macrophages[13]. This has been taken to mean that it is the macrophage and not the T cell that is defective. But if the defect were in fact due to the absence of certain T cell clones as a result of somatic selection, then one would expect the F_1 to be unable to respond to antigens presented on nonresponder macrophages. Thus the only way to test unambiguously whether the defect is in the macrophage or the T cell repertoire would be by using fully allogeneic nonresponder macrophages to present the antigen.

In order to do this, Ishii *et al.* had to overcome a technical difficulty: normally, T cells would be expected to mount an immune response to the allogeneic macrophages alone, and this would obscure any proliferative response to the combination of allogeneic Ia and antigen. They therefore had first to remove any T cells reactive to the macrophage alloantigens. This was accomplished by stimulating the T cell population with the macrophages in the absence of antigen and killing the proliferating alloreactive T cells by exposure to bromodeoxyuridine and light. The remaining T cell population was then restimulated with the same nonresponder macrophages, but now in the presence of antigen. The interesting result which they obtained was that these T cells could respond to the antigen in association with the nonresponder macrophages. Even if the T cells were obtained from one nonresponder strain and thus could not see the antigen in association with their own macrophages, they could respond to the same antigen in association with the macrophages from another nonresponder strain. These results suggest that a nonresponder T cell population is only blind to the antigen in the context of its own syngeneic macrophages. This in turn argues strongly against several of the theoretical models discussed above. First, if T cells exist which can recognize the antigen and nonresponder Ia molecules, then genes must exist in the species which encode for these receptors either as direct products or as somatic variants. Second, if T cells can respond to the antigen and nonresponder Ia molecules, then the Ia molecules themselves cannot be defective in binding and presenting the antigen. Therefore, these experiments can be interpreted as arguing against determinant selection models and by default supporting the somatic T cell selection models for generating Ir gene defects.

This is not the first report of nonresponder macrophages presenting antigen to responder T cells. In 1973 Kapp *et al.*[14] demonstrated that nonresponder macrophages could stimulate a helper T cell-supported antibody response from responder spleen cell populations. However, these experiments were criticized because the investigators did not deplete the responding population in alloreactive cells, which might have produced false positive results through complex interactions between lymphocyte subsets and the release of soluble recruitment molecules (so-called positive allogeneic effects)[15]. Even in the experiments reported by Ishii *et al.* one has to ask whether the authors have sufficiently depleted the T cell population in alloreactive cells to eliminate all such effects. They demonstrate that the nonresponder macrophages alone do not induce a proliferative response, suggesting by the criterion of cell division that alloreactivity has been removed. However, given the fact that all the strain combinations reported on in the paper produced a response, one has to worry whether residual alloreactivity, such as the release of recruitment factors, still remains. The authors state that they have some negative results using strain combinations with only partial histocompatibility differences, but these combinations might also generate reduced allogeneic effects.

Nonetheless, the implications of the findings reported in this paper have been supported by recent results from other laboratories. Pierce *et al.*[16] reported that responder T cell populations contain cells capable of helping B cells from nonresponders to make antibody, whereas (responder x nonresponder) F_1 populations lacked such T cells. Even more striking have been the data obtained with cloned T cell populations. Clark and Shevach[17] have isolated several clones from a responder strain of guinea pigs that respond to antigen presented on nonresponder macrophages from a different strain, but not to nonresponder macrophages alone, or to the same antigen presented on responder macrophages. This last result is analogous to that of the Ishii experiments. The beauty of the cloning experiments is that they totally eliminate the possibility of a false positive allogeneic effect since alloreactive T cells have been entirely removed. On the other hand, the problem with the cloning results published so far is that they only describe a few examples. These could represent rare phenotypes and thus might not be sufficient to explain Ir gene effects, which require the absence or lack of stimulation of whole families of T cells with similar specificities[7]. Overall, however, the data from both types of experiments would seem to complement one another, the clones addressing the doubts about alloreactivity and the Ishii experiments addressing the doubts about population size. Thus it would appear that these collective experiments represent a strong challenge to determinant selection models. It remains to be seen how and if this challenge will be met. □

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THE MOVEMENTS OF JUPITER'S ATMOSPHERE

Mr. Darwin describes the bands on Jupiter as "due to the trades and anti-trades" set in motion by the action of solar radiation on the solid body of the planet as are the trade-winds of the earth. Many other eminent astronomers still appear to accept this time-honoured explanation of the phenomena.

Have they reflected on the revelations supplied by the low specific gravity of Jupiter? There is no form of matter with which we are acquainted that could exist at a mean density of about one-fourth of that of the earth, while subject to the enormous pressure due to the mass of Jupiter, unless it were sufficiently hot to render the formation of a solid crust on its surface quite impossible. In order to attribute terrestrial solidity to either Jupiter, Saturn, or Neptune we must invent a new kind of matter as infusible as platinum, and far lighter than hydrogen, or endow it with absolute incompressibility.

These planets, if composed of any of the chemical elements or compounds known to us, can only retain their low density under the enormous pressure of their masses by the agency of proportionately counteracting heat-repulsion. At and about their centres this may be so far overcome by the superincumbent pressure as to produce solid nuclei.

Assuming the existence of such a central nucleus of Jupiter surrounded by a great fluid envelope, how will it be affected by the gravitating reaction of the satellite, supposing the compression to give it a specific gravity exceeding the mean specific gravity of its envelope?

It will obviously perform an eccentric rotation, or reeling, within the envelope. This motion must be very irregular and complex, owing to the different periods and the varying relative positions of the satellites. The effect of such internal reeling upon the surrounding gaseous mass explains far more efficiently than any possibility of solar radiation, the disturbances indicated by the ever-changing belts and spots of this planet.

W. MATTIEU WILLIAMS

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