females are immunized with the paternal form before mating, the production of paternal allotype will be completely or chronically suppressed in more than half the progeny by the time they are 6 months old.

The Herzenbergs showed that an active factor associated with T lymphocytes is responsible for chronic allotype suppression. They irradiate BALB/c mice to destroy the animals' immune system and prevent graft rejection, and then inject them with spleen cells from unsuppressed hybrid progeny. The injected spleen cells produce both maternal and paternal allotypes. However, when a mixture of equal numbers of normal and suppressed hybrid spleen cells are injected, much less of the paternal allotype is produced. In fact, the cells produce as little as do chronically suppressed spleen cells when only they are injected. Treating the suppressed spleen cells with an antiserum that destroys T cells destroyed the suppressing activity, which again shows that T cells can prevent antibody production.

Thus, T cells can suppress antibody production in a number of experimental systems. Many investigators are coming to the conclusion that this suppression is essential for normal control of immune responses. If that is the case, then malfunction of that control could result in either an excess of antibodies, as in allergic reactions, or production of aberrant antibodies, such as the autoantibodies found in autoimmune conditions, or a lack of antibodies and consequent immunodeficiency.

The immune system does not normally attack the body's own tissues, but occasionally this tolerance for self is lost and autoimmunity results. A number of severe debilitating diseases, including rheumatoid arthritis and systemic lupus erythematosus (SLE), are thought to be autoimmune conditions. One of the characteristics of these diseases is the presence of autoantibodies, that is, antibodies against self.

Many investigators think that suppressor T cells normally prevent the production of autoantibodies and that a deficiency of suppressors may there-

Astronomers Steer Students Away

In spite of the difficulties that new Ph.D.'s in astronomy have finding jobs, the ranks of astronomy graduate students are still growing rapidly, according to a recent study by the National Academy of Sciences. The astronomy manpower committee, chaired by Leo Goldberg at Kitt Peak National Observatory, advised that the production of new Ph.D.'s be reduced and found that many of them had received insufficient diversification in their graduate work, especially with respect to physics courses. The committee took the unusual step of recommending that the standard letter excerpted below be sent to all prospective astronomy students.

Dear Student:

Traditionally, the most desirable positions for a young astronomer have been those at large universities or major observatories where one could devote a substantial fraction of time to research. However, the increased numbers of graduate students in the physical sciences, the overall decreased enrollment at the undergraduate levels, and the decreased funds available for research have combined to make the prospect of obtaining such positions increasingly difficult. Opportunities will be available, of course, but the ratio of candidates to available positions at current rates of Ph.D. production is projected to be more than 4 to 1.

You should now seriously consider whether your interest in the field is so great that you wish to devote five more years of hard study to astronomy, knowing that at the end of those years the main job openings will probably be in fields entirely different from astronomy.

With the subject matter of the physical sciences changing so rapidly, it is not uncommon for a person who acquires knowledge and research experience in one field to change later to an allied one. The positions which will be available to you will be filled on a highly competitive basis, and you must be very honest with yourself in assessing your chances of success not only for completion of the Ph.D. but also in the competition for employment. We will do everything possible to advise you carefully as to your chances for future success in both graduate school and your intended career.

For the teachers of astronomy, a review of the graduate curricula and attitudes toward student job prospects were recommended.—W.D.M. fore contribute to the etiology of autoimmunity. For example, Alfred Steinberg and his colleagues at the National Institute of Arthritis, Metabolism, and Digestive Diseases, and Norman Talal of the University of California Medical Center in San Francisco, have found that there is a progressive decrease with age in suppressor effects in New Zealand Black (NZB) mice. This strain of mouse is frequently used by investigators studying autoimmunity because the animals spontaneously develop a disease that closely resembles SLE in humans.

According to Steinberg, as NZB mice age, they give an increased antibody response to immunization with pneumococcal polysaccharide, the same bacterial antigen used by Baker. There is no such increase in the response of another strain of mice that is not subject to autoimmune disease. The fact that thymus cells from young NZB mice reduced the excessive antibody response of the older animals supports the hypothesis that it is caused by loss of suppressor T cells. Steinberg also observed a decrease in cell-mediated immunity as NZB mice aged.

The losses of suppressor functions appear before other thymic functions decrease in NZB mice and before the onset of autoimmune symptoms. The cause of the loss of suppressor T cells is unknown. Viruses and genetic factors probably play a role. Investigators have also suggested that a deficiency of thymic hormones may be involved (*Sci*ence, 28 March, p. 1183).

Having too many suppressor T cells can be just as harmful as having too few, according to Thomas Waldmann and his colleagues at the National Cancer Institute. They have been investigating an immunodeficiency disease called common variable hypogammaglobulinemia that is characterized by inability of the affected individual to make adequate quantities of antibodies.

This disease, like certain other immunodeficiencies, may be caused by intrinsic defects in B cells or their precursors that prevent them from maturing into antibody-secreting cells or that block synthesis and release of antibodies. The NCI group, however, showed that lymphocytes from at least one group of patients inhibited antibody secretion by cultured lymphocytes of normal individuals who had been stimulated with pokeweed mitogen. Pokeweed mitogen is a lectin, that is, a plant protein that can mimic the natural effects of antigens by stimulating