found," says Schmidt. In other words, it pays to be degenerate, but even among magnetic white dwarfs, where conditions are extreme, fields tend to run no higher than tens of millions of gauss. Only one other star has a known magnetic field in this extreme range — another degenerate white dwarf, catalogued as Grw +70° 8247, which has about 300 MG.

An account of the determination of PG 1031+234's magnetic field will appear in the Oct. 1, 1986 ASTROPHYSICAL JOURNAL. Joining Schmidt in the work were Steven C. West and James Liebert of the Steward Observatory, Richard F. Green of Kitt Peak National Observatory in Tucson and H.S. Stockman of the Space Telescope Science Institute in Baltimore.

PG 1031+234 is unique in another important respect: It rotates, and does so very fast for a white dwarf or any other kind of star, going around once in three hours and 24 minutes. Rotation allows astronomers to study the structure of the star's field from many different perspectives. Grw +70° 8247 does not rotate.

The structure of PG 1031+234's magnetic field is also somewhat complicated. There is a basic dipole structure similar to the earth's magnetic field, although the white dwarf's dipole is more widely angled to its rotation axis than is the case on earth. Added to the dipole is a large magnetic spot, a place where magnetic field lines emerge but continue straight out into space rather than bending around to another pole as a dipole field does. The spot is similar to the magnetic spots seen on the sun but is a great many times as strong.

Both the rotation and magnetic field appear to be relics of PG 1031+234's youth, eons before it became a white dwarf. White dwarf status comes at the end of a star's life. It results from a tremendous collapse under the influence of the star's own gravitational self-attraction. A star the size of the sun can collapse to the size of the earth.

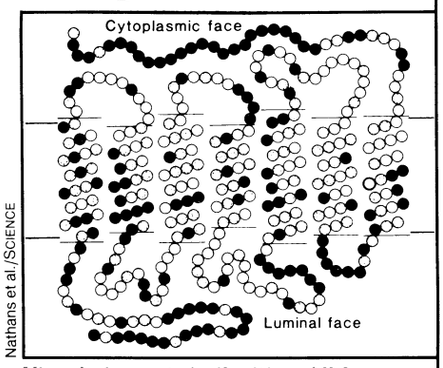
Such a collapse causes degeneracy, which in physicists' language has nothing to do with morals or ethics. It merely means that a large number of things are jammed together into only a few available energy states. To put it another way, the familiar structures of atoms with their well-defined shells of orbiting electrons are crushed out of existence. What remains is a collection of closely packed nuclei swimming in a "sea" of detached electrons.

Rotations and magnetic fields that already exist can be conserved through such a collapse. The collapse will greatly increase the strength of the magnetic field and the speed of the rotation. Thus the extreme values present in PG 1031+234 could have arisen from relatively ordinary values in a relatively ordinary progenitor star.

— D.E. Thomsen

The genes behind vision's palette

The human brain visualizes the world as a mixture of three primary colors, sensed by pigmented cells in the eye. This view of color vision evolved over centuries of investigation, but has now for the first time been directly demonstrated. Genes that correspond to the red, green and blue color-vision pigments have been identified by Jeremy Nathans, Darcy Thomas and David S. Hogness of Stanford University. Unexpected aspects of their findings give clues to how color vision evolved and may still be evolving.



Nathans et al./SCIENCE

Visual pigment similarities: All four human pigments and bovine rhodopsin have the same amino acid in the locations indicated by empty circles; similar amino acids at the stippled circles; and at least one "nonconservative" amino acid difference at the filled circles.

Tests on color-blind subjects provided critical information in the identification of the pigment genes. Color blindness is caused by the absence of a normal copy of one of these genes, the scientists have demonstrated in collaboration with Thomas P. Piantanida of S.R.I. International in Menlo Park, Calif., and researchers at Roswell Park Memorial Institute in Buffalo, N.Y. Furthermore, they traced a common condition of slightly altered color vision to the presence of an abnormal pigment gene. The brains of people with this condition portray colors as if they were using a slightly different set of paints.

"Through the application of modern recombinant DNA techniques and the analysis of genetic variants, a problem as old as the human effort to understand the real world has been brought to a higher, and most satisfactory, level of understanding," says David Botstein of Massachusetts Institute of Technology in the April 11 SCIENCE in an essay accompanying the color-vision research reports.

The key to the research success was the prediction that all the eye's pigment genes would have similarities due to a common evolutionary origin. Because one single-stranded DNA will bind to another resembling its complementary strand, an isolated gene can be used to search for related DNA sequences.

Nathans and his colleagues first used a gene that had already been identified as that of the bovine visual pigment called rhodopsin. With it they located the gene for the corresponding human pigment, which is used for vision in dim light but not for color vision. Then, with this human rhodopsin gene, they were able to identify three similar DNA sequences. They found the green- and the red-pigment genes on the X chromosome and the blue-pigment gene on the chromosome known as number 7.

Analyses of the genes indicate that a common ancestral DNA segment produced three genes: one that evolved to become the rhodopsin gene, a second that became the blue-pigment gene, and a third that duplicated in more recent evolution to become the green- and red-pigment genes.

The most surprising finding is that the X chromosome of people with normal color vision often contains two or even three copies of the green-pigment gene. The frequent presence of duplicate green-pigment genes "gives evolution some material to experiment with," says Piantanida.

The variation in green-pigment gene number seems to arise from unequal exchanges of DNA between paired chromosomes. These swaps also produce the chromosomes lacking a color-vision gene, in this way creating color blindness. Sometimes the exchanges appear to occur within genes. The result is genes that are hybrids of the red- and green-pigment genes. These hybrids underlie what has been a puzzling defect in color vision.

Among U.S. Caucasian men, 8 percent have defects in their red-green color vision. The most common defect is more subtle than an inability to distinguish red from green. It is observed when the men are asked to mix red and green light to match a certain shade of yellow. Those with "anomalous trichromatism" produce a different shade than does someone with normal color vision. Nathans and his colleagues have demonstrated that these men have, instead of one normal pigment, a hybrid pigment with different light-absorption characteristics.

Now that the visual pigment genes have been identified, scientists expect to be able to obtain for the first time adequate amounts of the pigments for biochemical study.

The intriguing question remains: During fetal development, how does each visual cell determine which pigment it must produce? *— J.A. Miller*