Search Antibody Database

Found 1 matching record:

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MAb ID	50-69 (SZ-50.69, 50-69D, 50.69, 50-6910)
HXB2 Location	Env DNA(7959..8033)
Author Location	gp41(BH10)
Research Contact	Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY
Epitope	(Discontinuous epitope)
Ab Type	gp41 cluster I
Neutralizing	
Species (Isotype)	human(IgG2κ)
Patient	
Immunogen	HIV-1 infection
Keywords	ADCC, antibody binding site, antibody generation, antibody interactions, antibody polyreactivity, antibody sequence, binding affinity, complement, dendritic cells, enhancing activity, immunotoxin, kinetics, mimotopes, neutralization, rate of progression, review, subtype comparisons, vaccine antigen design, vaccine-induced immune responses, variant cross-reactivity

Env Epitope Map

Notes

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- 50-69:The authors selected an optimal panel of diverse HIV-1 envelope glycoproteins to represent the antigenic diversity of HIV globally in order to be used as antigen

candidates. The selection was based on genetic and geographic diversity, and experimentally and computationally evaluated humoral responses. The eligibility of the envelopes as vaccine candidates was evaluated against a panel of antibodies for breadth, affinity, binding and durability of vaccine-elicited responses. The antigen panel was capable of detecting the spectrum of V2-specific antibodies that target epitopes from the V2 strand C (V2p), the integrin binding motif in V2 (V2i), and the quaternary epitope at the apex of the trimer (V2q). *Yates2018* (**vaccine antigen design**, **vaccine-induced immune responses**, **binding affinity**)

- 50-69: Directed antibody-dependent cellular cytotoxicity, ADCC. *Tyler1990* (**ADCC**)
- 50-69: The complexity of the epitopes recognized by ADCC responses in HIV-1 infected individuals and candidate vaccine recipients is discussed in this review. 50-69 is discussed as the Cluster I (HR2)region-targeting, non-neutralizing anti-gp41 mAb exhibiting ADCC activity and having a discontinuous epitope. *Pollara2013* (**ADCC**, **review**)
- 50-69: The capacity of 50-69 to block completely the activity of the anti-HIV peptide T20 was investigated. T20 inhibited the fusion or syncytia formation between co-cultured CHO-WT cells expressing HIV-1 HXB2 envelope glycoprotein on their surface and HeLaT4 cells. 50-69 was not able to block the anti-fusion effect of T20. *Vincent2012* (**antibody interactions**)
- 50-69D: Study demonstrated that polyreactivity is common among human gp41 cluster II (98-6, 167-D and 126-6)but not cluster I (240D, 246D, 50-69D) antibodies. However, unlike 2F5, cluster II MAbs bind strongly to oligomeric forms of Env gp140 but not to gp41 peptide complexes, suggesting that polyreactivity is necessary but not sufficient for neutralization. *Dennison2011a* (**antibody polyreactivity**)
- 50-69: 50-69 recognized trimeric, dimeric and monomeric forms of cross-linked sgp140(-) Env glycoprotein, but it precipitated monomeric and dimeric forms less efficiently, indicating that the conformation of the cluster I region can be influenced by the oligomeric state of HIV-1 envelope proteins. *Yuan2009* (**antibody binding site**)
- 50-69D: The Ig usage for variable heavy chain of this Ab was as follows: IGHV:1-69*01, IGHD:2-21, D-RF:2, IGHJ:4. Non-V3 mAbs preferentially used the VH1-69 gene segment. In contrast to V3 mAbs, these non-V3 mAbs used several VH4 gene segments and the D3-9 gene segment. Similarly to the V3 mAbs, the non-V3 mAbs used the VH3 gene family in a reduced manner. *Gorny2009* (**antibody sequence**)
- 50-69: 50-69 reacted with maltose-binding protein MBP32, containing both HR1 and HR2 domains of gp41, but did not react with MBP37 and MBP44, containing only the HR2 domain, nor with MBP-HR1, containing only the HR1 domain. In addition, 50-69 bound to MBP44/N36 and MBP-HR1/C34 complexes reaching a plateau at a concentration of ˜; 1 µg/ml. In ELISA, 50-69 reacted with the complex formed between MBP-HR1 and H44 (His-targeted protein) and C34, but failed to recognize the mixture of MBP-HR1 and T20, MBP3 and C34, and MBP3 and H44. In addition, 50-69 failed to recognize the peptide complex N36/C34. *Vincent2008* (**antibody binding site**)
- 50-69: This Ab was used in the analysis of clade C gp140 (97CN54) antigenicity and was shown to bind to this molecule. 50-69 was also used in a competition assay where it was shown to mildly inhibit binding of N3C5 Ab and relatively inhibit binding of N03B11 Ab

(43-61%), indicating proximity of their epitopes. *Sheppard2007a* (**antibody binding site**, **antibody interactions**, **variant cross-reactivity**, **binding affinity**)

- 50-69: To test the immunogenicity of three molecularly engineered gp41 variants on the cell surface their reactivity with 50-69 Ab was assessed. The reactivity of 4cSSL24 variant was comparable to gp160 while the other two variants were not recognized by this Ab since the epitope for this Ab was not present in these variants. *Kim2007* (**binding affinity**)
- 50-69: Point mutations in the highly conserved structural motif LLP-2 within the intracytoplasmic tail of gp41 resulted in conformational alternations of both gp41 and gp120. The alternations did not affect virus CD4 binding, coreceptor binding site exposure, or infectivity of the virus, but did result in decreased binding and neutralization by certain MAbs and human sera. 50-69 exhibited similar levels of binding to both the LLP-2 mutant and wildtype viruses, indicating that its epitope was not altered by the mutation. *Kalia2005* (**antibody binding site**, **binding affinity**)
- 50.69: 50.69 was found to bind to both monomeric and oligomeric gp41. Binding of this Ab to H9/IIIB-infected cells gave a strong signal which was increased by sCD4 pretreatment. Binding to H9/MN-infected cells gave a low signal which increased dramatically with sCD4 pretreatment. Sera from both long-term survivors and AIDS patients inhibited binding of 50.69 to H9/IIIB-infected cells. *Usami2005* (**antibody binding site**, **rate of progression**)
- 50-69: This Ab was shown to inhibit HIV-1 BaL replication in macrophages but not in PHA-stimulated PBMCs. It is suggested that inhibition of HIV replication occurs by an IgG-FcγR-dependent interaction leading to endocytosis and degradation of HIV particles. *Holl2006* (**neutralization**, **dendritic cells**)
- 50-69: Increased binding of 50-69 Ab to gp41 in the presence of CD4 was abrogated by the small molecule HIV-1 entry inhibitor IC9564, suggesting that IC9564 arrests gp120 into a fusion-incompetent conformation unable to expose 50-69 epitope. *Huang2007* (**antibody binding site**)
- 50-69: Sera from two HIV+ people and a panel of MAbs were used to explore susceptibility to neutralization in the presence or absence of glycans within or adjacent to the V3 loop and within the C2, C4 and V5 regions of HIV-1 SF162 env gp120. SF162 and each of the five glycosylation mutants studied were all neutralization resistant to 50-69. V3 glycans tended to shield V3 loop, CD4 and co-receptor MAb binding sites, while C4 and V5 glycans shielded V3 loop, CD4, gp41 but not co-receptor MAb binding sites. Selective removal of glycans from a vaccine candidate may enable greater access to neutralization susceptible epitopes. *McCaffrey2004* (**antibody binding site**, **vaccine antigen design**)
- 50-69: The role of serine proteases on HIV infection was explored. Trypsin decreased the binding of most Env MAb tested and diminished cell fusion of H9 cells infected with HIV-1 LAI virus (H9/IIIB) to MAGI cells. In contrast, thrombin increased the binding of MAbs to gp120 epitopes near the CD4 and CCR5 binding sites, and increased cell fusion. Binding of 17b and F105 was decreased by trypsin, but increased by thrombin. gp41 MAbs 246D, 98.6, 50-69, were decreased by trypsin, unaltered by thrombin, while NAb 2F5 binding

was increased by thrombin. Thrombin may increase HIV-induced cell fusion in blood by causing a conformational activating shift in gp120. *Ling2004* (**antibody binding site**)

- 50-69: This is one of 24 MAbs and Fabs in this database that bind to the highly immunogenic gp41 cluster I region (aa 579 - 604). Only one of these has any neutralizing potential, clone 3. *Gorny2003* (**review**)
- 50-69: Anti-gp41 MAbs were tested in a cell-cell fusion system to investigate the antigenic changes in gp41 during binding and fusion. Cluster I MAbs 50-69, F240, 240-D,3D6, and 246-D recognize a nonhelical hydrophobic region, positions 598-604, that forms a disulfide loop in the six-helix bundle. Cluster II MAbs 98-6 and 126-6 recognized residues 644-663 of gp41, a portion of the second heptad repeat. These MAbs were found to behave similarly, so 50-69 and 98-6 were used as representatives. Exposure of cluster I and cluster II epitopes required CD4 expression on HIV HXB2 Env expressing HeLa target cells, but not the CXCR4 co-receptor. Binding to CD4 exposed hidden cluster I and II epitopes. The MAbs were found to bind to gp120/gp41 complexes, not to gp41 after shedding of gp120, and were localized to at fusing-cell interfaces. Kinetic and binding results indicate that these MAbs are exposed in transitional structures during the fusion process, possibly the prehairpin intermediate prior to co-receptor binding, although other intermediate structures may be involved. They do not bind once syncytia begin to show extensive cytoplasmic mixing. These MAbs failed to inhibit fusion. The NAb 2F5 has a very different behavior in this study. *Finnegan2002* (**antibody binding site**, **kinetics**)
- 50-69: Called 50-69D. Alanine mutations were introduced into the N- and C-terminal alpha-helices of gp41 to destabilize interhelical packing interactions in order to study their inhibitory effect on viral infectivity. These mutations were shown to inhibit viral replication though affecting the conformational transition to the fusion-active form of gp41, and allow increased inhibition by gp41 peptides. 2F5 sensitivity is increased in the mutated viruses, presumably because 2F5s neutralization activity is focused on the transition to the fusion active state. No other gp41 MAb against tested, including NC-1, 50-69D, 1281, 98-6D, 246-D and F240, neutralized the parental or the fusion-deficient mutated viruses. *Follis2002* (**antibody binding site**)
- 50-69: NIH AIDS Research and Reference Reagent Program: 531.
- 50-69: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 -- six gave significant neutralization at 2 to 10 ug/ml: 2F5, 50-69, IgG1b12, 447-52D, 2G12, and 670-D six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98-6, and 1281 -- no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50-69 and 98-6, as well as 98-6 and 2F5. *Verrier2001* (**antibody interactions**)
- 50-69: This paper primarily concerns 4E10 and Z13, MAbs that both bind proximally to the 2F5 binding site to a conserved epitope, and that neutralize some primary isolates from clades B, C, and E -- MAb 50-69 binding to infected cells is enhanced by sCD4, while 4E10 and Z13 binding is essentially unaltered. *Zwick2001b* (**antibody binding site**)
- 50-69: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity -- 50-69 bound the majority of isolates although binding was moderate to weak -- specifies

discontinuous binding site range as aa 579-613. *Nyambi2000* (**variant cross-reactivity**, **subtype comparisons**)

- 50-69: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared -- no MAb was oligomer specific, but gp41 MAb 50-69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98-6, 167-D, 1281, 1342, and 1379) did not show a preference. *Gorny2000b* (**antibody binding site**)
- 50-69: A cluster I epitope that binds to rgp41 567-647, recognizes a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41 -- this MAb doesn't react with either of the peptides N51 or C43 individually -- MAbs 50-69 and 1367 had similar properties -- MAb 50-69 bound the fusogenic form of the protein in liquid phase. *Gorny2000a* (**antibody binding site**)
- 50-69: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605-609 (TTAVP) and 597-609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D -- 5/6 enhancing MAbs identified to date bind to the immunodominant region 579-613 -- identifies non-contiguous W596-G597-C598 and C604-T605 as minimal epitope. *Mitchell1998* (**antibody binding site**)
- 50-69: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library -- 50-69 maps to an immunodominant domain in gp41 -- three groups of peptides were selected, one which seems most closely related to gp41 sequence peptide consensus is WGCxx(RK)(x n)LxC -- the analogous gp41 sequence WGCSGKLIC is present in most M group clades, except D with a common L to H substitution. *Boots1997* (**mimotopes**)
- 50-69: Binding of anti-gp120 MAbs IgG1b12 or 654-30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50-69. *Stamatatos1997* (**antibody interactions**)
- 50-69: Used to test exposure of gp41 upon sCD4 binding. *Klasse1996*
- 50-69: Binds to a linear epitope located in the cluster I region -- binding of 50-69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2. *Binley1996* (**antibody binding site**)
- 50-69: Prebinding of anti-V3, and CD4i MAbs 48d and 17b, but not anti-V2 neutralizing MAbs, expose the 50-69 epitope. *Poignard1996b* (**antibody interactions**)
- 50-69: Does not neutralize HIV-1 LAI. *McDougal1996* (**variant cross-reactivity**)
- 50-69: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells. *Manca1995*
- 50-69: Preferentially binds oligomer -- binding increased after pretreatment of infected cells with sCD4 -- binding domain overlaps site that is critical for gp120-gp41 association. *Sattentau1995* (**antibody binding site**)
- 50-69: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation. *Chen1995* (**antibody binding site**)
- 50-69: Epitope described as cluster I, 601-604, conformational -- does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs. *Laal1994* (**antibody binding site**, **antibody interactions**)

- 50-69: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 -- complement mediated virolysis of MN and IIIB in the presence of sCD4. *Spear1993* (**complement**)
- 50-69: Called SZ-50.69 -- binds to an epitope within aa 579-613. *Eddleston1993* (**antibody binding site**)
- 50-69: Two fold increase in binding to gp120 in the presence of bound sCD4. *Sattentau1991* (**antibody binding site**)
- 50-69: Enhances HIV-1 infection *in vitro* -- synergizes with huMAb 120-16 *in vitro* to enhance HIV-1 infection to level approaching that found in polyclonal anti-HIV serum. *Robinson1991* (**antibody interactions**, **enhancing activity**)
- 50-69: The epitope is affected by the conformation conferred by the two cysteines at amino acids 598 and 604. *Xu1991* (**antibody binding site**)
- 50-69: Kills HIV-infected cells when coupled to deglycosylated ricin A chain. Antibody generation paper. Antibodies were derived from 58 HIV+ patients. Synthesized by immortalization of peripheral blood cells with Epstein-Barr virus. *Gorny1989* (**antibody generation**, **immunotoxin**)
- 50-69: Reacts preferentially with gp160 oligomer, compared to gp41 monomer. *Pinter1989* (**antibody binding site**)
- 50-69: Combined with deglycosylated A chain of ricin is toxic to lines of HIV-infected T cells (H9) and monocytes (U937). *Till1989* (**immunotoxin**)

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