# Heavyweight star sparks fight

How massive can a star be? Or, rather, how massive can a star be and still be stable? The proposal that there is a star in the Tarantula nebula that is 3,000 times as massive as the sun has inspired some theoretical astronomers to strenuous protests. One hundred to 150 times as massive as the sun was as heavy as many of them used to think a star could get.

The star in question is cataloged as R136a, and it seems to be powering the ionization of a large part of the Tarantula nebula. The Tarantula nebula, which is in the Large Magellanic Cloud, is the most massive region of ionized hydrogen visible in the local group of galaxies. Studies of the energy balance of the nebula indicated that most of the power for the ionization had to come from a very small central region. And about a year ago, after studies of its ultraviolet output, a group of researchers proposed that that tiny core is inhabited by a single star, 3,000 times as massive as the sun (SN: 1/17/81, p. 36). Now, at last week's meeting of the American Astronomical Society in Boulder, Colo., Dennis Ebbetts of the University of Wisconsin and Peter Conti of the University of Colorado report that a spectrum of the visible light from R136a is very similar to that of an early type Wolf-Rayet star. This is the kind of youthful, active star that might be expected in such a position.

The spectrum covers the range from 3,700 angstroms to 6,700 angstroms and was taken by Conti at the Cerro Tololo Interamerican Observatory. The spectrum shows broad but faint emission lines characteristic of the WN class of Wolf-Rayet star and absorption lines of the Balmer series of hydrogen and of neutral and ionized helium.

The most plausible supposition, according to Ebbetts and Conti, is therefore that the center of the Tarantula contains a single star more or less of this type. It would be extremely hot — 65,000 kelvins — and would have a radius 80 times the sun's; in the solar system it would occupy all the space inside the orbit of Venus. It would live about a million years, a very quick life for a star, and it would be 3,000 times as massive as the sun. The major difficulty is the presence of the helium absorption lines. They cannot come from within the star. The neutral helium ones could come from a shell around the star. The ionized helium ones have to come from something else, maybe some cloud in front of the star.

At this point came theoretical objection. Nolan Walborn of the Goddard Space Flight Center protested vigorously that such a star would be so unstable that it would never form. It would just not be able to get its mass together. He argued for an alternative interpretation: that there is a very dense cluster of stars of ordinary mass there.

Ebbetts's response is that it is difficult to explain the spectrum as the composite of a cluster. Furthermore, stellar statistics are against such a thing, he contends. It would require packing 1,000 ordinary type 0 stars and several dozen of types 03 and 04 into an area about a parsec across. It just requires too many stars in too narrow a space and a cluster packed far more densely than usual. Furthermore, 03's and 04's are rare stars. Getting that many of them together in one place again defies the odds. Another possibility is a binary star, but since the mass has to be there somehow, that solution doesn't escape the supermass problem.

At this point neither side seems to have convinced the other. Further observations will see whether R136a is variable in either optical or ultraviolet. Further theorizing is probable, too. As one astronomer said, theoreticians work with the data in hand. For years nobody saw a star more than 60 times the sun's mass, so the theorists made theories to show why a star bigger than, say, 100 solar masses could not exist. Confronted with the suggestion of such a thing, their first response is to say, "We already proved it can't exist." Later they may take another look. —*D.E. Thomsen*

# Simple replication mimics chromosome

A test-tube system with all the ingredients for initiating replication of a bacterial chromosome has been reported by Stanford University biochemists. For many years, laboratories around the world have searched for a "soup" that would mimic the natural process by which synthesis of DNA begins. The initiation of DNA synthesis is significant in controlling the rate at which a cell reproduces, so knowledge of the initiation process is crucial to questions such as why embryonic cells reproduce more extensively than most adult cells and why cancer cells show rapid and continuous growth. The biochemical system reported by Robert S. Fuller, Jon M. Kaguni and Arthur Kornberg in the December PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES is expected to allow scientists finally to identify and analyze the factors that control initiation of bacterial chromosome replication.

To obtain the new system for initiating replication, the Stanford biochemists worked with plasmids (small rings of DNA) genetically engineered to contain the DNA segment where synthesis always begins in the main bacterial chromosome. The researchers broke open bacteria to obtain a fluid containing a complex assortment of cellular components. They found that DNA synthesis of the plasmids would not begin in this fluid. But when Kornberg and colleagues used a salt, ammonium sulfate, to precipitate some cell components and thus remove them from the fluid, DNA synthesis did begin. The precipitation either concentrated ingredients necessary for initiation or removed ingredients that inhibit the operation. Further experiments showed that the test-tube system requires for its activity proteins recognized from genetic data as essential for normal chromosome replication, and it shares a variety of other features with replication initiation in intact bacteria. The test-tube system has already demonstrated that cellular membranes are not essential for enzymes to interact with the DNA sequence for the initiation of synthesis. Kornberg now plans to sort out the components of this biochemical "machine" and analyze precisely what substances are required. —*J. A. Miller*

# Reversed genetics of thalassemia

Scientists have turned around genetics to discover the detailed mechanism of an inherited disease. Traditional genetic techniques had already identified the human gene defective in $\beta^-$ thalassemia, a disease in which the patient has insufficient amounts of the beta subunit of the blood protein globin. Last year the exact DNA sequence of the gene from each of two patients was worked out. The same single nucleotide subunit change distinguished each of the defective genes from the normal sequence. The defect was not in the region of the gene that codes for the blood protein, but in one of the two noncoding, intervening regions.

How could an altered "silent" stretch of DNA cause such biological havoc? Meinrad Busslinger, Nikos Moschonas and Richard A. Flavell of the National Institute for Medical Research in Mill Hill, London, used an approach called reversed genetics. They started with the gene in question and did experiments to see how it is expressed. They put one $\beta^+$ thalassemia gene from a Turkish Cypriot patient into a plasmid and introduced the plasmid into human (HeLa) cells in laboratory culture. The investigators report in the December CELL that the problem is abnormal splicing of the intervening sequence from the messenger RNA molecules. The single mutation in the DNA sequence causes a pattern (CTATTAG) within the intervening sequence that resembles the site (CCCTTAG) where messenger RNA normally is clipped. Busslinger and colleagues find that with the defective gene, more than 90 percent of the RNA molecules are snipped at the abnormal site. The aberrant splicing shifts the messenger RNA reading frame, so it cannot be translated into functional protein. The incorrect splicing also allows unspliced RNA to accumulate. —*J. A. Miller*