Metabolism
www.metabolismjournal.com

Muscle grip strength predicts incident type 2 diabetes: Population-based cohort study

Joule J. Li[a,b,*], Gary A. Wittert[b], Andrew Vincent[b], Evan Atlantis[c], Zumin Shi[d], Sarah L. Appleton[a,b], Catherine L. Hill[a], Alicia J. Jenkins[e], Andrzej S. Januszewski[e], Robert J. Adams[a]

[a] The Health Observatory, School of Medicine, University of Adelaide
[b] Freemasons Foundation Centre for Men's Health, School of Medicine, University of Adelaide
[c] School of Nursing and Midwifery, Western Sydney University
[d] Population Research and Outcome Studies, School of Medicine, University of Adelaide
[e] NHMRC Clinical Trials Centre, Sydney Medical School, University of Sydney

ARTICLE INFO

Article history:
Received 11 February 2016
Accepted 22 March 2016

Keywords:
Skeletal muscle
Lean mass
Dual energy X-ray absorptiometry
Muscle strength
Grip strength

ABSTRACT

Objectives. To determine the longitudinal relationship of muscle mass and strength with incident type 2 diabetes, and previously unstudied mediating effects of testosterone and inflammation.

Methods. Community-dwelling male participants (aged ≥35 years) of the Men Androgen Inflammation Lifestyle Environment and Stress (MAILES) Study underwent biomedical assessment in 2002–2006 and 2007–2010, including hand grip strength (dynamometer), testosterone and inflammatory markers. Body composition (dual-energy X-ray absorptiometry) was assessed at baseline only. Incident type 2 diabetes was defined as a self-reported doctor diagnosis, diabetes medication use, fasting plasma glucose ≥7.0 mmol/L, or glycated haemoglobin ≥6.5% (48 mmol/mol) at follow-up, that was not present at baseline.

Results. Of n = 1632 men, incident type 2 diabetes occurred in 146 (8.9%). Muscle mass was not associated with incident type 2 diabetes. Grip strength was inversely associated with incident type 2 diabetes [unadjusted odds ratio (OR) per 5 kg: 0.87, 95% confidence interval (CI): 0.80–0.95; adjusted OR, 95% CI: 0.87, 0.78–0.97]. Arm muscle quality (grip strength divided by arm lean mass) was similarly associated with incident type 2 diabetes. Testosterone, IL-6 and TNF-α did not significantly mediate the associations. The population attributable fraction of type 2 diabetes from low grip strength was 27% (13–40%), assuming intervention could increase strength by 25%.

Conclusions. Reduced muscle strength, but not reduced muscle mass, is a risk factor for incident type 2 diabetes in men. This is not mediated by testosterone or inflammation. Intervention could prevent a substantial proportion of disease.

Abbreviations: ASM, appendicular skeletal muscle mass; ASMI, appendicular skeletal muscle mass index; DXA, dual-energy xray absorptiometry; FAMAS, Florey Adelaide Male Ageing Study; FMI, fat mass index; MAILES, Men Androgen Inflammation Lifestyle Environment and Stress; NWAHS, North West Adelaide Health Study; MET, metabolic equivalent; PAF, population attributable fraction.

* *Corresponding author at:* The Health Observatory Basil Hetzel Institute, Discipline of Medicine, University of Adelaide, The Queen Elizabeth Hospital Campus, Woodville Rd, Woodville, South Australia, 5011, Australia. Tel.: +61 404 787 603; fax: +61 8 8222 6042.
E-mail address: joule.li@adelaide.edu.au (J.J. Li).

http://dx.doi.org/10.1016/j.metabol.2016.03.011
0026-0495/

1. Introduction

The prevalence of type 2 diabetes has been increasing worldwide [1], in association with rising obesity. However, increased adiposity contributes to only 37–77% of incident type 2 diabetes, depending on the population studied [2,3]. Thus, identification of new and potentially modifiable risk factors is needed to inform strategies for prevention.

Multiple cross-sectional studies have shown type 2 diabetes is inversely associated with skeletal muscle mass [4,5] and strength [6,7]. Muscle strength is also inversely associated with the development of insulin resistance [8]. However, there are few longitudinal studies that have investigated the role of muscle mass and strength in the development of type 2 diabetes. Only three longitudinal studies have investigated muscle strength and incident type 2 diabetes, and they showed conflicting results which may due to methodological limitations from an inability to identify undiagnosed cases [9–11]. Furthermore, only one previous study has investigated muscle mass and incident type 2 diabetes, which found that changes in muscle mass did not predict incident type 2 diabetes [12]. However, that study relied on bioelectrical impedance analysis instead of gold standard measures such as dual-energy X-ray absorptiometry (DXA) [12]. The association between reduced skeletal muscle mass or strength and incident type 2 diabetes is therefore unclear.

Testosterone is a determinant of muscle mass and strength [13], and low testosterone has also been associated with type 2 diabetes in men [14]. Therefore low testosterone may mediate the association between skeletal muscle dysfunction and type 2 diabetes in men. Similarly, inflammation predicts decline in skeletal muscle mass and strength [15], while also predicting incident type 2 diabetes [16]. Hence inflammation may also mediate any association between skeletal muscle and incident type 2 diabetes. These mechanisms have not been previously explored.

We therefore aimed to investigate the association between measures of skeletal muscle mass and strength with incident type 2 diabetes in a prospective community-dwelling cohort of men, and whether testosterone or inflammation mediates that association.

2. Methods

2.1. Cohort participants

The Men Androgen Inflammation Lifestyle Environment and Stress (MAILES) study is a longitudinal cohort of community-dwelling men, and has been described previously [17]. In brief, MAILES consists of two concurrent prospective cohorts: the Florey Adelaide Male Ageing Study (FAMAS) [18] and the age-matched men from the North West Adelaide Health Study (NWAHS) [19]. The two cohorts are largely representative of the male population of South Australia, and used the same methodology for random population sampling.

Detailed demographic, comorbidity (doctor diagnosed diabetes, cardiovascular disease), hand dominance, and risk factor data (smoking, physical activity) were collected by self- and follow-up were conducted in 2 hospital-based clinics using standardised and reproducible study protocols, including grip strength and blood pressure measurement, anthropometry, and fasting blood samples (lipids, glucose, glycated hemoglobin, testosterone and inflammatory markers). Hypertension was defined as any of: self-report, a clinically measured systolic blood pressure of ≥140 mmHg (mean of two or three readings), diastolic blood pressure of ≥90 mmHg (mean of two or three readings), or use of anti-hypertensive medication. Metabolic syndrome was defined by International Diabetes Federation criteria. Mild (walking), moderate, and vigorous physical activity levels were determined by the Australian National Health Survey Physical Activity instrument and converted into metabolic equivalents (METs). In the NWAHS, depression was defined as a score of ≥21 on the Center for Epidemiological Studies Depression Scale, whereas in FAMAS, depression was defined as a score of ≥12 on the Beck Depression Inventory. Cardiovascular disease was defined as a self-report of doctor-diagnosed myocardial infarction, angina, stroke or transient ischemic attack.

Approval for MAILES was obtained from the Human Research Ethics Committees of the North West Adelaide Health Service and the Royal Adelaide Hospital. All participants gave written informed consent. Baseline data were obtained in 2002–2006 and follow-up data in 2007–2010. Fig. 1 shows the participant flowchart. Almost all (96%) participants were born in Australia or Western Europe (including the UK/Ireland).

2.2. Skeletal muscle measures

Grip strength was measured with a Jamar analog hand dynamometer in the NWAHS cohort (Lafayette Instrument Company, Lafayette, IN) or a Smedley analog hand dynamometer (Stoelting Corporation, Wood Dale, IL) in the FAMAS cohort [17]. To account for any systematic differences in the type of hand dynamometer used, we included cohort as a covariate in adjusted statistical analyses. Main analyses used the mean of three measurements in the dominant hand. Sensitivity analyses used the maximum (i.e. peak) measurement recorded in either hand. We also undertook sensitivity analyses to account for: 1) bodily (e.g. hand) pain, as determined by the bodily pain scale of the SF-36; 2) self-reported current smoking at baseline; and 3) use of systemic corticosteroids at follow-up.

Body composition was measured at baseline in a sub-set of the cohort (n = 1181): NWAHS participants aged ≥50 years [20], and all FAMAS participants [18]. Whole-body, arms, and legs lean mass and fat mass were measured by DXA using default settings on either a pencil-beam (DPX+, Lunar software v4·7e) or fan-beam (Prodigy DF+ 14759, Encore software v9·15) densitometer. Both machines were from GE Lunar (Madison, WI) and provided similar results [21]. Appendicular skeletal muscle mass (ASM) was calculated by summing the lean mass of arms and legs. ASM and fat mass were normalised for height by dividing by height squared to generate ASM index (ASMI), and fat mass index (FMI), respectively [20,22].

Arm muscle quality (grip strength corrected for arm lean mass) was calculated by dividing the sum of the grip strength in both hands by arms lean mass [23,24]. Sensitivity analyses were also undertaken using arm muscle quality calculated with peak grip strength.

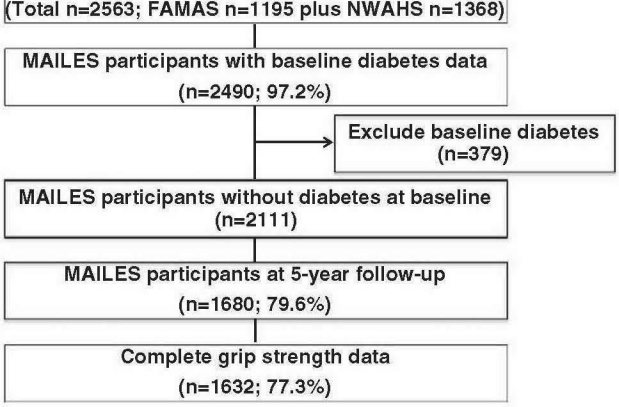

Fig. 1 – Flow of MAILES participants throughout the study. MAILES, Men Androgen Inflammation Lifestyle Environment and Stress study; FAMAS, Florey Adelaide Male Aging Study; NWAHS, North West Adelaide Health Study.

2.3. *Type 2 diabetes*

Type 2 diabetes was defined as: previous doctor diagnosis, diabetes medication use, fasting plasma glucose (FPG) ≥7.0 mmol/L (≥126 mg/dl), or glycated haemoglobin (HbA1c) ≥6.5% (48 mmol/mol). Diabetes medication use was identified by prescription of Anatomical Therapeutic Chemical classification A10 drug(s) from the Pharmaceutical Benefits Scheme of Australia. FPG was quantified using an automated chemistry analyzer system (Olympus AU5400; Olympus, Tokyo, Japan). HbA1c was measured by high-performance liquid chromatography using a spherical cation exchange gel (CV, 2% at 6% of total haemoglobin).

2.4. *Testosterone and inflammatory markers*

Serum total testosterone was measured by validated stable-isotope dilution liquid chromatography–tandem mass spectrometry (API-5000, Applied Biosystems/MDS SCIEX, Ontario, Canada) [25]. All measurements were undertaken on morning samples after an overnight fast; further detail is available in previous publications [17,26]. Serum interleukin-6 (IL-6) and serum tumour necrosis factor-alpha (TNF-α) were measured by enzyme-linked immunosorbent assay (ELISA) (R&D Systems, Minneapolis, MN) [17].

2.5. *Statistical analysis*

Statistical analysis was using SPSS version 20 (SPSS, IL) and R version 3.2.1 (R Foundation for Statistical Computing, Vienna, Austria). Differences in baseline characteristics between groups were determined using t-tests (numerical variables) or χ^2-tests (categorical variables). The unadjusted and adjusted associations between skeletal muscle measures and incident type 2 diabetes, and any interaction effects, were determined using binary logistic regression models. Collinearity was assessed using the variance inflation factor (VIF). All VIF values in this study were less than 3.

We used binary logistic regression models further adjusted for testosterone and inflammatory markers to determine if additional adjustment for these variables attenuated the association between grip strength (or arm muscle quality) and incident type 2 diabetes. To formally quantify any mediating effect of testosterone or inflammatory markers, we undertook mediation analysis using the R mediation package. For testosterone, we also undertook a sensitivity analysis that excluded participants taking medications known to affect serum testosterone; major health problems including prostate cancer, orchidectomy, and primary testicular disease; and outlier values including total testosterone >40 nmol/L, luteinising hormone >12 U/L, follicular stimulating hormone >8 U/L, and sex hormone-binding globulin >100 nmol/L [26].

To quantify the proportion of incident type 2 diabetes cases that could be prevented if grip strength was to be increased, we calculated the population attributable fraction (PAF) using the R *attribrisk* package. There is no widely accepted cut-off value for low grip strength, and resistance training increases muscle strength by 25–30% after 3 months [27]. Hence we calculated PAFs based on five potential "intervention targets", as follows: 1) all participants increased grip strength by 25%, 2) all participants increased grip strength by 10%, 3) participants increased grip strength to our cohort's mean (47.4 kg), 4) participants increased grip strength to one standard deviation above the cohort mean (57.0 kg) and 5) participants increased grip strength to one standard deviation below the cohort mean (37.2 kg).

There were <2% missing data for all confounders, except physical activity, which had 6.4% missing. Data for missing confounders, but not the primary predictor or outcome variable, were imputed as either the median (numerical variables) or the modal category (categorical variables). Data were not imputed for serum total testosterone, IL-6, or TNF-α. Sensitivity analyses using only the complete, non-imputed, dataset (n = 1493) were also conducted.

2.6. *Role of the funding source*

The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

3. Results

Diabetes data were available in n = 2490 men (Fig. 1). Excluding n = 379 men with diabetes at baseline, n = 2111 were included in this study, of which follow-up data were available in n = 1680 (79.6%) men. Grip strength data was available in n = 1632 (77.3% of the n = 2111 included in this study). Incident type 2 diabetes occurred in 146 men (8.9%) over a median follow-up of 4.95 (IQR 4.35–5.00) years. Baseline DXA measurements were available in a sub-sample of 1181 men (414 from NWAHS and 767 from FAMAS). Table 1 shows the baseline characteristics of the cohort, overall as well as grouped by whether the men developed incident type 2 diabetes or not. Compared to those that did not develop type 2 diabetes ('never diabetes') during follow-up, the participants that developed incident type 2 diabetes were at baseline: older, had higher body mass index (BMI), waist circumference (WC) and serum IL-6, and had higher prevalence of impaired FPG, family history of diabetes, hypertension, vascular disease. They had lower annual income, education level, serum total testosterone and grip strength. In the DXA sub-study, compared to the participants that did not develop type 2 diabetes, participants that developed type 2 diabetes had higher total fat mass and fat mass index, and had lower lean mass percentage, and arm muscle quality. The Pearson correlation coefficient between grip strength and arm muscle quality was 0.68.

Table 2 shows the associations of grip strength, arm muscle quality, and lean mass measures with incident type 2 diabetes. In both unadjusted and adjusted logistic regression models, grip strength and arm muscle quality were significantly inversely associated with incident type 2 diabetes. In unadjusted models, lean mass percentage was inversely associated with incident type 2 diabetes, but these associations did not persist after adjustment. In models that were mutually adjusted for either grip strength or whole-body lean mass (Model 3), grip strength

Table 1 – Baseline characteristics of the cohort.

	Overall (n = 1632)	Never diabetes (n = 1486)	Incident type 2 diabetes (n = 146)	p-value
Age (years)	54.1 ± 11.4	53.7 ± 11.4	57.7 ± 10.9	p < **0.001**
Annual income				p < **0.001**
<$40,000	42.5 (694)	41.1 (610)	57.5 (84)	
$40,000–$79,999	38.8 (633)	39.5 (587)	31.5 (46)	
≥$80,000	18.7 (305)	19.4 (289)	11.0 (16)	
Education				p = **0.001**
High school or lower	31.4 (513)	30.1 (447)	45.2 (66)	
Trade	30.5 (498)	30.8 (457)	28.1 (41)	
Diploma/Certificate	24.5 (400)	24.9 (370)	20.5 (30)	
Bachelor or higher	13.5 (221)	14.3 (212)	6.2 (9)	
Physical activity ≥600 MET min/week	41.3 (674)	41.3 (614)	41.1 (60)	p = 0.958
FPG				p < **0.001**
<5.5 mmol/L	88.1 (1437)	90.8 (1349)	60.3 (88)	
5.5–6.1 mmol/L	9.7 (158)	7.9 (117)	28.1 (41)	
6.1–6.9 mmol/L	2.3 (37)	1.3 (20)	11.6 (17)	
Family history of diabetes	28.2 (461)	27.3 (406)	37.7 (55)	p = **0.008**
Current smoking	19.4 (316)	19.7 (293)	15.8 (23)	p = 0.247
BMI (kg/m^2)	28.0 ± 4.2	27.9 ± 4.1	29.9 ± 4.8	p < **0.001**
Waist circumference (cm)	99.5 ± 11.2	99.0 ± 11.0	104.8 ± 12.3	p < **0.001**
Hypertension	49.5 (808)	47.6 (707)	69.2 (101)	p < **0.001**
HDL <1.0 mmol/L	13.4 (218)	12.7 (189)	19.9 (29)	p = **0.015**
Triglycerides >1.7 mmol/L	34.6 (564)	33.4 (497)	45.9 (67)	p = **0.003**
Metabolic syndrome	29.5 (481)	27.0 (401)	54.8 (80)	p < **0.001**
Depression	8.2 (134)	7.9 (117)	11.6 (17)	p = 0.113
Cardiovascular disease	7.2 (117)	6.7 (100)	11.6 (17)	p = **0.028**
Total testosterone(nmol/L)	17.3 ± 5.7	17.6 ± 5.7	15.0 ± 5.7	p < **0.001**
Serum IL-6 (pg/mL)	2.0 ± 1.8	1.9 ± 1.7	2.3 ± 2.6	p = **0.025**
Serum TNF-α (pg/mL)	1.9 ± 2.7	1.9 ± 2.8	1.7 ± 0.9	p = 0.494
Grip strength (kg)	47.1 ± 9.9	47.4 ± 9.9	44.6 ± 9.4	p = **0.001**
DXA sub-study	(n = 1181)	(n = 1062)	(n = 119)	
Total lean mass (kg)	58.1 ± 7.7	58.0 ± 7.7	58.7 ± 7.5	p = 0.339
Lean mass percentage (%)	69.0 ± 6.6	69.3 ± 6.6	66.6 ± 5.5	p < **0.001**
ASM (kg)	26.3 ± 3.8	26.3 ± 3.8	26.2 ± 3.8	p = 0.825
ASMI (kg/m^2)	8.6 ± 1.1	8.6 ± 1.0	8.7 ± 1.0	p = 0.200
Total fat mass (kg)	23.5 ± 8.3	23.1 ± 8.2	27.0 ± 8.1	p < **0.001**
Fat mass index (kg/m^2)	8.0 ± 2.8	7.6 ± 2.7	9.0 ± 2.7	p < **0.001**
Arm muscle quality (kg/kg)	12.4 ± 2.2	12.5 ± 2.2	11.5 ± 2.0	p < **0.001**

Data are presented as % (n), or mean ± standard deviation. P-values (t-test or χ^2-test) compare the incident diabetes group with the never diabetes group. MET, metabolic equivalents; FPG, fasting plasma glucose; BMI, body mass index; HDL, high density lipoprotein; DXA, dual-energy X-ray absorptiometry; ASM, appendicular skeletal muscle mass; ASMI, appendicular skeletal muscle mass index.

muscle quality, and lean mass measures.

	n	Unadjusted	Model 2	Model 3
		OR (95%CI)	OR (95%CI)	OR (95%CI)
Grip strength	1632	**0.87 (0.80–0.95)**	**0.87 (0.78–0.97)**	**0.85 (0.74–0.98)**
Arm muscle quality	1172	**0.81 (0.74–0.89)**	**0.85 (0.75–0.95)**	**0.84 (0.75–0.94)**
Whole-body lean mass	1181	1.06 (0.94–1.21)	0.96 (0.82–1.13)	1.03 (0.87–1.24)
Lean mass percentage	1181	**0.76 (0.67–0.87)**	0.91 (0.77–1.12)	0.96 (0.80–1.20)
ASM	1181	0.97 (0.76–1.25)	0.80 (0.57–1.11)	0.93 (0.64–1.34)
ASMI	1181	1.14 (0.94–1.38)	0.97 (0.76–1.24)	1.08 (0.83–1.39)

Odds ratios (95% confidence intervals) for incident diabetes are per 5 kg unit increases in grip strength, lean mass and ASM, per 1 kg/kg unit increases in arm muscle quality, per 5% unit increases for lean mass percentage, and per 1 kg/m^2 increases in ASMI. Significant associations highlighted in bold. ASM, appendicular skeletal muscle mass; ASMI, appendicular skeletal muscle mass index. Model 2: adjusted for age, income, cohort, waist circumference, fasting plasma glucose, physical activity, hypertension, triglycerides and family history of diabetes. Model 3: Model 2 plus mutual adjustment for either whole-body lean mass or grip strength.

and arm muscle quality were significantly inversely associated with incident type 2 diabetes independent of whole-body lean mass, but the lean mass measures were not significantly associated with incident type 2 diabetes independent of grip strength.

Table 3 shows the proportion mediated by testosterone, IL-6, and TNF-α of the associations of grip strength and arm muscle quality with incident type 2 diabetes. Further adjustment for serum total testosterone slightly attenuated the odds ratios for grip strength. The association between grip strength and incident type 2 diabetes was reduced to borderline significance ($p = 0.051$). The association between arm muscle quality and incident type 2 diabetes remained significant after testosterone adjustment. Further adjustment for serum IL-6 and TNF-α also did not attenuate any of the associations. In mediation analysis, testosterone, IL-6, and TNF-α did not significantly mediate the association between grip strength or arm muscle quality and incident type 2 diabetes.

Table 4 shows the PAFs of incident type 2 diabetes due to low grip strength. The PAFs ranged from 3% (95% CI: 0.1–6%) to 29% (95% CI: 8–49%), depending on the intervention target chosen.

Stratified analyses are shown in Figs. 2 and 3. Fig. 2 shows associations of grip strength with incident type 2 diabetes, stratified by age, FPG, cohort, general adiposity, central adiposity, family history of diabetes, and serum total testosterone. The interaction effects were not significant for age, baseline FPG, cohort, family history of diabetes, or serum total testosterone. However, there were significant interaction effects for WC ($p = 0.002$) and BMI ($p = 0.001$). The inverse association of grip strength with type 2 diabetes was stronger in men with lower central adiposity (OR for WC <102 cm, 0.80 [95% CI: 0.68–0.94] vs. WC ≥102 cm, 0.88 [95% CI: 0.76–1.03]) and lower general adiposity (OR for BMI <30 kg/m^2, 0.79 [95% CI: 0.69–0.92] vs. BMI ≥30 kg/m^2, 0.94 [95% CI: 0.80–1.13]).

Fig. 3 shows associations of arm muscle quality with incident type 2 diabetes, stratified by age, FPG, cohort, general adiposity, central adiposity, family history of diabetes, serum total testosterone, and lean mass per arm. The interaction effects were not significant for age, baseline FPG, cohort, family history of diabetes, serum total testosterone, or lean mass per arm. However, there were significant interaction effects for WC ($p = 0.001$) and BMI ($p < 0.001$). The inverse association of arm muscle quality with type 2 diabetes was stronger in men with lower central adiposity (OR for WC <102 cm, 0.70 [95% CI: 0.59–0.83] vs. WC ≥102 cm, 0.93 [95% CI: 0.80–1.09]) and lower general adiposity (OR for BMI <30 kg/m^2, 0.71 [95% CI: 0.62–0.83] vs. BMI ≥30 kg/m^2, 1.03 [95% CI: 0.86–1.23]).

Sensitivity analyses are presented in the online Supplementary Data (Appendix A). Table A1 shows the analyses using peak grip strength (instead of mean dominant hand grip strength) and peak arm muscle quality (instead of overall arm muscle quality), as well as the analyses additionally adjusting for SF-36 bodily pain scale, current smoking, and systemic corticosteroid use at follow-up. Table A2 shows sensitivity analyses limited to the non-imputed complete dataset. The results of these sensitivity analyses were similar to the main analyses. The sensitivity analysis for testosterone mediation also yielded similar results to the main analysis (proportion mediated of grip strength 4% [95% CI: −70 to 71%], $p = 0.49$; proportion mediated of arm muscle quality 1% [95% CI: −30 to 24%], $p = 0.90$).

4. Discussion

In a large prospective community-dwelling sample of men, we found that grip strength and arm muscle quality were inversely associated with incident type 2 diabetes. Neither testosterone nor inflammation significantly mediated these associations. These associations, robust to multiple sensitivity analyses, were consistent across age, impaired fasting glucose, cohort, family history, and serum total testosterone strata. However, while these associations were especially strong in non-obese men, they were not significant in obese men. Our findings suggest that measurement of grip strength has a particularly important role in identifying non-obese men at risk of type 2 diabetes.

Only three previous studies have investigated the association between muscle strength and incident type 2 diabetes [9–11]. The largest study of those found inconclusive results: the adjusted association between grip strength and incident diabetes was not significant in the main analyses, but was significant in the sensitivity analyses [11]. However, that study relied on self-report to exclude baseline diabetes, with incident diabetes defined via self-report, identification on death case report forms, or identification on hospitalisation case report

DOCKET ALARM

Explore Litigation Insights

Docket Alarm provides insights to develop a more informed litigation strategy and the peace of mind of knowing you're on top of things.

Real-Time Litigation Alerts

Keep your litigation team up-to-date with **real-time alerts** and advanced team management tools built for the enterprise, all while greatly reducing PACER spend.

Our comprehensive service means we can handle Federal, State, and Administrative courts across the country.

Advanced Docket Research

With over 230 million records, Docket Alarm's cloud-native docket research platform finds what other services can't. Coverage includes Federal, State, plus PTAB, TTAB, ITC and NLRB decisions, all in one place.

Identify arguments that have been successful in the past with full text, pinpoint searching. Link to case law cited within any court document via Fastcase.

Analytics At Your Fingertips

Learn what happened the last time a particular judge, opposing counsel or company faced cases similar to yours.

Advanced out-of-the-box PTAB and TTAB analytics are always at your fingertips.

API

Docket Alarm offers a powerful API (application programming interface) to developers that want to integrate case filings into their apps.

LAW FIRMS
Build custom dashboards for your attorneys and clients with live data direct from the court.

Automate many repetitive legal tasks like conflict checks, document management, and marketing.

FINANCIAL INSTITUTIONS
Litigation and bankruptcy checks for companies and debtors.

E-DISCOVERY AND LEGAL VENDORS
Sync your system to PACER to automate legal marketing.