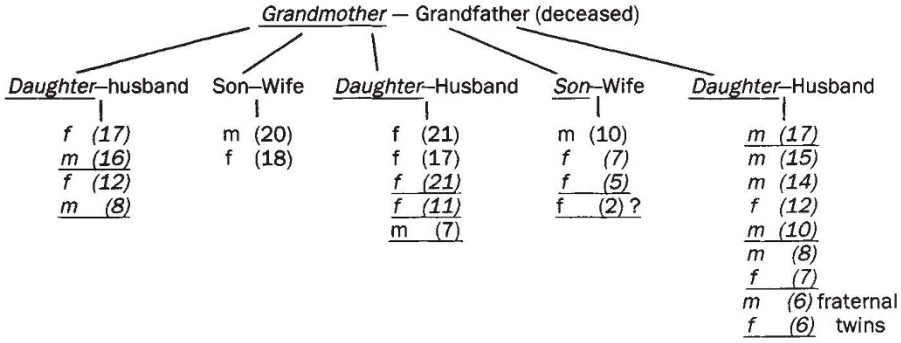
Feature-blind grammar and dysphasia

SIR—Developmental dysphasia — the inability of apparently normal children to acquire language normally — is well-known, but precisely what is wrong with their language and the causes of their errors are not. I now describe the outcome of testing for dysphasia the members of a large family spanning three generations (results shown in the figure).



Subjects underlined were those diagnosed as dysphasic. Italics denote those subjects that were part of this study, age of subjects is given in parentheses. The two year old had been only tentatively diagnosed as dysphasic.

I administered fourteen tests, adapted from a battery of tests for aphasia[1], to almost all the members of the family, both normal and dysphasic (the former provided a control group), I also gathered data from samples of writing and from interviews. Not all language skills were equally impaired.

Four of the tests required aptitude with syntactical-semantic features. The responses of the dysphasic members of the family to those tests were significantly different from those of the normal ($P = 0.01$), but the responses of the two groups to the other tests were not significantly different. For several of the tests, the responses of 15 independent normals were available, but did not differ significantly from the responses of the normal family members.

By way of illustration, the dysphasics do not differ significantly from normals in their ability to point correctly to pictures instantiating reflexives ("He washes him" versus "He washes himself"), possessives ("The mother's baby" versus "The baby's mother") and negative passives ("The car is not being pulled by the truck"). But the two groups differ significantly in their ability to change tenses, to construct regular plurals for nonsense words, to detect a particular class of grammatical errors and to correct them.

For example, in a tense-changing test consisting of 10 items such as "Every day he kisses his nanny. Yesterday he ———", the median score of the normals is 9, for the dysphasics, 3. Similarly, in a test of grammatical features consisting of 30 items such as "The boy eats three cookies", the median score for normals is 29 but that for dysphasics only 18.

While the language of dysphasic adults seems normal at first sight, careful testing shows that the normality is only apparent. Those affected may have learned strategies for coping with language, but their underlying grammar is still severely impaired. They may, for example, produce correct plurals for known words, but they lack a general rule for producing plurals. The dysphasics perform as normals in a task requiring that they point to "The book" rather than "The books", but when they are given a picture of an imaginary animal called a "wug", they cannot say whether a group of these animals would be called "wugs".

This and further evidence from the samples of writing suggests that the dysphasics, instead of being able to infer general rules about the signifiers of grammatical features from a few salient examples as normal children do, must learn each word as a separate lexical item. They have learned that the word "books" refers to several objects used in reading in much the way that normal speakers have had to learn that "children" refers to several young people. They appear not to know that there is a general feature of English grammar for signifying the number of nouns, determiners and verbs.

These findings are consistent with others. Clahsen[2], using data from the German language, and I (using data from refs 3 and 4) have shown that the language errors typical of dysphasia can be accounted for by the impairment of one particular grammatical faculty — the accurate usage of syntactical-semantic features of language such as the significance of number, gender, animacy, proper names, tense and aspect. Grammatically consequent skills are also impaired, as in the selection of apt determiners and the omission of subject pronouns before untensed verbs. Yet other grammatical skills, such as the judgement that the sentence "He puts" is ungrammatical, are unimpaired.

The deficits with which we are concerned show up in spontaneous speech, writing, grammatical judgement and repetition. Because the deficits are apparent in all aspects of language, their roots probably lie in the underlying grammar rather than in a peripheral processing system. Because the language skills that are not impaired are at least as complex as those which are, it is unlikely that the underlying deficit is one of cognition as such.

The distribution of dysphasia in this one large family suggests that it may be due to one dominant gene; M. Pembry (Institute of Child Health, London) is searching for appropriate genetic markers. Though there has not, to my knowledge, previously been such a clear family history of dysphasia, there have been anecdotal observations of dysphasia in different members and different generations of the same family.

Naturally, a single family cannot prove that the dysphasia described here is genetic, but we shall soon be undertaking a study of the patterns of occurrence of dysphasia in families and testing our feature-blindness hypothesis on a larger scale. A similar study of genetic factors in severe language disorders is being followed by B. Sahlen (Lund University, Sweden); R. Frackiowak (MRC Cyclotron Unit, Hammersmith Hospital, London) and D. Bub (Montreal Neurological Institute) will be investigating the neurological correlates of the disorder and F. Vargha-Khadem (Institute of Child Health) will be looking at cognitive aspects of the deficit.

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1. Paradis, M. in *The Assessment of Bilingual Aphasia* (Erlbaum, Hillsdale, New Jersey, 1987).
2. Clahsen, H. *Linguistics* **27**, 897–920 (1989).
3. Gopnik, M. *A Featureless Grammar in a Dysphasic Child (McGill Working Papers in Linguistics*, McGill University, Montreal, 1986).
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Folding proteins

SIR—Recombinant DNA technology and protein engineering make it feasible to produce substantial quantities of any protein with a specified amino acid sequence, but many potentially useful applications are impeded because the protein is produced in inclusion bodies, in an insoluble unfolded form that must be solubilized and folded to yield a biologically active product[1]. Often, for reasons which are not clear, the solubilized protein will not fold as readily as would be expected. Our experience with the production of recombinant bovine pancreatic trypsin inhibitor (BPTI) may suggest a possible explanation for inclusion body formation that has implications for the methods used to