Specific Language Impairment (SLI) is defined by a failure to acquire age-appropriate language, where not explained by factors such as general ability. The disorder is shown to have a strong familial transmission and high prevalence (around 5% of children). Because it is both common, and has high socio-educational impact ¹, research on biological mechanisms and early identification of at risk children has been highlighted as an important strategy in reducing the disease burden ²,³.

In the case of the related language disorder of dyslexia—problems with written language—, the pattern of familial transmission has been shown to be due almost entirely to genes after the age of 5 ⁴, and rapid progress has been made in moving to gene identification, with 11 sites of interest and at least 3 very strong candidate functional genes.

Relative to dyslexia, however, SLI has been little studied. In part this is because of difficulty in identifying a reliable specific phenotype ⁵. Gathercole and Baddeley recently suggested that a core deficit in language processing involves a defect in short-term storage of speech sounds. This deficit is assessed reliably using a non-word repetition paradigm, and work from the SLI consortium suggests that it is heritable, and explains a portion of variance in SLI. In this paper, we present the first data on the heritability of the NWR phenotype in an unselected sample of 967 families of adolescent twins, along with multivariate modeling of the genetic correlations between dyslexia and language impairment.

3 Fisher SE, Francks C: Genes, cognition and dyslexia: learning to read the genome. Trends Cogn Sci 2006; 10 250-257.