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Association of childhood adiposity measures with adulthood knee cartilage defects and bone marrow lesions: a 25-year cohort study

T. Meng †, S. Thayer ‡, A. Venn †, F. Wu †, F. Cicuttini §, L. March ||, T. Dwyer †¶, A. Halliday ‡, M. Cross ||, L.L. Laslett †, G. Jones †, C. Ding †§#*, B. Antony †

† *Menzies Institute for Medical Research, University of Tasmania, Hobart, Australia*
‡ *Department of Radiology, Royal Hobart Hospital, Hobart, Australia*
§ *Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia*
|| *Institute of Bone and Joint Research, University of Sydney, Sydney, Australia*
¶ *The George Institute for Global Health, Nuffield Department of Obstetrics & Gynaecology, University of Oxford, Oxford, United Kingdom*
# *Clinical Research Centre, Zhujiang Hospital, Southern Medical University, Guangzhou, China*

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SUMMARY

Objective: To describe the associations between childhood adiposity measures and adulthood knee cartilage defects and bone marrow lesions (BMLs) measured 25 years later.
Methods: 327 participants from the Australian Schools Health and Fitness Survey (ASHFS) of 1985 (aged 7–15 years) were followed up 25 years later (aged 31–41 years). Childhood measures (weight, height and skinfolds) were collected in 1985. Body mass index (BMI), overweight status and fat mass were calculated. Participants underwent 1.5 T knee magnetic resonance imaging (MRI) during 2008–2010, and cartilage defects and BMLs were scored from knee MRI scans. Log binomial regressions were used to examine the associations.
Results: Among 327 participants (47.1% females), 21 (6.4%) were overweight in childhood. Childhood adiposity measures were associated with the increased risk of adulthood patellar cartilage defects (Weight relative risk (RR) 1.05/kg, 95% confidence interval (CI) 1.01–1.09; BMI 1.10/kg/m^2, 1.01–1.19; Overweight 2.22/yes, 1.21–4.08; fat mass 1.11/kg, 1.01–1.22), but not tibiofemoral cartilage defects. Childhood adiposity measures were not significantly associated with adulthood knee BMLs except for the association between childhood overweight status and adulthood patellar BMLs (RR 2.87/yes, 95% CI 1.10–7.53). These significant associations persisted after adjustment for corresponding adulthood adiposity measure.
Conclusion: Childhood adiposity measures were associated with the increased risk of adulthood patellar cartilage defects and, to a lesser extent, BMLs, independent of adulthood adiposity measures. These results suggest that adiposity in childhood has long-term effects on patellar structural abnormalities in young adults.
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Introduction

Osteoarthritis (OA) is the most common joint disease, which is characterised by joint structural changes including cartilage degradation and subchondral bone abnormalities[1]. About 13% of women and 10% of men aged 60 years or older have symptomatic knee OA[2], with no approved disease modifying treatments available. Thus, identifying modifiable factors that can prevent knee OA is critically important.

Cartilage defects and bone marrow lesions (BMLs) are important imaging biomarkers for the incidence and progression of knee OA. They are common in both healthy individuals and symptomatic OA patients[3–5], and are associated with knee pain[6,7] knee cartilage volume loss[8,9] and subsequent knee replacement surgery[10,11] in most studies, although not all associations were consistent[12–15]. However, little is known about factors that are associated with cartilage defects and BMLs in young adults, who may not yet have

* Address correspondence and reprint requests to: C. Ding, Clinical Research Centre, Zhujiang Hospital, Southern Medical University, Guangzhou, Guangdong, China.
E-mail addresses: tao.meng@utas.edu.au (T. Meng), shuan.thayer@ths.tas.gov.au (S. Thayer), alison.venn@utas.edu.au (A. Venn), feitong.wu@utas.edu.au (F. Wu), flavia.cicuttini@monash.edu (F. Cicuttini), lyn.march@sydney.edu.au (L. March), terry.dwyer@mcri.edu.au (T. Dwyer), andrewhalliday@hotmail.com (A. Halliday), maritac@med.usyd.edu.au (M. Cross), laura.laslett@utas.edu.au (L.L. Laslett), graeme.jones@utas.edu.au (G. Jones), Changhai.Ding@utas.edu.au (C. Ding), benny.eathakkattuantony@utas.edu.au (B. Antony).

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established knee OA. This information may help develop intervention during early life to reduce the burden of knee OA in later life[16].

Obesity has long been recognized as a risk factor for the incidence and progression of knee OA[17], but the role of childhood adiposity in knee OA in later life is not well studied as most of current evidence is derived from middle-aged or older adults[18]. Wills *et al.* reported that body mass index (BMI) as early as 11 years in females and 20 years in males was independently associated with knee OA at the age of 53[19]. Similarly, Gelber *et al.* reported that BMI in young men, aged 20–29 years, was associated with the increased risk of subsequent knee OA[20]. In addition, we reported that the childhood adiposity measures were associated with higher risk of knee pain in adulthood 25 years later[21]. These findings suggest that adiposity measures during early life may have long-term effects on knee joint in later life. This comes particularly important as the prevalence of overweight and obesity has increased in children and adolescents in both developed and developing countries during 1980–2013[22]. However, there is a paucity of information about the effects of childhood adiposity measures on adulthood cartilage defects and BMLs. Therefore, we aimed to describe longitudinal associations between adiposity measures in childhood and knee cartilage defects and BMLs in adulthood 25 years later.

Materials and methods

Participants

The Australian Schools Health and Fitness Survey (ASHFS) was completed in 1985 on a nationwide sample of schoolchildren ($n = 8498$, aged 7–15 years), and a wide range of health-related measures were collected through field and technical tests. The Childhood Determinants of Adult Health (CDAH) Study was a 20-year follow-up ($n = 2410$, aged 26–36 years) of children who participated in ASHFS and was completed during 2004–2006, adulthood health-related measures were collected during the CDAH Study. The CDAH Knee Cartilage Study ($n = 330$, aged 31–41 years) was a sub-study of the CDAH Study and the participants completed knee magnetic resonance imaging (MRI) scans during 2008–2010.

We used the following strategy to recruit participants from the CDAH Study. The CDAH Study participants ($n = 764$) residing in metropolitan Melbourne and Sydney were contacted by mail and invited to participate in the CDAH Knee Cartilage Study. Participants who agreed to participate ($n = 529$, response percentage 69%) were assessed for their eligibility. Exclusion criteria for this study were being pregnant, having had diseases that might affect knee cartilage such as rheumatoid arthritis, or having a contraindication for MRI. Eighty participants were excluded either because of the exclusion criteria or because they changed their mind. The remaining 449 participants were asked to complete a short computer-assisted telephone interview (CATI). History of knee injury or surgery was not collected in childhood in the ASHFS study and, therefore, telephone interviews included history of knee injury in childhood. Knee injury was recorded in response to the question, "Have you had a knee injury requiring non-weight-bearing treatment for more than 24 h or surgery?" Participants were requested to have an MRI scan at Epworth Hospital in Melbourne or North Shore Private Hospital in Sydney. Some participants ($n = 119$) did not undergo MRI after enrolling in the study due to the long distance, work or family commitments, moving interstate, becoming pregnant by the time of MRI, or changing their mind. Eight MRI scans were not readable for cartilage defects and three for BMLs due to the absence of adequate sequences. Therefore, these MRI scans were not included for cartilage defects or BMLs assessments. There are 322 participants included in analyses for cartilage defects and 327 for BMLs. A flowchart of the selection of participants for this study is shown in Fig. 1.

This study was approved by the Southern Tasmania Health and Medical Human Research Ethics Committee (HREC), the Monash University HREC and the Northern Sydney and Central Coast Area HREC. All participants provided written informed consent. At baseline, all children provided assent and parents provided written informed consent.

Anthropometric measurements

Weight was measured to the nearest 0.5 kg in 1985 and 0.1 kg during follow-up, with shoes, socks and bulky clothing removed. Height was measured to the nearest 0.1 cm (with shoes and socks removed) using a stadiometer. BMI was calculated as weight in kilograms divided by height in meters squared, at both time points. Overweight status in childhood was defined according to age and sex-specific cut-off points[23]. Adulthood overweight status was defined as a BMI >25 kg/m^2.

Triceps, biceps, subscapular, and supra-iliac skinfolds were measured at locations determined by reference to anatomical landmarks[24] to the nearest 0.1 mm by using Holtain Skinfold Calipers in 1985 and Slim Guide Skinfold Calipers (SPRI Products) during CDAH Study. Body density was estimated from the log of the sum of four skinfolds using age-specific regression equations[24–26]. Estimate of percent body fat was derived from body density[27], and fat mass was estimated by percent body fat in kilograms: fat mass = fat% × weight.

MRI measurements

Participants had an MRI scan of their knees in the CDAH Knee Cartilage Study. MRI scans were obtained from 2 hospitals, which used the same type of machine (General Electric Medical Systems, Milwaukee, WI, USA). Knees were imaged on a 1.5 T whole-body magnetic resonance unit with use of a commercial transmit-receive extremity coil. The following image sequences were used: (1) a T1-weighted, fat-suppressed 3-dimensional (3D) spoiled gradient-recalled acquisition in the steady state; flip angle 55°; repetition time 58 msec; echo time 12 msec; field of view 16 cm; 60 partitions; 512 × 512-pixel matrix; acquisition time 11 min, 56 s; 1 acquisition. Sagittal images were obtained at a partition thickness of 1.5 mm and an in-plane resolution of 0.31 × 0.31 mm (512 × 512 pixels). (2) Proton density-weighted fat-suppressed two-dimensional fast spin-echo coronal images at a partition thickness of 3.3 mm and an in-plane resolution of 0.31 × 0.31 mm (512 × 512 pixels); repetition time 3800 msec; echo time 45 msec.

Knee cartilage defects were measured as previously reported[28] in an ordinal scale using the T1-weighted spoiled gradient-recalled sagittal MR images and proton density-weighted fast spin-echo coronal MR images together. Grade 0 indicated a normal cartilage, and Grade 1 indicated focal blistering and low-signal intensity area in T1-weighted sagittal images or high-signal intensity area in proton density-weighted images with intact surface/bottom. Grade 2 indicated a loss of thickness of <50% on surface/bottom of the cartilage. Grade 3 represented a loss of thickness >50%, and Grade 4 indicated a full-thickness chondral wear with exposure of subchondral bone. A prevalent cartilage defect was defined as a cartilage defect score of ≥2 at any site within that compartment. Intraobserver reliability expressed as an intraclass correlation coefficient (ICC) ranged from 0.89 to 0.94.

BMLs were identified using the sagittal images reformatted from coronal proton density-weighted images and then scored as the

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