matters arising

Culture and genetic variability

CHAKRABORTY found[1], by using gene frequencies for seven serological markers to compute genetic distance, that 'only geographical distance seems to be important, explaining the trend in genetic variabilities' in seven Chilean Indian populations. The correlation coefficients he presented were 0.716, 0.775, and 0.901, for correlations between genetic distance–geographical distance, genetic distance–cultural dissimilarity, and geographical distance–cultural dissimilarity, respectively. He then presented the results from a stepwise regression, and found that while geographical distance explained 51.31% of the genetic variation, cultural dissimilarity explained only an additional 8.88%. He did not, however, use as the first variable in the stepwise regression the one which had the highest correlation with the dependent variable. Culture alone accounts for 60.06% (r^2) of the genetic variation in these data. If geographical distance is then added, the additional variation explained would be less than 0.2%. These figures are, of course, approximations subject to slight rounding error from the published tables. It is indeed difficult to assess the relative importance of variables which are themselves highly correlated, as Chakraborty states. But his data clearly do not support his conclusion that of the variables measured, only geographical distance is an important correlate of genetic distance. From these data, culture is at least of equal importance.

It would be profitable to use these data to consider the relative effects of genetic and geographical distance upon culture. Geographical distance apparently explains 81.18% of cultural diversity as measured in this study. This leaves a smaller, but possibly significant, proportion of cultural variation which might be explained by the genetic measure.

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1 Chakraborty, R. *Nature* 264, 350–352 (1976).

CHAKRABORTY REPLIES—Greene[1] contests my assertion regarding the relative importance of geographical versus cultural separation as a correlate of genetic variation in seven Chilean populations[2]. In so doing, she notes that in the stepwise regression procedure I used geographic distance as the first variable even when the cultural dissimilarity index had a higher product moment correlation with genetic distance (the dependent variable). Two reasons justify my choice of geography as the first independent variable to be entered in the regression analysis, one being theoretical, the other pragmatic.

Take, for instance, cultural variability as the dependent variable. Evaluation of the relative effects of geographical and genetic distances on cultural variability indicate that 82.41% of the cultural variability is explained by geographical distance ($P < 0.0005$), whereas the additional effect of genetic distance is 4.01%, which is statistically insignificant ($0.10 > P > 0.05$). This alone may suggest that geographical distance should be the first variable of interest.

The pragmatic view concerns the choice of the characteristics determining cultural diversity. As elaborated elsewhere[3], it is difficult to ascertain that cultural attributes are independent of geography. For example, we classified the seven populations on the basis of their types of subsistence, housing and architecture, economics, mode of gathering food, etc., which do have strong geographic components embedded in them. Thus, the strong association between cultural affinity and geographic proximity is indeed historical in these seven populations.

Furthermore, a stepwise regression method often under-represents the importance of the last variable to go in, particularly when the independent variables are significantly correlated with the dependent one. A logical explanation of causal relationship in such an event has to based on an understanding of the process producing the covariation between the variables, and not based on which independent variable has the highest correlation with the dependent one.

Greene's criticism, therefore, should be viewed with caution, although any generality of my analysis needs further examination, possibly from studies of other populations with comparable ethnographic accounts.

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Do viruses use calcium ions to shut off host cell functions?

IN discussing the shut-off of host cell functions by poliovirus, Carrasco and Smith[1] ask: "How does a viral protein change the ionic environment inside the cell?" Perhaps it forms channels across the external membrane[2]. For example, a viral structural protein might generate holes by assembling into hexamers, pentamers, or extended lattices in a membrane.

Although Carrasco and Smith emphasised Na^+ ions, divalent ions are probably more important controllers of metabolism. In general, Ca^{2+}, Mg^{2+} and polyamine ions tend to form much more specific links with polyelectrolytes than Na^+ or K^+ do[3], and the Debye–Hückel concept of ionic strength grossly underestimates the relative binding of, say, Ca^{2+} compared with K^+, to membranes and viruses[4]. Many enzymic activities depend upon divalent ions, while relatively few respond to physiological changes in monovalent ion concentrations.

Of the two main divalent ions in biology, it is Ca^{2+}, not Mg^{2+}, that usually acts as a signal. This is because Ca^{2+} is actively pumped to at least a thousandfold concentration difference across membranes[5], whereas Mg^{2+} is much nearer equilibrium[6], probably because it permeates more easily. The fact that cells respond more to changes in extracellular Mg^{2+} than Ca^{2+} has misled some workers into putting a false emphasis upon the ions' relative roles[7].

The early effects of many lytic viruses can readily be interpreted as a consequence of increased flow of Ca^{2+} into

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 gearshift 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* paper[1], and which is the subject of a more detailed paper[2], 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 ions[2].

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 O_3, Kolb and Elgin[1] 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 assumed[2]. 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 suggested[2] 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[3] 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 catalogue[4] 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 Millman[5] 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 ratio[6] of 27:1. Since the mean visual meteor rate[6] 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 Hughes[6] 3.1×10^{-4} h^{-1}. Though there are uncertainties in $[O_3]$ 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 Hughes[7].

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 Lindblad[8] for Perseid meteor trans 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 ($^1D_2 \leftarrow {}^1S_0$) 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 $O(^1S_0)$ state most effectively occurs with O and O_2. 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. Plavec[9] 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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