

Poster Presentations

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Epidemiology, risk factors for disease or disease progression

**THU0675 ASSOCIATION OF GLUCOSE HOMEOSTASIS MEASURES AND METABOLIC SYNDROME WITH KNEE CARTILAGE DEFECTS AND CARTILAGE VOLUME IN YOUNG ADULTS**

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## Abstract

**Background** Diabetes mellitus and knee osteoarthritis (OA) were commonly coexisting, and metabolic syndrome (MetS) shared many pathways with knee OA. However, the effects of glucose homeostasis and MetS on knee cartilage in young adults were unknown.

**Objectives** To describe the associations of glucose homeostasis measures and MetS measures with knee cartilage defects and cartilage volume in young adults.

**Methods** Australian young adults from the Childhood Determinants of Adult Health Study were selected to undergo knee magnetic resonance imaging (MRI) scans during 2008-2010 (aged 31-41 years). Fasting blood sample, waist circumference and blood pressure measures were collected during 2004-2006 (aged 26-36 years). Glucose, insulin, triglyceride and high-density lipoprotein cholesterol (HDL-C) were measured using serum samples. Homeostatic model assessment 2-insulin resistance (HOMA2-IR), HOMA2-beta cell function (HOMA2-β), HOMA2-insulin sensitivity (HOMA-S) were calculated using HOMA2 calculator (version 2.2.3 available from <http://www.dtu.ox.ac.uk/homacalculator>) according to fasting glucose and fasting insulin. MetS was defined when at least three of the following five components were present: high waist circumference (male  $\geq 102$  cm, female  $\geq 88$  cm), high

fasting glucose ( $\geq 5.6$  mmol/L), high serum triglycerides ( $\geq 1.7$  mmol/L), low HDL-C (male  $< 1.03$  mmol/L, female  $< 1.3$  mmol/L), and high blood pressure ( $\geq 130/85$  mmHg). Cartilage defects and cartilage volume were measured from MRI scans. Data were analysed using log binomial or linear regressions and were adjusted for age, gender, body mass index and physical activity.

**Results** Among 328 participants (47.3% were females), 40 (12.7%) had hyperglycaemia and 21 (6.7%) had MetS. Glucose homeostasis measures (except fasting glucose) were associated with tibiofemoral cartilage defects (Fasting insulin: relative risk (RR) 1.05/mU/L, 95% confidence interval (CI) 1.01 to 1.08; HOMA2-IR: 1.44, 1.08 to 1.92; HOMA2- $\beta$ : 2.59, 1.33 to 5.07; HOMA2-S: 0.36, 0.18 to 0.72), but not patellar cartilage defects. There were no associations between glucose homeostasis measures and knee cartilage volume. MetS measures were not associated with either cartilage defects or cartilage volume, except the associations between high waist circumference and tibiofemoral cartilage defects (RR 2.32, 95% CI 1.18 to 4.54) and between low HDL-C and tibiofemoral cartilage defects (RR 1.99, 95% CI 1.08 to 3.69).

**Conclusion** Insulin resistance was associated with higher risk of tibiofemoral cartilage defects amongst young adults. MetS was not associated with neither cartilage defects nor cartilage volume. These suggest that glucose homeostasis, but not MetS, may play a role in cartilage damage in young adults and may lead to knee OA in later life.

**Disclosure of Interests** None declared

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