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Tow separate mechanisms must be distinguished in the events underling chronic liver diseases. Such diseases may arise from a chronic response to a persisting antigen or toxin. Alternatively the damage may result from production within the host of an auto-antigen, this giving rise to an auto-immune reaction.

Recent theories on the sequence of events leading to the development of chronic liver diseases are centered on defect in T cell function (Dudly et al., 1972 and Eddleston Williams, 1974).

The association of $HLA-A_1$ and $HLA-B_8$ with HB_s -Ag negative chronic active hepatitis raises the possibility that this must be a marker of genetic susceptibility to certain infections or toxic agents. However attempts to confirm this and to correlate other liver diseases with particular HLA-antigens have been disappointing (O'Brien et al., 1986).

Present knowledge suggest that a series of separate but probably related immunological mechanisms are involved in both the initiation and perpetuation of many liver disorders.

The aim at the current study is to evaluate the immune functions in children with chronic liver disease and whether or not they have any role in the pathogenesis or pathology of such disease.