in cellular DNA (refs 1, 2) and amino acid substitutions in proteins[3,4]. But, the main conclusions of our paper did not depend on knowing the proper corrections to apply, if any, for the lengthened generation times in the higher apes and man as compared to the Old World monkeys. The conclusions were that (1), nucleic acid sequences related to the genome of the genetically transmitted baboon type C virus can be detected in the cellular DNA of all Old World monkeys and apes; (2), the baboon type C virus specific sequences can be used to discriminate between those Old World monkeys and apes whose habitat is Africa (baboon, mangabey, African green monkey, colobus, chimpanzee, gorilla) and those whose habitat is Asia (various macaques, langurs, gibbon, orangutan); (3), the data obtained with human DNA closely fit the pattern observed with the Asian apes. The suggestion was therefore made that most of man's evolution since his divergence from the other apes had occurred in Asia rather than in Africa.

Sarich and Cronin point out that the hypothesis that animals with longer generation times accumulate fewer mutations per unit of chronological time as suggested by Kohne[1], Lovejoy[5], and others is subject to a direct test. The accumulation of nucleotide substitutions along the gibbon lineage, where the generation length has changed less, compared to Old World monkeys, should be more than it is along the human lineage where the time of sexual maturity and the maximum lifetime potential[6] have markedly increased. Although we agree with him that this is experimentally testable, the differences would be small. If man had a common ancestor with the chimpanzee and gorilla as recently as 4 Myr ago as Sarich has proposed[7,8], then the effect of the recent prolonged generation time of ancestral man would be even more difficult to detect. We would not conclude that the cellular DNA data as reported in the manuscript was performed in a sufficiently rigorous fashion to 'prove' the lack of effect of a generation time. In our initial studies we did not see a difference along the two lineages, but more refined studies directed specifically at that question might reveal small differences; such a study is currently in progress.

Our data concerning the relationships among the cellular DNA of Old World monkeys and apes agree quite well with the extensive immunological studies of Sarich, Cronin, and others. For example, we state that 'the extent of nucleic acid sequence divergence among chimpanzee, gorilla, and man is three to four times less than that between any of these three Hominidae and the Old World monkeys'. But, there is also a considerable body of palaeontological evidence suggesting that apes and hominids had differentiated long before the time Sarich and associates place the chimp–gorilla–man common ancestor[6,9]. With methods for dating fossils improving, this is now a substantial body of data we felt should not be ignored. It will be intriguing to see if the traditional palaeontological approach can be correlated to the data being obtained with respect to protein and nucleic acid evolution, or whether the discrepancy in the two methodologies is as large as Sarich suggests.

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Complementation of immune response genes for (T,G)–A–L

Two years ago we reported the results of experiments which demonstrated the *in vivo* complementation of immune response genes required for a response to (T,G)–A–L (ref. 1). Subsequent experiments seemed to confirm and extend this result[2]. Unfortunately others have been unable to repeat our results in as much as they find no evidence for complementation in the response to (T,G)–A–L (ref. 3). Recently in attempts to repeat our original findings with (B10.M × B10.Br) F_1 we also have been unable to do so but now find responses to (T,G)–A–L similar to those reported by others[3]. We wish, therefore, to express our reservations concerning complementation in the (B10.M × B10.Br)F_1, in the hope that further study of this phenomenon by ourselves and others will clarify the conditions in which genetic complementation in the response to (T,G)–A–L can occur.

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High explosive analogue of the Tunguska Event

Jones[1], observes the existence of an important, but little known experiment. The numerical conclusions however are incorrect, using only the data cited in the letter. Using the ratio 35,000 to 130 as the diameters of symmetric explosion phenomena for the Tunguska event and the 1966 experiment cited by Jones, respectively, and the relevant scaling law, the corresponding equivalent explosive source strength for the Tunguska event would appear to be of the order of $50 \times (35{,}000/130)^3$, or about 10^9 t, not the 10^5 t cited. Without accurately computing all the necessary corrections for energy partitioning, actual hydrodynamics, etc., this would provide an estimate of source strength about two orders of magnitude higher than ref. 3 cited therein concludes.

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Jones replies: Augenstein is correct, and the calculated values should have read 10^5 kt TNT and 200 Mt nuclear equivalent, these being minimum estimates. The Tunguska event devastated an area 35 km in diameter! It is commented, however, that the very low height of burst, almost a surface burst at this yield, should be retained despite the absence of a crater at Tunguska. I have discussed elsewhere (Suffield Reports 217 and 281), experimental evidence that high yield but low energy density explosions on the surface do not cause craters but do produce airblast and surface seismic effects similar to those from high density explosions. Forest blowdown, for example, is insensitive to the initial energy density but depends upon the total energy release. The lack of a crater at Tunguska is thus evidence of the low density, presumably cometary nature of the bolide.

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β-Globin mRNA in Ferrara β⁰-thalassaemia

In their paper on the presence of β-mRNA in Ferrara type β^0-thalassaemia, Ottolenghi *et al.*[1] stated that our laboratory had obtained "similar findings" to their's with material from one of their patients. Because of possible misinterpretations of that very general statement, we present here the limited data which we did obtain using Ferrara material.

Professor Conconi kindly provided us with ribosomes from one of his patients so that we could attempt to