In dermatomyositis, the structural similarity between a membrane protein expressed on the surface of capillary endothelial cells in muscle and skin and a protein antigen derived from the pathogen causes the antibodies generated against the pathogen to also bind to host cells. Via a breakdown in the deletion of autoimmune CD4+ helper T lymphocytes in the thymus, these cells provide activation signals to autoreactive B cells that orchestrate autoimmune reactions that become self-perpetuating and does not require the presence of the initial triggering pathogen.