Fasting Compared With Nonfasting Lipids and Apolipoproteins for Predicting Incident Cardiovascular Events

Samia Mora, MD, MHS; Nader Rifai, PhD; Julie E. Buring, ScD; Paul M Ridker, MD, MPH

Background—Although guidelines recommend measuring fasting lipids for initial screening of adults without cardiovascular disease (CVD), recent studies suggest that nonfasting triglycerides may be superior to fasting. Whether fasting status alters associations of nontriglyceride lipids with CVD is unclear.

Methods and Results—In a prospective study of 26,330 healthy women (19,983 fasting; 6,347 nonfasting), associations of baseline lipids with incident CVD (754 fasting; 207 nonfasting) were examined over an 11-year follow-up. Except for triglycerides, lipid concentrations differed minimally (<5%) for fasting versus nonfasting. However, stronger associations with CVD were noted for fasting total cholesterol (adjusted fasting hazard ratio [HR], 1.22 per 1-SD increment; 95% CI, 1.14 to 1.30; nonfasting HR, 1.07; 95% CI, 0.93 to 1.21), low-density lipoprotein (LDL) cholesterol (fasting HR, 1.21; 95% CI, 1.13 to 1.29; nonfasting HR, 1.00; 95% CI, 0.87 to 1.15), apolipoprotein B-100 (fasting HR, 1.36; 95% CI, 1.27 to 1.45; nonfasting HR, 1.20; 95% CI, 1.05 to 1.36), non–high-density lipoprotein (HDL) cholesterol (fasting HR, 1.29; 95% CI, 1.21 to 1.38; nonfasting HR, 1.15; 95% CI, 1.01 to 1.31), and apolipoprotein B-100/A-1 ratio (fasting HR, 1.39; 95% CI, 1.30 to 1.48; nonfasting HR, 1.18; 95% CI, 1.09 to 1.27). Compared with fasting levels, nonfasting HDL cholesterol, apolipoprotein A-1, and total/HDL cholesterol ratio had similar associations, and triglycerides had a stronger association, with CVD. Significant interactions were seen for LDL cholesterol and apolipoprotein B-100/A-1 ratio with fasting status (P for interaction=0.03 and <0.001, respectively).

Conclusions—This study demonstrates that HDL cholesterol, triglycerides, total/HDL cholesterol ratio, and apolipoprotein A-1 predict CVD when measured nonfasting. By contrast, total, LDL, and non-HDL cholesterol, in addition to apolipoprotein B-100 and B-100/A-1 ratio, provide less useful CVD risk information when nonfasting, despite small changes in their concentrations. Guidelines for lipid screening may need to consider these differences. (Circulation. 2008;118:993-1001.)

Key Words: apolipoproteins • lipids • women

Current guidelines recommend measurement of a fasting lipid profile for cardiovascular risk assessment.1,2 Lipids are traditionally measured after an 8- to 12-hour fast to minimize the influence of postprandial lipemia.3 Ingestion of a typical fat-containing meal results in higher triglyceride levels and smaller changes in low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol.4 The third report of the National Cholesterol Education Program Adult Treatment Panel (Adult Treatment Panel III) recommends that initial screening should include a fasting lipid profile that includes total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides.1 The guidelines allow for the measurement of total and HDL cholesterol in the nonfasting state4 because levels of these 2 lipids are altered minimally when measured in fasting or nonfasting blood.5,6 Non-HDL cholesterol, a secondary target of therapy in Adult Treatment Panel III, may also be used in the nonfasting state.1

Clinical Perspective p 1001

In apparent contrast to these guidelines, 3 studies have suggested that nonfasting triglycerides may better or similarly predict cardiovascular disease (CVD) events than fasting
levels. It has been increasingly recognized that postprandial responses, such as those relating to glucose and triglyceride metabolism, may trigger a number of proatherosclerotic and prothrombotic processes, including inflammation, oxidative stress, and vasoconstriction. It is unknown whether nonfasting status alters the association of nontriglyceride lipids and apolipoproteins with CVD. However, if postprandial effects do not substantially weaken the association of nontriglyceride lipids and apolipoproteins with CVD, then measurement of nonfasting lipids may have many practical advantages for clinical practice. Therefore, we conducted this study in a large prospective cohort of initially healthy women (1) to evaluate levels of lipids and apolipoproteins as a function of time after a typical meal and (2) to determine whether fasting compared with nonfasting status alters the association of these lipids and apolipoproteins with incident CVD.

**Methods**

**Study Population**

Study participants were enrolled in the Women’s Health Study, a recently completed randomized, double-blinded, placebo-controlled clinical trial of low-dose aspirin and vitamin E in the primary prevention of CVD and cancer in US female healthcare professionals. Eligible participants were apparently healthy women, aged ≥45 years, who were free of self-reported CVD or cancer at study entry (1992–1995), with follow-up for incident CVD through February 2006. At the time of enrollment, participants gave written informed consent and completed questionnaires on demographics, medical history, medications, and lifestyle factors. They were also asked to provide a blood sample. Participants were requested, but not required, to have the sample drawn in the morning before eating, and they reported the number of hours since their last meal before the blood draw and the time of day for the blood draw. In total, 27,748 women had baseline measurements on the lipids and apolipoproteins of interest. After exclusion of 1418 women because of missing data on the time since last meal, there were 26,330 women for analysis. The study was approved by the institutional review boards of the Brigham and Women’s Hospital (Boston, Mass). 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.

**Baseline Plasma Measurements**

EDTA blood samples were obtained at the time of enrollment and stored in vapor phase liquid nitrogen (−170°C). In a laboratory certified by the National Heart, Lung, and Blood Institute/Centers for Disease Control and Prevention Lipid Standardization program, baseline samples were thawed and analyzed for standard lipids and apolipoproteins. Total, LDL, and HDL cholesterol were assayed directly with reagents from Genzyme Corporation (Cambridge, Mass) and Roche Diagnostics (Indianapolis, Ind) with the use of a Hitachi 911 autoanalyzer. Apolipoproteins A-1 and B-100 were measured with immunoturbidimetric assays (DiaSorin, Stillwater, Minn).

**Definition of Fasting Status**

Participants whose last meal was ≥8 hours before their blood draw comprised the fasting sample (n=19,983), and those who had eaten within 8 hours of their blood draw comprised the nonfasting sample (n=6347). The study population was also divided into groups according to time since last meal by 2-hour intervals of <2 hours (n=990), 2 to <4 hours (n=2782), 4 to <6 hours (n=1702), 6 to <8 hours (n=872), 8 to <10 hours (n=1530), 10 to <12 hours (n=3490), 12 to <14 hours (n=8550), 14 to <16 hours (n=5196), and ≥16 hours (n=1426).

**Ascertainment of CVD Events**

The primary end point of interest was a composite end point of incident CVD (nonfatal myocardial infarction, percutaneous coro-
nary intervention, coronary artery bypass grafting, nonfatal stroke, or cardiovascular death). During the 11-year follow-up period, women reported the end points of interest on follow-up questionnaires every 6 or 12 months. All events were adjudicated by an end points committee.

### Statistical Analysis

Statistical analyses were performed with the use of STATA version 8.2 (STATA Corporation, College Station, Tex). Statistical comparisons between the fasting and nonfasting groups were obtained from Student t tests for continuous variables expressed as means, from Kruskal-Wallis tests for variables expressed as medians, and from chi² tests for categorical variables. The levels of the lipids and apolipoproteins were examined as a function of time since the last meal divided into 2-hour intervals.

Values shown are HRs (95% CIs) adjusted for age, randomized treatment assignment, smoking status, menopausal status, postmenopausal hormone use, blood pressure, diabetes, and body mass index. P value for linear trend was obtained with the median value for each quintile. \( P_{\text{interaction}} \) was obtained from likelihood ratio tests for interaction with fasting/nonfasting status and the lipid variable in relation to CVD.

**Additionally adjusted for total and HDL cholesterol.**

Following guidelines from the Department of Health and Human Services, lipids and apolipoproteins were divided into quintiles on the basis of the distribution among women not taking hormone replacement. Because the distributions differed between fasting and nonfasting participants for some of the lipid measurements, quintile cut points were defined separately in each of the fasting and nonfasting samples. To address whether the results may differ on the basis of the last meal divided into 2-hour intervals, analyses were repeated per 1-SD increment within strata of time since last meal divided into 2-hour intervals. Cox proportional hazard regression models were used to calculate the hazard ratios (HRs) and 95% CIs according to these quintiles and per 1-SD increments.
To examine the extent to which each lipid or lipoprotein biomarker was associated with incident events, we considered each lipid variable in a separate model that adjusted for nonlipid risk factors (age, randomized treatment assignment, smoking status, menopausal status, postmenopausal hormone use, blood pressure, diabetes, and body mass index). Analyses for triglycerides were additionally adjusted for total and HDL cholesterol based on prior work from this cohort.8 To address any potential confounding from time of day for the blood draw, we additionally adjusted the multivariable models for time of blood draw, which did not affect the findings.

**Table 3. Associations of Lipids and Apolipoproteins With CVD, According to Fasting Status and Postmenopausal Hormone Use**

<table>
<thead>
<tr>
<th></th>
<th>Non–Hormone Users (n=3331 Nonfasting; n=11,258 Fasting)</th>
<th>Hormone Users (n=2801 Nonfasting; n=8,688 Fasting)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Per 1 SD</td>
<td>𝑃</td>
</tr>
<tr>
<td><strong>Lipid concentrations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.10 (0.93–1.30)</td>
<td>0.28</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.27 (1.17–1.38)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.06 (0.89–1.27)</td>
<td>0.49</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.26 (1.16–1.37)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>0.68 (0.54–0.86)</td>
<td>0.002</td>
</tr>
<tr>
<td>Fasting</td>
<td>0.74 (0.65–0.84)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglycerides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.22 (1.10–1.37)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.27 (1.18–1.36)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglycerides*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.12 (0.96–1.30)</td>
<td>0.15</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.07 (0.97–1.18)</td>
<td>0.19</td>
</tr>
<tr>
<td><strong>Apolipoprotein concentrations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apolipoprotein B-100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.25 (1.06–1.47)</td>
<td>0.009</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.40 (1.28–1.53)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Apolipoprotein A-1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>0.80 (0.64–1.00)</td>
<td>0.05</td>
</tr>
<tr>
<td>Fasting</td>
<td>0.74 (0.65–0.84)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Combined lipid measures</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-HDL cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.19 (1.01–1.41)</td>
<td>0.04</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.35 (1.24–1.47)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total/HDL cholesterol ratio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.34 (1.16–1.55)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.42 (1.31–1.53)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Apolipoprotein B-100/A-1 ratio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfasting</td>
<td>1.18 (1.08–1.29)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting</td>
<td>1.42 (1.31–1.53)†</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values shown are HRs (95% CIs) adjusted for age, randomized treatment assignment, smoking status, menopausal status, postmenopausal hormone use, blood pressure, diabetes, and body mass index. 𝑃 value for linear trend was obtained with the median value for each quintile.

*Additionally adjusted for total and HDL cholesterol.

†Likelihood ratio test for interaction with fasting/nonfasting status: LDL cholesterol for hormone users, 𝑃 for interaction = 0.06; apolipoprotein B-100/A-1 for non–hormone users, 𝑃 for interaction = 0.0002.
Results

Table 1 shows the baseline characteristics of participants according to fasting or nonfasting status. Nonfasting women were slightly younger, had less prevalent hypertension, had more diabetes, and were less likely to be postmenopausal than fasting women. Compared with fasting lipids, nonfasting lipids were modestly (by ∼1% to 5%) but statistically significantly lower for total cholesterol, LDL cholesterol, apolipoprotein B-100, non-HDL cholesterol, total/HDL cholesterol ratio, and apolipoprotein B-100/A-1 ratio, with no significant difference for HDL cholesterol or apolipoprotein A-1. As anticipated, triglycerides were higher in the nonfasting women (by ∼15%).

During a median follow-up of 11.4 years, a total of 961 first CVD events occurred (3.34 events per 1000 person-years of follow-up), which affected 754 of 19,983 fasting women (3.8%) and 207 of 6347 nonfasting women (3.3%). Associations of each lipid variable with incident CVD were examined according to nonfasting or fasting quintiles and per 1-SD increments, in separate Cox regression models that considered each lipid variable, one at a time, and adjusted for nonlipid risk factors (Table 2). Nonfasting levels of total cholesterol and LDL cholesterol were not associated with CVD (adjusted nonfasting HR per 1-SD increment in total cholesterol 1.07; 95% CI, 0.93 to 1.21; P=0.35; and for LDL cholesterol, 1.00; 95% CI, 0.87 to 1.15; P=1.0). By contrast, fasting levels of both total cholesterol and LDL cholesterol were significantly associated with CVD (fasting HRs, respectively, of 1.22 [95% CI, 1.14 to 1.30] and 1.21 [95% CI, 1.13 to 1.29]; P<0.001 for both).

Stronger associations with CVD were also noted for fasting compared with nonfasting levels of apolipoprotein B-100 (fasting HR, 1.36; 95% CI, 1.27 to 1.45; nonfasting HR, 1.20; 95% CI, 1.05 to 1.36), non-HDL cholesterol (fasting HR, 1.29; 95% CI, 1.21 to 1.38; nonfasting HR, 1.15; 95% CI, 1.01 to 1.31), and apolipoprotein B-100/A-1 ratio (fasting HR, 1.39; 95% CI, 1.30 to 1.48; nonfasting HR, 1.18; 95% CI, 1.09 to 1.27). Fasting and nonfasting associations of HDL cholesterol, apolipoprotein A-1, and total/HDL cholesterol ratio with CVD were comparable. A statistically significant interaction was seen between fasting/nonfasting status and LDL cholesterol for incident CVD (P for interaction=0.03) and for apolipoprotein B-100/A-1 ratio (P for interaction<0.001), with a borderline significant interaction between total cholesterol and CVD (P for interaction=0.10). Consistent with our prior report, both fasting and nonfasting triglycerides were positively associated with CVD, but further adjustment for total cholesterol and HDL cholesterol weakened the association of fasting triglycerides with CVD.

Figure 1. Median and 25th to 75th percentile values for lipids and apolipoproteins as a function of time since the last meal.
Including time of day for blood draw in the multivariable models did not change our findings.

When we compared the association of fasting and nonfasting lipids and apolipoproteins in women grouped according to baseline use of postmenopausal hormones (Table 3), a similar pattern was observed. The associations of lipids and apolipoproteins with CVD were overall somewhat stronger in the women not taking hormones, but the effect of nonfasting status was similar to that seen in the entire cohort. A statistically significant interaction was seen between fasting/nonfasting status and apolipoprotein B-100/A-1 for incident CVD in non–hormone users \( (P \text{ for interaction} <0.001) \), and a borderline significant interaction was seen between LDL cholesterol and CVD in hormone users \( (P \text{ for interaction}=0.06) \).

Next, to determine the effect of time since the last meal on concentrations of lipids and apolipoproteins, we plotted the median, 25th percentile, and 75th percentile postprandial values by 2-hour intervals (Figure 1). Except for triglycerides, there were no substantial changes in the distributions of lipid and apolipoprotein concentrations as a function of time since the last meal. The highest levels of triglycerides were noted 4 to 5 hours postprandially, which was seen in women with HDL cholesterol \(<50 \text{ or } \geq 50 \text{ mg/dL} \). Figure 2 shows the adjusted HRs (95% CIs) for CVD of each of the lipid and apolipoprotein concentrations (per 1-SD increments) depending on the time since the last meal before the blood draw. For total cholesterol, LDL cholesterol, and non-HDL cholesterol, significant associations with CVD were noted only after at least 10 hours postprandially. By contrast, the strongest associations for the other lipids and apolipoproteins were noted 6 to 8 hours postprandially.

**Discussion**

In this prospective study of 26,330 initially healthy women, the concentrations of lipids and apolipoproteins differed minimally when measurements were performed on nonfasting compared with fasting blood, except for triglycerides, which were higher when nonfasting. However, the associations with CVD were stronger for fasting compared with nonfasting measurements of total cholesterol, LDL cholesterol, apolipoprotein B-100, non-HDL cholesterol, and the apolipoprotein B-100/A-1 ratio. By contrast, the associations with CVD were similar for fasting and nonfasting HDL cholesterol, apolipoprotein A-1, and the total/HDL cholesterol ratio and stronger for nonfasting triglycerides. These observations suggest that nonfasting blood draws may be highly effective and practical when limited to HDL cholesterol, total/HDL cholesterol ratio, and triglycerides. However, these data also suggest that a fasting sample is preferred if
risk assessment is based on total cholesterol, LDL cholesterol, or non-HDL cholesterol. Prior studies have found lower concentrations of LDL cholesterol postprandially and higher triglycerides, with a similar magnitude of difference in our study compared with prior studies that used a typical non–high-fat meal.\textsuperscript{6,15,16} Our finding that there was no significant difference between fasting and nonfasting measurements of HDL cholesterol and apolipoprotein A-1 is also consistent with other studies.\textsuperscript{17,18}

However, to our knowledge, this is the first study that prospectively compares the association of a comprehensive panel of lipids and apolipoproteins with CVD depending on the time to last meal. Although prior studies have evaluated the effect of food intake on lipid and apolipoprotein concentrations,\textsuperscript{6,16,17,19–22} the influence of postprandial time on the predictive value of lipids and apolipoproteins, other than triglycerides, is scarce. In a prior case-control report examining 683 postmenopausal healthy women that included some who were nonfasting, the results were not analyzed according to fasting status except for triglycerides, which showed similar prediction in the fasting or nonfasting state.\textsuperscript{23} The Apolipoprotein-related MOrtality RISK (AMORIS) study included a large proportion of women, with a third of participants who were nonfasting, but associations of lipids and apolipoproteins with fatal myocardial infarction were not reported according to fasting status.\textsuperscript{24} Other prospective studies that included a large number of nonfasting participants had data only on men and did not have comparisons with fasting lipids.\textsuperscript{25–27} Although differences between fasting and nonfasting concentrations of lipids and apolipoproteins, except for triglycerides, were small and clinically insignificant in our study, the strength of the association of various lipids and apolipoproteins with CVD differed by fasting/nonfasting status. Total cholesterol and LDL cholesterol showed the most marked differences in their predictive value, although different associations were also found for apolipoprotein B-100, non-HDL cholesterol, and the apolipoprotein B-100/A-1 ratio, all of which were stronger in the fasting state. This finding suggests that performing a nonfasting measurement of total cholesterol or non-HDL cholesterol, which is currently believed to be acceptable, may need to be interpreted with caution.

As suggested by Figure 2, even stronger associations may be observed within 6 to 8 hours postprandially for lipids and apolipoproteins that represent part of the atherogenic dyslipidemia of the metabolic syndrome, such as HDL cholesterol and triglycerides, whereas longer durations of fasting (>10 to 12 hours) may be required for total cholesterol and LDL cholesterol. The underlying biological explanation for this is unclear but may relate to the time course of postprandial triglyceride metabolism because 4 to 8 hours is the time of...
peak triglycerides, and by 8 hours, triglyceride concentrations have returned to fasting concentrations in most individuals.28

The present study has several limitations. Time to last meal was self-reported, and we did not have both fasting and nonfasting measurements in the same individuals. Lipid measurements were only available once at baseline, and results could not be corrected for potential regression dilution bias. We only had data on women, although no substantial differences were noted in women who were or were not taking hormones. Our study included healthcare professionals who were mostly white, apparently healthy, and recruited from a variety of geographic locations across the United States; thus, it is unclear if our results would be applicable to other ethnic populations or men. Our statistical power was less in nonfasting than in fasting women. Finally, this was a primary prevention population, and further studies are needed before the data can be extended to secondary prevention populations that are frequently treated with lipid-lowering medications.

Strengths of the present study include the large number of healthy women participants with comprehensive measurements of a panel of lipids and apolipoproteins, including the direct measurement of standard lipids. Additionally, detailed information on cardiovascular risk factors was available, allowing for the control for potential confounding by these factors, such as the time of day of blood draw and hormone use. Finally, previous studies have not examined the influence of fasting status on the predictive ability of various lipids and lipoproteins, other than triglycerides, all of which was possible in this study because of the large number of participants in subgroups divided by time since the last meal.

In summary, this study demonstrates that HDL cholesterol, triglycerides, total/HDL cholesterol ratio, and apolipoprotein A-1 predict CVD when measured nonfasting. By contrast, total, LDL, and non-HDL cholesterol, in addition to apolipoprotein B-100 and B-100/A-1 ratio, may provide less useful CVD risk information when measured nonfasting, despite small changes in their concentrations. Guidelines for lipid screening may need to consider these differences.

Sources of Funding

The Women’s Health Study is supported by grants HL-43851 and CA-47988 from the National Heart, Lung, and Blood Institute and the National Cancer Institute and by grants from the Donald W. Reynolds Foundation, Leukod Foundation, and Doris Duke Charitable Foundation, with additional support from an Investigator-Initiated Studies Program from Merck. Dr Mora is supported by grants from the American Heart Association (0670007N), Sandra Daugherty Foundation, and Lerner Research Young Investigator Award. The funding agencies played no role in the design, conduct, data management, analysis, or manuscript preparation related to this manuscript.

Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Current guidelines recommend measurement of a fasting lipid profile for cardiovascular risk assessment. Recent studies suggest that nonfasting triglycerides may better or similarly predict cardiovascular disease (CVD) events than fasting levels. Measurement of nonfasting lipids may have many practical advantages for clinical practice, but it is unknown whether nonfasting status alters the association of nontriglyceride lipids and apolipoproteins with CVD. Using prospectively collected data from the Women’s Health Study with 26,330 healthy women (19,983 fasting; 6,347 nonfasting) followed over an 11-year period, we examined the association of CVD with baseline lipids and apolipoproteins according to time from last meal. We found that the concentrations of lipids and apolipoproteins differed minimally when measurements were performed on nonfasting (<8 hours from last meal) compared with fasting blood (≥8 hours from last meal), except for triglycerides, which were higher when nonfasting. However, the associations with CVD were stronger for fasting compared with nonfasting measurements of total cholesterol, low-density lipoprotein cholesterol, apolipoprotein B-100, non–high-density lipoprotein (non-HDL) cholesterol, and the apolipoprotein B-100/A-1 ratio. By contrast, the associations with CVD were similar for fasting and nonfasting HDL cholesterol, apolipoprotein A-1, and the total/HDL cholesterol ratio and stronger for nonfasting triglycerides. These observations suggest that nonfasting blood draws may be highly effective and practical when limited to HDL cholesterol, total/HDL cholesterol ratio, triglycerides, and apolipoprotein A-1. However, these data also suggest that a fasting sample is preferred if risk assessment is based on total cholesterol, low-density lipoprotein cholesterol, non-HDL cholesterol, apolipoprotein B-100, and apolipoprotein B-100/A-1 ratio.
Fasting Compared With Nonfasting Lipids and Apolipoproteins for Predicting Incident Cardiovascular Events
Samia Mora, Nader Rifai, Julie E. Buring and Paul M Ridker

Circulation. 2008;118:993-1001; originally published online August 18, 2008;
doi: 10.1161/CIRCULATIONAHA.108.777334
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/118/10/993

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/