CBA/CaHN-Btk^{xd}/J mice have a mutation in the Bruton’s tyrosine kinase gene (Btk), and are a model of human X-linked immunodeficiency. They have a B-lymphocyte-specific defect that results in an inability to launch an antibody response to thymus-independent type II antigens, although they do produce normal amounts of antibody in response to some protein antigens. They have low serum IgM and IgG3 and a reduced number of B-cells. Moreover, the B-cells that are present have a reduced surface IgM to IgD ratio, which suggests a disorder in B-cell maturation.
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