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Review

Epidemiology

Epidemiology of osteoarthritis of the hand and wrist

LN Banks[1], TR Lindau[2]*

Abstract

Introduction

Osteoarthritis is more common than any other joint disease, and patients often ask what the cause is, enquiring about environmental causes, such as previous occupation. The available literature suggests that factors such as increasing age, gender (female), obesity and genetics may all lead to the development of osteoarthritis of the hand or wrist. It is a multifactorial, heterogenous and complex disease.

This review article presents the evidence that aging, female gender and hereditary factors are the most compelling culprits in the culmination of the primary hand and wrist osteoarthritis.

Materials and methods

Extensive literature search was carried out.

Results

As people age, the risk of developing osteoarthritis increases, with over 90% of those over 80 years of age being afflicted. For patients over the age of 60 years, the rate at which osteoarthritis progresses also increases. The prevalence of osteoarthritis at the base of the thumb has been shown to increase more rapidly in women than in men, with earlier peak prevalence in women. There is questionable evidence about hormonal links and hand osteoarthritis, rather chromosomes, genes and human leukocyte antigen-types are linked.

Co-morbidities, infection, trauma and joint laxity (repetitive trauma) may also lead to the development of the secondary osteoarthritis.

Discussion

A genetic predisposition appears to be the most powerful predictor of osteoarthritis. Hand osteoarthritis affects predominantly women, and their hand arthritis is more likely to progress faster than in males.

Conclusion

Aging, being female and hereditary factors are the most compelling culprits in the culmination and progression of the primary hand and wrist osteoarthritis.

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Introduction

Osteoarthritis (OA) is more common than any other joint disease, affecting around eight million people in the United Kingdom[1,2]. Worldwide, it has been estimated to be the fourth leading cause of disability[3].

In the hand and wrist, the most common site of OA is at the base of the thumb (trapezio-metacarpal joint (TMC) joint or first carpo-metacarpal (CMC I) joint), with radiographic changes indicative of OA (joint space narrowing, osteophytes, cyst formation, sclerosis) found in up to 81% of the elderly population[4] (Figures 1–5). The most popular radiologic OA classification among studies is the Kellgren and Lawrence classification, 1957[5–16]. There may be some question as to how robust it is as a classification system and that by using it, errors in classification (18%) of cases may be generated[15,17].

In today's increasingly litigious society, patients often want to know what has 'caused' their arthritis fuelled by example, *'disability benefits attorneys'* claiming that 'synthetic chemicals' may be to blame[18].

Hand OA includes the thumb base, metacarpophalangeal (MCP) joints, distal interphalangeal (DIP) and proximal interphalangeal (PIP) joints (Figure 1). OA may be either primary or secondary.

The primary OA is a multifactorial, heterogenous and complex disease.

The secondary OA is caused by co-morbidities, infection, trauma and joint laxity (repetitive trauma) (Table 1).

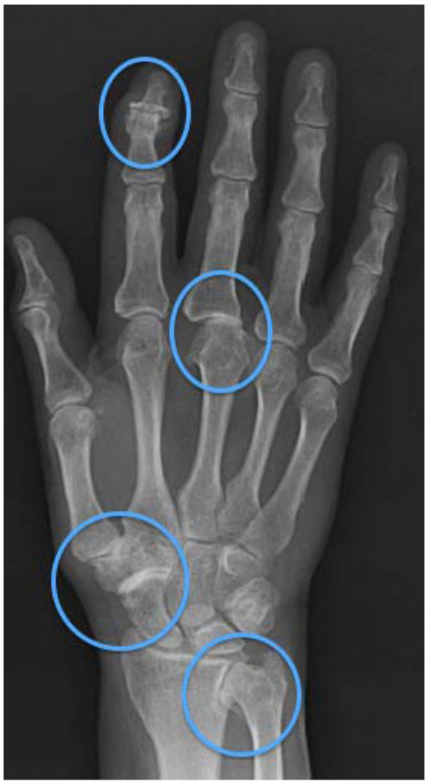

Figure 1: Radiograph of a patient with hand and wrist (index DIPJ, middle MCPJ, CMCJ-I, scapho-trapezio-trapezoid, radio-scaphoid and distal radioulnar joint OA).

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Table 1: Classification of OA and probable associations	
OA type	**Probable associations**
Primary	Increasing age Gender (female) Genetics Race Occupation (heavy labour or repetitive hand use) Obesity
Secondary	Trauma Co-morbidities Infection

OA: osteoarthritis.

This article reviews the literature on the epidemiology of the hand and wrist so that patients may be better informed when they present to their physician.

Materials and methods

Search methods

The following resources were searched:

Evidence-based reviews (The Cochrane Library, DARE, HTA Database); National Guidelines Clearing House and Specialist Libraries (MEDLINE / CINAHL / BNI / AMED / EMBASE/HBE/PubMed, TRIP database, SUMSearch) with no search limits enforced.

The following search terms were used: 'epidemiology', 'osteoarthritis', 'hand', 'wrist ± joint', 'age factors', 'continental population groups', 'occupation' 'occupation related phenomena', 'casualty/trauma', 'genetic epidemiology', 'hand joint ± pain', 'hand osteoarthritis', 'wrist disease ± wrist pain', 'race ± race difference', 'gender ± sex'.

Results

Primary OA

Age

As people age, the risk of developing OA, not surprisingly, increases, with around 10% of those over the age of 60 years and over 90% of those over 80 years of age being afflicted[2,19]. In a Baltimore Longitudinal Study of Aging[6], it was found that the prevalence of OA in both the DIP and PIP joints becomes progressively higher as the age of the subjects increased[6]. It was also found that OA progresses in those joints at a faster rate in the older population, compared with subjects who were less than 60 years of age[6].

Sex/gender

Generally, OA is known to affect women more than men, and there is no difference with OA in the hand[7,19,20]. The prevalence of OA at the base of the thumb has also been shown to increase more rapidly in women than in men (94% versus 85% in over 80 years of age) and is more likely to lead to '*complete joint destruction*' in the female population[19] (Figures 1 and 2). The peak prevalence in women is also earlier than in men (60–79 years vs. 75–84 years, respectively)[21].

The genes that have been implicated in the development of OA have been postulated to act differently between the sexes[22]. There is conflicting evidence between an obvious female hormonal relationship and hand OA[23]. A cross-sectional Tasmanian study (n = 348; Cooley[24]) noted a high prevalence of hand OA (65–70%)[24]. Their preliminary findings suggested that exposure to oestrogen (endogenous or exogenous) around the time of disease onset may have a '*priming*' effect on the severity of DIP joint OA, while breast-feeding in earlier life may be protective for CMC joint OA[24]. This is contradicted by findings without relationship between female hormonal aspects and OA of the hand (hip and knee)[23].

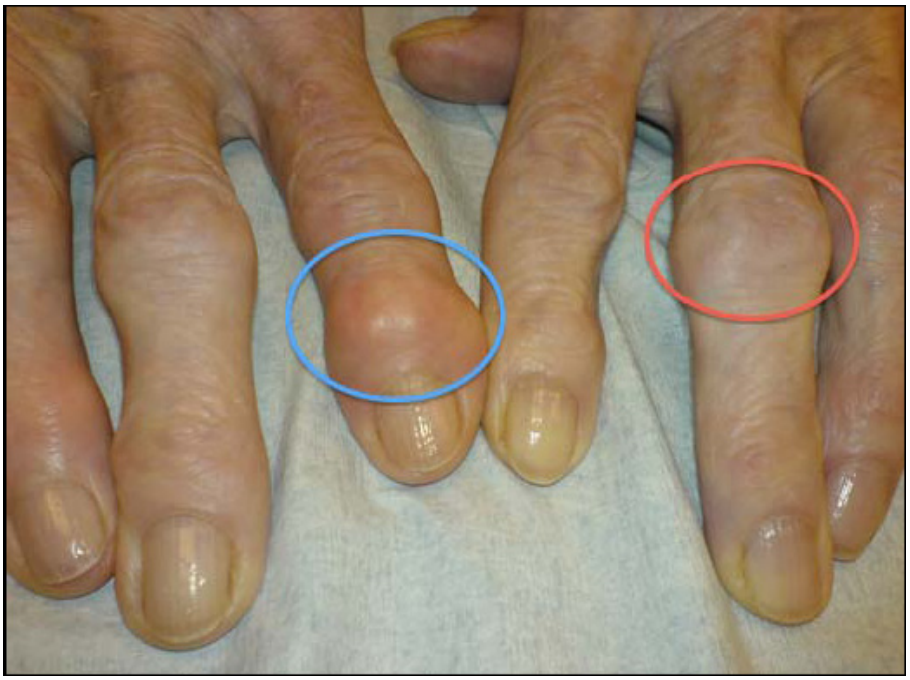

Figure 2: Clinical features of hand OA (Heberden's nodes highlighted in blue and Bouchard's in red).

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Data taken from the Baltimore longitudinal study of aging[25] have demonstrated that people with radiographic OA 'lose bone at different rates than those with normal radiographs and that this relationship varies between the site of OA and the site of measurement of body mass density'. In 437 Caucasian subjects (aged 20 years and above) looked at radiographically, it was found that females with OA of the hand had a 'significantly greater adjusted rate of bone loss at the radius than women with normal hand radiographs'. This difference was not noted in men for hand OA[25].

Genetics

Genetic factors are 'strong determinants of the disease'[26]. Studies looking at family history epidemiology and clustering, twin studies and associations between rare genetic disorders have all given information for the evidence of genetic factors on OA.

A Swedish group looked at the prevalence of radiographic hand OA with radiographic knee OA (after meniscectomy) in 170 patients and concluded that there were hereditary and environmental risk factors for OA, rather than hand OA being a sequelae post-knee surgery[8]. Twin studies have shown that the influence of genetic factors is between 39% and 65% in radiographic OA of the hand in women[22]. Twin studies examining OA of the hand, knee, hip and spine have suggested that OA has a heritability of 50–59%, and that there is a substantially increased risk of developing hand OA in first-degree relatives (siblings, parents, offspring)[26]. Table 2 illustrates proposed hereditary linkages to OA.

In explaining the often-haphazard way in which OA presents in one subject, it has been hypothesised that the genes that are in question may act on different sites within the body and on different disease features within those body sites[26] (Table 2). It has been highlighted in the literature that

Table 2: Proposed linkages, amongst others, to primary OA

Linkages to OA		Authors
Chromosomes	2q 9q 11q 12 16p	Cicuttini et al. (1995)[17] Spector et al. (1996)[22] Doherty (2008)[27]
Genes	Vitamin D receptor AGC1 (aggrecan) insulin-like growth factor-1 Oestrogen receptor alpha Transforming growth factor beta CRTM (cartilage matrix protein) CRTL (cartilage link protein)	Spector(2004)[26]
HLA	HLA-A1B8 HLA-B8	Brodsky et al. (1979)[50] Pattrick et al. (1989)[51] Doherty (2008)[27]
Collagen	II IX Type II pro-collagen (COL2A1)	Spector (2004)[26] Doherty (2008)[27]
Others	α1 antitrypsin isoform patterns	Pattrick et al. (1989)[51] Doherty (2008)[27]

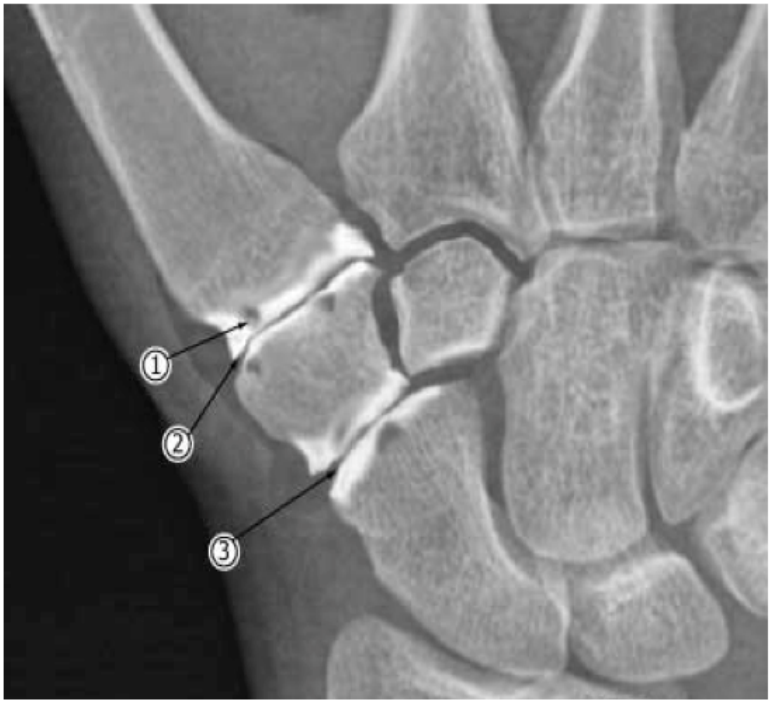

Figure 3: Radiograph of a patient with OA at the base of the thumb (CMCJ-I) and between the trapezium and the scaphoid. 1 = cystic changes, 2 = sclerosis and osteophyte formation, 3 = loss of joint space.

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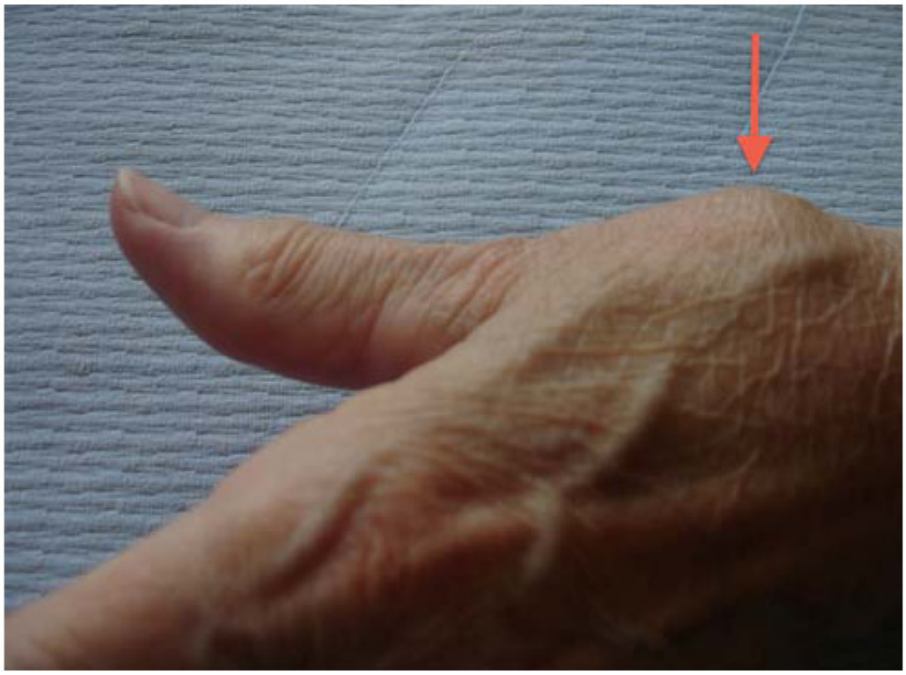

Figure 4: Clinical features of OA at the base of the thumb (CMCJ-I), illustrating a characteristic 'shoulder' or 'squaring' of the joint (highlighted by the arrow).

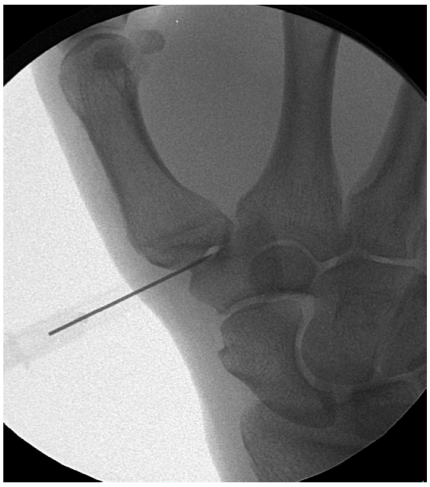

Figure 5: Early treatment of CMCJ-I OA – intra-articular steroid and local anaesthetic injection using radiological guidance.

gene association in OA is difficult given the 'case definition, late age of phenotype expression and other confounding (environmental, constitutional) factors'[27].

A study of 1086 twins from the Twins UK Adult Twin Registry examined leucocyte telomere length (LTL), which is a bioindicator of ageing and compared this with radiographic hand OA[14]. After adjustment for age, sex, body mass index (BMI) and smoking, it was found that a shorter LTL—equivalent to around 11 years of annual loss in normal people—was associated with hand OA (radiographic) and disease severity[14]. They suggested that potential shared mechanisms existed between OA and ageing, implicating 'oxidative stress and low-level chronic inflammation in both conditions'[14]. Another twin study found that OA affecting in particular the PIP joints, failed to show any genetic effect[22].

An inherited type of OA is associated with familial calcium pyrophosphate. This disorder leads to crystals (containing calcium) being deposited in the joint tissue. The disease has an autosomal mode of inheritance and can progress to severe degenerative OA[22]. It may be argued that OA here is secondary to pyrophosphate deposits.

Race

A Japanese study (n = 551 women; Toba[10]) found that the prevalence of OA was lower in the thumb CMC joint and higher (22%–30%) in the thumb IP joint compared with those in Caucasian women (7%–9%) reported previously[10]. They concluded, however, that the pattern of disease of (radiographic) hand OA was similar among the Japanese and Caucasian female population in terms of symmetry and carpal row affected.

In neighbouring China, population-based surveys including 241,169 adults indicated that the prevalence of symptomatic OA ranged from 5% to 21% with the prevalence of hand OA being much lower in Chinese than in Caucasian populations[28].

Another Asian (Korea) study found that the prevalence of thumb IP joint OA was higher than in Caucasians with a lower incidence of thumb CMC joint OA[16]. This has been postulated in a couple of studies to be associated with the use of chopsticks when eating, with different load forces through the thumb[10,16].

Occupation

There is little in the literature to prove a causal effect between occupation and hand/wrist OA.

There is, however, a strong trend to support that 'heavy work causing pressure on the hands' predisposes to OA of the hand[29,30], further supported by Asian studies who believe that OA is associated with heavy physical occupational activity or repetitive hand use[3,31]. A large population-based survey in China found that Chinese coal miners had a much higher prevalence of hand (and hip) OA than the general population[28]. It has further been concluded that 'accumulation of daily activities may contribute to the incidence of hand OA at different sites'[10].

In contrast to this, there was 'no association between the rate of hand

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OA progression and anthropometric features, life-style factors or familial effects' in Caucasians (n = 263; Russian Federation) found[11]. They further concluded that occupation, alcohol and nicotine consumption did not have a significant role to play in hand OA.

A small German cross-sectional study comparing young, high-level climbers (n = 37) with a non-climbing control group (n = 12) concluded that there was no statistically significant difference between the groups. They felt that 'intensive training and climbing lead to adaptive reactions'[32].

Obesity

Obesity affects over one-fifth of the UK's adult population, and its prevalence is increasing[33]. Excess adipose tissue produces humoral factors, which may alter the metabolism of the articular cartilage. It has been hypothesised that the leptin system/adipokine (a non-glycosylated protein secreted by adipocytes) may be a link between the metabolic abnormalities in obesity and an increased risk of OA[33,34]. Adipose tissue is a real endocrine organ releasing several substances including adipokines. Rat experiments have clearly shown that leptin may act as a catabolic factor in the progression of OA[34].

Although obesity (BMI greater than 30 kg/m^2 [World Health Organisation]) is well known to be associated with OA of the hip and knee, there is also evidence that this is the case in the hand and wrist, possibly caused by the metabolic abnormalities[33,35]. An American study (n = 1276) found that obesity was significantly associated with OA of the hands and that 'greater baseline relative weight was also associated with greater subsequent severity of OA of the hands'[35]. BMI is also positively related to radiological changes (n = 608), but applying logistic regression analyses did not demonstrate an independent positive relation to CMC I-joint OA[15].

A large Russian study (n = 1871) found no relationship between BMI and prevalence and severity of hand OA[9]. In contrast to this, the same first author published a study of 704 Turkmen subjects (again, after adjustment for age) and found that females with severe obesity (BMI greater than 35 kg/m^2) had a higher risk of developing hand OA than their counterparts of normal BMI[11].

Secondary OA

Trauma

Traumatic articular defects predispose individuals to the secondary OA. It is known that the presence of subchondral haematomas in distal radial fractures can lead to early onset of mild (radiographic) OA of the wrist and worse outcomes for patients[36]. This has been found to be the case even in some extra-articular distal radial fractures[36]. Intra-articular fractures of the distal radius are likely (68%) to produce radiological evidence of the secondary OA but may not necessarily change patients' functional and evaluation measures[37].

Bennett's fractures (an intra-articular fracture of the base of the first metacarpal) have been shown to do poorly when conservatively managed, with a high incidence of joint degeneration and functional problems (after mean 26 years)[38].

Intra-articular fractures of the phalanges seem to fair better. There is little in the literature about long-term follow-up, but although patients sustaining these types of fractures may well develop radiological osteophytes or cystic changes (17%), they do not necessarily go on to develop OA or symptoms[39].

Joint laxity, common in young women has also been associated with premature degenerative joint changes, especially at the base of the thumb[31]. It is thought that repeated loading of subluxed joints, that is, repetitive trauma, in women younger than 50 years of age, may explain why one-third of women over the age of 50 years show radiological evidence of degenerative changes at the thumb base[40].

Co-morbidity

Haemochromatosis arthropathy (a genetic disorder causing the body to absorb an excessive amount of iron from the diet, leading to iron deposition in the joints) is a secondary OA. These patients tend to be younger and predominantly male, when compared with the primary OA[13]. They also tend to have worse function, with more joint tenderness and more frequent and severe involvement of the metacarpophalangeal and wrist joint compared with individuals with the primary OA[13].

A population-based study (n = 819; Russian Federation)[12] found statistically significant evidence providing an association between radiographic hand OA and co-morbidities. They found that individuals with ischaemic heart disease had higher values of radiographic hand OA, compared with lower values in individuals with gastrointestinal diseases[12].

Infection

Septic (bacterial) arthritis can affect any articular surface, with the wrist and less commonly, the finger joints being the targets.

Despite a brief presence of bacteria in a joint, enzymes from the neutrophils can destroy cartilage and bone in a matter of hours, to days resulting in permanent disability from the secondary OA, with increased mortality associated with delayed presentations/diagnosis[41]. In general, haematogenous spread of the bacteria is the most common, but foreign bodies near the finger joints, trauma or recent hand surgery can also introduce local infection.

The most common microorganisms affecting the hand are *Staphylococcus aureus*, *Streptococcus pyogenes* and an increasing number of Gram-negative bacteria[42,43].

In a German review (n = 40) of bacterial infection in the hand IP joints,

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only 25% of the affected joints were preserved[44]. The remainder underwent surgery (joint resection and external fixation, followed by arthrodesis). Another study looking at pyarthrosis in the hand (n = 110) came to the same conclusion, that a significant number (25%) of patients end up with either an arthrodesis or an amputation[45].

A Swiss study (n = 31) suggested that in septic arthritis of the finger joints, an unfavourable outcome occurred when cartilage damage was observed at the time of surgery, and they suggested that in those cases, the primary arthrodesis with the use of an external fixator was indicated[46].

A UK study (n = 26) suggested a more conservative approach and admitted that the majority (54%) had restricted range of motion at discharge, but at follow-up (mean 54 months), overall motion and function appeared to improve in the longer term[47].

In septic arthritis of the wrist, open drainage or arthroscopic washout needs to be done expediently. There is some evidence that arthroscopic washout in isolated wrist sepsis means that patients have a shorter stay and fewer subsequent washouts[48]. Septic arthritis of the wrist, compared with the finger joints, seems to be less aggressive in causing a permanent disability[43].

Discussion

The authors have referenced some of their own studies in this review. These referenced studies have been conducted in accordance with the Declaration of Helsinki (1964), and the protocols of these studies have been approved by the relevant ethics committees related to the institution in which they were performed. All human subjects, in these referenced studies, gave informed consent to participate in these studies.

It has been concluded that 'ageing, female gender, genotype, heavy work and injuries predispose to OA in the hand'[29]. This is supported by the available literature that suggests that age, gender, race, obesity, occupation and genetics are all factors that lead to the primary OA of the hand or wrist. It is a multifactorial, heterogenous and complex disease.

A genetic predisposition appears to be the most powerful predictor of OA. Hand OA affects predominantly women and their hand arthritis is more likely to progress faster than in males.

There is, however, little in the evidence to prove a direct causal effect between occupation and the development of hand and wrist OA, although repetitive and heavy manual labour is thought by several to be one of the many factors leading to OA.

One problem studying epidemiology of OA of the hand and wrist is the fact that most of the published studies examine radiological data rather than clinical and do not necessarily include symptomatic individuals. This is due to the fact that radiography is the only diagnostic method we can control. It has been stated that 'there is no absolute clinical, radiological, or pathological standard against which epidemiological definitions of hand OA can be tested'[49].

Further research examining gene–gene and gene–environment interaction (especially looking at hormones, obesity, occupation) studies may give further insights into the pathogenesis of hand OA.

Conclusion

The evidence based upon this review is conclusive – there are many factors involved in the development of the primary hand and wrist OA. Aging, being female and hereditary factors are the most compelling culprits in the culmination and progression of the primary hand and wrist OA.

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Library Services, Royal Derby Hospital.

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