theme that can be derived from our study and the study by Bikdeli et al. (2) is that the burden of infective endocarditis hospitalization rates in the United States is high and rising, which ultimately confers to higher health care expenditure and morbidity. Hence, a multispecialty collaborative effort is needed to understand the factors responsible and identify the strategies to halt this rising trend. We believe that the ongoing monitoring of the impact of the prophylaxis guidelines and appropriate updates on the basis of such data is an essential step in this regard.

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http://dx.doi.org/10.1016/j.jacc.2015.07.071

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

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How to Control Residual Risk During the Statin Era?

The Schwartz et al. (1) paper reported that among patients with acute coronary syndrome treated effectively with statins, fasting triglycerides (Tg) predict long-term and short-term cardiovascular risk. Triglyceride-rich lipoproteins may be an important additional target for therapy (1).

Lowering low-density lipoprotein cholesterol (LDL-C) is the primary target in the management of dyslipidemia in patients at high risk of cardiovascular disease. However, patients who have achieved LDL-C levels below the currently recommended targets may still experience cardiovascular events. This may result, in part, from elevated Tg levels (2). Atherogenic dyslipidemia, characterized by high Tg, low levels of high-density lipoprotein cholesterol (HDL-C), and small, dense LDL particles, is a typical phenotype of dyslipidemia in subjects with insulin resistance and metabolic syndrome. On the other hand, raised Tg concentrations are strongly associated with low concentrations of HDL-C, and the past 15 years have been dominated by HDL research, with less focus on Tg. However, the understanding from genetic studies and randomized trials that low HDL-C might not be a cause of cardiovascular disease as originally thought has generated renewed interest in raised Tg (3). Indeed, a study investigated the causal role of HDL-C and Tg using multiple instrumental variables for Mendelian randomization and reported that the genetic findings supported a causal effect of Tg on coronary heart disease risk (4).

It is obvious that statins are the first-line drug for the treatment of dyslipidemia. However, a strategy to reduce high Tg and modify the small, dense LDL particles is required. In this regard, statin-based combined with fibrates, peroxisome proliferator-activated receptor agonists may be recommended in addition to therapeutic lifestyle changes if patients still experience cardiovascular events. The cost effectiveness of these combinations should also be evaluated (5).

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http://dx.doi.org/10.1016/j.jacc.2015.07.072

Please note: Dr. Koh has reported that he has no relationships relevant to the contents of this paper to disclose.

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