

Antibody-dependent cellular cytotoxicity (ADCC), also called antibody-dependent cell-mediated cytotoxicity, is an immune mechanism through which Fc receptor-bearing effector cells can recognize and kill antibody-coated target cells expressing tumor- or pathogen-derived antigens on their surface. Numerous associations between ADCC activity, Fc receptor polymorphisms, and clinical outcomes have been observed in both the settings of vaccination and monoclonal antibody therapy. Here, the effector cells and receptors involved in ADCC are introduced, followed by a description of the four main stages and mechanisms leading to the antibody-dependent effector-mediated killing of the target cell: (1) Recognition of the target cell and Fc receptor cross-linking on the surface of the effector cell; (2) phosphorylation of immunoreceptor tyrosine-based activation motifs (ITAMs) by cellular src kinases within the effector cell; (3) triggering of three main downstream signaling pathways in the effector cell, resulting in cytotoxic granule polarization and release; and (4) killing of the target cell via the predominant perforin/granzyme cell death pathway. Further, a summary and a discussion are presented in relation to case studies in which in vitro ADCC activity correlates with protection against infectious diseases and outcomes in monoclonal antibody therapy of cancer in vivo. The means by which these mechanisms are currently being exploited by recombinant antibody engineering, and a path toward a future in which designed vaccines take advantage of variant ADCC activity are also discussed. Throughout the chapter, attention is drawn to the fact that, while the majority of ADCC studies have been based on research using peripheral blood mononuclear cells in which NK cells have been assumed to be the main effectors, questions remain unanswered about ADCC mediated by non-NK cell populations in peripheral blood and in mucosal compartments.

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Antibody-Dependent Cellular Cytotoxicity (ADCC) Mediated by Human Killer Lymphocytes (K-Cells) (1) Certain cytolytic T lymphocytes (CTL) may destroy target cells after contact interaction Commonly, the effector cells exhibiting ADCC have surface receptors for the Fc-part of Download to read the full chapter text. Fc effector functions such as antibody-dependent cell-mediated cytotoxicity Keywords: monoclonal antibody, ADCC, CD89, Fc γ RI, tandem, . E:T ratios were when KC cells were used as effector cells, and .. Kelton W, Mehta N, Charab W, Lee J, Lee C-H, Kojima T, Kang TH, Georgiou G.. Introduction - Results - Discussion - Methods. Once activated through Fc receptors by antibodies bound to target cells, NK cells This antibody-dependent cell-mediated cytotoxicity (ADCC) of tumor cells is utilized in .. activation (74), which is elicited by the CH2 domain on antibodies (75). inducible-1 gene (RAE-1) permits natural killer cell-mediated rejection of a. Spring BioProcessing Journal [Vol/No.1]. Since cellular cytotoxicity (ADCC), complement-dependent cytotoxicity cal function such as antibody-dependent cellular cytotoxicity . ADCP assay using flow cytometry readouts for confir- mation and the data in accordance with EP and USP statistics chapters. Chapter 1 - Antibody-Dependent Cellular Cytotoxicity (ADCC) (1) Recognition of the target cell and Fc receptor cross-linking on the surface of the effector cell;. Antibody-dependent cellular cytotoxicity (ADCC), involves the Fc γ RIIIa (or ortholog . Figure 1. Common in vitro influenza Fc-receptor assays. Virus Neutralization by HIV-1 Env-Specific Monoclonal Antibodies. Benjamin von Bredow, a Juan IgG constant (Fc) domain can recruit cellular mediators of anti- body-dependent cell-mediated

cytotoxicity (ADCC) and phago- mond PW, Olsen OA, Phung P, Fling S, Wong C-H, Phogat S, Wrin. T, Simek.

Antibody-dependent cellular cytotoxicity (ADCC) has recently been Therapeutic antibodies fully lacking the core fucose of the Fc Shigeru Iida,; Reiko Kuni-Kamochi,; Katsuhiko Mori,; Hirofumi View ArticlePubMedGoogle Scholar; Schauer U, Stemberg F, Rieger CH, Borte M, Schubert S, Riedel F.

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