

## Lensing of supernova neutrinos?

SIR—The detection of neutrinos<sup>1,2</sup> from supernova SN1987A has led to an explosion of speculative analyses<sup>3-7</sup>. These place a heavy burden of interpretation upon time delays between the individual neutrino events detected on Earth. We would like to point out that the bimodal distribution of the Kamiokande detections could be explained if the neutrino burst from the supernova were split by an intervening gravitational lens. This wildly speculative hypothesis has nevertheless the merit of being testable in the future.

The twelve Kamiokande II neutrino events have arrival times<sup>1</sup> of 0.00, 0.11, 0.30, 0.32, 0.51, 0.69, 1.54, 1.73, 1.92, 9.22, 10.43, 12.43 s and fall into two bunches with nine events around 1 s and three near 10 s. We do not know the intrinsic spread of the neutrino events, but if we assume it to be less than a few seconds then the intrinsic spread of the first bunch is less than the time between the two bunches by a factor of two. Suppose the supernova emitted only a single burst of neutrinos with an intrinsic spread of a few seconds which subsequently encountered the gravitational field of a compact intervening mass,  $M$ , en route to Earth. This mass will act as a gravitational lens to produce two (in the case of a point lens) or more (for a distributed lens not dominated by a compact core) neutrino paths. Arrival time differences for the two neutrino signals will, for the point mass lens, be

$$\Delta t \sim \frac{4GM}{c^3} \sim 10 \left( \frac{M}{5 \times 10^5 M_\odot} \right) \text{s}$$

We assume that the ratio of the apparent intensities along the two neutrino paths is 1:3 as indicated by the relative abundance of neutrino events in the two detected bunches. Hence, we see that the observed delay of  $\sim 10$  s between the two bunches of neutrinos could be accounted for by an intervening object with  $M \sim 5 \times 10^5 M_\odot$ . Assuming the intensity ratio 1:3, with the lensing object at 25 kpc and the supernova at 50 kpc, the image separation angle produced by a point lens is

$$\Delta\theta \sim 0.6'' \left( \frac{M}{5 \times 10^5 M_\odot} \right)^{1/2}$$

Such image-splitting is probably too fine to be resolved at present but future radio, optical, and certainly Space Telescope observations, could check the existence of lensing by searching for the second image of the supernova.

The lens candidate must have a mass  $\sim 5 \times 10^5 M_\odot$  within a radius  $\sim 0.07$  pc if the lens lies at a distance of  $\sim 25$  kpc — midway between us and the supernova where the probability of lensing is greatest, although still very small,  $\sim 10^{-6}$ .

This means that a black hole with a Schwarzschild radius of  $5 \times 10^{-6}$  pc, a compact star cluster, or a compact cluster of 'Jupiters' would be ideal candidates. Both have been proposed for other reasons<sup>9,10</sup>. If one were to assume that a uniform population of such lens candidates constituted the dark halo of our own Galaxy then the chance<sup>11</sup> that one would be found interposed between us and a supernova in the Large Magellanic Cloud is about  $10^{-6}$ .

A more speculative lens candidate is a loop or line of cosmic vacuum string. There has been a recent claim by Cowie and Hu that such an object has been observed producing a unique signature of four pairs of double galaxy images as expected if a string loop passed through four galaxies. A curved cosmic string having mass per unit length  $\mu$  produces image splitting with angle<sup>12</sup>

$$\Delta\theta \leq \frac{8\pi G\mu}{c^2}$$

A string parameter of  $G\mu/c^2 \sim 10^{-7}$  for a string located roughly midway between us and the supernova and approximately perpendicular to the line of sight would also produce the observed delay of  $\sim 10$  s between arrival of the two neutrino signals. The string has to curve, and presumably therefore be part of a loop, for the apparent intensities of the two neutrino signals to be unequal. A string of this sort will produce an image separation of  $\sim 0.25''$ .

Finally, we should also consider the consequences of including the smaller sample of neutrino data from the IMB detector<sup>2</sup>. IMB saw eight neutrino events at times 0.00, 0.42, 0.65, 1.15, 1.57, 2.69, 5.01 and 5.59 s but with an unknown time origin relative to the Kamiokande events

and a higher detection energy threshold. These two uncertainties make it difficult to compare the two data sets with the information publicly available. If we assume that the time origins are the same then we can draw either of two conclusions concerning the lensing hypothesis. One possibility is that the IMB data provide support for the view that the neutrino arrival times are randomly spread between 0 and 12 s and have no significant sub-structure. Despite the reasonableness of this view it clearly has not been adopted by authors deducing neutrino masses<sup>3-6</sup>. The other possibility is to take seriously the bunching of two IMB events near 5 s displayed by the two sets of data in conjunction with each other. In this case the lens hypothesis could imply the existence of three images as predicted for any smooth bounded lens with the intensities of the images in the ratio 1:0.3:0.3.

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## Intracellular pH and cytoplasmic free Ca<sup>2+</sup>

SIR—The title of a recent letter to *Nature*<sup>1</sup> contains a specific conclusion, that "activation of sodium-proton exchange is a prerequisite for Ca<sup>2+</sup> mobilization in human platelets." Siffert and Akkerman conclude that "an increase in intracellular pH forms an essential step in the cascade of events required to increase cytoplasmic free Ca<sup>2+</sup> ..." I think that published data from my own laboratory<sup>2</sup>, and from other groups<sup>3,5</sup>, argue strongly against this view.

Siffert and Akkerman report that replacement of Na by choline suppresses thrombin-evoked internal discharge of Ca<sup>2+</sup> and slows and reduces Ca<sup>2+</sup> influx. Sage and I, however, showed<sup>2</sup> that replacement of Na by choline had no significant effect on the extent of the rise in intracellular Ca<sup>2+</sup> concentration, [Ca<sup>2+</sup>]<sub>i</sub>, evoked by thrombin or platelet-activating factor in the presence or absence of external Ca<sup>2+</sup>. Also, complete replacement of Na by K had no effect on internal discharge of Ca<sup>2+</sup>. Our data appear to refute

unequivocally the main conclusions of Siffert and Akkerman. I cannot readily explain the discrepancy in the experimental findings; their results might be explained by the use of forskolin and gel-filtration in preparing their platelets, or by the presence of impurities in their sodium substitute. In fact, one result in Siffert and Akkerman's own work seems to show that Na<sup>+</sup>/H<sup>+</sup> exchange was not an "essential step". Their Fig. 3 shows that at higher concentrations of thrombin there was no significant effect of Na removal on [Ca<sup>2+</sup>]<sub>i</sub> rise or on aggregation.

The rightward shift in the thrombin dose-response curves produced by sodium removal, as shown in their Fig. 3, is similar to that reported by Connolly and Limbird<sup>3</sup> and attributed to a requirement for Na in the generation of thromboxane A<sub>2</sub>. Siffert and Akkerman did not specify the state of cyclooxygenase in their platelets, but presumably they used drug-free donors and their results and their interpretation could be analogous to those of Connolly and Limbird. The finding that ethylisopropyl-amiloride can have similar