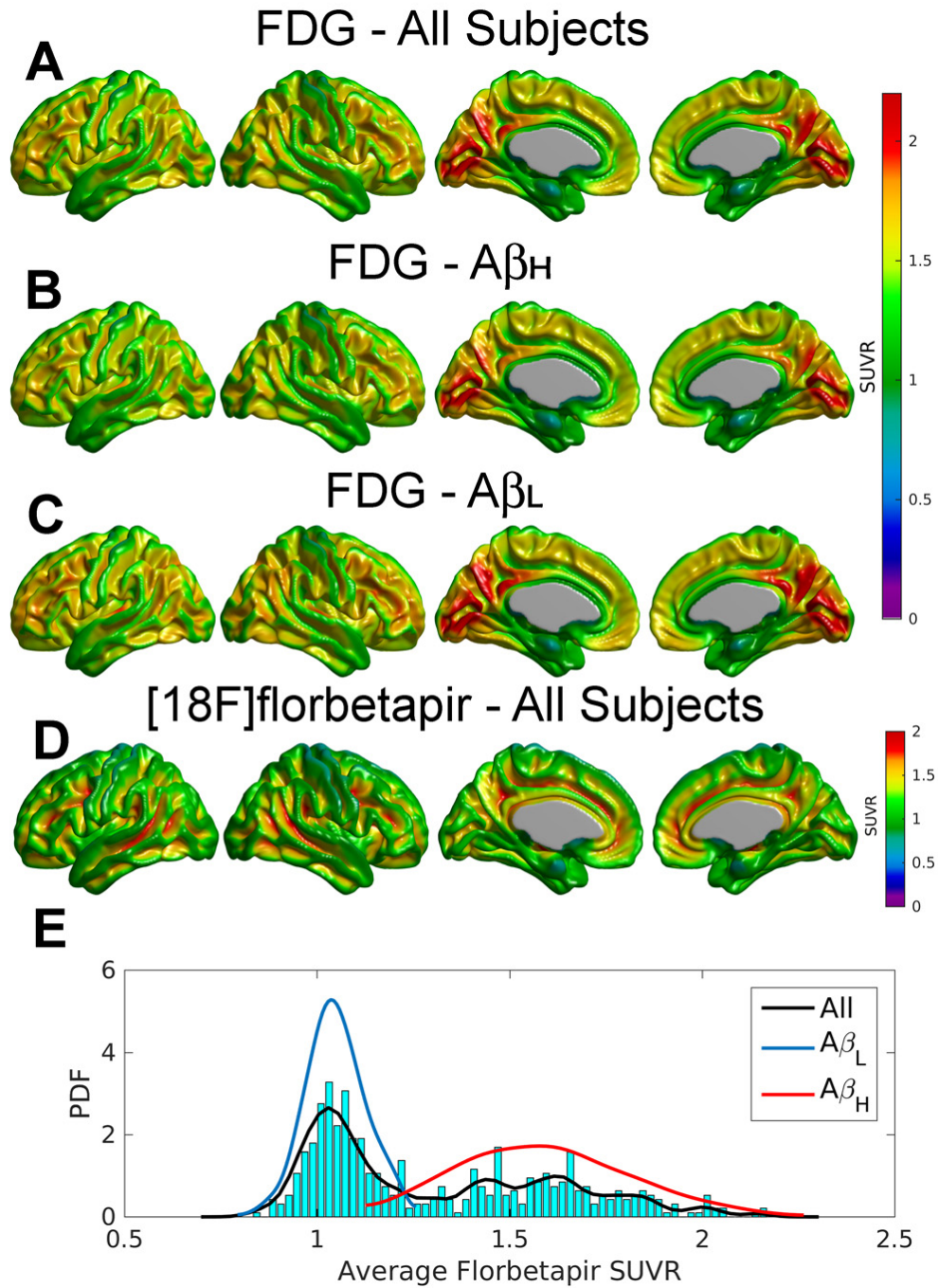
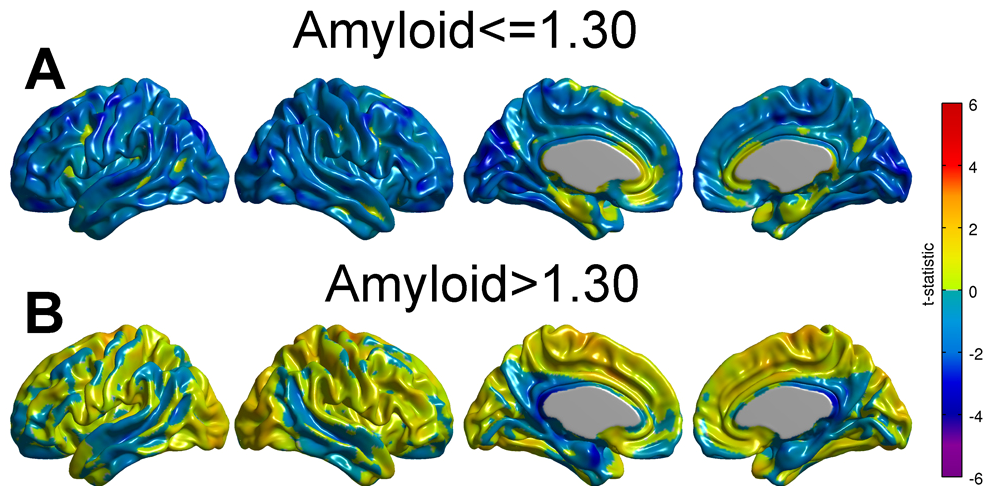


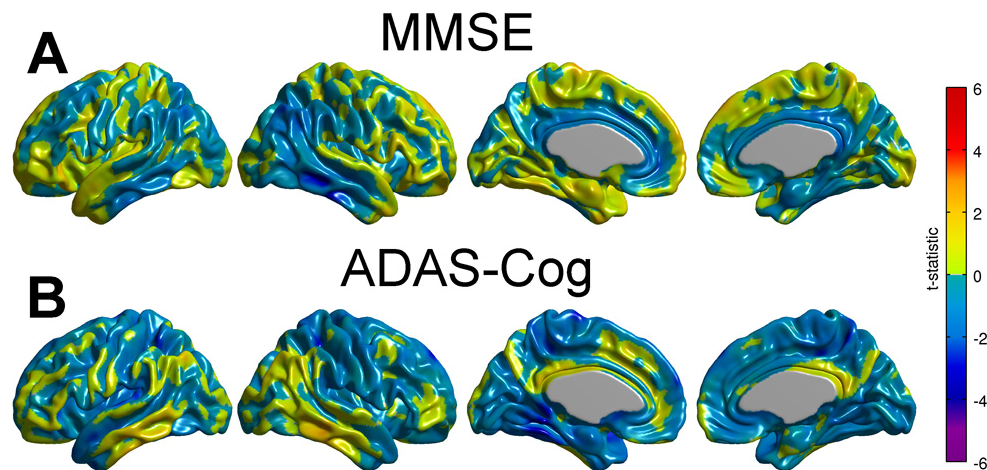
## Supplementary Figures



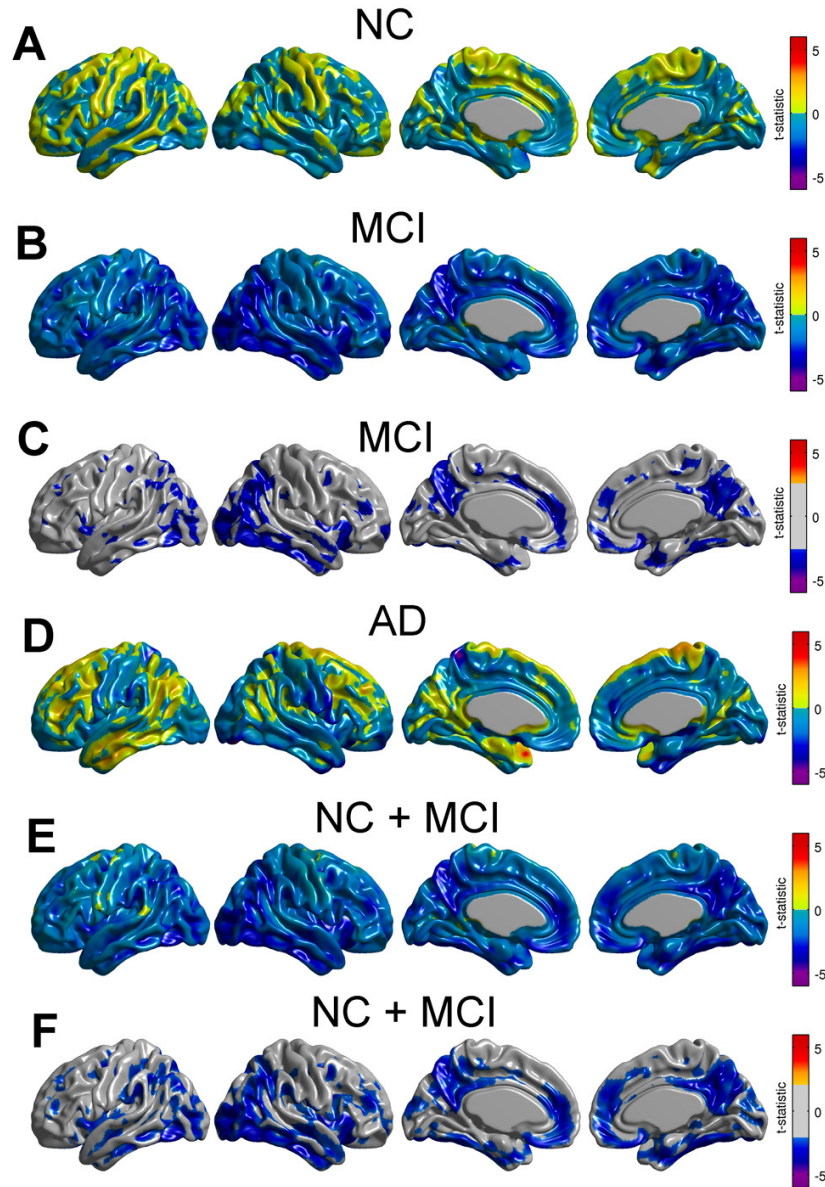
**Supplementary Figure 1.** (A, B, C) Average [18]FDG SUVR maps for all subjects,  $A\beta_H$ , and  $A\beta_L$  groups, respectively. (D) Average [18]florbetapir SUVR map for all subjects. (E) Distribution of the [18]florbetapir SUVR<sub>ROI</sub> across the whole sample, as well as estimation of the probability distribution functions (PDFs) for the  $A\beta_L$  and  $A\beta_H$  groups.



**Supplementary Figure 2.** Statistical significance of the relationship between  $\beta$ -amyloid burden and glucose metabolism for Amyloid  $\leq 1.30$  and Amyloid  $> 1.30$ , respectively. The SUVR cutoff value of 1.30 shows two different patterns.



**Supplementary Figure 3.** Relationship between glucose metabolism and  $\beta$ -amyloid burden modulated by MMSE and ADAD-Cog. Multiple comparisons thresholding of these t-statistic parametric maps using FDR at 0.05 did not show any regions of statistical significance that would support a strong modulation effect.



**Supplementary Figure 4.** Significance of APOE  $\epsilon 4$  status on metabolism corresponding to NC, MCI, AD subjects and combined NC/MCI cohort. Results (A, B, D, and E) are shown without thresholding. A statistically-significant main effect of genotype was only found for MCI subjects (B,C) and the NC/MCI cohort (E,F). Results (C) and (F) are shown at an FDR-corrected threshold of 0.05.

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