

Cracked plate caused telescope collapse

The sudden collapse last November of the 300-foot-diameter radiotelescope at Green Bank, W. Va., probably resulted from the fracture of a single, highly stressed steel plate, according to an investigation by an independent panel of engineers reporting to the National Science Foundation (NSF). The panel found that parts of the telescope, which was constructed in 1962 of a fine aluminum mesh held by steel supports, experienced far greater stress than would be allowed in a structure built today.

"From the beginning of its life, the structure was marginal with respect to structural failures of a minor or perhaps major nature," the panel reports. Designed to last only 10 years, the radiotelescope simply wore out.

The plate in question was a critical component in the instrument's support structure and endured great stresses whenever the telescope was moving. The plate itself was hidden from view and could not be examined routinely without dismantling the telescope. A metallurgical analysis of the remains of the plate revealed small cracks, which apparently grew until the plate suddenly failed.

Attention is now likely to focus on plans to replace the instrument. NSF's top choice for its next astronomy facility is an observatory geared to searching for gravitational waves. Known as the Laser Interferometer Gravitational Wave Observatory, the observatory would consist of two facilities located near the east and west coasts and cost about \$100 million. However, West Virginia politicians insist that NSF should give higher priority to replacing the collapsed radiotelescope with a modern instrument, at a cost of \$75 million (SN: 11/26/88, p.342).

The new age of the sun

The sun's age, as measured from the time it entered the main sequence of stellar evolution, can't be determined directly. It has to be inferred from the ages of the oldest meteorites — a difficult task because astronomers must establish a connection between the time of the sun's birth and the formation of meteorites. Recent observations of young stars in star-forming regions are starting to supply that connection.

These young stars, not yet on the main sequence of stellar evolution when they achieve core temperatures high enough to fuse hydrogen into helium, are surrounded by disks of dust and gas, out of which meteorites form. On the basis of this evidence, David B. Guenther of Yale University in New Haven, Conn., now calculates the sun's age at 4.49 billion years, somewhat less than the 4.7 billion years commonly used in the standard solar model. Guenther reports his results in the April 15 *ASTROPHYSICAL JOURNAL*.

"The well-determined ages of the meteorites, the theories describing the origin of the solar system, improved to fit recent infrared and radio observations of star-forming regions, and the pre-main-sequence evolutionary calculations have all combined to permit, for the first time, a relatively precise determination of the age of the sun," Guenther says. "Because the error in the age is small and because the solar model is relatively insensitive to small changes in the age, we now have grounds for using the sun's age as a fixed parameter of the standard solar model."

Guenther's calculations show that the 200-million-year difference in age between the old and new estimates has little effect on present theoretical models of how the sun has evolved. Characteristics such as the helium abundance and amount of internal mixing remain relatively unchanged. At the same time, the sun's new age is more consistent with the age of the oldest known meteorites, which are 4.53 billion years old. Such meteorites would have formed before the sun entered the main sequence of stellar evolution.

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Scientists home in on tooth enamel gene

Applying a new molecular "probe" to human chromosomes, researchers have found the approximate location of the gene that tells cells to make the major protein in tooth enamel. The protein, called amelogenin, is one of two proteins that provide the biological scaffolding around which mineralization occurs during tooth development. Many scientists believe it plays an active, regulatory role in tooth development as well.

The researchers say a determination of the gene's exact location should boost their understanding of a rare, hereditary weakness of tooth enamel called amelogenesis imperfecta. And because tooth enamel mineralization resembles the process of bone formation, isolation of the amelogenin gene may aid in detecting genes involved in inherited bone defects.

Enamel, the outermost coating of teeth, forms the hardest tissue in the vertebrate body. Its production begins with a matrix of amelogenin, produced by cells called ameloblasts, along with the less abundant protein enamelin. During the process of enamel maturation, the proteins are gradually replaced by crystals of a mineral compound called hydroxyapatite.

Mature teeth are composed of about 99 percent mineral crystals and less than 1 percent protein. But while present, amelogenin plays key roles in tooth development, perhaps in part by helping to exclude water from tooth tissue. Water content affects the size and arrangement of hydroxyapatite crystals.

Eduardo C. Lau of the University of Southern California in Los Angeles and his colleagues used a genetic probe made from mouse amelogenin DNA to look for a similar sequence on human chromosomes. The experiments allowed Lau and his co-workers to narrow the human amelogenin gene's location to relatively small genetic "neighborhoods" on both the X and Y chromosomes in humans — neighborhoods where other genes affecting tooth morphology are known to reside.

Once the researchers find the gene's exact location, they hope to pursue one or more molecular biological approaches to understanding the exact role amelogenin plays in biomineralization and the types of gene defects that can disrupt tooth development. Experiments could involve genetically engineered mice that either under- or overproduce the human protein, or that produce various defective versions of the protein, Lau told *SCIENCE NEWS*.

Meanwhile, the researchers continue to study people with amelogenesis imperfecta in families known to carry the tooth defect. The disorder, which leaves teeth with little or no enamel coating, affects about one in 14,000 individuals in the United States.

A precise identification of the amelogenin gene could lead to a genetic test capable of screening for the disease in members of high-risk families, says Lau, who reports the new findings in *GENOMICS* (Vol.4, No.2).

Post-chemotherapy PCR aids in prognosis

Since scientists developed the gene-amplifying procedure called polymerase chain reaction (PCR) just three years ago, they have applied it to a host of experiments testing for the presence of tiny amounts of DNA (SN: 4/23/88, p.262). Now researchers in London, England, have found PCR useful for detecting remnants of cells containing cancer-causing gene rearrangements in lymphoma patients following chemotherapy.

PCR performed on blood or tissue samples should help physicians tell whether a cancer has spread and may prove useful for monitoring treatment effectiveness, say St. Mary's Hospital Medical School researcher D. Cunningham and his colleagues in the April 1 *LANCET*.

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