

Commentaries

DOI: 10.1017.S0142716406060395

Beyond words: Phonological short-term memory and syntactic impairment in specific language impairment

The assessment of nonword repetition in children goes back at least to 1974, when the Goldman–Fristoe–Woodcock Auditory Skills Battery was published, including a subtest (Sound Mimicry) assessing nonword repetition (Goldman, Fristoe, & Woodcock, 1974). Nevertheless, it was not until 20 years later, when Gathercole and Baddeley (1990) reported a study of short-term memory in children with specific language impairment (SLI), that a theoretical framework was developed linking deficits in nonword repetition to impaired language acquisition. Gathercole’s Keynote in this issue (2006) tells the story of how this initial study revealed a striking nonword repetition deficit in children with SLI, complementing work on typically developing children showing a major role of phonological short-term memory (STM) in word learning. As she points out, the story is a complex one: phonological STM is not the only skill tapped by the nonword repetition task, and children may do poorly for different reasons. Furthermore, relationships between nonword repetition and word learning may be reciprocal, with vocabulary level affecting children’s ability to segment nonwords efficiently and retain them in memory. However, the original finding, that deficient nonword repetition is a strong correlate of SLI, has stood the test of time, to the extent that poor performance on this test has been used successfully as a marker of a heritable phenotype in molecular genetic studies of SLI (Newbury, Monaco, & Bishop, 2005).

According to the theoretical framework presented by Gathercole (2006), word learning should proceed slowly in SLI, and this is indeed usually the case. Nevertheless, for most children, vocabulary is less impaired than syntax (see Leonard, 1998, for review). This raises the question of whether phonological STM is implicated in acquisition of syntax as well as vocabulary.

This issue was raised in 1998 by Baddeley, Gathercole, and Papagno, who presented a theory of the phonological loop as a language learning device, arguing that the ability to retain small amounts of phonological information in STM evolved as a human characteristic that had selection advantage because it facilitated language acquisition. In that paper they reviewed evidence that, in typically developing children, nonword repetition, a measure of phonological loop capacity, was related not only to vocabulary learning but also to acquisition of syntax.

This theoretical account was attractive to those working on SLI, because it suggested that a deficit in a single specialized memory system could potentially account for the whole gamut of linguistic deficits seen in language-impaired children, without needing to invoke domain-specific impairment of specialized

syntactic mechanisms (Joanisse & Seidenberg, 1998). It is possible to formulate at least two hypotheses that predict a link between weak nonword repetition and poor syntax in SLI. The first corresponds to a storage account, and maintains that incoming speech needs to be held in a temporary buffer while syntactic analysis is carried out. This operation will be hampered if there is rapid decay of phonological material in the short-term store. The second hypothesis predicts that poor syntactic abilities will follow if the child persists in analyzing incoming speech at the level of the syllable, rather than identifying individual phonemic segments. As Gathercole (2006) notes in her Keynote, this could lead to poor nonword repetition; however, in addition, the child may not recognize that words such as “walked,” “hopped,” and “laughed” all end with the same sound, and thus fail to extract a morphosyntactic rule of past tense formation.

Although both hypotheses seem plausible, more recent work suggests that they are almost certainly wrong. The first indication of this came from simple correlations between measures of nonword repetition and syntax in children with SLI, which were far weaker than would be predicted if phonological STM were a major factor in syntax acquisition. For instance, Norbury, Bishop, and Briscoe (2001) found no significant correlation between nonword repetition and a measure of production of verb inflections in 14 children with SLI aged from 7 to 10 years. More powerful evidence for independence of phonological STM and syntax comes from Bishop, Adams, and Norbury (2006), who studied a sample of 173 6-year-old twin pairs, selected to include a high proportion of children with language impairments. Twins provide a means of disentangling genetic and environmental influences on language ability. Monozygotic (MZ) twins are genetically identical, whereas dizygotic (DZ) twins share on average 50% of alleles from segregating genes (i.e., genes that have different allelic forms in different people). If genes are implicated in causing a disorder, we expect cases of concordance (i.e., both twins have the disorder) to be more numerous in MZ than in DZ twins. More complex methods of analysis can be used to study quantitative scores on language measures, to estimate the extent to which deficits are under genetic influence. Furthermore, by looking at cross-concordance across language measures, one can test whether two different deficits are influenced by the same genetic factors. Bishop et al. (2006) first looked at nonword repetition. Heritability of poor nonword repetition was not as high in this 6-year-old sample as in previous samples of older twins. However, it was noted that in 6-year-olds, nonword repetition was correlated with articulation skills, and the pattern of errors suggested that some children did poorly because they could not produce the sounds correctly even in very short nonwords (see also Sahlén, Reuterskiöld-Wagner, Nettelbladt, & Radeborg, 1999). To obtain a purer index of phonological STM, a derived measure was computed, reflecting the score on longer nonwords, after adjusting for level of performance on the shortest nonwords. This measure was more sensitive to language impairment and gave higher estimates of heritability than the raw unadjusted measure, incidentally supporting Gathercole’s (2006) claim that articulatory limitations do not explain the link between SLI and nonword repetition. The next step was to look at heritability of other language measures, including tests of syntactic ability. These, too, were highly heritable. However, the correlation between syntactic measures and phonological STM, although significant, was weak ($<.3$), and bivariate genetic

analysis suggested that different genes were implicated in causing risk for weak phonological STM and poor syntactic skill. This result throws into question theoretical accounts that aim to explain all the linguistic deficits in SLI in terms of an underlying limitation of phonological STM capacity.

A remaining puzzle is why there should be any correlation between phonological STM and syntax if they have independent origins. A hint comes from the finding by Gathercole, Tiffany, Briscoe, Thorn, and The ALSPAC Team (2005) that one can have children who have poor nonword repetition skills but normal scores on language tests. This is surprising, given that deficient nonword repetition is a strong correlate of language impairment. One possible explanation raised by Gathercole (2006) is that weak phonological STM may lead to SLI only if it is accompanied by other cognitive risk factors. Our data on syntax fit well with that interpretation, and suggest that the child who just has weak phonological STM or just has slow mastery of syntax may show few overt language difficulties, but if both these deficits occur in combination, the impact on everyday language skills is much more severe.

The field has shown considerable advances based on the initial insights of Gathercole and colleagues, who recognized that the deceptively simple task of repeating nonwords taps a fundamental building block of language learning. However, the more we learn, the clearer it becomes that any single factor explanation of SLI is inadequate to explain the phenomenon.

REFERENCES

- Baddeley, A., Gathercole, S., & Papagno, C. (1998). The phonological loop as a language learning device. *Psychological Review*, *105*, 158–173.
- Bishop, D. V. M., Adams, C. V., & Norbury, C. F. (2006). Distinct genetic influences on grammar and phonological short-term memory deficits: Evidence from 6-year-old twins. *Genes, Brain and Behavior*, *5*, 158–169.
- Gathercole, S. E. (2006). Nonword repetition and word learning: The nature of the relationship [Keynote]. *Applied Psycholinguistics*, *27*, 513–543.
- Gathercole, S. E., & Baddeley, A. D. (1990). Phonological memory deficits in language disordered children: Is there a causal connection? *Journal of Memory and Language*, *29*, 336–360.
- Gathercole, S. E., Tiffany, C., Briscoe, J., Thorn, A. S. C., & The ALSPAC Team. (2005). Developmental consequences of poor phonological short-term memory function in childhood: A longitudinal study. *Journal of Child Psychology and Psychiatry*, *46*, 598–611.
- Goldman, R., Fristoe, M., & Woodcock, R. W. (1974). *Goldman–Fristoe–Woodcock Auditory Skills Battery*. Circle Pines, MN: American Guidance Service.
- Joanisse, M. F., & Seidenberg, M. S. (1998). Specific language impairment: A deficit in grammar or processing? *Trends in Cognitive Sciences*, *2*, 240–247.
- Leonard, L. B. (1998). *Children with specific language impairment*. Cambridge, MA: MIT Press.
- Newbury, D. F., Bishop, D. V. M., & Monaco, A. P. (2005). Genetic influences on language impairment and phonological short-term memory. *Trends in Cognitive Sciences*, *9*, 528–534.
- Norbury, C. F., Bishop, D. V. M., & Briscoe, J. (2001). Production of English finite verb morphology: A comparison of SLI and mild–moderate hearing impairment. *Journal of Speech, Language, and Hearing Research*, *44*, 165–178.
- Sahlén, B., Reuterskiöld-Wagner, C., Nettelbladt, U., & Radeborg, K. (1999). Non-word repetition in children with language impairment—Pitfalls and possibilities. *International Journal of Language and Communication Disorders*, *34*, 337–352.

Dorothy V. M. Bishop
University of Oxford