and Chau favour a black body spectrum, and in fact the data on the recently discovered X-ray pulsar Cir XR-1 seem to be a fractionally better fit to the black body spectrum than to thermal bremsstrahlung. Henriksen, Feldman and Chau's work may also be relevant to the X-ray source GX 340+0 which has recently been determined to have in all probability a black body spectrum, although there is no evidence so far that GX 340+0 is pulsating. This is the only object so far for which the resolution is adequate to distinguish between the black body and thermal bremsstrahlung models.

The model by Henriksen, Feldman and Chau is based on the generation of heat in the crust of what they call a wobbling neutron star, and the periodicity of about 5 seconds that is observed in the X-ray data is attributed to the free nutation period. It seems too much to hope, however, that all the pulsating X-ray sources will fit neatly into this scheme. Although knowledge of the characteristics of pulsed X-ray sources is still in a confused state, two classes of sources are emerging. On the one hand are the sources Cen XR-3 and NP 0532 (the pulsar in the Crab Nebula which is detectable at frequencies from the gamma ray to the radio band), which are characterized by well-defined periodicities, implying that some phenomenon involving the whole star —such as rotation—is involved in the timing mechanism. On the other hand, however, are sources such as Cyg X-1 and Sco X-1 which seem to emit short trains of pulses whose periodicities can be different from one pulse train to the next. Some periodic phenomenon involving only the atmospheres of the objects seems the likeliest explanation that will allow this kind of irregularity.

Nor is this to say that further sources in the first class will be susceptible to the treatment of Henriksen, Feldman and Chau. For the present, other more established views based on the population of radio pulsars seem to give a good account of the X-ray emission from NP 0532, and such models would also presumably work for PSR 0833-45 if and when X-ray emission is detected from this other fast pulsar. For the time being, then, the deep pulses from Cen XR-3 containing 70 per cent of the radiated power (2–6 keV) seem to be in a class of their own.

GX3+1

Two Candidates

ALL but two possible candidates for the optical counterpart of the X-ray source GX3+1 have been eliminated by two successful lunar occultation experiments, which have reduced the uncertainty in its known position by a large factor. The Skylark rocket flights which have produced this improvement were carried out from Woomera for the University of Leicester group (on September 27 this year) and for the University College, London, group (on October 24). It was necessary to take advantage of the rare occultation of the source by the Moon (see *Nature Physical Science*, **233**, 106 ; and **234**, 2 ; 1971) because in the best error circle available previously there were a great many stars, but none of them had a sufficiently striking spectrum to make it a likely candidate as an optical counterpart of the X-ray source.

Both groups have now completed the reduction of their data, and their results have been combined to give an improved fix on GX3+1. This fix is so good that the exact positions of faint stars in the area have had to be redetermined for comparison with the X-ray data. Two possible candidates have been found, lying one either side of the error box and close to RA 17h 44m 51.5s dec −26°33′50″. Either GX3+1 is associated with one of these stars (numbers 14 and 15 in the notation of Kunkel *et al.*, *Astrophys. J. Lett.*, **161**, L169 ; 1970), or it is fainter than 18th magnitude and lies between them, or it is fainter than 21st magnitude and lies somewhere else in the error box.

Interaction between HL-A Antigen Determinants

HL–A ANTIGENS, like other genetically based polymorphisms in man, have acquired a mystique which has tended to isolate discussion to the habitués. The meaning of "HL" is itself a matter of some doubt. By usage it has come to be accepted as an abbreviation for "human leucocyte", but its propositors (see Amos, *Science*, **159**, 659; 1968) meant it to stand for "human histocompatibility locus".

It seems that HL–A antigens are controlled chiefly, but perhaps not exclusively, by two closely linked genetic loci often known as the first and the second. At each locus a number of alleles are recognized the activity of which results in the synthesis of various antigens designated (for the most part) HL–A1, HL–A2 and so on. Unfortunately, consecutive numbers can relate to antigens associated with different loci; for example, HL–A8 is determined by locus 2; HL–A9 is determined by locus 1. This muddle has arisen because the antigens and their controlling genes were not discovered in a nice neat sequence. The antigens are usually recognized by the use of specific cytotoxic or complement-fixing antisera used on leucocyte or platelet populations *in vitro*. A report by Legrand and Dausset in next Wednesday's *Nature New Biology* (December 29) reveals an interesting facet of these tests.

The leucocytes from a single human individual can carry four different antigens determined by the first and second loci, that is, when heterozygous at both loci. Legrand and Dausset took the cells from such an individual (HL–A3, 5/10,12; 3 and 10 determined by genes at the first locus, 5 and 12 by genes at the second locus), and incubated them with a selected antibody (say anti-HL–A3) in the absence of complement. When the cells showed no further capacity for the absorption of anti-HL–A3 antibody they were incubated with one of the other three relevant antisera and the specific cytotoxic effect of the resulting supernatants was quantitated. By reference to the gene loci it can be said that sequential incubation with anti-HL–A3 and with then anti-HL–A10 is an allelic combination, anti-HL–A3 followed by anti-HL–A5 is a *cis* combination, and anti-HL–A3 followed by anti-HL–A12 is a *trans* combination. There are four of each kind of combination of different antisera. Legrand and Dausset found that when the antigens were attacked in the *cis* combination incubation with the first antiserum inhibited the absorption of the second antiserum. Further studies on genotyped patients established that this was a general rule.

This finding that there can be interference between combining sites on the cell surface which are determined by "adjacent" genes on a chromosome is in agreement with previous studies with mouse (H–2) histocompatibility antigens. The inference to the geneticist, as indicated by Legrand and Dausset, may be that two HL–A genes in the *cis* position cooperate in the building of a single molecule. To the transplanter it may well be that if an antibody to a particular antigen can be elicited which can block combination of antibody with another antigen (in the *cis* position), certain typing mismatches may be of no consequence. It would also be of interest to determine whether the inhibiting antibody prevented attack by cytotoxic cells.

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