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## Authors' Reply to Comments on “Cardiovascular Risk Profile of Transgender Women with HIV: A U.S. Healthcare Database Study”

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### Keywords

HIV; transgender; women; cardiovascular disease risk

### Authors' Reply:

We appreciate the comments by Streed et al. on our research letter “Cardiovascular Risk Profile of Transgender Women with HIV.” In our letter, we presented data on cardiovascular disease risk factors among transgender women with HIV identified through the Partners HIV Cohort (n=23). Data points were extracted through retrospective review of electronic health records (EHR).

We agree with Streed et al. that among transgender women with HIV, history of hormonal therapies would be expected to influence the CVD risk factor profile. Several factors precluded us from disentangling hormonal-CVD risk factor relationships among our studied cohort of transgender women with HIV. Such factors include a relatively small overall sample size (single health network system), heterogeneity of documented hormonal therapies, and inadequate EHR capture of non-prescribed hormonal therapies in this group.

We further agree with Streed et al. that insights furnished through *comparison* of CVD risk factor profiles hinge on the selected comparator group. Our comparator group was developed from within the Partners HIV Cohort matched to the group of transgender women on birth-assigned sex, age, and race (n=92). As noted, we selected cisgender men as our comparator group to address the following specific question: For people with HIV (PHIV) with male nascent sex, how does gender identification influence the CVD risk factor profile?

Comments by Streed et al. highlight the need for further research focused on CVD risk among transgender women with HIV. Such work, particularly if prospective in nature,

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should be attentive to history of prescribed and non-prescribed hormonal therapies. Moreover, through inclusion of additional comparator groups (e.g. cisgender women with HIV, transgender women without HIV), such work will help address distinct, important questions.

Finally, Streed et al. question the relevance of our primary finding that hemoglobin levels are lower among transgender women with HIV than among cisgender men with HIV. Streed et al. suggest that among transgender women receiving gender-affirming hormonal therapy, hemoglobin levels should be compared to those of cisgender women. Like Streed et al., we recognize that suppression of endogenous androgen production is *anticipated* to result in reduced erythropoiesis and attendant dampening of circulating hemoglobin levels. However, we posit that there remains room for study on how reduced hemoglobin levels influence health and disease among transgender women with HIV. Of note, among PHIV, anemia has been independently associated with acute myocardial infarction<sup>1</sup>. Further, select investigations have suggested a graded relationship between hemoglobin levels and mortality among PHIV, potentially contributing to sex-differences in mortality in this group<sup>2</sup>. For the broader population of transgender women, Endocrine Society guidelines recommend monitoring of select cardiometabolic parameters expected to be influenced by gender-affirming hormonal therapy – e.g. lipid profile<sup>3</sup>. For transgender women with HIV, additional attention to changes in hemoglobin potentially induced by anti-androgen medical and/or surgical therapy may be warranted. This type of attention would acknowledge previously established relationships between red blood cell indices and morbidity/mortality in HIV<sup>12</sup>.

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