

MMTV will be of particular interest in relation to our present proposal.

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Prediction of protein structure from sequence

SIR—In a recent News and Views article, Stephen Harrison¹ urges caution "in predicting aspects of protein structure from sequence, what we need to recognize are entire domains or significant sub-domains—not just elements of secondary structure". He also points out "The poor correlation between local sequence and secondary structure has been aptly demonstrated by Kabasch and Sander², who show that the same pentapeptide in different proteins has fundamentally different backbone configurations in each".

I wish to point out two items which should caution the reader against accepting such advice without question.

In our original paper on the prediction of protein secondary structure³ (also see ref. 4), it was pointed out with great care that any specific sequence, for example five residues, will assume a conformation dependent upon the neighbouring sequences on both the amino and carboxyl termini until a sequence is reached, of usually four residues, which will either change conformation (β -turn), or will be unable to maintain a secondary structure [$\langle P_{\alpha} \rangle$ or $\langle P_{\beta} \rangle < 1.0$]. Thus it is an expected observation, rather than a novel finding, that a pentapeptide, joined to different sequences may assume dissimilar conformations. Thus the Kabasch and Sander² discovery, used to discredit predictive schemes, reaffirms rather than discredits the predictive algorithm. Harrison's¹ emphasis on domain structure rather than secondary structure to evaluate biological functional units has been expounded with great elegance by Rossman⁵ and is a well accepted theme. However, domains are constructed by in-

teractions of secondary structures, so let us not put the cart before the horse. To understand the formation of domains one must know the secondary structures first. Thus before one has a complete three-dimensional structure of a protein, derived from X-ray crystallographic studies, it can be extremely useful to predict secondary structures and look for homologies with other proteins to find interesting relationships.

I agree with Harrison¹ that the paper of Kabasch and Sander² should be carefully read as it displays how a false conclusion can be drawn from an interesting fact.

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On the possibility of inductive probability

SIR—We here make a second attempt to explain the reason for our hesitation in accepting the Popper-Miller thesis¹. We agree with all of their mathematical relations. Our problem is with the interpretation. We offer again two reasons and frame them here without using the negative signs of our first letter², which appear to have drawn excessive attention to themselves.

First, consider the (correct) result for $p(e) < 1$

$$s(h \leftarrow e, e) \equiv p(h \leftarrow e|e) - p(h \leftarrow e) < 0 \quad (1)$$

Popper and Miller identify the ampliative (or "loosely speaking inductive") part relative to any evidence e of any hypothesis h , with $h \leftarrow e$ given e . They infer from (1) that that part of h which goes beyond e is always countersupported by e . (We use their wording in both these sentences.) Our difficulty with this is that $s(k \leftarrow e, e) < 0$ for all values of k and in particular for $k = h$ and $k = \bar{h}$. One might have expected that if the inductive part relative to e of h is countersupported by e , then it would at least be possible for the inductive part relative to e of \bar{h} to be supported by e . But on Popper and Miller's interpretation the inductive part relative to e of both h and \bar{h} is always countersupported by e . This suggests to us that there is something astray with this interpretation, and it led us to observe in ref. 2 that a different definition of terms or some restriction on their interpretation seems to be required. Our interpretation of (1) was explained in our comment on our theorem A, and is rather simple: Since $p(h \leftarrow e)$ still allows \bar{e} to occur, (1) shows that this probability is reduced if \bar{e} is ruled out.

Second, one can understand the import-

ance of discussions of the extent to which 10 or 20 supportive occurrences e support a hypothesis h as a general statement, as is done in statistical theory. If a penny comes up "heads" 10 times (e_1, e_2, \dots) is it a penny with two heads (h)? There is a structured relation between e and h . It is, however, difficult to see how statements about induction can go very far if they are based only on probability algebra which leaves h and e as arbitrary propositions. In our letter we noted that the arguments in this correspondence had used only probability algebra, and that they involve the handicap of being independent of any inferential relationship between e and h .

Mathematically speaking, we would say that the support function $s(x, y)$ is indeed useful, as we noted in our comments on our theorem B. It is its division into the suggested "deductive" and "nondeductive" or (loosely speaking) inductive components (and particularly, the interpretation of the letter) that has given rise to our difficulties.

We are grateful to Karl Popper and David Miller for the courtesy of some private exchange of opinions.

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The form of maps in the brain

SIR—Further to Professor Ettliger's pertinent comments on flying fox somatotopy¹, may I suggest that, if we accept the approach of metric tensor modelling for cortex as well as cerebellum², there would seem to be no requirement for any particular coordinate reference frame, provided that the sensorimotor connections to the central nervous system always preserve the tensorial relationships. Admittedly, my personal interpretation of the metric tensor model leaves me asking why we (and presumably some other vertebrates) need to construct a cartesian four-dimensional space-time at all.

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Selfish DNA and the origin of introns—Correction

IN this piece of Scientific Correspondence (by T. Cavalier-Smith, *Nature* **315**, 283-284; 1985) the word "not" was omitted from line 14 on page 284. The sentence should begin: "But nuclear tRNA does not depend on specific intron sequences . . ."