Seminars in Arthritis and Rheumatism 43 (2014) 429–436

Contents lists available at ScienceDirect

Seminars in Arthritis and Rheumatism

journal homepage: www.elsevier.com/locate/semarthrit

# Incidence of total knee and hip replacement for osteoarthritis in relation to the metabolic syndrome and its components: A prospective cohort study

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ARTICLE INFO

*Keywords:*
The metabolic syndrome
Osteoarthritis
Total knee replacement
Total hip replacement

ABSTRACT

*Objective:* To examine whether components of metabolic syndrome (MetS), either singly or additively, were associated with the incidence of severe knee and hip OA, and whether these associations were independent of obesity assessed by body mass index (BMI).
*Methods:* Twenty thousand, four hundred and thirty participants who had blood lipids, anthropometric and blood pressure measurements during 2003–2007 were selected from the Melbourne Collaborative Cohort Study. MetS was defined as central obesity assessed by waist circumference and any two of raised triglyceride level, reduced HDL cholesterol level, hypertension or impaired fasting glycaemia. The incidence of total knee and hip replacement was determined by linking cohort records to the Australian Orthopaedic Association National Joint Replacement Registry.
*Results:* Six hundred and sixty participants had knee OA and 562 had hip OA. After adjustment for age, gender, country of birth, education, physical activity and BMI, central obesity [hazard ratio (HR) 1.59, 95% confidence interval (CI) 1.25–2.01] and hypertension (1.24, 1.05–1.48) were associated with increased risk of knee OA. The accumulation of MetS components was associated with knee OA risk, independent of BMI: one component, 2.12 (1.15–3.91); two components, 2.92 (1.60–5.33) and three or more components, 3.09 (1.68–5.69). No statistically significant associations were observed for hip OA.
*Conclusion:* Cumulative number of MetS components and central obesity and hypertension were associated with increased risk of severe knee OA, independent of BMI. No associations were observed with severe hip OA. These findings suggest that the pathogenesis of knee and hip OA differ and that targeting the management of MetS may reduce the risk of knee OA.
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## Introduction

Osteoarthritis (OA) is a major health problem with significant morbidity and disability associated with OA of the knees and hips. OA resulted in a total of 71.1 million years lived with disability in 2010, an increase of 64% since 1990 [1]. Although several drugs and nutriceuticals have been evaluated in clinical trials to determine whether they are able to slow the structural progression of OA, currently there are no registered disease-modifying OA drugs. Therefore, understanding the role of modifiable risk factors is important for improving prevention. Obesity [high body mass index (BMI) or waist circumference] is widely recognised as the most important modifiable risk factor for knee OA [2,3], although the evidence for hip OA is less consistent [2,4]. The available evidence suggests that obesity increases the risk of OA through both increased loading [5] and metabolic mechanisms [6,7].

There has been an increasing interest in the relationship between OA and the metabolic syndrome (MetS), the clustering of abdominal obesity, dyslipidaemia (low high-density lipoprotein (HDL) and triglyceridaemia), hyperglycaemia and hypertension [8].

Sources of support: The recruitment of the Melbourne Collaborative Cohort Study was funded by Vic Health and Cancer Council Victoria. This study was funded by a programme Grant from the National Health and Medical Research Council (NHMRC; 209057), Capacity Building Grant (251533), and Enabling Grant (396414), and was further supported by infrastructure provided by the Cancer Council Victoria. Dr Wluka is the recipient of NHMRC Career Development Fellowship (Clinical, level 1, #545876).
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0049-0172/$ - see front matter © 2014 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.semarthrit.2013.07.013

MetS was found to be 5.26 times more common in those with OA compared with those with no OA, and the individual components of the MetS were also more prevalent in people with OA in the NHANES III survey [9]. However, the Malmö Diet and Cancer study reported only central obesity to be associated with increased risk of knee OA independent of BMI, and neither MetS nor components of MetS to be associated with hip OA [10].

The Research on Osteoarthritis/Osteoporosis Against Disability (ROAD) 3-year follow-up study focused on the relationship between components of MetS defined as obesity measured by BMI, hypertension, dyslipidaemia and IFG with the incidence and progression of radiological knee OA in a Japanese population [11]. They reported that BMI, systolic blood pressure and dyslipidaemia were associated with incident knee OA [11], but the associations for systolic blood pressure and dyslipidaemia were no longer statistically significant after adjusting for BMI [11]. Moreover, this study found an increased risk of knee OA progression associated with an increase in the number of MetS components [11].

There is now an increasing body of evidence from epidemiological, proteomic, genetic and *in vitro* studies examining potential shared pathogenic mechanisms between the MetS and knee OA [12,13] that supports the inclusion of OA as a component of MetS [13], but it is still unclear whether MetS is a pathway to OA or whether OA and MetS simply coexist through commonly shared risk factors of age and obesity.

One method for defining OA is based on joint replacement [4,10]. This definition signifies severe clinical knee and hip OA, which is relevant to the symptomatic disease burden and economic impact of OA. Thus, in a large prospective cohort study, we examined the relationship between the components of MetS and the accumulation of components of MetS and the incidence of total knee and hip replacement due to severe OA.

## Patients and methods

### *Study participants*

The Melbourne Collaborative Cohort Study (MCCS) is a prospective cohort study of 41,514 participants (24,469 women) between the ages of 27 and 75 years at baseline [14]. Participants were recruited via the Electoral Rolls, advertisements and community announcements in local media, between 1990 and 1994. Southern European migrants to Australia (including 5411 Italians and 4525 Greeks) were deliberately oversampled to extend the range of lifestyle exposures and to increase genetic variation. The study protocol was approved by The Cancer Council Victoria's Human Research Ethics Committee. Follow-up was conducted by record linkage to Electoral Rolls, electronic phone books and the Victorian Cancer Registry and death records.

From 2003 to 2007, 28,046 participants (68% of the original MCCS participants) were followed up with face-to-face interviews. Of these, 20,430 had data available for all anthropometric measurements, blood pressure and blood lipids (Fig. 1), and thus were included for data analysis.

### *Anthropometric measurements*

Height, weight, waist circumference and blood pressure (BP) were measured at the 2003–2007 follow-up visit according to written protocols based on standard procedures [15]. Weight was measured to the nearest 0.1 kg using digital electronic scales, height and waist circumference were measured to the nearest 1 mm using a stadiometer and a metal anthropometric tape, respectively. BMI was calculated as weight in kilograms divided by the square of height in metres. For BP recording, three

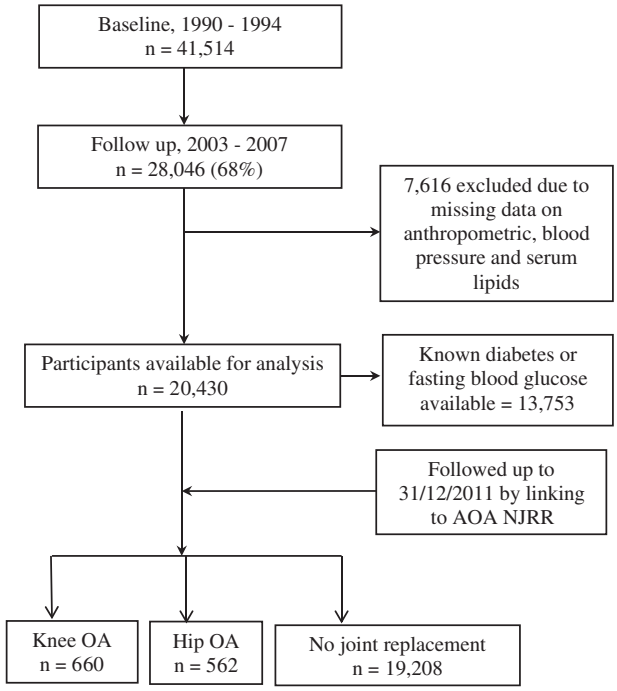


**Fig. 1.** Flowchart of data collection.

consecutive measurements were taken using a Dinamap 1846SX automatic BP monitor (Critikon, Tampa, FL). The average of the second and third BP measurements was used in the analysis.

### *Assessment of demographic, lifestyle factors and physical activity*

At baseline, information was obtained regarding demographic and lifestyle factors, including date of birth, country of birth and highest level of education. Physical activity over the last 6 months was assessed by asking specific questions regarding the frequency of vigorous activity, less vigorous activity and walking. Walking and less vigorous activity frequencies were added together with twice the frequency of vigorous activity to compute a total physical activity level for each person. Physical activity was then categorised into four groups—none (0), low ( > 0–4), moderate ( > 4–6) and high ( > 6) [16].

### *Plasma assays*

Plasma lipid and glucose levels were measured by routine assays. Levels of serum lipids (triglyceride and HDL cholesterol) were measured using blood collected between 2003 and 2007. Total TG was measured enzymatically using a Hitachi 917 instrument (Boehringer Mannheim Corp., Indianapolis, IN). HDL cholesterol was measured using the HDL-C plus 2nd generation, Roche kit (Roche Diagnostics Australia Pty Ltd., Castle Hill, New South Wales, Australia) on a Hitachi 917 instrument (Boehringer Mannheim Corp, Indianapolis, IN) [17]. Since most of the participants were not fasting during 2003–2007 follow-up, plasma glucose levels measured from fasting blood collected at baseline (1990–1994) using a Kodak Ektachem analyzer (Rochester, NY) were the glucose levels used in the analysis.

### *Identification of incident primary knee and hip joint replacement*

Cases were identified from the AOA NJRR. The Registry began data collection in September 1999 and implementation was introduced in a staged fashion in each of the Australian States and Territories. Victoria commenced data collection in 2001 [18]. The Registry monitors hip and knee joint replacements. It has

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