Burden and Rates of Treatment and Control of Cardiovascular Disease Risk Factors in Obesity: The Framingham Heart Study

Esther A. Molenaar MSc, Shih-Jen Hwang PhD, Ramachandran S. Vasan MD, Diederick E. Grobbee MD PhD, James B. Meigs MD MPH, Ralph B. D’Agostino Sr PhD, Daniel Levy MD, Caroline S. Fox MD MPH

From the Julius Center for Health Sciences and Primary Care (EAM, DEG), University Medical Center Utrecht, Utrecht, The Netherlands; Municipal Health Service Utrecht (EAM), Utrecht, The Netherlands; National Heart, Lung and Blood Institute’s Framingham Heart Study (CSF, SJH, RSV, DL), Framingham, Massachusetts; Department of Endocrinology, Diabetes, and Hypertension, the Brigham and Women’s Hospital (CSF), Harvard Medical School, Boston, Massachusetts; Department of Cardiology & Preventive Medicine (RSV), Boston University School of Medicine, Boston, MA; Department of Mathematics and Statistics (RBD), Boston University, Boston, Massachusetts; Division of General Internal Medicine (JBM), Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts.

Corresponding Author:
Caroline S. Fox MD MPH
NHLBI’s Framingham Heart Study
73 Mt. Wayte Ave Suite #2
Framingham MA 01702
foxca@nhlbi.nih.gov

Running Title: Cardiovascular Disease Risk Factors in Obesity

Received for publication 19 December 2007 and accepted in revised form 25 March 2008.

Additional information for this article can be found in an online appendix at http://care.diabetesjournals.org.
Objective Obesity is associated with an increased risk for cardiovascular disease (CVD). We sought to determine rates of treatment and control of CVD risk factors among normal weight, overweight and obese individuals in a community-based cohort.

Research Design and Methods Participants free of CVD (n=6801; mean age 49 years; 54% women) from the Framingham Offspring and Third Generation cohorts who attended the seventh Offspring examination (1998-2001) or first Third Generation (2002-2005) examination were studied.

Results Obese participants with hypertension were more likely to receive antihypertensive treatment (62.3%) than normal weight (58.7%) or overweight individuals (59.0%; p=0.002), but no differences in hypertension control across BMI subgroups among participants with hypertension were observed (36.7% [normal weight], 37.3% [overweight], and 39.4% [obese]; p=0.48). Rates of lipid-lowering treatment were higher among obese participants with elevated LDL cholesterol (39.5%) as compared to normal weight (34.2%) or overweight participants (36.4%; p=0.02), but control rates among those with elevated LDL cholesterol did not differ across BMI categories (26.7% [normal weight], 26.0% [overweight], and 29.2% [obese]; p=0.11). There were no differences in diabetes treatment among participants with diabetes across BMI groups (69.2% [normal weight], 50.0% [overweight], 55.0% [obese]; p=0.54), but obese participants with diabetes were less likely to have fasting blood glucose <126 mg/dL (15.7%) as compared to normal weight (30.4%) or overweight participants (20.7%; p=0.02).

Conclusions These findings emphasize the suboptimal rates of treatment and control of CVD risk factors among overweight and obese individuals.
Obesity affects more than one-third of the adult population in the United States. Excess weight is associated with multiple cardiovascular disease (CVD) risk factors, including hypertension, dyslipidemia, diabetes, and the metabolic syndrome.

Although the incidence and mortality of CVD have declined markedly during the past decades, some studies suggest that the increasing prevalence of obesity and diabetes may have slowed this rate of decline (1). In addition, recent data suggests that the prevalence of chronic kidney disease is increasing, in part due to the increasing rates of diabetes (2). Unfortunately, the efficacy of current therapies for obesity including lifestyle and pharmacologic interventions, is limited (3). While bariatric surgery is an effective method of weight loss among severely obese individuals, eligibility criteria limit its use to only the most significantly affected patients.

Given the current limitations of effective weight loss therapies, minimizing the risk of complications of obesity and diabetes due to CVD risk factors is essential. Few studies have focused on a comprehensive approach to CVD risk factor burden, treatment, and control among obese individuals. Therefore, the aim of this study is to examine the burden of CVD risk factors as well as rates of treatment and control among normal weight, overweight and obese individuals in an unselected population-based cohort. As abdominal fat accumulation is strongly associated with metabolic and CVD risk factors, and as recent guidelines have emphasized the importance of measuring waist circumference (WC) as part of clinical cardiovascular risk assessment, we also studied individuals with and without abdominal obesity.

**RESEARCH DESIGN AND METHODS**

**Study sample:** The Framingham Heart Study is a population-based prospective cohort study that commenced in 1948, consisting of 5209 men and women in the original cohort. In 1971, 5124 men and women were enrolled into the Framingham Heart Study Offspring cohort, including the children and spouses of the children of the original cohort. Starting in 2002, 4095 participants who had at least one parent in the Offspring cohort, were enrolled into the Framingham Heart Third Generation Study. Approximately every 4 years Offspring participants underwent examinations; the design and methodology of the Offspring and Third Generation cohort have been previously described (4,5).

For the current study, the study sample consisted of Offspring and Third Generation participants who attended the seventh (1998-2001) and first (2002-2005) examination cycle, respectively.

Of 7634 participants (3539 Offspring, 4095 Third Generation participants) eligible participants, we excluded those with prevalent CVD (n=463), body mass index (BMI) <18.5 kg/m² or incomplete BMI data (n=196), type 1 diabetes (n=15), missing waist circumference (WC) values (n=105) and missing covariate data (n=54), resulting in 6801 eligible participants.

The study protocol was approved by the institutional review boards of the Boston University Medical Center. All subjects provided written informed consent. The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

**Measurements and definitions:** Height and weight were directly measured using a standardized protocol. BMI was calculated by dividing weight in kilograms by the square of the height in meters. General obesity was defined according to the World Health Organization/ National Institutes of Health
Cardiovascular Disease Risk Factors in Obesity

classification scheme. WC was measured at the level of the umbilicus. Abdominal obesity was defined as a WC ≥88 cm (women) and 102 cm (men).

CVD risk factor assessment: Assessment of CVD risk factors (including fasting blood testing) was based on measurements obtained during a single examination. Hypertension was defined as systolic blood pressure of at least 140 mm Hg or a diastolic blood pressure of at least 90 mm Hg (based on the average of 2 readings), or current use of antihypertensive medication for hypertension. Serum cholesterol levels were measured in a fasting state. Participants with elevated low-density lipoprotein cholesterol (LDL-C) levels according to their CVD risk level as classified by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATPIII) algorithm, or those receiving lipid-lowering agents were defined as having elevated LDL-C levels. Type 2 diabetes was defined as a fasting blood glucose level of at least 126 mg/dL (7.0 mmol/L) or current use of insulin and/or hypoglycemic treatment for diabetes. High-performance liquid chromatography was used to measure hemoglobin A1c levels with an assay coefficient of variation of less than 2.5%.

Treatment and control of CVD risk factors: To determine rates of treatment, the number of participants receiving medication for each individual condition was divided by the number of all participants with the condition. Rates of control were determined by dividing the number of participants classified as controlled by the total number of individuals with the condition. Control of hypertension was defined as either a blood pressure of less than 140/90 mm Hg or 130/80 mm Hg for participants with diabetes (6). Control of LDL-C levels was determined using participant’s specified treatment goal according to the NCEP ATPIII algorithm. Diabetes control rates were assessed by dividing the number of individuals with fasting blood glucose less than 126 mg/dL by the number of all participants with diabetes. A hemoglobin A1c level of less than 7.0% was additionally used to calculate rates of glycemic control in the Offspring cohort only.

Statistical analysis: Prevalence and rates of treatment and control of CVD risk factors were compared among individuals in the three BMI categories. For each risk factor, the age-sex adjusted proportion of participants with the condition that were treated and controlled was calculated; 95% confidence intervals (95% CI) were abstracted from the logistic regression models. In all analyses, the global p-values were obtained from models using the generalized estimation equation (GEE) to account for familial correlation, except for analyses where sample sizes were too small to permit the GEE. In this case, ANOVA p-values were calculated. Low high-density lipoprotein cholesterol (HDL-C) levels were defined as less than 50 mg/dL (1.29 mmol/L) in women and less than 40 mg/dL (1.03 mmol/L) in men or current use of lipid-lowering agents. Rates of dual control of hypertension and elevated LDL-C levels and triple control of hypertension, elevated LDL-C and fasting blood glucose were calculated and compared across the three BMI categories.

The following secondary analyses were performed. Participants were stratified by abdominal obesity. In addition, general obesity was further categorized in Stage I (BMI 30-<35 kg/m²) and Stage II or higher (BMI ≥35 kg/m²) obesity; the latter is further in the text simply indicated as Stage II obesity. Participants were also stratified by age (younger than 50 years or 50 years and older) and sex.

Statistical analyses were performed using SAS statistical software, version 8. A 2-tailed p<0.05 and p<0.01 were considered statistically significant for primary and secondary analyses, respectively.
RESULTS

Overall, 36.1% of the study participants (mean age 49 ± 13 years, 54% women) were normal weight, 38.2% were overweight, and 25.7% were obese; 47.7% had abdominal obesity. The characteristics of the study participants are displayed in Table 1.

Hypertension: The prevalence of hypertension increased significantly with increasing BMI category (p<0.001, Table 2). Among those with hypertension, obese participants were more likely to be treated (62.3%) as compared to normal weight (58.7%) or overweight participants (59.0%; p=0.002). However, control rates were uniformly poor and did not differ by BMI category (36.7% [normal weight], 37.3% [overweight], and 39.4% [obese]; p=0.48).

Elevated LDL Cholesterol: Elevated LDL-C increased with increasing BMI categories (p<0.001, Table 2). Obese participants with elevated LDL-C were more likely to be treated with lipid lowering agents (39.5%) as compared to normal weight (34.2%) or overweight participants (36.4%; p=0.02). Less than one third of the participants were controlled and rates of control did not differ by BMI category (p=0.11).

Type 2 diabetes

Despite higher prevalence rates of diabetes with increasing BMI (p<0.001, Table 2), there were no differences in hypoglycemic treatment (69.2% [normal weight], 50.0% [overweight], 55.0% [obese]; p=0.54), or differences in prevalence of optimal hemoglobin A1c levels across BMI categories in the Offspring cohort (50.0% [normal weight], 58.8% [overweight], 47.7% [obese]; p=0.26) among participants with diabetes. Obese participants were less likely to have fasting blood glucose <126 mg/dL (15.7%) than normal weight (30.4%) or overweight (20.7%; p=0.02) participants.

Combinations of risk factors

The number of CVD risk factors among BMI categories is displayed in Online Appendix Figure 1; only 6.0% of obese participants had no CVD risk factors. Dual control of hypertension and elevated LDL-C was uniformly low and did not differ by BMI category (19.1% [95% CI; 12.6%-27.0%] [normal weight], 12.1% [95% CI; 9.0%-15.8%] [overweight], and 16.0% [95% CI; 12.7%-19.8%] [obese]; p=0.94). Rates of triple control of hypertension, LDL-C and diabetes were low and showed no differences by BMI category (p=0.15): none of the normal weight participants with hypertension, elevated LDL-C and diabetes (n=17) achieved optimal triple control (0%; 95% CI 0%-0%), only 3 out of 52 overweight participants (5.9%; 95% CI 1.2%-16.2%), and only 2 out of 131 obese individuals achieved optimal triple control (1.6%; 95% CI 0.2%-5.5%).

Secondary analyses: When results were stratified by abdominal obesity, findings were not materially different (Online Appendix Table 1).

In analyses stratified by age, among older participants, obese individuals with hypertension were more likely to receive antihypertensive treatment (74.1%) as compared to those with normal weight (67.4%) and overweight (67.5%; p=0.006, Online Appendix Table 2), whereas hypertension treatment rates among participants younger than 50 years were uniformly lower and similar across BMI categories (p=0.26). Age-stratified analyses of hypoglycemic treatment demonstrated that in participants younger than 50 years of age, obese individuals with diabetes were less likely to receive treatment (39.3%) as compared to overweight individuals with diabetes (50.0%; p=0.006, Online Appendix Table 2).

In sex-specific analyses, obese men were more likely to receive anti-hypertensive treatment (56.9%) as compared to normal weight men (50.9%) or overweight men (53.5%; p=0.006, Online Appendix Table 3), whereas treatment rates among women were
uniformly the same across BMI categories (p=0.15). A similar pattern of sex differences was observed for lipid-lowering treatment and control of elevated LDL-C (Online Appendix Table 3). Sex-specific analyses of elevated glucose control demonstrated that among women, obese individuals with diabetes were less likely to have fasting blood glucose <126 mg/dL (12.8%) as compared to normal weight (30.8%) or overweight individuals (32.3%; p<0.001, Online Appendix Table 3), whereas the rates among men were uniformly the same across BMI categories.

When the obesity category was further broken down into Stage I vs. Stage II obesity, no difference in treatment or control of hypertension (Figure 1A), elevated LDL-C (Figure 1B) and diabetes (Figure 1C) was observed despite higher prevalence of hypertension and diabetes among participants with Stage II obesity.

**CONCLUSIONS**

**Principal findings:** Despite the higher burden of CVD risk factors among participants with obesity from the Framingham Heart Study, rates of treatment and control of CVD risk factors are suboptimal among overweight and obese individuals. Among participants with obesity, only one in four with hypertension achieved recommended blood pressure levels, less than one third with elevated LDL-C had optimal control of elevated LDL-C and only one in six participants with diabetes achieved fasting blood glucose <126 mg/dL. Dual and triple control of CVD risk factors were uniformly poor across all BMI categories.

**Hypertension:** High blood pressure is associated with an increased risk of mortality and morbidity from stroke, coronary heart disease and congestive heart failure (7) and is more frequent in obese individuals as compared to lean individuals (8). Obese participants were more likely to receive antihypertensive treatment but were not more likely to be controlled. Overall, potential reasons for poor blood pressure may include unrecognized hypertension, poor adherence to medication regimen (9) and failure to initiate or intensify therapy when indicated (10). In addition, the pathophysiology of obesity-related hypertension may differ from hypertension among non-obese individuals due to the presence of excess adipose tissue. Potential mechanisms that link adipose tissue to hypertension include alterations in the renin-angiotensin system, activation of the sympathetic nervous system, insulin resistance, sodium and volume retention and renal dysfunction (11). These mechanisms may have important implications for the effectiveness of antihypertensive therapy in obese individuals. Clinical trial data has shown that beta-blockers alone (12), or in combination with doxazosin (13), more effectively lower blood pressure in obese than in lean hypertensive individuals. Clinical trials have consistently shown that angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are associated with reductions in the risk of new-onset type 2 diabetes (14), and there is growing evidence that drugs blocking the renin-angiotensin system may be beneficial in the management of hypertension in obese individuals (15). Current treatment guidelines do not provide specific recommendations for obese individuals regarding blood pressure targets and specific treatment. This may be due to lack of randomized clinical trials that have focused specifically on this question.

**Elevated LDL Cholesterol:** Obese participants with elevated LDL-C were more likely to receive lipid-lowering therapy but rates of control of LDL-C among affected individuals did not differ across BMI categories. Among all participants with elevated LDL-C, less than one third were well-controlled.

High levels of LDL-C are an important modifiable risk factor in the
Cardiovascular Disease Risk Factors in Obesity

development of CVD. Many primary and secondary prevention trials have demonstrated the efficacy and safety of statins in reducing CVD risk. Therefore, it is surprising that LDL control was so poor among obese participants. There are several potential reasons for this. The current National Cholesterol Education Program guidelines do not specifically target obesity as a high-risk condition warranting lower LDL targets for lipid lowering. In addition, few clinical trials have studied the efficacy of statins in BMI subgroups on intermediate markers of CVD demonstrating increased benefit among obese individuals as compared to non-obese individuals or consistent effects across subgroups. Lastly, clinical trials studying the efficacy of statins on cardiovascular outcomes in BMI subgroups are lacking.

Type 2 diabetes

The prevalence of diabetes has increased substantially over the last several decades (16), likely due to increases in obesity, and the prevalence of obesity among individuals with diabetes increased by 50% between 1970 and 1989 (17). Rates of CVD associated with type 2 diabetes are high (18), and recent increases in chronic kidney disease may be due in part to increases in obesity and diabetes (2).

Despite this, we observed similarly low rates of treatment and control of hemoglobin A1c levels across BMI subgroups. There may be several potential reasons for the observed poor glycemic control. First, diabetes may be unrecognized and therefore untreated. Secondly, clinical trial data demonstrating CVD event reduction in the setting of optimal glycemic control is lacking. However, improved blood glucose control reduces the risk of chronic kidney disease and diabetic retinopathy (19). Third, patients may be noncompliant with regard to treatment regimens, including diet and exercise recommendations. Undesirable side effects due to antidiabetic agents, in particular weight gain in the setting of insulin treatment may limit treatment adherence as well. Lastly, clinical trials suggest that diabetes is more difficult to control among obese individuals (20).

We have shown that material differences in treatment of diabetes do not exist across BMI categories and that obese individuals are less likely to achieve optimal fasting blood glucose levels. The majority of diabetes occurs in obese individuals. Our results highlight the vast numbers of untreated and uncontrolled diabetes in this subgroup.

Control of combinations of risk factors: Rates of dual and triple control of CVD risk factors were uniformly poor across BMI categories in our study sample. Clustering of metabolic abnormalities contributes cumulatively to CVD risk and complicates treatment (21). This data emphasizes the importance of a treatment regimen aimed at multiple risk factors.

Clinical implications and future research: The suboptimal rates of diabetes treatment and control of CVD risk factors in obese participants in the current study are of particular concern given the increasing rates of overweight and obesity among US adults. Without substantial improvements in CVD risk factor treatment and control rates among obese individuals, the medical and financial burden of CVD events may grow substantially in the next several decades. There is a paucity of clinical trial data specifically testing interventions in obese subgroups to determine whether more intensive risk factor management or obesity-specific treatment and control guidelines would result in decreased CVD outcomes. Additionally, there is a need for more effective pharmacotherapy for obesity.

Strengths and limitations: Strengths of our study include the examination of a large, population-based sample of women and men with a broad age spectrum and standardized
assessment of anthropometric measures and CVD risk factors and treatment. Several limitations should be acknowledged. We used guidelines for treatment that were not necessarily in place at the time of data collection. However, the aim of current study was to characterize the burden of CVD risk factors using the most contemporary data available. The data collection period spanned from 1998 to 2005 and rates of treatment or control of CVD risk factors may have changed during this period. Participants of the Offspring cohort were followed for several years and may have benefited with respect to risk factor reduction as the findings of each examination are reviewed and letters are sent to the physician. However, our rates of treatment and control of CVD risk factors are similar to data from national surveys (22), suggesting that the rates of treatment and control mirror national data. Further, the data in the present study from the Third Generation cohort represent their first examination, minimizing this concern. The Framingham Heart Study Offspring and Third Generation cohort participants are primarily white, therefore the generalizability of our findings to other racial groups may be limited. Lastly, we did not examine the reasons for low rates of treatment and control.

**Conclusion:** Rates of treatment and control of CVD risk factors are suboptimal among overweight and obese individuals in the Framingham Heart Study.

**ACKNOWLEDGEMENTS**

**Funding Sources** The Framingham Heart Study is supported by the National Heart, Lung and Blood Institute (N01-HC-25195). MSc. Molenaar is supported by the Netherlands Organization for Scientific Research (NWO) and the Netherlands Heart Foundation (NHF). Dr. Vasan is supported in part by 2K24 HL 04334 (NHLBI). Dr. Meigs is supported in part by K24 DK080140 (NIDDK).

**Disclosures**

None.
Reference List


Cardiovascular Disease Risk Factors in Obesity

Table 1: Characteristics of study participants within different BMI categories. Data shown as mean ± standard deviation for continuous variables, and n (percent) for dichotomous variables.

<table>
<thead>
<tr>
<th></th>
<th>BMI 18.5-&lt;25</th>
<th>BMI 25-&lt;30</th>
<th>BMI ≥30</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>46 ± 14</td>
<td>50 ± 13</td>
<td>51 ± 13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female (%)</td>
<td>1727 (70.3)</td>
<td>1093 (42.1)</td>
<td>866 (49.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic Blood Pressure, mm Hg</td>
<td>115 ± 16</td>
<td>122 ± 16</td>
<td>127 ± 16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic Blood Pressure, mm Hg</td>
<td>71 ± 9</td>
<td>76 ± 9</td>
<td>79 ± 9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting Blood Glucose, mg/dL</td>
<td>91 ± 12</td>
<td>98 ± 17</td>
<td>107 ± 29</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>187 ± 34</td>
<td>199 ± 37</td>
<td>198 ± 35</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LDL cholesterol, mg/dL</td>
<td>107 ± 31</td>
<td>122 ± 32</td>
<td>120 ± 31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL</td>
<td>61 ± 17</td>
<td>52 ± 15</td>
<td>48 ± 14</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglycerides, median (25/75 percentiles), mg/dL</td>
<td>78 [58, 109]</td>
<td>109 [76, 161]</td>
<td>131 [93, 184]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>22.5 ± 1.6</td>
<td>27.3 ± 1.4</td>
<td>34.6 ± 4.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist Circumference, cm</td>
<td>82.6 ± 7.6</td>
<td>96.6 ± 7.2</td>
<td>113.7 ± 12.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Current smoker (%)</td>
<td>407 (16.6)</td>
<td>386 (14.9)</td>
<td>247 (14.1)</td>
<td>0.14</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; SD, standard deviation; LDL, low-density lipoprotein; HDL, high-density lipoprotein; WC, waist circumference

SI conversion factors: To convert glucose to mmol/L, multiply mg/dL values by 0.0555; total, LDL and HDL cholesterol to mmol/L, multiply mg/dL values by 0.0259; triglycerides to mmol/L, multiply mg/dL values by 0.0113

* Global GEE age- and sex-adjusted p-value, except for age which is sex-adjusted and sex, which is age-adjusted
### Table 2: Age-and sex-adjusted rates of hypertension, elevated levels of low lipoprotein cholesterol, type 2 diabetes, treatment and control among BMI categories

<table>
<thead>
<tr>
<th></th>
<th>% of Participants (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BMI 18.5-&lt;25</td>
</tr>
<tr>
<td></td>
<td>N=2458</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td>Prevalence</td>
<td>11.5 (10.2-12.9)</td>
</tr>
<tr>
<td>Treatment</td>
<td>58.7 (49.8-67.1)</td>
</tr>
<tr>
<td>Control</td>
<td>36.7 (30.3-43.5)</td>
</tr>
<tr>
<td>Elevated LDL-C levels</td>
<td></td>
</tr>
<tr>
<td>Prevalence</td>
<td>12.7 (11.3-14.2)</td>
</tr>
<tr>
<td>Treatment</td>
<td>34.2 (28.2-40.6)</td>
</tr>
<tr>
<td>Control</td>
<td>26.7 (21.5-32.4)</td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>Prevalence</td>
<td>1.4 (0.9-1.9)</td>
</tr>
<tr>
<td>Treatment</td>
<td>69.2 (38.6-90.9)</td>
</tr>
<tr>
<td>Hb A1c &lt;7.0% †</td>
<td>50.0 (18.7-81.3)</td>
</tr>
<tr>
<td>FPG &lt;126 mg/dL</td>
<td>30.4 (13.2-52.9)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; BMI, body mass index; LDL-C, low-density lipoprotein cholesterol; Hb A1c, Hemoglobin A1c level; FPG, fasting plasma glucose

SI conversion factor: To convert fasting plasma glucose to mmol/L, multiply mg/dL values by 0.0555

* Global GEE p-value

† Data only available for Offspring cohort
Figure 1 – Prevalence and rates of treatment and control of hypertension (Panel A), elevated levels of low-density lipoprotein cholesterol (Panel B), and type 2 diabetes (Panel C) among normal weight, overweight, obese Stage I, and obese Stage II participants. GEE p-values represent obesity Stage I vs. obesity Stage II adjusted for age and sex.

* p<0.001