Atherosclerosis 233 (2014) 363–369

Contents lists available at ScienceDirect

Atherosclerosis

journal homepage: www.elsevier.com/locate/atherosclerosis

Obesity and risk factors for cardiovascular disease and type 2 diabetes: Investigating the role of physical activity and sedentary behaviour in mid-life in the 1958 British cohort☆

Chris Power*, Snehal M. Pinto Pereira, Catherine Law, Myung Ki

Centre for Paediatric Epidemiology and Biostatistics, UCL Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK

ARTICLE INFO

Article history:
Received 1 November 2013
Received in revised form
7 January 2014
Accepted 15 January 2014
Available online 27 January 2014

Keywords:
Body mass index
Cardiovascular diseases
Glucose metabolism
Physical activity
Sedentary behaviour
TV-viewing

ABSTRACT

Objective: A key public health priority is to minimise obesity-related health consequences. We aim to establish whether physical activity (PA) or less sedentary behaviour ameliorate associations of obesity with biomarkers for cardiovascular disease (CVD) and type 2 diabetes.
Methods: Data on obesity (33 y), PA (42 y), TV-viewing and health biomarkers (45 y) are from the 1958 British birth cohort ($N = 9377$).
Results: Obesity was associated with an adverse biomarker profile for CVD and type 2 diabetes. For PA, men active ≥1/week had 1.09% (0.28, 1.90) lower diastolic blood pressure (DBP) than less active men; triglycerides were 2.08% (0.52, 3.64) lower per unit higher PA (on 4-point scale). TV-viewing was independently associated with several biomarkers, e.g. per unit higher TV-viewing (on 4-point scale) DBP was raised by 0.50% (0.09, 0.90) and triglycerides by 3.61% (1.58, 5.64). For both TV-viewing and PA, associations with HbA1c were greatest for the obese ($p_{interaction} \leq 0.04$): compared to a reference value of 5.20 HbA1c% in non-obese men viewing 0–1 h/day, HbA1c% differed little for those viewing >3 h/day; among obese men HbA1c% was 5.36 (5.22, 5.51) and 5.65 (5.53, 5.76), for 0–1 and >3 h/day respectively. For PA in non-obese men, the reduction associated with activity ≥1/week was negligible compared to a reference value of 5.20 HbA1c% for those less active; but there was a reduction among obese men, HbA1c % was 5.50 (5.40, 5.59) vs 5.66 (5.55, 5.77) respectively.
Conclusion: Reduced TV-viewing and prevention of infrequent activity have greatest beneficial associations for glucose metabolism among the obese, with benefits for other biomarkers across obese and non-obese groups.

1. Introduction

Health-damaging effects of obesity have been found for mortality, cardiovascular disease, type 2 diabetes and associated risk factors such as blood pressure and lipids [1]. There are also many studies suggesting protective effects of physical activity (PA) and deleterious effects of sedentary lifestyles on all-cause and cardiovascular disease (CVD) mortality[2–4], type 2 diabetes and associated biomarkers, such as blood pressure[5–8]. Some studies demonstrate that obesity, PA and sedentary behaviour have separate contributions to CVD and diabetes-related outcomes [2,4,5,9–13]. In addition to the observational evidence for type 2 diabetes, benefits of PA have been shown in intervention studies that emphasise weight control, PA and dietary modification [14,15]. With increasing trends in obesity, substantial proportions of the global population are now at risk of obesity-related ill-health [16,17]. Strategies to halt the rising trend in obesity are important, but action is needed simultaneously for generations already affected. Thus, a key public health priority is to minimise obesity-related health consequences. In this context, potentially modifiable factors, such as PA and sedentary lifestyles, should be considered.

Observational studies suggest that obese adults may reap greater health benefits from being physically active or less sedentary than the non-obese. In a Finnish study, the impact of leisure-time PA on the risk of death from ischaemic heart disease was stronger in men whose body mass index (BMI) was ≥27 kg/m^2 [18]. For type 2 diabetes, a review of 8 studies found that obese groups who were physically inactive had an increased risk greater than the additional effect of each factor (obesity and inactivity) separately [19], whilst an earlier study had reported stronger protective effects of activity among obese individuals [9]. Yet, not all studies show

* Corresponding author. Tel.: +44 (0)207 905 2106; fax: +44 (0)20 7905 2381.
E-mail address: christine.power@ucl.ac.uk (C. Power).

0021-9150/$ – see front matter © 2014 The Authors. Published by Elsevier Ireland Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.atherosclerosis.2014.01.032

greater benefits of activity amongst obese groups for type 2 diabetes [20] or coronary heart disease [2,5]. Research is scarce on whether benefits of activity vary for obese and non-obese groups across multiple CVD and diabetes biomarkers and whether there are corresponding patterns for sedentary behaviour. The lack of evidence for sedentary behaviour is important given that such behaviours are increasing [21]. As argued elsewhere, interactive effects of activity (or sedentary behaviour) and obesity imply that prevention of either reduces type 2 diabetes risk by abolishing the separate effects of each factor as well as the increased risk due to the interaction between them [19].

We investigated whether obesity-CVD or diabetes biomarker associations can be lessened by PA or lower sedentary behaviour in the 1958 British birth cohort. Our aim was to determine whether obesity, PA and sedentary behaviour were independently associated with biomarkers for CVD and type 2 diabetes and, also, whether PA or sedentary behaviour moderated the obesity − biomarker associations. We investigated the association between obesity in early adulthood (33 y) with CVD and diabetes biomarkers twelve years later (at 45 y) together with PA and sedentary behaviour (indicated by TV-viewing) recorded before or at 45 y. This temporal sequence was used to follow the ordering for potential interventions to reduce the health burden associated with obesity.

2. Methods

2.1. Study sample

The 1958 cohort consists of 17,638 males and females followed from birth during one week, March 1958, in England, Scotland and Wales [22]. Information was collected at several ages throughout child and adulthood. At 45 y, individuals still in contact with the study, and who at 42 y had not required a proxy interview ($n = 11{,}971$) were invited to a home-based clinical assessment by a nurse; the 9377 (78%) who responded were broadly representative of the total surviving cohort [23]. The 45 y survey included blood collection, to which participants gave written informed consent. Ethical approval was given by the South-East Multi-Centre Research Ethics Committee (ref: 01/1/44).

2.1.1. Outcomes

All CVD and diabetes biomarkers were obtained at 45 y from measurements taken by nurses using standardised protocols. After participants were seated for five minutes, blood pressure was measured three times (Omron 705CP, Tokyo, Japan); average systolic and diastolic blood pressure (SBP and DBP) values were used. Non-fasted venous blood samples were obtained and posted to a central laboratory. Glycosylated haemoglobin (HbA1c) levels were measured using ion exchange high performance liquid chromatography. Total-, HDL-cholesterol and triglyceride levels were analysed by an autoanalyzer (Olympus AU640, Japan) using enzymatic methods. LDL-cholesterol was calculated using the Friedewald formula [24], except when triglyceride levels were >4.5 mmol/l. Nurses recorded currently prescribed medications (observed from packaging) from which we identified type 2 diabetes, anti-hypertensive and lipid-lowering medications.

2.1.2. Obesity, PA and sedentary behaviour

Height and weight were measured at 33 y using standardised protocols and BMI calculated as weight/height^2 (kg/m^2). Obesity was defined as BMI $\geq$ 30 kg/m^2. Also, we identified i) the top BMI quintile at 33 y (men $\geq$ 28 kg/m^2; women $\geq$ 27 kg/m^2); ii) 42 y obesity, from BMI based on self-reported weights and heights; and iii) 45 y obesity, from waist circumference (measured midway between the lower ribs and iliac crest) using cut-offs: $\geq$102 cm for men; $\geq$88 cm for women.

Leisure-time PA at 42 y was assessed using a question about frequency of regular activity (to aid recall, several activities were provided as examples) categorised into four groups: $\leq$2–3 times/month, once/week, 2–3 times/week and 4–7 times/week. For leisure-time sedentary behaviour we used self-reports at 45 y of average daily TV-viewing in the previous year, in four groups (0–1 to $\geq$3 h/day).

2.1.3. Covariates

Several covariates were identified from the literature. Birthweight was recorded prospectively, measured in pounds and ounces, and converted into kilograms. Smoking, reported at 33 y, was categorised as never, ex-smoker, or current smoker. Socioeconomic position (SEP), based on the Registrar General's Social Classification of their occupation at 33 y, was categorised into four groups: I (professional) or II; IIINM; IIIM; and IV or V (unskilled). Highest qualification by 33 y was categorised into five groups: none, some, O-levels, A-levels or degree. Longstanding illness, disability or infirmity limiting daily activities were identified at 33 y. Diet at 33 y included consumption frequency of fruit (five categories: <1 day/wk to >1/day), chips (five categories: 1+/day to never) and alcohol (five categories: most days to never). For women, hormone replacement therapy (HRT), oral contraceptive (OC) and menopausal status were ascertained at 45 y. Hypertension (SBP $\geq$ 140 mmHg or DBP $\geq$ 90 mmHg [25] or on medication for high blood pressure) was a covariate for some biomarkers.

2.2. Statistical analyses

To facilitate comparison, all biomarkers were log-transformed and multiplied by 100; in analyses, regression coefficients are interpreted as symmetric percentage differences in means [26]. To avoid possible bias in associations that can occur by excluding participants using medications or by ignoring medication use, we followed the recommendation to correct biomarker levels [27]. For individuals on medication for high blood pressure ($n = 424$) we corrected values by +10 mmHg for DBP and SBP respectively [27]; for oral medication for type 2 diabetes ($n = 122$) +1% in absolute terms for HbA1c [28] and for lipid-lowering drugs ($n = 126$) +25% for total-cholesterol, +54% for LDL-cholesterol, +18% for triglycerides, −5% for HDL-cholesterol [29]. Individuals with type I diabetes ($n = 56$) were excluded from analysis of HbA1c.

Preliminary work showed that several 33 y obesity, 42 y PA and 45 y TV-viewing associations with biomarkers varied by sex ($p_{\text{interaction}} < 0.02$), hence, we assessed associations separately for men and women. In preliminary analysis we also found no trend in blood pressure or HbA1c across categories of increasing activity frequency, except between the least active ($\leq$2–3 times/month) and others. Thus, for these biomarkers we treated activity as a dichotomous variable, but as a continuous variable for other biomarkers. Associations for TV-viewing and biomarkers showed trends across the four categories, so TV-viewing was treated as continuous in all analyses. Linear regression models were undertaken as follows: Model 1, unadjusted; Model 2, adjusted for covariates; and Model 3, adjusted for covariates plus the two other main exposures (i.e. BMI, PA or TV-viewing as appropriate). To account for co-morbidity, adjustments were made for hypertension and total- and HDL-cholesterol. For all biomarkers, we tested interactions (i.e. effect modification) between obesity and (i) PA or (ii) TV-viewing. To establish whether any interactions ($p < 0.05$) were due to differences in adiposity concurrent with biomarker measurement, we further adjusted for 45 y BMI and waist