METABOLIC SYNDROME PRECEDES THE ONSET OF HIP AND KNEE PAIN AND THE RISK IS NOT MODIFIED BY DIET OR CHANGES IN BMI

Max Yates1,2, Jordan Tsigarides3, Shabina Hayat4, Robert Luben5, Jack Dainty1,2

1University of East Anglia, Norwich, United Kingdom
2University of Cambridge, MRC Epidemiology Unit, Cambridge, United Kingdom
3Norfolk and Norwich University Hospital, Norwich, United Kingdom
4University of Cambridge, UK

Background: Excess weight and components of the metabolic syndrome have been associated with knee osteoarthritis (OA). However, the precise timing of when this risk operates in the natural history of the disease is unknown. An understanding of the sequence in which risk factors operate in OA is important for delivering effective preventative interventions.

Objectives: We examined longitudinal data from a large prospective population-based cohort to assess the association between metabolic syndrome components and the subsequent development of either knee or hip over 10 years of follow-up.

Methods: Known risk factors data were obtained at enrolment to the EPIC-Norfolk cohort between 1993 and 1997. Data were available on anthropometric variables, smoking status and lipid metabolites. Knee and hip pain were self-reported at 18 months, 3 and 10 years when respondents answered whether they had had pain in their knee or hip on most days in the preceding month. Metabolic syndrome components were defined in line with the Alberti formula. Logistic regression was used to investigate metabolic syndrome components: waist circumference (men >102cm, women >88cm), low HDL (men <1.0mmol/L, women <1.3mmol/L), high triglycerides (>1.7mmol/L) and their association to incident pain, and any effect-modification of dietary patterns and changes in BMI over time.

Results: Amongst 20,517 respondents (age at enrolment 59.7 years (SD 9.2), 56.5% female) there were 3,886 who reported knee pain and 2,467 who reported hip pain at 18 months. By the end of follow-up there were 2619 who had developed incident knee pain and 1752 who had developed incident hip pain. In a logistic model adjusted for age and sex, significant associations were seen for incident pain for every-smoking (knee: odds ratio 1.22, 95% CI 1.11, 1.33 p<0.001), (hip: odds ratio 1.21, 95% CI 1.08, 1.35 p<0.001) and obesity (knee: OR = 1.70 95% CI 1.51, 1.91 p<0.001), (hip: OR = 1.68 95% CI 1.47, 1.92 p<0.001). After adjustment for obesity, components of the metabolic syndrome associated with pain included waist circumference (knee: OR = 1.17 95% CI 1.03, 1.33 p<0.019), (hip: OR = 1.37 95% CI 1.17, 1.59 p<0.001) and for knee pain, low HDL (OR = 1.15 95% CI 1.05, 1.27 p<0.003) and high triglycerides (OR = 1.10 95% CI 1.00, 1.04 p=0.043). These associations were not modified by dietary patterns nor changes in weight over the follow-up interval.

Conclusion: This longitudinal analysis shows that metabolic syndrome is a predictor for the future onset of hip and knee pain over an interval of 10 years, and the risk is not modified by diet or subsequent weight change. These data suggest that preventative strategies need to be targeted in the early disease course, and should include a range of measures including smoking cessation and those that prevent the onset of metabolic syndrome.