the resulting solar cells and greatly reducing their efficiency, a problem that was only recently resolved by making the die from extremely pure graphite. Silicon carbide and silica (silicon dioxide) have also been considered as die materials. Nonetheless, frequent replacement of dies and crucibles is an inherent part of the concept. Mlavsky hopes eventually to grow ribbons 5 centimeters wide and 125 micrometers thick for periods of at least 16 hours—a schedule that would yield ribbons about 50 meters long—but he believes that ribbons even half as long would prove economically feasible. The major uncertainty still to be resolved is how much the die will dissolve and whether impurities will accumulate to unacceptable levels within that period.

In theoretical studies closely tied to the experiments, Chalmers is modeling the crystal growth process to find optimum manufacturing conditions. There is a trade-off, for example, between the advantages of maintaining a high temperature gradient in the molten silicon and the desire to keep the temperature near the die as low as possible. Experimental problems include the development of heat shields to obtain the desired temperature distribution in and around the crucible and die and the development of equipment to pull a long ribbon of silicon.

Even if all the production problems of the edge-defined growth technique have not been resolved for silicon, the ribbons produced so far do represent a major breakthrough. They contain some grain boundaries and other crystal defects, but few enough that impurities have been a larger problem. To make solar cells, phosphorus is diffused into pieces of the ribbon (which is already doped with boron) to form a semiconductor junction, a grid of contacts to collect the current is deposited on the surface, and the silicon is coated with an antireflective material. Centralab, a major solar cell manufacturer located in El Monte, California, has made and tested cells from some of Tyco's ribbon, and they confirm the 10 percent efficiency obtained. "It's good silicon," says P. Iles of Centralab's research facility.

Iles believes that the availability of silicon ribbon will permit mechanization, if not automation, of the cell manufacturing process, although little has been done as yet. Several years ago, in fact, an ad hoc group of scientists from several industries and research laboratories with interests in silicon products studied the prospects for large-scale production of solar cells, starting with the at that time doubtful assumption that silicon ribbon yielding 10 percent efficient cells was available. They concluded that ribbon could be converted on a large scale to solar cell arrays for something like $22 per kilogram. Assuming that the cost of making raw high-grade silicon can be reduced substantially (it now costs an extravagant $66 per kilogram), Mlavsky estimates that solar panels might be sold at less than $400 per kilowatt (peak power at full sunlight) of capacity, or roughly the equivalent of electric power produced conventionally with oil at $11 per barrel.* Mass production, which these estimates assume, is still clearly some time away, because a large market for solar cells has yet to develop and because the shift from handcrafted production techniques analogous to those in a Swiss watch factory has only just begun.

Crystal growing techniques other than the edge-defined method are also being investigated, although most observers believe the Tyco process to be the best bet at present, because its feasibility is already established. Considerable effort is beginning to be expended on reducing the cost of solar cell production with existing methods. Most firms in the business are reducing prices for cells intended for terrestrial use and attempting to find new applications for their product ($5 per watt in very large quantities was the lowest price cited to *Science*, and remote installations such as lookout towers operated by the Forest Service are currently a prime market). How rapidly new production techniques are brought into operation and how cheap solar cells will eventually be will depend a great deal on the amount of money invested in the effort, which now depends largely on the National Science Foundation's fledgling solar energy research program. But it seems inescapable that solar power in the form of photovoltaic cells could become a reality well before the end of the century.

—ALLEN L. HAMMOND

* A comparison with the cost of power delivered to the consumer is more favorable. A 2-kilowatt rooftop panel costing $1000 and delivering an average of 10 kilowatt-hours per day would pay for itself in about 6 years at the electric rates prevalent in the Washington, D.C., area (about $0.05 per kilowatt-hour).

Autoimmune Diseases in Animals: Useful Models for Immunology

Autoimmune diseases are associated with reactions of an organism's immune system to its own cells. The mechanism is believed to involve, among other things, a loss of tolerance for antigens of a specific tissue or organ such as the thyroid gland, the skeletal muscles, or the myelin sheath of the central nervous system. One approach to understanding this phenomenon is the study of an autoimmune disease that can be produced in animals—experimental allergic encephalomyelitis (EAE). Recent investigators have dealt with such questions as how self-tolerance is lost and how the components of the immune system interact.

Numerous theories to explain loss of self-tolerance have been advanced, but none of them have been experimentally established. Proponents of various theories differ as to whether autoimmunity is caused by the appearance of new mutant cells of the immune system which lack self-tolerance or whether it results from a defect in a control mechanism that normally maintains self-tolerance. In what may be the first step in deciding among these theories, S. Orgad and I. Cohen of the Weizmann Institute in Israel used the fact that the etiology of EAE is well described to design an experiment that provides evidence consistent with the theory that autoimmunity results from a defect in a control mechanism.

When an animal develops EAE, it undergoes a specific autoimmune response: namely, it produces sensitized T lymphocytes (thymus-derived cells of the immune system) that react to a component of the myelin sheath (the structure that insulates axons of the central nervous system). This component is commonly called myelin basic protein (BP). The reaction of T cells to BP results in a destruction of the myelin sheath, whereupon the animal becomes paralyzed and in most cases dies.

Orgad and Cohen sought to deter-

mine whether normal T lymphocytes could lose their self-tolerance if they were maintained outside the body. Accordingly, they obtained T lymphocytes from the thymus glands of rats and cultured them in vitro with a crude extract of soluble proteins (antigens) from the brains and spinal cords of genetically similar rats. This crude extract contained BP, which is an antigen of the central nervous system that can induce EAE.

When the T lymphocytes cultured in the presence of the central nervous system antigens were injected into healthy rats, these animals developed brain lesions like those associated with EAE. But in the group of control rats, which were injected with T lymphocytes cultured in vitro without central nervous system antigens or were injected with central nervous system antigen alone, the animals did not develop the disease. In order to induce EAE in a healthy animal, the animal must be injected with T cells from an animal with EAE or it must be injected with BP together with an emulsion called Freund's complete adjuvant, which specifically stimulates the production of sensitized T cells. Thus the results of Orgad and Cohen are consistent with the hypothesis that self-tolerance depends on mechanisms that operate in vivo to regulate the sensitization of T lymphocytes. The results do not rule out the possibility that those T lymphocytes that are unaffected by these control mechanisms are mutant lymphocytes.

Studying the mechanisms that regulate self-tolerance is difficult because of the interactions between the two major components of the immune system: the T lymphocytes, which can become sensitized to antigens and will subsequently destroy cells that carry those antigens, and the B (bone-marrow derived) lymphocytes, which react to antigens by secreting antibody molecules that bind to those antigens. Sensitized T cells are known to cause EAE when injected into animals, but the role of B cells is less clear. Sensitized T cells must be present before B cells produce antibody to BP. However, J. Howard and N. Gonatas of the University of Pennsylvania Medical School in Philadelphia were able to show that B cells are not by themselves sufficient for the development of EAE.

Howard and Gonatas completely depleted Lewis rats (a strain that is highly susceptible to EAE) of both T and B lymphocytes. They removed the thymus (which is necessary for T cell production) from the animals and then irradiated them so that they could no longer produce B cells. (B cells are produced in the bone marrow, which no longer functions after a large dose of radiation.) These rats were then injected with bone marrow from genetically similar rats whereupon they had B lymphocytes but no T cells. Howard and Gonatas attempted but failed to produce EAE in these animals. But when the rats were injected with T cells from other rats, they were susceptible to EAE.

Are Antibodies Involved?

Although the role of antibody in the pathogenesis of EAE is controversial, many investigators believe that the production of antibodies to BP is not a factor in the development of the disease. Some experiments are consistent with the hypothesis that antibodies to BP may be associated with resistance to or recovery from EAE. For example, D. McFarlin of Emory University in Atlanta showed that, if animals with EAE are kept alive (they are usually killed after they exhibit symptoms of the disease), they often recover and then may experience a second, this time fatal, attack of EAE. Associated with this initial recovery is an increase in circulating antibodies to BP. However, M. Kies of the National Institute of Mental Health in Bethesda, Maryland, points out that no one has yet been able to protect an animal from EAE by inoculating it with antibody-containing serum from an animal that has the disease. Thus evidence that antibody production is a protective mechanism is still weak.

In contrast to McFarlin's results, Kies and her colleagues have found that antibody concentrations may not change when guinea pigs recover from EAE. They caused the animals to produce a great deal of antibody to BP when they developed EAE. They were able to cure these animals by injecting them with BP together with Freund's incomplete adjuvant (which differs from Freund's complete adjuvant in that it does not contain killed mycobacteria and thus does not stimulate T cell production). When the guinea pigs were cured of the disease, their antibody concentrations did not significantly change. Thus Kies postulates that the positive response to treatment with BP may result from some change in the concentration of circulating T lymphocytes rather than from a change in the concentration of antibodies to BP.

In support of her hypothesis, Kies points out that D. Rowley of the University of Chicago recently showed such an effect of an antigen on T lymphocytes of rats. Rowley induced rats to produce T lymphocytes that were sensitive to a given antigen. He then injected the rats with this antigen and found that the number of specifically sensitized T cells in the rats' thoracic ducts was subsequently reduced.

The hypothesis that the presence of an antigen is associated with a decrease in the number of specifically sensitized T cells is also consistent with a result of S. Levine of New York Medical College in New York. By transferring to rats T cells that were sensitive to BP, Levine could induce EAE in these animals. However, the development of EAE in recipient rats was delayed or prevented if BP was injected together with the sensitized T cells.

Kies together with E. Alvord of the University of Washington in Seattle and their associates are now using their knowledge of the complete amino acid sequence of the guinea pig BP to investigate the biochemical basis of T cell sensitization and antibody production. They have discovered that different parts of the BP molecule have different effects on the immune system of guinea pigs and have identified sites that appear to be necessary for antigen specificity, for antigen production, and for EAE development.

In addition to studying mechanisms by which self-tolerance can be lost and relations between T and B cells, investigators are using EAE to study such fundamental problems as genetic influences on autoimmune responses (various inbred strains of animals differ markedly in their susceptibility to EAE) and the curious role that the killed mycobacteria in Freund's complete adjuvant play in the induction of sensitized T cells. Since sensitized T cells are involved in many allergic reactions, research on EAE is related to understanding several specific reactions as well as to solving fundamental problems in immunology. Because this disease is so well characterized, it is proving invaluable to those who are attempting to study the immune system.

—GINA BARI KOLATA

Additional Reading

1. S. Orgad and I. Cohen, *Science* **183**, 1083 (1974).
→ B. F. Driscoll, A. J. Kramer, M. W. Kies, *ibid.* **184**, 73 (1974).