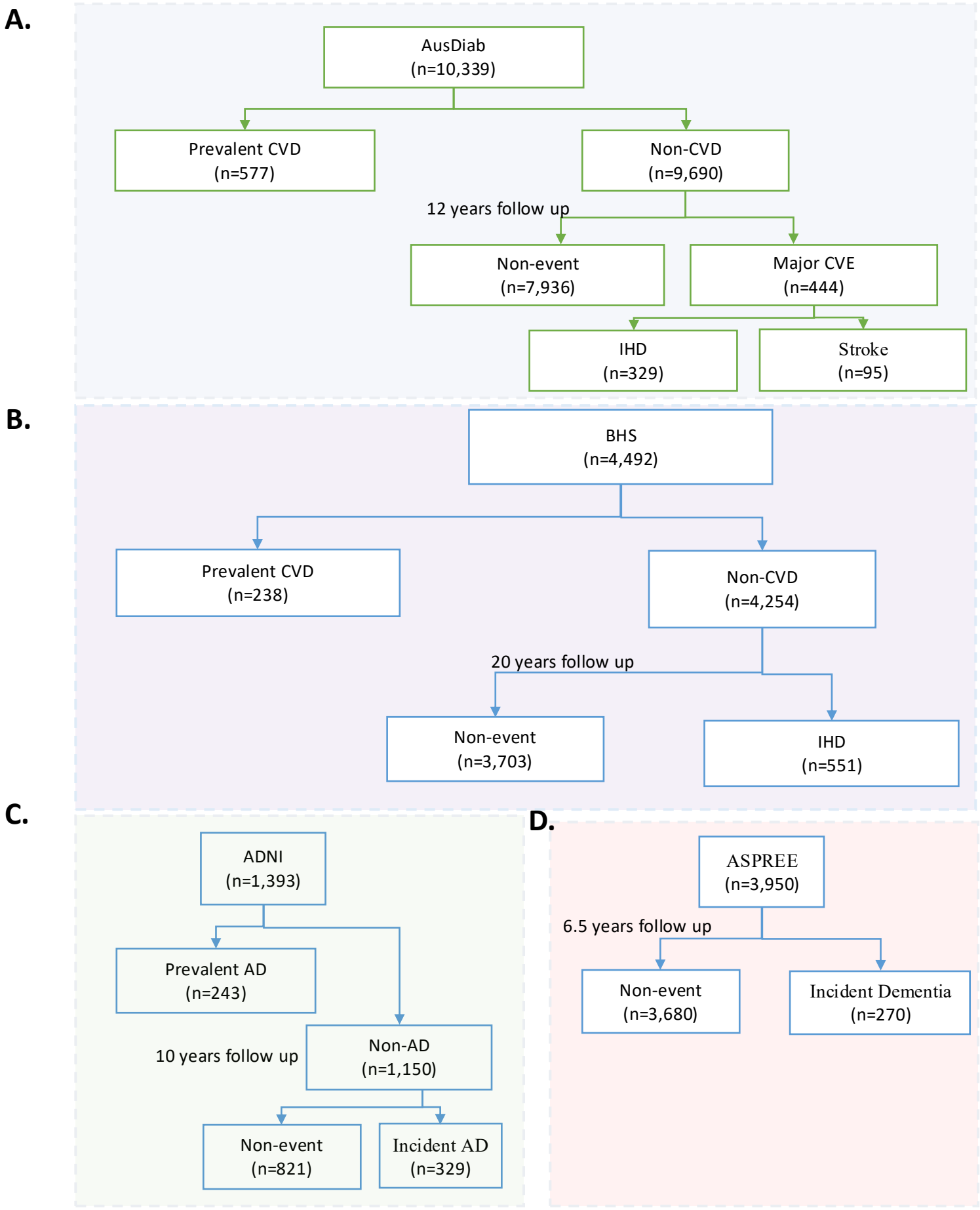
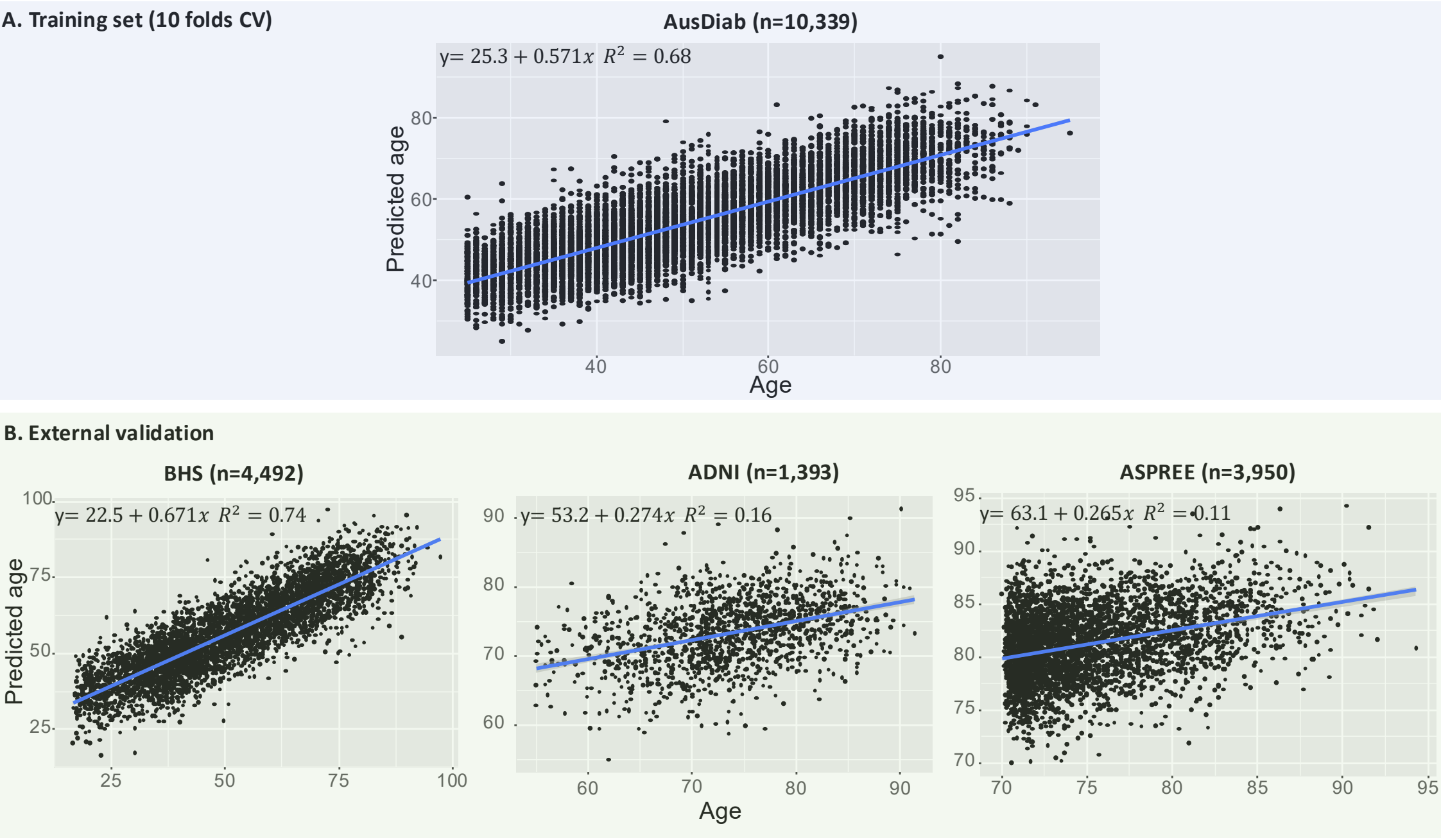
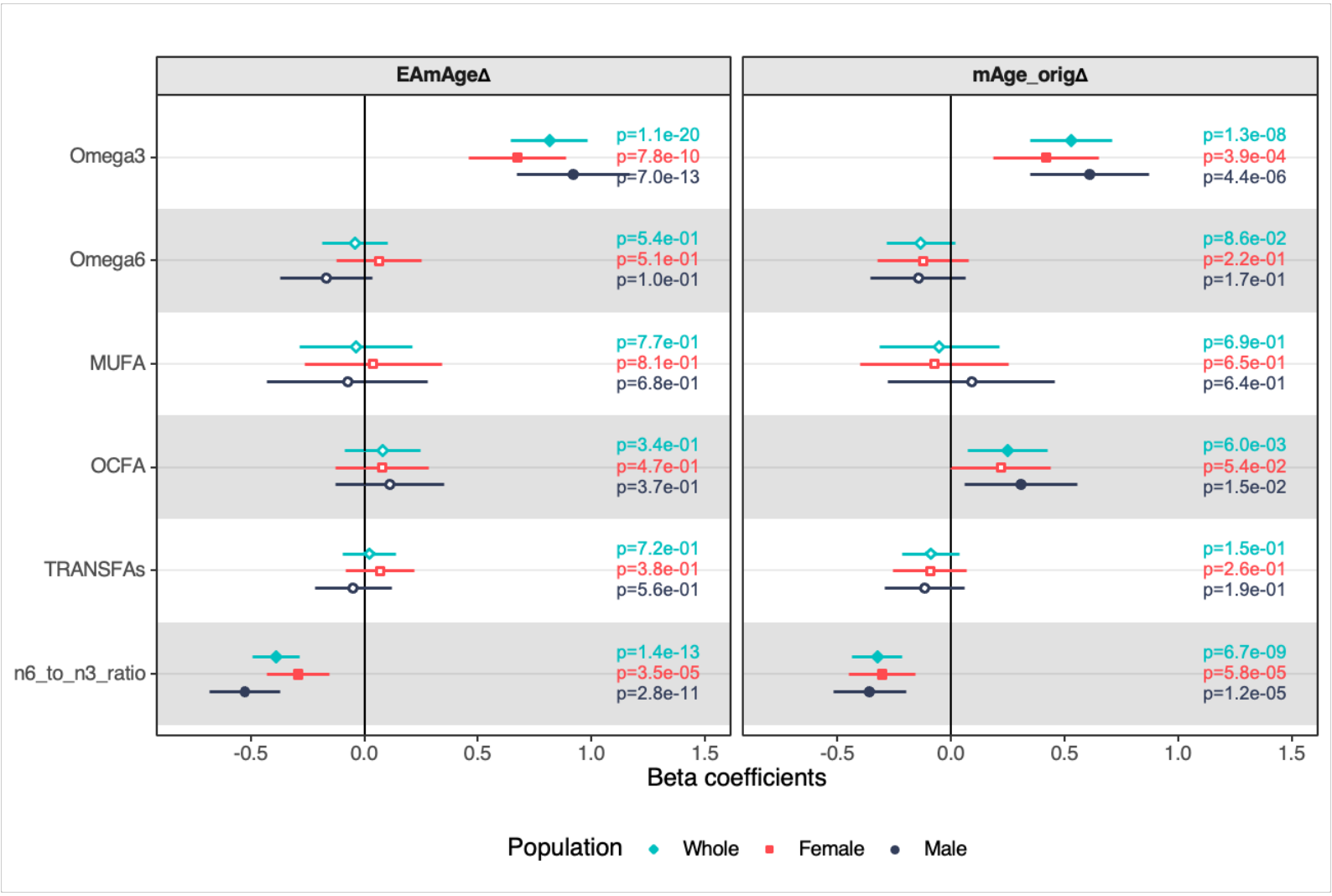


Supplementary Figure 1. Details of clinical outcomes of the AusDiab (A), BHS (B), ADNI (C), and ASPREE (D) cohorts.

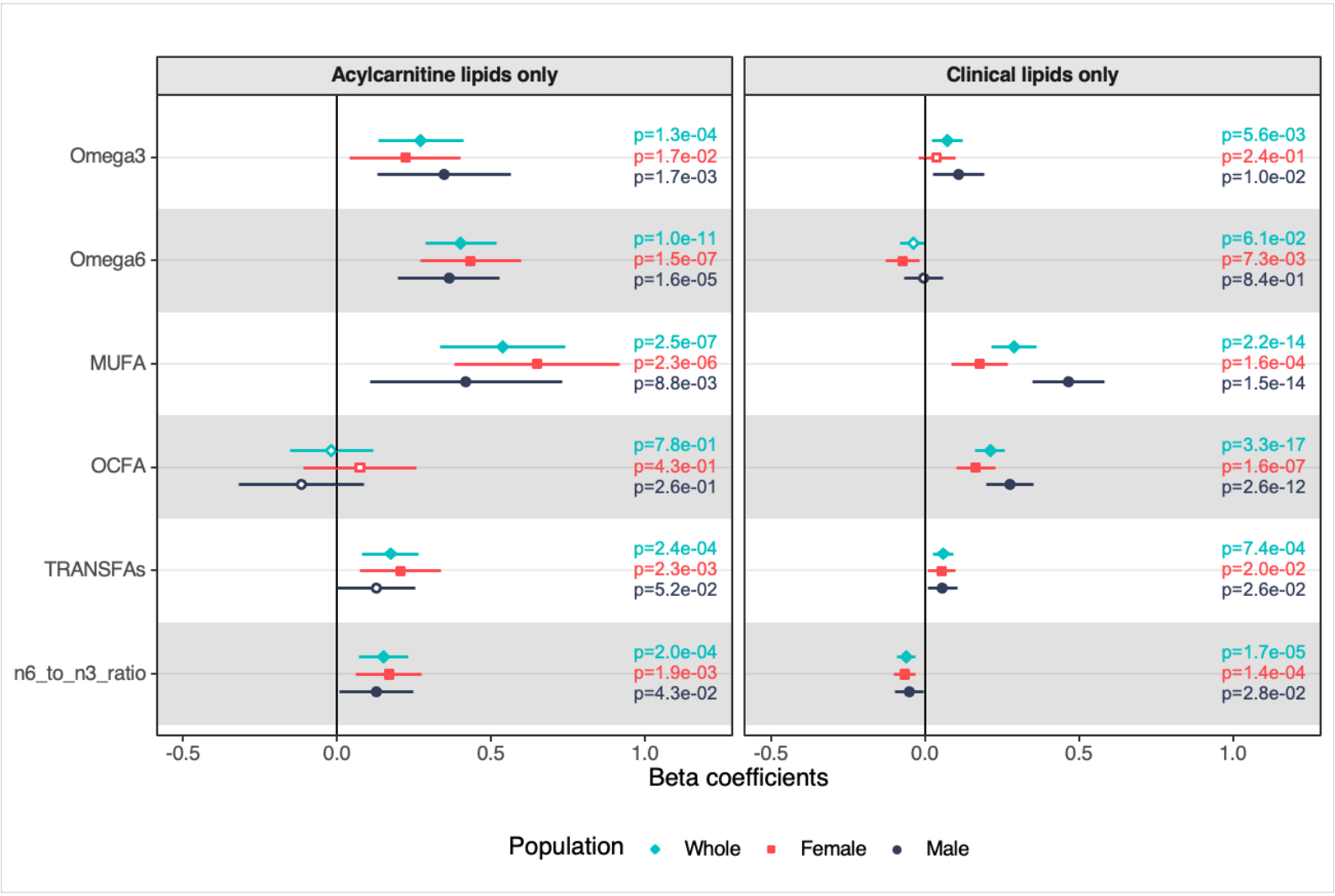


Supplementary Figure 2. The prediction performance of metabolic age modelling (ridge) in AusDiab, BHS, ADNI, and ASPREE.
Ridge regression model was built on AusDiab (n=10,339) under 10 folds cross validation (A). Further, the model built on the full AusDiab data set was externally validated on BHS (n=4,492; B). Additionally, the model built on AusDiab (age≥60; n=2,608) was validated on two elderly clinical populations - ADNI (N=1,393; C) and ASPREE (N=3,950; D).

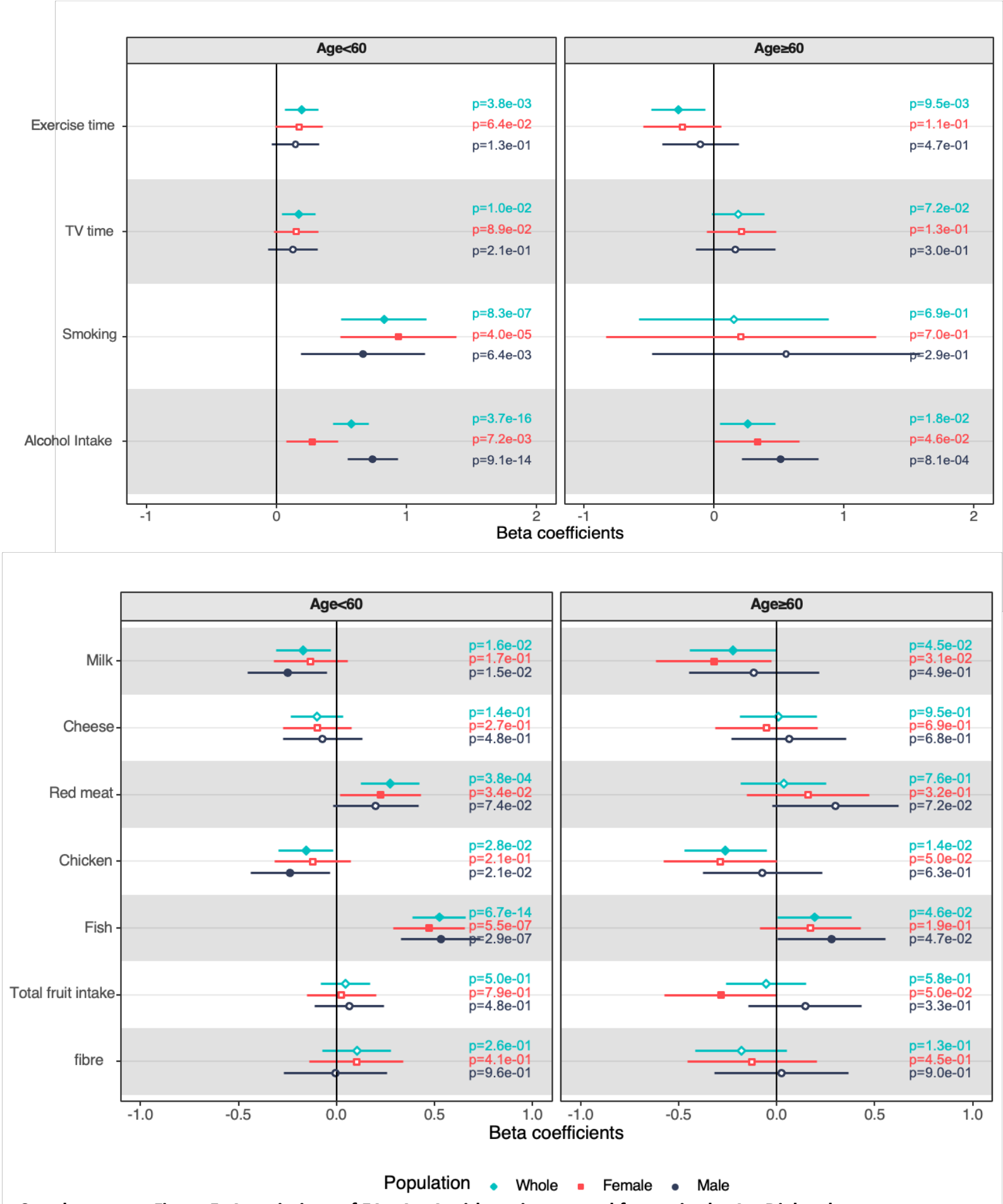


Supplementary Figure 3. Associations of mAge_origΔ and EAmAgeΔ with different combinations of dietary fatty acids.

Separate linear regression model were carried out to examine different combinations of dietary fatty acids with two sets of mAgeΔ with or without environmental adjustment.



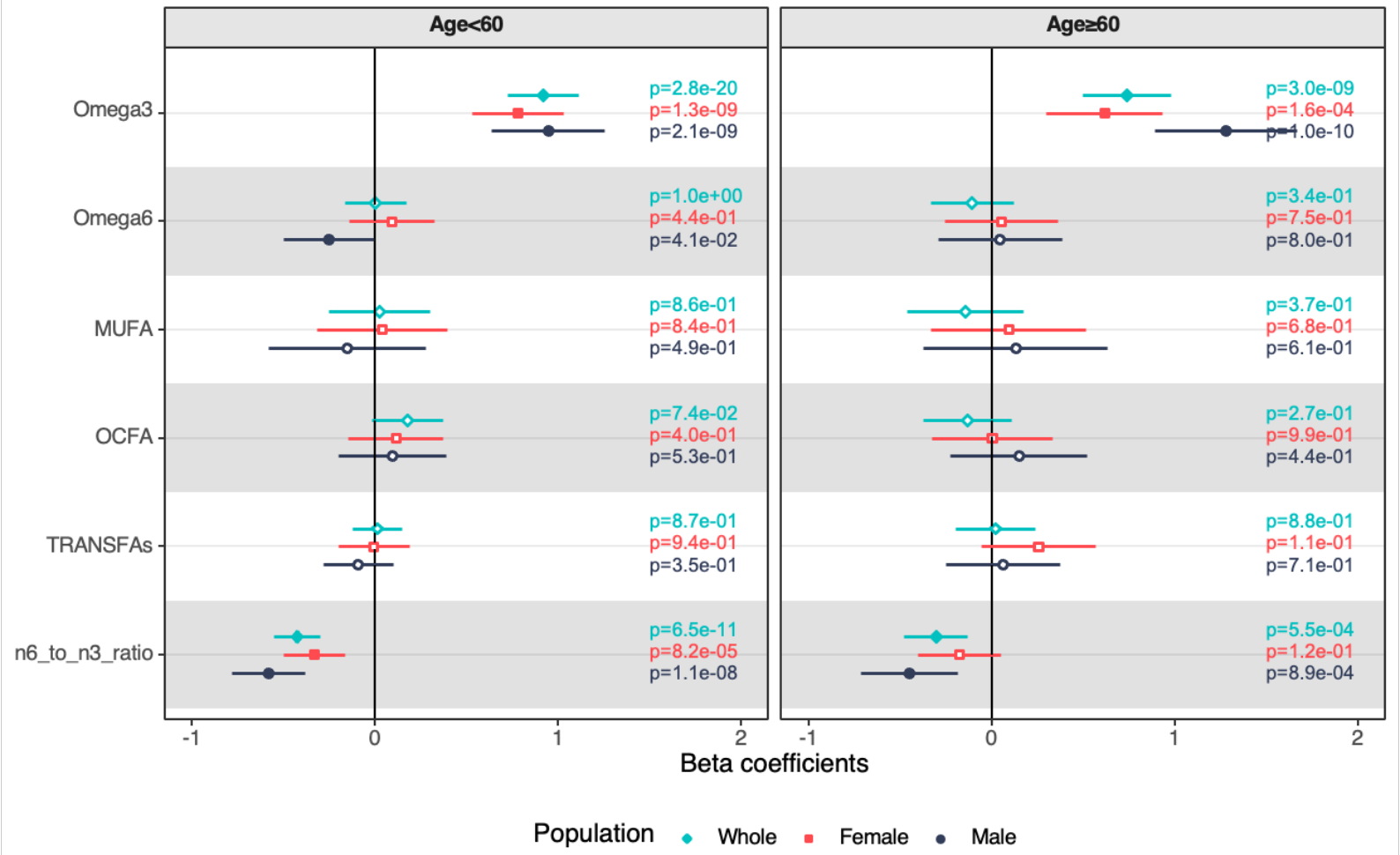
Supplementary Figure 4. Associations of EAmAgeΔ with different combinations of dietary fatty acids.
EAmAge models were separately built on Acylcarnitine lipids only (left panel) and clinical lipid including HDL-C, TG, and TC (right panle).



Supplementary Figure 5. Associations of EAmAgeΔ with environmental factors in the AusDiab cohort.

Separate linear regression model were carried out to examine different environmental factors with EAmAgeΔ scores on younger group (age<60; Left panel) and older group (age≥60; Right panel).

Smoking status (current smoker vs. non-smoker) was treated as a categorical variable. All other continuous environmental factors were inverse rank normalized and are interpreted per standard deviation change.



Supplementary Figure 6. Associations of EAmAgeΔ with environmental factors in the AusDiab cohort.
Separate linear regression model were carried out to examine different environmental factors with EAmAgeΔ scores on younger group (age<60; Left panel) and older group (age≥60; Right panel).