

IV. Conclusion

1. Con A deteriorated hepatic lesions undergoing murine GVHR due to MHC class II disparity histopathologically with the elevation of transaminase levels. It is suggested that IFN- γ might be involved in the progression mechanism. With this inducible model, it might be possible to analyze factors for the down-regulation of autoimmune-related liver injury.
2. GVHR hepatic lesions induced in MHC class II disparate F1 mice were worsened by the administration of antibodies against IL-10. Neutralizing IL-10 caused elevated levels of both IFN- γ and IL-4 mRNA. These findings suggest that IL-10 might play an important role in down-regulating autoimmune-related hepatic lesions.
3. Although the deterioration of inflammatory changes in liver was observed in the present studies, these modified GVHR mice do not progress into fibrosis or cirrhosis. The repeated injury of liver may be required. In addition, the role of the effector cells (such as CD8⁺ T cells, NKT cells) and the participation of other cytokines in these GVHR hepatic lesions have to be examined in future experiments.