epitope 1 is recognized by WM63, J4-57, and J-51; epitope 2 is recognized by T155 (WM68), K31, and Tü145; epitope 3 is defined by mAb (T120 (LOMn25); and epitope 4 is identified by mAb HuLym3 (see introductory diagram).

Function

There has been no specific function assigned to CD48 in humans, although a recent study in the murine system clearly demonstrated that CD48 is a ligand for CD2 [9]. However, no CD58 counterpart has been identified in rodents thus far and, in contrast to humans, CD48 is found on rodent endothelium [12]. Thus, CD48 in rodents may play the same role as CD58 in humans.

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AS1.3 CD55 cluster report

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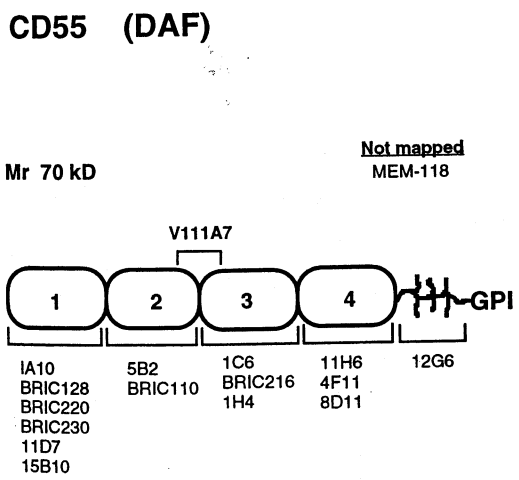

CD55 (decay accelerating factor, DAF) was clustered in the Fourth Workshop [1] with four monoclonal antibodies (mAb). Three CD55 mAb were studied in the Fifth Workshop, S016 (MEM-118) and S031 (IA10) in the Adhesion Section and T158 (143-30) in the T-cell section.

Cellular expression

CD55 is widely distributed. Previous reports described the absence of CD55 on NK cells [2]; however, a recent study with a more sensitive flow cytometer found low levels of CD55 on CD56+ cells [Solomon *et al.*, AS1.12]. Studies in this Workshop confirmed previous studies [1] demonstrating the absence of CD55 antigen on the surface of affected cells of

patients with paroxysmal nocturnal haemoglobinuria (PNH) and cell lines with the glycosylphosphatidylinositol (GPI)-anchor-deficient phenotype [Solomon *et al.*, AS1.12; Klickstein *et al.*, AS1.7; Schubert *et al.*, AS1.10].

Immunochemistry

Immunoprecipitation studies from the Fourth Workshop and the literature, as well as the preclustering analysis, demonstrate a single, broad 70–75 kDa band, that migrates slightly more slowly upon reduction [1,2]. Studies from this Workshop confirmed the presence of a GPI anchor on all cells studied [Solomon *et al.*, AS1.12; Klickstein *et al.*, AS1.7; Schubert *et al.*, AS1.10]. Hořejší and colleagues found coprecipitating protein kinase activity with CD55 mAb [Angelisová *et al.*, AS1.8] as with mAb to other GPI-anchored antigens. Studies with specific glycosidases have shown the presence of both N- and O-linked carbohydrate.

Molecular cloning

Two groups independently identified cDNA clones for CD55 [3,4]. The translated cDNA contains 347 amino acids and the extracellular domain is comprised of four short consensus repeats (SCRs) typical of members of the regulator of complement activation (RCA) gene family. The membrane proximal region is serine- and threonine-rich and is the site of O-glycosylation as determined by deletion mutagenesis [5]. The gene for CD55 has been mapped to the RCA locus at chromosome 1 band q32, together with the genes for the homologous proteins CD21, CD35, and CD46 [6].

Transfectant and epitope analysis

A recent study in the literature examined a panel of CD55 deletion mutants where each of the four SCRs or the serine/threonine-rich region was individually deleted. This study found that the blocking mAb to CD55, 1C6 and 1H4, mapped to SCR3, while other mAb mapped elsewhere (see introductory diagram) [5]. Expression of the CD55 mutants in CHO cells and assay for protection of the transfected cells from complement-mediated lysis showed that deletion of SCR1 had no effect on DAF function, while deletion of any one of the other SCRs or deletion of the serine/threonine rich region resulted in loss of function. Substitution of an unrelated sequence for the serine/threonine-rich region restored function, suggesting it serves a spacer function [5].

Function

CD55 was initially identified as an integral membrane protein of erythrocytes that accelerated the decay of C3 convertase deposited on the cell surface [2]. CD55 does not have cofactor activity for factor I mediated C3b inactivation nor does it mediate rosetting with particles coated with C3 or C4 fragments. CD55 and CD59 are the two GPI-anchored complement regulatory proteins absent on affected haemopoietic cells of patients with PNH [2,7]. The identification of patients with selective deficiency of CD55 [8,9] or CD59 [10] demonstrated that only the CD59-deficient genotype resulted in a haemolytic phenotype, thus indicating that CD59 is probably more important than CD55 in protecting cells from complement-mediated lysis; however, the number of patients studied has been small. Like other GPI-anchored antigens, CD55 may have a signalling function in some cell types [11].

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