

carefully-compiled homogeneous samples of QSOs.

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STEWART AND HAWKINS REPLY—it seems clear that there is no essential disagreement between Wills' response and the primary result of our original paper¹; that if one takes the evolutionary law and luminosity function for quasars widely found in the literature and models the (*L*, *z*) plot one finds a disagreement with the observations, whether one uses the large list of Burbidge *et al.*² or the complete sample of Wills and Lynds³, in the sense that there is a shortage of very luminous objects. The question now arises as to why this disagreement with the observations exists. Wills accepts that the evolution law and luminosity function are correct and solves the problem by involving a 'cutoff' in the luminosity of quasars, that is by saying that that for some reason a quasar cannot be more luminous than 10^{24} W Hz⁻¹ at 2,500 Å.

An attempt was made¹ to discuss the distribution in the (*L*, *z*) plot without invoking a cutoff by relaxing some of the assumptions, in particular by allowing the evolution rate to be an adjustable parameter and maintaining the luminosity function unchanged, although allowing rather large error bars. Wills is not strictly correct in saying that the two cannot be distinguished as it is clear¹ that the slope of the upper envelope in the (*L*, *z*) diagram at low redshift is determined by the index of the luminosity function alone. The result¹ was that the distribution of quasars in (*L*, *z*) could be accounted for very naturally with low or zero evolution, and in particular the absence of very luminous objects was shown to be a simple geometrical effect, thus removing the need for a cutoff in quasar luminosity.

It is obvious that if one regards the results of previous measurements of quasar evolution as being beyond reproach one is forced to introduce a luminosity cutoff and all Wills' results follow. However, as this widely held position is based on the results of a single test—the *V/V_m* test—it seems to us that one ought to look more closely at this and

ask if it is really giving the right result. This question is treated more fully in another paper⁴.

If one insists that the *V/V_m* test is giving the right answer then Wills is quite right and a cutoff, evolution and so on are all required. However, given the results of ref. 1 it seems desirable to re-examine the *V/V_m* test, as it may be possible to abandon two *ad hoc* hypotheses, that is, quasar evolution and luminosity cutoff, if its results turn out to be incorrect.

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Mixed ligand chelate therapy for plutonium and cadmium poisoning

I HAVE accumulated information leading me to doubt the veracity of results published in a report¹ and a reply² of which I was the co-author. I have repeated the experiments personally, and thus far find that in the stated conditions¹ I do not confirm these results. The experiments reported for cadmium poisoned mice, for example, did not, contrary to data provided me, include experimental controls for the individual chelants at the stated doses. Further, treatment was given 5 min post-Cd and not 1 h, as stated. At the latter time no survival is observed. Neither can I confirm the results described for plutonium decorporation by diethylenetriaminepentaacetic acid/salicylic acid (DTPA/SA). In fact, from potentiometric titration studies which I now carry out as a screening method for the identification of mixed ligand chelate (MLC) systems potentially useful for biological applications, I find that the DTPA/SA systems do not form significant levels of MLCs with a cation similar to Pu(IV), namely Th(IV). However, DTPA and catechol or Tiron do form MLCs but that with EDTA instead of DTPA is superior.

I believe that MLC phenomena constitute a potentially superior approach for the treatment of metal poisons. However, the extent to which this is realised will be determined by the results of my own experiments and those of others. In the meantime, I am forced to conclude that the previously published results¹ are invalid.

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Mixed ligand chelation therapy

SCHUBERT AND DERR¹ have reported that mixed ligand chelates (MLCs) of DTPA and salicylic acid (SA) markedly enhance the clearance of ²³⁹Pu from mice. We have examined this procedure in rats (196–224-d-old females, HMT strain, from the MRC Radiobiology Unit, Harwell) using similar injection procedures and similar analytical procedures. Analysis for ²³⁹Pu in excreta from animals caged singly, six animals per group, as well as the kidneys, livers, spleens and carcasses, showed that there was no significant difference between either the DTPA group or the DTPA/SA group.

We have also investigated the extraction of ²³⁹Pu into heptane and found no evidence that Pu(EDTA)SA is lipophilic. Unfortunately the extraction procedure was not described; however, our extraction procedure has been used routinely to demonstrate the interaction of ²³⁹Pu⁴⁺ with phospholipids².

Furthermore, we have found that intravenously injected NaSA (2.0 mmol per kg) is acutely lethal to rats and it is known that 10–30 g of NaSA is lethal to man³; the injection procedure of Schubert and Derr would necessitate intravenous administration of 22 g of NaSA to man every 3 d.

After completing our work we learnt that Schubert had withdrawn some of his claims, but we still view his optimism for MLC therapy with caution. For MLC therapy to be successful it will require administration of a chelating agent sufficiently lipophilic to offset the hydrophilicity of the Pu DTPA complex. There is no reason to believe that such diverse species enter cells in synchrony; in addition, we might wonder if serum citrate could not be part of an MLC with Pu and DTPA. It is more likely that synergistic chelation therapy⁴ will contribute more to toxic metal decorporation than MLC therapy.

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