears to occur in 20% to 30% of those with established disease [31, 33, 67].

It is of great importance to establish quality of life changes during the natural course of the disease. Given that the concordance between radiographic progression and quality of life has not been formally studied in longitudinal studies, extrapolation from pathology is necessary. A recent study of the validity of various definitions of radiographic progression suggests that several have strong associations with knee pain [68]. Given that pain is a principal determinant of quality of life, it may be assumed that decrements in quality of life and pathology are paralleled. For some people, orthopaedic surgery is possible (e.g. knee or hip replacement) and quality of life may suddenly increase. These issues will be further explored in subsequent reports.

6 Opportunities for impacting on Osteoarthritis in Australia

OA is a complex disease, about which much remains unknown. Opportunities for reducing disease burden can be postulated based on theoretical considerations and available evidence, despite incomplete knowledge of the natural course of the disease and how this is influenced by alternative approaches to management.

6.1 Primary prevention

As noted, the incidence of OA is related to a number of modifiable risk factors: obesity, occupational/recreational injury and possibly quadriceps strength, and diet. As these risk factors are potentially amenable to public health interventions and targeted clinical programs, there is an a priori case for suggesting OA may be preventable, or at least that the incidence of OA may be reduced through programs to modify lifestyle behaviours. In sum, primary prevention is a plausible approach to the reduction of disease burden in OA.

6.2 Management for persons with diagnosed OA.

There are various types of management options to reduce disease burden for persons with OA. Management is primarily concerned with symptom relief rather than modification of the disease process. Specifically management is focused on pain relief and maintaining as far as possible the capacity to engage in normal daily activities, and reduce disability.

Opportunities for management of OA have been classified into the following broad groups:
1. Non-medical / non-surgical
2. Medical / pharmacological (including intra-articular pharmacologic)
3. Surgical

Each of these branches of health care will be discussed in detail in subsequent reports, particularly the evidence surrounding the clinical effectiveness of each component. Each is now introduced with respect to the stage of the disease at which they tend to be used, and from which segment of the health care sector they are administered.

### 6.2.1 Non-medical / non-surgical

This is the broadest category of OA treatment. It includes chronic disease self-management programs, allied health services notably physiotherapy, nutriceuticals and complementary/alternative medicines.

Recognition of the role for patient self efficacy as a central component of the management of chronic disease has become more apparent in recent years and is evidenced by the recent Federal Government Chronic Disease Prevention and Management Initiative ([http://www.health.gov.au:80/publth/chronic/index.htm](http://www.health.gov.au:80/publth/chronic/index.htm)). The aims of such programs include disease education, lifestyle modification, psychosocial therapies, peer-support, and general augmentation of self-efficacy. They may also result in reduced use, or more appropriate use of pharmaceuticals.

Such programs may be delivered in various ways; by consumer groups and can be peer lead, supported by allied health personnel, or delivered by general practitioners. General practitioners are generally the first contact for people who are developing the pain and stiffness associated with OA onset. Policy initiatives akin to the recent dietary counselling required prior to the administration of GP-prescribed cholesterol lowering drugs may be possible. For example, practice guidelines may be developed where weight loss and physical activity counselling may be implemented alongside administration of pharmaceutical treatments.

Support for healthy lifestyle behaviours in those with OA and at risk could well be implemented jointly with programs targeted at other chronic diseases such as CVD and diabetes where the risk factors are similar.

Allied Health is a potentially large component of OA management. It can comprise physiotherapy which may be applied directly for symptom management, or it may be applied pre and post surgery thus facilitating rehabilitation. It can be focussed on pain relief and/or involve specific strategies to enhance activities of daily living. It can comprise many forms of direct application (massage, transcutaneous electrical nerve, heat etc) or support to patients through specification of stretches or, more generally, exercise programs.

Occupational therapy involves assistance to patients to directly maintain/enhance their activities of daily living through, for instance, the provision of aides and supports, and specific techniques and skills. Such intervention may not only be directly beneficial but also prevent falls and further disability.

Osteopathy and chiropractic treatments also involve massage, stretches and manipulations. Other complementary medicine techniques include acupuncture, herbs, homoeopathy, magnets etc many of which are untested and the prevalence of usage is unknown.
A final class of non-medical / non-surgical treatments are the range of ‘complementary medicines’. These include nutriceuticals (refined nutrients or nutrient-like compounds) such as glucosamine and chondroitin, which are claimed to reduce symptoms and disease progression through modifying biochemical pathways. Other complementary medicines suggested for use in OA include herbs, homeopathics, meditation, traditional Chinese medicine, naturopathy, and many others.

6.2.2 Pharmacological treatments

The most common pharmacological treatments are simple analgesics, particularly paracetamol. This is usually administered early in the disease process and continues to provide some relief even in the later stages of the disease. Non-steroidal anti inflammatory drugs (NSAIDs) are administered to reduce pain and inflammation. The ‘new’ NSAIDs, COX-2 inhibitors, appear to have a similar effect as traditional NSAIDs, but with fewer gastric side effects. They may however be associated with certain renal and cardiac side effects, and are much more expensive than traditional NSAIDS. For late stage disease, where other forms of pain control have failed, opioids are sometimes administered.

6.2.3 Intra-articular pharmacological treatments

In situations where the disease is severe, painful and inflamed, corticosteroids may be injected into the site with the aim to reduce discomfort. Hyaluronans, the shock absorbing substance usually found between cartilage and bone, may also be injected into the joint with the aim of reducing pain. Tidal irrigation, or ‘washing out the joint with salty water’ is sometime undertaken.

6.2.4 Surgical treatments

The main form of surgical treatment is joint replacement (arthroplasty), which can offer a sudden reduction in pain and increased movement for most people. In late-stage disease one or more joints may be totally replaced. The modern materials used in replaced joints tend to last between 5 and 15 years, and the prostheses are generally replaced after a period of 10-15 years [71]. A common procedure in early OA, particularly for athletes, is arthroscopy, which is designed to clean up damaged tissue within the joint.

6.3 OA prevention and management: opportunities for priority setting

To make decisions about the allocation of resources to reduce the disease burden associated with OA, a combination of the best features of the evidenced-based Program Budgeting and Marginal Analysis (EBMA) model and the Health-Sector-Wide Disease-Based Model (HSW-DBM) has been adopted as the Priority Setting Framework [1]. This Framework requires the selection of a comprehensive set of intervention options and the conduct of comparative cost-effectiveness analyses to establish the most and least cost-effective means to intervene to reduce disease burden, from OA.

Thus an important task in this framework is the selection of a set of interventions on which to conduct cost-effectiveness analyses. An understanding of the opportunities to intervene to reduce disease burden is central to that choice.

Using the results of such comparative cost-effectiveness analyses it should be possible to establish a mix of preventive and management strategies that will minimise the disability burden.
due to OA. Once specific intervention strategies are selected for these analyses, which need to be drawn from all the possibilities, data on effectiveness and costs must then be collated to enable cost-effectiveness ratios to be developed for each intervention. In this way those options that are more cost-effective and those least cost-effective can be identified and resource shifts recommended.
Appendix I  Summary of other musculoskeletal conditions

Rheumatoid arthritis (RA)

RA affects 1% to 2.5% of the population and is more common in women (60% to 70%). Although the disease is most obvious in the joints, it is a systemic disease affecting other organs. It is characterised by a persistent and symmetrical peripheral inflammatory arthritis and about 70% of cases occur between the ages of 25 and 59 years [71, 72].

The cause of rheumatoid arthritis is unknown, although infection and autoimmunity have been proposed. Genetic factors play a small but definite role in some families. The classical presentation is of a woman in her mid-thirties with pain, stiffness and swelling of several weeks duration in the small joints of the hands, wrists and feet. Later in the course of the disease about 25% of people develop rheumatoid nodules, hardened lumps under the skin often around bony sites such as elbows, hips, heels, and back of the head. Cartilage and bone destruction occurs if joint inflammation persists. This leads to deformation of joints and leads to immobility. In addition to joint deterioration, people more severely affected may also experience weight loss, low-grade fever and malaise.

In general, 25% of people with rheumatoid arthritis remain fit for all normal activities, 40% have moderate impairment of function, 25% are quite badly disabled and about 10% become bound to a wheelchair.

Early diagnosis and appropriate treatment can limit the progression of the disease in some cases, but generally the disease slowly progresses and the average life span is reduced by about 7 years in males and 3 years in females [73-75]. Rheumatologists generally believe that the clinical picture of rheumatoid arthritis has improved considerably from that of a generation ago due to modern treatments, however this has been difficult to establish [73, 76].

Fibromyalgia

Fibromyalgia was first conceived as a diagnosis in 1981 to characterise widespread pain and increased sensitive to pressure at several specific anatomical sites [77]. It occurs predominantly in women and affects approximately 2-3% of people in industrialised societies. Men are less commonly affected (10%) when compared with the prevalence in women (90%). The condition is not considered to be a formal medical syndrome by all physicians [78, 79] however the brief description of the condition above, forms the basis of the American College of Rheumatology diagnostic criteria.

The etiology is very poorly understood. Dysfunction of the neuroendocrine system that leads to aberrant central pain mechanisms with central sensitisation has been suggested [80].

It is thought to be a disorder of pain amplification due to increased sensitivity of the pain sensory system. Management of simple fibromyalgia involves education regarding the nature of the problem, an exercise program and advice on stress management. The management may be described as “flexible and holistic” and may involve relaxation programs, physical therapies, cognitive behavioural therapy and analgesic medication.
Systemic Lupus Erythematosus (SLE)

Systemic lupus erythematosus is an autoimmune disease that affects many organs of the body. A significant genetic component to susceptibility is recognised and some environmental risks are known. It occurs worldwide but a higher incidence has been found in the US among blacks and Hispanics, as well as among Australian Aborigines [81]. It occurs far more commonly in women compared with men (10:1) and predominantly in younger women. The prevalence is approximately 0.1% [82]. It shortens life expectancy, causes substantial morbidity. About 90% of people survive ten years [71].

Over the past 4 decades, the incidence of SLE has nearly tripled, but there has been a small improvement in survival, which is likely to be due to a combination of improved recognition of early disease and better approaches to therapy [82, 83].

It is a chronic inflammatory disease that affects many parts of the body, including joints, kidneys, blood cells, heart and lungs. Common symptoms include arthritis, sensitivity to light, rashes, skin ulcers and fevers.

Gout

Gout is a metabolic disease characterised by recurrent attacks of acute arthritis affecting one or more joints of the extremities, increased serum uric acid concentration (hyperuricemia), and deposits of monosodium urate monohydrate crystals in the articular cartilage and synovial membrane. These crystals, when released into synovial fluid, provoke an inflammatory response. The US prevalence of gout is approximately between 0.4% and 0.8% and is about three times more prevalent in men than women [71]. Treatment involves dietary changes and by joint aspiration and NSAIDs, or colchicine.

Back pain

Back pain is a common disorder but a clear description of the epidemiology of the disorder is hindered by the lack of a clear definition [84] and problems with research designs [85]. In Victoria, back pain accounts for 25% of all Workers Compensation Claims, 40% of all long-term claims and nearly 50% of the cost of all claims [86]. Management follows recent evidence that continuing or resuming ordinary activities is more effective than rest and that early investigation and referral to a specialist are unwarranted in most cases. Population-based education programs appear to improve the management and reduce disability [87].

Osteoporosis

Osteoporosis is defined as a reduction of bone mass (or density) or the presence of a fragility fracture. Reduction in bone tissue is accompanied by deterioration in the architecture and strength of the skeleton leading to a markedly increased risk of fracture and substantial pain and disability. It occurs more frequently in older people as bone tissue is progressively lost. In women, the loss of ovarian function at menopause (typically after age 50) precipitates bone loss such that most women meet the criteria for osteoporosis by age 70. In a US study, 12% of the population was estimated to have clinical signs and symptoms of the disease [71]. A recent study of Australian women which examined bone mineral density of the spine, femoral neck, or midforearm indicated that osteoporosis was present in 0.9% of those aged 40-44 years and 87% of those aged 79 of older [88].
There are several known potentially modifiable risk factors which include current cigarette smoking, low body weight, oestrogen deficiency, early menopause (<45 years) or bilateral ovariectomy, prolonged premenstrual amenorrhea (>1 year), low calcium intake, alcoholism and inadequate physical activity.

Osteoporosis is characterised by long bone fractures and/or other fractures (e.g., vertebral, rib, and pelvic fractures). Treatment frequently involves management of acute fractures as well as treatment of the underlying disease. Hip fractures almost always require surgical repair if the patient is to become ambulatory again. Procedures may include pins and plates, and arthroplasties. Surgical procedures are followed by intense rehabilitation in an attempt to return patients to their normal function. Small fractures are usually managed supportive care only, with no specific orthopaedic treatment.
References


