Adiponectin and Coronary Heart Disease
A Prospective Study and Meta-Analysis

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Background—There is uncertainty about the association between circulating concentrations of adiponectin and coronary heart disease (CHD) risk. We report new data from a prospective study in the context of a meta-analysis of previously published prospective studies.

Methods and Results—We measured baseline adiponectin levels in stored serum samples of 589 men with fatal CHD or nonfatal myocardial infarction and in 1231 controls nested within a prospective study of 5661 men (aged 40 to 59 years) recruited during 1978–1980, as well as in paired samples obtained 4 years apart from 221 of these participants. Baseline adiponectin concentrations correlated (P<0.0001) positively with HDL cholesterol (r=0.33) and inversely with C-reactive protein (r=−0.11) and BMI (r=−0.21), and the year-to-year consistency of adiponectin values was comparable to those of blood pressure and total cholesterol levels. No significant difference between median adiponectin levels at baseline was observed between cases and controls (10.2 versus 10.8 μg/mL; P=0.5), despite the fact that body mass index, HDL, and C-reactive protein were all significant predictors of events in this cohort. The odds ratio for CHD was 0.89 (95% CI, 0.67 to 1.18) in a comparison of men in the top third of adiponectin concentrations compared with those in the bottom third, similar to a meta-analysis (including the present study) of 7 prospective studies involving a total of 1318 CHD cases (odds ratio, 0.84 [95% CI, 0.70 to 1.01]).

Conclusions—In contrast to the strong associations previously reported between adiponectin levels and risk of type 2 diabetes, any association with CHD risk is comparatively moderate and requires further investigation. (Circulation. 2006;114:623-629.)

Key Words: cardiovascular diseases ■ metabolism ■ risk factors

There is considerable interest in the relationship of the adipocyte-derived protein adiponectin to both type 2 diabetes and coronary heart disease (CHD). Adiponectin is a 244-amino acid protein that, despite being derived solely from adipose tissue, is paradoxically reduced in obesity. Circulating adiponectin levels, ranging from 0.5 to 30 μg/mL in humans, are reportedly ~1000-fold higher than circulating levels of other hormones such as insulin and leptin. Prospective epidemiological studies have suggested that elevated adiponectin concentrations are associated with greater insulin sensitivity and reduced risk of type 2 diabetes, apparently independent of obesity and other potential confounders. Thus, the development of interventions that raise adiponectin levels has been proposed as a target to improve insulin sensitivity and glucose tolerance and possibly to prevent CHD. Adiponectin has been proposed to protect against cardiovascular disease by other proposed mechanisms. Adiponectin is strongly antiinflammatory, acting through the nuclear factor-κB pathway, downregulates adhesion molecule expression on endothelial cells, and enhances lipid clearance in numerous animal models. In accord with such observations, exogenous adiponectin administration protects against development of atherosclerosis in apolipoprotein E-deficient mice.

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In humans, however, the evidence thus far has been somewhat conflicting. In the Health Professionals Study, a doubling of baseline adiponectin level was reported to be associated with a statistically significant 20% reduction in myocardial infarction (MI) risk in multivariate analyses, after adjustment for age, smoking, hypertension history, lipids, glycemic control, and C-reactive protein (CRP). The results from this study, based on 266 incident MI cases, have suggested that adiponectin is a major mechanistic link (“common soil”) between diabetes and increased CHD risk. However, subsequent investigations in similarly sized studies have not reported significant associations between adiponec-
tin levels and CHD risk. To help clarify the evidence, we report new data from the prospective British Regional Heart Study (BRHS), which involves almost 600 incident CHD deaths and events, more than twice as many as in the previous largest study. It also includes information on repeat measurements made 4 years apart in 221 study participants to quantify and correct for within-individual variations in the measurement of adiponectin levels. To place our results in context, we also report a meta-analysis of all available prospective studies of adiponectin and CHD risk, involving a total of >1300 CHD cases from 7 prospective studies based in general Western populations.

Methods

Participants

In 1978–1980, 7735 men were randomly selected from general practices in each of 24 British towns and invited to take part in the BRHS (response rate, 78%). Nurses administered questionnaires, made physical measurements, recorded an ECG, and, in 5661 men in 18 of the towns, collected nonfasting venous blood samples from which serum was stored at −20°C for subsequent analysis. All men were followed up for all-cause mortality and cardiovascular morbidity, and follow-up has been achieved for 99% of the cohort. A nested case-control study of major CHD events was established within the cohort. Eligible cases were 279 men who died from CHD and 364 men who had nonfatal MI before 1996. Fatal cases were ascertained through National Health Service Central Registers on the hospital records confirming the diagnosis in accordance with World Health Organization criteria. Cases were “frequency matched” with 1278 controls on town of residence and age in 5-year bands of general practitioner record reviews. Controls were randomly selected from among men surviving to the end of the study period free from incident CHD. Because of limited sample availability, adiponectin measurements were available for 589 cases and 1231 controls.

Laboratory Methods

Laboratory measurements were made blind to participants’ disease status, with samples from patients and controls randomly distributed among assay plates. Adiponectin levels were assessed with the use of a sensitive enzyme-linked immunosorbent assay (ELISA) (R&D Systems, Oxford, UK); the intra-assay and the interassay coefficients of variability were each <7.5%. This ELISA adiponectin method correlated well (r=0.91, P<0.0001) with a radioimmunoassay adiponectin assay (Linco, St Charles, Mo) in an analysis of 80 samples from men and women in our laboratory. Because of fluctuations of adiponectin levels over time, case-control comparisons of measurements made at baseline can underestimate the magnitude of any associations with CHD risk. Hence, adiponectin measurements were made in pairs of samples collected at an interval of 4 years in 221 healthy individuals to help quantify the within-individual variation and correct for regression dilution bias. Methods for the measurement of other risk factors in the BRHS and their association with CHD risk have been reported previously.

Statistical Analysis

Association between parameters was examined with the use of Spearman correlations. We prespecified that case-control comparisons of measurements made at baseline can underestimate the magnitude of any associations with CHD risk. Hence, adiponectin measurements were made in pairs of samples collected at an interval of 4 years in 221 healthy individuals to help quantify the within-individual variation and correct for regression dilution bias. Methods for the measurement of other risk factors in the BRHS and their association with CHD risk have been reported previously.

| TABLE 1. Baseline Characteristics of Controls and Men Who Developed Major CHD (Fatal/Nonfatal) During the Follow-Up |
|---|---|---|
| Characteristics | Cases (n=589) | Controls (n=1231) | P |
| Questionnaire | | | |
| Age, y | 52.6±5.2 | 52.5±5.3 | Frequency matched |
| Current smoker, n (%) | 303 (51.5) | 517 (42.1) | <0.001 |
| Evidence of coronary disease, n (%) | 189 (32.2) | 216 (17.6) | <0.001 |
| Physician-diagnosed diabetes, n (%) | 16 (2.7) | 19 (1.5) | 0.096 |
| >2 Drinks alcohol per day, n (%) | 121 (20.5) | 278 (22.7) | 0.298 |
| Nonactive, n (%) | 271 (46.5) | 489 (40.3) | 0.017 |
| Nonmanual occupation, n (%) | 406 (69.1) | 768 (62.4) | 0.006 |
| Physical measurements | | | |
| BMI, kg/m² | 25.9±3.4 | 25.4±3.3 | 0.001 |
| Systolic blood pressure, mm Hg | 151.7±21.8 | 146.7±20.9 | <0.001 |
| Diastolic blood pressure, mm Hg | 85.8±13.8 | 82.8±13.2 | <0.001 |
| Blood sample | | | |
| Total cholesterol, mmol/L | 6.6±1.06 | 6.2±0.99 | <0.001 |
| HDL cholesterol, mmol/L | 1.09±0.27 | 1.15±0.27 | <0.001 |
| Triglycerides, median (IQR), mmol/L | 1.88 (1.38–2.80) | 1.67 (1.14–2.41) | <0.001 |
| Log triglycerides | 0.67±0.53 | 0.52±0.54 | <0.001 |
| CRP, median (IQR), mg/L | 2.4 (1.3–5.2) | 1.4 (0.5–3.3) | <0.001 |
| Log CRP | 0.90±1.09 | 0.34±1.22 | <0.001 |
| Adiponectin, median (IQR), µg/mL | 10.22 (7.23–13.94) | 10.75 (7.30–14.85) | |
| Log adiponectin | 2.31±0.51 | 2.33±0.54 | 0.500 |

Values are provided as mean±SD or number (%) if not stated otherwise. IQR indicates interquartile range.
in the middle third, and further adjustment for BMI, total cholesterol, HDL cholesterol, and log triglycerides made little difference to the odds ratios (Table 3). Smoking status was fitted as a 4-level variable: never smoked, ex-smoker, ≤20 cigarettes per day, and >20 cigarettes per day; physical activity was fitted as a 3-level variable; alcohol intake was fitted as an 8-level variable; social class was divided into 3 categories: manual, nonmanual, and armed forces; and age, body mass index (BMI), and blood-based covariates were fitted as continuous variables, unless stated otherwise.

Correction for regression dilution was made by dividing the regression coefficients (and their standard errors) that related risk to blood-based covariates were fitted as continuous variables, unless stated otherwise.

TABLE 2. Comparisons of Levels of Risk Factors and Other Characteristics in Controls by Thirds of Adiponectin

<table>
<thead>
<tr>
<th>Variables, Classic Risk Factors</th>
<th>Bottom (n=411)</th>
<th>Middle (n=410)</th>
<th>Top (n=410)</th>
<th>P* (for Trend)</th>
<th>P† (for Trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y‡</td>
<td>51.8±0.3</td>
<td>52.3±0.3</td>
<td>53.4±0.3</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>173 (42.3)</td>
<td>162 (39.6)</td>
<td>182 (44.4)</td>
<td>0.903</td>
<td>0.519</td>
</tr>
<tr>
<td>&gt;2 Drinks alcohol per day, n (%)</td>
<td>91 (22.3)</td>
<td>91 (22.1)</td>
<td>96 (23.7)</td>
<td>0.575</td>
<td>0.382</td>
</tr>
<tr>
<td>Nonactive, n (%)</td>
<td>150 (37.0)</td>
<td>158 (39.0)</td>
<td>181 (44.8)</td>
<td>0.050</td>
<td>0.031</td>
</tr>
<tr>
<td>Evidence of CHD at entry, n (%)</td>
<td>75 (18.3)</td>
<td>71 (17.4)</td>
<td>70 (17.1)</td>
<td>0.403</td>
<td>0.607</td>
</tr>
<tr>
<td>History of diabetes at baseline, n (%)</td>
<td>8 (1.9)</td>
<td>4 (1.0)</td>
<td>7 (1.7)</td>
<td>0.852</td>
<td>0.989</td>
</tr>
<tr>
<td>Nonmanual occupation, n (%)</td>
<td>251 (61.2)</td>
<td>238 (58.3)</td>
<td>279 (67.9)</td>
<td>0.065</td>
<td>0.040</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>6.23±0.05</td>
<td>6.24±0.05</td>
<td>6.13±0.05</td>
<td>0.151</td>
<td>0.780</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.05±0.01</td>
<td>1.15±0.01</td>
<td>1.26±0.01</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Log triglycerides, mmol/L</td>
<td>0.68±0.03</td>
<td>0.51±0.03</td>
<td>0.37±0.03</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.0±0.2</td>
<td>25.7±0.2</td>
<td>24.5±0.2</td>
<td>&lt;0.001</td>
<td>NA</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>148.2±1.0</td>
<td>146.2±1.0</td>
<td>145.7±1.0</td>
<td>0.082</td>
<td>0.940</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>83.8±0.6</td>
<td>82.5±0.6</td>
<td>82.1±0.6</td>
<td>0.054</td>
<td>0.668</td>
</tr>
<tr>
<td>Log CRP, mg/L</td>
<td>0.55±0.06</td>
<td>0.29±0.07</td>
<td>0.21±0.07</td>
<td>&lt;0.001</td>
<td>0.038</td>
</tr>
</tbody>
</table>

Values are provided as mean±SE and number (%) adjusted for age and town. NA indicates not applicable.
*Model adjusted for age and town.
†Model adjusted for age, town, and BMI.
‡Adjusted for town only.

TABLE 3. Odds Ratios of CHD in Men in the Top Third of Adiponectin Distribution of Controls Relative to Those With Values in the Bottom Third

<table>
<thead>
<tr>
<th>Thirds</th>
<th>All men</th>
<th>Excluding men with preexisting CHD</th>
<th>Excluding men with preexisting diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases (n=411)</td>
<td>Controls (n=410)</td>
<td>Model A</td>
</tr>
<tr>
<td>I (lowest)</td>
<td>207</td>
<td>411</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>219</td>
<td>410</td>
<td>1.03 (0.82-1.31)</td>
</tr>
<tr>
<td>III (highest)</td>
<td>163</td>
<td>410</td>
<td>0.76 (0.59-0.98)</td>
</tr>
<tr>
<td>P</td>
<td>0.039</td>
<td>0.135</td>
<td>0.736</td>
</tr>
</tbody>
</table>

Values are odds ratios (95% CI); Model A is adjusted for age and town; Model B, adjusted for age, town, and BMI; Model C, adjusted for age, town, BMI, total cholesterol, HDL cholesterol, and log triglycerides; and Model D, adjusted for age, town, BMI, total cholesterol, HDL cholesterol, log triglycerides, smoking, alcohol, physical activity, social class, and systolic blood pressure. Adiponectin thirds are as follows: I, <3.32 μg/mL; II, ≥3.32 and <13.33 μg/mL; III, ≥13.33 μg/mL.
regression dilution factor calculated from the resurvey measurements made.23 Meta-analysis was done of studies published before November 2005 with >1 year’s follow-up with the use of search, abstraction, and data synthesis methods that have been described previously24 and with the use of nonfatal MI or CHD death as end points. Corresponding authors of all identified studies were contacted to provide supplementary limited tabular data (6 of 7 contacted authors responded). Results of studies were combined with the use of inverse variance weighted averages of log odds ratios. A regression dilution correction factor of 0.58 (derived from the resurvey in the present study) was applied.

Results of studies were combined with the use of inverse variance weighted averages of log odds ratios. A regression dilution correction factor of 0.58 (derived from the resurvey in the present study) was applied to the studies identified in the meta-analysis. Heterogeneity was assessed by standard χ² tests and the I² statistic, which describes the percentage of variation in the log odds ratios that is attributable to genuine differences across studies rather than random error.25 Odds ratios are given with 95% CIs, and 2-sided probability values are used.

We confirm that the authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

Present Prospective Study

Established coronary risk factors were significantly different between cases and controls in the expected directions, although there was no statistically significant difference in mean adiponectin levels (Table 1).

Associations of Adiponectin With Other Measured Risk Factors

Among controls, there were statistically significant (P<0.0001 unless otherwise stated) positive associations of adiponectin levels with age (r=0.14), HDL cholesterol (r=0.33), and prevalence of physical inactivity (P=0.05) and an inverse association with triglycerides (r=−0.25), BMI (r=−0.21), and CRP (r=−0.11). After adjustment for BMI, the relationships between adiponectin and age, physical activity, HDL cholesterol, triglycerides, and CRP remained statistically significant. The associations of adiponectin levels with smoking status, alcohol intake, preexisting CHD, history of diabetes, total cholesterol, systolic blood pressure, and diastolic blood pressure were not statistically significant (Table 2).

Within-Person Variation in Adiponectin Levels

In an analysis of repeat adiponectin measurements made in 221 participants ~4 years apart, the long-term stability of adiponectin levels, as determined by the within-person correlation coefficient, was 0.58 (95% CI, 0.49 to 0.66). This value is comparable to those obtained for the 4-year within-person correlation coefficient for LDL cholesterol, systolic blood pressure, and CRP previously reported for participants in this study.26

Adiponectin and CHD Risk

In a comparison of men in the top third of baseline adiponectin levels with those in the bottom third (with cut points defined by the distribution among controls), the age- and town-adjusted odds ratio for CHD was 0.76 (95% CI, 0.59 to 0.98; Table 3). After additional adjustment for BMI and other established coronary risk factors, the odds ratio was reduced to 0.89 (95 CI, 0.67 to 1.18), and no statistically significant trends were observed. After correction for regression dilution bias, the odds ratio was 0.79 (95% CI, 0.46 to 1.33). Exclusion of men with preexisting CHD and diabetes made no material difference in the findings (Table 3). Similar results were observed when analyses were repeated on the basis of a comparison of fifths of the same distribution (data not shown). When the continuous relation between baseline log₁₀ adiponectin and CHD was examined, the age- and town-adjusted odds ratio for a 50% increase in adiponectin levels was 0.97 (95% CI, 0.90 to 1.05), and this was reduced to 1.02 (95% CI, 0.93 to 1.12) after further adjustment for other coronary risk factors.

Meta-Analysis of Available Prospective Studies of Adiponectin Concentrations and CHD Risk

We identified 7 published prospective reports on adiponectin and CHD (including the present study) in Western populations, with a total of 1313 CHD cases (weighed mean age at entry, 59 years; weighted mean follow-up, 9.7 years; Table 4). There was little evidence of heterogeneity among the findings of these studies (χ²=8.4; P=0.21; I²=29% [95% CI, 0% to 69%]). A combined analysis of all 7 available
prospective studies yielded an odds ratio of 0.84 (95% CI, 0.70 to 1.01) (Figure) in a comparison of extreme thirds of adiponectin values (0.74 [95% CI, 0.54 to 1.02] after correction for regression dilution), which was similar to the odds ratio obtained in the 4 studies restricted to essentially general populations (odds ratio, 0.81 [95% CI, 0.67 to 0.97]; test for heterogeneity: \( \chi^2 = 0.07; P = 0.79 \)). The summary odds ratio was not sensitive to choice of fixed or random effects models, as described in the figure legend.

Discussion

The present data have demonstrated that the year-to-year variation of adiponectin levels within individuals is comparable to those of blood pressure and blood cholesterol levels. Furthermore, in agreement with previous reports,5,13,14,27 the data indicate that circulating adiponectin levels are inversely associated with adiposity, triglycerides, and CRP and positively associated with HDL cholesterol. However, in contrast to some previous suggestions, the present evidence, involving 589 incident cases of CHD and 1231 controls plus a meta-analysis of 6 previous studies,5,12–16 indicates that any association between adiponectin levels and CHD risk is unlikely to be strong. Indeed, in contrast to associations with CHD previously observed in the same participants in the BRHS for a range of established risk factors (eg, smoking, blood pressure, blood lipids) and emerging markers (eg, CRP), the inverse associations observed with adiponectin levels were comparatively modest and not statistically significant.

In contrast to the situation with CHD, reported associations between adiponectin levels and the risk of incident type 2 diabetes are more extreme, albeit derived from much smaller studies. For example, Spranger et al6 reported that subjects with adiponectin in the top quarter of adiponectin levels had an odds ratio of 0.3 (95% CI, 0.2 to 0.7; \( P = 0.005 \)) for development of type 2 diabetes compared with subjects in the bottom quarter, even after adjustment for measures of adiposity and glycohemoglobin. Comparable results were obtained by Choi et al4 in a prospective study of Korean individuals and Lindsay et al28 in a study of Pima Indians. Some support for the idea that adiponectin levels are more strongly related to type 2 diabetes than to CHD is suggested by observations that newer drugs with beneficial effects on insulin resistance and glucose intolerance (such as the peroxisome proliferator-activated receptor-\( \gamma \) agonists and the selective cannabinoid-1 receptor blocker rimonabant) increase adiponectin concentrations.29,30

Strengths and Limitations

This is the largest prospective study of circulating concentrations of adiponectin and CHD thus far reported, almost doubling the number of cases available. The study was population based, had high response and follow-up rates, and featured robust ascertainment of new CHD cases.17,18 Adjustments for a wide range of potential confounders and for the effect of regression dilution were possible. Of interest, the correlation coefficient of 0.58 for adiponectin recorded 4 years apart in the same subset of BRHS participants was nearly identical to that reported in a 3-year follow-up study in Korean subjects (\( r = 0.63 \)).4 The BRHS demonstrates the expected associations of other risk factors with subsequent events, and thus it is an informative study with which to put into perspective any association between adiponectin and the risk of CHD. Finally, a meta-analysis of previous prospective studies helps to put the new findings in context.

The study had some potential limitations. The study included only men and cannot necessarily be extrapolated to women. However, a recent study in women (in whom adiponectin levels are generally higher) also reported no significant association of adiponectin with incident CHD.13 The samples were stored at \(-20^\circ \text{C} \) and analyzed with an ELISA assay; most previous studies have used radioimmunoassays. However, adiponectin is not known to be influ-
enced by delayed measurement, prolonged storage, or repeated freeze-thaw cycles. Moreover, we and others noted excellent correlation between radioimmunoassay and ELISA methods for measurement of adiponectin concentration\textsuperscript{(r}>0.90). The adiponectin concentrations observed among male controls in the present study were similar to those reported in previous prospective studies of male populations (and, as would be expected on the basis of previous work, about one third lower than levels measured in women) by either ELISA or radioimmunoassay (Table 4).\textsuperscript{3,12} Adiponectin concentrations also demonstrated the expected associations with other risk factors such as HDL cholesterol, BMI, and CRP in this study, and validation of the assay methodology is suggested by the reasonably high degree of year-to-year consistency observed in the paired samples, similar to those previously noted for LDL cholesterol, systolic blood pressure, and CRP in this study population.\textsuperscript{26}

Finally, although differing molecular forms of adiponectin may reflect differing biological effects (with the high-molecular form being potentially associated with slightly greater insulin sensitivity\textsuperscript{33}), commercially available radioimmunoassay or ELISA methods are, at present, unable to distinguish between the lower-weight trimer-dimer form of adiponectin and the high-molecular complexes; separation and measurement of such fractions have generally been conducted by labor-intensive methods. A recent ELISA for measurement of the high-molecular-weight fraction has been proposed recently but requires further validation.\textsuperscript{34} Future studies, using such novel assays, should therefore aim to determine whether there is a specific association between particular forms of adiponectin and CHD.

Conclusions

We have shown that the magnitude of association between circulating adiponectin concentrations and CHD risk is weaker than previously suspected and that more reliable investigation of any moderate associations will require further studies involving much larger sample sizes. It is unlikely that the strength of association of circulating adiponectin with CHD is comparable to that previously reported with type 2 diabetes, suggesting that it may be premature to conclude that adiponectin is part of any “common soil” for type 2 diabetes and CHD.

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Disclosures

None.

References


CLINICAL PERSPECTIVE

Researchers continue to search for new risk factors that may help to better predict risk for cardiovascular disease. The adipocyte-derived hormone adiponectin represents one such factor. Adiponectin paradoxically decreases in concentration with rising obesity and insulin resistance, and, in animal models, recombinant adiponectin protects against vascular disease. Consistent with such data, high baseline adiponectin concentrations in men predicted a significantly lower risk of myocardial infarction in the first prospective study reported ~2 years ago. Such prediction was independent of traditional risk factors. However, results from subsequent, similarly sized prospective studies in men and women have been less convincing. We therefore measured adiponectin in a large prospective study of 589 coronary heart disease (CHD) cases and 1231 controls and added these new data to a meta-analysis of previously published prospective studies. Baseline adiponectin concentrations correlated ($P<0.0001$) positively with HDL cholesterol and inversely with C-reactive protein and body mass index. However, despite these correlations with known risk factors and other markers, there was no significant difference between baseline median adiponectin levels in cases versus controls (10.2 versus 10.8 µg/mL; $P=0.5$). The odds ratio for CHD was 0.89 (95% CI, 0.67 to 1.18) in a comparison of men in the top third of adiponectin concentrations compared with those in the bottom third, similar to a meta-analysis of 7 prospective studies involving a total of 1318 CHD cases (odds ratio, 0.84 [95% CI, 0.70 to 1.01]). We conclude that despite the strong associations previously reported between adiponectin levels and risk of type 2 diabetes, any association with CHD risk is comparatively moderate and requires further investigation.
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In the article, “Adiponectin and Coronary Heart Disease: A Prospective Study and Meta-Analysis” by Sattar et al that published in the August 15, 2006, issue (Circulation. 2006;114:623–629), the assay method attributed to the study of Zoccali et al in Table 4 was incorrectly stated as the assay by Linco; it was an in-house ELISA and done on frozen rather than fresh samples. In addition, with respect to the study of Wolk et al in the same table, frozen rather than fresh blood samples were used.

The authors regret these errors.

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