INTRODUCTION: The exact mechanism involved in the variation of the host response to acute and chronic infections and the development of lesions due to endogenous chronic or latent virus is not completely understood. Since recurrent lesions appear in herpes simplex virus (HSV) infected patients with high serum antibody levels, investigators believe that some other immune mechanism plays a predominant role. Recent investigations have shown that macrophages and lymphocytes (T) are key factors in controlling chronic HSV infections in mice. An elegant series of experiments by Rager-Zisman and Allison using cyclophosphamide-treated HSV infected mice has indicated that protection against this virus infection is predominantly cell-dependent. Their studies seem to imply that humoral antibodies alone play no major role in the recovery of the host. However, they suggest that the data tend to support the theory that antibody-dependent cell-mediated cytotoxicity (ADCC) may well be operative in this specific incidence. Rouse and coworkers have recently confirmed this suggestion. In their studies, the role of the immune mechanism responsible for the recovery of cattle from acute IBV infections, resistance to re-infection and the recrudescence of chronic or latent infection has not until recently been the subject of critical, refined scientific evaluation. The role of environmentally-induced pathogenesis in IBV infections has been investigated, but careful, controlled scientific analysis is difficult. The role ofshift in normal viral behavior during cabinets, nutritional deficiencies have also been suggested. Some of the most intriguing theories is the fact that certain groups of viruses may suppress the cell-mediated immune (CMI) response of acutely infected animals or depress CMI in chronically infected animals thereby allowing recurrent disease to develop. Nevertheless, have been shown to affect the CMI response in other animals. The recent reports of reactivation of IBV infection in young calves by experimental other virus infections tend to support this hypothesis.

Davis and Carmichael studied the effect of cell-mediated and antibody upon the cell-mediated immune response during primary and recurrent infections. They suggested that suppression of cell-mediated immunity, as measured by lymphocyte transformation in whole blood cultures, occurred during CS-induced recrudescence. This adrenocorticotrophic hormone (ACTH) and tricyclic antidepressant induced recrudescence without a companion immunosuppression. Rouse and co-workers have recently published a series of papers concerning their investigations into the immune mechanisms of acute and chronic IBV infections. Their conclusion is that almost all well-known immune mechanisms play a role in the recovery from the maintenance of the recurrence of IBV infections.

Recent investigations have shown that chronic IBV virus infections both from vaccine and field strains could be reinduced with corticosteroid in virtually all previously infected animals and that altered lymphocyte response occurred. In contrast, recrudescence with B lymphocytes and neutrophils functional was altered with cyclophosphamide.