MATTERS ARISING

Rebuttal of criticisms of remote viewing experiments

MARKS[1] has argued that our first two remote viewing (RV) studies (with subjects Price and Hammid)[2] should be discounted as evidence for the RV hypothesis. He claims that sensory cues in the subject-generated RV transcripts provide sufficient information to permit judges to blind-match transcripts to target sites on an artefactual basis. The evidence, however, does not support this claim.

First, with regard to the Hammid series. When one examines the details of Marks' criticism (see ref. 3) one finds that the criticism about cueing is based on an error-in-fact on the part of Marks; he erroneously assumed that the target list given to judges was in the order of target usage, that is, unrandomized. That is incorrect. That list, which Marks used to formulate his criticism, was randomized by random number generator to prevent the very possibilities he raises. When this fact is taken into account, one finds that his criticism is voided at its foundation. Specifically, his criticism is derived from the fact that the transcripts were dated; however, the dates do not provide the useful cue information posited by Marks—they would have done so only under the (incorrect) assumptions about target order made by Marks. As a result, Marks' attempt to associate the transcripts to the target sites on the basis of the alleged cues failed.

With regard to the Price series. While not questioning the data collection procedures or commenting on the near-photographic accuracy of some of the individual descriptions, Marks and Kammann hypothesized in their first critique that the successful judging of the Price series might be due to sensory cues in the transcripts that provide information as to, for example, whether a given trial was early or late in the series[4]. In response to this we arranged to have the series independently rejudged, using transcripts from which the suggested cues had been removed. The rejudging resulted in the same seven out of nine target/transcript matchings as in the original study, indicating that it was the quality of the transcripts themselves, rather than the presence of cues, that was responsible for the successful outcome[2]. This rejudging is rejected by Marks because of the potential confounding factor of the publication of the original study.

This leads us to a second re-analysis of the Price series for which, in order to give the Marks' hypothesis its best chance, we assume all potential cues remain and are used to maximum advantage; we then rigorously assess the consequences. In the strongest case for cues the probability of a correct match can at most be increased from one-ninth to one-third; in another case from one-ninth to one-seventh; and in two other cases from one-ninth to one-eighth. The remaining five cases gain no direct advantage from cues, just indirect advantage from not using as possibilities the ones already used in the more advantageous cases. When one takes into account the resulting constraints due to cues, the number of possible target/transcript matchings is reduced from $9! = 362{,}880$ to 68,760 combinations, by exact count. Assuming that the cues are used to maximum advantage, we find that the significance level associated with obtaining at least seven matches in nine trials (as was done) in a forced-choice, non-independent assignment of transcripts to target sites (the most conservative statistic) is only reduced from $P = 10^{-4}$ to $P = 3.9 \times 10^{-4}$, still a quite significant result. (A paper containing the details of this analysis has been published elsewhere[5].) Thus, the Marks hypothesis that the presence of extraneous cues accounts for the significance of the Price series result receives no support.

We would summarize by first indicating that we recognize that our original RV studies are not without flaw, and the potential effect of possible cues deserves critical examination. However, in our continuing re-examination of this work we find that the suggested cueing artefacts, although providing some potential for confounding, are yet well below the magnitude necessary to account for the strength of the results. As a result, this re-examination of the data has provided yet additional support for the hypothesis indicating the existence of a human remote sensing capability. This conclusion is supported by the continuing replication of RV experiments in our own and other laboratories[6,7] for which the Marks concerns no longer apply due to the increased sophistication of the procedures in use.

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Expression of herpesvirus-induced antigens in human cervical cancer

DREESMAN *ET AL.*[1] have described the expression of two herpes simplex virus type 2 (HSV-2) antigens, designated ICSP 11/12 and ICSP 34/35, in 38% of cervical tissues with pathological findings of severe dysplasia or carcinoma. Their undocumented statement that previous studies used antisera against HSV virions and gave rise to equivocal results is inaccurate and generates controversy where none exists. Indeed, the expression of HSV-2 antigens in cervical anaplastic tissue has been consistently and unequivocally demonstrated using antisera prepared against extracts of HSV-2-infected cells that contain both structural and non-structural viral proteins. To our knowledge, there is no report describing the failure to identify HSV-2 antigens in cervical anaplastic tissue when such have been studied. Had the authors' comment on the equivocal nature of previous results been valid, their data demonstrating the presence of two antigens, the viral identity of which may be questionable to some critics, in only 38% of patients with anaplastic findings could hardly support their conclusion that HSV-2 is a factor in human cervical cancer[1].

Using antisera against total viral antigens, HSV-2 antigens were demonstrated by immunodiffusion and crossed immunoelectrophoresis in extracts of cervical tumour cells[2–4]. Similarly prepared antisera were used by Athanasiu *et al.*[5] in immunofluorescent staining of frozen sections of cervical tumour biopsies. Expression of HSV-2 antigens was described in 8 of 24 (33.3%) specimens from cervical carcinoma, a percentage remarkably similar to that described by Dreesman *et al.*[1], using similar specimens. The observation that viral antigens are preferentially expressed in cells on the peripheral part of the carcinoma mass towards the vagina and/or cervical canal[6,7] has led some investigators to conclude that the proportion of reactive patients could be greatly increased by studying the cervical cells that have naturally exfoliated or have been induced to exfoliate artificially. Immunoflorescent staining of such specimens with antisera against total viral antigens in six independent studies[6–11] has demonstrated the expression of HSV-2 antigens in cells of 61–81.2% of patients with dysplasia and 92–100% of those with invasive cancer or carcinoma *in situ* (CIS). This compares with a reactivity of 0–9.4% in normal women (Table 1).

We showed previously that antisera against an antigenic fraction designated AG-e, that consists of two viral proteins (ICP 12, ICP 14)[12], stains cervical anaplastic cells from patients with dysplasia, CIS or invasive cancer, but not from normal women[10]. Consistent with this finding, antisera against ICP 12 or ICP 14 also stain the cells from patients with these pathological findings[11]. It is significant that ICP 12 shares a number of

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