We fully agree with the authors that basically, similar to other previously such conclusion can be drawn, we would like to make the following comments:

(1) Transradial approach has been initially described more than 15 years ago and it has become popular mainly outside US. Today, it is the best method to minimize the risks of access site complications and bleeding after percutaneous coronary interventions (PCIs).

(2) Bleeding post-PCI is a dreadful consequence, which increases acute morbidity and mortality, prolongs hospitalization, and costs millions to health systems. Major bleeding post-PCI is now recognized as a strong independent predictive factor of mortality. Patients at higher risk of bleeding are also those who benefit the most from transradial approach.

(3) Clinical scenarios associated with increased risk of bleeding such as primary and rescue PCI were excluded from Brasselet’s study.

(4) Transradial approach has also permitted the development of outpatient PCI practice even when maximized anticoagulation and high-risk patients are involved.

(5) Basically, similar to other previously published reports, this report shows that longer fluoroscopy time is associated with increased patient and physician exposure. Since patients are not exposed several times, the remote stochastic and non-stochastic risks associated with transradial approach remain negligible and certainly should not be weighted against the immediate risks of bleeding and/or access site complications.

(6) We fully agree with the authors that radiation exposure is an important issue most of the time under-evaluated by most operators. Although the authors claim optimized radiation protection, we notice that there is no mention of operators wearing leaded glasses nor are they visible on their pictures. Cataract is a non-stochastic risk, which means that the incidence would presumably be close to 100% if the operator reaches a certain threshold. In our institution, where transradial approach has been the default technique for the last 14 years, operators perform about 1000 diagnostic catheterizations and 200–300 PCI cases/year and have been strongly advised to wear leaded glasses (still not all operators wear them!). By keeping annual eye exposure below the recommended 150 mSv, a cardiologist could therefore work for more than 35 years before reaching the cataract threshold.

(7) From Figure 2, it appears that most of the difference in radiation exposure results from the diagnostic part. Indeed, difficulties in catheter progression through the arm and/or subclavian part can lead to additional fluoroscopy and is, however, recommended to avoid potential arterial damages. Thus, the practice of ad hoc procedures compared with diagnostic and PCI in separate procedures has an important role in limiting patient and staff exposure.

(8) Once the transradial approach becomes a common practice, there are several tricks that can help to reduce radiation exposure like exchanging catheters over wire placed in the ascending aorta without using fluoroscopy.

(9) Finally, from the report of Brasselet et al., those promoting transradial approach should take a step back and carefully revisit the radiation protection devices they currently use and look for further optimization. For example, instead of using 0.5 mm leaded glass, why not use thicker protection that would further reduce operator exposure!

In conclusion, transradial approach has been a major step forward in the practice of percutaneous coronary angiography and PCIs to the benefit of all patients. Tremendous improvements in radiation protection measures have been associated with a dramatic reduction in patient and staff radiation exposure over the last decade. It is time for ‘radialists’ to discuss with radiation protection specialists and the industry further means to reduce staff radiation exposure and hence, the associated risks.

References

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which was eventually published.\(^5\) Compared with that of von Eynatten et al., our cohort showed more CV deaths and composite CV events (comprising CV death, non-fatal myocardial infarction, acute coronary syndromes, stroke, and vascular surgery) (45 and 119, respectively) even despite a slightly shorter follow-up (median 3.8 years), thus providing a high statistical power to our study.

Noteworthy, at univariate Kaplan–Meier analysis, we found a higher CV death rate among those with a high plasma adiponectin than in those with a low plasma adiponectin, suggesting a paradoxical detrimental role of adiponectin on CV outcome. Nonetheless, a comparison of the two groups evidenced an unbalanced distribution of several covariates that can affect outcome, including major CV risk factors, rate of previous CV events, and ongoing medical treatment.

We therefore undertook a hierarchical multivariate Cox regression analysis where these potential confounders were considered. At variance with von Eynatten et al. who considered only the number of major epicardial coronary vessel affected, we could estimate the CAD burden by a modified Duke prognostic index score that furnishes an accurate estimation of the severity of the CAD extension. Moreover, at variance with von Eynatten et al. who considered only the use of angiotensin-converting enzyme-inhibitors and lipid-lowering drugs, we considered all the ongoing CV drugs.

This refined examination showed that the independent predictors of CV death were low left ventricular ejection fraction (LVEF) and calcium channel blockers treatment, while the strongest predictors of the composite CV end-point were the Duke prognostic index score followed by LVEF and age. However, no impact of plasma adiponectin on either CV death or the composite CV end-point was detected, thus confirming von Eynatten et al.’s findings.\(^1\)

Moreover, we exploited the use of the propensity score,\(^6\) a novel statistical technique which allows to consider all potentially relevant co-variables, without the limitations of variables adjustment imposed to the multivariate models by the limited number of cardiovascular deaths and/or events observed. Hence, we could consider the complete ongoing treatment. This additional analysis fully confirmed the results obtained with the multivariate analysis, thus confirming that in high-risk patients when potential confounders are taken into due consideration adiponectin do not carry any additional prognostic information over conventional CV risk factors.

These two independent studies testify that, when assessing the usefulness of novel biomarkers of CV disease in high-risk patients, the overall risk profile, LVEF, CAD atherosclerotic burden, medical history, and concurrent medical treatment should be carefully considered before jumping to conclusions that might affect clinical practice.

**References**


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doi:10.1093/eurheartj/ehn396
Online publish-ahead-of-print 9 September 2008

**Adiponectin and prognostic outcome in patients with coronary artery disease: reply**

The letter by Cesari et al.\(^1\) refers to yet unpublished data on the association between plasma adiponectin and prognostic outcome in patients with coronary heart disease (CHD). The authors exclusively focused on total adiponectin levels and did not consider its multimeric isoforms, such as the high-molecular weight isoform. In accordance with our study,\(^2\) adiponectin levels did not yield additional predictive value for secondary cardiovascular disease events in addition to traditional cardiovascular risk factors after appropriate adjustment for covariates and consequently may not be regarded as an independent biomarker for future risk in patients with prevalent CHD. However, we like to emphasize distinct differences regarding patient selection and design of the two studies resulting in important consequences for data interpretation.

We recruited incident CHD patients out of an in-hospital rehabilitation programme only if they were admitted within 3 months after the acute event.\(^2\) As secondary prevention efforts usually start immediately after the acute event in the acute hospital, we targeted patients at the earliest stage for a possible secondary outcome follow-up. Impaired left ventricular function (LVF) is a common clinical complication in advanced CHD, substantially associated with heart morbidity and mortality. Compared with our cohort, patients in the study by Cesari et al. were presumably in more advanced stages of CHD with already impaired LVF. This may explain the increased CV death rate in their study. Furthermore, they reported a significant positive association between adiponectin and CV death, which was previously shown by other studies comprising patients with chronic heart failure (CHF).\(^3\) In an earlier work, we found also a positive association between adiponectin and a marker of heart failure.\(^4\) However, it is generally accepted that several traditional risk factors require different interpretation in patients with CHF, and high adiponectin in CHF patients could simply be a risk marker for severe complications of CHD, such as heart failure. In our cohort, the majority of patients had normal LVF, a possible reason for the lack of a positive association between adiponectin and CHD death in the study. Nevertheless, the concordant findings of Cesari et al. and our study clearly suggest that adiponectin may not emerge as a promising target for secondary risk measures, neither in incident CHD nor any later in the course of coronary atherosclerosis.

We fully agree with Cesari et al. that traditional risk factors should be targeted first and that the individual course of CHD and related complications should have a major impact on planning secondary prevention.