



Identifying metabolic syndrome in a clinical cohort: Implications for prevention of chronic disease[☆]

Allison Martin^{a,*}, Elizabeth P Neale^a, Marjka Batterham^b, Linda C Tapsell^a

^a School of Medicine, Faculty of Science, Medicine and Health, University of Wollongong, NSW 2522, Australia

^b Statistical Consulting Centre, University of Wollongong, NSW 2522, Australia

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ABSTRACT

In the clinical setting, calculating cardiovascular disease (CVD) risk is commonplace but the utility of the harmonised equation for metabolic syndrome (MetS) (Alberti et al., 2009) is less well established. The aims of this study were to apply this equation to an overweight clinical cohort to identify risk factors for being metabolically unhealthy and explore associations with chronic disease.

Baseline data were analysed from a lifestyle intervention trial of Illawarra residents recruited in 2014/2015. Participants were aged 25–54 years with a BMI 25–40 kg/m². Data included MetS, CVD risk, insulin sensitivity, weight, body fat, diet, peripheral artery disease (PAD), physical activity, socio-economic position and psychological profile. Backward stepwise regression tested the association of covariates with MetS status and linear or logistic regression tested associations between MetS and risk of CVD, coronary heart disease, PAD and insulin resistance. 374 participants were included in the analysis with 127 (34.0%) categorised with MetS. Covariates significantly and positively associated with MetS were higher BMI (odds 1.26, $p < 0.01$) and older age (odds 1.08, $p < 0.01$). MetS participants ($n = 351$) had a 4.50% increase in CVD risk and were 8.1 and 12.7 times (respectively) more likely to be at risk of CHD and insulin resistance, compared to participants without MetS.

The utility of the harmonised equation in the clinical setting was confirmed in this overweight clinical cohort. Those classified as having MetS were more likely to be older, overweight/obese individuals and they had a substantially higher risk of developing CVD and insulin resistance than those without MetS.

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1. Introduction

A decline in metabolic health often precedes cardiovascular disease (CVD)¹ and diabetes mellitus, so there are benefits in early identification and intervention. Definitions of “metabolically unhealthy” vary. Investigators have frequently used either the absence of metabolic syndrome (MetS), high insulin sensitivity, or a combination of both to define metabolic health in overweight or obese individuals (Hinnouho et al., 2013; Årnlöv et al., 2010). A global definition of MetS now exists (Alberti et al., 2009) that unifies previously published equations with an agreement that central obesity is not an obligatory component. The harmonised definition of MetS is defined as having three of the following: elevated waist circumference (country specific guidelines), triglycerides ≥ 1.7 mmol/L*, systolic blood pressure ≥ 130 or diastolic ≥ 85

mm Hg*, glucose ≥ 100 mg/dL and reduced HDL cholesterol < 1.0 mmol/L (males) or < 1.3 mmol/L (females) (or drug treatment as indicated*) (Alberti et al., 2009). Recently published studies have also been found to use previous MetS equations (Hinnouho et al., 2013; Shab-Bidar et al., 2014; de Castro and Scorsatto, 2015). While risk calculations of CVD are readily available (D’Agostino et al., 2008; Wilson et al., 1998), the identification of MetS is not commonly used in practice and could help initiate early intervention for disease prevention.

MetS often occurs in the presence of obesity which is known to cause a decline in life expectancy due to its associated metabolic and cardiovascular comorbid disorders. Clinical practice guidelines for obesity defer to treatment with diet, physical activity and behavioural support (Council NHaMR, 2013). However, recent research suggests that not all obese individuals have the same metabolic risk profile (Phillips, 2013) and this has implications for personalising treatment. In the first instance it may be relevant to distinguish individuals at high risk for obesity-related metabolic diseases by identifying the presence of MetS.

In addition to traditional risk factors such as obesity, many other factors have been suggested as being associated with MetS, such as chronic stress (Epel et al., 2004); socioeconomic position (Brunner et al., 1997); cardiorespiratory fitness (LaMonte et al., 2005), peripheral artery

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* Corresponding author at: Room 120, Building 32, Illawarra Health and Medical Research Institute, University of Wollongong, NSW 2522, Australia.

E-mail address: allisonh@uow.edu.au (A. Martin).

¹ List of abbreviations: metabolic syndrome (MetS), body mass index (BMI), ankle brachial index (ABI), systolic blood pressure (SBP), diastolic blood pressure (DBP), international physical activity questionnaire (IPAQ), depression anxiety stress scale (DASS-21), cardiovascular disease (CVD), coronary heart disease (CHD).

disease (PAD) (Wild et al., 2006), and dietary saturated fatty acids (Shab-Bidar et al., 2014). These all have implications for the mode of treatment, particularly in relation to the nature of diet, exercise and psychological support.

The aims of this study were to apply the harmonised equation for calculation of MetS to a clinical cohort and to identify risk factors for being metabolically unhealthy, as well as assess presence of MetS and risk of chronic disease.

2. Methods

2.1. Study design and setting

This paper reports on a secondary analysis of data from the HealthTrack study, a 12 month healthy lifestyle randomised clinical trial. Participants were randomised to receiving either usual care (general advice), or individualised dietary advice with exercise prescription and health coaching, with or without a healthy food supplement (walnuts). Participants were recruited from the Illawarra region of NSW between May 2014 and April 2015. Inclusion criteria for the study were: permanent residents of the Illawarra region (NSW, Australia), adults aged 25–54 years, and with a body mass index (BMI) in the range 25–40 kg/m². Exclusion criteria were: unable to communicate in English; severe medical conditions, impaired ability to participate; suffering from immunodeficiency; reported illegal drug use or regular alcohol intake (>50 g/day). Ethical approval was granted by the University of Wollongong/Illawarra Shoalhaven Local Health District Human Research Ethics Committee (Health and Medical) (HE 13/189). Full written consent was obtained prior to study commencement. The study is registered with the ANZ Clinical Trial Registry (ANZCTR12614000581662).

Full details of the protocol and methodology have previously been described (Tapsell et al., 2015). In summary, at baseline 377 participants had *body weight* (kg) and *%body fat* measured using scales with a bio-electrical impedance component. *Fasting blood lipids* (total cholesterol, LDL, HDL, triglycerides) and *blood glucose* were collected and tested through a registered pathology service (Southern IML Pathology). *Systolic (SBP) and diastolic blood pressure (DBP)* was measured using the Omron BP-203RPEIII VP-1000 device (Omron Health Care, Kyoto, Japan). Arterial stiffness (baPWV) and arterial occlusion (*ankle brachial index - ABI*) data were also collected from this device, giving a direct measure of peripheral artery disease. An ABI of <0.90 or >1.40 was classified as indicative of peripheral artery disease, ABI between 0.90 and 1.00 was classified as at risk of peripheral artery disease, and ABI between 1.01 and 1.39 was classified as healthy. *Dietary intake* was assessed using 4 day food records completed by participants, which included one weekend day. Dietary data was analysed using FoodWorks software (Version 7, 2012, Xyris Software, Spring Hill, QLD, Australia). Food intake data was converted to energy and macronutrient intake using the AUSNUT2007 food composition database (AUSNUT, 2007). *Physical activity* was assessed using the International Physical Activity Questionnaire (IPAQ) short form survey questions (Craig et al., 2003) and by a scientific grade pedometer (Yamax Digiwalker SW200, Pedometers Australia) worn for 4 consecutive days to confirm an average number of steps per day. *Psychological profile* was assessed using the Physical and Mental Quality of Life Assessment (SF-12) (Jenkinson et al., 2001) and the Depression Anxiety Stress Scale (DASS-21) (Lovibond SHAPFL, 1995). *Socio economic position* was approximated by the use of level of education attained and the average household income, which were both self-reported in an on-line population survey completed during screening for the randomised controlled trial. These covariates were included in the analysis as they are related to the three components of the lifestyle intervention which were implemented in the HealthTrack study after the baseline assessment, those being: dietary counselling, exercise advice and psychological coaching.

MetS was calculated using the harmonised definition (Alberti et al., 2009), with waist circumference thresholds set at the AHA/NHLBI

(ATP III) levels of ≥ 102 cm for males and ≥ 88 cm for females. Participants were categorised as MUO if MetS was present or MHO if no MetS was present. Framingham CVD and coronary heart disease (CHD) risk were calculated based on the published CVD equations (D'Agostino et al., 2008) and CHD score sheets (Wilson et al., 1998). A risk percentage of <10% was categorised as 'low risk', and 10% or more were categorised as 'moderate to high risk' (<1% had a high risk of CVD and CHD, therefore moderate and high risk categories were combined). Similarly, for ABI the peripheral artery disease category (≤ 0.90) was combined with the at risk category (0.91–1.00) for analysis. Insulin resistance was defined as participants prescribed hypoglycaemic agents or a glycated haemoglobin (HbA1c) level >6%.

2.2. Statistical analysis

The statistical package used for analysis was IBM SPSS Statistics (version 21.0, IBM Corp, Chicago IL, 2012). Analyses included baseline data from all participants recruited into the HealthTrack study. The cross-sectional associations between physiological and lifestyle covariates and those classified as having MetS were analysed using backward stepwise logistic regression. Covariates included demographic (age, BMI, gender, % body fat); psychological (DASS-21, SF12 mental scale); physical activity (IPAQ, steps/day); dietary (total kilojoules, total fat, mono-unsaturated fatty acid (MUFA), polyunsaturated fatty acid (PUFA) and saturated fat); and social (education level, household income). Predictors which achieved a *p* value <0.2 in univariate analysis were assessed for inclusion in the multivariate model. The final model was checked using forward elimination (data not shown). Linear regression was used to examine associations between incidence of MetS and CVD risk, while logistic regression was used to test associations between MetS and CHD risk, peripheral artery disease and insulin resistance.

3. Results

Of the 377 participants randomised, data required for calculation of MetS was available for 374, and 127 participants (34.0%) were classified with MetS (Table 1). Multivariate analysis demonstrated that having a higher BMI (odds 1.26, *p* < 0.01) and older age (odds 1.08, *p* < 0.01) were significantly and positively associated with having MetS (Table 2). None of the other psychological, physical, dietary or social factors had a significant association with MetS classification.

To examine CVD and CHD estimated risk, data were available for *n* = 351 participants (Table 3). The median risk of experiencing cardiovascular disease within the next 10 years for all participants (interquartile range) was 3.89% (1.83–7.00). Most participants (88–92%) were in the low risk (<10%) category. Using linear regression, participants classified with MetS had a 4.50% increase in CVD risk compared to no-MetS participants (odds 4.50 (95% CI: 3.72, 5.28), *p* < 0.01). Using logistic regression, it was identified that MetS participants were 8.1 times more likely to be at risk of CHD than others (odds 8.07 (95% CI: 3.16, 20.62), *p* < 0.01).

A total of *n* = 26 (6.9%) of participants were categorised as being insulin resistant (15 on prescribed hypoglycaemic medication, 11 with HbA1c >6%). Using logistic regression, MetS participants were found to be 12.7 times more likely to be insulin resistant (odds 12.73 (95% CI: 4.28, 37.85), *p* < 0.01). In contrast, analysis of available data for MetS and ABI (*n* = 372) found that MetS did not significantly predict peripheral artery disease (odds 0.91 (95% CI: 0.55, 1.51), *p* = 0.72).

4. Discussion

Obesity is a public health problem that contributes significantly to the increasing prevalence of chronic diseases such as diabetes mellitus and CVD. However, given that metabolic risk may differ between obese individuals, the identification of those with MetS and the factors associated with this risk may assist in developing approaches to

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