Binary precursor for planet?

SIR — Bailes *et al.*[1] have recently discovered a pulsar, PSR 1829–10, which shows time-delay variations, indicating that it is orbited by an object of planet mass. It is surprising both that a planet has apparently survived the supernova explosion that formed the neutron star and that the planet is now in a circular six-month orbit. We have devised a plausible explanation, in which the neutron star is relatively old and was in a wide orbit around a massive star. Be X-ray binaries such as this are relatively common[2]. When the companion star swelled up to become a giant, it transferred mass to the neutron star which thereby acquired a massive disk. We assume that the disk both spun up the neutron star and, after the supernova explosion of the companion, that the outer parts formed planets, at least one of which is massive enough to cause the observed time-delay variations.

To present our argument in more detail: we require that the progenitor system was an X-ray binary, for example a Be X-ray binary, with a relatively long orbital period of at least 5 yr. This means that the neutron star is not captured by frictional drag in the envelope of its companion when it becomes a red giant (it escapes 'spiral-in'). The neutron star may have been spun down to a spin period of minutes due to the propeller effect[3] in the matter expelled by the companion in its wind and equatorial disk. When the massive companion does swell to become a giant, the wind increases in strength with $\dot{M}_{wind} \sim 10^{-5}\ M_\odot\ \mathrm{yr}^{-1}$ and also slows down. If the wind velocity is less than the orbital velocity of the neutron star, it will accrete a fraction of $\dot{M}_{wind}$ equal to the square of the mass ratio of the stars, which is about 1 per cent in the case considered (where the mass of the giant is about 10 times that of the neutron star). This implied accretion rate is super-eddington. The material cannot all be accepted by the neutron star, so a large disk of mass $> 10^{-3} M_\odot$ accumulates around it provided that this phase lasts longer than 10^4 yr. The disk extends out to the roche lobe, which means that its outer radius is several AU.

When the companion explodes in a supernova, the binary system becomes unbound. Most of the disk will survive if it subtends a small angle at the companion or is shielded from objecta in the orbital plane. It then resembles the inner parts of the early proto-solar system. The central part will accrete onto the neutron star, thereby spinning it up until it reaches an equilibrium with the keplerian rotation of the disk at the magnetospheric radius. Our chief assumption now is that the outer parts of the disk cool and form planets. This should occur after the luminous companion has exploded if the inner parts shield the outer parts from the X-rays. By analogy with our own Solar System, we suppose that it is reasonable for the planet masses to be similar to or greater than that of the Earth. The planets will, of course, have orbits of low eccentricity.

The observed large spin period derivative of the pulsar indicates that it lies above the classical spin-up line for neutron stars[4], which could argue against our model. We note that this line, which is theoretically derived from (uncertain) models of the disk–magnetosphere interaction at the eddington limit, does not necessarily apply to a high magnetic field neutron star such as is considered here. (For instance, the neutron star may accrete at a super-eddington rate like the 10^{39} erg s^{-1} X-ray pulsar, AO538–66, in the Large Magellanic Cloud[5] since it has a strong magnetic field which can channel the accretion flow and beam the outgoing radiation.) We also note that a similar scheme to ours may be relevant to the X-ray pulsar 1E2259+586 (ref. 6 and references therein) which is in a supernova remnant and appears to be powered by accretion yet has no measured binary motion. It has a period of nearly 7 s, which is much larger than the 0.3-s period of PSR 1829–10. In our scheme it may just have had a much shorter spin-up phase than PSR 1829–10 and is now a single object surrounded by, and accreting from, a massive disk.

The advantage of our scheme is that it involves only a simple extrapolation from known systems and it does not require a sequence of improbable events. Planetary systems around stars do not have to be common. We predict that there should be another neutron star (from the ex-Be companion) near PSR 1829–10 and possibly an old supernova remnant (this depends on when the companion exploded). We also expect that PSR 1829–10 has further planets also in circular orbits.

A. C. FABIAN
Ph. PODSIADLOWSKI
Institute of Astronomy,
Madingley Road,
Cambridge CB3 0HA, UK

1. Bailes, M., Lyne, A. G. & Shemar, S. L. *Nature* **352,** 311–313 (1991).
2. van den Heuvel, E. P. J. & Rappaport, S. A. in *Physics of Be Stars* (eds A. Slettebak & T. P. Snow) 291–307 (Cambridge Univ. Press, 1987).
3. Illarionov, A. F. & Sunyaev, R. A. *Astr. Astrophys.* **39,** 185–195 (1975).
4. Battacharya, D. & van den Heuvel, E. P. J. *Phys. Rep.* **203,** 1–124 (1991).
5. Skinner, G. K. *et al. Nature* **297,** 568–570 (1982).
6. Davies, S. R., Wood, K. S. & Coe, M. J. *Mon. Not. R. astr. Soc.* **245,** 268–274 (1990).

■ See also Letters on pages 827 and 829.

CJD discrepancy

SIR — Palmer *et al.*[1] report that homozygosity, Met/Met or Val/Val, at codon 129 of the prion protein (PrP) confers genetic predisposition to sporadic Creutzfeldt–Jakob disease (CJD) in the United Kingdom. But this is not true in Japanese patients with sporadic CJD, where either homozygous or heterozygous Val 129 is related to the phenotype of disease.

We tested 21 unrelated Japanese patients with sporadic CJD, who lack mutations at PrP codons 102, 117, 178, 198 and 200 or insertion mutations seen in inherited spongiform encephalopathies. One is homozygous for Val 129, four are heterozygous, and the remaining sixteen are homozygous for Met 129. Because population data from 179 unrelated Japanese people show a much higher prevalence of codon 129 homozygosity (164 Met/Met 129, 15 Met/Val and no Val/Val), we conclude that Japanese patients with sporadic CJD do not carry an excess of homozygous Val 129 or Met 129 (χ^2=1.40, P>0.1). The Japanese population is in Hardy–Weinberg equilibrium for the alleles (χ^2=0.34, P>0.1), but the allele frequencies (Met:Val 129, 0.958:0.042) vastly differ from those found in the United Kingdom (0.625:0.375).

It is interesting that clinical and pathological features depend on Val 129. Japanese patients without Val 129 had a shorter clinical course (mean 17.0 months, s.d. 8) with visual impairment, personality change or hallucination at the onset of the disease. Myoclonus and periodic synchronous discharges in the EEG were always observed, but pathology showed no amyloid plaques in the brain using anti-human PrP antiserum. In contrast, all five patients with Val 129 had a longer course of disease (55.6 months, s.d. 30), starting with ataxia, amnesia or parkinsonian symptoms. Not all of them showed myoclonus or periodic synchronous discharges, but all had marked amyloid plaques in the brain which stained with anti-human PrP antiserum. These clinical and pathological features are rather similar to those of Gerstmann–Sträussler–Scheinker disease (GSS). We also tested two unrelated families (N.N. and K.M.) with GSS. One individual from N.N. and two sisters from K.M. had similar clinical and pathological features and were heterozygous for Val 129. Sequence analysis in all CJD and GSS patients with Val 129 revealed no other mutations in PrP. These results suggest that among Japanese CJD patients, individuals who are either homozygous or heterozygous for Val 129 will express the GSS phenotype and should be categorized as GSS, just like patients with sporadic