

Search Antibody Database

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[MAB ID](#) 2558

[HXB2
Location](#) Env

[Env
Epitope
Map](#)

[Author
Location](#) Env(92UG037)

[Research
Contact](#) Dr. Zolla-Pazner, Veterans Affairs Center, NY, NY.
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[Epitope](#)

[Subtype](#) A, CRF02_AG

[Ab Type](#) gp120 V3 // V3 glycan (V3g)

[Neutralizing](#) tier 1, tier 2 [View neutralization details](#)

[Species
\(Isotype\)](#) human

[Patient](#)

[Immunogen](#)

[Country](#) Uganda

[Keywords](#) antibody binding site, antibody generation, antibody lineage, antibody sequence, assay or method development, binding affinity, mimotopes, neutralization, structure, subtype comparisons, vaccine antigen design, variant cross-reactivity

Notes

Showing 9 of 9 notes.

- 2558: Neutralizing and binding activity of 18 anti-V3 MAbs from Cameroonian and Indian patients were compared. The Cameroonian patients had CRF01_AG (Env corresponding

to clade A) and Indian patients had clade C. MABs from Cameroonian patients were significantly more cross-neutralizing than those from India, suggesting the preference for CRF02_AG for vaccine design. This MAB was from patient from Cameroon and the subtype of the infecting virus was CRF02_AG. [Andrabi2013](#) (neutralization)

- 2558: VH5-51 gene segment was used by 18 of 51 (35%) anti-V3 MABs. This study analyzed the crystal structure of 5 Fabs encoded by VH5-51/VL lambda genes. Each Fab interacted with key residues at the same 7 positions in the crown of the V3 loop, although the amino acids could vary, suggesting that while V3 is variable in sequence and structurally flexible, a common structure is retained across strains. MAb 2558 interacted with amino acids R304, K305, I307, H308, I309, F317, Y318 of MN V3 peptide. Most of MAb 2558 contact residues were also present at the corresponding positions of the germline VH5-51 gene. All 18 VH5-51 using MABs were studied with a constrained peptide mimotope which preserved the 3D of the VH5-51 derived MABs 2219, 2557, 1006, but did not react with other anti-V3 MABs that recognize different V3 epitopes. 14/18 (2558 included) were reactive with the mimotope, compared to only 1/30 non-VH5-51 MABs. [Gorny2011](#) (mimotopes, antibody sequence, structure, antibody lineage)
- 2558: 2558 neutralizing activity was assessed against pseudoviruses expressing Envs of diverse HIV-1 subtypes from subjects with acute and chronic infection. IC50 neutralization activity was also statistically assessed based on the area under the neutralization curves (AUC). 2558 was able to neutralize 4/57 viruses in U87-based assay and 15/41 viruses in TZM-based assay, including Tier 1 and Tier 2 viruses, viruses of subtypes B, C, D, AG, and viruses from both chronic and acute infections. AUC analysis revealed that 17/57 viruses in the U87-based assay, and 17/41 viruses in the TZM-based assay, were significantly neutralized by this Ab. Thus, the AUC method has the ability to detect low levels of neutralizing activity that otherwise may be missed. [Hioe2010](#) (assay or method development, neutralization, variant cross-reactivity)
- 2558: Two V3-scaffold immunogen constructs were designed and expressed using 3D structures of cholera toxin B (CTB), V3 in the gp120 context, and V3 bound to 447-52D MAB. The construct (V3-CTB) presenting the complete V3 was recognized by 2558 MAB and by the large majority of other MABs (18/24), indicating correctly folded and exposed MAB epitopes. V3-CTB induced V3-binding Abs and Abs displaying cross-clade neutralizing activity in immunized rabbits. Short V3-CTB construct, presenting a V3 fragment in conformation observed in complex with 447-52D, was recognized by 2558 and by 9/24 other MABs. [Totrov2010](#) (vaccine antigen design, binding affinity, structure)
- 2558: The Ig usage for variable heavy chain of this Ab was as follows: IGHV:5-51*03, IGHD:1-26, D-RF:3, IGHJ:4. There was a preferential usage of the VH5-51 gene segment for V3 Abs. The usage of the VH4 family for the V3 Abs was restricted to only one gene segment, VH4-59, and the VH3 gene family was used at a significantly lower level by these Abs. The V3 Abs preferentially used the JH3 and D2-15 gene segments. [Gorny2009](#) (antibody sequence)
- 2558: This Ab was shown to neutralize SF162 and the neutralization sensitivity increased in the SF162 variant with a JR-FL V3 loop, SF162(JR-FL V3). In contrast, no

neutralization was observed of the SF162(JR-FL V1/V2) variant and was somewhat restored in the SF162(JR-FL V1/V2/V3) variant, indicating that the masking of the V1/V2 loop plays a much greater role in restricting neutralization sensitivity than the variations in V3. This Ab was shown to neutralize viruses with V3 sequences from several different subtypes (B, F, A1, H, C, CRF02_AG and CRF01_AE). This Ab failed to neutralize SF162(JR-FL V1/V2) with V3 derived from CRF02_AG but not A1 and C, indicating effective V1/V2-mediated masking of some HIV-1 clades. The effect on the neutralization sensitivity of the residue at the crown of the V3 loop (position 18) was shown to be low for this Ab. [Krachmarov2006](#) (neutralization, variant cross-reactivity, subtype comparisons)

- 2558: This MAb was derived from plasma from a patient with env clade A virus with the GPGQ V3 motif. When cross-reactivity was tested, this Ab bound to both the V3subtypeB-fusion protein containing GPGR motif and V3subtypeA-fusion protein containing GPGQ motif. This Ab was also shown to be able to neutralize both clade B psSF162 (GPGR) and clade C psMW965 (GPGQ) virus and the majority of subtype B and non-B primary isolates. [Gorny2006](#) (neutralization, variant cross-reactivity, binding affinity, subtype comparisons)
- 2558: Sera from subtype A infected individuals from Cameroon have antibodies that react strongly with subtype A and subtype B V3 loops in fusion proteins, and neutralize SF162 pseudotypes, while sera from 47 subtype B infected individuals reacted only with subtype B V3s. Sera from Cameroon did not neutralize primary A or B isolates, due to indirect masking by the V1/V2 domain rather than due to loss of the target epitope. 2557 was derived from a person infected with a clade A or CRF02 virus, and binds to A and B V3 loops. Neutralization of JR-FL and SF162(UG V3) by anti-V3 MAbs 2557, 2558, 2601, but not subtype A primary isolates despite binding to the subtype A V3 loops, suggested masking by V1V2 blocking of neutralization by these antibodies. [Krachmarov2005](#) (antibody binding site, variant cross-reactivity, subtype comparisons)
- 2558: MAbs 2557 and 2558 were produced from individuals who were infected with the CRF02AG virus subtype and living in Cameroon. V3 MAb neutralization is influenced by retaining the epitope, exposure on the intact virion, mobility during CD4-induced conformational change, and affinity. Anti-V3 MAbs selected using V3 peptides neutralize less effectively than V3 MAbs selected using fusion proteins or gp120, suggesting antigenic conformation is important. This antibody was selected using an A clade fusion protein, 92UG037. It is unusual in that it is a V3 antibody selected for conformational aspects using an A clade virus, with a V3 GPGQ tip -- clade B viruses are usually used and have GPGR tips. It cross-neutralizes and binds B clade HIV SF162. [Gorny2004](#) (antibody binding site, antibody generation)

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Showing 9 of 9 references.

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