Available online at
ScienceDirect
www.sciencedirect.com
Elsevier Masson France
EM|consulte
www.em-consulte.com/en
Diabetes & Metabolism 41 (2015) 463–469

Original article

The metabolic syndrome and cancer: Is the metabolic syndrome useful for predicting cancer risk above and beyond its individual components?

J. Harding [a,b,*,1], M. Sooriyakumaran [a,b,1], K.J. Anstey [c], R. Adams [d], B. Balkau [e], T. Briffa [f], T.M.E. Davis [g], W.A. Davis [g], A. Dobson [h], G.G. Giles [b,i,j], J. Grant [k], M. Knuiman [f], M. Luszcz [l], P. Mitchell [m], J.A. Pasco [n,o], C. Reid [p], D. Simmons [q,r], L. Simons [s], A. Tonkin [p], M. Woodward [t,u], J.E. Shaw [a,b,1], D.J. Magliano [a,b,1]

[a] *Department of Clinical Diabetes and Epidemiology, Baker IDI Heart and Diabetes Institute, Melbourne, Australia*
[b] *Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia*
[c] *Research School of Population Health, the Australian National University, Canberra, Australia*
[d] *The Health Observatory Discipline of Medicine, the University of Adelaide, Adelaide, Australia*
[e] *Inserm, U1018, Centre for Research in Epidemiology and Population Health, France*
[f] *School of Population Health, the University of Western Australia, Crawley, Australia*
[g] *School of Medicine and Pharmacology, the University of Western Australia, Fremantle, Australia*
[h] *School of Population Health, the University of Queensland, Brisbane, Australia*
[i] *Cancer Epidemiology Centre, the Cancer Council Victoria, Melbourne, Australia*
[j] *Centre for Epidemiology and Biostatistics, School of Population and Global Heath, the University of Melbourne, Melbourne, Australia*
[k] *Population Research & Outcome Studies, the University of Adelaide, Adelaide, Australia*
[l] *Flinders Centre for Ageing Studies, Flinders University, Adelaide, Australia*
[m] *Westmead Millennium Institute, the University of Sydney, Sydney, Australia*
[n] *IMPACT Strategic Research Centre School of Medicine, Deakin University, Geelong, Australia*
[o] *NorthWest Academic Centre, Department of Medicine, the University of Melbourne, St Albans, Australia*
[p] *School of Public Health and Preventive Medicine, Monash University, Melbourne, Australia*
[q] *School of Medicine, University of Western Sydney, Campbelltown, Australia*
[r] *Department of Rural Health, the University of Melbourne, Shepparton, Australia*
[s] *UNSW Australia Lipid Research Dept, St Vincent's Hospital, Sydney, Australia*
[t] *The George Institute for Global Health, the University of Sydney, Sydney, Australia*
[u] *The George Institute for Global Health, Nuffield Department of Population Health, University of Oxford, UK*

Received 19 February 2015; accepted 22 April 2015
Available online 30 May 2015

Abstract

Aims. – The metabolic syndrome (MetS) is a risk factor for cancer. However, it is not known if the MetS confers a greater cancer risk than the sum of its individual components, which components drive the association, or if the MetS predicts future cancer risk.

Materials and methods. – We linked 20,648 participants from the Australian and New Zealand Diabetes and Cancer Collaboration with complete data on the MetS to national cancer registries and used Cox proportional hazards models to estimate associations of the MetS, the number of positive MetS components, and each of the five MetS components separately with the risk for overall, colorectal, prostate and breast cancer. Hazard ratios (HR) and 95% confidence intervals (95%CI) are reported. We assessed predictive ability of the MetS using Harrell's c-statistic.

Results. – The MetS was inversely associated with prostate cancer (HR 0.85; 95% CI 0.72-0.99). We found no evidence of an association between the MetS overall, colorectal and breast cancers. For those with five positive MetS components the HR was 1.12 (1.02-1.48) and 2.07 (1.26-3.39) for overall, and colorectal cancer, respectively, compared with those with zero positive MetS components. Greater waist circumference (WC) (1.38; 1.13-1.70) and elevated blood pressure (1.29; 1.01-1.64) were associated with colorectal cancer. Elevated WC and triglycerides were (inversely) associated with prostate cancer. MetS models were only poor to moderate discriminators for all cancer outcomes.

* Corresponding author. Level 4, 99 Commercial Road, Melbourne, VIC, Australia, 3004. Tel.: +61 3853 21582; fax: +61 3853 21100.
E-mail address: jessica.harding@bakeridi.edu.au (J. Harding).
[1] Joint authorship.

http://dx.doi.org/10.1016/j.diabet.2015.04.006
1262-3636/© 2015 Elsevier Masson SAS. All rights reserved.

Conclusions. – We show that the MetS is (inversely) associated with prostate cancer, but is not associated with overall, colorectal or breast cancer. Although, persons with five positive components of the MetS are at a 1.2 and 2.1 increased risk for overall and colorectal cancer, respectively, and these associations appear to be driven, largely, by elevated WC and BP. We also demonstrate that the MetS is only a moderate discriminator of cancer risk.
© 2015 Elsevier Masson SAS. All rights reserved.

Keywords: Cancer; Epidemiology; Metabolic syndrome; Prediction

1. Background

The metabolic syndrome (MetS) is defined by a group of metabolic risk factors that have a tendency to cluster together in one individual-obesity (particularly central obesity), hypertension, dyslipidaemia and insulin resistance [1,2]. These factors, separately and jointly, have been associated with several chronic diseases, in particular cardiovascular disease (CVD) [3] and type 2 diabetes [4]. There is emerging evidence that the MetS may also be important in the development of some cancers [5].

A recent meta-analysis reported that the MetS is associated with low to modest increased risks for colorectal, post-menopausal breast, bladder, pancreas, endometrium and liver cancers [5], but for prostate cancer, evidence is conflicting. Some studies report an increased risk [6], others report a decreased risk [7], and others report no association with the MetS [5]. Mechanisms linking the MetS and cancer are not well understood. The association may partially be explained by the presence of obesity, and overt hyperglycaemia, both of which have been repeatedly associated with increased risks for some common cancers and a decreased risk for prostate cancer [8,9]. There is also some evidence to suggest that elevated blood pressure (BP) is associated with an increased cancer risk [10] while high-density lipoprotein (HDL) cholesterol has been shown to have an inverse association with cancer [11]. It is not yet known whether the strength of the association between the MetS and cancer is greater than the sum of its individual components, which individual components may be driving this association, or whether the MetS is a useful predictor of future cancer risk.

Using a large pool of prospective studies, we report the risks for overall, and the three most common site-specific cancers, colorectal, prostate and breast cancer associated with the components of the MetS, both separately and jointly. We additionally investigate whether the MetS is a useful measure for discriminating cancer risk.

2. Methods

2.1. Study population

The Australian and New Zealand Diabetes and Cancer Collaboration (ANZDCC) is a pooled cohort comprising 18 prospective studies in Australia and New Zealand with data on 153,025 men and women. All included cohorts were comprised of adults, except Fremantle Diabetes Study (FDS), a diabetes cohort, which also included some adolescents with type 1 diabetes. Details of sampling procedures, study designs, and methods for each of the studies have been described [12]. In brief, investigators of cohort studies conducted in the region from 1983 onwards with data on diabetes and the MetS, and with a minimum sample size of 1000 were invited to participate in the ANZDCC study. For the current analysis, we included studies that had collected data on all five MetS components in order to determine MetS status (five cohorts; $n = 59{,}630$). We further excluded participants with missing data on any of the five MetS components ($n = 37{,}392$); a cancer diagnosis prior to their baseline date ($n = 905$); and missing data on smoking and education status ($n = 305$). A total of 20,468 participants (men = 9437; women = 11,031) with complete data were included in the final data analysis.

2.2. Data linkage

The ANZDCC cohort was linked to the Australian Cancer Database (ACD), a register of all primary, malignant cancers diagnosed in Australia since 1982, and t*he National Death Index* (NDI). Linkage was performed by the Australian Institute of Health and Welfare (AIHW) and the Western Australian Data Linkage Unit (FDS only) using first name, second name, last name, gender, and date of birth [13]. Cancer status of the cohort was determined until 31 December 2008 for the Australian Diabetes Obesity and Lifestyle Study (AusDiab), Crossroads Undiagnosed Disease Study (CUDS) and the North West Adealaide Health Study (NWAHS); 31 August 2010 for the Melbourne Collaborative Cohort Study (MCCS); and 31 October 2012 for FDS. We set a match link rate of 97.70% (true matches/correct links) with link accuracy of 97.92% (1.08% expected to be false positive links). Twenty-seven percent of links underwent clerical review, performed by AIHW. This match link rate has shown to be a reliable cut-off in similar studies [14]. Cancer was defined using the International Classification of Disease 10th Revision (ICD-10) codes as follows: overall cancer (C00-C97, D45-D46, D47.1, D47.3); colorectal (C18-C20); prostate (C61); breast (C50).

2.3. Definition of covariates

All participants were measured for weight, height, waist circumference (WC), BP, fasting plasma glucose, serum HDL cholesterol and triglycerides by trained staff adhering to standardised protocols at baseline. Information on education and smoking status was collected by questionnaires. These risk

Download English Version:

https://daneshyari.com/en/article/3258996

Download Persian Version:

https://daneshyari.com/article/3258996

Daneshyari.com