Fasting Versus Nonfasting Triglycerides and the Risk of Cardiovascular Events

Gennady Nosovitsky

Pacific University
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Abstract

Background: ATP III guidelines suggest a 9- to 12-hour fast before obtaining lipid levels. Various publications have challenged this practice citing postprandial hypertriglyceridemia as a risk for cardiovascular events. Although this association is not entirely certain, it does raise into question the requirement for obtaining fasting lipoprotein measurements.

Method: Exhaustive literature search was conducted using multiple search engines, with keywords related to lipids, postprandial time and cardiovascular events. Relevant studies were assessed for quality of evidence using GRADE.

Results: Three prospective cohort studies met inclusion criteria. All studies included fasting and nonfasting plasma triglycerides, which were about ~15% higher in the nonfasting group. First study had 19 983 fasting and 6347 nonfasting female participants. Cardiovascular events HR for postprandial hypertriglyceridemia was 1.22 (95% CI 1.12-1.33) and 1.23 (95% CI 1.16-1.30) in the fasting. Further adjusting for total and HDL-cholesterol, showed an HR 1.17 (95% CI 1.04-1.31) and 1.07 (95% CI 1.00-1.15), respectively. Second study included 20 118 fasting and 6391 nonfasting female participants. When adjusted for all possible variables, the nonfasting group showed an HR of 1.98 (95% CI 1.21-3.25) and 1.09 (95% CI 0.85-1.41) for the fasting group. Third study included 2809 men, who, at baseline, had both fasting and nonfasting triglycerides measured. At an 8-year follow-up, cardiovascular event HR was 1.64 (95% CI 1.17-2.29) and 1.46 (95% CI 1.03-2.07) for fasting and nonfasting triglycerides ≥200 mg/dL, respectively. At 18-year follow-up, HR for cardiovascular-associated death in the fasting cohort was 0.93 (95% CI 0.61-1.41) versus 1.60 (95% CI 1.05-2.45) for nonfasting.

Conclusion: All of the studies reviewed showed nonfasting triglycerides to be an independent risk factor for cardiovascular events. However, risk with fasting levels was not as consistent. At this time, fasting is standard practice, but in certain patients, checking nonfasting lipids may be appropriate.

Keywords: Lipid, lipoprotein, triglyceride, fasting, nonfasting, cardiovascular events

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Fasting Versus Nonfasting Triglycerides and the Risk of Cardiovascular Events

Gennady Nosovitsky

A Clinical Graduate Project Submitted to the Faculty of the
School of Physician Assistant Studies
Pacific University
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Clinical Graduate Project Coordinator: Annjanette Sommers, PA-C, MS
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**Conclusion:** All of the studies reviewed showed nonfasting triglycerides to be an independent risk factor for cardiovascular events. However, risk with fasting levels was not as consistent. At this time, fasting is standard practice, but in certain patients, checking nonfasting lipids may be appropriate.

**Keywords:** Lipid, lipoprotein, triglyceride, fasting, nonfasting, cardiovascular events
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List of Abbreviations

1-SD 1-Standard Deviation
ATP III Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults – Adult Panel III Guidelines
BMI Body Mass Index
CABG Coronary Artery Bypass Grafting
CHD Coronary Heart Disease
CI Confidence Interval
CRP C-Reactive Protein
CV Cardiovascular
GRADE Grading of Recommendations, Assessment, Development and Evaluations
HDL-C High-density Lipoprotein Cholesterol
HMG-CoA HydroxyMethylGlutaryl Coenzyme A
HR Hazard Ratio
HRT Hormone Replacement Therapy
LDL-C Low-density Lipoprotein Cholesterol
MI Myocardial Infarction
NIH National Institute of Health
NS Not Statistically Significant
PCI Percutaneous Coronary Intervention
TC Total Cholesterol
TC:HDL Total Cholesterol to High-density Lipoprotein Ratio
**Fasting Versus Nonfasting Triglycerides and the Risk of Cardiovascular Events**

**BACKGROUND**

Routine measurement of cholesterol has become a cornerstone of primary care, in both screening and monitoring of hyperlipidemia, given the well established link between cardiovascular disease and hyperlipidemia. Current recommendation guidelines on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults – Adult Panel III (ATP III) from the National Institute of Health (NIH) suggest a 9- to 12-hour fast before obtaining lipid or lipoprotein levels. However, if nonfasting lipids are obtained, only Total Cholesterol and high-density lipoprotein (HDL) levels should be used for any clinical decision making, given the variability in triglycerides and thus low-density lipoproteins (LDL), since LDL levels are calculated based on measured triglycerides. Various publications however, challenge the current paradigm of measuring fasting lipoproteins, especially triglycerides, and argue for nonfasting measurements instead. Recent studies have shown postprandial hypertriglyceridemia as a risk factor for developing cardiovascular events and thus posing a question of whether it is really necessary to obtain fasting lipids. If the data demonstrates a push towards lifting the recommendation for fasting lipoprotein measurements, this may not only influence the practice of many primary care providers, but can also increase compliance with lipid testing, since it does not require patients to return for follow-up, without jeopardizing patient care.
METHODS

An exhaustive literature search was conducted using the following search engines: MEDLINE-Ovid, Web of Science, CINAHL and EBMR Multifile; using the following keywords: nonfasting, postprandial, lipids, triglycerides and cardiovascular event. References of all eligible studies were also reviewed for relevant articles. Inclusion criteria used: adult population, studies having both fasting and a nonfasting group and the influence on cardiovascular events. All studies investigating drug intervention on lipid profiles and outcomes were excluded. All relevant studies were assessed for quality of evidence using the Grading of Recommendations, Assessment, Development and Evaluation (GRADE).

RESULTS

Initial literature search resulted in 192 articles. After reviewing the articles based on the eligibility criteria and removing duplicates, a total of three studies met the criteria. All three studies were prospective cohort studies.²,⁶,¹²

Mora et al

This was a prospective cohort trial¹² which evaluated plasma lipid concentration at various postprandial times and determined if fasting versus nonfasting status differs in predicting cardiovascular events.¹²

Fasting status was defined as having last meal ≥8-hours prior to phlebotomy. Two groups were compared, fasting versus nonfasting, and then further divided according to time since last meal. The nonfasting female cohort comprised of 6347 participants. The fasting group included 19,983 female participants. Each group was then subdivided based on time since last meal in 2-hour intervals. The nonfasting
group was divided into the following groups: <2-hours (n=991), 2 to <4 (n=2782), 4 to <6 (n=1702), and 6 to 8 (n=872); while the fasting group was divided into the following groups: 8 to <10 (n=1321), 10 to <12 (n=3490), 12 to <14 (n=8550), 14 to <16 (n=5196) and >16-hours (n=1426). The primary end-point recorded was the incidence of a cardiovascular event, which the study defined as nonfatal myocardial infarction (MI), percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), nonfatal stroke or cardiovascular-related death.\(^\text{12}\)

During the 11.4-year follow up, there were a total of 961 cardiovascular events, 754 (3.8%) in the fasting group and 207 (3.3%) in the nonfasting group. When compared with fasting lipids, nonfasting lipids showed about 1-5% lower concentration of total cholesterol (TC), low-density lipoprotein (LDL), apolipoprotein-B-100, non-high-density lipoprotein (non-HDL), and apolipoprotein-B-100:A-1 ratio levels. Triglycerides, however, were about 15% higher in the nonfasting group. The results (Table 2) further showed a significant risk of a cardiovascular event in the setting of elevated postprandial triglycerides when compared with fasting concentration, HR 1.22 (95% CI 1.12-1.33) and 1.23 (95% CI 1.16-1.30), respectively. However, when further adjusted for total and HDL-cholesterol, showed an HR 1.17 (95% CI 1.04-1.31) and 1.07 (95% CI 1.00-1.15), for nonfasting and fasting, respectively. However, the study did find that for TC, LDL, apolipoprotein-B-100, non-HDL, and apolipoprotein-B-100:A-1 ratio, fasting levels were better associated with a cardiovascular event. HDL, apoliprotein-A-1, and TC:HDL ratio were comparable. The study also assessed to see if time of day had any influence on lipid levels or risk and this was found to not be of any clinical significance.\(^\text{12}\)
The study did have some limitations. First, only women participated in the study. Thus the results may not be able to be applied to male and non-white patients in a clinical setting. However despite the limitations, the study does suggest fasting lipids may not be necessary when assessing levels of HDL or triglycerides.\textsuperscript{12}

\textbf{Bansal et al}

This is a prospective cohort study\textsuperscript{2} which compared fasting and nonfasting triglycerides and the incidence of a cardiovascular event in a female population. Of the 26,509 women participants, there were 20,118 in the fasting group and 6,391 in the nonfasting group. The outcomes assessed were cardiovascular events, which included myocardial infarction, ischemic stroke, coronary revascularization, or cardiovascular related-death. Data analyses was adjusted for aspirin, age, blood pressure, smoking status and hormone replacement therapy (HRT).\textsuperscript{2} Fasting was defined as nothing by mouth for at least 8-hours prior to phlebotomy. Follow-up was 11-years.

Each group was subdivided into three-tertiles, which were based on plasma triglyceride concentration. Each tertile was then subanalysed using three adjustment-models, which controlled for different variables. Model 1 adjusted for age, blood pressure, smoking, and the use of HRT; Model 2 adjusted for all variables in Model 1 plus additionally for total and high-density lipoprotein cholesterol (HDL-C); Model 3 included Model 2 plus diabetes mellitus, body mass index (BMI), and high C-reactive protein (CRP) levels.\textsuperscript{2} Data was reported in terms of hazard ratios, as illustrated in Table 3.

When controlling for all variables, nonfasting triglycerides showed higher HR for tertile-3, triglyceride concentration \(\geq 171\) mg/dL, compared to fasting levels.
Further data review, showed statistical significance for a link between postprandial triglycerides concentration and cardiovascular events when lipid levels were checked 2- to 4-hours after a meal, but not when measured within 2-hours.²

In their comments, the authors describe that postprandial hypertriglyceridemia showed an independent risk for a CV event, compared with that of fasting concentration, and was most significant at 2- to 4-hours postprandially.² However, the study does have several limitations. First, participants were not randomized to the assigned groups, though baseline characteristics were similar between the groups. Second, only female participants were included in the study and thus extrapolating the data to male patients is probably not recommended.² Third, no ethnicity of the study participants was provided, thus applying the data to other ethnic groups maybe somewhat problematic. However, despite the limitations, the study does ultimately recommend moving towards measuring postprandial rather than fasting lipoprotein concentrations, citing study strengths of “large sample size, extended follow-up time with validated outcomes and measurement of both fasting and nonfasting levels of triglycerides within the same cohort.”² The authors also point out that “postprandial levels are a more robust indicator of cardiovascular risk, perhaps because the greater variability of postprandial levels captures important information about an individual’s metabolism.”²

Eberly et al

This was a prospective cohort trial comparing both fasting and nonfasting triglyceride measurements and to determine the implication for developing coronary heart disease (CHD) based on triglyceride concentration. The study included 2809 men, who,
at baseline, had both fasting and nonfasting lipoprotein measurement, with particular emphasis on plasma triglyceride concentration.6

The authors report baseline mean triglyceride concentration of 187 mg/dL (±135 mg/dL) and 284 mg/dL (±193 mg/dL) for the fasting and nonfasting groups, respectively. Of the 2809 participants, 874 (31%) had fasting triglyceride levels ≥200 mg/dL, compared to 1724 (61%) in the nonfasting group.6

Follow-up was approximately 25.4 years. During that time period, there were a total of 117 deaths (13.4%) in the fasting group (HR, 1.24, 95% CI 0.97-1.60) among those with triglyceride concentration of ≥200 mg/dL, compared to 226 deaths (13.1%) in the nonfasting group (HR, 1.26, 95% CI 0.98-1.62). There were also reports of 211 deaths (10.9%) in participants with triglyceride concentration of <200 mg/dL in the fasting group, versus 102 deaths (9.4%) in the nonfasting group (Table 4).6 The authors reported a cardiovascular event HR of 1.64 (95% CI 1.17-2.29) and 1.46 (95% CI 1.03-2.07) for fasting and nonfasting plasma triglycerides ≥200 mg/dL, respectively, at 8-year follow-up. However, at the 18-year follow-up, HR for cardiovascular-associated death in the nonfasting cohort was 1.60 (95% CI 1.05-2.45), compared to 0.93 (95% CI 0.61-1.41) in the fasting.6

The authors describe a limitation of not determining time since last meal.6 However this may not indicate a limitation, but rather represent true everyday clinical practice. Thus, in addition to the results, the authors make a case for the utilization of nonfasting lipid levels given the convenience of obtaining nonfasting measurement, higher prevalence for postprandial hypertriglyceridemia and similar risk compared to that of fasting.
DISCUSSION

It is well documented that hyperlipidemia is a risk factor for cardiovascular disease and fasting lipoprotein measurements, according to ATP III recommendation guidelines, is currently considered the standard of care when assessing a patient’s lipid profile. In a clinical setting this creates an inconvenience for patients and providers alike. However recent studies have raised doubt as to the need to measure fasting lipids and thus changing clinical practice.

The studies in this review directly compared fasting versus nonfasting lipid concentration as a risk factor for cardiovascular events. Two of the studies focused specifically on triglyceride concentration, whereas Mora et al included all fractions of a clinically available lipid panel.

When examining the correlation between fasting versus nonfasting plasma triglyceride concentration and the risk for developing cardiovascular events, Mora et al and Eberly et al both showed a statistically significant correlation for such events in the setting of hypertriglyceridemia, without regard to time since last food intake. In the Mora et al study, each 1-SD increase in plasma triglyceride concentration demonstrated an HR of 1.23 (95% CI 1.16-1.30) and 1.22 (95% CI 1.12-1.33) for fasting and nonfasting levels, respectively. Although, when further adjusted for total cholesterol and HDL, the HR for fasting was 1.07 (95% CI 1.00-1.15) and 1.17 (95% CI 1.04-1.31) for nonfasting. Eberly et al similarly reported a cardiovascular event HR of 1.64 (95% CI 1.17-2.29) and 1.46 (95% CI 1.03-2.07) for fasting and nonfasting plasma triglycerides ≥200 mg/dL, respectively, at 8-year follow-up. However, at the 18-year follow-up, HR for cardiovascular-associated death in the nonfasting cohort was 1.60 (95% CI 1.05-
2.45), compared to 0.93 (95% CI 0.61-1.41) in the fasting (Table 4). Furthermore, in the Bansal et al\textsuperscript{2} study, when adjusted for all possible variables such as age, blood pressure, smoking, HRT, HDL, diabetes, BMI, and high CRP levels (Model 3), the nonfasting tertile-3 group showed an HR of 1.98 (95% CI 1.21-3.25) compared to 1.09 (95% CI 0.85-1.41) for the fasting group.\textsuperscript{2}

Despite the finding of postprandial hypertriglyceridemia as a risk for a cardiovascular event, Mora et al\textsuperscript{12} also found a statistically significant risk for each 1-SD increment increase in measured low-density lipoprotein (LDL) when obtained ≥9-hours after a meal (HR 1.21 [95% CI 1.13-1.29]) compared to a nonfasting measurement (HR 1.00 [95% CI 0.87-1.15]). This was despite statistically significant lower LDL concentration in the nonfasting group,\textsuperscript{12} which was consistent with other studies showing lower postprandial LDL concentrations.\textsuperscript{4,5,11,21,28} The authors in the Mora et al\textsuperscript{12} study, however, did not provide an explanation as to why fasting LDL showed to be a better predictor for cardiovascular disease, compared to nonfasting, despite lower concentrations postprandially. The authors also did not comment as to whether any of the study participants were on statin (HMG-CoA inhibitor) lipid-lowering therapy. Although it is unlikely for the study participants to be on any lipid-lowering agents during the time of the trial, it is possible that those in the fasting group were counseled more aggressively given their higher fasting LDL levels.

The data showing statistically significant risk with postprandial hypertriglyceridemia presents clinicians with flexibility when obtaining lipoprotein levels in a nonfasting individual. In a clinical setting, providers frequently have to decide whether to have the afternoon patient come back in the morning after at least a 9-hour
fast or to allow for nonfasting measurements. This may provide clinicians with some
guidance on interpreting nonfasting lipoprotein measurements. If a nonfasting lipid
concentration is obtained, providers can base their clinical decision-making on
triglycerides and total:HDL-cholesterol ratio, with an understanding that the LDL levels
will be lower by approximately 1-5%.12 Consequently, this poses an important clinical
question as to how providers proceed with regard to pharmacological interventions given
that most lipid-lowering trials were done with participants being in the fasting state,16
making nonfasting lipid measurements to some extent ambiguous. Future large
multicenter clinical trials will be very useful in clarifying how lipid-lowering agents may
influence cardiovascular event outcomes in the setting of postprandial
hypertriglyceridemia.

Using the GRADE system, studies2,6,12 were reviewed for quality of
evidence (Table 1). All three individual studies showed “low” level in the assessment of
the quality of evidence. In the Bansal et al2 and Mora et al12 studies, the authors reported
several limitations. First, time to last meal was patient-self-reported. Second, the study
only included female participants “who were mostly white,”12 although the exact
ethnicity percentage was not reported.2,12 Although these limitations preclude clinicians
from extrapolating the data to males and potentially other ethnic groups, it was felt that
this did not warrant a downgrade. All other categories (inconsistency, indirectness,
imprecision and publication bias) were assessed and no discrepancies were noted. Bansal
et al2 and Mora et al12 both had a significantly large cohort of participants, with Mora et
al12 also reporting comprehensive data for all clinically available lipoprotein
measurements.
CONCLUSION

Despite the recommendation for obtaining plasma lipoprotein measurements in the fasting state, several studies have questioned this practice, raising the argument that postprandial hypertriglyceridemia is a risk factor for cardiovascular events. All of the studies reviewed herein did show nonfasting plasma triglycerides to be an independent risk factor for cardiovascular events. However, when it came to fasting plasma triglycerides, the same consistency and correlation across the studies could not be found. Although, at this time, using a fasting lipid profile is standard practice – given the results of the aforementioned studies – in certain patients obtaining nonfasting lipid profile may be more convenient as compliance may be an issue. However, there are currently no formal recommendations to suggest this practice and further studies are needed to corroborate the findings mentioned above.


<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Limitations</th>
<th>Inconsistency</th>
<th>Indirectness</th>
<th>Imprecision</th>
<th>Publication Bias</th>
<th>Quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mora et al</td>
<td>Prospective Cohort</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Unlikely</td>
<td>Low</td>
</tr>
<tr>
<td>Bansal et al</td>
<td>Prospective Cohort</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Unlikely</td>
<td>Low</td>
</tr>
<tr>
<td>Eberly et al</td>
<td>Prospective Cohort</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Unlikely</td>
<td>Low</td>
</tr>
</tbody>
</table>
**TABLE 2** Mora et al study, cardiovascular events rate, adjusted hazard ratios

<table>
<thead>
<tr>
<th>Cardiovascular Events, No (%)</th>
<th>Fasting (n = 19 983)</th>
<th>Nonfasting (n = 6347)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>754 (3.8%)</td>
<td>207 (3.3%)</td>
</tr>
</tbody>
</table>

**Adjusted Hazard Ratios (95% CI)**  
per 1-SD increment increase

<table>
<thead>
<tr>
<th></th>
<th>Fasting</th>
<th>Nonfasting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>1.23 (1.16-1.30)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.22 (1.12-1.33)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Triglycerides, adjusted for total and HDL-cholesterol</td>
<td>1.07 (1.00-1.15)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>1.17 (1.04-1.31)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total:HDL-cholesterol ratio</td>
<td>1.36 (1.27-1.45)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.28 (1.15-1.44)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>1.22 (1.14-1.30)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.07 (0.93-1.21)&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
<tr>
<td>LDL</td>
<td>1.21 (1.13-1.29)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.00 (0.87-1.15)&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>0.79 (0.72-0.86)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.75 (0.64-0.89)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Non-HDL</td>
<td>1.29 (1.21-1.38)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.15 (1.01-1.31)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Apolipoprotein A-1</td>
<td>0.82 (0.75-0.89)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.86 (0.73-1.00)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Apolipoprotein B-100</td>
<td>1.36 (1.27-1.45)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.20 (1.05-1.36)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Apolipoprotein B-100:A-1 ratio</td>
<td>1.39 (1.30-1.48)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.18 (1.09-1.27)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>P<0.05, <sup>*</sup>P=NS

**TABLE 3** Bansal et al study, fasting and nonfasting Hazard Ratios for each tertile

<table>
<thead>
<tr>
<th>Fasting triglycerides, mg/dL</th>
<th>Tertile 1</th>
<th>Tertile 2</th>
<th>Tertile 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤90</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1 [reference point]</td>
<td>1.63 (1.31-2.02)</td>
<td>2.23 (1.82-2.74)</td>
</tr>
<tr>
<td>Model 2&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1 [reference point]</td>
<td>1.27 (1.02-1.59)</td>
<td>1.32 (1.03-1.68)</td>
</tr>
<tr>
<td>Model 3&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1 [reference point]</td>
<td>1.21 (0.96-1.52)</td>
<td>1.09 (0.85-1.41)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nonfasting triglycerides, mg/dL</th>
<th>Tertile 1</th>
<th>Tertile 2</th>
<th>Tertile 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤104</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1 [reference point]</td>
<td>1.48 (0.95-2.29)</td>
<td>2.53 (1.69-3.79)</td>
</tr>
<tr>
<td>Model 2&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1 [reference point]</td>
<td>1.31 (0.83-2.05)</td>
<td>1.94 (1.21-3.10)</td>
</tr>
<tr>
<td>Model 3&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1 [reference point]</td>
<td>1.44 (0.90-2.29)</td>
<td>1.98 (1.21-3.25)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Adjusted Hazard Ratios (95% CI) for age, blood pressure, smoking, and use of hormone therapy  
<sup>b</sup>Adjusted HR (95% CI) model 1 plus total and high-density lipoprotein cholesterol (HDL-C)  
<sup>c</sup>Adjusted HR (95% CI) for model 2 plus diabetes mellitus, body mass index, and high C-reactive protein levels.
<table>
<thead>
<tr>
<th></th>
<th>Triglyceride concentration, mg/dL</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;200</td>
<td>≥200</td>
<td></td>
</tr>
<tr>
<td><strong>FASTING</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular Events, 8-year follow-up, No (%) [HR (95% CI)]</td>
<td>102 (5.3%)</td>
<td>73 (8.4%)</td>
<td>[1.64 (1.17-2.29)]</td>
</tr>
<tr>
<td>HR (95% CI) for death, 18-year follow-up</td>
<td>n/a</td>
<td>0.93 (0.61-1.41)</td>
<td></td>
</tr>
<tr>
<td>Deaths, 25-year follow-up, No (%) [HR (95% CI)]</td>
<td>211 (10.9%)</td>
<td>117 (13.4%)</td>
<td>[1.24 (0.97-1.60)]</td>
</tr>
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<tr>
<td><strong>Non-FASTING</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular Events, 8-year follow-up, No (%) [HR (95% CI)]</td>
<td>50 (4.6%)</td>
<td>125 (7.3%)</td>
<td>[1.46 (1.03-2.07)]</td>
</tr>
<tr>
<td>HR (95% CI) for death, 18-year follow-up</td>
<td>n/a</td>
<td>1.60 (1.05-2.45)</td>
<td></td>
</tr>
<tr>
<td>Deaths, 25-year follow-up, No (%) [HR (95% CI)]</td>
<td>102 (9.4%)</td>
<td>226 (13.1%)</td>
<td>[1.26 (0.98-1.62)]</td>
</tr>
</tbody>
</table>