

# PLINX204 PSYCHOLINGUISTICS

## LANGUAGE ACQUISITION – 2006 -1

### The genetic basis for language acquisition: Overview

1. The ‘innateness hypothesis’ and its implications.

- a. Human infants *versus* kittens and computers
- b. Linguistic variety: Arabic, Bengali, Chinese ... Xhosa, Yoruba, Zulu.

Nature *versus* nurture. “... part of our linguistic knowledge is innate and part is learned ... [but] ‘innate’ and ‘learned’ are not technical scientific terms but belong to the pre-theoretical stage of investigation ... any property will be the result of an interaction between what was specified in the genome and what is in the environment”. (Smith, 2004:37).

2. **Types of evidence:**

- a. **A priori evidence:** Fodor’s (1975, 1980) ‘Language of thought’ infinite regress argument.

"... to learn a language you already need to know a language" (Smith, 1989:65, paraphrasing Fodor). To learn *tabescent* you need a representational system rich enough to express 'progression', 'emaciation', etc. For a critique, see Spelke & Tsivkin (2001); Viger (2005).

- b. **Learnability** see Stromswold, 2001.

Even in the absence of negative evidence, and even in conditions of sometimes severe deprivation, either social or cognitive, children characteristically acquire their first language quickly and easily.

- c. **Linguistic:** For a summary, see Smith, 2005, ch. 9 (“Backlash”)
  - i. **Species-specificity.**

Species traits are genetically encoded. Language is characteristic of humans and, up to severe pathology, seems to be independent of intelligence. Even if bonobos or dolphins also had language, this would not impugn the claim that humans were genetically predisposed to acquire language. However, evolutionary considerations (FLB *versus* FLN, see Hauser et al, 2002) suggest that we share much of our ‘language faculty’ with other species, presumably likewise as a result of genetic evolution.

- ii. **Speed and age dependence** of acquisition. Critical periods.

It takes much less time to learn English than genetics, and one can do the former at an age when the latter is impossible. (This is a form of the Poverty of the Stimulus argument). For a detailed critique, see Sampson (1989 or 1999).

iii. Convergence among grammars.

Speakers appear to converge on much the same grammar irrespective of intelligence and of their exposure to different samples of the language. In pathological populations (e.g. Down Syndrome, Williams Syndrome children), the innate component is more important than in the typically developing population, because of the putative unavailability of intelligence-based alternative routes to acquisition. (This is a form of the Poverty of the Stimulus argument). For a detailed critique, see Sampson (1989 or 1999).

iv. Universals both of grammars and the stages of their acquisition.

Languages have universal properties: e.g. structure-dependence, exploitation of c-command; specific categories, such as Noun and Verb; X-bar theory; constraints on lexicalisable concepts; fixed order of emergence – e.g. peripheral negation, etc. For a detailed critique, see Sampson (1989 or 1999).

v. Emergent categories (Clark, 2001)

Categories such as classifiers, or middles, or the notion ‘source’, or inherent *versus* temporary properties are reflected in children’s over-extensions. Children appear to create only those categories which are licensed by UG. (This is a form of the Poverty of the Stimulus argument).

vi. Parametric cascades (Baker, 2001).

Learning one fact of one’s first language seems to bring with it knowledge of other facts without exposure to relevant data: e.g. the pro-drop parameter. (This is a form of the Poverty of the Stimulus argument).

vii. Domain-specificity

In problem solving behaviour (Smith & Tsimpli, 1995:140-142), structure-independent operations (in Epun) were impossible to learn when embedded within the language faculty, but were easy in a general problem-solving domain.

viii. Poverty of the stimulus

We end up knowing more than we have been exposed to or can plausibly be taken to have inferred on the basis of input data. It is especially important in accounting for intuitions of ill-formedness. The primary linguistic data are ‘restricted and degenerate’. The classical Poverty of the Stimulus argument is exemplified for structure dependence by e.g. the nature of question formation: *Is the man who is tall in the room?* where sundry possible acquisitional hypotheses are ruled out a priori, and children appear not to entertain such (e.g. arithmetic) hypotheses. For a detailed critique, see Sampson (1989 or 1999).

ix. Modality neutrality

Language acquisition seems to be neutral as between the signed and spoken domain, suggesting an abstract linguistic substructure. (Petitto, 2005).

d. **Behavioural genetics**

i. Heritability of language disorders.

Some disorders of language (e.g. SLI) appear to be ‘inherited’. Evidence comes from epidemiology and familial aggregation; the KE family. (Bishop, 2003; Marcus & Fisher, 2003; Smith, 2004:131-3).

ii. Twin, Adoption and Linkage studies.

Differences between identical and fraternal (MZ/DZ) twins suggest genetic determinants of many linguistic properties. (The TEDS – Twin Early Development Study - project). Confirmation comes from adoption studies e.g. (CAP - Colorado Adoption Project), and ‘Linkage studies’. (Plomin & Dale, 2000, Plomin et al, 2000, ch.5).

iii. Individual differences in the rate of language acquisition by children and proficiency in the adult correlate with known genetic differences. (Stromswold, 2001; Plomin et al, 2002).

iv. Variance in language disorders.

Written vs. spoken language abilities and disorders. Multivariate genetic analysis. (Plomin & Dale, 2000; Stromswold, 2001). “... about half of the variance in tests of verbal ability can be ascribed to genetic differences among individuals” (Plomin & Dale, 2000:39).

v. Modularity vs. modularisation; Continuity vs. discontinuity.

Plomin et al, 2002; Stromswold, 2001; Friederici, 2005; Karmiloff-Smith, 1992.

e. **Molecular genetics.**

i. FOXP2

Elementary genetics: from genotype to phenotype. Mendel’s laws; chromosomes, genes, alleles, polymorphism. Pleiotropy vs. convergence – i.e. polygenic phenomena (autism). Epigenetic considerations. The KE family revisited; of mice and men. Epidemiology shows that certain pathologies must be genetically controlled; we are now in a position to identify the genes involved. (Plomin et al, 2000; Russell, 2006; Stromswold, 2001; Newbury et al, 2005; Müller (2004), Elman et al. 1996.)

ii. QTL (Quantitative Trait Locus) analysis.

Most genetic disorders are attributable not to a single gene, but to a large number of closely located genes. Developmental dysphasia and dyslexia. Spoken and written language. NWR (Non-word repetition) and STM (Short-term memory); The relation to SLI. Risk-factor models.

f. **Neuro-anatomy and brain-imaging**

i. PET (Positron Emission Tomography) and the genetics of Phonology. (Müller, 2004; Friederici, 2005).

ii. ERP (Event-related potential) studies. (Friederici, 2005).

g. **Language acquisition in pathology**

i. Autism and SLI.  
Müller, 2004; Bartlett et al, 2002; Bishop, 2003.

ii. Williams syndrome and Down syndrome.

Scerif & Karmiloff-Smith, 2005; Zukowski, 2004.

3. **The challenge of Minimalism**

Is anything innate specific to language? We are probably still not in a position where we can even say categorically that *anything* in the genome is dedicated exclusively to language (or to the structure of the knee ...). Chomsky, 1995b, 2005.

4. **Risk factor analysis:** the importance of multi-variate analysis (see Newbury et al, 2005:530).

