suming that these are the particles that comprise the galaxy's dark mass halo, they argue, swarms of them will constantly be passing through the sun. Whenever one of them collides with a particle of ordinary matter, it will tend to lose most of its kinetic energy and go into orbit in the interior. Thus, over the 4.6-billion-year lifetime of the sun, the cosmions must have accumulated into just the kind of haze that Faulkner and Gilliland originally suggested. Indeed, the picture is remarkably consistent: if the cosmion collision cross section is the right order of magnitude for efficient thermal transport in the sun (roughly 10^{-36} square centimeter), then the present density of cosmions in the sun can be calculated—and it works out to one part in 10^{-12}, exactly what is needed for suppressing neutrino production to the observed levels.

With this encouragement, Faulkner and Gilliland have returned to the problem (*4*), and are currently collaborating with Press and his co-workers on a detailed and self-consistent calculation of the suppression effect.

Unfortunately, there does remain one dark cloud. The most obvious and plausible candidate particle is the photino, which is the hypothetical partner of the photon in the popular theory of supersymmetry. But it does not quite seem to work. The problem is that the photinos, if they exist at all, tend to annihilate each other when they meet. (More precisely, the photino is its own antiparticle.) This annihilation process means that they could never accumulate in the sun to the levels just mentioned. In fact, the upper limit on the density works out to be three or four orders of magnitude too low to affect the solar neutrino problem.

Since the same problem plagues many of the other plausible candidates, the cosmion/WIMP solution to the solar neutrino problem has to be rated as little more than an intriguing suggestion. On the other hand, its internal consistency and its relationship with other cosmological phenomena make it particularly attractive. And given the current state of particle physics, it is always possible that the cosmion particles, when and if they are ever observed in the laboratory, will have exactly the properties they need.—**M. MITCHELL WALDROP**

References

1. M. M. Waldrop, *Science* **228**, 978 (1985).
2. G. Steigman, C. L. Sarazin, H. Quintana, J. Faulkner, *Astron. J.* **83**, 1050 (1978).
3. D. N. Spergel and W. H. Press, *Astrophys. J.* **294**, 663 (1985); W. H. Press and D. N. Spergel, to appear in *ibid.* **296**, 679 (1985).
4. J. Faulkner and R. L. Gilliland, to appear in *ibid.* (1 December 1985).

Fixing Nitrogen Without Molybdenum?

The metal molybdenum has for many years been considered a sine qua non for biological nitrogen fixation, the reduction of molecular nitrogen to ammonia by soil- and water-dwelling microorganisms. A variety of evidence indicates that the metal is part of the catalytically active site of nitrogenase, the enzyme that performs the reduction. Perhaps not surprisingly then, when Paul Bishop of North Carolina State University in Raleigh first proposed about 5 years ago that the bacterium *Azotobacter vinelandii* has an alternative system for fixing nitrogen—one that may not require molybdenum—the suggestion was greeted with more than a little skepticism.

However, recent research by Bishop and his colleagues and also by investigators from the Agriculture and Food Research Council Unit of Nitrogen Fixation at the University of Sussex, England, has confirmed that *A. vinelandii* has a second nitrogen-fixing system. As Christine Kennedy of the Sussex group told participants in the Sixth International Symposium on Nitrogen Fixation,* "Work of the past year has established that the alternative system is a reality."

Much of the recent evidence comes from studies of *A. vinelandii* mutants in which the genes coding for the nitrogenase proteins were specifically deleted or inactivated. If the bacterium had only the conventional nitrogen-fixing system, the mutants ought to lose the ability to reduce nitrogen, but this is not the case. For example, Bishop with Robert Eady of Sussex, found that a mutant in which all three nitrogenase structural genes had been deleted carries out the nitrogen-fixing reactions about as effectively as the wild type bacterium, but only when deprived of molybdenum. In contrast, the wild type must have the metal in the culture medium if it is to reduce nitrogen.

These results and others indicate, Bishop says, that the alternative system is activated in *A. vinelandii* by molybdenum starvation. The system may be an adaptation to molybdenum-poor soils. How widespread it is among nitrogen-fixing organisms remains to be determined.

The Bishop group is working to identify the enzymes that participate in the alternative pathway. Normal nitrogenase has two protein components. The molybdenum-iron protein contains both metals and reduces nitrogen with electrons transferred to it by the second protein, which contains only iron. The *A. vinelandii* deletion mutant lacks both of these, but Bishop and John Chisnell, also of North Carolina State, find that it has two new proteins that work together to reduce nitrogen. The larger one may play a role analogous to the molybdenum-iron protein, Bishop suggests. Although physiological studies indicate that molybdenum does not participate in nitrogen reduction by the alternative pathway, purification and analysis of the protein will be necessary to confirm that it does not contain the metal. The smaller of the two new proteins may be the equivalent of the iron protein.

The genes that code for the enzymes of the alternative system are also being sought. Robert Robson of the Sussex group has found that another *Azotobacter* species, *A. chroococcum*, has at least two versions of the *nifH* gene, which codes for the iron protein. One is part of the conventional nitrogen-fixing system; it is located in a cluster with the genes for the molybdenum-iron protein. The second *nifH* gene, which is about 90 percent identical to the first, is not a part of that cluster and may be involved in some fashion in the alternative system. Nevertheless, its role is unclear.

Although the Bishop group has evidence that the second *nifH* is transcribed into messenger RNA in *A. vinelandii* in response to molybdenum deficiency, it apparently does not code for either of the alternative nitrogenase proteins identified by Bishop and Chisnell. *Azotobacter vinelandii* may contain yet a third *nifH* gene.

The recent work has verified the existence of the alternative nitrogen-fixing system and shown that it is activated by molybdenum deficiency, but has left unanswered many questions about the operation of the system and its interaction with the con-

*The symposium was held at Oregon State University in Corvallis on 4 to 10 August.

ventional pathway. Moreover, the absence of molybdenum from the alternative system has not been conclusively demonstrated, although Bishop predicts that this will prove to be the case.

The possibility that biological nitrogen fixation may take place without the intercession of molybdenum is especially intriguing in view of the mysteries still surrounding the function of the metal in the normal nitrogenase. Despite all the evidence implicating molybdenum as an essential component of the enzyme, no one knows yet exactly what it does, as several presentations at the symposium demonstrated. If the alternative system does prove to be molybdenum-free, comparison of its workings with those of the conventional system might lead to new insights into the biochemistry of biological nitrogen fixation.

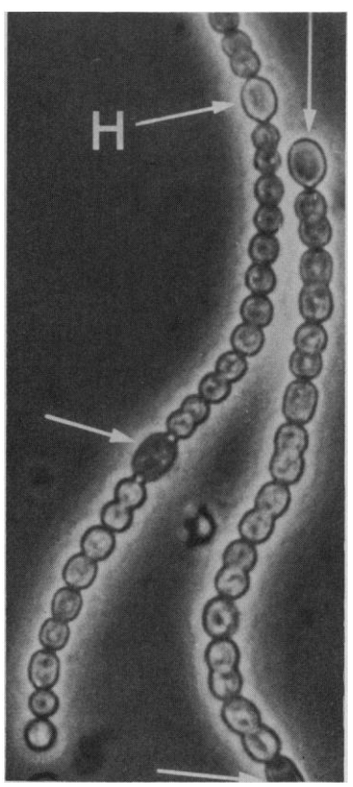
Gene Rearrangements in a Prokaryote

Gene rearrangements during development are generally thought to be restricted to the differentiating cells of higher organisms. The now classic example is the rearrangement of antibody genes during the maturation of antibody-producing cells. However, at the nitrogen-fixation symposium, Robert Haselkorn of the University of Chicago described an unusual example of a developmentally regulated gene rearrangement in a prokaryote, the photosynthetic cyanobacterium *Anabaena* 7120.

When *Anabaena* fails to obtain the fixed nitrogen it needs from the environment, roughly 10 percent of the cells differentiate to form structures called heterocysts. As they differentiate, the cells undergo a series of changes in which they acquire the ability to fix nitrogen for the remaining vegetative cells. In particular, the genes of the *nif* (nitrogen fixation) complex become turned on in the heterocysts and they begin making the nitrogen-reducing enzyme nitrogenase. They also stop producing oxygen, which would otherwise irreversibly poison the enzyme.

In *Klebsiella pneumoniae*, the prototype nitrogen-fixing bacterium, the three genes that code for the nitrogenase proteins, *nifK*, *nifD*, and *nifH* (in the order in which they appear in the genome) are contiguous and are transcribed into messenger RNA as a single unit. But studies of the *Anabaena nif* complex revealed a significant difference.

According to Haselkorn and his colleagues Doug Rice and Barbara Mazur, in the nonnitrogen-fixing, vegetative cells of *Anabaena*, *nifK* is separated from the other two genes by 11 kilobases of apparently unrelated DNA. James Golden of the Chicago group found that during heterocyst differentiation the 11-kilobase sequence is excised as a circle and the three genes become joined and transcribed as they are in *Klebsiella*. The *Anabaena* genome contains only the one copy of the 11-kilobase segment, the ends of which carry identical 11-base-pair sequences.



Anabaena *filaments*

When Anabaena *is deprived of fixed nitrogen, approximately one cell in 10 develops into a heterocyst (arrows).* [*Source: H. Fleming and R. Haselkorn,* Proc. Natl. Acad. Sci. U.S.A. ***70****, 2727 (1973)*]

In addition to the excision of this fragment, there is a second rearrangement to the right of the *nifH* gene during heterocyst development that brings in new DNA from at least 25 kilobases away, according to Martin Mulligan, also of Chicago, and Golden. This rearrangement may also be an excision, but the element removed is not the same as the one excised from within the structural genes.

Removal of the 11-kilobase sequence from the nitrogenase structural genes may be necessary for synthesis of the enzyme. Close examination showed that the sequence actually occurs within the *nifD* gene, although close to the end of its protein-coding region. "Clearly this element interrupts the *nifD* reading frame," Haselkorn explains. "I suspect that only the rearranged genes can give rise to active nitrogenase." He notes, however, that his colleagues are not fully convinced that this is the case. There is a possibility that the unrearranged, truncated *nifD* gene is capable of producing a functional product, although Haselkorn points out that he has never detected any evidence that such a product is actually made.

Introduction of the cloned *Anabaena nif* genes into *Escherichia coli* cells showed that the 11-kilobase fragment can also be excised in this bacterium. These experiments allowed Golden and Peter Lammers of the Haselkorn group to identify a region in the fragment that encodes a protein necessary for the excision. The protein is likely to be a "recombinase," an enzyme that binds to and joins the 11-base pair repeats at the ends of the sequence and results in its removal as a circle. This mode of excision resembles that of certain viral DNA's that integrate into bacterial genomes and are also excised under appropriate conditions. The results suggest that the 11-kilobase sequence might be of viral origin.

What role the sequence might play in the vegetative cells of *Anabaena* that would account for its maintenance there is unclear. It is not needed to prevent nitrogenase synthesis. There are other control mechanisms for that. Nevertheless, the Haselkorn group has found the sequence in *Anabaena* strains collected around the world, a result which indicates that its presence does provide some sort of advantage for the organism.

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