Neuroscience Campus Amsterdam

Stability in anxious depression as a function of genes and environment. A longitudinal twin study from age 3 to 65 years

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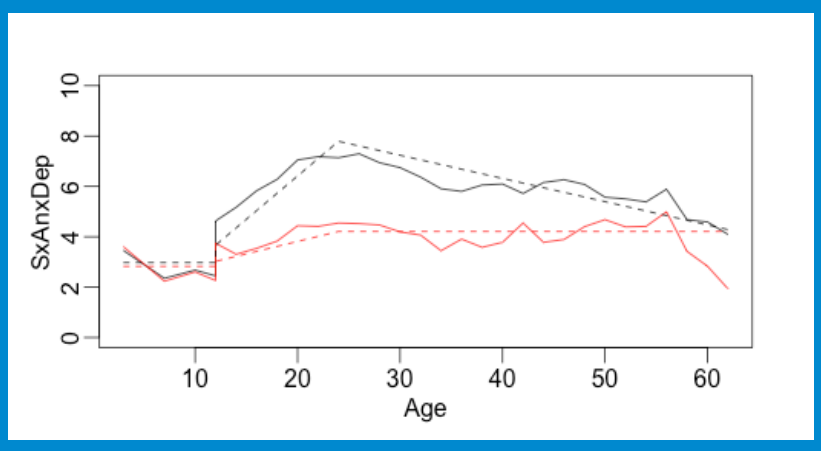
Introduction

Knowledge on the longitudinal etiology and the stability of genetic and environmental influences on symptoms of anxiety and depression (SxAnxDep) across the lifespan is limited.

Methods

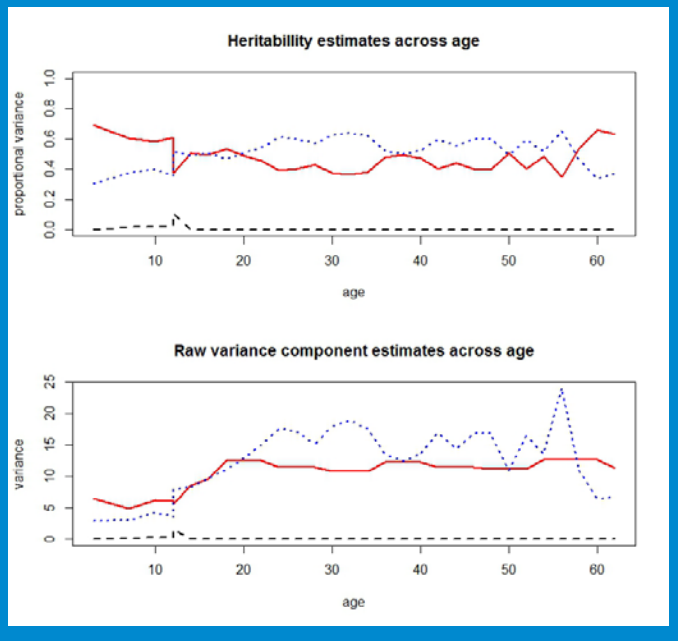
Longitudinal data on SxAnxDep were collected in 7,863 monozygotic and 15,815 dizygotic twin pairs registered at the Netherlands Twin Register (NTR). The majority of twins participated in more than one survey. A genetic simplex model was chosen to analyze the cohort sequential data. This model allows the assessment of the stability in the effects of genetic and environmental factors over time by modeling the extent to which effects are transmitted from one time point to the next, and the extent to which “new” effects (innovations) come into play.

Means



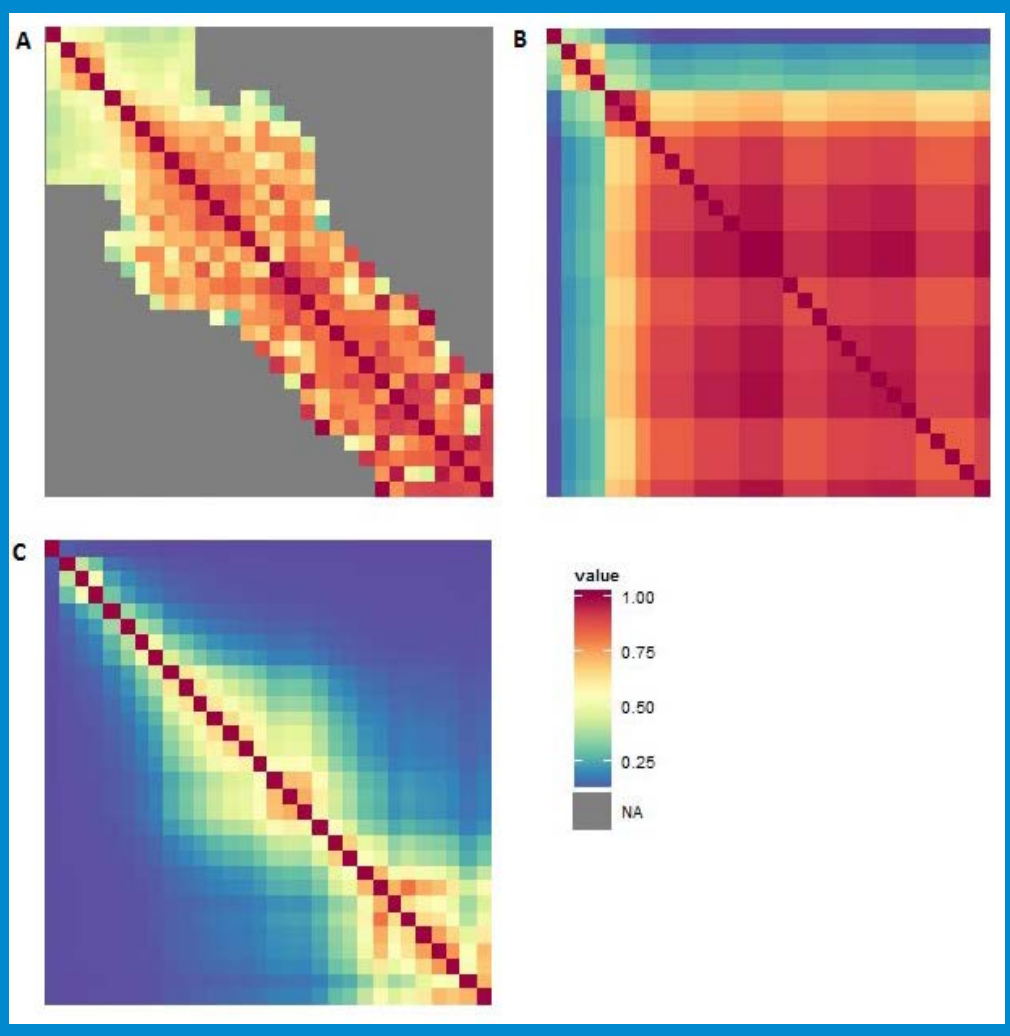
Mean SxAnxDep scores over time for men (red) and blue (women) Dashed lines are the best fitting WLS regression . Female SxAnxDep increase more than male scores in adolescence and remain significantly higher until age 50

Variance



Genetic (red) unique environmental(blue) and shared (black) components of variance. Above the variance components relative to the total variance (heritabillity) below the raw variance components.

Covariance



Heat map of A) observed correlations between all age groups, B) model derived genetic correlations between all age groups and C) model derived environmental correlations between all age groups. Age groups span 2 years and range from age 3 (upper left) to 65 (bottom right) years of age.

Results and Discussion

Over age, there was a significant increase in mean scores with sex differences emerging during adolescence (see Mean plot). Heritability was high in childhood and then decreased to around 30-40% during adulthood. This decrease in heritability was due to an increase in unique environmental variance (see Figure at the left). Phenotypic stability was moderate in children (correlations between ages around .5) and increased in adolescence (r=.6), young adults (r=.7) and after age 32 (r=.8) (see covariance plot). Stability was mostly explained by genetic factors. During childhood and adolescence there was also significant genetic innovation, which was absent in adults. Unique environmental effects contributed only to short term stability. These observations are consistent with and explain the low –as compared to other major psychiatric disorders- heritability of major depressive disorder.

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