

In normal immune responses, CD4+ helper T lymphocytes would not recognise self-peptides on HLA class II receptors since they would have been deleted in the thymus. In dermatomyositis, a breakdown in this mechanism causes autoreactive CD4+ helper T lymphocytes that can recognise self-peptides on HLA class II DQA1*0501 or DQB1*0301 receptors to escape from the thymus. These cells are activated by dendritic cells and later by macrophages and can now provide T cell help to autoreactive B cells. Activation of the B cells leads to the generation of autoimmune antibody secreting plasma cells, as well as new memory cells and also induces higher affinity and isotype switched B cells.

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