Lean body mass and risk of type 2 diabetes - a Danish cohort study

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Abstract

Purpose Excess body fat is a commonly known risk factor for type 2 diabetes. However, whether lean body mass, or fat free mass, could have a protective effect against type 2 diabetes, remains unclear. The aim of this study was to explore the association between lean body mass, fat mass and type 2 diabetes.

Methods This study used data from the Danish Diet, Cancer and Health cohort of 37,053 men and women, aged 50–64 years at baseline (1993–1997). The exposure was measurements of body composition using bioelectrical impedance analysis. Incident diabetes during follow-up was determined through linkage to the Danish National Diabetes Register. Cox proportional hazards regression analysis was used to estimate HR and 95%CI for the association between lean body mass and incident type 2 diabetes, with and without adjustment for fat mass. A sensitivity analysis was performed, excluding cases of incident type 2 diabetes within the first 2 years of follow-up.

Results When adjusted for fat mass, the main analysis showed non-linear inverse association between lean body mass and risk of diabetes for men, but not for women. However, the sensitivity analysis found no association for either men or women.

Conclusions Lean body mass was not associated with incident type 2 diabetes when excluding cases that may have been subclinical at baseline. The results imply that public health should focus on reduction of fat mass for diabetes prevention.

Keywords Type 2 diabetes · Lean body mass · Fat mass · Body composition

Introduction

An estimated 422 billion people have diabetes worldwide and the prevalence is rising. Diabetes is a major cause of disability and premature death, and thus focus on prevention is important [1]. Commonly known risk factors for type 2 diabetes include overweight and obesity, as defined by excess body fat, and these are directly associated with incidence of type 2 diabetes [2, 3]. However, the role of lean body mass, or fat free mass, remains unclear, and only few prospective cohort studies have been performed. Some studies suggest an inverse association between lean body mass and development of type 2 diabetes [4–6], while others show no association [7, 8]. The existing literature on this

topic is sparse and includes a broad range of methods and study populations, making comparison between studies difficult, in contrast to studies focusing on fat mass, which have consistently shown positive association between obesity and risk of type 2 diabetes across study populations [9, 10].

Skeletal muscle mass is a main component of lean body mass, and helps to regulate blood glucose levels by storing glucose, and converting it to energy [11]. Total lean body mass could therefore potentially reduce risk of type 2 diabetes by facilitating stable blood sugar levels, thus preventing hyperglycemia. On the other hand, the role of fat tissue in the development of type 2 diabetes includes the release of free fatty acids (FFA) which reduce insulin secretion from pancreatic β -cells. This contributes to insulin resistance in muscle tissue and can lead to hyperglycemia [12]. Fat tissue and lean body mass thus affect blood glucose levels, in opposite directions, and the net contributions to the development of type 2 diabetes are unclear.

To investigate how lean body mass and fat tissue affect the development of type 2 diabetes, this study aimed to explore associations between lean body mass, fat mass and type 2 diabetes in a large, prospective cohort study.

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Materials and methods

Study design

This cohort study used data from the Diet, Cancer and Health study, which recruited participants from December 1993 to May 1997, including a clinic visit in either Aarhus or Copenhagen where anthropometry was measured, and data from self-administered questionnaires was collected [13].

Subjects

People were invited to participate in the Diet, Cancer and Health cohort if they met the following inclusion criteria: 50–64 years of age at the time of invitation, born in Denmark, living in or near Aarhus or Copenhagen, the two largest cities in Denmark and not previously diagnosed with cancer. Eligible cohort participants were identified and located through the Danish Civil Registration system, which assigns an individual 10-digit number to every person living in Denmark [14].

For this study, participants were excluded if they reported diabetes at entry to the study, or had a diagnosis of diabetes prior to baseline in the National Diabetes Registry, if they were missing covariate information, or if their values for lean body mass(%) and fat mass(%) were implausible. While there is no general consensus on specific cut-off for ranges for plausible fat percentage [15], participants with a fat percentage lower than 5% for men and 12% for women were excluded from the study, as these values were considered implausible.

Exposures

Measurements of body composition were obtained by bioelectrical impedance analysis (BIA), performed by trained technicians at the study centers. Measurements were obtained using a 50 Hz single frequency device (BIA 101-F, Akern/RJL, Florence Italy), as described elsewhere [16]. Body composition was calculated using algorithms including sex, age, weight and height, developed previously based on data from validated BIA measurements of 139 Danish adults [17]. Absolute values for lean body mass and fat mass were calculated based on the relative measurements of lean body mass and fat mass, respectively, and the total weight of each participant. In this study, lean body mass(%) is defined as total body mass, minus fat mass(%).

Other variables

Measurements of height and weight were taken by trained lab technicians at the study centers during the clinical visit. Height was measured to the nearest 0.5 cm, with the participants standing without shoes. Weight was measured by digital

scales with the participants wearing light clothing, and recorded to the nearest 0.1 kg. [18]. Information on other covariates, including physical activity (>/< 30mins/day), smoking (never, former or current smoker), alcohol intake (no alcohol intake, <10 g/d, <20 g/d, <30 g/d or > 30 g/d) and educational history (none, short, medium or long tertiary education) was obtained by completion of a self-reported questionnaire, filled in by the participants at the study centers [13].

Outcome

Incident cases of diabetes during follow-up were obtained by register linkage to the Danish National Diabetes Register. A person was registered in the National Diabetes Register as a case if he/she was either registered in the National Patient Register with a diagnosis of diabetes, was registered as a diabetic patient receiving chiropody in the National Health Service Register, had five blood-glucose measurements in a 1 year period in the National Health Service Register, had two blood-glucose measurements per year in 5 consecutive years in the National Health Service Register, or purchased anti-diabetic drugs, as recorded in the Danish National Prescription register. The date of inclusion in the register was therefore not the date of diabetes diagnosis, but the date on which one of the above inclusion criteria was met. Incidence data in the National Diabetes Register was complete from August 1997 onwards. The National Diabetes Register did not differentiate between types of diabetes [19].

Information regarding death and emigration was obtained by linking the records from the Diet, Cancer and Health cohort to those of the Civil Registration System, which contains complete information on death and emigration for all people living in Denmark.

All cohort participants were followed from entry to the study to censoring, which was the age of diagnosis in National Diabetes Register, death, emigration or end of follow-up on 31.12.2011, whichever came first.

Ethics

All cohort participants provided informed consent to participate and the study was approved by relevant Ethical Committees and the Danish Data Protection Agency [13]. The present study was approved by the Danish Data Protection Agency in December 2017.

Statistical analysis

Cox Proportional Hazards Regression models were used to estimate hazard ratios (HR) and 95% confidence intervals (CI) for the association between lean body mass and incident type 2 diabetes, with age as the underlying time-variable. Age at recruitment was defined as entry into the study, and exit was

defined as age at censoring. The associations between lean body mass and type 2 diabetes were modeled as 4-knotted, restricted cubic splines, using the mean as reference. Analyses were performed separately for men and women, to account for sex differences in body composition.

Initially, the association between lean body mass and incident type 2 diabetes was investigated (Model 1a) and then adjusted for fat mass (Model 1b). Then a sensitivity analysis was performed with the aim to investigate any differences between short- and long-term risk. The sensitivity analysis excluded incident diabetes cases that occurred within the first 2 years of follow-up, with the aim to assess a potential difference in the association between lean body mass and all cases or later-onset cases. Finally, we assessed the association between fat mass (kg) and incident type 2 diabetes, with and without adjustment for lean body mass, in the full cohort (Model 2a and 2b).

All models included adjustment for age, physical activity, smoking, alcohol intake and education, and were stratified by quartiles of inclusion date and performed separately for each sex. Potential confounders were entered into all models as categorical variables, as shown in Table 1. The proportional hazards assumption could not be rejected for any of the analyses ($p > 0,05$). All statistical analyses were performed in Stata/IC version 15.1, with a statistical significance level of 5%.

Results

Of the 160,725 people who met the inclusion criteria, and received an invitation, 57,053 people, including 27,190 men and 29,863 women, chose to participate [13]. Of these, 569 people were excluded due to cancer diagnosed prior to enrolment, 161 people were excluded due to self-reported type 2

Table 1 Baseline characteristics of all study participants (total $n = 54,295$) and cases of incident type 2 diabetes

	Men		Women	
	All Participants ($N = 25,770$)	Cases ($N = 3973$)	All Participants ($N = 28,525$)	Cases ($N = 3178$)
Age at entry (mean(sd))	56,1 (4,4)	56,4 (4,4)	56,2 (4,4)	56,8 (4,4)
Lean body mass (kg) (mean(sd))	60,6 (6,1)	62,3 (6,7)	44,4 (4,5)	46,0 (5,2)
Fat mass (kg) (mean(sd))	22,3 (7,4)	26,8 (8,4)	24,4 (8,7)	30,3 (10,5)
Lean body mass (%) (mean(sd))	73,7 (5,4)	70,5 (5,1)	65,5 (6,6)	61,3 (6,4)
Fat mass (%) (mean(sd))	26,3 (5,4)	29,5 (5,1)	34,5 (6,6)	38,7 (6,4)
Physical activity (%(N))				
<30 min/day	62,0% (15,949)	68,0% (2702)	59,0% (16,821)	64,0% (2036)
>30 min/day	38,0% (9821)	32,0% (1271)	41,0% (11,704)	36,0% (1142)
Smoker (%(N))				
Never	25,9% (6670)	21,4% (854)	43,6 (12,485)	41,0% (1307)
Former	34,7% (8925)	36,2% (1436)	23,6% (6709)	23,3% (742)
Current	39,5% (10,175)	42,4% (1683)	32,8% (9331)	35,6% (1129)
Alcohol intake (%(N))				
0 g/day	1,8% (454)	1,9% (74)	2,62% (752)	3,8% (121)
<10 g/day	22,2% (5721)	24,1% (959)	49,3% (14,067)	53,9% (1709)
<20 g/day	27,3% (7056)	26,2% (1042)	27,1% (7742)	22,8% (727)
<30 g/day	11,3% (2906)	9,8% (386)	4,52% (1293)	4,4% (140)
≥30 g/day	37,44% (9641)	38,1% (1513)	16,4% (4704)	15,1% (481)
Education (%(N))				
No tertiary education	9,8% (2531)	12,8% (511)	19,1% (5444)	26,0% (827)
Short tertiary education	13,7% (3517)	14,9% (593)	31,6% (8988)	31,1% (986)
Medium tertiary education	42,3% (10,893)	43,2% (1714)	38,0% (10,863)	34,6% (1101)
Long tertiary education	34,2% (8829)	29,1% (1155)	11,3% (3230)	8,3% (264)
Body Mass Index (BMI) categories (%(N))				
BMI <25	35,3% (9089)	16,5% (655)	52,2% (14,877)	26,7% (850)
BMI 25–30	50,1% (12,911)	50,6% (2008)	34,3% (9786)	39,8% (1266)
BMI >30	14,6% (3769)	32,9% (1310)	13,5% (3862)	33,5% (1062)

diabetes at baseline, 1222 people were excluded due to a diagnosis of diabetes in the National Diabetes Register at baseline, 770 people were excluded due to missing information and 36 people were excluded due to implausible values of fat percentage. These exclusions resulted in a total of 54,295 eligible participants, including 25,770 men and 28,525 women, for this study. During a total of 765,682 person years of follow-up and an average follow-up time of 14.1 years, 7151 cases of incident diabetes were recorded, including 3973 men and 3178 women.

Participants who developed diabetes during follow-up had lower lean body mass(%) and a higher BMI at baseline than the full cohort (Table 1). Participants who developed diabetes during follow-up were less likely to report being physically active, were more likely to be current smokers, to be of lower educational attainment, and to report a history of hypertension or hypercholesterolemia at baseline, than the full cohort.

Baseline characteristics for the early incident cases, who were excluded in the secondary analysis, revealed poorer health compared to the later incident cases, with early cases being more likely to have a BMI > 30, a higher fat percentage, and be less physically active than the later cases.

Lean body mass was positively associated with type 2 diabetes in both sexes (model 1a). However, when adjusting for fat mass, a weak inverse association with risk of type 2 diabetes was seen in men, but not in women (Fig. 1).

The sensitivity analysis excluding cases occurring within the first 2 years of follow up ($n=465$), showed no inverse association between lean body mass and risk of type 2

diabetes, either for men or for women, when adjusted for fat mass (Fig. 2).

Fat mass was statistically significantly positively associated with incident type 2 diabetes in both sexes, and the association remained largely unchanged after adjustment for lean body mass (Fig. 3).

Discussion

This prospective cohort study aimed to investigate how lean body mass and fat tissue affect the development of incident type 2 diabetes. In the main analyses, we observed that a high level of lean body mass was associated with a slightly lower risk of type 2 diabetes in men after adjusting for fat mass, but not in women. In sensitivity analyses excluding cases occurring within the first 2 years of follow-up, we found no association between lean body mass and type 2 diabetes for either sex. High levels of fat (kg) were associated with a higher risk of type 2 diabetes, as previously identified in other studies [9, 10]. This association persisted when adjusting for lean body mass.

This study has strengths as well as limitations. Only 286 (0.53%) participants emigrated during follow-up, limiting the concern for selection bias. The use of BIA to determine body composition has previously been validated against DXA and has been found to be a valid tool, with very little difference in results of the measurements produces by these two methods [20–23], limiting concern for misclassification of the exposure. The National Diabetes Register does not differentiate between types of diabetes. However, given the age of the

Fig. 1 The association between lean body mass and incident type 2 diabetes, modelled by 4-knotted restricted cubic splines for men and women ($n=54,295$). Models 1a were adjusted for age, physical activity, smoking, alcohol consumption and education. Models 1b include additional adjustment for fat mass. The solid lines show HR while the dotted lines show 95% CI, with reference point at the mean for lean body mass.

Fig. 2 The association between lean body mass and incident type 2 diabetes, modelled by 4-knotted restricted cubic splines for men and women, after exclusion of incident cases within the first 2 years of follow up were excluded ($N = 465$). Models 1a were adjusted for age, physical activity, smoking, alcohol consumption and education. Models 1b on the right include additional adjustment for fat mass. The solid lines show the association (HR) while the dotted lines show 95% CI, with reference point at the mean for lean mass (kg)

study population diagnoses of type 2 diabetes, rather than other types, are most likely in this study. Registration in the National Diabetes Register is independent of exposure measurement in the cohort, limiting concern for differential misclassification of the outcome. Nevertheless, because incident type 2 diabetes in this study was determined upon registration in the National Diabetes Register, which in many cases occurs

when a patient needs treatment for their condition, incident cases registered early on in the follow-up period may actually have had type 2 diabetes prior to entry to the study. People with type 2 diabetes have been found to have an accelerated loss of muscle mass, compared to healthy individuals of a similar age [24, 25]. If the baseline measurements of lean body mass were lower for people who already had type 2 diabetes,

Fig. 3 The association between fat mass and incident type 2 diabetes, modelled by 4-knotted restricted cubic splines for men and women. Models 2a were adjusted for age, physical activity, smoking, alcohol consumption and education. Models 2b on the right include additional adjustment for lean body mass. The solid lines show HR while the dotted lines show 95% CI, with reference point at the mean for fat mass

unregistered in the National Diabetes Register, than for healthy participants, this could induce an association between lean body mass and type 2 diabetes, as a result of reverse causation. To assess if this was the case, we performed a sensitivity analysis, in which we excluded cases registered within the first 2 years of follow-up. The difference between the two analyses indicates that reverse causation may have caused the inverse association in the main analysis, as the association was attenuated in the sensitivity analysis. However, if the initial cases were those most sensitive to the development of diabetes as a result of their body composition, compared to later cases, this exclusion may introduce selection bias into the study. We adjusted for known confounders, which did not alter the strength of the observed associations. Nevertheless, residual confounding cannot be excluded. The study population was largely white, and caution should be taken when extrapolating results to populations of other ethnicity, for whom anthropometry may associate differently with risk of diabetes [26, 27].

Existing knowledge regarding the regulation of blood glucose supports the plausibility of an inverse association between lean mass and risk of type 2 diabetes [11]. lean body mass plays a role in reducing blood sugar, while fat tissue reduces insulin secretion, which can in turn lead to hyperglycemia [12]. Thus, a person with greater lean body mass could have a lower risk of developing type 2 diabetes than a person with less lean body mass. However, this study indicates that this is not the case.

Previous studies have defined lean body mass in different ways, using calculated measurements such as “Muscle Mass Index” [4] and “Relative Muscle Mass” [5]. These differences in definition of the exposure along with the variations between studies mentioned above, means comparison of existing results with those of this study must be done with some caution. Previous studies of the association between lean body mass and type 2 diabetes have unsurprisingly shown differing results, with some studies identifying an inverse association [4–6] and others not [7, 8]. The difference in results of the analysis of the association between lean body mass and type 2 diabetes may be due to differences in the analytical approach, where some studies include adjustment for fat mass, and others do not [5, 6]. One study analyzed tertiles of “Muscle Mass Index” and risk of type 2 diabetes, adjusting for fat percentage as a dichotomous variable [4]. However, the absolute values of fat are likely to vary between the tertiles, which would not be captured in this analytical approach. As a result, the observed inverse associations may actually be due to low fat mass, and not high lean body mass. Of the two existing studies that do not identify an association between lean body mass and type 2 diabetes, one included adjustment for total body fat [7]. The other [8] assessed differences between intervals of lean body mass, and similarly found no association, which is in line with the results of this study. However, several

studies are in agreement that fat mass is an important risk factor for developing type 2 diabetes [9, 10, 12].

Conclusions

This study suggests that lean mass is not significantly associated with risk of incident type 2 diabetes for men or for women when early cases, that may have been subclinical at entry to the study, are excluded. The findings may be used in a public health context to inform health practitioners and the general public alike, that focus should be on reducing absolute fat mass, when the main aim is to reduce diabetes risk.

Compliance with ethical standards

Conflict of interest The authors declared that they have no conflict of interest.

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