

GEOSECS and Transient Tracers in the Ocean (TTO) studies considered by Fanning. Our results are presented in the table and suggest that the surface waters of this area of the Sargasso Sea are deficient in both nitrogen and phosphorus for most of the time, rather than phosphorus-deficient as suggested by Fanning. The two data sets are different in that one provides geographical coverage and the other is a time series at a single point, but we would argue from the station S data set that the situation suggested by Fanning is not necessarily the general case.

A more fundamental point, however, involves the extrapolation of nutrient detectability, as described above and in Fanning's paper, with nutrient limitation because there is clear evidence that high rates of primary productivity can be sustained at very low nutrient concentrations by intense nutrient recycling⁴. Therefore considerations of nutrient limitation based on rates of nutrient consumption and carbon production would be more appropriate.

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Genetic basis of grammar defect

SIR—Vargha-Khadem and Passingham¹ and Fletcher² in their responses to my Scientific Correspondence present no data or arguments to challenge my hypothesis³ that there is a deficit in the ability of my subjects to construct an underlying grammar for abstract morphemes like number and tense. They suggest that other subjects in this heterogeneous clinical category have other problems. That is to be expected, but it is also true that the kind of errors that I cite are widely reported in the literature. They say that the subjects in the family in question make additional kinds of errors, some of which I would dispute, but they fail to show that they are relevant to the hypothesis which I have proposed.

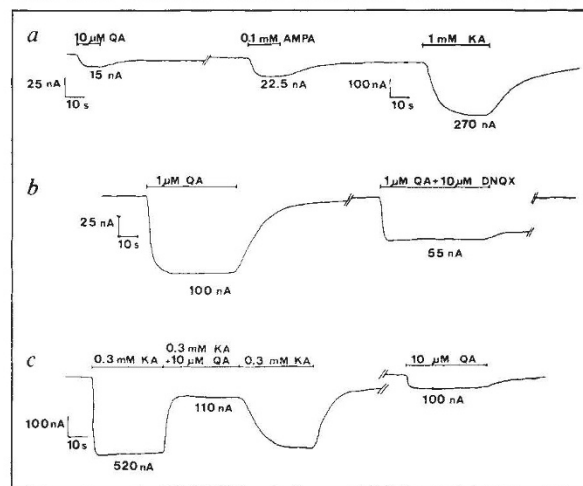
What really is at stake here are not the data, but rather how to account for them. My explanation hypothesizes that the subjects are impaired because they cannot

How many NMDA receptors?

SIR—The distinction between glutamate receptors coupled to ion channels is based on their selective activation by different agonists. Three main subtypes have thus been defined: NMDA, kainate and

response suggests the existence of two separate non-NMDA receptors.

We have further characterized the GluR-K1 clone expressed in oocytes and find that it indeed responds to kainate



but also to quisqualate (see figure). Responses induced by kainate and quisqualate are blocked by DNQX, and GluR-K1 has a higher affinity for quisqualate than for kainate. (The maximally effective quisqualate concentration elicited only about a tenth of the maximal response to kainate.) During a prolonged application of kainate, addition of quisqualate reduces the recorded current to the level obtained with quisqualate alone (see figure). Currents induced by kainate, quisqualate and domoate display similar sensitivity to membrane potential and show pronounced inward rectification.

Our demonstration that a single protein expressed from a single complementary DNA responds to quisqualate and kainate with the same current-voltage curve supports the hypothesis that the *in vivo* responses to these agonists are mediated by the same non-NMDA glutamate ionotropic receptors.

Currents induced in *Xenopus* oocytes injected with GluR-K1 transcripts. Records from oocytes clamped at -60 mV. *a*, Responses to quisqualate (QA), kainate (KA) and AMPA (5 ng transcripts). *b*, Antagonism of QA by DNQX. *c*, During a prolonged application of KA, QA reduced the KA response to the level attained with QA alone. Traces *b*, *c* are from the same oocyte injected with 20 ng transcripts. Technical details available from J.R. on request.

quisqualate receptors^{1,2}, although the last two may activate the same receptor³. The recent report by Hollmann *et al.*⁴, of the cloning of GluR-K1 complementary DNA encoding a protein of relative molecular mass 100,000 which, after *in vitro* transcription and expression in *Xenopus* oocytes, induces kainate but no quisqualate

current-voltage curve supports the hypothesis that the *in vivo* responses to these agonists are mediated by the same non-NMDA glutamate ionotropic receptors.

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construct language rules at the necessary level of abstraction⁴. Your correspondents^{1,2} prefer an explanation which hypothesizes that the subjects are impaired because they cannot hear or cannot pronounce certain sounds, and this indirectly affects the grammar carried by these sounds. A wide range of data can be explained by my hypothesis but not by theirs. The data clearly show that it is not the marker *ed* or *s* which is impaired, but the underlying grammatical categories of number and tense. Moreover, the data confirm that all such features are impaired, even those like aspect, for which the surface marking is produced.

There is now evidence from twin studies⁵ and from large statistical studies⁶ which is consistent with the hypothesis that developmental language impairments may be linked to an autosomally dominant

gene. The exciting question is what does this gene do? Does it affect some peripheral aspect of language processing which only indirectly has consequences for language, or does it directly affect the capacity to acquire language? The abstract morpheme hypothesis supports the latter view and provides a more complete account of the data.

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