

cells. In some cases structural components of the virus perhaps act as the ionophores^{1,8,9}, while in others new proteins may need to be synthesised. The many phenomena observed in practice would then be merely the different facets of a fundamental gear-shift in polyelectrolyte-counterion interactions within the cell.

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CARRASCO AND SMITH REPLY—We find the proposal by Durham that changes in calcium ion concentration following infection by viruses are responsible for alterations in host cell functions very attractive. Our model to explain the shut-off of protein synthesis, which was briefly summarised in our *Nature* paper¹, and which is the subject of a more detailed paper², is in fact very similar. We also favour a model in which viral structural proteins form lattices in the cellular membrane and leave holes which act in the same way as some ionophores to allow the passage of ions².

We believe, however, that the shut-off of host cell protein synthesis is best explained by a change in Na⁺ ion concentration within infected cells, and we presented experimental evidence showing that sodium ions can have a differential effect on protein synthesis *in vitro* and indirect evidence that such changes in sodium ion concentration could occur *in vivo*¹. While we have not examined the effects of calcium ions in detail, a preliminary screening of the effects of many mono- and divalent cations on protein synthesis *in vivo* and *in vitro* failed to reveal a differential effect by calcium ions.

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Sodium emission in persistent meteor trains

Using theoretical estimates of the rate coefficient of the association of Na with O₃, Kolb and Elgin¹ have shown that the catalytic effect of sodium in releasing the store of recombination energy of free atmospheric atomic oxygen is more powerful than was previously assumed². Drawing on evidence from measured Na D-line intensities, Kolb and Elgin estimate a branching ratio for the production of excited Na(²P) from NaO reduction of $f \sim 0.05$, which, using the model previously suggested² implies that at 90 km sufficient photon emission occurs for an enduring train to result from a meteor of magnitude about -6. It is instructive to relate this estimate to observation. It is known that the trains of small duration show an initial emission decay at 90 km of about 0.2 mag s⁻¹ and since the radiation probably results from the interaction of ionic constituents of the train so that the reaction rate varies inversely as the meteor column cross-sectional area and hence inversely as time, a decay of 5 mag would be expected between about 1 s and 100 s. We may infer that any train luminosity having a duration $t \gtrsim 100$ s must be due to a catalytic (sodium) mechanism. In Olivier's catalogue⁴ 55% of trains ($t > 10$ s) were produced by high velocity shower meteors, Perseids, Orionids and Leonids. In the duration-magnitude characteristics given for 3700 trains by Millman⁵ the average meteor magnitude corresponding to a duration of 100 s is in the range -7.5 to -5 for velocities 60–70 km s⁻¹. From the extensive surveys of Hoffmeister (see ref. 4) and Olivier, one visual meteor in 780 results in a train of duration $t > 10$ s. Using ref. 5 the difference in magnitudes between meteors responsible for trains of 10 s and 100 s duration is 2.5 implying a corresponding incident meteor flux ratio⁶ of 27:1. Since the mean visual meteor rate⁶ to a single observer is 9.7 h⁻¹ the observed occurrence frequency of trains $t > 100$ s is 4.6×10^{-4} h⁻¹. In comparison the cumulative flux of meteors having magnitudes brighter than -6 for an individual observer is, using Hughes⁹ 3.1×10^{-4} h⁻¹. Though there are uncertainties in [O₃] and f and variations in Na abundance, the sodium cycle process is strongly supported as a source of persistent meteor train luminosity. This conclusion may be viewed in the light of the review of train characteristics by Hughes⁷.

Hughes considered evidence for the mechanisms responsible for persistent meteor train emission and on the basis of visual observations^{8,9} concluded that

the evidence supported a mechanism that is closely associated with the level of meteoric ionization rather than with meteoroid mass or meteor luminosity. It is of importance to emphasise that based on the observations^{8,9} the conclusion of Hughes is inappropriate. The results of Lindblad⁸ for Perseid meteor trains are confined to very short durations, t : for only 2% of the trains was $t > 3$ s while for 90% $t < 1.5$ s indicating that the observed light was associated with the meteor wake emission and in particular the well known OI 5577 Å feature. Indeed the data of Lindblad permit the determination of the effective lifetime τ (decay constant) of the emitting species. It is straightforward to show that the gradient of train duration-meteor magnitude plot is given by $-\tau(\log_e 10)/1.51$ and using Lindblad (Fig. 11B) $\tau = 0.21 \pm 0.05$ s. In comparison, the radiative lifetime of the OI (¹D₂ ← ¹S₀) transition is 0.74 s. However the great majority of observed trains were in the 90–100 km height interval (Lindblad, Fig. 18) where deactivation of the O(¹S₀) state most effectively occurs with O and O₂. Using recommended quenching coefficients and known atmospheric concentrations the quenching rate at 95 km is ~ 2.9 s⁻¹ resulting in an effective emission lifetime of 0.23 s. Plavec⁹ does not state the durations of trains in his analysis. We note, however, that according to Plavec (a), the trains disappeared in a few seconds, (b), there was concern with the effects of observer reaction times on duration measurements (c), 50% of Perseid meteors yielded trains in contrast to the much lower proportion at lower meteor velocities. The results indeed coincide with what is now known about the green line emission (the 5,577 Å feature was not identified until 1958). It is quite clear that the observations of Lindblad and Plavec do not refer to the true rare enduring train phenomenon.

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