Galactic halo gamma rays

SIR — Recent reports[1,2] on the isotropy of the 117 observed and located γ-ray bursts (GRBs) and the $V/V_{max} = 0.34 \pm 0.03$ value for them has far-reaching consequences. V/V_{max} is the average over the bursts of $(C_p/C_{lim})^{-3/2}$, where C_p is the peak count of a burst and C_{lim} is the limiting count that triggers detection[3]. For a spherical distribution of sources with a luminosity function that is spatially invariant, $V/V_{max} < 0.5$ when their density drops outwards. Thus, the observed value suggests that the distribution of sources is either local around the Solar System, or local around the Milky Way, or of cosmological dimensions.

I would like to consider the possibility of a distribution of about 50 kpc around our Galaxy which, for this number of bursts, is not excluded by the reported lack of clustering in the direction of the Large Magellanic Cloud, in view of its relatively small mass, or by the Sun's off-centre position. Because galactic escape speeds are of the order of 300 km s^{-1}, any objects coming from the Galaxy would occupy a far larger volume, unless their velocity is finely tuned or unless they are able to produce only GRBs during a limited, well-selected epoch of their lifetime. Otherwise, we must assume that the distribution is pregalactic. Unless very different from galactic ones in origin or in ability to produce GRBs, pregalactic neutron stars would be associated with too much supernova debris to be compatible with the chemical composition of the Galaxy, if their number greatly exceeds that of the former; if they are less numerous, it is not clear why no trace of the galactic distribution was observed.

With the reported detection threshold, maximum intrinsic luminosities in the case of a ~ 50 kpc distribution are several 10^{39} erg s^{-1}. We have suggested that accreting, rapidly rotating black holes are the sources for at least some of the GRBs[4–6]. An attractive feature of this model was that its Compton–Penrose spectra, characterized by the electron rest-energy, were similar to those observed. These spectra are generated by Compton scattering in the inner ergosphere of photons that are emitted by the infalling matter and are travelling inwards. On arrival at the inner ergosphere they become extremely blueshifted in the local inertial frame and come out of the collisions with the locally orbiting electrons having energies around the electron rest energy. The very fast black-hole rotation then counteracts gravity on these photons and, if the optical depth is right, they emerge to infinity with negligible redshift.

I now wish to point out that if the 'dark matter' halo of our Galaxy consists of ~ 1% (by total mass) ~ 10^{-13} $M_\odot$ asteroids and if the bulk of the mass is in ~ 10 – 30 $M_\odot$ black holes rotating very close to the extreme Kerr limit, then an asteroid will come to within a Roche lobe distance of a black hole about once a day, the observed GRB rate. In this encounter the asteroid would become bound, would break up and would accrete onto the black hole to produce the GRB when matter hits the inner ergosphere of the black hole. Asteroid–asteroid collisions would not significantly deplete that reservoir.

This particular composition of the halo could obtain if the fragmentation process in the pregalactic halo terminates with $\lesssim 30$ $M_\odot$ collapsing clouds, of which the central part eventually becomes a black hole but the exterior, which acquires metallicity from the nucleosynthesis, forms a disk to take up the excess angular momentum. The disk then fragments into the asteroids, some of which subsequently become unbound.

With the increased sensitivity of future X-ray detectors, such black holes, if they exist, could be seen as they pass through the galactic disk with galactic escape speeds and accrete interstellar clouds.

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AIDS response

SIR — Maddox[1] and Anderson[2], discussing the results of Stott *et al.*[3,4] appear to differ on the key issue of whether or not the xenoreactive monkey sera bound SIV-encoded proteins (although Stott *et al.*[4] reported no such reactivity was detected with purified envelope proteins). But both authors[1,2] implicate some protective cross-reactive immunity between SIV-encoded proteins and cellular (presumably histocompatibility) antigens.

This interpretation overlooks at least two readily testable alternative explanations of the phenomena described. First, HIV budding from a host cell might incorporate cell-surface molecules, providing both the initial xenoimmunizing and subsequent neutralizing epitopes. Second, immunization with cellular components from a $CD4^+$ line might induce anti-CD4 antibodies which could block subsequent viral access to $CD4^+$ cells. Watanabe *et al.*[5] have demonstrated just such an anti-self CD4 mechanism in rhesus monkeys protected from challenge with SIV following soluble CD4 treatment.

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SIR — Stott *et al.*[4] present evidence that antibodies against proteins of human lymphoblastoid cells in sera from macaques immunized with fixed, uninfected cells (or partially purified, inactivated whole SIV) might correlate with protection from infection after challenge with live cell-free SIV grown in the same human cells. This raises the possibility that vaccine protection in macaques immunized with SIV vaccines might be influenced by immune responses to cellular proteins, as the vaccine and challenge viruses used in previous studies were grown in human cells[4]. In this regard, reports describing vaccine protection against SIV infection in macaques have not included an analysis of antibodies against cellular proteins.

We have been evaluating antibody responses in rhesus macaques immunized with whole, inactivated SIV, and subsequently challenged with live cell-free SIV. The vaccine and challenge stocks of SIV were grown in the human lymphoblastoid cell line CEM×174. Virions used for vaccination were purified by centrifugation through a glycerol bed and inactivated with psoralen and ultraviolet light. After intravenous challenge with live cell-free SIV (50 MID_{50}), all 10 animals that received the SIV vaccine were protected from infection, whereas all 6 animals immunized with HBsAg (as a control vaccine) became infected[6]. While performing assays for neutralizing antibodies on plasmas from these animals, we noticed abnormal cell clumping and growth inhibition of CEM×174 cells at dilutions approaching the neutralizing titre. These effects were observed in plasmas from SIV-vaccinated animals but not in plasmas from HBsAg-vaccinated animals, prompting us to suspect that anti-cell antibodies were present in the SIV vaccinees.

To characterize further the anti-cell antibodies, we performed the following experiments. First, we showed that anti-cell antibodies were present in plasma from the SIV vaccinees by using a fixed-cell (H9 and CEM) immunofluorescence assay. In contrast, no anti-cell antibodies were found in plasmas from HBsAg vaccinees or, importantly, macaques that had been experimentally infected with