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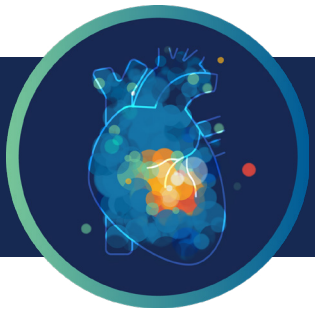
# EXPERT INSIGHTS: Q&A WITH DR. SALVATORE BRUGALETTA



**Dr. Salvatore Brugaletta, MD, PhD**
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Access the full publication: **Preventive percutaneous coronary intervention versus optimal medical therapy alone for the treatment of vulnerable atherosclerotic coronary plaques (PREVENT)**

## In plain language, how would you summarize the findings from this trial to a patient with stable atherosclerotic cardiovascular disease in your clinic?

In our clinical practice, we usually treat those coronary stenoses that are flow-limiting and possibly responsible for stable angina. This study looks forward and beyond the status quo, by evaluating the efficacy and safety of a preventive treatment of those coronary stenoses which would not usually be treated at all, because they are not flow-limiting.

The reason behind this preventive treatment is related to the particular composition of these coronary plaques, which defines them as vulnerable and as a possible source of future coronary events. In this sense, the study shows that if we treat these stenoses preventively by stent placement, future events may be prevented.

## This study adopted an open-label design, which exposes the trial to potential bias. Do you think a sham-controlled, single-blinded study design may have lent greater validity to the study's findings?

I think that the study is valuable despite its open-label design. Nevertheless, a sham-controlled and double-blinded design may have contributed to a higher validity to the study's findings. In particular I think a double-blinded design would have been of value, as doctors who will be making the follow-up may be influenced in their medical treatment choice by whether a stent was implanted or not. Such a design, however, would have been difficult to perform.

**Given that just 6-7% of the randomized cohort had thin-cap fibroatheroma (TCFA) noted on optical coherence tomography or radiofrequency intravascular ultrasound, do you think the vulnerable coronary plaques treated in the PREVENT trial were "vulnerable" enough?**

First of all, I am puzzled by how a radiofrequency intravascular ultrasound may identify a thin cap, as the resolution of this imaging technique is above the thickness we want to identify. In any case, to reliably and reproducibly identify a vulnerable plaque is in itself absolutely difficult. Although we may assume that the TCFA identified in the study were indeed vulnerable, there could be many other plaques not vulnerable at the time of the study inclusion but which then become vulnerable over time. The biology underpinning a vulnerable plaque is indeed very variable.

**One could argue the results of this study are not generalizable enough given the recruitment of subjects restricted to the Asia-Pacific region, relatively low female representation and the early use of bioresorbable vascular scaffolds for stenting of vulnerable plaques. What further evidence is required in your view, before preventive percutaneous coronary intervention (PCI) of vulnerable atherosclerotic plaque could become prime time?**

I agree, that is indeed a possibility. However, we are lucky as there are at least two other studies currently ongoing: COMBINE-INTERVENE (NCT05333068) and VULNERABLE (NCT05599061) testing the same hypothesis. So in 3 years we will have more evidence in this field.

**Given the emergence of PCSK9-targeted therapies, encouraging results from trials of a novel CETP inhibitor, large scale cardiovascular outcomes trials of lipoprotein(a) lowering and guideline-directed management of coronary inflammation, do you think there will be a place for preventive PCI of vulnerable plaque in the future?**

This is a very interesting question. Results from all the trials coming in this field will tell us if there is a place for preventive PCI of vulnerable plaque. In my opinion preventive PCI should be evaluated on top of maximum and best-in-class treatment, as preventive PCI is stabilizing a short coronary plaque, whereas medical treatment is stabilizing the whole coronary tree.



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