on small-scale impacts[6] indicate that some material may be spalled at speeds approaching 85 % of the impact velocity. It is clear that a 50–75-km-diameter transient crater (which would produce a final crater comparable in diameter to the observed large craters on earth) could eject roughly a million cubic meters of boulders of more than one meter diameter from the fecund zone. The diameter of the boulders is estimated by the fragmentation theory of Grady and Kipp (discussed in refs 1–3), which predicts the fragment diameter l_{GK}

$$l_{GK} = \frac{2Ta}{\rho v_{ej}^{2/3} U^{4/3}}$$

where T is the tensile strength of the rock (taken here to be 0.1 GPa). The fragment diameter estimated by this equation is shown in the figure (*b*).

It seems likely that the impacts that produced the craters on Earth that are greater than 100 km in diameter[7] would each have ejected millions of tons of near-surface rocks carrying viable microorganisms into interplanetary space, much in the form of boulders large enough to shield those organisms from ultraviolet radiation, low-energy solar cosmic rays, and even galactic cosmic rays. Under such circumstances spores might remain viable for long periods of time. Even microorganisms active at the time of ejection might have been preserved for considerable periods by lopholization in hard vacuum. Viable organisms preserved by this process were recovered from the Surveyor 3 camera assembly after three years on the lunar surface[8].

No computations have yet been performed to indicate how long Earth ejecta would take to reach Mars. Similar computations on the migration of Mars ejecta to Earth[9] indicate mean transit times of millions of years; the probability of a direct orbit is exceedingly low. Once at Mars, boulders falling to the surface may be slowed by the thin atmosphere and fragmented by aerodynamic stresses at low altitudes, exposing their interiors. This process is most important for objects of less than a meter diameter[10,11].

Although terrestrial organisms in these rocks would have the opportunity to colonize the planet, it seems unlikely they would find conditions suitable for propagation. Planets of the Solar System should therefore not be thought of as biologically isolated: from time to time large impacts may inoculate Mars and the other planets of the inner Solar System with a sample of terrestrial life.

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Leukocyte L1 protein and the cystic fibrosis antigen

SIR—Since its initial description as a major leukocyte protein[1], L1 has been characterized and its expression localized. Briefly, it is a calcium-binding, heat-stable multi-chain protein, with a relative molecular mass (M_r) of about 36.5 (ref. 2). Increased L1 plasma concentration is found in many infectious and malignant conditions[3,4], and L1 is present in leukocytes[5], macrophages[6] and epithelial cells[7].

Two-dimensional electrophoresis of purified L1 shows that it corresponds to the leukocyte polypeptides RA 6–7 which are increased in rheumatoid arthritis[8]. Purified L1 from human granulocytes separates into three polypeptide chains, on two-dimensional isoelectric focusing/SDS gel electrophoresis; the M_r and *p*I of polypeptides I, II and III are M_r 12.5, 13.3 8.3; *p*I 5.5, 6.0, 7.0 (H.B.B. and M.K.F., unpublished). Polypeptides I and II cannot be distinguished by rabbit or rat polyclonal or mouse monoclonal antibodies. The amino-acid compositions of polypeptides I and II are similar[9], and their small differences in *p*I and mass probably result from post-translational modifications and/or deamidation. Polypeptide chains III and I/II are called the L1 light and heavy chain, respectively.

Odink *et al.*[10] recently described two macrophage proteins, MRP-8 and MRP-14, the former of which is nearly identical to the cystic fibrosis (CF) antigen[11]. The sequences deduced from cDNA encoding the MRP-8 and MRP-14 polypeptides[10] are identical to the partial amino-acid sequences of L1 light and L1 heavy chains, respectively[9]. Post-translational modifications are, however, observed in L1 light-chain positions 1 and 6. Sequences of tryptic peptides derived from the C-terminal region of the L1 light chain all agree with the MRP-8 cDNA sequence from position 292 but no peptides corresponding to the deduced CF antigen C-terminal sequence[11] were detected. Like Odink *et al.*, we suggest there is a sequencing error from nucleotide 293 of the CF antigen.

Overall, there can be no doubt that the MRP-8/CF antigen and MRP-14 are identical to the L1 light and heavy chains, respectively. Note that, like the L1 antigen[7], the CF antigen is present in squamous mucosal epithelia[11]. Until an appropriate name can be coined on the basis of a putative biological function, we propose that the terms L1 antigen, L1 light chain and L1 heavy chain should be used.

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Return of the 'last ribo-organism'

SIR—In accusing us of a profound misunderstanding, Maizels and Weiner[1] continue to illustrate the problems encountered as molecular biologists attempt to model the 'RNA world'.

For example, they write that cofactors containing fragments of RNA would emerge in a world of protein catalysts because "nucleotides are far better catalysts than proteins for many reactions". This is wrong. The nucleotide fragments of RNA cofactors are incidental to their chemical reactivities[2]. In the RNA world model, their existence is a 'vestige' of early RNA metabolism[3]. Cofactors that emerged after the breakthrough to the 'protein world', such as biotin[4], are not ubiquitous, lack RNA fragments, and have reactivities reflecting the improved catalytic power of proteins[2,4].

Indeed, Maizels and Weiner's writing is generally confused about vestigiality. If an RNA unit performs a selectable function in the modern world that is intrinsic to the chemistry of RNA and could not be performed as well by proteins, the function is probably not a vestige of the RNA world but has arisen more recently. Therefore, the modern function and intrinsic chemistry of introns and tRNA-like structures