Reply to “Cardiovascular risk of tenofovir disoproxil fumarate or tenofovir alafenamide fumarate in HBV patients”

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Authors’ contribution
PN Cheng drafted the manuscript. ML Yu reviewed and finalized the manuscript.

Conflicts of Interest
The authors have no conflicts to disclose.
We are grateful for the comments [1] from Prof. Hong and Prof. Choi to our Editorial [2]. The issue of risk of cardiovascular disease (CVD) of tenofovir diproxil fumarate (TDF) and tenofovir alafenamide (TAF) treatment is based on the unexpected changes of lipid profiles in chronic hepatitis B (CHB). Current literature revealed controversial results between TAF treatment and lipid changes [3,4]. In contrast, lipid-lowering effects of TDF presented with lower levels of total cholesterol, triglyceride, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol are well established [5,6]. Our previous study also showed that lower lipid profiles was observed in TDF-treated patients when compared with entecavir-treated patients [6]. How TDF works on lipid profiles is still unclear. Recently, we examine the lipid loading capacity of cholesterol and triglyceride in each very low-density lipoprotein (VLDL) and LDL-C particle [7] in CHB patients treated with TDF or entecavir. Comparing with entecavir-treated patients, reduced level of apolipoprotein B100 (apo-B) in LDL particle but similar apo-B level in VLDL particle were observed in TDF-treated patients (unpublished data). The results indicate that TDF treatment could enhance hepatic uptake of LDL by LDL receptor during the metabolized pathway of VLDL to LDL and subsequently leads to lower lipid profiles. However, how this feature works on lipids and the association long-term risk of CVD are still elusive. Finally, we totally agree that well-designed and prospective studies address the association of risk of CVD and long-term use of TDF or TAF are strongly require to validate the findings from Prof. Hong and Prof. Choi [8].
Reference


