

PLINX204 PSYCHOLINGUISTICS

LANGUAGE ACQUISITION – 2006 - 5

Behavioural ('population') genetic evidence (2)

Twin, Adoption and Linkage studies

Basic reading: Stromswold, 2001 (esp. §§10-15); Plomin & Dale, 2000; Friederici, 2005.

1. Background

99.9% of DNA base pairs are the same for all human beings. So our individual uniqueness is due to that 0.1% (plus environment). Following on from last week's "change of perspective", note that language could be 100% genetically determined but everyone could be genetically identical, so in normals, *no* variance would be attributable to genetics. (Consider number of fingers versus weight or BMI). Need to keep apart normal and pathological development. Alpert syndrome (oligodactyly); Laurence-Moon-Biedl syndrome (polydactyly). In language, normal and abnormal development are both subject to genetic influence, though not necessarily the influence of the same genes.

2. Aggregation

Nature versus nurture: Family aggregation studies show that the incidence of language disorders is greater among relatives of people with language impairment ('proband') than controls. In seven studies, the incidence of positive family history ranged from 24% to 78% for probands, from 3% to 46% for controls. This is suggestive of genetic determination but is compatible with the Deviant Linguistic Environment Hypothesis: that such **children are impaired because they are exposed to deviant language**. Similarly, we are not born knowing words – we learn them. The quantity and quality of parental language correlates with children's rate of language development; schools differ in the quality of the educational experience they provide; more rapidly developing children elicit different language behaviour from their caretakers. However, DLEH makes the wrong predictions: (Stromswold, 2001:651).

i. The most severely impaired children should come from families whose members have severe language impairments, but children with profound language impairments were less likely to have positive family histories of language impairment than children with moderate language impairments.

ii. The type of impairment should be the same, but is consistently different.

iii. Parents who have recovered from a spoken language impairment should be no more likely to have children with impairments than parents who were never affected. This is contrary to fact.

iv. If mothers have primary responsibility for child-rearing, then Mother-child correlations should be greater than father-child and sibling-child correlations. In fact, mother-child correlations were weakest.

3. Twins

Identical (MZ) and fraternal (DZ) twins. MZ \approx 100% genetically identical; DZ \approx 50% (same as any other siblings). {This is not entirely straightforward because (for epigenetic

reasons) MZ twins may have (e.g.) different fingerprints; DZ twins may have more than 50% in common (cf. tissue graft rejection)}. MZ twins have twice as much genetic material in common as DZ twins, so if MZ twins show no more similarity for a trait than do DZ twins the implication is that that trait is not under genetic control. ‘Heritability’ refers to the proportion of phenotypic variation that can be attributed to genetic variation. The statistics are complex: Stromswold (2001) gives a good survey. Her meta-analysis shows that concordance rates are significantly higher for MZ twins: for spoken language disorders they are about 10% higher than for written language disorders, but consistently about 10% higher for MZ than DZ in both. There is a significant genetic role in dyslexia, word blindness, SLI, ‘poor composite language score’, deficient vocabulary, etc. For instance on the Edinburgh articulation test, MZ SLI twins had significantly higher correlation coefficients than DZ SLI twins.

i. In analyses of stuttering zygoty was seen to be relevant back in 1945: MZ 90%; DZ 7%. Biological (genetic) aetiology was out of fashion, so an alternative explanation “telepathic identity” was suggested (Felsenfeld & Drayna, 2001:154). Same result from Australian Twin Project: 50 MZ pairs – 20% concordant; DZ – 3% concordant. 71% of the variance = genetic; 29% = environmental (Felsenfeld & Drayna, 2001:161-2).

ii. Heritability of vocabulary development: Stromswold (2001:668) reports that MZ correlations were greater than DZ correlations in all 8 studies.

iii. In 36 tests involving Phonological awareness, Morphosyntax, The onset of the two-word stage and Language comprehension, MZ correlation were greater than DZ for 33 of the tests (Stromswold, p.680). High correlation between vocabulary and grammar, low correlation between verbal and non-verbal cognition.

iv. TEDS (Twins Early Development Study) (Plomin & Dale, 2000).
> 3000 sets of twins born in 1994.

Twins at age 2 were given the MacArthur Communicative Development Inventory, testing:

- a. production of root words – a vocabulary measure [.25]
- b. extent to which words are combined into sentences - a grammar measure [.39]
- c. use of language for displaced reference (talking about non-present items) [.22]

The test also included:

- d. ‘Non-verbal performance’ [.25]
- e. ‘General cognitive ability’ [.20]

Heritability estimates are shown in [square brackets].

v. TEDS data at 2 years were also used to conduct a multivariate genetic analysis within language, comparing (a, b, c) above. Correlations show that genetic effects largely overlap. [.61] for a/b; [.65] for a/c; [.53] for b/c

Genetic correlation between language and non-verbal cognitive development is smaller: [.30] for a/d; [.29] for b/d; [.40] for c/d (cf. also Stromswold, p.663).

vi. “In TEDS, the heritability of vocabulary is slightly but significantly greater for boys (26%) than for girls (19%)” (P&D 2000:41).

vii “... articulation of the phoneme /r/ is largely the result of genetic factors, whereas environmental factors play a greater role in the articulation of the phonemes /l/, /w/ and /j/”. (Stromswold, 2001:673).

viii. The most striking contrast between linguistic and non-linguistic performance is on Non-Word Repetition (heritability 100%) *versus* pure tone repetition (heritability 0%).

ix. Pathological *versus* normal development

“... genetic factors account for some of the individual differences in linguistic ability for both normal people and people who suffer from developmental language disorders. For both written- and spoken-language impairments, MZ twins are significantly more likely to be concordant for language disorders than are DZ twins... heritable factors typically accounted for over half of the variance in language-impaired people’s linguistic abilities”. (Stromswold 2001:704). DF extremes analysis¹ – avoids the crudeness of dichotomies caused by reliance on concordance data.

x. Group vs. individual heritability (73% *versus* 25%) indicates that genetic factors are far more important for the lowest 5% of the distribution than for the group as a whole. The genetic correlation between V(erbally IQ) and P(erformance IQ) is modest for the normal range [.30], but high [1.0] for probands selected for low V (p.46), suggesting that lumping them together for ‘IQ’ purposes is misguided.

xi. The effect of genes *increases* over time:

a. “As children get older, heritable factors and nonshared environmental factors play an increasing role in spoken vocabulary, and shared environmental factors play a decreasing role” (Stromswold, 2001:670-671).

b. “[G]eneral cognitive ability shows only modest heritability in infancy and early childhood with increasing heritability in middle childhood and adolescence” (Spinath et al 2004:445).

c. “... shared environmental influence declines in importance from childhood to adolescence, and heritability increases during this period” (Spinath et al, 2004:453).

4. Adoption

Adoption studies work in two directions: the adoptees and their unadopted siblings share genetics but not environment, the adoptees and their adopters’ children share environment but not genetics. Twin and adoption studies give the same results.

i. “The longitudinal Colorado Adoption Project ... shows **increasing genetic influence on verbal ability from childhood through adolescence**... [such] increasing heritability illustrate[s] the extent to which genetics and environment may be interdependent... Although it is possible that new genes turn on during development, it is more likely that the explanation for this finding is that experience ... is affected by the child’s own characteristics, which are in turn influenced by the child’s genotype, and this phenomenon becomes increasingly important with development”. (Plomin & Dale, 2000:39; my emphasis).

ii. “The results of CAP indicate that for normal and impaired children genetic factors affect language abilities more than shared environmental factors do. Stromswold, p.694.

“Adopted children who had language-impaired biological relatives were almost three times more likely to suffer from language disorders than adopted children who did not have language-impaired relatives” (Stromswold, 2001:704-5).

¹ An analysis of familial resemblance that uses quantitative scores of the relatives of probands rather than just assigning a dichotomous diagnosis to the relatives and assessing concordance.

iii. "... language disability tends to be more strongly under the influence of genetic factors than does language ability in the normal range" (Spinath et al, 2004:452).

iv. "... some genes associated with language disability and language ability may show stronger effects in boys than in girls"... the greatest effect on language disability and ability in early childhood is shared environmental influence" (ibid).

5. Linkage Continuity, maturation, modularity and modularisation

i. "Written language disorders are genetically heterogeneous" Stromswold, p.698-9, with e.g. dyslexia being linked to six distinct genetic loci. It is extremely likely that spoken language disorders too are genetically heterogeneous, despite lack of much evidence beyond the KE family.

ii. "Recent neurophysiological work with infants and young children indicates that brain mechanisms underlying phonological, semantic and syntactic processing are quite similar to that of adults, even in early development" (Friederici, 2005:482) "... whereas second language learners use qualitatively different processes" (id. p.481).

iii. Continuity as an evolutionary default: "Evolutionary change is biased towards modification of pre-existing mechanisms, and away from the construction of wholly new devices" (Schoenemann, 2005:82).

iv. Multivariate genetic analyses contradict Karmiloff-Smith's (1992) modularisation hypothesis. (Plomin & Dale, 2000:45).

- a. Early low V/P correlation (each of verbal and performance factors seem to be autonomous from early on), allied with high heritability for V.
- b. Later higher V/P correlation (suggesting convergence), allied with high, perhaps increasing, heritability for V.

Contradicts the predictions of modularisation, as opposed to Fodorian modularity.

v. "Recent neurophysiological work with infants and young children indicates that brain mechanisms underlying phonological, semantic and syntactic processing are quite similar to that of adults, even in early development" (Friederici, 2005:482) "... whereas second language learners use qualitatively different processes" (id. p.481). This constitutes further support for continuity and against the usage-based account of e.g. Tomasello. (cf. Tomasello (2000a, b) and Smith (2005, ch. 9)).

Additional reference

Felsenfeld, S. & D. Drayna (2001) "Stuttering and genetics". In: Gerber, S.A. (ed) (2001) *The Handbook of Genetic Communicative Disorders*. San Diego; Academic Press; pp. 151-174.

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