

# Letters

## COMMENT & RESPONSE

### Cardiovascular Risk Prediction in Patients With Human Immunodeficiency Virus

**To the Editor** We read with interest the article by Feinstein et al<sup>1</sup> in the February issue of *JAMA Cardiology*. Despite the well-known increased risk of cardiovascular disease in patients with human immunodeficiency virus (HIV), there do not seem to be good prediction models to accurately estimate risk. Feinstein et al<sup>1</sup> noted that the accuracy of the prediction models may be particularly low in women and Hispanic individuals and that the equations may work best in patients at moderate to high risk. Furthermore, there was no evidence that adding HIV-specific risk factors to algorithms based on traditional risk factors, such as the Pooled Equations, increased the performance of these models.

These results are remarkably similar to the findings we published in 2016 in a European longitudinal cohort of 2550 white patients with HIV.<sup>2</sup> Among 2550 patients with HIV (median age, 46.5 years; 34% female), we reported a 3.98 per 1000 patient-years hard cardiovascular event rate, while Feinstein et al<sup>1</sup> reported an event rate of 4.4 per 1000 patient-years. Like Feinstein et al,<sup>1</sup> we recorded mostly myocardial infarctions (MIs) and reported a similar and fairly low accuracy of traditional or HIV-specific prediction algorithms. Feinstein et al<sup>1</sup> suggested that consideration should be given to lowering the risk thresholds to capture more events in the lower-risk groups. However, even this approach was shown to be ineffective in our publication.<sup>2</sup> We submit that the size of the populations studied was different, with several more events recorded by Feinstein et al,<sup>1</sup> but the results were the same.

Of note, another recent publication by Crane et al<sup>3</sup> highlighted an important difference in the type of MIs recorded in the general population and patients with HIV; while only about a quarter of MIs are type 2 in the general population, over half of the MIs recorded in patients with HIV are type 2. This type of MI has a very different pathogenesis from type 1 MI, and often, plaque rupture and intravascular acute thrombosis are not

the mechanisms subtending the events but rather, for example, sepsis, vasospasm due to cocaine use, anemia, and acute volume fluctuations. In the study by Crane et al,<sup>3</sup> these events occurred more frequently in women and Hispanic individuals where the Pooled Equations appear to underestimate cardiovascular events. Hence, it may be inappropriate to use algorithms that were calibrated to predict an atherosclerotic event in the general population to estimate the risk of events that in more than half of the cases are nonatherosclerotic in patients with HIV. Future studies may need to address the calibration of various algorithms to separate the risk of type 1 MI from type 2 MI.

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1. Feinstein MJ, Nance RM, Drozd DR, et al. Assessing and refining myocardial infarction risk estimation among patients with human immunodeficiency virus: a study by the centers for AIDS Research Network of Integrated Clinical Systems. *JAMA Cardiol*. 2017;2(2):155-162.
2. Raggi P, De Francesco D, Manicardi M, et al. Prediction of hard cardiovascular events in HIV patients. *J Antimicrob Chemother*. 2016;71(12):3515-3518.
3. Crane HM, Paramsothy P, Drozd DR, et al; Centers for AIDS Research Network of Integrated Clinical Systems (CNICS) Cohort. Types of myocardial infarction among human immunodeficiency virus-infected individuals in the United States. *JAMA Cardiol*. 2017;2(3):260-267.