

# PLINX204 PSYCHOLINGUISTICS

## LANGUAGE ACQUISITION – 2006 - 9

### Language acquisition in pathology (2) Williams syndrome & Down syndrome

Basic reading: Ring & Clahsen, 2005; Scerif & Karmiloff-Smith, 2005; Zukowski, 2004.  
Additional reading: Schaner-Wolles, 2004; Grzeschik, 2004; Perovic, 2001.

Beware of the hype.

1. Outline of the issues:
  - a. What is WS? What is DS?
  - b. What are the linguistic properties of each?
    - i. Are these properties the same or do they (doubly) dissociate?
    - ii. Are they correlated with intelligence or other cognitive phenotypes?
    - iii. Do they indicate 'deviance' or (just) 'delay'?
    - iv. Is the phenotype static over time (from child to adult)?
  - c. What are the genetic aetiologies of each?
    - i. Can these be correlated with any putative linguistic differences? More generally, is it feasible to effect any mapping between specific genes and specific cognitive phenotypes? That is: do 'pure selective deficits' exist?
    - ii. Can they be used as evidence for or against specific linguistic theories (e.g. Minimalism *versus* LFG or HPSG), or cognitive theories (e.g. Modularity *versus* Modularisation)?
2. Williams(-Beuren) syndrome: A rare condition (infantile hypercalcaemia) in which children have a variety of genetically conditioned physical characteristics (supravalvular aortic stenosis, elfin features, stellate iris, moderate to severe intellectual impoverishment, but excellent face recognition ability and extremely fluent language.

WS is a sporadic disorder affecting 1 in 20,000 births and associated with hemizygous microdeletion of 1.5 – 1.7 million base pairs from chromosome 7q11.23 encompassing more than 20 genes, including the elastin (ELN) gene.

3. Down syndrome: A common condition characterised by muscular hypotonia, thick or protruding tongue, general motor and intellectual delay, mechanical problems, heart defects, sensory deficits (especially deafness), cognitive retardation, typically to 4-5 year old level in the adult, and considerable linguistic variability from case to case,

DS is the commonest cause of mental retardation, occurring in 1 in 600 births; in 95% of cases it is due to trisomy 21 – i.e. 3 copies of chromosome 21. The other 5% of cases are due to (Robertsonian) translocation between chromosomes 14 and 21 (4%), and mosaicism (a mix of normal and trisomy 21 cells in different tissues (1%).

4. The complexity of the phenotype (from Scerif & Karmiloff-Smith (2005:128):

WS has a strikingly uneven profile in the adult, with relative strengths in language and face-processing and weaknesses in visuo-spatial cognition and numerical processing ... infant studies show a different pattern with delayed vocabulary comprehension equivalent to DS subjects (whose later language is delayed compared with WS adults) and to younger normal controls. WS infants are normal for the discrimination of small quantities but grossly impaired numerically when older. By contrast DS infants were extremely delayed on the same measure, though DS adults surpass WS adults numerically. That is the genotype – phenotype relation is complex, with developmental asymmetries of the kind shown in (5):

5.

	<u>WS infants</u>	<u>WS adults</u>	<u>DS infants</u>	<u>DS adults</u>
<i>Language</i>	weak <sup>1</sup>	strong	weak	weak
<i>Face processing</i>	weak	strong <sup>2</sup>	weak	weak
<i>Numerical ability</i>	normal <sup>3</sup>	very weak	very weak	better than WS

6. Deviance versus delay:

All claims are contested but the majority view has been that language development is deviant in WS (see especially Karmiloff-Smith and her colleagues), but merely delayed in DS (for discussion, see Rondal, 1995, who describes a case of a woman who attained essentially flawless French). If the condition is mild, DS subjects may continue to learn their first language until (at least) puberty. Post-pubertal learning is (probably) restricted to non-parametric development (evidence for a critical period). Linguistically outstanding DS individuals “developed their excellent morphosyntactic and phonological abilities between approximately 4 and 10 years C[hronological] A[ge]” (Rondal & Comblain, 1996:2.2). The major differentiating factor is whether WS/DS subjects show patterns of development which are distinct from patterns found in TD children. My tentative conclusion today (following Perovic, 2001; Ring & Clahsen, 2005 and Zukowski, 2004) will be, contrary to this received wisdom, that the knowledge of language of DS children is sometimes ‘deviant’ and that the knowledge of language of WS children is typically (only) delayed. The exploitation of that knowledge in performance may obscure this simple dichotomy.

7a. Karmiloff-Smith et al (1997:258) claim that “WS language follows a different path to normal acquisition and may turn out to be more like second language acquisition”: that is, subjects acquire language by ‘deviant mechanisms’ – their *knowledge* is abnormal and their performance is based on ‘auditory rote memory’.

b. Zukowski’s (2004) experimental results suggest that WS subjects’ knowledge of language is developmentally normal. Test material involved the production of negative questions such as *What flavour of ice-cream don’t you like?/ What flavour of ice-cream do*

<sup>1</sup> In terms of grossly delayed vocabulary comprehension.

<sup>2</sup> But note that this may not be done the same way that normals do it.

<sup>3</sup> In terms of the discrimination of small quantities.

*you not like?* which are very rare in spontaneous speech. WS subjects “did not produce any developmentally abnormal errors” (Zukowski, 2004:113): i.e. they are normal in their development, but get stuck at a stage where characteristic errors are still made.

8. TD children make mistakes on negative questions but not on positive questions:

- a. *Where can't your dogs sleep?* [target]
- b. *Where can your dogs can't sleep?* [auxiliary doubling error]
- c. *Where your dogs can't sleep?* [lack of subject-auxiliary inversion]
- d. *Where can your dogs can sleep?* [does not occur]
- e. *Where your dogs can sleep?* [does not occur]

9. Results: {From Zukowski, 2004:112}

Variable	Affirmative question trials		Negative question trials	
	WS (287)	Controls (292)	WS (176)	Controls (184)
Target question	90%	99%	49%	49%
No auxiliary raising	10%	1%	26%	7%
Doubled auxiliary	0%	0%	9%	18%
Unraised <i>not</i>	-	-	14%	23%

10. Corroboration: {From Ring & Clahsen, 2005}

a. “The two core modules [lexicon and computational system] are dissociated in WS such that the computational system is not affected while lexical representations and/or their access procedures are impaired” (Ring & Clahsen, 2005:480). (e.g. the frequency effects characteristic of normals appear not to obtain).

b. In DS there are enhanced levels of lexical skill relative to reduced levels of morphosyntax, and qualitative differences between TD and DS children (e.g. Perovic).

11. The evidence: Passive and Binding. Theory dependent possible parallels between passive and binding – ‘A-chain formation’ links them for e.g. Reuland, 2001; nothing links them for e.g. LFG (passive is a lexical alternation, binding a principle of semantic interpretation), so different results may also support different theories.

a. Binding tested with STOP (Syntactic Test Of Pronominal reference, van der Lely & Stollwerck, 1997): *Is Mowgli tickling him/himself?* [Picture of Mowgli tickling Baloo Bear] Subjects had to answer yes/no respectively.

b. Passives tested with TAPS (Test of Active and Passive Sentences, van der Lely, 1996): *The man eats the fish/ The fish is eaten by the man/ The fish is being eaten/ The fish is eaten* On being prompted with one of these sentences, subjects had to choose one of four pictures:  
A man eating a fish/ A fish eating a man/ An eaten fish on a plate/ The remains of a man.

12a. Binding: DS did better on pronouns than on reflexives; WS showed no difference and were as good as, or even better than, the controls.

b. Passives: DS had more difficulties interpreting passives than active sentences (usually their errors involved ‘reversal’ responses); WS subjects had no systematic errors and were not statistically distinguishable from the controls.

c. WS are roughly error-free on both, DS have problems with both, controls are fine on both.

d. The DS difficulty with reflexives (while having no problem with non-reflexives) supports Perovic’s (2001) results showing deviance not just delay for DS subjects: i.e. no normal population has this problem – (contrast the ‘delay of principle B effect’).

13. WS/DS “different genetic etiologies are associated with different specifically linguistic patterns of impairment” (Ring & Clahsen, 2005:479). But how?

14. “Genetic duplications [trisomy 21] are known to have a variety of effects yielding, for example, more rigid cell walls or an increased gradient of regulatory proteins ... but how these effects should bring about particular patterns of linguistic impairment [A-chain formation] is unknown ... [nonetheless] it might indeed be the case that this particular grammatical structure is under genetic control” (Ring & Clahsen, 2005:497-498).

#### Additional references

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