Published in final edited form as:

Am J Prev Med. 2015 December ; 49(6): 850–858. doi:10.1016/j.amepre.2015.05.025.

Grip Strength as a Marker of Hypertension and Diabetes in Healthy Weight Adults

Arch G. Mainous III, PhD[1,2], **Rebecca J. Tanner, MA**[1], **Stephen D. Anton, PhD**[3], and **Ara Jo, MS**[1]

[1]Department of Health Services Research, Management, and Policy, University of Florida, Gainesville, Florida

[2]Department of Family and Community Medicine, University of Florida, Gainesville, Florida

[3]Department of Aging and Geriatric Research, University of Florida, Gainesville, Florida

Abstract

Introduction—Muscle strength may play a role in cardiometabolic disease. We examined the relationship between hand grip strength and diabetes and hypertension in a sample of healthy weight adults.

Methods—In 2015, we analyzed the National Health and Nutrition Examination Survey 2011–2012 for adults aged ≥20 years with healthy BMIs (between 18.5 and <25 kg/m^2) and no history of cardiovascular disease (unweighted *n*=1,469; weighted *n*=61,672,082). Hand grip strength was assessed with a dynamometer. Diabetes was based on hemoglobin A1c level and reported diabetes diagnosis. Hypertension was based on measured blood pressure and reported hypertension diagnosis.

Results—Individuals with undiagnosed diabetes compared with individuals without diabetes had lower grip strength (51.9 vs 69.8, *p*=0.0001), as well as among individuals with diagnosed diabetes compared with individuals without diabetes (61.7 vs 69.8, *p*=0.008). Mean grip strength was lower among individuals with undiagnosed hypertension compared with individuals without hypertension (63.5 vs 71.5, *p*=0.008) as well as among individuals with diagnosed hypertension compared with those without hypertension (60.8 vs 71.5, *p*<0.0001). In adjusted analyses controlling for age, sex, race, smoking status, and first-degree relative with disease, mean grip strength was lower for undiagnosed diabetes (β= −10.02, *p*<0.0001) and diagnosed diabetes (β= −8.21, *p*=0.03) compared with individuals without diabetes. In adjusted analyses, grip strength was lower among individuals with undiagnosed hypertension (β= −6.6, *p*=0.004) and diagnosed hypertension (β= −4.27, *p*=0.04) compared with individuals without hypertension.

Address correspondence to: Arch G. Mainous III, PhD, Department of Health Services Research, Management and Policy, Health Sciences Center, University of Florida, PO Box 100195, Gainesville FL 32610. arch.mainous@ufl.edu.

No financial disclosures were reported by the authors of this paper.

Conclusions—Among healthy weight adults, combined grip strength is lower in individuals with diagnosed and undiagnosed diabetes and hypertension.

Introduction

According to the WHO,[1] approximately 347 million people have diabetes worldwide. Diabetes is a chronic and disabling disease that affects 8.3% of the U.S. population, nearly 26 million adults.[2] The prevalence of diagnosed diabetes has increased in recent years.[3] However, a substantial proportion of people with diabetes in the U.S. are undiagnosed.[4] Similar to diabetes, hypertension is a highly prevalent condition in the U.S. population, which has implications for cardiovascular morbidity and mortality, yet a substantial proportion are undiagnosed.[5] Approximately one in three U.S. adults has high blood pressure, with more than one third of these individuals unaware of their hypertension.[6]

There has been a staggering increase in the prevalence of obesity over the past 30 years in the U.S.[7] Furthermore, global studies have reported that increased BMI is one of the factors most strongly associated with diabetes and hypertension.[8,9] Extensive evidence indicates that obese individuals are at higher risk for both diabetes[10–20] and hypertension[21–30] than individuals of healthy weight. Consequently, healthcare providers now view obese individuals as an important high-risk target population. The focus on obese individuals, however, may lead to missed opportunities for investigation of undetected disease in healthy weight individuals. Individuals of a healthy weight may have “normal weight obesity,” a condition characterized by high body fat and lower lean muscle mass at a healthy BMI.[31] Recent reports indicate “normal weight obesity” is associated with increased risk of developing cardiovascular disease and diabetes.[31–33]

A potential marker for guiding detection of undiagnosed disease among patients at normal weight is grip strength. Grip strength is noninvasive and easy to measure. Grip strength is associated with both Type 2 diabetes and cardiovascular disease[34,35] and has been shown to be lower in older men with Type 2 diabetes compared with nondiabetic controls.[36] This study also showed that grip strength was negatively correlated with blood hemoglobin A1c (HbA1c) levels. Similarly, de Carvalho e Silva et al.[37] found that grip strength was incrementally lower in individuals with diabetes and osteoarthritis, respectively, compared with healthy controls. Despite these emerging associations, grip strength is rarely used as a marker of disease risk in primary care settings and has also been under-investigated as a cue to screen for diabetes or hypertension.

The purpose of this study was to examine the relationship between hand grip strength and undiagnosed diabetes and hypertension among adults of healthy weight in a nationally representative sample.

Methods

We analyzed the National Health and Nutrition Examination Survey (NHANES) for the years 2011–2012. NHANES is a large, nationally representative survey that samples the non-institutionalized population of the U.S. using a stratified multistage probability sample design. The National Center for Health Statistics (NCHS) uses a multilevel weighting system to account for survey design and nonresponse. This allows the study to provide nationally representative population estimates of the U.S. The current study focused on adults aged ≥20 years who reported never having been told they have cardiovascular disease (coronary heart disease, heart attack, or stroke) and who had a BMI between 18.5 and <25 kg/m^2.[38] A total of 1,469 adults with no history of cardiovascular disease had BMIs in the healthy range, representing 61,672,082 Americans.

Measures

Isometric handgrip strength was measured using a handgrip dynamometer (Takei digital grip strength dynamometer). Grip strength was measured three times using both hands in a standing position. We used the combined grip strength in kilograms, which is the sum of the largest reading from each hand. Individuals who only had one hand tested were not included in the sample, as NHANES does not calculate combined grip strength for these individuals.

Individuals who had an HbA1c level of ≥6.5% and who were never told they had diabetes (excluding gestational) were considered to have undiagnosed diabetes.[39] Individuals who reported they had been told by a healthcare provider that they had diabetes were considered to have diagnosed diabetes. Individuals who had never been told they had diabetes and had an HbA1c level of <6.5% were considered not to have diabetes.

Blood pressure was measured using a sphygmomanometer during a physical examination at a mobile examination center. Three measures of blood pressure were obtained, with a fourth attempt for those who had a previous measurement interrupted or incomplete. Mean systolic and mean diastolic blood pressure were calculated. Undiagnosed hypertension was defined as having a mean systolic blood pressure of ≥140 or a mean diastolic blood pressure of ≥90, for respondents who reported never having been told by a physician that they had high blood pressure or hypertension.[40] Diagnosed hypertension was defined as having ever been told by a physician that they had high blood pressure or hypertension. No hypertension was defined as no previous hypertension diagnosis, and having a systolic blood pressure of <140 and a diastolic blood pressure of <90. It should be noted that although a more updated classification of hypertension was released in 2014, we felt that it was important to use the classification available to clinicians at the time of the data collection in 2011–2012.[41]

Age was self-reported and divided into two categories: 20–64 and ≥65 years. Race/ethnicity was also self-reported and was categorized as non-Hispanic white, non-Hispanic black, Hispanic, and Asian/other. Sex was self-reported.

Because family history of a particular disease will increase suspicion of potential undetected disease, we thought that it was important to account for first-degree relatives with diabetes or heart attack/angina. NHANES asks respondents if they have a close biological (blood) relative (e.g., father, mother, sister, or brother) with a heart attack or angina before age 50 years or diabetes.[42] Having a first-degree relative with diabetes was defined as individuals who reported having a close biological relative ever having been diagnosed with diabetes. A first-degree relative diagnosed with heart attack or angina was defined as individuals who reported a close biological relative who had been diagnosed with a heart attack or angina by a health professional before age 50 years.

We controlled for smoking status, as smoking has been associated with the development of sarcopenia.[43] NHANES respondents are asked if they have smoked at least 100 cigarettes in their lives, and if they are currently smoking some day, every day, or not at all.[44] Smoking status was categorized as never smoker (having smoked <100 cigarettes in one's life), current smoker (having smoked ≥100 cigarettes in life, and reporting smoking some or every day), and former smoker (having smoked ≥100 cigarettes in life, and reporting no current smoking).

Statistical Analysis

To account for the complex sampling design of the NHANES, we used SUDAAN, version 11 for all data analyses. The analyses utilized the nesting capability in SUDAAN with variables provided in NHANES (that account for stratum-level differences and primary sampling unit differences) for all analyses, along with the appropriate weight identified by NCHS for the type of data being used in our analyses. Utilizing these weights and sampling design variables allowed us to account for the complexity of the sampling design in performing univariate analyses, two-sample *t*-tests, and linear regression models and make population estimates for the non-institutionalized adult population of the U.S. Analysis was conducted in 2015 of data collected in 2011–2012.

We examined demographic (age, race, sex) and health-related (smoking status, first degree relatives diagnosed with disease) characteristics of the respondents. We also examined the differences in mean combined grip strength by disease status for hypertension and diabetes using two sample *t*-tests. We used two-sample *t*-tests because SUDAAN does not support analysis of variance computations. In order to test differences in means, we computed *t*-tests examining each pair of disease statuses (no disease, diagnosed disease, and undiagnosed disease) for diabetes and for hypertension. We computed a linear regression model assessing the impact of diabetes status (no diabetes, diagnosed diabetes, and undiagnosed diabetes) on combined grip strength, controlling for age, sex, race, smoking, and first-degree relatives with diabetes. We computed a linear regression model assessing the impact of hypertension status (no hypertension, diagnosed hypertension, and undiagnosed hypertension) on combined grip strength, controlling for age, sex, race, smoking, and first-degree relatives with a history of heart attack/angina. We used listwise deletion of missing cases for the regression analyses.

In addition to the main analysis of all adults, we also examined the relationship between grip strength and disease status among adults aged ≥45 years, as the U.S. Preventive Services Task Force and American Diabetes Association recommend screening individuals aged ≥45 years.[39,45] Individuals who self-reported an age of ≥45 years were included in this analysis. This subgroup was examined using the same analytic methods described for the main analysis. Analysis of individuals aged <45 years was not possible because of limited sample size.

Results

Demographic characteristics for the full sample as well as hypertension and diabetes status are shown in Table 1. Table 1 also provides demographic for individuals with diagnosed/

undiagnosed hypertension and diagnosed/undiagnosed diabetes. The results presented in Table 2 showed that grip strength among individuals with undiagnosed diabetes or diagnosed diabetes was significantly lower than that of individuals without diabetes. Similarly, individuals with undiagnosed hypertension or diagnosed hypertension had significantly lower grip strength than individuals without diagnosed hypertension.

Tables 3 and 4 present the results of the multivariate analyses controlling for demographics and having a first-degree relative with disease. As with the unadjusted results, the adjusted results for both diabetes and hypertension showed a significant relationship of lower grip strength among individuals with undiagnosed disease or diagnosed disease compared with individuals with no disease.

Age 45 Years and Older

In the subgroup analysis focusing on individuals aged ≥45 years only, mean combined grip strength was lower among individuals with diagnosed diabetes (mean=58.3, SE=2.5) and undiagnosed diabetes (mean=49.4, SE=2.4) compared with individuals who had no diabetes (mean=63.4, SE=0.8). Two-sample *t*-tests indicated that means for diagnosed diabetes and undiagnosed diabetes were significantly different (p=0.002), as were means for undiagnosed diabetes and no diabetes (p<0.0001). The means for diagnosed diabetes versus no diabetes were not significantly different (p=0.06).

Mean grip strength was also significantly lower among individuals with diagnosed hypertension (mean=56.3, SE=1.5) and individuals with undiagnosed hypertension (mean=62.4, SE=3.1) compared with those without hypertension (mean=66.0, SE=1.6). Two-sample *t*-tests indicated that the means of diagnosed hypertension and no hypertension were significantly different (p=0.0005). The means of diagnosed hypertension and undiagnosed hypertension were not significantly different (p=0.1). The means of undiagnosed hypertension and no hypertension were also not significantly different (p=0.4).

Linear regression analyses indicated that diabetes diagnosis status was associated with combined grip strength (diagnosed diabetes, β= −7.83 p=0.02; undiagnosed diabetes, β= −9.24, p<0.0001; no diabetes, ref). Hypertension status was also associated with lower combined grip strength (diagnosed hypertension, β= −5.31, p=0.002; undiagnosed hypertension, β= −5.95, p=0.01; no hypertension, ref).

Discussion

We found that among adults with healthy BMIs without a history of cardiovascular disease, lower grip strength was associated with undiagnosed diabetes and undiagnosed hypertension as well as diagnosed disease. Grip strength was significantly lower among individuals with diagnosed or undiagnosed disease even after controlling for common demographics and family history of disease. To our knowledge, this is the first study to report these associations in a sample that included young, middle-aged, and older adults. This work suggests that grip strength may have potential utility as a quick, noninvasive marker to cue physicians and increase suspicion of undiagnosed cardiometabolic disease, specifically diabetes and hypertension.

There are a number of reasons why grip strength could be related to hypertension and diabetes. First, the difference in muscle strength between individuals with and without diabetes and hypertension could reflect lower muscle quality (muscle strength/muscle mass or muscle strength/body weight). A growing body of evidence strongly suggests that age-related changes in skeletal muscle composition, specifically the accumulation of lipids within skeletal muscle fibers, contributes to poor muscle quality and can lead to metabolic disorders such as insulin resistance. Poor muscle quality has been found to be directly related to changes in body composition and functional capacity during aging,[46,48] as well as poor cardiorespiratory function,[49] risk for disability,[50] and mortality[51,52] in older adults. In addition to changes in muscle quality, muscle mass has consistently been found to decline during aging (i.e., sarcopenia). This loss of muscle mass appears to be primarily due to the progressive loss of Type II muscle fibers and motor neurons[53] and is related to a corresponding reduction in skeletal muscle strength.[54,55] Moreover, sarcopenia is an emerging risk factor for metabolic disorders and hypertension.[56,58] Because skeletal muscle is a primary site for glucose uptake and deposition, loss of muscle mass and muscle quality can promote insulin resistance, and lead to the development of metabolic syndrome and diabetes.[59–61]

The specific biological mechanism linking low grip strength and sarcopenia with hypertension and Type 2 diabetes is not known, but sarcopenia and impaired metabolic states share common cellular and molecular characteristics. For example, increased fat infiltration into muscle bundles can lead to the accumulation of intermuscular adipose tissue,[62,63] which has been found to adversely affect mitochondrial function[64,65] and insulin signaling.[66] Converging evidence suggests that reductions in mitochondrial function have a pivotal role in the pathogenesis of muscle degradation[67,68] and cardiometabolic disorders.[69–71] Increased levels of oxidative stress and chronic molecular inflammation, which are commonly observed during aging, also appear to directly contribute to the development of sarcopenia and a host of cardiometabolic disease conditions, including hypertension and diabetes. The activation of specific inflammatory signaling pathways[72] can lead to cellular apoptosis (programmed cell death),[73] which been shown in several clinical conditions to negatively affect muscle mass and function.[74–76] Of note, regular exercise, which has consistently been shown to lower blood pressure and improve glycemic control,[77,78] may improve metabolic function and avert sarcopenia by improving mitochondrial function and reducing inflammation, oxidative damage, and consequent atrophy and apoptosis of skeletal muscle myocytes.

Second, the finding of lower grip strength among diabetics in particular could be caused by peripheral neuropathy[79] or diabetic hand syndrome. Diabetic hand syndrome is characterized by limited joint mobility or diabetic cheiroarthropathy, flexor tenosynovitis, and Dupuytern disease,[80] which can cause significant morbidity and may negatively impact grip strength.[81] Handgrip strength has been shown to be an indicator of autonomic damage in individuals with diabetes.[82]

Although the current findings are suggestive of the clinical utility of grip strength as a marker for undiagnosed disease, the benefit of grip strength as a prescreening strategy requires more controlled research. Disease status was associated with lower grip strength, even controlling for age, smoking, and first-degree relatives with the disease. However, having a better understanding of the positive and negative predictive value of grip strength as a screening tool for either hypertension or diabetes is necessary before making strong conclusions regarding its benefit. Because primary care physicians must deal with undifferentiated patient populations, they must therefore use different patient characteristics to guide screening and grip strength may help in their decision making. In the same way that BMI in the overweight or obese categories risk stratifies patients and cues physicians to the need for screening, grip strength could help primary care physicians with adult patients who present with healthy BMIs. Because nearly all patients have their blood pressure taken in an office visit, regardless of whether the patient is obese the additional value of a grip strength assessment for hypertension may be limited. For diabetes, grip strength could be a quick and easy option to increase suspicion among normal weight individuals and guide blood test screening. This method of prescreening requires only a dynamometer and requires little time to complete, but has the potential to identify patients who could benefit from screening.

An additional area of future research could be to provide normative data for grip strength related to risk of disease. Not only could that be age and sex normed but it could be potentially normed for different ethnic groups. The variation in genetic admixture would have to be carefully considered if one were to norm based on self-reported ethnicity. For example, WHO has considered different BMI standards for healthy weight for different ethnics groups and, in particular, Asians.[83] However, because of variation even among "Asians," they ultimately recommended that the current WHO BMI cut off points (18.5–24.99, 25.0–29.99, and >30 kg/m^2) should be retained as the international classification.[84]

Limitations

This study has a number of limitations. First, undiagnosed disease status was made on the basis of one physical exam and blood analysis, which may not be sufficient to determine diabetes or hypertension status. However, we used HbA1c level, a measure that accounts for longer-term glucose impairment to diagnose diabetes. Blood pressure measurements were also attempted up to four times, in order to obtain three readings, which were then averaged. Second, we only examined grip strength as a measure of muscle strength. Although our study did not allow for more-intense measures of muscle mass or muscle strength, we focused on grip strength because other underlying causes germane to diabetes and hypertension (e.g., neuropathy) may be manifested in this measure. Third, this study is cross-sectional in nature, and cannot speak to whether diseases such as diabetes and hypertension cause lower grip strength; it only shows that they are associated with it.

Conclusions

Our findings indicate lower grip strength is associated with undiagnosed and diagnosed diabetes and hypertension, and the presence of these two conditions is associated with lower grip strength among individuals with a healthy BMI. This finding may have clinical utility for providers in that it points toward a quick, easily administered prescreening tool that can indicate which healthy weight patients may benefit from more intensive screening.

Acknowledgments

Stephen D. Anton is funded in part by NIH/National Institute on Aging (NIA) grant P30AG028740 through the Claude D. Pepper Older American's Independence Center. The NIH/NIA had no role in study design; collection, analysis, and interpretation of the data; writing the report; or the decision to submit the report for publication.

References

1. WHO. [Accessed February 19, 2015] Diabetes. Fact sheet no. 312. WHO. 2013. www.who.int/diabetes/en/. 2013.
2. CDC. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the U.S. 2011 www.cdc.gov/diabetes.
3. Geiss LS, Wang J, Cheng YJ, et al. Prevalence and incidence trends for diagnosed diabetes among adults aged 20 to 79 years, United States, 1980–2012. JAMA. 2014; 312(12):1218–1226. http://dx.doi.org/10.1001/jama.2014.11494. [PubMed: 25247518]
4. Selvin E, Parrinello CM, Sacks DB, Coresh J. Trends in prevalence and control of diabetes in the United States, 1988–1994 and 1999–2010. Ann Intern Med. 2014; 160(8):517–525. http://dx.doi.org/10.7326/M13-2411. [PubMed: 24733192]
5. Wall HK, Hannan JA, Wright JS. Patients with undiagnosed hypertension: hiding in plain sight. JAMA. 2014; 312(19):1973–1974. http://dx.doi.org/10.1001/jama.2014.15388. [PubMed: 25399269]
6. Nwankwo, T.; Yoon, SS.; Burt, V.; Gu, Q. [Accessed February 19, 2015] Hypertension among adults in the United States: National Health and Nutrition Examination Survey, 2011–2012. www.cdc.gov/nchs/data/databriefs/db133.pdf.
7. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999–2008. JAMA. 2010; 303(3):235–241. http://dx.doi.org/10.1001/jama.2009.2014. [PubMed: 20071471]
8. Liu L, Yin X, Morrissey S. Global variability in diabetes mellitus and its association with body weight and primary healthcare support in 49 low- and middle-income developing countries. Diabet Med. 2012; 29(8):995–1002. http://dx.doi.org/10.1111/j.1464-5491.2011.03549.x. [PubMed: 22150805]
9. Courcoulas AP, Christian NJ, Belle SH, et al. Weight change and health outcomes at 3 years after bariatric surgery among individuals with severe obesity. JAMA. 2013; 310(22):2416–2425. http://dx.doi.org/10.1001/jama.2013.280928. [PubMed: 24189773]
10. Westlund K, Nicolaysen R. Ten-year mortality and morbidity related to serum cholesterol. A follow-up of 3,751 men aged 40–49. Scand J Clin Lab Invest Suppl. 1972; 127:1–24. [PubMed: 4640618]
11. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. J Chronic Dis. 1979; 32(8):563–576. http://dx.doi.org/10.1016/0021-9681(79)90119-X. [PubMed: 468958]
12. Medalie JH, Papier C, Herman JB, et al. Diabetes mellitus among 120,000 adult men. I. 5-year incidence and associated variables. Isr J Med Sci. 1974; 10:681–697. [PubMed: 4851353]
13. Colditz GA, Willett WC, Stampfer MJ, et al. Weight as a risk factor for clinical diabetes in women. Am J Epidemiol. 1990; 132:501–513. [PubMed: 2389754]
14. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of U.S. adults. Am J Epidemiol. 1997; 146(3):214–222. http://dx.doi.org/10.1093/oxfordjournals.aje.a009256. [PubMed: 9247005]
15. Despres JP, Nadeau A, Tremblay A, et al. Role of deep abdominal fat in the association between regional adipose tissue distribution and glucose tolerance in obese women. Diabetes. 1989; 38(3):304–309. http://dx.doi.org/10.2337/diab.38.3.304. [PubMed: 2645187]
16. Haffner SM, Mitchell BD, Hazuda HP, Stern MP. Greater influence of central distribution of adipose tissue on incidence of non-insulin-dependent diabetes in women than men. Am J Clin Nutr. 1991; 53(5):1312–1317. [PubMed: 2021139]

17. Sparrow D, Borkan GA, Gerzof SG, Wisniewski C, Silbert CK. Relationship of fat distribution to glucose tolerance. Results of computed tomography in male participants of the Normative Aging Study. Diabetes. 1986; 35(4):411–415. http://dx.doi.org/10.2337/diab.35.4.411. [PubMed: 3956878]

18. Lundgren H, Bengtsson C, Blohme G, Lapidus L, Sjostrom L. Adiposity and adipose tissue distribution in relation to incidence of diabetes in women: results from a prospective population study in Gothenburg, Sweden. Int J Obes. 1989; 13(4):413–423. [PubMed: 2793297]

19. Ohlson LO, Larsson B, Svardsudd K, et al. The influence of body fat distribution on the incidence of diabetes mellitus. 13.5 years of follow-up of the participants in the study of men born in 1913. Diabetes. 1985; 34(10):1055–1058. http://dx.doi.org/10.2337/diab.34.10.1055. [PubMed: 4043554]

20. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willet WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care. 1994; 17(9):961–969. http://dx.doi.org/10.2337/diacare.17.9.961. [PubMed: 7988316]

21. Brown CD, Higgins M, Donato KA, et al. Body mass index and the prevalence of hypertension and dyslipidemia. Obes Res. 2000; 8(9):605–619. http://dx.doi.org/10.1038/oby.2000.79. [PubMed: 11225709]

22. Association of Life Insurance Medical Directors of America. Blood Pressure Study. Chicago: The Society; 1980. Society of Actuaries.

23. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure. Findings in hypertension screening of 1 million Americans. JAMA. 1978; 240(15):1607–1610. http://dx.doi.org/10.1001/jama.1978.03290150053024. [PubMed: 691146]

24. Criqui MH, Mebane I, Wallace RB, Heiss G, Holdbrook MJ. Multivariate correlates of adult blood pressures in nine North American populations: The Lipid Research Clinics Prevalence Study. Prev Med. 1982; 11(4):391–402. http://dx.doi.org/10.1016/0091-7435(82)90043-3. [PubMed: 7122431]

25. Dyer AR, Elliott P. The INTERSALT study: relations of body mass index to blood pressure. INTERSALT Co-operative Research Group. J Hum Hypertens. 1989; 3(5):299–308. [PubMed: 2810326]

26. Ballantyne D, Devine BL, Fife R. Interrelation of age, obesity, cigarette smoking, and blood pressure in hypertensive patients. BMJ. 1978; 1:880–881. http://dx.doi.org/10.1136/bmj.1.6117.880. [PubMed: 638505]

27. Brennan PJ, Simpson JM, Blacket RB, McGilchrist CA. The effects of body weight on serum cholesterol, serum triglycerides, serum urate, and systolic blood pressure. Aust N Z J Med. 1980; 10(1):15–20. http://dx.doi.org/10.1111/j.1445-5994.1980.tb03412.x. [PubMed: 6929670]

28. Havlik RJ, Hubert HB, Fabsitz RR, Feinleib M. Weight and hypertension. Ann Intern Med. 1983; 98(5):855–859. http://dx.doi.org/10.7326/0003-4819-98-5-855. [PubMed: 6847025]

29. Loggie JM, Horan MJ, Hohn AR, Gruskin AB, Dunbar JB, Havlik RJ. Juvenile hypertension: highlights of a workshop. J Pediatric. 1984; 104(5):657–663. http://dx.doi.org/10.1016/S0022-3476(84)80939-7.

30. MacMahon SW, Blacket RB, Macdonald GJ, Hall W. Obesity, alcohol consumption and blood pressure in Australian men and women. The National Heart Foundation of Australia Risk Factor Prevalence Study. J Hypertens. 1984; 2(1):85–91. http://dx.doi.org/10.1097/00004872-198402000-00015. [PubMed: 6530540]

31. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. Eur Heart J. 2010; 31(6):737–746. http://dx.doi.org/10.1093/eurheartj/ehp487. [PubMed: 19933515]

32. Jean N, Somers VK, Sochor O, Medina-Inojosa J, Llano EM, Lopez-Jimenez F. Normal-weight obesity: implications for cardiovascular health. Curr Atheroscler Rep. 2014; 16:464.http://dx.doi.org/10.1007/s11883-014-0464-7 [PubMed: 25342492]

33. Marques-Vidal P, Pécoud A, Hayoz D, et al. Normal weight obesity: relationship with lipids, glycaemic status, liver enzymes and inflammation. Nutr Metab Cardiovasc Dis. 2010; 20(9):669–675. http://dx.doi.org/10.1016/j.numecd.2009.06.001. [PubMed: 19748248]

34. Park SW, Goodpaster BH, Strotmeyer ES, et al. Decreased muscle strength and quality in older adults with type 2 diabetes: the health, aging, and body composition study. Diabetes. 2006; 55(6): 1813–1818. http://dx.doi.org/10.2337/db05-1183. [PubMed: 16731847]

35. Sénéchal M, McGavock JM, Church TS, et al. Cut points of muscle strength associated with metabolic syndrome in men. Med Sci Sports Exerc. 2014; 46(8):1475–1481. http://dx.doi.org/10.1249/MSS.0000000000000266. [PubMed: 25029165]

36. Leenders M, Verdijk LB, van der Hoeven L, et al. Patients with type 2 diabetes show a greater decline in muscle mass, muscle strength, and functional capacity with aging. J Am Med Dir Assoc. 2013; 14(8):585–592. http://dx.doi.org/10.1016/j.jamda.2013.02.006. [PubMed: 23537893]

37. de Carvalho e Silva F, Jakimiu FO, Skare TL. Diabetic hands: a study on strength and function. Diabetes Metab Syndr. 2014; 8(3):162–165. http://dx.doi.org/10.1016/j.dsx.2014.04.020. [PubMed: 25220919]

38. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report. Bethesda MD: National Heart, Lung, and Blood Institute; 1998. NHLBI Obesity Education Initiative Expert Panel on the Identification, Evaluation, and Treatment of Obesity in Adults.

39. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 2012; 35(S1):S64–S71. http://dx.doi.org/10.2337/dc12-s064. [PubMed: 22187472]

40. Rosendorff C, Lackland DT, Allison M, et al. Treatment of hypertension in patients with coronary artery disease: A scientific statement from the American Heart Association, American College of Cardiology, and American Society of Hypertension. J Am Coll Cardiol. 2015 In press, corrected proof. http://dx.doi.org/10.1016/j.jacc.2015.02.038.

41. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014; 311(5):507–520. http://dx.doi.org/10.1001/jama.2013.284427. [PubMed: 24352797]

42. [Accessed April 3, 2015] National Health and Nutrition Examination Survey 2011–2012 Data Documentation, Codebook, and Frequencies: Medical Conditions. National Center for Health Statistics. 2013 Sep. wwwn.cdc.gov/nchs/nhanes/2011-2012/MCQ_G.htm#MCQ300a.

43. Steffl M, Bohannon RW, Petr M, Kohlikova E, Holmerova I. Relation between cigarette smoking and sarcopenia - meta analysis. Physiol Res. 2014 Dec 22. [Epub ahead of print].

44. National Health and Nutrition Examination Survey 2011–2012 Data Documentation, Codebook, and Frequencies: Smoking - Cigarette Use. [Accessed April 3, 2015] National Center for Health Statistics. 2013 Sep. http://wwwn.cdc.gov/nchs/nhanes/2011-2012/SMQ_G.htm.

45. Draft Recommendation Statement: Abnormal Glucose and Type 2 Diabetes Mellitus in Adults: Screening. [Accessed March 10, 2015] U.S. Preventive Services Task Force. 2014 Oct. www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementDraft/screening-for-abnormal-glucose-and-type-2-diabetes-mellitus.

46. Goodpaster BH, Carlson CL, Visser M, et al. Attenuation of skeletal muscle and strength in the elderly: The Health ABC Study. J Appl Physiol. 2001; 90(6):2157–2165. [PubMed: 11356778]

47. Kelley DE, Slasky BS, Janosky J. Skeletal muscle density: effects of obesity and non-insulin-dependent diabetes mellitus. Am J Clin Nutr. 1991; 54(3):509–515. [PubMed: 1877507]

48. Peterson MD, Liu D, Gordish-Dressman H, et al. Adiposity attenuates muscle quality and the adaptive response to resistance exercise in non-obese, healthy adults. Int J Obes (Lond). 2011; 35:1095–1103. http://dx.doi.org/10.1038/ijo.2010.257. [PubMed: 21139562]

49. Barbat-Artigas S, Dupontgand S, Fex A, Karelis AD, Aubertin-Leheudre M. Relationship between dynapenia and cardio-repiratory functions in healthy postmenopausal women: novel clinical criteria. Menopause. 2010; 18(4):400–405. http://dx.doi.org/10.1097/gme.0b013e3181f7a596. [PubMed: 21107297]

50. Al Snih S, Markides KS, Ottenbacher KJ, Raji MA. Hand grip strength and incident ADL disability in elderly Mexican Americans over a seven-year period. Aging Clin Exp Res. 2004; 16(6):481–486. http://dx.doi.org/10.1007/BF03327406. [PubMed: 15739601]

51. Al Snih S, Markides KS, Ray L, Ostir GV, Goodwin JS. Handgrip Strength and Mortality in Older Mexican Americans. J Am Geriatr Soc. 2002; 50(7):1250–1256. http://dx.doi.org/10.1046/j.1532-5415.2002.50312.x. [PubMed: 12133020]
52. Newman AB, Kupelian V, Visser M, et al. Strength, but not muscle mass, is associated with mortality in the health, aging and body composition study cohort. J Gerontol A Biol Sci Med Sci. 2006; 61(1):72–77. http://dx.doi.org/10.1093/gerona/61.1.72. [PubMed: 16456196]
53. Larsson L, Sjodin B, Karlsson J. Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22--65 years. Acta Physiol Scand. 1978; 103(1):31–39. http://dx.doi.org/10.1111/j.1748-1716.1978.tb06187.x. [PubMed: 208350]
54. Abe T, Thiebaud S, Loenneke JP, Ogawa M, Mitsukawa N. Association between forearm muscle thickness and age-related loss of skeletal muscle mass, hand grip and knee extension strength and walking performance in old men and women: pilot study. Ultasound Med Biol. 2014; 40(9):2069–2075. http://dx.doi.org/10.1016/j.ultrasmedbio.2014.05.003.
55. Hughes VA, Frontera WR, Wood M, et al. Longitudinal muscle strength changes in older adults: Influences of Muscle Mass, physical activity, and health. J Gerontol A Biol Sci Med Sci. 2001; 56(5):B209–B217. http://dx.doi.org/10.1093/gerona/56.5.B209. [PubMed: 11320101]
56. Hulens M, Vansant G, Lysens R, Claessens AL, Muls E, Brumagne S. Study of differences in peripheral muscle strength of lean versus obese women: an allometric approach. Int J Obes Relat Metab Disord. 2001; 25(5):676–681. http://dx.doi.org/10.1038/sj.ijo.0801560. [PubMed: 11360150]
57. Moon SS. Low skeletal muscle mass is associated with insulin resistance, diabetes, and metabolic syndrome in the Korean population: the Korea National Health and Nutrition Examination Survey (KNHANES) 2009–2010. Endocr J. 2014; 61(1):61–70. http://dx.doi.org/10.1507/endocrj.EJ13-0244. [PubMed: 24088600]
58. Han K, Park YM, Kwon HS, et al. Sarcopenia as a determinant of blood pressure in older Koreans: findings from the Korea National Health and Nutrition Examination Surveys (KNHANES) 2008–2010. PLoS One. 2014; 29(9(1)):e86902. http://dx.doi.org/10.1371/journal.pone.0086902. [PubMed: 24489804]
59. Klip A, Paquet MR. Glucose transport and glucose transporters in muscle and their metabolic regulation. Diabetes Care. 1990; 13(3):228–243. http://dx.doi.org/10.2337/diacare.13.3.228. [PubMed: 2407478]
60. Sayer AA, Dennison EM, Syddall HE, Gilbody HJ, Phillips DI, Cooper C. Type 2 diabetes, muscle strength, and impaired physical function: the tip of the iceberg? Diabetes Care. 2005; 28(10): 2541–2542. http://dx.doi.org/10.2337/diacare.28.10.2541. [PubMed: 16186295]
61. Cetinus E, Buyukbese MA, Uzel M, Ekerbicer H, Karaoguz A. Hand grip strength in patients with type 2 diabetes mellitus. Diabetes Res Clin Pract. 2005; 70(3):278–286. http://dx.doi.org/10.1016/j.diabres.2005.03.028. [PubMed: 15878215]
62. Janssen I, Ross R. Linking age-related changes in skeletal muscle mass and composition with metabolism and disease. J Nutr Health Aging. 2005; 9(6):408–419. [PubMed: 16395513]
63. Manini TM, Buford TW, Lott DJ, et al. Effect of dietary restriction and exercise on lower extremity tissue compartments in obese, older women: a pilot study. J Gerontol A Biol Sci Med Sci. 2014; 69(1):101–108. http://dx.doi.org/10.1093/gerona/gls337. [PubMed: 23682155]
64. Bayeva M, Gheorghiade M, Ardehali H. Mitochondria as a therapeutic target in heart failure. J Am Coll Cardiol. 2013; 61(6):599–610. http://dx.doi.org/10.1016/jjacc.2012.08.1021. [PubMed: 23219298]
65. Verdejo HE, del CA, Troncoso R, et al. Mitochondria, myocardial remodeling, and cardiovascular disease. Curr Hypertens Rep. 2012; 14(6):532–539. http://dx.doi.org/10.1007/s11906-012-0305-4. [PubMed: 22972531]
66. Shulman GI. Cellular mechanisms of insulin resistance. J Clin Invest. 2000; 106(2):171–176. http://dx.doi.org/10.1172/JCI10583. [PubMed: 10903330]
67. Short KR, Bigelow ML, Kahl J, et al. Decline in skeletal muscle mitochondrial function with aging in humans. Proc Natl Acad Sci U S A. 2005; 102(15):5618–5623. http://dx.doi.org/10.1073/pnas.0501559102. [PubMed: 15800038]

68. Rooyackers OE, Adey DB, Ades PA, Nair KS. Effect of age on in vivo rates of mitochondrial protein synthesis in human skeletal muscle. Proc Natl Acad Sci U S A. 1996; 93(26):15364–15369. http://dx.doi.org/10.1073/pnas.93.26.15364. [PubMed: 8986817]
69. Schrauwen-Hinderling VB, Kooi ME, Schrauwen P. Mitochondrial function and diabetes; consequences for skeletal and cardiac muscle metabolism. Antioxid Redox Signal. 2015 Mar 26. Epub ahead of print. http://dx.doi.org/10.1089/ars.2015.6291.
70. Montgomery MK, Turner N. Mitochondrial dysfunction and insulin resistance: an update. Endocr Connect. 2015; 4:R1–R15. http://dx.doi.org/10.1530/EC-14-0092. [PubMed: 25385852]
71. Crescenzo R, Bianco F, Mazzoli A, Giacco A, Liverini G, Iossa S. Mitochondrial efficiency and insulin resistance. Front Physiol. 2015; 5:512. http://dx.doi.org/10.3389/fphys.2014.00512. [PubMed: 25601841]
72. Phillips T, Leeuwenburgh C. Muscle fiber specific apoptosis and TNF-alpha signaling in sarcopenia are attenuated by life-long calorie restriction. FASEB J. 2005; 19(6):668–670. [PubMed: 15665035]
73. Chung HY, Cesari M, Anton S, et al. Molecular inflammation: underpinnings of aging and age-related diseases. Ageing Res Rev. 2009; 8(1):18–30. http://dx.doi.org/10.1016/j.arr.2008.07.002. [PubMed: 18692159]
74. Leeuwenburgh C. Role of apoptosis in sarcopenia. J Gerontol A Biol Sci Med Sci. 2003; 58(11): 999–1001. http://dx.doi.org/10.1093/gerona/58.11.M999. [PubMed: 14630880]
75. Marzetti E, Calvani R, Bernabei R, Leeuwenburgh C. Apoptosis in skeletal myocytes: a potential target for interventions against sarcopenia and physical frailty - a mini-review. Gerontology. 2012; 58(2):99–106. http://dx.doi.org/10.1159/000330064. [PubMed: 21952604]
76. Marzetti E, Privitera G, Simili V, et al. Multiple pathways to the same end: mechanisms of myonuclear apoptosis in sarcopenia of aging. Scientific World Journal. 2010; 10:340–349. http://dx.doi.org/10.1100/tsw.2010.27. [PubMed: 20191247]
77. Brook RD, Appel LJ, Rubenfire M, et al. Beyond medications and diet: alternative approaches to lowering blood pressure : A Scientific Statement From the American Heart Association. Hypertension. 2013; 61(6):1360–1383. http://dx.doi.org/10.1161/HYP.0b013e318293645f. [PubMed: 23608661]
78. Lackland DT, Voeks JH. Metabolic syndrome and hypertension: regular exercise as part of lifestyle management. Curr Hypertens Rep. 2014; 16:492.http://dx.doi.org/10.1007/s11906-014-0492-2 [PubMed: 25190022]
79. Koopman RJ, Mainous AG 3rd, Liszka HA, et al. Evidence of nephropathy and peripheral neuropathy in U.S. adults with undiagnosed diabetes. Ann Fam Med. 2006; 4(5):427–432. http://dx.doi.org/10.1370/afm.577. [PubMed: 17003143]
80. Al-Matubsi HY, Hamdan F, Alhanbali OA, Oriquat GA, Salim M. Diabetic hand syndromes as a clinical and diagnostic tool for diabetes mellitus patients. Diabetes Res Clin Pract. 2011; 94(2): 225–229. http://dx.doi.org/10.1016/j.diabres.2011.07.012. [PubMed: 21831469]
81. Savaş S, Köroğlu BK, Koyuncuoğlu HR, Uzar E, Celik H, Tamer NM. The effects of the diabetes related soft tissue hand lesions and the reduced hand strength on functional disability of hand in type 2 diabetic patients. Diabetes Res Clin Pract. 2007; 77(1):77–83. http://dx.doi.org/10.1016/j.diabres.2006.10.020. [PubMed: 17141353]
82. Petrofsky J, Prowse M, Remigio W, et al. The use of an isometric handgrip test to show autonomic damage in people with diabetes. Diabetes Technol Ther. 2009; 11(6):361–368. http://dx.doi.org/10.1089/dia.2008.0094. [PubMed: 19459764]
83. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004; 363(9403):157–163. http://dx.doi.org/10.1016/S0140-6736(03)15268-3. [PubMed: 14726171]
84. WHO. [Accessed April 6, 2015] BMI Classification. http://apps.who.int/bmi/index.jsp?introPage=intro_3.html&.

Table 1

Sample Characteristics

	Full sample(%)	Diagnosed/ undiagnosed diabetes(%)	Diagnosed/ undiagnosed hypertension(%)
Unweighted sample size	1,469	97	363
Weighted sample size	61,672,082	2,741,535	12,748,322
Sex			
Male	44.8	54.7	44.8
Female	55.2	45.3	55.2
Age			
20–64	85.7	61.7	61.5
65 and older	14.3	38.3	38.5
Race			
Non-Hispanic white	67.9	54.1	71.4
Non-Hispanic black	8.3	13.8	11.3
Hispanic	10.7	10.4	6.4
Asian/Other	13.2	21.7	10.9
First degree relative diagnosed with diabetes	26.8	55.4	29.2
First degree relative diagnosed with heart attack/angina	10.4	10.7	12.3
Smoking status			
Current smoker	24.2	29.5	24.6
Former smoker	19.2	33.1	29.7
Never smoker	56.5	37.4	45.7
Diabetes			
Diagnosed diabetes	3.9	--	10.8
Undiagnosed diabetes	0.8	--	2.0
No diabetes	95.3	--	87.2
Hypertension			
Diagnosed hypertension	14.6	41.9	--
Undiagnosed hypertension	6.7	13.1	--
No hypertension	78.7	45.0	--

Table 2

Comparison of Mean Combined Grip Strength by Disease Status Using t-tests for Statistical Significance

	Mean	SE	*p*-value
Diabetes			
Diagnosed Diabetes	61.7	2.86	
Undiagnosed Diabetes	51.9	2.91	
No Diabetes	69.8	0.69	
Hypertension			
Diagnosed Hypertension	60.8	1.61	
Undiagnosed Hypertension	63.5	2.37	
No Hypertension	71.5	0.84	
Diabetes			
Diagnosed diabetes vs undiagnosed diabetes			**0.002**
Diagnosed diabetes vs no diabetes			**0.008**
No diabetes vs undiagnosed diabetes			**<0.0001**
Hypertension			
Diagnosed hypertension vs undiagnosed hypertension			0.42
Diagnosed hypertension vs no hypertension			**<0.0001**
No hypertension vs undiagnosed hypertension			**0.008**

Note: Boldface indicates statistical significance ($p<0.05$).

Table 3

Linear Regression of Diabetes Status and Combined Mean Grip Strength

	β Coefficient	*p*-value
Intercept	89.02	**<0.0001**
Sex		
Male	.	.
Female	−30.38	**<0.0001**
Age		
18–64	.	.
65+	−15.71	**<0.0001**
Race		
Non-Hispanic white	.	.
Non-Hispanic black	2.23	0.15
Hispanic	−2.9	**0.02**
Asian	−4.46	**0.001**
First degree relative diagnosed with diabetes		
No first degree relative diagnosed with diabetes	.	.
First degree relative diagnosed with diabetes	−0.04	0.96
Smoking status		
Current smoker	1.14	0.37
Former smoker	1.68	0.35
Never smoker	.	.
Diabetes diagnosis		
Diagnosed diabetes	−8.56	**0.03**
Undiagnosed diabetes	−9.76	**<0.0001**
No diabetes	.	.

Note: Boldface indicates statistical significance (p<0.05).

Table 4

Linear Regression of Hypertension Status and Combined Mean Grip Strength

	β Coefficient	*p*-value
Intercept	89.64	**<0.0001**
Sex		
Male	.	.
Female	−29.95	**<0.0001**
Age		
18–64	.	.
65+	−14.01	
Race		
Non-Hispanic white	.	.
Non-Hispanic black	2.00	0.21
Hispanic	−3.70	**0.006**
Asian	−5.38	**<0.0001**
First degree relative with heart attack		
No first degree relative diagnosed with heart attack	.	.
First degree relative diagnosed with heart attack	−0.24	0.90
Smoking status		
Current smoker	.80	0.53
Former smoker	1.50	0.37
Never smoker	.	.
Hypertension diagnosis		
Diagnosed hypertension	−4.93	**0.03**
Undiagnosed hypertension	−6.82	**0.003**
No hypertension	.	.

Note: Boldface indicates statistical significance (p<0.05).