case of the Galaxy, however, the fields of 2–3 μG are not random, but run along the galactic arms.)

Both limits are substantially smaller than the limit, $q/e \geq 10^{-13}$, obtained by Bernstein and Ruderman[2] from arguments on solar energy losses. It is indeed remarkable that from these first ever observations of neutrinos from a supernova such stringent limits on their charge can be obtained.

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Growth transformation by *v-myc*

SIR—A recent report by Casalbore *et.al.*[1] showed that the avian retrovirus MC29, which contains only the *v-myc* oncogene, stimulates the growth of neuroretina (NR) cells from 7-day-old embryos, under conditions where uninfected cells can be cultivated for 10–15 population doublings. The authors conclude that the *v-myc* oncogene is sufficient to induce "growth transformation" of chick NR cells and that "their results differ considerably from the experiments of Bechade *et al.* (ref. 2)".

Indeed, we have reported that viruses containing only the *v-myc* oncogene fail to induce proliferation of normal NR cells from 7-day-old chick embryos which, in our experimental conditions, do not multiply *in vitro*, while oncogenes such as *v-src*[3] and *v-mil*[4] induce rapid and massive proliferation of these cells. In the experiments of Casalbore *et al.*, the *v-myc* gene appears to enhance the growth rate of NR cells that are already undergoing cell division. Therefore, we believe that their results support our conclusion that *v-myc* is not sufficient to induce NR cell multiplication, because it depends on specific culture conditions, that are not required by *v-src* or *v-mil*, in order to alter the growth properties of these cells.

Yet, even under these "optimized culture conditions in which efficient virus spread allows a rapid uniform viral infection" the authors showed that only 30% of multiplying NR cells release MC29, while over 80% of NR cells infected with MH_2, containing both the *v-mil* and *v-myc* oncogenes, were found to produce virus. To explain these differences, they suggest that MC29-infected NR cells are more sensitive to environmental effects that would delay expression of the *v-myc* protein. A simpler and more obvious explanation would be that the concomitant expression of *v-mil*, in MH_2-infected NR cells, allows synthesis of the *v-myc* gene product in the majority of NR cells.

Casalbore *et al.* conclude that their results "suggest a novel experimental approach to generating clonal strains from short-lived early neuronal precursors otherwise not easily amenable to adequate characterization". Indeed, we have already reported that it is possible to generate clonal strains that exhibit typical neuronal properties with quail NR cultures transformed by Rous sarcoma virus[4,5].

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The primate trade and the origin of AIDS viruses

SIR—There is now[1] compelling evidence for an African origin of the viruses causing AIDS. It seems that the human AIDS viruses are descended from a monkey virus, probably via the passage to man of the simian T-lymphotropic virus (STLV-3), now called SIV, isolated[2] from healthy African green monkey (AGM) *Cercopitecus aethiops*, which causes an AIDS-like disease in captive rhesus macaque monkeys[3]. Thus, an apparently harmless monkey virus has probably given rise to a human virus which has evolved into the HIVs, but it remains difficult to explain why human HIV seropositivity is recognized in Africa only after the 1950s.

Is it possible that these events are linked with the beginning, in the 1950s, of a massive trade of monkeys from Africa to western countries, mainly the United States, coinciding with the beginning of tissue-culture technology? In the 1950s, the introduction of tissue cultures in research into human enteroviruses, and in studies of the preparation and control of polio vaccines, caused a massive request for monkeys, and many primate stabularies were created where different species of monkeys often lived together. AGM has been one of the monkeys used most for kidney cultures for enterovirus studies. This caused an unprecedented human manipulation of AGM by Africans involved in the capture and maintenance of these monkeys, and stabulary and laboratory personnel of western countries. All this might have vastly increased the odds of an accidental passage of SIV from AGM to other monkeys and to humans. The lag in the appearance of widespread human infection might have been due to the adaptation of the virus to its new host and to the behavioural characteristics of people in the different countries.

Finally, the susceptibility of rhesus monkeys and other primates to SIV raises the concern of the safety conditions used for the selection and maintenance of monkeys entering primate stabularies in general, and of those monkeys used by industrial polio vaccine suppliers in particular; taking into account that blood-cell contaminants (adherent mononuclear cell clusters) are always present in primary *in vitro* monkey kidney cell cultures.

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Contingent contagious constraints

SIR—Hoyle and Wickramasinghe[1] present arguments in the cometary debate which are, "contingent on constraints that they themselves have imposed". Where they have argued that, "the available epidemiological evidence is consistent with the model where the causative agent (or a trigger for it) is airborne, "they have analysed epidemics in an eccentric manner of their own devising which is entirely innocent of the techniques of mathematical epidemiology[2] — an approach which would be strange enough were the authors non-numerate but which is truly extraordinary for scientists with such considerable mathematical powers[3].

A key feature of contagious processes is that they can exhibit great variability. This is especially likely to be the case when the values of parameters are near threshold, and processes in which variances are greater than means are not uncommon. In their analyses[2], however, Hoyle and Wickramasinghe repeatedly rely on the binomial distribution (for which the variance is less than the mean) and their claims to demolish theories of person to person transmission on this basis are no more impressive than would be those of a medical statistician claiming to have overturned classical physics because he had discovered that force was not proportional to velocity.

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Erratum

Reference 2 in J.H. Hecht's letter in *Nature* **328**, 765: 1987, should have been: Saslaw, W.C. & Gaustad, J.E. *Nature* **221**, 160–162 (1969).