The nature-nurture controversy in its most recent avatar: A response to Shukla and Vasanta

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The study of language acquisition in children has long been at the core of the nature versus nurture controversy among researchers from several disciplines whose common endeavour was the understanding of the acquisition of language in humans. Over the years the pendulum has swung in both directions but the 20th century has perhaps witnessed the greatest debates on the topic, increasingly within a scientific framework. Each side of the controversy has been supported or criticized on the basis of evidence, both experimental and clinical. The reign of the nurturists was embodied by the Skinnerian theory of 'verbal behaviour' in the first half of the last century. This was dealt a body blow by Chomskyan innatists by the middle of the century. By the latter half of the century the innatists were themselves under severe criticism largely for two reasons. First, hard evidence for the presumed underlying biological predisposition for language acquisition was lacking. Second, there was a neglect of, and lack of accounting for, several factors that were shown to govern the acquisition of language use in a social context.

Concurrent events at the turn of this century, in the disciplines of speech-language pathology and psycholinguistics on the one hand, and genetics and molecular biology on the other, have once again revived the ferocity of the debate. The chief provocation this time is the documentation of a claimed specific deficit, exclusively in language acquisition, in a number of individuals of a given family (the KE family). There were said to be no significant intellectual or other cognitive deficits in these individuals. To top it all, a defective gene, FOXP2 (subsequently dubbed the 'language gene'), was identified in these individuals. In contrast, in Williams syndrome (WS), a rare autosomal genetic disorder, linguistic abilities were reported to be spared in the presence of serious deficits in cognitive skills, particularly visuo-motor skills. WS has been cited as a developmental disorder with a clear dissociation of language from other cognitive skills. Assuming that behaviourally defined specific language impairment (SLI) and the language-associated traits exhibited by the KE family can be treated as the same phenotypic trait, this would seem to imply something interesting about the double dissociation seen in SLI and WS, both of which have clear genetic deficits. One could conclude that intelligence and language were not irrevocably interconnected as had been previously thought. The theory that our ability to use language is innate and based in our biology, not acquired in training, seemed to be reinforced. However, the claims of exclusivity with regard to the language disorder in the affected members of the KE family was subsequently revised when more extensive testing showed that some among them also tended to score below average in nonverbal intelligence tests (Vargha-Khadem *et al* 1995). Similarly the claim that language development is normal in WS has been questioned with the documentation of pragmatic language impairments (Laws and Bishop 2004).

In this volume, Shukla, the protagonist of the position that language acquisition in humans is innate, presents his argument within the framework of Chomsky's theory of principles and parameters (Shukla 2005). He argues for an innate biological component determined by genetic and cellular factors intrinsic to the developing brain, as

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Abbreviations used: DD, Developmental dystexia; MLU, mean length of utterance; SLI, specific language impairment; WS, Williams syndrome.

the base for the internal computational capacity (principle) that underlies the use of language for the various external functions. The observed grammatical properties of languages are seen as being derived from the interaction between this core syntax (merge) and the two interfaces – semantic (conceptual-intentional) and output (sensory-motor), with the variations in languages (parameters) seen as arising from the different solutions to interfacing. A further distinction is made between the 'core syntax' and the' interface' aspects of language. While the former is termed 'language-as–computation' the latter is presented as 'language-as–communication'.

The creolization of pidgin languages and the naturalization of 'sign language' after a couple of generations are interpreted as being supportive of the existence of such a biological base. It is conceded that 'linguistic input' triggers the language computational system and some of Shukla's work and that of his colleagues is presented in support of a possible though as yet hypothetical, trigger. Further, Shukla argues that language, as computation is the precursor to the totality of the language function.

Shukla then works out parallels between this supposition and other biological systems, such as – the reflection of the sensory systems in the topographic maps of the cortex in humans (visual and somatosensory) and other mammals. He raises the question of how 'merge' might have been acquired, by examining the acquisition of novel function in biological evolution, in particular the 'duplicate and evolve' pattern believed to hold true generally. The assumption is that if recursion existed in another system, a similar phenomenon in the conceptual – intentional system might have set the stage for the evolution of language as communication.

It is important to note that Shukla repeatedly emphasizes the distinction between 'language-as-computation' and 'language-as-communication', possibly forestalling the criticisms of the nurturists. However this does not explain if and why this computational capacity is deemed to be unique to language, and that too specifically to syntax. It could be similar to or a part of, a computational capacity that may underlie other cognitive skills such as mathematics or music. For instance, it was claimed that children with WS are noted to be remarkable in their musical abilities, though this observation has not stood the test of documentation.

It is also now clear that the speech disorder in SLI goes beyond an isolated syntactic deficit and encompasses motor speech production deficits (severe verbal dyspraxia). Those with SLI are also reported to function below par on phonemic discrimination and parsing, auditory comprehension, lexical access and meta-linguistic skills such as judgment of grammaticality. There is in fact substantial impairment of both receptive and expressive language. In fact given the range of deficits in SLI, subtypes are now being identified. Further while intelligence is relatively spared, there is some lowering of IQ (Pinker 2001; Lai *et al* 2001).

One of the major criticisms against the innatist view of language acquisition has been from researchers who have focused on aspects of language use that can only be acquired in a social context. This is the position of Vasanta who stresses the importance of social context in language (Vasanta 2005). She draws attention to the considerable visuo-spatial information that accompanies speech in day-to-day communication and the effects of the very process of development in language acquisition, irrespective of whether it started off as being delayed or deviant. The arguments presented against the innatist position which views language as a purely cognitive, non-communicative event are summarized, followed by a discussion of the problems of characterizing genotype-phenotype relations in neuro-developmental disorders such as developmental dyslexia (DD) and WS. Pinker's case for double dissociation in SLI versus WS as evidence for genetic double dissociation (Pinker 1999, pp 255–262) is countered with Karmiloff-Smith's argument against this in terms of the static vs dynamic characterizations of these disorders. Illustrating her point Vasanta considers Pennington's evidence for a genetic basis of DD, which relates deficits in phonological awareness and word retrieval difficulties to deficits in chromosomes 6 and 15. She counters this evidence with the argument that differences in phenotype may arise as a result of differences in factors such as orthography. It is now being increasingly documented (Karanth 2003) that the nature of the orthography can be an influential factor in the manifestation of the different types of dyslexia. Parallels may be seen in the manifestation of agrammatism in English versus say Hebrew, or the grammatical deficits in English speaking versus Italian speaking SLI.

The isolation of a specific deficit in the use of grammar, particularly in the expressive language of adults with a focal lesion in the Broca's area, had a couple of decades ago led to the notion that this is indeed the seat of grammar in the human brain. Subsequent studies made it clear that the extent of grammatical difficulties or degree and type of agrammatism and its overall nature (namely a pure expressive disorder versus an expressive and receptive disorder) was determined not only by the location and size of the lesion but also to a considerable extent by the characteristics of the language spoken by the individual. Patients who spoke highly inflected agglutinative languages like Hebrew in which the grammatical notations are expressed to a greater extent by bound morphemes, for instance, appeared to be less agrammatic as compared to those who spoke relatively uninflected languages like English; leading researchers such as Grodzinsky (1984) to argue that phonological accounts of agrammatism while

possibly accounting for agrammatism in English are far less attractive for Hebrew. This has also been our experience with agrammatism in speakers of agglutinative Indian languages like Kannada and Hindi with a differential loss experienced by bilingual speakers one of whose languages is agglutinative, in this case Kannada; while the other is nonagglutinative, in this case English (Rangamani 1991). Such a comparison lends support to the idea that despite the biological underpinnings or neural representations, differences in the manifestation of language disorders consequent to neural damage may be a reflection of the nature of the language or languages involved.

Similarly it has been documented that the areas of syntactic weakness in children with SLI will vary on the basis of language type. Italian speaking children with SLI are reported to differ from their English speaking counterparts both quantitatively and qualitatively in the way they treat grammatical inflections, once again the syntactic difficulties are more apparent in English as compared to the inflected Italian (Leonard 2000). This would suggest that the manifestation of SLI is linked to the nature of the language and not solely due to the deficit in the computational system (MERGE) that Shukla argues it as being.

Vasanta then presents evidence that questions the dissociation between visuo-spatial abilities and language abilities in WS, and points out that phenotypic outcome cannot be used to make generalizations about impaired or intact modules in the initial state. For instance, the manner in which those with WS learn the lexicon is said to involve different processes as compared to their normal peers, resulting in a number of atypicalities (Thomas and Karmiloff-Smith 2003). Others are reporting similar findings in other domains of language acquisition in WS. Levy (2004) found different grammatical profiles in two children with WS when compared to typically developing controls matched on mean length of utterance (MLU). The former performed better than MLU matched (but chronologically younger) controls on several grammatical markers but not for linguistic variables that are semantic-conceptual in nature. This leads to the question of whether language acquisition in children with WS is atypical, asynchronous or of a unique developmental pace. In Shukla's distinction one could argue that what is seen in WS is a normally developing 'language as computation' aspect, with the 'language as communication' aspect being disturbed. However, since WS also has clearly defined genetic components, it would imply that these aspects of language too have an underlying genetic basis, perhaps affecting other neural circuitry.

Further, just as some lowering of intellectual performance is being documented in SLI, a supposedly 'pure' language disorder, delays of onset in language including prelinguistic communicative gestures, vocabulary acquisition and grammar are all reported to be delayed in onset in children with WS (Laws and Bishop 2004). Apparently, language in children with WS is a strength as compared to non-verbal abilities but in standard tests of language they do poorly as compared to norms. In addition Laws and Bishop (2004) documented pragmatic impairments in children with WS, who performed significantly poorly as compared to children with Down's syndrome (DS) and SLI in a test of pragmatic skills, particularly on such aspects of pragmatics as inappropriate initiation of conversation and the use of stereotyped conversation.

Given that it now appears that neither SLI nor WS are clear cases of 'pure' language versus cognitive deficits and that both of them do indeed show signs of deficits in the relatively stronger area one could in fact argue, that these are due to deficits at the level of the interaction between merge and the two interfaces – semantic (conceptual-intentional) in the case of WS and output (sensory-motor) in the cased of SLI. The 'parameters' would then influence the extent of the variations across languages in children with these conditions.

Vasanta ends by raising issues concerning clinical management. She emphasizes that searching for behavioural phenotypes within a model that gives importance to environment and developmental time scale is more productive for the purposes of assessment and remediation than determining the genetic bases of the disorder using expensive DNA based tests, a caution that practitioners in countries like India should take heed of.

To summarize, the proposed double dissociation in WS and SLI has become the focus of a renewed debate on biodeterminism of human language capacities. The initial excitement about this dissociation is more tempered now because of the evidence from molecular biology (Lai *et al* 2001) that the biological deficit is more in the form of a trigger, rather than the source. It is important to note that the researchers involved postulate two things. One is that FOXP2 may have a causal role in the development of the normal brain circuitry that underlies language and speech; the other is that FOXP2 haplo-insufficiency in the brain at a key stage of embryogenesis leads to abnormal development of neural structures that are important for speech and language, with possible ramifications for other cognitive capacities (Lai *et al* 2001). Additional support for the role of FOXP2 in the evolution of language has come from different sources. Among these are the differences in DNA sequence of the FOXP2 gene in humans as compared to other mammals, and the time frame in which these changes occurred, which appears to match the time frame of the appearance of language in humans. That the evidence for the biological basis of language is growing cannot be disputed. However this does not necessarily mean that what is inherited is the 'universal grammar'. Many affected members of the KE family had

severe speech difficulties along with deficits in syntax and this was true of the unrelated case CS as well. SLI may turn out to be dependent on additive influences of many genes of small effect (Robert Plomin, cited by Bishop 2004). The dissociation between language and intelligence is also not as complete as it was made out to be. IQ in some affected members of the KE family is indeed significantly lower than in the unaffected individuals (Lai *et al* 2001; Vargha-Khadem *et al* 1995).

Incidentally, along with the advances made in genetic studies, developments in psycholinguistics are now making possible a more careful documentation of linguistic patterns, which were not seen or identified in these children 2–3 decades ago. A few decades ago, children with language disorders were evaluated with tools developed within the framework of adult language disorders or with gross measures of language such as vocabulary size or MLU, which focused on any one aspect of language. Today, with the growth of psycholinguistics and its impact on the study of childhood language disorders, we have tools that have been designed to capture the developmental process and those that focus on particular aspects of language, including the use of language for social communication. In addition there is an emphasis on the need for evolving theoretically motivated language indices rather than conventional clinical criteria. Bishop (2004) for instance argues for the use of 'nonword repetition', for a person's risk status. Nonwords are combinations of phonemes/syllables that follow the phonotactic rules of a language but are not words that are in existence in the vocabulary of the given language. Thus while holding the potential for becoming a lexical item in the language they are not in fact lexical items. Nonwords are often used in psycholinguistic experiments for teasing out effects of variables such as familiarity, for instance, in reading tasks. In her study Bishop found that children with SLI and those with a history of slow language development performed poorly on nonword repetition tasks. Further she reports that not only did identical twins resemble each other strongly in this skill while fraternal twins did so to a much lesser extent; but also in two children with SLI a significant linkage to this skill was found for a locus on chromosome 16. Bishop (2004) therefore suggests that nonword repetition could be a 'promising behavioural marker of underlying genetic risk' (p. 159).

These indices, as compared to regular language tasks are likely to be less influenced by phenotypic variations such as those seen in the cases of agrammatism.

We are yet to understand the complexities involved in mapping the neurological underpinnings of language acquisition in normal typically developing children. Just as the study of the underlying neuroanatomical lesions in the different types of aphasias led to a gross but nevertheless consistent mapping of the adult brain for language, the genetic study of children with language impairments could lead to a better understanding of the biological basis of language acquisition. When taken together the psycholinguistic and molecular genetic approaches have the potential for unravelling the mystery of language acquisition in children. Such investigations are only beginning and it is likely that we will witness many a new avatar of this age old debate before we understand the exact roles of nature and nurture in language acquisition.

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