We thank Moulton et al. for the valuable comments. We carefully considered practice effects when designing our study, and improvements in cognitive function cannot be attributed to retest effects alone.

First, retest effects are attributed to the content of neuropsychological tests and the context of administration. In this study, cognitive testing was 1 of 14 tests conducted in an hour-long assessment, making it especially difficult for patients to recall or learn content through repeated testing.

Second, the largest retest effects typically occur between the first two assessment cycles (at kidney transplantation [KT] and 1-month post-KT) and dissipate thereafter. In a postpublication sensitivity analysis, we refined trajectories in more short-term intervals: knots at 1-month, 3-months, 6-months, and 1-year post-KT. We observed improvements between KT and 1-month in both frail (slope = 0.39 points/wk) and nonfrail recipients (slope = 0.30 points/wk), which may represent a combination of retest effects and KT recovery effects. However, between 3- and 6-months post-KT, we saw continued substantial improvement in cognitive function among frail (slope = 0.32 points/wk) and plateaued trajectories among nonfrail recipients in spite of dissipating retest effects. This supports our original inferences that both groups improved in the short-term due in large part to recovery of kidney function.

Third, based on prior studies, retest effects may not differ by group demographics and factors, and thus are unlikely to differ by frailty.

Furthermore, we made the critical decision not to adjust for depression for two reasons: (1) the frailty phenotype incorporates exhaustion, measured using two items from the Center for Epidemiologic Studies Depression Scale (CES-D), and (2) we believe depression to be a potential mediator on the causal pathway. As Moulten et al. suggested, depression is common and related to frailty in KT. Based on the frailty phenotype model, the underlying process of multisystem dysfunction catalyzes depressive symptoms, potentially leading to clinical depression.

Depressive symptoms were also linked to cognitive dysfunction in KT; studies are needed to assess the temporality of this association. As such, we strongly believe depression may be one pathway linking frailty and cognitive decline in KT.

Nevertheless, we excluded recipients with severe symptoms (CES-D ≥ 16) at KT (n = 74, 11.5%) in a postpublication sensitivity analysis. We found inferences remained robust: nonfrail (slope = +0.14 points/wk) and frail (slope = +0.18 points/wk) recipients experienced improvements by 3-months post-KT. After 1-year, frail recipients experienced significant declines in cognitive function (slope = −0.03 points/wk), while nonfrail recipients plateaued (slope = 0.01 points/wk).

Given the aforementioned arguments, we believe the limitations brought forth by Moulten et al. have little, if any, impact on previously published inferences.

DISCLOSURES

None.

FUNDING

This study was supported by the National Institute on Aging (grants: F32AG053025 K01AG043501 K01AG050699 P30AG021334 P50AG00514 R01AG042504 R01AG055781 T32AG000247) and National Institute of Diabetes and Digestive and Kidney Diseases (grants: F30DK116658 K24DK101828 R01DK114074).

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