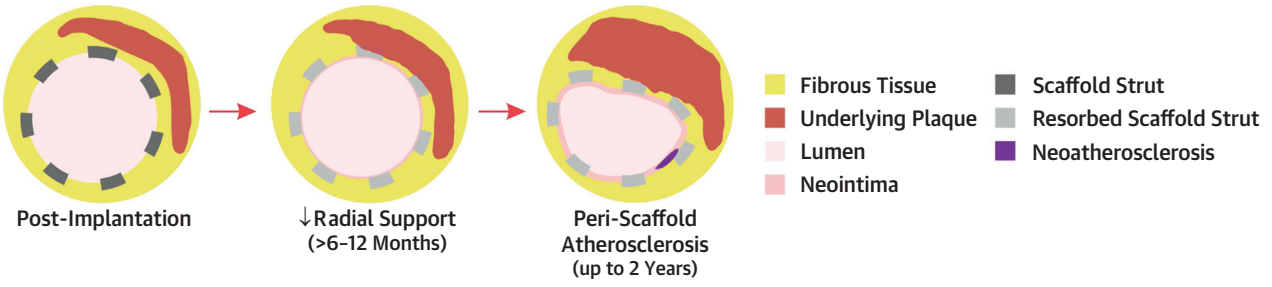
JACC VOL. 72, NO. 9, 2018
AUGUST 28, 2018:1060–7

FIGURE 1 Assessment of Atherosclerosis Development Around a Bioresorbable Scaffold



Following loss of the scaffold mechanical support during bioresorption, native atherosclerotic plaque progression might result in inward strut dislocation. In-scaffold neoatherosclerosis development would be seen inside the struts. These changes could be observed during a timeframe when strut remnants are still visible with intravascular imaging.

Bioresorbable scaffolds could serve as an ideal means to provide valuable insights into the mechanisms of periscaffold atherosclerosis development owing to the decreasing radial strength during bioresorption and the black box-like appearance without dorsal shadowing of the optically translucent polymeric struts. That would be interesting if the scaffolded segments could be serially assessed during an earlier timeframe during the bioresorption process when the radial strength of the scaffold has been lost, but the strut remnants are still visible (which could be a period from 6 months up to 2 years post-implantation) and may move freely due to neighboring mechanical forces (e.g., plaque progression or vessel remodeling). As the mechanical integrity of the degrading scaffold diminishes over time, native atherosclerotic plaque ingrowth may lead to dislocation of the strut remnants by pushing them toward the lumen (**Figure 1**). Accordingly, prospective serial optical coherence tomography assessments of bioresorbable scaffolds, combined with intravascular ultrasound to account for potential negative vessel remodeling, with meticulous observation of the strut footprints are warranted.

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https://doi.org/10.1016/j.jacc.2018.05.069

Please note: Both authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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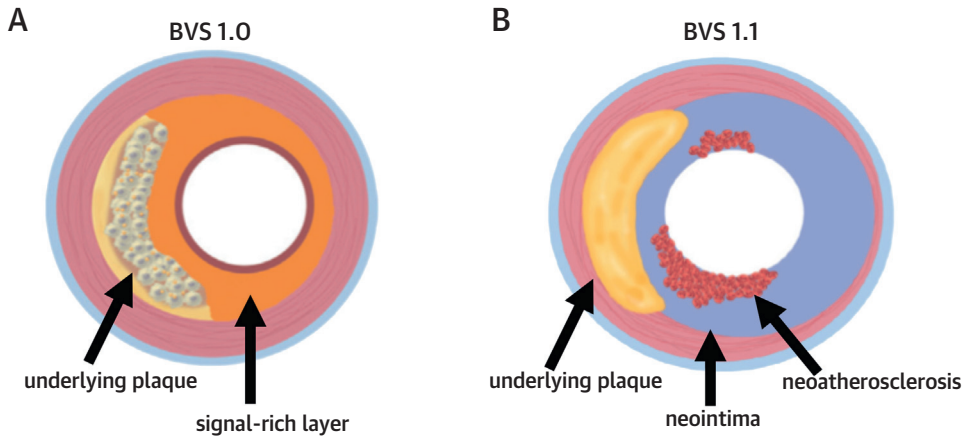
REPLY: In-Scaffold Neoatherosclerosis

An Overestimated Entity?

We appreciate Drs. Andreou and Alexopoulos for their valuable comment on the study "Neoatherosclerosis 5 Years After Bioresorbable Vascular Scaffold Implantation" (1). They commented on the possible mechanism that native atherosclerotic plaque ingrowth behind the scaffold may lead to dislocation of the strut remnants by pushing them toward the lumen, which might explain the incidence of in-scaffold atherosclerosis. The term "neoatherosclerosis" has been adopted on the hypothesis that atherosclerosis within the stent/scaffold does not communicate with the underlying native atherosclerosis. Our results cannot make a denial of their theory because our analysis was based on this hypothesis of neoatherosclerosis, and we did not focus on underlying plaque assessment. Therefore, we agree that underlying atherosclerotic plaque may play a role of in-scaffold atherosclerosis growth (2). We also concede that the bioresorbable scaffold is a suitable device to prove their theory by using serial optical coherence tomography (OCT) assessment.

FIGURE 1 Discrepancy Between BVS 1.0 and 1.1



Karanasos, A. et al. J Am Coll Cardiol. 2014

Moriyama, N. et al. J Am Coll Cardiol. 2018

(A) Signal-rich, low-attenuating tissue layer that covered thrombogenic plaque components at 5 years after BVS 1.0 implantation. Reprinted with permission from Karanasos et al. (3). **(B)** Atherosclerotic intima within in-scaffold segments at 5 years after BVS 1.1 implantation. Reprinted with permission from Moriyama et al. (1). BVS = bioresorbable vascular scaffold.

In the current study, OCT assessment of bioresorbable vascular scaffold (BVS) 1.1 supported that atherosclerotic changes with luminal narrowing occurs only within in-scaffold segments, but not in out-scaffold segments. By contrast, Karanasos et al. (3) reported a good tissue response with late liminal enlargement and development of a healthy tissue layer that covered thrombogenic plaque components after BVS 1.0 implantation. The fundamental difference between BVS 1.1 and BVS 1.0 is nothing but the mechanical radial force of BVS. Therefore, their theory that intimal atherosclerosis comes from underlying plaque could not fully explain this obvious discrepancy between 2 intergenerational scaffolds (**Figure 1**). The resorption rate and geometry of BVS 1.0 differs from those in BVS 1.1 due to divergence of radial force (4). This can have the potential to explain the distinct healing responses. Hence, BVS 1.1 itself may be associated with in-scaffold atherosclerosis at 5 years. Accordingly, larger studies including early-phase serial imaging assessment are warranted to confirm where in-scaffold atherosclerosis comes from.

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https://doi.org/10.1016/j.jacc.2018.06.039

Please note: The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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Left Ventricular Ejection Fraction and Global Longitudinal Strain

Prognostic When Not Load Dependent?

In their recent publication, Park et al. (1) demonstrate that global longitudinal strain (GLS) had strong