well-tolerated drug as regards myopathy in our study on Indian patients."[1] I have some comments on this work. First, the number of subjects in this study is very small and might have a limitation in further interpretation Second, the actual mechanism of zidovudine myopathy is believed to be due to a zidovudine-induced mitochondrial DNA depletion.[2] The corresponding mechanisms include azidothymidine (AZT) -induced oxidative stress, direct inhibition of mitochondrial bioenergetic machinery, and mitochondrial depletion of L-carnitine.[3] Based on these mechanisms, there should be no race difference of this zidovudine adverse effect. The higher prevalence in the Western population might be due to the reason that is given by Authier *et al*., that "lengthening of the survival of HIV-infected individuals has been associated with an increasing prevalence of iatrogenic conditions".[4]

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Zidovudine-induced myopathy

Sir,

I read the recent publication on zidovudine-induced myopathy with a great interest.[1] Sagar *et al*., concluded that "Zidovudine myopathy may be a constraint for use of the drug in the Western population; however, it is a