New insights into disease-specific absence of complement factor H related protein C in mouse models of spontaneous autoimmune diseases.

Mehta G, Ferreira VP, Skerka C, Zipfel PF, Banda NK (2014) New insights into disease-specific absence of complement factor H related protein C in mouse models of spontaneous autoimmune diseases. *Mol Immunol* 62(1), 235-248.

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PubMed

Abstract

Complement factor H (CFH) protein is an inhibitor of the alternative pathway of complement (AP) both in the fluid phase and on the surface of host cells. Mouse and human complement factor H-related (CFHR) proteins also belong to the fH family of plasma glycoproteins. The main goal of the current study was to compare the presence of mRNA for two mCFHR proteins in spontaneously developing autoimmune diseases in mice such as dense deposit disease (DDD), diabetes mellitus (DM), basal laminar deposits (BLD), collagen antibody-induced arthrits (CAIA) and systemic lupus erythematosus (SLE). Here we report for the first time that the CFHR-C mRNA was universally absent in the liver from three strains of lupus-prone mice and in a diabetic-prone mouse strain. The mRNA levels (pg/ng) for CFH and CFHR-B in MRL-lpr/lpr, at 9wks and 23wks were 707.2±44.4, 54.5±5.75 and 729±252.9, 74.04±22.76, respectively. The mRNA levels for CFH and CFHR-B in NZB/NZW mice, at 9wks and 54wks were 579.9±23.8, 58.8±1.41 and 890.3±135.2, 63.30±9.2, respectively. CFHR-C protein was absent in the circulation of MRL-lpr/lpr and NZB/NZW mice

before and after the development of lupus. Similarly, mRNA and protein for CFHR-C was universally absent in liver and other organs and in the circulation of NOD mice before and after the development of DM. In contrast, the mRNAs for CFH, CFHR-B and CFHR-C were universally present in the liver from mice with and without DDD, BLD and CAIA. The levels of mRNA for CFHR-B in mice with and without BLD were ~4 times higher than the mice with lupus. The complete absence of mRNA for CFHR-C in lupus and diabetic-prone strains indicates that polymorphic variation within the mouse CFHR family exists and raises the possibility that such variation contributes to lupus and diabetic phenotypes.

Beteiligte Forschungseinheiten

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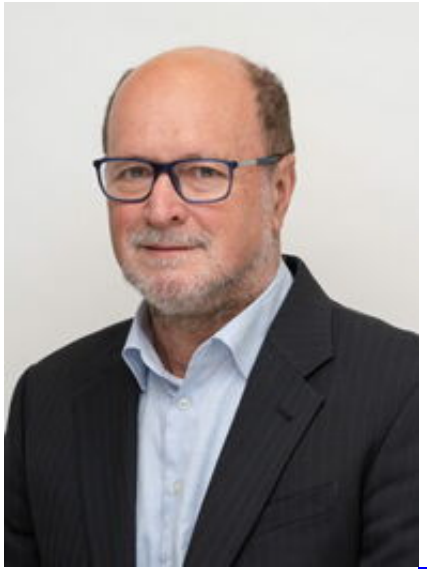

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Identifier

doi: S0161-5890(14)00159-X

PMID: 25033230