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Complete versus target vessel revascularization in ST-elevation myocardial infarction—analysis of results from published meta-analysis of randomized controlled trials

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We thank Weng *et al.* for their interest in our recent paper where we performed an updated meta-analysis of the benefits of non-culprit revascularization during an ST elevation myocardial infarction (STEMI) (1). The authors used trial sequence analysis (TSA) to further explore the results of our meta-analysis. The results of their TSA (2) confirmed the lower incidence of major adverse cardiac events and repeat revascularization among patients undergoing non-culprit lesion intervention. They also hypothesized that our finding of similar all-cause mortality may be a type II error (false negative) and the demonstration of lower myocardial infarction may be a type 1 error (false positive).

TSA methodology has been recently employed by researchers to combine an information size calculation for meta-analysis with the threshold of statistical significance. It is being used as a tool to quantify reliability of statistical analysis but depends on predetermined assessment of clinically significant changes, the definitions of which can somewhat arbitrary (3).

The quality of any meta-analysis depends on the heterogeneity of the included studies; the higher the heterogeneity, the greater the risk of introducing bias. In particular, in the absence of an individual patient level meta-analysis, meta-analyses including observational data cannot account for confounding inherent to the original studies. To overcome this risk of bias and to increase the strength of our meta-analysis, we included only randomized controlled trials which decreases the risk of bias since by design patients are matched at baseline in randomized trials. We included funnel plots to assess for bias in studies and this revealed minimal bias. We also performed a sensitivity analysis for outcomes of interest by excluding the study with maximum weight which did not alter the results. A meta regression was done to show the effect of follow up period on major outcomes of interest. It is also possible that random effect models may provide artificial large weights to smaller trials. To avoid this, we showed both fixed and random effect analysis for all outcomes to avoid bias from possible inappropriate weighing by random analysis.

It should be noted that the outcome of recurrent myocardial infarction included a total of six studies in our analysis but the paper by Weng *et al.* (2) showed only five studies as being included which altered certain outcomes. Also, the authors incorrectly mentioned that our article showed a false positive result for recurrent MI whereas, in our study, this was actually a negative (no difference) outcome. The variable definitions and threshold for considering a lesion as significant by either angiography or fractional flow reserve could also affect the outcomes which cannot be accounted for even by a TSA.

The clear message delivered from our meta-analysis (1) and from the author's TSA (2) is that multivessel revascularization is associated with lower major adverse cardiac events and target vessel revascularization. In general, both Weng's (2) TSA and our own meta-analysis (1) demonstrate only beneficial changes without any signal of harm when non-culprit revascularization is performed. This further increases our confidence in our results, although the magnitude of benefit in individual end points is certainly

up for debate and will be clarified in upcoming large randomized trials.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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