**Supplemental Material**

**Sensitivity Analyses**

 *Alternate model.*Considering that high perceived stress could be both a cause and symptom of depressive symptoms, we tested an alternative model in which perceived stress was modeled as a mediator of the association between depressive symptoms and memory. While the negative direct association between depressive symptoms and memory remained the same, there was no indirect association between depressive symptoms and memory via perceived stress (completely standardized indirect effect = -0.02, SE= 0.01 95% CI [-0.05, 0.01]).

 *Memory Type.* Associations among perceived stress, depressive symptoms, and memory may differ across memory task types (e.g., recall versus recognition), thus the above analyses were repeated with each of the three individual components of the SRT (initial learning, delayed recall, and recognition) as separate memory outcomes. Effects of perceived stress, both directly and indirectly through depressive symptoms, were the same as the original analyses for initial learning and delayed recall, but not recognition (completely standardized indirect effect = -0.02, SE= 0.02, 95% CI [-0.05, 0.01]). Specifically, there was no association between depressive symptoms and recognition memory (β = -0.05, SE= 0.03, *p*= .21), nor a total effect of perceived stress on recognition (β = -0.02, SE= 0.05, *p*= .56).

 *MCI.*While participants did not meet clinical criteria for dementia (see Richard et. al, 2013 for details on the diagnosis protocol in WHICAP), they comprised a broad range of cognitive abilities, including mild cognitive impairment (see Manly et al., 2005 for operationalization criteria).Prior research has shown that associations between stress and cognition may differ among cognitively healthy older adults and those with some impairment (Peavy et al., 2009), thus, a separate sensitivity analysis excluding individuals with mild cognitive impairment was conducted (model n= 465). Results of this mediation model did not differ from the original findings (completely standardized indirect effect = -0.03, SE= 0.01, 95% CI [-0.05, -0.003]).

 *Income.*Of the many indicators of socioeconomic status, education is the most strongly associated with cognition (Lee, Kawachi, Berkman, & Grodstein, 2003) and was thus included in the primary analytic models. A separate sensitivity analysis covarying for monthly household income as an additional marker of socioeconomic status led to a drop in the overall sample due to missing data on income (model n= 538). Although the total effect of perceived stress on episodic memory and the association between perceived stress and depressive symptoms did not change from the original model, the relationship between depressive symptoms and episodic memory did not remain (β = -0.07, SE= 0.02, *p*= .07). Thus, the indirect effect of stress on memory was no longer statistically significant (completely standardized indirect effect = -0.02, SE= 0.01 95% CI [-0.05, 0.00]).

 *Health conditions.* While vascular diseases have previously been shown to have the strongest associations with late life cognitive impairment (Snyder et al., 2015), a sensitivity analysis expanding the physical health covariate to account for the presence of 12 additional health conditions (stroke, arthritis, chronic obstructive pulmonary disease, thyroid disease, liver disease, renal disease, ulcer, peripheral vascular disease, cancer, Parkinson's disease, multiple sclerosis, and essential tremor) was conducted. Results were virtually identical to the original mediation model (completely standardized indirect effect = -0.03, SE= 0.01, 95% CI [-0.05, -0.01]).

 *Time between assessments.* WHICAP participants often completed the ancillary psychosocial battery and core WHICAP tests (e.g., episodic memory) on different dates. Thus, number of days between measurement of predictor and exposure variables was added as a covariate. Addition of this covariate did not alter the original pattern of findings (completely standardized indirect effect = -0.03, SE= 0.01, 95% CI [-0.06, -0.01]).

References

Lee, S., Kawachi, I., Berkman, L. F., & Grodstein, F. (2003). Education, Other Socioeconomic Indicators, and Cognitive Function. *American Journal of Epidemiology*, *157*(8), 712–720. https://doi.org/10.1093/aje/kwg042

Manly, J. J., Bell-McGinty, S., Tang, M.-X., Schupf, N., Stern, Y., & Mayeux, R. (2005). Implementing Diagnostic Criteria and Estimating Frequency of Mild Cognitive Impairment in an Urban Community. *Archives of Neurology*, *62*(11), 1739. https://doi.org/10.1001/archneur.62.11.1739

Peavy, G. M., Salmon, D. P., Jacobson, M. W., Hervey, A., Gamst, A. C., Wolfson, T., … Galasko, D. (2009). Effects of Chronic Stress on Memory Decline in Cognitively Normal and Mildly Impaired Older Adults. *American Journal of Psychiatry*, *166*(12), 1384–1391. https://doi.org/10.1176/appi.ajp.2009.09040461

Snyder, H. M., Corriveau, R. A., Craft, S., Faber, J. E., Greenberg, S. M., Knopman, D., … Carrillo, M. C. (2015, June 1). Vascular contributions to cognitive impairment and dementia including Alzheimer’s disease. *Alzheimer’s and Dementia*. Elsevier Inc. https://doi.org/10.1016/j.jalz.2014.10.008