



(86) Date de dépôt PCT/PCT Filing Date: 2013/09/12
(87) Date publication PCT/PCT Publication Date: 2014/03/20
(45) Date de délivrance/Issue Date: 2024/06/18
(85) Entrée phase nationale/National Entry: 2015/03/12
(86) N° demande PCT/PCT Application No.: US 2013/000210
(87) N° publication PCT/PCT Publication No.: 2014/042669
(30) Priorités/Priorities: 2012/09/12 (US61/700,252);
2012/10/01 (US61/708,466); 2013/02/13 (US61/764,421)

(51) Cl.Int./Int.Cl. *C07K 14/155* (2006.01),
A61K 39/12 (2006.01), *A61P 31/18* (2006.01)

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(54) Titre : IMMUNOGENES INDUCTEURS D'ANTICORPS DU VIH-1

(54) Title: HIV-1 ANTIBODY EVOLUTION IMMUNOGENS

(57) Abrégé/Abstract:

The present invention relates, in general, to HIV-1 and, in particular, to broadly neutralizing HIV-1 antibodies, and to HIV-1 immunogens and to methods of using such immunogens to induce the production of broadly neutralizing HIV-1 antibodies in a subject (e.g., a human).



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(12) INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)

(19) World Intellectual Property
Organization
International Bureau



(43) International Publication Date
20 March 2014 (20.03.2014)

(10) International Publication Number
WO 2014/042669 A1

(51) International Patent Classification:

C07K 14/155 (2006.01) **A61P 31/18** (2006.01)
A61K 39/12 (2006.01)

(21) International Application Number:

PCT/US2013/000210

(22) International Filing Date:

12 September 2013 (12.09.2013)

(25) Filing Language:

English

(26) Publication Language:

English

(30) Priority Data:

61/700,252 12 September 2012 (12.09.2012) US
61/708,466 1 October 2012 (01.10.2012) US
61/764,421 13 February 2013 (13.02.2013) US

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(81) **Designated States** (*unless otherwise indicated, for every kind of national protection available*): AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BN, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PA, PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SA, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW.

(84) **Designated States** (*unless otherwise indicated, for every kind of regional protection available*): ARIPO (BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW), Eurasian (AM, AZ, BY, KG, KZ, RU, TJ, TM), European (AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR), OAPI (BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, KM, ML, MR, NE, SN, TD, TG).

Declarations under Rule 4.17:

— *of inventorship (Rule 4.17(iv))*

Published:

— *with international search report (Art. 21(3))*

— *before the expiration of the time limit for amending the claims and to be republished in the event of receipt of amendments (Rule 48.2(h))*

(54) **Title:** ANTIBODY EVOLUTION IMMUNOGENS

(57) **Abstract:** The present invention relates, in general, to HIV-1 and, in particular, to broadly neutralizing HIV-1 antibodies, and to HIV-1 immunogens and to methods of using such immunogens to induce the production of broadly neutralizing HIV-1 antibodies in a subject (e.g., a human).

WO 2014/042669 A1

HIV-1 ANTIBODY EVOLUTION IMMUNOGENS

TECHNICAL FIELD

The present invention relates, in general, to HIV-1 and, in particular, to
5 broadly neutralizing HIV-1 antibodies, and to HIV-1 immunogens and to
methods of using such immunogens to induce the production of broadly
neutralizing HIV-1 antibodies in a subject (e.g., a human).

BACKGROUND

Induction of HIV-1 envelope (Env) broadly neutralizing antibodies
10 (BnAbs) is a key goal of HIV-1 vaccine development. BnAbs can target
conserved regions that include conformational glycans, the gp41 membrane
proximal region, the V1/V2 region, glycans-associated C3/V3 on gp120, and
the CD4 binding site (CD4bs) (Walker et al, Science 326:285-289 (2009),
Walker et al, Nature 477:466-470 (2011), Burton et al, Science 337:183-186
15 (2012), Kwong and Mascola, Immunity 37:412-425 (2012), Wu et al, Science
329:856-861 (2010), Wu et al, Science 333:1593-1602 (2011), Zhou et al,
Science 329:811-817 (2010), Sattentau and McMichael, F1000 Biol. Rep. 2:60
(2010), Stamatatos, Curr. Opin. Immunol. 24:316-323 (2012)). Most mature
BnAbs have one or more unusual features (long heavy chain third
20 complementarity determining regions

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[HCDR3s], polyreactivity for non-HIV-1 antigens, and high levels of somatic mutation) suggesting substantial barriers to their elicitation (Kwong and Mascola, *Immunity* 37:412-425 (2012), Haynes et al, *Science* 308:1906-1908 (2005), Haynes et al, *Nat. Biotechnol.* 30:423-433 (2012), Mouquet and Nussenzweig, *Cell Mol. Life Sci.* 69:1435-1445 (2012), Scheid et al, *Nature* 458:636-640 (2009)). In particular, CD4bs BnAbs have extremely high levels of somatic mutation suggesting complex or prolonged maturation pathways (Kwong and Mascola, *Immunity* 37:412-425 (2012), Wu et al, *Science* 329:856-861 (2010), Wu et al, *Science* 333:1593-1602 (2011), Zhou et al, *Science* 329:811-817 (2010)). Moreover, it has been difficult to find Envs that bind with high affinity to BnAb germline or unmutated common ancestors (UCAs), a trait that would be desirable for candidate immunogens for induction of BnAbs (Zhou et al, *Science* 329:811-817 (2010), Chen et al, *AIDS Res. Human Retrovirol.* 23:11 (2008), Dimitroff, *MAbs* 2:347-356 (2010), Ma et al, *PLoS Pathog.* 7:e1002200 (2001), Pancera et al, *J. Virol.* 84:8098-8110 (2010), Xiao et al, *Biochem. Biophys. Res. Commun.* 390:404-409 (2009)). Whereas it has been found that Envs bind to UCAs of BnAbs targeting gp41 membrane proximal region (Ma et al, *PLoS Pathog.* 7:e1002200 (2001), Alam et al, *J. Virol.* 85:11725-11731 (2011)), and to UCAs of some V1/V2 BnAb (Bonsignori et al, *J. Virol.* 85:9998-10009 (2011)), to date, heterologous Envs have not been identified that bind the UCAs of CD4bs BnAb lineages (Zhou et al, *Science* 329:811-817 (2010), Xiao et al, *Biochem. Biophys. Res. Commun.* 390:404-409 (2009), Mouquet et al, *Nature* 467:591-595 (2010), Scheid et al, *Science* 333:1633-1637 (2011), Hoot et al, *PLoS Pathog.* 9:e1003106 (2013)), although Envs that bind CD4bs BnAb UCAs should exist (Hoot et al, *PLoS Pathog.* 9:e1003106 (2013)).

Eighty percent of heterosexual HIV-1 infections are established by one transmitted/founder (T/F) virus (Keele et al, *Proc. Natl. Acad. Sci. USA* 105:7552-7557 (2008)). The initial neutralizing antibody response to this virus

arises approximately 3 months after transmission and is strain-specific (Richman et al, Proc. Natl. Acad. Sci. USA 100:4144-4149 (2003), Corti et al, PLoS One 5:e8805 (2010)). This antibody response to the T/F virus drives viral escape, such that virus mutants become resistant to neutralization by autologous plasma

5 (Richman et al, Proc. Natl. Acad. Sci. USA 100:4144-4149 (2003), Corti et al, PLoS One 5:e8805 (2010)). This antibody-virus race leads to poor or restricted specificities of neutralizing antibodies in ~80% of patients; however in ~20% of patients, evolved variants of the T/F virus induce antibodies with considerable neutralization breadth, e.g. BnAbs (Walker et al, Nature 477:466-470 (2011),

10 Bonsignori et al, J. Virol. 85:9998-10009 (2011), Corti et al, PLoS One 5:e8805 (2010), Gray et al, J. Virol. 85:4828-4840 (2011), Klein et al, J. Exp. Med. 209:1469-1479 (2012), Lynch et al, J. Virol. 86:7588-7595 (2012), Moore et al, Curr. Opin. HIV AIDS 4:358-363 (2009), Moore et al, J. Virol. 85:3128-3141 (2011), Tomaras et al, J. Virol. 85:11502-11519 (2011)).

15 There are a number of potential molecular routes by which antibodies to HIV-1 may evolve and, indeed, types of antibodies with different neutralizing specificities may follow different routes (Wu et al, Science 333:1593-1602 (2011), Haynes et al, Nat. Biotechnol. 30:423-433 (2012), Dimitrov, MAbs 2:347-356 (2010), Liao et al, J. Exp. Med. 208:2237-2249 (2011)). Because the initial

20 autologous neutralizing antibody response is specific for the T/F virus (Moore et al, Curr. Opin. HIV AIDS 4:358-363 (2009)), some T/F Envs might be predisposed to binding the germline or unmutated common ancestor (UCA) of the observed BnAb in those rare patients that make BnAbs. Thus, although neutralizing breadth generally is not observed until chronic infection, a precise

25 understanding of the interplay between virus evolution and maturing BnAb lineages in early infection may provide insight into events that ultimately lead to BnAb development. BnAbs studied to date have only been isolated from individuals who were sampled during chronic infection (Walker et al, Science

326:285-289 (2009), Burton et al, Science 337:183-186 (2012), Kwong and Mascola, Immunity 37:412-425 (2012), Wu et al, Science 329:856-861 (2010), Wu et al, Science 333:1593-1602 (2011), Zhou et al, Science 329:811-817 (2010), Bonsignori et al, J. Virol. 85:9998-10009 (2011), Corti et al, PLoS One 5:e8805
5 (2010), Klein et al, J. Exp. Med. 209:1469-1479 (2012)). Thus, the evolutionary trajectories of virus and antibody from the time of virus transmission through the development of broad neutralization remain unknown.

Vaccine strategies have been proposed that begin by targeting unmutated common ancestors (UCAs), the putative naïve B cell receptors of BnAbs, with
10 relevant Env immunogens to trigger antibody lineages with potential ultimately to develop breadth (Wu et al, Science 333:1593-1602 (2011), Haynes et al, Nat. Biotechnol. 30:423-433 (2012), Scheid et al, Nature 458:636-640 (2009), Chen et al, AIDS Res. Human Retrovirol. 23:11 (2008), Dimitroff, MAbs 2:347-356 (2010), Ma et al, PLoS Pathog. 7:e1002200 (2011), Xiao et al, Biochem. Biophys. Res. Commun. 390:404-409 (2009), Alam et al, J. Virol. 85:11725-11731 (2011),
15 Mouquet et al, Nature 467:591-595 (2010)). This would be followed by vaccination with Envs specifically selected to stimulate somatic mutation pathways that give rise to BnAbs. Both aspects of this strategy have proved challenging due to lack of knowledge of specific Envs capable of interacting with
20 UCAs and early intermediate (I) antibodies of BnAbs.

The present invention results, at least in part, from studies that resulted in the isolation of the CH103 CD4bs BnAb clonal lineage from an African patient, CH505, who was followed from acute HIV-1 infection through BnAb development. The studies show that the CH103 BnAb lineage is less mutated
25 than most other CD4 binding site BnAbs, and may be first detectable by as early as 14 weeks after HIV-1 infection. Early autologous neutralization by antibodies in this lineage triggered virus escape, but rapid and extensive Env evolution in and near the epitope region preceded the acquisition of plasma antibody

neutralization breadth defined as neutralization of heterologous viruses. Analysis of the cocrystal structure of the CH103 Fab and a gp120-core demonstrated a novel loop binding mode of antibody neutralization.

SUMMARY OF THE INVENTION

5 In general, the present invention relates to HIV-1 and to broadly neutralizing HIV-1 antibodies. More specifically, the invention relates to HIV-1 immunogens and compositions comprising same. The invention further relates to methods of inducing the production of broadly neutralizing HIV-1 antibodies in a subject (e.g., a human) and to compounds and compositions suitable for use in
10 such methods.

Objects and advantages of the present invention will be clear from the description that follows..

BRIEF DESCRIPTION OF THE DRAWINGS

Figures 1A-1D. Development of neutralization breadth in donor CH505
15 and isolation of antibody. Fig. 1A, Shown are HIV-1 viral RNA copies and reactivity of longitudinal plasmas samples with HIV1-1 YU2 core gp120, RSC3 and negative control Δ RSC3 proteins. Fig. 1B, PBMCs from week 136 was used for sorting CD19⁺, CD20⁺, IgG⁺, RSC3⁺ and RSC3 Δ 371I⁻ memory B cells (0.198%). Cells indicated as orange, blue and green dots yielded mAbs CH103,
20 CH104 and CH106, as identified by index sorting. Fig. 1C, Shown are HIV-1 neutralization potency and breadth of CH103 antibody. A neighbor joining phylogenetic tree created by neighbor joining method (NJ tree PHYLIP package software) of 196 HIV-1 Envs representing major circulating clades is colored according to IC50 of neutralized virus by CH103. Fig. 1D, Cross competition of

CH103 binding to YU2 gp120 by the indicated HIV-1 antibodies, and soluble CD4-Ig was determined in ELISA.

Figures 2A-2D. CH103-clonal family with time of appearance, V_HDJ_H mutations, and HIV-1 Env reactivity. Phylogenies of V_HDJ_H (Fig. 2A) and V_LJ_L (Fig. 2B) sequences from sorted single memory B cells and pyrosequencing. Figure was produced using DNA sequences and the EBI bioinformatics server with ancestral reconstructions performed using dnaml maximum likelihood. Neighbor joining was used to illustrate the correspondence of sampling date and read abundance in the context of the clonal history. Within time-point V_H monophyletic clades are collapsed to single branches; variant frequencies are indicated on the right. Isolated mature antibodies are red, pyrosequencing-derived sequences are black. The inferred evolutionary paths to observed matured antibodies are bold. Fig. 2C, CH103 lineage with the inferred intermediates (circles, I1-4, I7 and I8), and percentage mutated V_H sites and timing (blue), indicated. Fig. 2D, Binding affinity (Kd, nM) of antibodies to autologous CH505 (left box) and heterologous B.63521 were measured by SPR (right box).

Figures 3A-3D. Structure of antibody CH103 in complex with the outer domain of HIV-1 gp120 (OD). Fig. 3A, Overall structure of complex with gp120 polypeptide depicted in red ribbon and CH103 shown as a molecular surface (heavy chain in green and light chain in blue). Fig. 3B, Superposition of OD bound by CH103 (red) and core gp120 bound by CH103 (gray) with polypeptide shown in ribbon representation. Fig. 3C, CH103 epitope (green) on OD (red) with the initial CD4-binding site superposed (yellow boundaries) in surface representation. Fig. 3D, Sequence alignment of outer domains of the crystallized

gp120 shown on the first line and diverse HIV-1 Envs recognized by CH103 (SEQ ID NOS 23-28, respectively, in order of appearance). Secondary structure elements are labeled above the alignment with gray dashed lines indicating disordered regions. Symbols in yellow or green denote gp120 OD contacts for CD4 and CH103, respectively, with open circles representing main-chain contacts, open circles with rays representing side-chain contact, and filled circles representing both main-chain and side-chain contacts.

Figures 4A-4D. CH103 paratope, critical residues, and required immune precursors. Fig. 4A, Overall structure of complex with variable domains of CH103 depicted in ribbon representation and gp120 shown as a molecular surface. The color scheme is the same as in Fig. 3A. Fig. 4B, CH103 paratope surface displayed on top of an underlying polypeptide ribbon. The surface is colored and labeled by contributing antibody components. Fig. 4C, CH103 paratope surface colored by maturation states of the underlying residues. Unmutated residues are colored magenta while affinity matured residues are colored green and light blue for heavy and light chains respectively. Fig. 4D, Sequence alignment of heavy (SEQ ID NOS 29-42, respectively, in order of appearance) and light (SEQ ID NOS 43-47, respectively, in order of appearance) chains of CH103 clonal lineage members. Framework and CDR residues are labeled, as are residues that interact with the gp120 (open circle, main chain interaction; open circle with rays, side chain interactions; filled circle, both main chain and side chain interactions). The unmutated paratope residues are highlighted in magenta and the maturation-gained paratope residues are highlighted in green for heavy chain and blue for light chain.

Figure 5. Sequence Logo displaying variation in key regions of Ch505 Envs. The frequency of each amino acid variant per site is indicated by its height, deletions are indicated by grey bars. The first recurring mutation, N279K, appears

at week 4 (open arrow). The timing of BnAb activity development (from Fig. 8 and Table 1) is on the left. Viral diversification, which precedes acquisition of breadth, is highlighted by vertical arrows to the right of each region. CD4 and CH103 contact residues, and amino acid position numbers based on HIV-1 HXB2, are shown along the base of each Logo column.

Figures 6A and 6B. Development of neutralization breadth in the CH103-clonal lineage. Fig. 6A, Phylogenetic CH103 clonal lineage tree showing the IC50 ($\mu\text{g/ml}$) of neutralization of either the autologous T/F (C.CH505), heterologous tier clades A (A.Q842) and B (B.BG1168) viruses as indicated. Fig. 6B, Interplay between evolving virus and developing clonal lineage mapped on to models of CH103-developmental variants and contemporaneous virus. The outer domain of HIV gp120 is depicted in worm representation, with worm thickness and color (white to red) mapping the degree of per-site sequence diversity at each time point. Models of antibody intermediates are shown in cartoon diagram with somatic mutations at each time point highlighted in spheres and colored red for mutations carried over from I8 to mature antibody, cyan for mutations carried over from I4 to mature antibody, green for mutations carried over from I3 to mature antibody, blue for mutations carried over from I2 to mature antibody, orange for mutations carried over from I1 to mature antibody, magenta for CH103 mutations from I1. Transient mutations that did not carry all the way to mature antibody are colored in deep olive. The antibody (paratope) residues are shown in surface representation and colored by their chemical types as in Fig. 5.

Figures 7A and 7B. Hamming distance frequency distributions of sequences at (Fig. 7A) week 4 and (Fig. 7B) week 14. A model of the best fit

Poisson distribution is shown as a red line. Analysis of the sequence diversity in the first available sample (Fig. 7A) from subject CH505 using the Poisson Fitter tool (ref below) indicates that the sequences were a consistent with a star phylogeny and that the mutations were accumulating according to a Poisson distribution (goodness of fit $p = 0.11$). This is consistent with a single founder virus establishing the infection, with random accumulation of mutations prior to selection. The lambda parameter was 1.325, and assuming the mutation rate of 2.16×10^{-5} , the estimated time from the most recent common ancestor was 22 days (95% CI, 18-27). Given that the outer bound of this confidence interval is 27 days, it is highly like this sample was taken within 4 weeks of infection, thus this sampling time is called “week 4” as a conservative estimate. This timing estimate is further supported by Feibig staging at time of enrollment. By week 14 (Fig. 7B), the tree was no longer consistent with a star phylogeny or a Poisson distribution ($p \ll 10^{-10}$), indicating selection was well underway. Of note, although the mutation data at week 4 (Fig. 7A) is statistically consistent with a Poisson distribution, the observed number of pairwise sequence identities was somewhat reduced relative to expectation, and the observed number of Hamming distances of 1 and 2 are slightly more than expected. This is of interest as this shift is the a result of a single mutation in loop D, in a CH103 contact residue (N279K) -- so although the deviation from the Poisson was not significant, given its location it is possible that the site is a very early indicator of selection. (Giorgi et al, BMC Bioinformatics Oct 25;11:532 (2010), PMID: 20973976

Figure 8. Binding of plasma antibodies of CH505 patient over time to autologous transmitted/founder (T/F) and heterologous HIV-1 Env proteins. Plasma samples were longitudinally collected from HIV-1 patient CH505 starting from time of infection(in x axis) and tested for neutralization activity against the

autologous transmitted/founder (T/F) virus and heterologous HIV-1 Env pseudoviruses including subtype B (B) SF162, JRFL and BG1168) and subtype A in TZM-bl cell-based neutralization assays. Results were expressed as IC50 (reciprocal plasma dilution) (in y axis).

5 Figures 9A and 9B. Reactivity of antibodies in CH103 clonal lineage to HIV-1 Env resurfaced core3 (RSC3) and RSC3 mutant. Antibodies in CH103 clonal lineage were tested in dose range from 100 μ g to 0.0005 μ g/ml for binding to (Fig. 9A) HIV-1 Env RSC) and (Fig. 9B) RSC3 with P363N and D371I mutations in ELISA. Results are expressed as EC50 (μ g/ml) and are indicated
10 next to the individual antibodies. NB = no detectable binding.

 Figures 10A and 10B. SDS-PAGE analysis of recombinant HIV-1 Env gp140 and gp120 proteins*. HIV-1 Env gp120 and gp140 proteins were analyzed on SDS-PAGE under reducing condition (Fig. 10A) and gp140 proteins were analyzed on blue native PAGE for (Fig. 10B). Individual HIV-1 Env proteins
15 are identified on the top of gels. Fig. 10A, The HIV-1 gp120 and gp140 used in the study had no degradation under reducing condition in SDS-PAGE. Fig. 10B, Most heterologous HIV-1 Env gp140 Envs and all autologous CH505 gp140 Envs migrated predominantly as trimers and also contain dimer and monomer forms.

 Figures 11A-11D. Polyreactivity analysis of CH103 clonal lineage
20 antibodies by HEp-2 staining, ANA assays and protein array microchip analysis. Reactivity of antibodies in CH103 clonal lineage was assayed by indirect immunofluorescence staining (Fig. 11A) and by ANA assays (Fig. 11B). Pictures at magnification x 200 of immunofluorescence staining for individual antibodies are presented next to the antibody ID. Results of the reactivity of individual

antibodies with panel of autoantigens assayed by ANA is indicated (Fig. 11B). The intermediate antibody (I1) and CH106 were identified as reactive with HEp-2 cells and then selected for further testing for reactivity with human host cellular antigens (Figs. 11C and 11D) using Invitrogen ProtoArrays™. It was found that I1 (Fig. 11C) and CH106 ((Fig. 11D) exhibit specific autoreactivity and robust polyreactivity. Bound antibody was determined by immunofluorescence and relative fluorescence intensities for 9,400 recombinant human proteins in the 151K array (y-axis) is plotted against (x-axis) the homologous intensities in IA1 (Fig. 11C) and CH106 (Fig. 11D) arrays. All proteins are printed in duplicate on each array and each data point represents one fluorescence measurement. The diagonals in each graph represent equal fluorescence intensities (equivalent binding) by the I1, CH106 and 151K control Ab. Self-antigens bound by the I1 and CH106 are identified by high fluorescence intensity versus 151K and are indicated by circles. Polyreactivity is indicated by significant and general skewing from the diagonal. Autoantigens identified: BHMT2 (betaine-homocysteine methyltransferase 2); CENP-R (centromere protein R) [151K]; eEF-2K (eukaryotic elongation factor-2 kinase); UBE3A (ubiquitin-protein ligase E3A) [IA1 and CH106]; TGM2 (transglutaminase 2) [CH106]; NFKB1A (nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha); FAM184A (family with sequence similarity 184, member A) [I1].

Figures 12A and 12B. Crystal packing of the CH103-gp120 complex in P21 space group. Fig. 12A, A view of the crystal lattice. The two complexes in each asymmetric unit are marked with red and blue dashed lines and are shown in cartoon diagrams with gp120 in red and salmon, CH103 heavy chain in green and palegreen, and CH103 light chain in light blue and cyan. Fig. 12B, A close-up view of the lattice between two neighboring complexes. When extended core gp120 of clade C ZM176.66 from the VRC01 complex is superposed with its

ordered corresponding portions in the CH103 complex, the inner domain shown in magenta clashes with the neighboring complex, indicating inner domain of gp120 is not present in the CH103-gp120 crystal due to proteolytic degradation during crystal growth.

5 Figure 13. Pixel map and phylogenetic tree of HIV evolution over time in CH505. The pixel tool was used to illustrate the amino changes in the V1 to V5 region of the envelope; focus was on this region as it most critical CD4bs antibody susceptibility, and includes of all known CD4 binding contacts, which are indicated as black tic marks along the top of the figure. Blue tic marks
10 indicated CH103 contact residues, and the horizontal blue line indicates that part of gp120 that was used for the CH103 crystal structure (although the contact surface is mostly there, still quite a bit is missing that is important for CD4 and VRC01, which is why we use CD4 contacts to help define bits that may be important for CH103 binding in those missing regions). Each row is a
15 sequences, and they are ordered according to the phylogeny. Red bits indicate amino acid change relative to the TF virus, and black bits indicate either an insertion or deletion. The phylogenetic tree on the right was made with PhyML v2 [1] and the JTT substitution model [2] from the translated Env sequences. The tree was configured as a ladder and the T/F virus was reconstructed from
20 the first time point sequences obtained at week 4 after transmission. Colors indicated the estimated number of weeks from infection. The tree was rendered with APE v3.0-6 [3] and both used R v2.15.1 [4]. The arrow indicates the week 30-53 selective bottleneck. (Guindon et al, Syst. Biol. 52:696–704 (2003), Jones et al, Comput Applic Biosci 8: 275–282 (1992), Paradis et al,
25 Bioinformatics 20: 289–290 (2004), R Core Team. 2012. R: A language and environment for statistical computing. R Foundation for Statistical

Computing, Vienna, Austria. ISBN 3-900051-07-0)

Figure 14. Entropy map illustrating the per site diversity within each time point sampled in CH505. Full gp160 is shown, and CD4 and CH103 contact residues are highlighted. This figure shows the Shannon entropy of each position in the alignment, where the observed frequency of all in a position characters is considered, and a gap is treated as a character (Korber J Virol. 1994;68(11):7467-81). This provides a map of regional within-time point diversity spanning Env, and illustrates where mutations are concentrated and the relative diversity of key regions over time.

Figures 15A and 15B. A comparison of the speed of viral sequence evolution in CH505 in regions relevant to the CH103 epitope to other subjects. Fig. 15A, The distribution of sequence distances expressed as the percentage of amino acids that are different between two sequences, resulting from a pairwise comparison of all sequences sampled in a given time point. These are all homogeneous infection cases, so in acute infection there is very little mutation in the CH103 relevant regions, or elsewhere in the virus (left hand panels). By 24 weeks after enrollment (week 30 from infection in CH505, labeled month 6 here as it is approximate), extensive mutations have begun to accrue, focused in CH103 relevant regions (top middle panel), but not in other regions of Env (bottom middle panel). CH103 has the highest ranked diversity among 15 subject sampled in this time frame ($p=0.067$), indicating a focused selective pressure began unusually early in this subject. By 1 year (month 12 indicates samples taken between 10-14 months from enrollment), this region has begun to evolve in many individuals, possibly due to autologous NAb responses that come up later in

infection. Fig. 15B, Phylogenetic trees based on CH103 relevant regions. In this view, the extensive evolution away from the T/F virus by month 6, shown in gold, is particularly striking. The distance between sequences sampled in CH505 at month 6 and the T/F ancestral state were much greater than the sequences in the second most variable individual 704010042 (Wilcoxon rank sum, $p = 0.0003$: CH505, median = 0.064, range = 0.019 – 0.13, $N = 25$, and 704010042, median = 0.027, range = 0.009 – 0.056, $N = 26$).

Figure 16. Co-evolution of virus and antibody - interplay between maturation of antibody CH103 and sequence variability epitope in gp120. The sequence variability (within sample) at each time point is mapped on a gp120 structure that tracks the viral evolution over time from 14 weeks thru 100 weeks post-transmission. Entropy at each residue is color-coded as green to white to red to indicate no sequence variation to slight variation to high sequence variation. This extensive virus within-time point diversity coincides with maturing antibody lineages that ultimately develop breadth. Here, the somatic mutations are captured along the CH103 clonal lineage beginning with unmutated common ancestor (UCA) to I-8 to I-4 to I-3 to I-2 to I-1 and to mature CH103. The color balls in heavy (violet) and light (cyan) chains of antibody indicate the appearance/disappearance of somatic mutations during the evolutionary path according to the following scheme. Red balls: mutations appeared in I-8 and remained all the way thru maturation to CH103, Orange balls: mutations appeared in I-4 and remained thru maturation to CH103, Blue balls: mutations that appeared early but are lost before maturing to CH103, and Gray balls: mutations that appeared very late in maturation. Structure of Fab CH103-gp120 from ZM176.66 complex determined in this work is used to map these mutations. The sequence entropy at week 100 is used to pair with CH103 to simply illustrate the relative spatial locations of somatic mutations and sequence variability in gp120.

As discussed in the text, viral evolution with time tracks with neutralization breadth and this simple mapping supports that (i) T/F virus began to diversify very early in regions in or proximal to the epitope, and (ii) Somatic mutations that occur early in evolution and remain fixed in heavy chain tend to cluster
 5 near the gp120 contact region unlike those mutations that appear later.

Figures 17A and 17B. Amino acid (Fig. 17A) (SEQ ID NOS 48-445, respectively, in order of appearance) and nucleic acid (Fig. 17B) (SEQ ID NOS 446-843, respectively, in order of appearance) sequences. 703010505.TF is the transmitted/founder sequence and “W and number” indicates the week after
 10 transmission

Figure 18. Antibody-virus co-evolution in acutely infected patients followed to BnAb induction.

Figures 19A – 19D. Multivalent vaccine sequences. CH505 Env sequences (Figs 19A and 19B) (Figs 19A and 19B each disclose SEQ ID NOS
 15 844-860, respectively, in order of appearance), and CH505_D8gp120 or CH505 gp120 sequences (Fig. 19C) (SEQ ID NOS 861-878, respectively, in order of appearance) and corresponding cleavage site mutations (Fig. 19D) (SEQ ID NOS 879-896, respectively, in order of appearance) (underlined).

Figure 20. The HIV-1 arms race: isolation of broad neutralizing
 20 antibodies from chronically infected patient CH0505 followed from time of transmission.

Figure 21. The same virus clonal lineage tree of CH0505 shown in Fig. 20 - starred in the right panel are examples of sequential envs chosen for immunogens and starred on the tree on the left are env sequences in Fig. 17.

Figure 22. Contact region for CD4, VRC01 and b12 and the signature sites that impact VRC01 and b12 neutralization are under intense selective pressure in CH0505.

Figure 23. The number of pairwise differences in just the
 5 CD4/b12/VRC01 contact residues is also relatively high for CH0505.

Figure 24. Clonal lineage tree of Clone 103 from CH0505 - binding to CH0505 transmitted/founder Env gp140 (EC50 $\mu\text{g/ml}$).

Figure 25. Clonal lineage tree of Clone CH103 from CH0505 - neutralization of tier 2 CH0505 (EC50, $\mu\text{g/ml}$).

10 Figure 26. HIV-1 vaccine design. Figure 26 discloses SEQ ID NOS 897-898, respectively, in order of appearance.

Figure 27. Viral evolution during BnAb development in the HIV-1 infected individual (CH505). Figure 27 discloses SEQ ID NOS 899-908, respectively, in order of appearance.

15 Figure 28. Alignment of CH505 Env gp120 with RSC3. Figure 28 discloses SEQ ID NOS 909-910, respectively, in order of appearance.

Figure 29. Design for CH505 outer domain immunogen. Figure 29 discloses SEQ ID NO: 911, residues 1-65 of SEQ ID NO: 911, SEQ ID NO: 912, residues 1-65 of SEQ ID NO: 912 and SEQ ID NO: 913, respectively, in
 20 order of appearance.

Figure 30. Plasma binding ratio of RSC3 to RSC3delta371 proteins induced by CH505 Env variants alone or sequentially administered to BALB/c mice.

DETAILED DESCRIPTION OF THE INVENTION

The results of the study described in the Example that follows demonstrate that the binding of a T/F Env to a UCA B cell receptor of a BnAb lineage was responsible for the induction of broad neutralizing antibodies, thus providing a logical starting place for vaccine-induced CD4bs BnAb clonal activation and expansion. Importantly, the number of mutations required to achieve neutralization breadth was reduced in the CH103 lineage compared to most CD4bs BnAbs, although the CH103 lineage had reduced neutralization breadth compared to more mutated CD4bs BnAbs. By tracking viral evolution through early infection, it was found that intense selection and epitope diversification in the T/F virus preceded the acquisition of NAb breadth in this individual -- thus demonstrating the viral variants or combination of variants associated with development of BnAbs directly from autologous neutralizing antibodies and illuminating a pathway for induction of similar B cell lineages. (See viral envelope sequences (and encoding sequences) in Figs. 17A, B and 19A-D.) The envelopes to be used as immunogens can be expressed as full gp140, gp145 with transmembrane portions, gp120s, gp120 resurfaced core proteins, gp120 outer domain constructs, or other minimal gp120 constructs with portions of the CH103 contacts such as the gp120 D loop, the V5 loop and the CD4 binding site loop region expressed such that the UCA, and/or Intermediate antibodies and/or mature CH103, CH104, CH105, and CH106 mature antibodies bind to the immunogen constructs.

In accordance with the invention, immunization regimens can include sequential immunizations of Env constructs selected from Figs 17 and 19, or can involve prime and boosts of combinations of Envs, or the administration of "swarms" of such sequences (e.g., those in Fig. 19A-D). Immunogenic fragments/subunits can also be used as can encoding nucleic acid sequences.

Alternatively, the transmitted founder virus Env constructs can be used as primes, followed by a boost with the transmitted founder Env and sequential additions of Envs from progressively later times after transmission in patient CH505. Further, repetitive immunization can be effected with “swarms” of CH505 Envs (for
5 example, including various combinations of the proteins and nucleic acid sequences in Figs. 19A-D) ranging from, for example, 2 to 40 Envs. Examples of vaccine strategies of the invention are shown in Fig. 18.

The data provided in the Examples below have implications for understanding the B cell maturation pathways of the CH103 lineage and for
10 replicating similar pathways in a vaccine setting. First, it was demonstrated in CH505 that BnAbs were driven by sequential Env evolution beginning as early as 14 weeks after transmission, a time period compatible with induction of this type of BnAb lineage with a vaccine given the correct set of immunogens. Second, whereas heterologous Envs did not bind with UCAs or early intermediate
15 antibodies of this lineage, the CH505 T/F Env bound remarkably well to the CH103 UCA, and subsequent Envs bound with increased affinity to later clonal lineage members. Thus, immunizations with similar sequences of Env or Env subunits can be expected drive similar lineages. Third, the CH103 lineage is less complicated than those of the VRC01-class of antibodies because antibodies in
20 this lineage have fewer somatic mutations, and no indels, except CH103 V_L has a deletion of 3 amino acid residues in the LCDR1 region. The study described in the Example 1 below was in one patient. Nonetheless, in each BnAb patient, analysis of viral evolution should elucidate a similar pathway of evolved Envs that induce BnAb breadth. The observation that rhesus macaques infected with the
25 CCR5-tropic SHIV-AD8 virus frequently develop neutralization breadth (Shingai et al, Proc. Natl. Acad. Sci. USA 109:19769-19774 (2012)) indicates that certain envelopes may be more likely to induce breadth and potency than others.

Polyreactivity to host molecules in the CH103-lineage arose during affinity maturation in the periphery coincident with BnAb activity. This finding is compatible with the hypothesis that BnAbs may be derived from an inherently polyreactive pool of B cells, with polyreactivity providing a neutralization
5 advantage via heterologation of Env and host molecules (Mouquet et al, Nature 467:591-595 (2010), Alam et al, J. Immunol. 178:4424-4435 (2007)). Alternatively, as CH103 affinity maturation involves adapting to the simultaneous presence of diverse co-circulating forms of the epitope (Malherbe et al, J. Virol.. 85:5262-5274 (2011)), the selection of antibodies that can interact with extensive
10 escape-generated epitope diversification may be an evolutionary force that also drives incidental acquisition of polyreactivity.

Thus, in one embodiment, the present invention relates to a method of activating an appropriate naïve B cell response in a subject (e.g., a human) by administering the CH505 T/F Env or Env subunits that can include the gp145
15 with a transmembrane portion, gp41 and gp120, an uncleaved gp140, a cleaved gp140, a gp120, a gp120 subunit such as a resurfaced core (Wu X, Science 329:856-61 (2010)), an outerdomain, or a minimum epitope expressing only the contact points of CH103 with Env, i.e., the gp120 D loop, the V5 loop and the CD4 binding site loop region (the minimal epitope to avoid dominant Env non-
20 neutralizing epitopes), followed by boosting with representatives of the subsequently evolved CH505 Env variants (e.g., those in Figs. 17 and 19) either given in combination to mimic the high diversity observed *in vivo* during affinity maturation, or in series, using vaccine immunogens specifically selected to trigger the appropriate maturation pathway by high affinity binding to UCA and antibody
25 intermediates (Haynes et al, Nat. Biotechnol. 30:423-433 (2012)). DNA, RNA, protein or vectored immunogens can be used alone or in combination. In one embodiment of the invention, transmitted founder virus envelope (e.g., B.6240 (see also Fig. 17)) is administered to the subject (e.g., human) as the priming

envelope and then one or more of the sequential envelopes disclosed herein is administered as a boost in an amount and under conditions such that BnAbs are produced in the subject (e.g., human). By way of example, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17 or 18 envelopes (or subunits thereof) (e.g., from Fig. 19) can be used with one prime and multiple boosts.

The data provided in the Examples demonstrate the importance of studying subjects followed from the transmission event through the development of plasma BnAb activity for concomitant isolation of both T/F viruses and their evolved quasispecies along with the clonal lineage of induced BnAbs. The finding that the T/F Env can be the stimulator of a potent BnAb and bind optimally to that BnAb UCA is a critical insight for vaccine design, and makes possible the induction of BnAbs by targeting UCAs and IAs of BnAb clonal lineage trees (Haynes et al, Nat. Biotechnol. 30:423-433 (2012)).

The present invention includes the specific envelope proteins disclosed herein (e.g., those in Fig. 17A and Fig. 19A-D) and nucleic acids comprising nucleotide sequences encoding same (e.g., those in Fig. 17B). Preferred sequences (amino acid and nucleic acid) include those designated 703010505.TF, 703010505.w53.16, 703010505.w78.33 and 703010505.w100.B6. The envelope proteins (and subunits) can be expressed, for example, in 293T cells, 293F cells or CHO cells (Liao et al, Virology 353:268-82 (2006)). As indicated above, the envelope proteins can be expressed, for example, as gp120 or gp140 proteins and portions of the envelope proteins can be used as immunogens such as the resurfaced core protein design (RSC) (Fig. 28) (Wu et al, Science 329:856-861 (2010)); another possible design is an outer domain design (Fig. 29) (Lynch et al, J. Virol. 86:7588-95 (2012)). The invention includes immunogenic fragments/subunits of the envelope sequences disclosed herein, including fragments at least 6, 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, 120, 140, 160, 180, 200, 220, 240, 260, 280 300, 320 or more amino acids in length, as well as nucleic

acids comprising nucleotide sequences encoding such fragments and vectors containing same.

In other embodiments, the invention provides variants of the sequences in Fig. 17, wherein the variants comprise a mutation which repairs a trypsin cleavage site, thereby preventing protein clipping during Env protein production in a cell line, e.g., a CHO cell line. Non-limiting examples of such trypsin resistant variants are shown in Figure 19D (the portion of the document called constructs cleavage site mutations). In one embodiment, amino acid "A" at position 289 in CH0505TF 7gp120 is changed to "T", and amino acid "Q" at position 295 is changed to "D." The invention contemplates trypsin resistant Env variants that include changes at the corresponding positions in any of the Env sequences in Fig. 17.

The envelopes (immunogens) can be formulated with appropriate carriers using standard techniques to yield compositions suitable for administration. The compositions can include an adjuvant, such as, for example, alum, poly IC, MF-59 or other squalene-based adjuvant, AS01B or other liposomal based adjuvant suitable for protein immunization.

As indicated above, nucleic acid sequences (e.g., DNA sequences) encoding the immunogens can also be administered to a subject (e.g., a human) under conditions such that the immunogen is expressed *in vivo* and BnAbs are produced. The DNA can be present as an insert in a vector, such as a rAdenoviral (Barouch, et al. Nature Med. 16: 319-23 (2010), recombinant mycobacterial (i.e., BCG or *M smegmatis*) (Yu et al. Clinical Vaccine Immunol. 14: 886-093 (2007); *ibid* 13: 1204-11 (2006), or recombinant vaccinia type of vector (Santra S. Nature Med. 16: 324-8 (2010)).

Immunogens of the invention, and nucleic acids (e.g., DNAs) encoding same, are suitable for use in generating an immune response (e.g., BnAbs) in a patient (e.g., a human patient) to HIV-1. The mode of administration of the

immunogen, or encoding sequence, can vary with the particular immunogen, the patient and the effect sought, similarly, the dose administered. Typically, the administration route is intramuscular or subcutaneous injection (intravenous and intraperitoneal can also be used). Additionally, the formulations can be
 5 administered via the intranasal route, or intrarectally or vaginally as a suppository-like vehicle. Optimum dosing regimens can be readily determined by one skilled in the art. The immunogens (and nucleic acids encoding same) are preferred for use prophylactically, however, their administration to infected individuals may reduce viral load.

10 Previous attempts to use sequential immunizations with Env proteins that have developed over time in humans or animals that have developed neutralization breadth have failed, primarily because the viruses have been isolated but the envelope immunogens have not been matched to bind to the BnAbs themselves, i.e., they are not antigenic. That is, in the two studies that
 15 have isolated Envs over time in BnAb subjects, no transmitted founder viruses or subsequent (sequential) viruses were available and thus the correct Env immunogens to choose were not apparent (Malherbe et al, J Virol. 85:5262-74 (2011); Pisoni, Vaccine 30:5519-26 (2012)). What is different here is that both the BnAbs and the virus Env sequences that drove the induction and
 20 maturation of the BnAbs are known, and, thus, those envelopes can be chosen with mutations in the CD4 binding site or in regions that are important for CD4 binding site BnAb binding, such as V5 loop region (Zhou et al, Science 329:811-17 (2010); Wu et al, Science 333:1593-602 (2011)).

Certain aspects of the invention can be described in greater detail in the
 25 non-limiting Examples that follow. (See also U.S. Application No. 13/314,712, filed December 8, 2011 and PCT/US2012/000442, filed October 3, 2012.)

EXAMPLE 1

Experimental Details

In summary, serial blood samples were collected from a HIV-1 infected
 5 subject CH505 starting 4 weeks after infection up to 236 weeks after infection.
 MAbs CH103, CH104 and CH106 were generated by the isolation,
 amplification and cloning of single RSC3-specific memory B cells as described
 (Scheid et al, J. Immunol. Methods 343:65-67 (2009), Wu et al, Science
 329:856-861 (2010), Wu et al, Science 333:1593-1602 (2011), Zhou et al,
 10 Science 329:811-817 (2010), Scheid et al, Science 333:1633-1637 (2011)).
 V_HDJ_H and V_LJ_L 454 pyrosequencing was performed on samples from 5
 timepoints after transmission (Wu et al, Science 333:1593-1602 (2011)).
 Inference of unmutated ancestor (UCA), identification and production of clone
 members were performed using the methods as described (Liao et al, J. Exp.
 15 Med. 208:2237-2249 (2011)) (Kepler, T.B. submitted, 2012). Additional
 V_HDJ_H and V_LJ_L and V_LJ_L genes were identified by 454 pyrosequencing (Wu et
 al, Science 333:1593-1602 (2011), Liao et al, J. Exp. Med. 208:2237-2249
 (2011), Boyd et al, Sci. Transl. Med. 1:12ra23 (2009)) and select V_HDJ_H and
 V_LJ_L genes were used to produce recombinant antibodies as reported previously
 20 (Liao et al, J. Exp. Med. 208:2237-2249 (2011)). Binding of patient plasma
 antibodies and CH103 clonal lineage antibody members to autologous and
 heterologous HIV-1 Envs was measured by ELISA

25

and surface plasmon resonance (Alam et al, J. Virol. 85:11725-11731 (2011), Liao et al, J. Exp. Med. 208:2237-2249 (2011), Alam et al, J. Immunol. 178:4424-4435 (2007), Alam et al, J. Virol. 82:115-125 (2008)), and neutralizing activity of patient plasma and CH103 antibody clonal lineage members was determined in a

5 TZM-bl-based pseudovirus neutralization assay (Wu et al, Science 329:856-861 (2010), Seaman et al, J. Virol. 84:1439-1452 (2010), Montefiori, Cur. Protoc. Immunol., Chapter 12, Unit 12 11 (2005)). Crystallographic analysis of CH103 bound to HIV-1 outer domain was performed as previously reported (Zhou et al, Science 329:811-817 (2010)). The GenBank accession numbers for 292 CH505

10 Envs are KC247375-KC247667, for 459 V_HDJ_H are 174 V_LJ_L sequences of antibody members in CH103 clonal lineage are KC575845-KC576303 and KC576304-KC576477, respectively. Coordinates and structure factors for unbound CH103 Fab as well as CH103 Fab in complex with ZM176.66 outer domain have been deposited with the Protein Data Bank.

15 The methods used are described in greater detail below.

Study subject. Plasma and peripheral blood mononuclear cells (PBMC) were isolated from serial blood samples that were collected from a HIV-1 infected subject CH505 starting 6 weeks after infection up to 236 weeks after infection (Table 1) and frozen at -80°C and liquid nitrogen tanks, respectively. During this

20 time, no anti-retroviral therapy was administered. All work related to human subjects was in compliance with Institutional Review Board protocols approved by the Duke University Health System Institutional Review Board.

Inference of unmutated common ancestor (UCA) and identification of clone members. The variable regions of heavy- and light chain (V_HDJ_H and V_LJ_L)

25 gene segments were inferred from the natural pairs themselves. The posterior probabilities for these two gene segments are 0.999 and 0.993, respectively. The UA was first inferred from the natural pairs. Additional clonally related variable region sequences were then identified from deep sequencing and the estimate of

the UCA refined iteratively. All variable region sequences inferred were identified to have been rearranged to the same V_HDJ_H and J_H , and to have the correct CDR3 length. For each sequence, a count was made of the number of mismatches between the sequence and the presumed V_HDJ_H gene up to the codon for the second invariant cysteine. Each iteration was based on the CDR3 of the current posterior modal UA. For each candidate sequence, the number of nucleotide mismatches between its CDR3 and the UA CDR3 were computed. The sequence was rejected as a potential clone member if the z-statistic in a test for difference between proportion is greater than two (Zar, Biostatistical Analysis, entice-Hall, Inc., Upper Saddle River, NJ (1974)). Once the set of candidates has been thus filtered by CDR3 distance, the UA was inferred on that larger set of sequences as described (Haynes et al, Nat. Biotechnol. 30:423-433 (2012), Ma et al, PLoS Pathog. 7:e1002200 (2001), Liao et al, J. Exp. Med. 208:2237-2249 (2011)). The paper, Kepler, T.B., Reconstructing a B cell clonal lineage: I. Statistical Inference of Unobserved Ancestors, that describes the methods and their mathematical basis in detail has been deposited to the arXiv preprint collection at Cornell. If the new posterior modal UA differed from the previous one, the process was repeated until convergence was reached. Due to the greatest uncertainty occurring in the CDRH3, from the V_HDJ_H sequences derived from observed antibodies and sequences identified by 454 pyrosequencing, the 7 most likely VH UCA sequences were inferred resulting in 4 unique amino acid sequences that were all produced and assayed for reactivity with the transmitted/founder envelope gp140 (Table 5).

Isolation of V_HDJ_H and V_L genes and expression of V_HDJ_H and V_LJ_L genes as full-length IgG1 recombinant mAbs. The V_HDJ_H and V_LJ_L gene segment pairs of the observed CH103, CH104 and CH106 and the V_HDJ_H gene segment of CH105 were amplified by RT/PCR of flow sorted HIV-1 Env RSC3 (re-surface core3) -specific memory B cells using the methods as described previously

(Scheid et al, J. Immunol. Methods 343:65-67 (2009), Wu et al, Science 329:856-861 (2010), Wu et al, Science 333:1593-1602 (2011), Zhou et al, Science 329:811-817 (2010), Scheid et al, Science 333:1633-1637 (2011). Additional V_HDJ_H and V_LJ_L and V_LJ_L genes were identified by 454 pyrosequencing. Clonally
5 related V_HDJ_H and V_LJ_L sequences derived from either sorted single B cells or 454 pyrosequencing were combined and used to generate neighbor-joining phylogenetic trees (Figs 2A and 2B). Antibodies that were recovered from single memory B cells are noted in the figure in red, and bolded lines show the inferred evolutionary paths from the UCA to mature BnAbs. For clarity, related V_H
10 variants that grouped within monophyletic clades from the same time-point were collapsed to single branches, condensing 457 V_HDJ_H and 174 V_LJ_L variants to 119 and 46 branches, respectively, via the “nw_condense” function from the Newick Utilities package (v. 1.6) (Junier and Zdobnov, Bioinformatics 26:1669-1670 (2010)). The frequencies of V_HDJ_H variants in each B cell sample are
15 shown to the right of the V_HDJ_H tree in Fig. 2A, and were computed from sample sizes of 188,793, 186,626, and 211,901 sequences from weeks 53, 92, and 144, respectively. Two V_HDJ_H genes (IZ95W and 02IV4) were found at 14 weeks after transmission and paired with UCA V_LJ_L for expression as IgG1 mAbs. IZ95W mAb weakly bound the CH505 T/F Env gp140 with end-point titer of 11
20 ug/ml. Among heavy chain sequences in the tree, the mean distance of each to its nearest neighbor was calculated to be 8.1 nt. The cumulative distribution function shows that, while there are pairs that are very close together (nearly 30% of sequences are 1nt from its neighbor), 45% of all sequences differ by 6nt or more from its nearest neighbor. The probability of generating a sequence that
25 differs by 6 or more nucleotides from the starting sequence by PCR and sequencing is very small. The numbers of sequences obtained from a total of 100 million PBMC were within the expected range of 50-500 antigen-specific B cells.

Regarding the number of unique V_HDJ_H and V_LJ_L genes that have been isolated, this issue has been analyzed in a number of ways. First, the calculations have been clarified for the possible number of antigen-specific CD4bs memory B cells that could have been isolated from the samples studied. Five patient CH505
5 time points were studied with pyrosequencing with ~20 million PBMC per time point for a total of 100 million PBMC studied. In chronic HIV, there is a mean of 145 total B cells per μ l of blood, and a mean of 60 memory B cells per μ l of blood (Moir et al, The Journal of Infectious Diseases 197:572-579 (2008)). This high percent of memory B cells of ~40% of the total B cells in chronic HIV
10 infection is due to selective loss of naïve B cells in HIV infection. Thus, in 100 ml (100,000 μ l) of blood, there will be approximately 6 million memory B cells. If 0.1 to 1.0% are antigen specific, that that would be 6,000 to 60,000 antigen-specific B cells sampled, and if, of these, 5% were CD4bs antibodies, then from 300 to 3000 CD4 bs B cells would have been sampled in 100 million PBMC
15 studied. This is completely compatible and within the range of the calculations of the reviewer above (50 CD4 bs B cells per 5 million PBMC), since studied 100 million PBMC, there should, by these calculations, 1000 CD4bs B cells sampled. Either calculation therefore yields estimates that are completely compatible with the 474 V_HDJ_H genes amplified.

20 To further study the plausibility of sequences isolated, the second method of analysis used was as follows. Among heavy chain sequences in the tree, one can compute the distance of each to its nearest neighbor. The mean distance to the nearest neighbor is 8.1 nt. The cumulative distribution function shows that, while there are pairs that are very close together (nearly 30% of sequences are 1nt from
25 its neighbor), 45% of all sequences differ by 6nt or more from its nearest neighbor. The probability of generating a sequence that differs by 6 or more nucleotides from the starting sequence by PCR and sequencing is very small. It is believed that the number of genes represented in the sample is closer to 200 than

to 50, and most likely is larger than 200.

The third analysis performed was to compute the distance of each heavy chain sequences in the tree to its nearest neighbor. The mean distance to the nearest neighbor is 8.1 nt. Agglomerative clustering was used to prune the sequence alignment. At the stage where no pairs of sequences were 3 nucleotides
5 apart or closer, there were 335 of 452 sequences remaining; when no pairs are 6nt apart or closer, there are still 288 sequences remaining. Therefore, with this analysis, it is believed that the number of genes represented in the sample is closer to 300 than to 50, and may be larger. Thus, by the sum of these re-analyses, it is
10 believed that the number of genes in the trees in Fig. 2 are quite plausible.

The isolated Ig V_HDJ_H and V_LJ_L gene pairs, the inferred UCA and intermediate V_HDJ_H and V_LJ_L sequences, and select V_HDJ_H gene sequences identified by pyrosequencing were studied experimentally (Table 2) and used to generate a phylogenetic tree showing percentage of mutated V_H sites and time of
15 appearance after transmission (Figs 2C) and binding affinity (Fig 2D). The isolated four mature antibodies are indicated in red, antibodies derived from 454 pyrosequencing are indicated in black, and inferred-intermediate antibodies (I1-I4, I7, I8) are indicated by circles at ancestral nodes. The deep clades in this tree had modest bootstrap support, and the branching order and UCA inference were
20 somewhat altered when more sequences were added to the phylogenetic analysis (compare the branching order of Fig. 2C and Fig. 2A). The tree depicted in Figs. 2C and 2D was used to derive the ancestral intermediates of the representative lineage early in the study, and marked an important step in the analysis of antibody affinity maturation. The V_HDJ_H and V_LJ_L genes were synthesized
25 (GenScript, NJ) and cloned into pcDNA3.1 plasmid (Invitrogen, Grand Island, NY) for production of purified recombinant IgG1 antibodies as described previously (Liao et al, J. Virol. Methods 158:171-179 (2009), Liao et al, Immunity 38:176-186 (2013)). The V_HDJ_H genes of I1-I4, I7 and I8 as well as the

V_HDJ_H of CH105 were paired with either the V_L gene of the inferred UCA or I2 depending on the genetic distance of the V_HDJ_H to either the UCA or mature antibodies for expressing as full-length IgG1 antibodies as described (Liao et al, J. Meth. Virol. 158:171-179 (2009)) (Table 2).

5 *Production of recombinant HIV-1 Proteins.* HIV-1 Env genes including subtype B, 63521, subtype C, 1086, and subtype CRF_01, 427299, as well as subtype C, CH505 autologous transmitted/founder Env were obtained from acutely infected HIV-1 subjects by single genome amplification (Keele et al, Proc. Natl. Acad. Sci. USA 105:7552-7557 (2008)) codon-optimized by
10 employing the codon usage of highly expressed human housekeeping genes (Andre et al, Journal of Virology 72:1497-1503 (1998)), *de novo* synthesized (GeneScript, NJ) as gp140 or gp120 (AE.427299) and cloned into a mammalian expression plasmid pcDNA3.1/hygromycin (Invitrogen, Grand Island, NY). Recombinant Env glycoproteins were produced in 293F cells cultured in serum-
15 free medium and transfected with the HIV-1 gp140- or gp120-expressing pcDNA3.1 plasmids, purified from the supernatants of transfected 293F cells by using *Galanthus nivalis* lectin-agarose (Vector Labs, Burlingame, CA) column chromatography (Ma et al, PLoS Pathog. 7:e1002200 (2011)), Liao et al, Virology 353:268-282 (2006), Liao et al, Immunity 38:176-186 (2013)), and
20 stored at -80°C until use. Select Env made as CH505 T/F Env were further purified by superpose 6 column chromatography to trimer forms, and used in binding assays that showed similar results as with the lectin-purified oligomers.

Enzyme-Linked Immunoassay (ELISA). Binding of patient plasma antibodies and CH103 clonal lineage antibody members to autologous and
25 heterologous HIV-1 Envs was measured by ELISA as described previously (Liao et al, J. Exp. Med. 208:2237-2249 (2011), Liao et al, Immunity 38:176-186 (2013)). Plasma samples in serial 3-fold dilutions starting at 1:30 to 1:521,4470 or purified mAbs in serial 3-fold dilutions starting at 100µg/ml to 0.000µg/ml

diluted in PBS were assayed for binding to autologous and heterologous HIV-1 Envs. Binding of biotin-labeled CH103 at the subsaturating concentration was assayed for cross competition by unlabeled HIV-1 antibodies and soluble CD4 in serial 4-fold dilutions starting at 10µg/ml. The half maximal effective
5 concentration (EC50) of plasma samples and mAbs to HIV-1 Envs were determined and expressed as either the reciprocal dilution of the plasma samples or concentration of mAbs.

Surface plasmon resonance (SPR) affinity and kinetics measurements.

Binding K_d and rate constant (association rate k_a , dissociation rate k_d)
10 measurements of mAbs and all candidate UCAs to the autologous Env C. CH05 gp140 and/or the heterologous Env B.63521 gp120 were carried out on BIAcore 3000 instruments as described previously (Alam et al, J. Virol. 85:11725-11731 (2011), Alam et al, J. Immunol. 178:4424-4435 (2007), Alam et al, J. Virol. 82:115-125 (2008)). Anti-human IgG Fc antibody (Sigma Chemicals) was
15 immobilized on a CM5 sensor chip to about 15000 Response Unit (RU) and each antibody was captured to about 50-200 RU on three individual flow cells for replicate analysis, in addition to having one flow cell captured with the control Synagis (anti-RSV) mAb on the same sensor chip. Double referencing for each mAb-HIV-1 Env binding interactions was used to subtract non-specific binding
20 and signal drift of the Env proteins to the control surface and blank buffer flow respectively. Antibody capture level on the sensor surface was optimized for each mAb to minimize rebinding and any associated avidity effects. C.CH505 Env gp140 protein was injected at concentrations ranging from 2 to 25µg/mL and B.63521 gp120 was injected at 50 – 400 µg/mL for UCA and early intermediates
25 (IA8, IA4), 10-100µg/mL (IA3), and 1-25 µg/mL for the distal and mature mAbs. All curve fitting analysis were performed using global fit of to the 1:1 Langmuir model and are representative of at least three measurements. All data analysis was performed using the BIAevaluation 4.1 analysis software (GE Healthcare).

Neutralization assays. Neutralizing antibody assays in TZM-bl cells were performed as described previously (Montefiori, The Journal of Infectious Diseases 206:431-441 (2012)). Neutralizing activity of plasma samples in 8 serial 3-fold dilutions starting at 1:20 dilution and for recombinant mAbs in 8 serial 3-fold dilutions starting at 50ug/ml were tested against autologous and heterologous HIV-1 Env-pseudotyped viruses in TZM-bl-based neutralization assays using the methods as described (Wu et al, Science 329:856-861 (2010), Seaman et al, J. Virol. 84:1439-1452 (2010), Montefiori, The Journal of Infectious Diseases 206:431-441 (2012)). The data were calculated as a reduction in luminescence units compared with control wells and reported as IC50 in either reciprocal dilution for plasma samples or in µg/ml for mAbs.

Crystallization of antibody CH103 and its gp120 complex. The antigen binding fragment (Fab) of CH103 was generated by LyS-C (Roche) digestion of IgG1 CH103 and purified with protocols described previously (Zhou et al, Science 329:811-817 (2010)). The extended core gp120 of HIV-1 clade C ZM176.66 was used to form complex with Fab CH103. Briefly, deglycosylated ZM176.66 extended core gp120 that was produced using the method as described previously (Zhou et al, Science 329:811-817 (2010)) and Fab CH103 were mixed at a 1:1.2 molar ratio at room temperature and purified by size exclusion chromatography (Hiload 26/60 Superdex S200 prep grade, GE Healthcare) with buffer containing 0.35 M NaCl, 2.5 mM Tris pH 7.0, 0.02% NaN₃. Fractions of the Fab or gp120:CH103 complex were concentrated to ~10 mg/ml, flash frozen with liquid nitrogen before storing at -80°C and used for crystallization screening experiments.

Commercially available screens, Hampton Crystal Screen (Hampton Research), Precipitant Synergy Screen (Emerald BioSystems), Wizard Screen (Emerald BioSystems), PACT Suite and JCSG+ (Qiagen) were used for initial crystallization screening of both Fab CH103 and its gp120 complex. Vapor-

diffusion sitting drops were set up robotically by mixing 0.2 μ l of protein with an equal volume of precipitant solutions (Honeybee 963, DigiLab). The screen plates were stored at 20 °C and imaged at scheduled times with RockImager (Formulatrix.). The Fab CH103 crystals appeared in a condition from the JCSG+ kit containing 170 mM ammonium sulfate, 15% glycerol and 25.5 % PEG 4000. For the gp120:CH103 complex, crystals were obtained after 21 days of incubation in a fungi-contaminated droplet of the PACT suite that contained 200 mM sodium formate, 20% PEG 3350 and 100 mM Bistrispropane, pH 7.5.

X-ray data collection, structure determination and refinement for the gp120:CH103 complex. Diffraction data were collected under cryogenic conditions. Best cryo-protectant conditions were obtained by screening several commonly used cryo-protectants as described previously (Zhou et al, Science 329:811-817 (2010)). X-ray diffraction data were collected at beam-line ID-22 (SER-CAT) at the Advanced Photon Source, Argonne National Laboratory, with 1.0000 Å radiation, processed and reduced with HKL2000 (Otwinowski, Methods in Enzymology 276:307 (1997)). For the Fab CH103 crystal, a data set at 1.65 Å resolution was collected with a cryo-solution containing 20% ethylene glycol, 300 mM ammonium sulfate, 15% glycerol and 25 % PEG 4000 (Table 7). For the gp120:CH103 crystals, a data set at 3.20 Å resolution was collected using a cryo-solution containing 30% glycerol, 200 mM sodium formate, 30% PEG 3350 and 100 mM Bistrispropane, pH 7.5 (Table 7).

The Fab CH103 crystal was in the P2₁ space group with cell dimensions at a=43.0, b=146.4, c=66.3, α =90.0, β =97.7, γ =90.0 and contained two Fab molecules per asymmetric unit (Table 7). The crystal structures of Fab CH103 were solved by molecular replacement using Phaser (McCoy et al, J. Appl. Crystallogr. 40:658-674 (2007)) in the CCP4 Program Suite (Project, cta Crystallographica Section D 50:760 (1994)) with published antibody structures as searching models. The gp120:CH103 crystal also belonged to the P2₁ space group

with cell dimensions at $a=48.9$, $b=208.7$, $c=69.4$, $\alpha=90$, $\beta=107.2$, $\gamma=90.0$, and contained two gp120:CH103 complexes per asymmetric unit (Table 7). The high resolution Fab CH103 structure was used as an initial model to place the Fab CH103 component in the complex. With the Fab CH103 position fixed, searching
5 with the extended core gp120 of ZM176.66 in the VRC01-bound form as an initial model failed to place the gp120 component in the complex. After trimming the inner domain and bridging sheet from the gp120 model, Phaser was able to correctly place the remaining outer domain of gp120 into the complex without significant clashes. Analysis of the packing of the crystallographic lattice
10 indicated the lack of space to accommodate the inner domain of gp120, suggesting possible protease cleavage of the gp120 by the containing fungi during crystallization.

Structural refinements were carried out with PHENIX (Adams et al, Acta Crystallogr. D. Biol. Crystallogr. 58:1948-1954 (2002)). Starting with torsion-
15 angle simulated annealing with slow cooling, iterative manual model building was carried out on COOT (Emsley and Cowtan, Acta Crystallogr. D. Biol. Crystallogr. 60:2126-2132 (2004)) with maps generated from combinations of standard positional, individual *B*-factor, TLS refinement algorithms and non-crystallographic symmetry (NCS) restraints. Ordered solvents were added during
20 each macro cycle. Throughout the refinement processes, a cross validation (R_{free}) test set consisting of 5% of the data was used and hydrogens were included as riding model. Structure validations were performed periodically during the model building/refinement process with MolProbity (Davis et al, Nucleic Acids Res. 35:W375-383 (2007)) and pdb-care (Lutheke and von der Lieth, BMC
25 Bioinformatics 5:69 (2004)). X-ray crystallographic data and refinement statistics are summarized in Table 7. The Kabat nomenclature (Kabat et al, C. Sequences of Proteins of Immunological Interest, 5th Edition (1991)) was used for numbering of amino acid residues in amino acid sequences in antibodies.

Protein structure analysis and graphical representations. PISA (Krissinel and Henrick, J. Mol. Biol. 372:774-797 (2007)) was used to perform protein-protein interfaces analysis. CCP4 (Emsley and Cowtan, Acta Crystallogr. D. Biol. Crystallogr. 60:2126-2132 (2004)) was used for structural
 5 alignments. All graphical representation with protein crystal structures were made with Pymol (DeLano, The PyMOL Molecular Graphics System, DeLano Scientific, San Carlos, CA, USA. Org (2002)).

Polyreactivity analysis of CH103 clonal lineage antibodies by HEp-2 cell staining, ANA assays and protein array microchip. All antibodies in
 10 CH103 clonal lineage were assayed at 50µg/ml for autoreactivity to HEp-2 cells (Inverness Medical Professional Diagnostics, Princeton, NJ) by indirect immunofluorescence staining and a panel of autogens by ANA assays using the methods as reported previously (Haynes et al, Science 308:1906-1908 (2005)). The intermediate antibody (IA1) and CH106 were identified as reactive with
 15 HEp-2 cells and then selected for further testing for reactivity with human host cellular antigens using ProtoArray 5 microchip (Invitrogen, Grand Island, NY) according to the instructions of the microchip manufacturer. Briefly, ProtoArray 5 microchips were blocked and exposed to 2 µg/ml IA1, CH106 or an isotype-matched (IgG1, k) human myeloma protein, 151K (Southern
 20 Biotech) for 90 min at 4 °C. Protein-Ab interactions were detected by 1 µg/mL Alexa Fluor 647-conjugated anti-human IgG. The arrays were scanned at 635 nm with 10µm resolution using 100% power and 600 gain (GenePix 4000B scanner, Molecular Devices). Fluorescence intensities were quantified using GenePix Pro 5.0 (Molecular Devices). Lot-specific protein spot definitions
 25 were provided by the microchip manufacturer and aligned to the image.

Results

Isolation of the CH103 BnAb lineage

The CH505 donor was enrolled in the CHAVI001 acute HIV-1 infection cohort (Tomaras et al, J. Virol. 82:12449-12463 (2008)) approximately 4 weeks
 5 after HIV-1 infection (Fig. 7) and followed for more than 3 years. Single genome amplification of 53 plasma viral Env gp160 RNAs (5) from 4 weeks after transmission identified a single clade C transmitted/founder (T/F) virus. Serologic analysis demonstrated the development of autologous neutralizing antibodies at 14 weeks, CD4 binding site (CD4bs) antibodies that bound to a recombinant Env
 10 protein (resurfaced core, RSC3) (Wu et al, Science 329:856-861 (2010)) at 53 weeks, and evolution of plasma cross-reactive neutralizing activity from 41-92 weeks after transmission (Lynch et al, J. Virol. 86:7588-7595 (2012)) (Fig. 1, Table 1, Fig. 8). The natural variable regions of heavy- (V_HDJ_H) and light-chain (V_LJ_L) gene pairs of antibodies CH103, CH104, CH106 were isolated from
 15 peripheral blood mononuclear cells (PBMC) at 136 weeks after transmission by flow sorting of memory B cells that bound RSC3 Env protein (Scheid et al, J. Immunol. Methods 343:65-67 (2009), Wu et al, Science 329:856-861 (2010), (Scheid et al, Nature 458:636-640 (2009)) (Fig. 1B). The V_HDJ_H gene of antibody CH105 was similarly isolated, but no V_LJ_L gene was identified from the same
 20 cell. Analysis of characteristics of V_HDJ_H (V_H4 -59 [posterior probability, PP = 0.99], D3-16 (PP=0.74), J_H4 [PP= 1.00]) and V_LJ_L (V_L3 -1 [PP=1.00], J_L1 [PP=1.00]) rearrangements in mAbs CH103, CH104, CH105 and CH106 demonstrated that these antibodies were representatives of a single clonal lineage designated as the CH103 clonal lineage (Fig. 2, Table 2).

25 Neutralization assays using a previously described (Wu et al, Science 329:856-861 (2010), (Seaman et al, J. Virol. 84:1439-1452 (2010)) panel of 196 of geographically and genetically diverse Env-pseudoviruses representing the major circulated genetic subtypes and circulating recombinant forms

demonstrated that CH103 neutralized 55% of viral isolates with a geometric mean IC_{50} of 4.54 ug/ml among sensitive isolates (Fig. 1C, Table 3). ELISA cross-competition analysis demonstrated that CH103 binding to gp120 was competed by known CD4bs ligands such as mAb VRC01 and the chimeric protein CD4-Ig (Fig. 1D); CH103 binding to RSC3 Env was also substantially diminished by gp120 with P363N and Δ 371I mutations known to reduce binding of most CD4bs mAbs (Fig. 9) (Wu et al, Science 329:856-861 (2010), Lynch et al, J. Virol. 86:7588-7595 (2012)).

Molecular characterization of the CH103 BnAb lineage

The RSC3 probe isolated CH103, CH104, CH105, and CH106 BnAbs by single cell flow sorting. The CH103 clonal lineage was enriched by V_HDJ_H and V_LJ_L sequences identified by pyrosequencing PBMC DNA (Liao et al, J. Exp. Med. 208:2237-2249 (2011), Boyd et al, Sci. Transl. Med. 1:12ra23 (2009)) obtained 66 and 140 weeks after transmission and cDNA antibody transcripts (Wu et al, Science 333:1593-1602 (2011)) obtained 6, 14, 53, 92 and 144 weeks after transmission. From pyrosequencing of antibody gene transcripts, 457 unique heavy and 171 unique light chain clonal members were found (Figs. 2A, 2B). For comprehensive study, a representative 14 member BnAb pathway was reconstructed from V_HDJ_H sequences (1AH92U, 1AZCET and 1A102R) recovered by pyrosequencing, and V_HDJ_H genes of the inferred intermediate (I) antibodies (I1-I4, I7, I8) (Haynes et al, Nat. Biotechnol. 30:423-433 (2012), (Ma et al, PLoS Pathog. 7:e1002200 (2011)), Liao et al, J. Exp. Med. 208:2237-2249 (2011)) (Kepler, TB, Submitted, 2012) that were paired and expressed with either the UCA or I2 V_LJ_L depending on the genetic distance of the V_HDJ_H to either the UCA or mature antibodies (Fig. 2C, Table 2). The mature CH103, CH104 and CH106 antibodies were paired with their natural V_LJ_L . The CH105 natural

V_HD_HJ_H isolated from RSC3 memory B cell sorting was paired with the V_LJ_L of I2.

Whereas the V_HDJ_H mutation frequencies of the published CD4bs BnAbs VRC01, CH31 and NIH45-46 V_HDJ_H are 30-36% (Wu et al, Science 329:856-861 (2010), Wu et al, Science 333:1593-1602 (2011), Zhou et al, Science 329:811-817 (2010), Scheid et al, Science 333:1633-1637 (2011), (Bonsignori et al, J. Virol. 86:4688-4692 (2012)), the CH103 lineage CH103, CH104, CH105 and CH106 V_HDJ_H frequencies are 13-17% (Fig. 2C). Additionally, antibodies in CH103 clonal lineage do not contain the large (>3 nt) insertion or deletion mutations common in VRC01-class of BnAbs (1-3) with the exception of the V_LJ_L of CH103 which contained a 3 aa LCDR1 deletion.

It has been proposed that one reason CD4bs BnAbs are difficult to induce is heterologous HIV-1 Envs do not bind their UCAs (Zhou et al, Science 329:811-817 (2010), Xiao et al, Biochem. Biophys. Res. Commun. 390:404-409 (2009)), Scheid et al, Science 333:1633-1637 (2011)). The question presented was whether the CH505 T/F Env, the initial driving antigen for the CH103 BnAb lineage, would preferentially bind to early CH103 clonal lineage members and the UCA compared to heterologous Envs. Indeed, a heterologous gp120 T/F Env, B.63521, did not bind to the CH103 UCA (Fig. 2D) but did bind to later members of the clonal lineage. Affinity for this heterologous Env increased four orders of magnitude during somatic evolution of the CH103 lineage, with maximal K_d values of 2.4 to 7.0 nM in the mature CH103-CH106 mAbs (Fig. 2D). The CH103 UCA mAb also did not bind other heterologous T/F Envs AE.427299, B.9021 and C.1086 (Table 4), confirming lack of heterologous Env binding to CD4bs UCAs. Moreover, the gp120 Env RSC3 protein was also not bound by the CH103 UCA and earlier members of the clonal lineage (Fig. 9A) and no binding was seen with RSC3 mutant proteins known to disrupt CD4bs BnAb binding (Fig. 9B).

In contrast to heterologous Envs, the CH505 T/F Env gp140 bound well to all of the candidate UCAs (Table 5) with the highest UCA affinity of $K_d = 37.5$ nM. In addition, the CH505 T/F Env gp140 was recognized by all members of the CH103 clonal lineage (Fig. 2D). Whereas affinity to the heterologous T/F Env B.63521 increased by over four orders of magnitude as the CH103 lineage matured, affinity for the CH505 T/F Env increased by no more than ten fold (Fig. 2D). To directly demonstrate Env escape from CH103 lineage members, autologous recombinant gp140 Envs isolated at weeks 30, 53 and 78 postinfection were expressed and compared with the CH505 T/F Env for binding to the BnAb arm of the CH103 clonal lineage (Table 6, Fig. 10). Escape mutant Envs could be isolated that were progressively less reactive with the CH103 clonal lineage members. Envs isolated from weeks 30, 53 and 78 lost UCA reactivity and only bound intermediate antibodies 3, 2 and 1 as well as BnAbs CH103, CH104, CH105 and CH106 (Table 6). In addition, two Env escape mutants from week-78 viruses also lost either strong reactivity to all intermediate antibodies or to all lineage members (Table 6).

To quantify CH103 clonal variants from initial generation to induction of broad and potent neutralization, pyrosequencing of antibody cDNA transcripts from five time points, weeks 6, 14, 53, 92 and 144 weeks after transmission was used (Table 7). Two V_HDJ_H chains closely related to, and possibly members of, the CH103 clonal lineage were found (Fig. 2A, Table 7). Moreover, one of these V_HDJ_H when reconstituted in a full IgG1 backbone and expressed with the UCA V_LJ_L weakly bound the CH505 T/F Env gp140 at endpoint titer of 11 $\mu\text{g/ml}$ (Fig. 2A). These reconstructed antibodies were present concomitant with CH505 plasma autologous neutralizing activity at 14 weeks after transmission (Fig. 8). Antibodies that bound the CH505 T/F Env were present in plasma as early as 4 weeks after transmission (data not shown). Both CH103 lineage V_HDJ_H and V_LJ_L sequences peaked at week 53 with 230 and 83 unique transcripts, respectively.

V_HDJ_H clonal members fell to 46 at week 144, and V_LJ_L members were 76 at week 144.

Polyreactivity is a common trait of BnAbs, suggesting that the generation of some BnAbs may be controlled by tolerance mechanisms (Haynes et al, Science 308:1906-1908 (2005), Mouquet et al, Nature 467:591-595 (2010), Haynes et al, Hum. Antibodies 14:59-67 (2005)). Conversely, polyreactivity can arise during the somatic evolution of B cells in germinal centers as a normal component of B-cell development (Wardemann et al, Science 301:1374-1377 (2003)). The CH103 clonal lineage was evaluated for polyreactivity as measured by HEp-2 cell reactivity and binding to a panel of autoantigens (Haynes et al, Science 308:1906-1908 (2005)). While earlier members of the CH103 clonal lineage were not polyreactive by these measures, polyreactivity was acquired in concert with BnAb activity by the intermediate antibody I2, I1, and clonal members, CH103, CH104 and CH106 (Figs. 11A, 11B). The BnAbs CH106 and intermediate antibody I1 also demonstrated polyreactivity in protein arrays with specific reactivity to several human autoantigens, including elongation factor-2 kinase and ubiquitin-protein ligase E3A (Figs. 11C and 11D).

Structure of CH103 in complex with HIV-1 gp120

Crystals of the complex between Fab CH103 and the ZM176.66 strain of HIV diffracted to 3.15-Å resolution, and molecular replacement identified solutions for Fab CH103 and for the outer domain of gp120 (Fig. 3A). Inspection of the CH103-gp120 crystal lattice (Fig. 12) indicated the absence of the gp120 inner domain was likely related to proteolytic degradation of the extended gp120 core to an outer domain fragment. Refinement to R_{crystal}/R_{free} of 19.1%/25.3% (Table 8) confirmed a lack of electron density for gp120 residues N terminal to residue Val 255_{gp120} or C terminal to Gly472_{gp120} (gp120 residues are numbered according to standard HXB2 nomenclature), and no electron density was observed

for residues 301-324_{gp120} (V3), 398-411_{gp120} (V4) and 421-439_{gp120} (β 20-21). Superposition of the ordered portions of gp120 (gp120 residues are numbered according to standard HXB2 nomenclature) in complex with CH103 with the fully extended core gp120 bound by antibody VRC01 (Zhou et al, Science
 5 329:811-817 (2010)) indicated a highly similar structure (C α -rmsd 1.16 Å) (Fig. 3B). Despite missing portions of core gp120, the entire CH103 epitope appeared to be present in the electron density for the experimentally observed gp120 outer domain.

The surface bound by CH103 formed an elongated patch with dimensions
 10 of $\sim 40 \times 10$ Å, which stretched across the site of initial CD4 contact on the outer domain of gp120 (Fig. 3C). The gp120 surface recognized by CH103 correlated well with the initial site of CD4 contact; of the residues contacted by CH103, only eight of these residues were not predicted to interact with CD4. CH103 interacted with these residues through side-chain contact with Ser256_{gp120} in loop D, main-
 15 and side-chain contacts with His364_{gp120} and Leu369_{gp120} in the CD4-binding loop, and main- and side-chain contacts with Asn463_{gp120} and Asp464_{gp120} in the V5 loop (Fig. 3D). Notably, residue 463 is a predicted site of *N*-linked glycosylation in strain ZM176.66 as well as in the autologous CH505 virus, but electron density for an *N*-linked glycan was not observed. Overall, of the 22
 20 residues that mAb CH103 was observed to contact on gp120, 14 were expected to interact with CD4 (16 of these residues with antibody VRC01), providing a structural basis for the CD4-epitope specificity of CH103 and its broad recognition (Table 9).

Residues 1-215_{HC} on the antibody heavy chain and 1-209_{LC} showed well
 25 defined backbone densities. Overall, CH103 utilizes a CDR H3 dominated mode of interaction, although all six of the complementarity-determining regions (CDRs) interacted with gp120 as well as the light chain framework region 3 (FWR3) (Fig. 4A,B, Tables 10 and 11). It is important to note that $\sim 40\%$ of the

antibody contact surface was altered by somatic mutation, in two regions, in the CDR H2 and in the CDR L1, L2 and FWR3. In particular, residues 56_{HC}, 50_{LC}, 51_{LC} and 66_{LC} are altered by somatic mutation to form hydrogen bonds with the CD4-binding loop, loop D and loop V5 of gp120. Nevertheless, 88% of the CH103 V_HD_HJ_H and 44% of the V_λJ_λ contact areas were with amino acids unmutated in the CH103 germline, potentially providing an explanation for the robust binding of the T/F Env to the CH103 UCA (Figs. 4C, 4D, and Table 12).

Evolution of transmitted/founder Env sequences tracks acquisition of BnAb activity

Using single genome amplification and sequencing ((Keele et al, Proc. Natl. Acad. Sci. USA 105:7552-7557 (2008)), the evolution of CH505 *env* genes was tracked longitudinally from the T/F virus through 160 weeks post-transmission (Fig. 5, Fig. 12). The earliest recurrent mutation in Env, N279K (HIV-1 HXB2 numbering), was found at 4 weeks post-infection, and was in Env loop D in a CH103 contact residue. By week 14 additional mutations in loop D appeared, followed by mutations and insertions in V1 at week 20. Insertions and mutations in the V5 loop began to accumulate by week 30 (Fig. 5). Thus, the T/F virus began to diversify in key CD4 contact regions starting within 3 months of infection (Figs. 13, 14). Loop D and V5 mutations were directly in or adjacent to CH103/Env contact residues. Although the V1 region was not included in the CH103-Env co-crystal, the observed V1 CH505 Env mutations were adjacent to contact residues for CD4 and VRC01 so are likely to be relevant. It is also possible that early V1 insertions (Fig. 5) were selected by inhibiting access to the CD4bs in the trimer or that they arose in response to early T cell pressure. CD4 binding-loop mutations were present by week 78. Once regions that could directly impact CH103-lineage binding began to evolve (loop D, V5, the CD4 binding,

loop, and possibly V1), they were under sustained positive selective pressure throughout the study period (Fig. 5, Figs. 13, 14, Table 13).

Considerable within-sample virus variability was evident in Env regions that could impact CH103-lineage antibody binding, and diversification within these regions preceded neutralization breadth. Expanding diversification early in viral evolution (4-22 weeks after transmission) (Figs. 13, 14) coincided with autologous NAb development, consistent with autologous NAb escape mutations. Mutations that accumulated from weeks 41-78 in CH505 Env contact regions immediately preceded development of NAb breadth (Fig 5, Figs. 13, 14). By weeks 30-53, extensive within-sample diversity resulted from both point mutations in and around CH103 contact residues, and to multiple insertions and deletions in V1 and V5 (Fig. 14). A strong selective pressure seems to have come into play between weeks 30 and 53, perhaps due to autologous neutralization escape, and neutralization breadth developed after this point (Figs. 5, 13, 14). Importantly, due to apparent strong positive selective pressure between week 30 and week 53, there was a dramatic shift in the viral population that is evident in the phylogenetic tree, such that only viruses carrying multiple mutations relative to the T/F, particularly in CH103 contact regions, persisted after week 30. This was followed by extreme and increasing within time-point diversification in key epitope regions, beginning at week 53 (Fig. 14). Emergence of antibodies with neutralization breadth occurred during this time (Fig. 8, Table 1). Thus, plasma breadth evolved in the presence of highly diverse forms of the CH103 epitope contact regions (Fig. 5, Fig. 8).

To evaluate and compare the immune pressure on amino acids in the region of CH103 and CD4 contacts, a comparison was made of the frequency of mutations in evolving T/F sequences of patient CH505 during the first year of infection and in 16 other acutely infected subjects followed over time (Fig. 15). The accumulation of mutations in the CH505 virus population was concentrated

in regions likely to be associated with escape from the CH103 lineage (Fig. 15A), and diversification of these regions was far more extensive during the first six months of infection in CH505 than in other subjects (Fig. 15B). However, by one year into their infections, viruses from the other subjects had also begun to
5 acquire mutations in these regions. Thus, the early and continuing accumulation of mutations in CH103 contact regions may have potentiated the early development of neutralizing antibody breadth in patient CH505.

Neutralization of autologous and heterologous viruses and the CH103 lineage

Heterologous BnAb activity was confined to the later members (I3 and
10 later) of the BnAb arm of the CH103 lineage as manifested by their neutralization capacity of pseudoviruses carrying tier 2 Envs A.Q842 and B.BG1168 (Fig. 6A). Similar results were seen with Envs A.Q168, B.JRFL, B.SF162 and C.ZM106 (Tables 14 and 15). In contrast, neutralizing activity of clonal lineage members against the autologous T/F Env pseudovirus appeared earlier with measurable
15 neutralization of the CH505 T/F virus by all members of the lineage after the UCA except mAb 1AH92U (Fig. 6A). Thus, within the CH103 lineage, early intermediate antibodies neutralized the T/F virus, while later intermediate antibodies gained neutralization breadth, indicating evolution of neutralization breadth with affinity maturation, and CH103-CH106 BnAbs evolved from an
20 early autologous neutralizing antibody response. Moreover, the clonal lineage was heterogeneous, with an arm of the lineage represented in Fig. 6A evolving neutralization breadth and another antibody arm capable of mediating only autologous T/F virus neutralization. While some escape viruses are clearly emerging over time (Table 4), it is important to point out that, whereas escape
25 mutant viruses are driving BnAb evolution, the BnAbs remain capable of neutralizing the CH505 T/F virus (Fig. 6A). Of note, the earliest mutations in the heavy chain lineage clustered near the contact points with gp120, and these

remained fixed throughout the period of study, while mutations that accumulated later tended to be further from the binding site and may be impacting binding less directly (Fig. 10). Thus, stimulation of the CH103 BnAbs occurs in a manner to retain reactivity with the core CD4bs epitope present on the T/F Env. One
5 possibility that might explain this is that the footprint of UCA binding contracts to the central core binding site of the CH103 mature antibody. Obtaining a crystal structure of the UCA with the T/F Env should inform this notion. Another possibility is that because affinity maturation is occurring in the presence of highly diverse forms of the CD4bs epitope, antibodies that favor tolerance of
10 variation in and near the epitope are selected instead of those antibodies that acquire increased affinity for particular escape Envs. In both scenarios, persistence of activity to the T/F form and early viral variants would be expected. Fig. 6B and Fig. 16 show views of accumulations of mutations or entropy during the parallel evolution of the antibody paratope and the Env epitope bound by mAb
15 CH103.

Table 1. Plasma neutralization activity developed over time of in patient CH505 against the autologous transmitted/funder (T/F) and heterologous viruses.

Sample ID	Week after Infection	EC50, reciprocal dilution						
		MuLV	CH505	B.SF162	B.JRFL	A.Q168	A.842	B.BG1168
G770DM2V-12	6	<20	<20	<20	<20	<20	<20	<20
G770DPFL-12	7	<20	<20	<20	<20	<20	<20	<20
K770DQNT-15	8	<20	<20	<20	<20	<20	<20	<20
K770F1ZZ-12_16	14	<20	45	<20	<20	<20	<20	<20
A770FDB0-13	20	<20	157	29	<20	<20	<20	<20
C770FJKX-13	22	<20	267	28	<20	<20	<20	<20
F770G05F-11	30	<20	1,291	60	<20	<20	<20	<20
A770GN15-12	41	<20	1,636	154	56	<21	53	<20
A770H7QF-11	53	<20	1,701	237	244	32	183	<20
D8Z03WQG-03	66	<20	3,193	401	701	69	367	<20
K8Z047D8-04	78	<20	6,428	1,172	806	83	345	<20
A770JL0J-12	92	<20	3,396	1,534	522	92	293	<20
J770JXKQ-12	100	<20	2,464	1,066	619	94	473	25
K770KQ98-12	136	<20	4,985	4,651	2,085	172	433	35
A770KRJ3-13	138	<20	3,586	5,081	1,368	138	326	56
H770KSHS-12	140	<20	3,374	13,407	1,287	148	237	51
C770KW30-13	144	<20	4,665	8,354	905	118	237	56
F770LOXJ-12	152	<20	1,789	3,122	1,612	108	234	40
K8Z06JCY-03	160	<20	2,684	9,761	2,482	144	230	35
K770LJF6-12	176	<20	2,003	5,148	2,243	91	150	55
B770MDVN-11	208	<20	1,353	5,850	1,303	60	95	58
E770NW1S-11	233	<20	3,279	3,612	895	107	151	31
K8Z07X34-04	234	<20	3,033	4,887	1,712	103	232	37
D8Z07Y9M-06	236	<20	1,969	4,417	1,354	107	299	60
2F5*		>50	>50	0.69	2.26	1.62	11.71	57
								1.43

*EC50 values for positive control antibody 2F5 are presented as ug/ml. MuLV = murine leukemia virus as negative control.

Table 2. V(D)J rearrangement of the matured, and reverted unmutated ancestor and intermediate antibodies in CH103 clonal lineage.

Antibody ID	IgH_ID	VH	DH	JH	Mutation frequency	CDRH3 length ¹	Isotype	VL ID	VL	JL	Mutation frequency	CDRL3 length ¹
UCA	UCAVH	4-59*01	3-16*01	4*02	0.0%	15	IgG1	UCAVL	3-1*01	1*01	0%	10
I8	I8VH	4-59*01	3-16*01	4*02	3.6%	15	IgG1	UCAVL	3-1*01	1*01	0%	10
I7	I7VH	4-59*01	3-16*01	4*02	5.0%	15	IgG1	UCAVL	3-1*01	1*01	0%	10
I4	I4VH	4-59*01	3-16*01	4*02	6.9%	15	IgG1	UCAVL	3-1*01	1*01	0%	10
I3	I3VH	4-59*01	3-16*01	4*02	9.1%	15	IgG1	I2VL	3-1*01	1*01	10.0%	10
I2	I2VH	4-59*01	3-16*01	4*02	14.9%	15	IgG1	I2VL	3-1*01	1*01	10.0%	10
I1	I1VH	4-59*01	3-16*01	4*02	15.2%	15	IgG1	I2VL	3-1*01	1*01	10.0%	10
1AZCETI5	1AZCETI5VH	4-59*01	3-16*01	4*02	15.2%	15	IgG1	I2VL	3-1*01	1*01	10.0%	10
1AH92U	1AH92UVH	4-59*01	3-16*01	4*02	8.3%	15	IgG1	UCAVL	3-1*01	1*01	0%	10
1A102R16	1A102R16VH	4-59*01	3-16*01	4*02	7.7%	15	IgG1	I2VL	3-1*01	1*01	10.0%	10
CH106	CH106VH	4-59*01	3-16*01	4*02	16.0%	15	IgG1	CH106VL	3-1*01	1*01	11.2%	10
CH103	CH103VH	4-59*01	3-16*01	4*02	16.8%	15	IgG1	CH103VL	3-1*01	1*01	10.6%	10
CH104	CH104VH	4-59*01	3-16*01	4*02	14.9%	15	IgG1	CH104VL	3-1*01	1*01	10.6%	10
CH105	CH105VH	4-59*01	3-16*01	4*02	12.7%	15	IgG1	I2VL	3-1*01	1*01	10.0%	10

¹The HCDR3 and LCDR3 lengths of the CH103 lineage are similar to the median of HCDR3 and LCDR3 lengths of unrelated antibodies in pyrosequencing database or Genbank. Using the same 454 pyrosequencing dataset derived from three HIV infected subjects unrelated to the CH505 patient as the source of comparison, we find that the CH103 CDRH3 length of 45 nucleotides (15 aa) is the median value. The interquartile range is 39-54 nucleotides (13-18 aa). 9% of all heavy chains in this database have HCDR3 length = 45 nucleotides, this is the second most-frequent length, after 42 nucleotides. We used human L\lambda rearrangements from Genbank to compare the light chain. The CH103 light chain CDR3 is 30 nucleotides (10 aa) long. The median among Genbank human lambda chains is 33 nucleotides (11 aa). 24% of all human lambda chains have HCDR3 length = 30 nt, second-most frequent after 33nt.

Table 3a. Comparison of neutralization activity of CH103, and other CD4bs mAbs against 25 clade A Env-pseudoviruses.

Virus ID	Clade	IC50 ^a				IC80 ^a			
		VRC01	CH31	b12	CH103	VRC01	CH31	b12	CH103
1260.v5.c36	A	0.53		>50	>50	1.48		>50	>50
0330.v4.c3	A	0.06		>50	7.84	0.23		>50	31
0439.v5.c1	A	0.05	0.06	>50	8.37	0.24	0.26	>50	>50
0365.v2.c20	A		0.01	18.90	4.00		0.03	>50	40
3415.v1.c1	A	0.09	0.06	6.93	2.46	0.26	0.18	13.70	4
0718.v3.c11	A	0.22	0.13	12.10	29.60	4.99	0.41	43.80	>50
08-F1_F6_20	A	0.06		0.06	>50	0.32		1.30	>50
3B201.B42	A	0.34	0.04	0.52	9.19	1.11	0.13	3.77	40
0B539.2B13	A	0.09	>50	1.42	22.20	0.33	>50	7.40	>50
BI369.9A	A	0.15	0.01	14.90	>50	0.66	0.04	>50	>50
BS208.B1	A	0.03	0.02	0.04	>50	0.10	0.05	0.33	>50
ER2008.12	A	0.56	0.11	>50	>50	1.74	0.41	>50	>50
ER2018.11	A	0.07	0.39	49.20	40.00	0.40	2.32	>50	>50
NH1209.18	A	0.09	0.02	0.79	>50	0.30	0.10	3.39	>50
MB201.A1	A	0.24	0.02	>50	17.70	0.48	0.07	>50	>50
0B539.2B7	A	0.54	0.16	14.00	3.70	1.48	0.45	>50	14
MI369.A5	A	0.16	0.03	1.58	>50	0.77	0.08	36.40	>50
MS208.A1	A	0.15	0.07	0.13	>50	0.67	0.21	0.98	>50
Q23.17	A	0.09		>50	10.40	0.25		>50	12
Q259.17	A	0.05	5.09	>50	10.60	0.25	>50	>50	33
Q769.d22	A	0.02	0.03	>50	1.00	0.07	0.16	>50	2
Q842.d12	A	0.01	0.003	>50	1.46	0.02	0.01	>50	5
0209.14M.A2	A	0.02	0.01	>50	1.16	0.08	0.07	>50	5
RW020.2	A	0.30	0.004	10.70	23.60	0.87	0.03	21.60	47
UG037.8	A	0.04	0.02	>50	1.76	0.13	0.09	>50	11
Breadth	N=25 Titer <50	96	80	56	68	96	76	40	4
metric Mean^b		0.095	0.038	2.180	6.569	0.373	0.12	5.34	0

Values <1 µg/ml are indicated in red and values 1-50 µg/ml are in green.

Geometric means were calculated for neutralization sensitive viruses with an IC₅₀ or IC₈₀ value <50 µg/ml.

Results of 118 isolates summarized in Tables 3a, b and c are representatives of total of 196 isolates tested.

Table 3b. Comparison of neutralization activity of CH103, and other CD4bs mAbs against 39 clade B Env-pseudoviruses.

Virus ID	Clade	IC ₅₀ ^a				IC ₈₀ ^a			
		VRC01	CH31	b12	CH103	VRC01	CH31	b12	CH103
3988.25	B	2.10		2.21	9.84	>50		12.40	11.68
5768.04	B	0.10	0.10	5.62	7.08	0.94	2.37	>50	7.03
6101.10	B	0.10		>50	1.80	0.33		>50	2.25
6535.3	B	2.16		0.87	4.67	6.27		18.50	3.90
7165.18	B	>50		>50	>50	>50		>50	>50
45_01dG5	B				0.80				1.10
89.6.DG	B	0.46	0.06	0.08	1.40	1.58	0.33	0.49	1.56
AC10.29	B	1.43		1.86	>50	3.83		16.30	>50
ADA.DG	B	0.42	0.21	0.13	1.49	1.40	1.07	0.73	1.82
Bal.01	B	0.10		0.09	0.68	0.32		0.45	2.01
BG1168.01	B	0.45	0.65	>50	21.70	1.43	2.50	>50	>50
BL01.DG	B	>50	>50	>50	>50	>50	>50	>50	>50
BR07.DG	B	1.67	2.01	0.19	9.67	4.67	8.06	1.30	>50
BX08.16	B	0.28		0.92	1.15	0.68		8.16	1.07
CAAN.A2	B	1.06	>50	>50	>50	2.63	>50	>50	>50
CNE10	B	0.78		>50	26.90	1.87		>50	>50
CNE12	B	0.79		>50	26.40	2.19		>50	>50
CNE14	B	0.39		8.28	0.52	0.98		24.50	0.60
CNE4	B	0.87		>50	43.50	2.36		>50	>50
CNE57	B	0.54		16.60	>50	1.34		>50	>50
HO86.8	B	>50	>50	>50	>50	>50	>50	>50	>50
HT593.1	B	0.44	0.17	0.25	40.00	1.72	0.76	4.51	>50
HXB2.DG	B	0.04		0.00	0.12	0.08		0.01	0.23
JRCSF.JB	B	0.23		0.22	0.91	0.80		1.56	1.96
JRFL.JB	B	0.03		0.02	0.02	0.12		0.10	0.05
MN.3	B	0.03		<0.0006	0.19	0.07		0.002	0.54
PVO.04	B	0.39		>50	>50	0.99		>50	>50
QH0515.01	B	0.52	0.03	0.80	10.20	3.66	0.38	12.50	4.93
QH0692.42	B	1.16		0.79	25.20	3.00		3.05	>50
REJO.67	B	0.05		1.70	2.15	1.47		>50	5.38
RHPA.7	B	0.05		0.13	8.53	0.21		0.58	9.20
SC422.8	B	0.13		0.20	2.44	0.16		2.19	3.08
SF162.LS	B	0.24		0.03	0.84	0.33		0.15	1.19
SS1196.01	B	0.28		0.78	1.34	0.63		5.51	1.08
THRO.18	B	4.42		0.96	>50	0.65		8.76	>50
TRJO.58	B	0.08		>50	>50	15.10		>50	>50
TRO.11	B	0.34		>50	5.34	0.24		>50	5.15
WITO.33	B	0.11		9.67	7.35	1.09		>50	8.53
YU2.DG	B	0.06	0.072	1.46	1.80	0.27	0.23	6.34	2.00
Breadth	N=39	Titer <50	90	21	64	77	87	21	56
Geometric Mean^b			0.30	0.17	0.46	2.84	0.92	1.01	1.47

^aValues <1 µg/ml are indicated in red and values 1-50 µg/ml are in green.^bGeometric means were calculated for neutralization sensitive viruses with an IC₅₀ or IC₈₀ value <50 µg/ml.

Table 3c. Comparison of neutralization activity of CH103, and other CD4bs mAbs against 54 clade C Env-pseudoviruses.

Virus ID	Clade	IC50 ^a				IC80 ^a			
		VRC01	CH31	b12	CH103	VRC01	CH31	b12	CH103
286.36	C	0.10		0.90	8.36	1.36		3.18	18.80
288.38	C	1.52		>50	8.45	0.38		>50	19.10
0013095-2.11	C	0.14		>50	5.71	5.86		>50	25.00
001428-2.42	C	0.01		>50	1.51	0.32		>50	4.64
0077_V1.C16	C	1.04		0.16	>50	0.04		2.42	>50
00836-2.5	C	0.13		>50	28.60	3.65		>50	>50
0921.V2.C14	C			3.30	>50	0.52		12.70	>50
16055-2.3	C	0.11		>50	>50			>50	>50
16845-2.22	C	2.41	4.55	>50	>50	0.37	27.80	>50	>50
16936-2.21	C	0.11		>50	5.57	9.07		>50	34.10
25710-2.43	C	0.55		>50	>50	0.47		>50	>50
25711-2.4	C	0.71		23.40	17.20	1.56		>50	46.00
25925-2.22	C	0.56		>50	26.70	1.70		>50	>50
26191-2.48	C	0.20		1.44	>50	1.39		7.25	>50
3168.V4.C10	C	0.13		>50	10.30	0.65		>50	>50
3637.V5.C3	C	4.09		>50	>50	0.28		>50	>50
3873.V1.C24	C	0.95		>50	>50	11.00		>50	>50
6322.V4.C1	C	>50		>50	>50	0.33		>50	>50
6471.V1.C16	C	>50		>50	>50	>50		>50	>50
6631.V3.C10	C	>50		>50	>50	>50		>50	>50
6644.V2.C33	C	0.16		0.03	0.38	>50		0.36	1.51
6785.V5.C14	C	0.33		16.50	8.95	0.53		>50	27.60
6838.V1.C35	C			>50	29.00	0.87		>50	>50
96ZM651.02	C	0.53		>50	8.99			>50	>50
BR025.9	C	0.27	1.13	>50	>50	1.94	49.00	>50	>50
CAP210.E8	C	>50		>50	>50	1.08		>50	>50
CAP244.D3	C	0.86		>50	>50	>50		>50	>50
CAP45.G3	C	9.47		0.11	>50	2.36		3.65	>50
CNE30	C	0.93		3.81	>50	>50		18.10	>50
CNE31	C	0.96		18.40	>50	2.59		>50	>50
CNE53	C	0.11		>50	6.92	2.24		>50	>50
CNE58	C	0.12		>50	0.54	0.28		>50	1.46
DU123.06	C	13.60		0.40	>50	0.31		5.02	>50
DU151.02	C	7.70		16.00	>50	>50		>50	>50
DU156.12	C	0.08		1.41	>50	>50		4.90	>50
DU172.17	C	>50		0.55	>50	0.24		2.95	>50
DU422.01	C	>50		0.40	>50	>50		2.17	>50
MW965.26	C	0.04		0.002	0.01	>50		2.34	0.09
SO18.18	C	0.07	0.07	14.30	1.88	0.12	0.32	47.20	7.48
TV1.29	C	>50	>50	>50	>50	0.17	>50	>50	>50
TZA125.17	C	>50	>50	>50	>50	>50	>50	>50	>50
TZBD.02	C	0.07	0.09	>50	5.55	>50	0.34	>50	23.70
ZA012.29	C	0.25	0.02	>50	28.00	0.23	0.10	>50	>50
ZM106.9	C	0.25		>50	4.82	0.83		>50	8.11
ZM109.4	C	0.13		>50	21.20	0.64		>50	>50
ZM135.10a	C	1.28		>50	>50	0.39		>50	>50
ZM176.66	C	0.04	0.01	>50	0.23	6.16	2.27	>50	1.25
ZM197.7	C	0.62		>50	35.40	1.62	0.06	>50	>50
ZM214.15	C	0.88		4.78	41.40	0.25		>50	>50
ZM215.8	C	0.28		>50	2.57	1.55		>50	>50
ZM233.6	C	4.25		>50	7.35	3.04		>50	>50
ZM249.1	C	0.08		2.73	7.23	0.83		14.80	37.60
ZM53.12	C	0.84	4.76	>50	>50	23.10		>50	>50
ZM55.28a	C	0.14		>50	4.59	0.24	17.00	>50	14.30
Breadth	N=54 Titer <50	81	13	35	52	76	15	26	30
Geometric Mean ^b		0.366	0.219	1.224	5.23	0.870	1.574	4.881	8.11

^aValues <1µg/ml are indicated in red and values 1-50 µg/ml are in green.^bGeometric means were calculated for neutralization sensitive viruses with an IC₅₀ or IC₈₀ value <50µg/ml.

Table 4. Binding of antibodies in CH103 clonal lineage to heterologous HIV-1 Env proteins

Antibody	Binding to heterologous HIV-1 Env, EC50 (ug/ml)		
	AE.427299 gp120	B.9021 gp140	C.1086 gp140
UCA	NB	NB	NB
I8	NB	NB	NB
I7	NB	NB	NB
IA102RI6	NB	NB	NB
I4	NB	NB	36.2
IAZCET15	NB	>10	4.5
I3	NB	0.086	0.11
I2	NB	0.03	0.06
I1	NB	0.066	0.12
IAH92U	NB	3.2	0.16
CH104	NB	0.063	0.06
CH103	NB	0.5	0.07
CH106	NB	0.06	0.22
CH105	NB	0.09	0.11
NB = No dateable binding.			

Table 5. Affinity and kinetics of CH103 UCAs binding to autologous T/F CH505 gp140^a.

CH103UCAs	Binding affinity to autologous Envs	
	k_a ($\times 10^3$ M ⁻¹ s ⁻¹)	K_d nM
CH103UCA-1	26.7	37.5
CH103UCA-2,3,5 ^b	20.5	141.5
CH103UCA-4	27.2	36.8
CH103UCA-6	25.0	264.0

^a SPR binding rate constants and dissociation constant (K_d) was measured with each antibody captured on an anti-IgG (Fe specific) antibody surface and CH505 gp140 was injected in solution at concentrations ranging from 2 to 100 μ g/ml and as described in the online Methods section. Data is representative of at least two independent measurements.

^b Amino acid sequences encoded by V_HDJ_H of CH103UCAs-2, 4 - 6 are the same amino acid as shown in the alignment below.

DNA sequence alignment of V_HDJ_H CH103 UCAs: (SEQ ID NOS 1-6, respectively, in order of appearance)

CH103UCA-1	CAGGCTGTAGC TCCAGGAGTC GGGCCACAGG CTTGGTAAGC CTCTGAGAT TCTGTCTTTC ATTCCACAGT TCTTGGTGG CCCCATTAT ATTATATAT TGAATGGAT GGGCCAGCC TTAGGGAAG	150
CH103UCA-2		
CH103UCA-3		
CH103UCA-4		
CH103UCA-5		
CH103UCA-6		
CH103UCA-1	GACUGGAGTG GATTGGATAT ATTCTATTAA GTGGAGGAC CAACTACAC CTTCTCTCTA AATATGGAT CACATATCA GTAGACAGT CCAAGAACCA ATTCTCTTG AAGCTGAGT CTGAGACAC	150
CH103UCA-2		
CH103UCA-3		
CH103UCA-4		
CH103UCA-5		
CH103UCA-6		
CH103UCA-1	TACGTACAGG AGCTGTATT ACTGTGGAG CTTGACCCAGG GCGCACTTAS TCATGCTTA TTTTATATAC TGGGGCCAGG GAATCTTGT CACCGCTTC TCA	260
CH103UCA-2		
CH103UCA-3		
CH103UCA-4		
CH103UCA-5		
CH103UCA-6		

Amino acid sequence alignment of V_HDJ_H CH103 UCAs: (SEQ ID NOS 7-12, respectively, in order of appearance)

CH103UCA-1	QVQLQFSGPG LKRPSETLSL TCTVSGGIS SYVSWLRQF PGKLEWIGY IYYSGSTNYN PSLKSRVTIS VDTSKNQFSL KLSSTVAADT AVYYCASLPR GQLVNPFDY WGGTFLVTYS S	
CH103UCA-2		
CH103UCA-3		
CH103UCA-4		
CH103UCA-5		
CH103UCA-6		

Table 6. Reactivity of autologous Envs with antibodies in CH103 clonal lineage in ELISA.

Autologous Env	* Apparent binding affinity of autologous Envs to CH103 clonal lineage antibodies, EC50, ug/ml									
	UCA	18	14	13	12	11	CH105	CH103	CH104	CH106
CH0505 T/F	2	1.1	0.3	0.12	0.09	0.11	0.1	0.08	0.12	0.08
CH505.w30.e16	NB	>10	2.1	0.07	0.047	0.06	0.064	0.055	0.05	0.05
CH0505.w30.e23	NB	NB	>20	0.14	0.07	0.09	0.08	0.044	0.07	0.053
CH505.w53.e16	NB	NB	NB	0.066	0.03	0.05	0.05	0.03	0.036	0.032
CH505.w78.e7	NB	NB	NB	0.13	0.054	0.083	0.09	0.043	0.1	0.13
CH505.w78.e16	NB	NB	NB	NB	0.2	>10	0.3	1.2	0.19	0.14
CH505.w78.e38	NB	NB	NB	>100	>100	>10	>10	>10	>10	>10

* Env proteins highlighted in green had 2-fold or greater loss of binding affinity to antibodies in CH103 clonal lineage compared with the binding of transmitted/founder (T/F) Env to the same antibodies. NB = No detectable binding.

Table 7. V_HD_H sequences 2 genes (IZ95W and 02IV4) very similar to the CH103 VDJ genes, possible clonal members, identified by 454 sequencing and alignment with their UCA. V_HD_H genes of IZ95W and 02IV4 were produced as recombinant antibodies complemented with V_LJ_L genes of UCA and tested for binding to the autologous CH505 T F Env and heterologous HIV-1 Envs in ELISA assays. MA b IZ95W bound CH505 T F gp140 with end point titer of 11.1 ug/ml, but did not BIND with heterologous Envs, 6321, 9021, 1086C and 427299.

```

>UCA_VHDH
QVQLQESGPGLVKRPSETLSLTCTVSGGSISSYYWSWIRQPPGKGLEWIGYIIYSGSTNINPELSRVTTISVDTSRKNQFSLKLSVTAADTAVYYCASLPRGGLVNAVFDY
WGQGTLLVTVSS (SEQ ID NO: 7)
>IZ95W_VHDH
QVQLQESGPGLVKRPSETLSLTCTVSGGSISSYYWSWIRQPPGKGLEWIGYIIYSGSTNINPELSRVTTISVDTSRKNQFSLKLSVTAADTAVYYCASLPRGGLIIGYFDY
WGQGTLLVTVSS (SEQ ID NO: 13)
>02IV4_VHDH
QVQLQESGSGGLVAPSETLSLTCTVSGGSISSYYWSWIRQPPGKGLEWIGYIIYSGSTNINPELSRVTTISVDTSRKNQFSLKLSVTAADTAVYYCASLPRGGLIIGYFDY
WGQGTLLVTVSS (SEQ ID NO: 14)
>UCA_VHDH
QVQLQESGPG LVKRPSETLSL TCTVSGGSISS YYWSWIRQP FGRGLEWIGY IYYSGSTNIN
IZ95W_VHDH
02IV4_VHDH
-----S-----
-----S-----F-----60
>UCA_VHDH
PRLSRVTIS VDTSRKNQFSL KLSVTAADT AVYYCASLPR GGLVNAVFTY WGQGTLLVTVS
IZ95W_VH
02IV4_VH
-----I-----R-----ILG-----120
-----I-----R-----ILG-----120
>UCA_VHDH
S (SEQ ID NO: 7)
IZ95W_VHDH
- | (SEQ ID NO: 13)
02IV4_VHDH
- 121 (SEQ ID NO: 14)

```

Table 8. Crystallographic data collection and refinement statistics.

PDB accession code	CH103:gp120	Fab CH103
	To be deposited	To be deposited
Data collection		
Space group	<i>P</i> 21	<i>P</i> 21
Cell constants		
<i>a</i> , <i>b</i> , <i>c</i> (Å)	48.9, 208.7, 69.4	43.0, 146.4, 66.322
α , β , γ (°)	90.0, 107.2, 90.0	90.0, 97.7, 90.0
Wavelength (Å)	1.00	1.00
Resolution (Å)	50-3.20 (3.20-3.20)*	50-1.65(1.68-1.65)
<i>R</i> _{merge}	13.4 (44.4)	6.7(53.1)
<i>I</i> / σ <i>I</i>	9.6 (1.9)	30.0 (1.7)
Completeness (%)	89.4 (52.3)	98.4 (90.0)
Redundancy	3.4 (2.4)	3.4 (2.3)
Refinement		
Resolution (Å)	3.20	1.65
No. reflections	68,668	319,139
<i>R</i> _{work} / <i>R</i> _{free} (%)	19.1/25.3	17.8/20.1
No. atoms	17821	13319
Protein	8837	6428
Ligand/ion	154	0
Water	23	597
<i>B</i> -factors	88.7	28.3
Protein	88.7	27.7
Solvent	46.9	34.80
R.m.s. deviations		
Bond lengths (Å)	0.004	0.004
Bond angles (°)	0.735	0.967
Ramachandran		
Most favored regions (%)	92.5	96.8
Additional allowed regions (%)	7.0	2.8
Disallowed regions (%)	0.5	0.4

* Values in parentheses are for highest-resolution shell.

The antigen-binding fragment (Fab) of CH103 was screened for crystallization, either by itself or in complex with various strains of HIV-1 expressed with an extended gp120 core¹, which had been deglycosylated to protein-proximal N-acetyl glucosamines². Crystals of Fab CH103 by itself diffracted to 1.6-Å resolution, and the Fab CH103 structure was solved by molecular replacement and refined to *R*_{crystal}/*R*_{free} of 17.9%/20.1%.

¹Kwon YD, *et al.* (2012) Unliganded HIV-1 gp120 core structures assume the CD4-bound conformation with regulation by quaternary interactions and variable loops. *Proc Natl Acad Sci U S A* 109(15):5663-5668.

²Kwong PD, *et al.* (1999) Probability analysis of variational crystallization and its application to gp120, the exterior envelope glycoprotein of type 1 human immunodeficiency virus (HIV-1). *J Biol Chem* 274(7):4115-4123.

Table 9. Comparison of interactions between HIV-1 gp120 and CD4, CH103 and other CD4-binding site antibodies.

HIV-1 gp120			Residue-by-residue binding surface on HIV-1 gp120 (Å ²) *								
Region	Residue		CD4 (2NXY)	CH103 (XXXX)	b12 (2NY7)	b13 (3IDX)	F105 (3H11)	VRC01 (3NGB)	VRC03 (3SE8)	VRC- PG04 (3SE9)	NIH45- 46 (3U7Y)
	Number	Type									
α1	49	D									4.8
	96	W									7.1
	97	K						26.4	41.0	10.5	44.0
	99	D									22.1
	102	E									33.8
	105	H				2.9					
	108	I				2.8					
	109	I				28.7					
V1/V2	112	W				69.9					
	122	L								3.1	3.3
	123	T							8.5		5.6
	124	P	39.9					36.8	81.7	31.2	33.8
	125	L	6.7								
	126	C	61.5								
	127	V	30.8								
	196	C	5.5								
β4/5	198	T						12.7	51.3		
	199	S							13.4		
	210	F					5.3				
Loop B	226	L					11.8				
	244	T					5.7				
	255	V					18.1				
Loop D	256	S		56.6							
	257	T			15.0		10.9		6.9		
	275	V								26.2	11.1
	276	N						22.6	13.3	17.7	9.9
	278	T						84.4	73.0	35.2	36.7
	279	D	18.5	3.9			6.6	56.5	36.5	51.7	57.5
	280	N	51.1	94.9	13.0		14.3	70.2	76.3	72.0	69.7
	281	A	74.7		29.8	16.7	52.2	69.6	70.8	74.0	75.7
β15/ α3 CD4- binging loop	282	K	31.6					30.5	17.2	31.9	49.7
	283	T	18.2			12.6		11.9	8.2	10.6	5.2
	364	S		18.9							
	365	S	65.6	85.9	38.3	11.5	25.5	61.5	58.0	46.0	59.1
	366	G	24.6	16.7	44.5	29.5	53.8	22.0	23.3	22.0	21.1
	367	G	38.6	71.1	74.4	61.7	37.4	24.1	26.4	22.9	26.2
	368	D	69.5	64.0	64.6	87.0	87.3	48.6	54.2	51.2	47.4
	369	P		19.4	59.7	62.9	16.6				
β17	370	E	14.7	30.1	14.7	26.4	53.7		16.0		
	371	I	39.6	39.8	80.8	61.8	47.5	44.1	35.2	44.8	60.4
	372	V			30.9	25.8					
	373	T			17.3	11.7					
	375	S				9.2	7.0		3.3		
β18	382	F					36.2				
	384	Y			4.6	16.2	7.2				
	386	N			35.9	2.8					
β18	417	P			16.3						
	418	C			2.2						
	419	R			84.7	64.8					

Table 9 continued:

HIV-1 gp120			Residue-by-residue binding surface on HIV-1 gp120 (Å ²) *								
			HIV-1 gp120 interacting molecule (PDB code)								
Region	Residue		CD4 (2NXY)	CH103 (XXXX)	b12 (2NY7)	b13 (3IDX)	F105 (3H11)	VRC01 (3NGB)	VRC03 (3SE8)	VRC- PG04 (3SE9)	NIH45-46 (3U7Y)
	Number	Type									
β20/21 Bridging sheet	421	K				40.2	54.1				
	424	I					4.3				
	425	N	24.5			60.7	18.2		12.1		
	426	M	14.4			12.5	79.1		7.1		
	427	W	28.2					7.6	35.1		12.8
	428	Q						4.7			
	429	K	14.9				63.0	2.1	49.7	47.6	23.7
	430	V	111.5		31.1			58.3			57.3
	431	G			13.2				13.8	7.0	11.3
	432	K			47.8						8.0
β 23	455	T	15.5	18.6	28.3	3.5	9.6	31.2	24.0	32.9	31.9
	456	R	3.6		5.4			5.8	6.4	2.4	6.8
	457	D	37.4	49.1				46.4	43.7	27.1	45.8
Loop V5	458	G		32.5				50.5	35.2	39.4	44.9
	459	G	32.2	48.9				69.1	62.9	56.8	68.8
	460	N	64.8	53.3				37.1	63.9		24.1
	461	S		66.1				67.8	51.2	66.3	54.9
	462	N		36.2					28.9	26.7	6.6
	463	N		16.9				13.7	15.4		10.2
β24	465	S						9.7	9.4		8.7
	466	G						6.0			2.1
	467	I						11.3	15.9		17.8
	469	R	13.5	59.4				23.3	21.8	17.2	21.0
Outer domain exiting loop	471	G		4.2			4.2				
	472	G	20.5	6.6	20.5	6.3		8.4	23.7	3.6	4.8
	473	G	23.4		53.5	23.8	50.2	27.6	29.2	18.3	22.8
	474	D	37.2		26.0	25.3	43.9	17.3	3.0	7.6	30.5
	475	M	2.8		65.0		33.6				4.2
α5	476	R	9.3				40.9			21.9	24.8
	477	D	3.7				3.1				
	480	R									16.4

*: Residues with interacting surface area less than 2.0 Å² are not listed.

Table 9 discloses the residues at positions 122-127, 278-283, 364-373, 424-432, 458-463 and 471-475 as SEQ ID NOS 914-919, respectively.

Table 10. Interface between antibody CH103 and ZM176.66 gp120.**Supplementary Table 10a, Total buried surface areas across the interface of CH103 and HIV-1 gp120.**

Antibody	Interface on gp120 (Å ²)	Interface on CH103 (Å ²)	
		Total antibody area	Area contributed by UCA residues (% total)
Heavy chain	429	493	414 (84%)
Light chain	378	377	164 (44%)
Total	807	870	578 (66%)

Table 10b, Residue-by-residue buried surface area of gp120 residues that interact with CH103.

gp120 residue			Heavy chain interactions		Light chain interactions	
Region	Number	Type	Bond type*	Surface area (Å ²)	Bond type	Surface area (Å ²)
Loop B	256	SER	H	11.72		
Loop D	279	ASP				3.56
	280	ASN		12.38	H	39.83
CD4-binding loop	364	HIS		13.21		3.56
	365	SER	H	53.96		31.86
	366	GLY		17.71		3.61
	367	GLY	H	68.46		
	368	ASP	HS	65.44		
	369	LEU	H	18.72		
	370	GLU	HS	28.69		
	371	ILE		38.90		
Loop V5	455	THR		18.59		
	457	ASP		35.32		13.05
	458	GLY		9.87	H	24.12
	459	GLY				50.36
	460	ASN		50.77		50.77
	461	ASP			HS	69.50
	462	ASP				36.60
	463	ASN				15.46
• 24	469	ARG		23.84		34.44
	471	GLY		5.35		
	472	GLY		5.85		

* Bond type: H: Hydrogen, S: Salt bridge.

Detailed gp120:CH103 interface data was calculated on the EBI PISA server.

Table 10b discloses the residues at positions 364-371 and 457-463 as SEQ ID NOS 920-921, respectively.

Table 10c. Residue-by-residue buried surface areas of the CH103 paratope residues.

Chain	Region	Residue		Bond type*	Buried surface area (Å ²)	Contribution by Region (%)
		Number (Kabat)	Type			
H	CDR H1	33	TYR	H	32.31	3.7
H	CDR H2	50	TYR	H	21.67	11.8
H		52	PHE		12.11	
H		54	THR		7.68	
H		56	GLU	H	59.46	
H		58	ASN		2.01	
H	CDR H3	97	ARG	HS	82.19	41.1
H		98	GLY		23.45	
H		99	GLN	H	120.00	
H		100	LEU	H	27.19	
H		100A	VAL	H	55.44	
H		100B	ASN		50.10	
L	CDR L1	27	SER		3.68	8.0
L		31	THR		14.17	
L		32	ASN	H	52.10	
L	CDR L2	50	GLU	H	38.67	14.0
L		51	ASN	H	27.91	
L		52	TYR		32.84	
L		53	LYS	H	22.45	
L	FWR L3	65	SER		9.04	13.1
L		66	LYS	HS	43.83	
L		67	SER		25.47	
L		68	GLY		36.10	
L	CDR L3	91	TRP		70.62	8.1

* Bond type: H: Hydrogen, D: Disulphide bond, S: Salt bridge, C: Covalent link.

Detailed gp120:CH103 interface data was calculated on the EBI PISA server

Table 10c discloses the residues at positions 97-100B, 50-53 and 65-68 as SEQ ID NOS 922-924, respectively.

Table 11. Hydrogen bonds and salt bridges between CH103 and ZM176.66 gp120.

Hydrogen bonds							
Antibody CH103				Distance (Å)	gp120		
Chain	Number	Type	Atom		Atom	Type	Number
H	33	TYR	OH	2.17	OD1	ASP	368
H	50	TYR	OH	3.68	O	GLY	367
H	56	GLU	OE1	3.20	N	LEU	369
H	97	ARG	NH1	3.81	OG	SER	256
H	97	ARG	NH2	2.59	OE1	GLU	370
H	99	GLN	NE2	3.10	OG	SER	365
H	100	LEU	N	2.41	O	SER	365
H	100	LEU	N	3.22	OG	SER	365
H	100A	VAL	N	3.18	OG	SER	365
L	32	ASN	ND2	2.97	O	GLY	458
L	50	GLU	OE2	3.07	ND2	ASN	280
L	51	ASN	ND2	2.92	OD1	ASP	461
L	53	LYS	NZ	3.19	OD1	ASN	280
L	66	LYS	NZ	3.16	OD2	ASP	461

Salt bridges							
Antibody CH103				Distance (Å)	gp120		
Chain	Number	Type	Atom		Atom	Type	Number
H	97	ARG	NE	3.93	OD1	ASP	368
H	97	ARG	NE	2.58	OD2	ASP	368
H	97	ARG	NH2	2.59	OE1	GLU	370
H	97	ARG	NH2	2.73	OD2	ASP	368
L	66	LYS	NZ	3.65	OD1	ASP	461
L	66	LYS	NZ	3.16	OD2	ASP	461

Table 12. Residue-by-residue specification of unmutated versus mutated residues on antibody CH103.

Heavy chain mutations					
Region	ID	UCA	Mature	Paratope	Description and note
FWR 1	11	L	V		Shortens side chain on strand A
	14	P	S		Alters loop between strands A and B
	29	I	M		Enhances interactions with heavy chain Trp34
	30	S	G		Increases loop flexibility
CDR 1	31	S	G		Increases CDR I flexibility
	32	Y	T		Avoids clash with other heavy chain residues
FWR 1	37	I	L		Neutral mutation
	39	Q	L		Alters heavy/light chain interface
	40	P	S		Allows flexibility in strand C
CDR 2	52	Y	F	Yes	Polar to hydrophobic
	53	Y	H	Yes	Polar to basic
	54	S	T	Yes	Adds carbon to paratope interface
	56	S	E	Yes	Forms hydrogen bond with backbone amide of Leu369 in the CD4-binding loop
	60	N	S		Alters heavy/light chain interface
	65	S	G		Increases flexibility in loop between strand C' and D
FWR 3	68	T	S		Avoids clashes with neighboring residues
	75	K	E		Basic to acidic change in loop between strand D and E
	76	N	D		Polar to acidic change at the beginning of strand E
	81	K	R		Neutral mutation
	82A	S	R		Polar to basic change at C terminus of strand E
	91	Y	F		Polar to hydrophobic change at the heavy/light chain interface
CDR 3	101	D	R		Acidic to basic change at the first layer adjacent to paratope
	102	Y	N		Smaller side chain at the end of CDR H3
FWR 4	105	Q	R		Alters heavy/light chain interface
	107	T	S		Avoids clashes with neighboring residues
	110	T	S		Alters heavy/light chain interface
	112	S	T		Minor change at the end of strand G
	113	S	A		Avoids clashes with neighboring residues

(Table 12 continues to next page)

Table 12, continued:

Table 12 discloses residues 26-27C as SEQ ID NO: 925.

Light chain mutations

Region	ID	UCA	Mature	Paratope	Description and note
FWR 1	20	S	T		Surface residue in strand B
	26	D	A		Avoids clashes with neighboring residues
	27	K	.		Deletion reduces potential clashes with HIV-1 gp120
	27A	L	.		Deletion reduces potential clashes with HIV-1 gp120
	27B	G	.		Deletion reduces potential clashes with HIV-1 gp120
CDR 1	27C	D	S	Yes	Acidic to polar change
	31	K	T	Yes	Shorter side chain reduces potential clashes with HIV-1 gp120
	32	Y	N	Yes	Smaller side chain reduces potential clashes with HIV-1 gp120
	33	A	V		Bulker side chain increases packing of light chain core
	38	Q	V		Alters heavy/light chain interface
FWR 2	45	V	E		Surface residue at strand C'', hydrophobic to acidic change
	46	L	V		Alters heavy/light chain interface
	49	Y	F		Alters heavy/light chain interface
	50	Q	E	Yes	Forms hydrogen bond with Asn280 in gp120 loop D
CDR 2	51	D	N	Yes	Forms hydrogen bond with Asn461 in gp120 loop V5
	52	S	Y	Yes	Enhances interactions with gp120 loop D
	60	E	D		Surface residue in loop between strands C'' and D
	66	N	K	Yes	Forms hydrogen bond and salt bridges with Asn461 in loop V5
FWR 3	69	N	S		Shortens side chain in loop between strands D and E
	76	S	R		Polar to basic change and longer side chain at C terminus of strand E
	81	M	I		Shortens side chain in loop between strands E and F
CDR 3	90	A	V		Bulker side chain increases packing of light chain core
FWR 4	100	T	S		Neutral mutation in strand G and near heavy/light chain interface

To determine the frequency of germline antibodies that could potentially serve as unmutated common ancestors of a lineage line CH103, we have interrogated a combined dataset of 454 pyrosequences of three HIV infected subjects unrelated to the CH505 patient. Gene segment frequencies in this dataset demonstrate that the frequency of the VH4-59 gene is 4.2%, the JH4 is 49.7% and the frequency of HCDR3 length of the CH103 VH length (a 15mer) is 8.9%. The proportion of sequences with all three characteristics, if independent is VH4-59/JH4/CDR3 Length=15 is 1/540 with the actual count in the analyzed data set of the combination = 637/386853 = 1/607.

This frequency is clearly very common. The question that remains regards the prevalence of the relevant characteristics of CDR3.

For example, the HC CDR3 contact residues (from figure 4 of the paper) are RGQLVN (SEQ ID NO: 926) starting at position 4 in HCDR3 with the following conservative substitutions:

R: K; G: A; Q: E; L: I, V; V: I, V; N: D

We therefore use the HCDR3 motif: XXX(R/K)(G/A)(Q/E)(L/V/I)(L/V/I)(N/D)nX, and scanned our pyrosequencing heavy-chain dataset for its occurrence. This motif occurred 10 times among the 337567 in-frame HCDR3 in our pyrosequencing database.

If we allow positions other than the fourth (which contains the R/K necessary for the salt bridge) to vary we obtain the table below. The number of positions at which the observed HCDR3 differs from the CH103 HCDR3

the table below. The number of positions at which the observed HCDR3 differs from the CH103 HCDR3 motif is on the left, and the number out of 337567 HCDR3 sequences is on the right. All of the CDR3 in this table have R or K at position 4.

distance	number of sequences out of 337567
0	10
1	71
2	1028

An appropriate light-chain UCA is also likely to be readily available. We downloaded 2312 rearranged human lambda V-region sequences from Genbank and analyzed them for comparison. The CH103 light chain uses IGLV3-1 and IGLJ1. These genes are found in 9.6% and 15.5% respectively of all sequences in the Genbank lambda database. The CH103 light chain is 30 nt long, as are 23.7% of the Genbank lambdas. The single contact residue in the light-chain CDR3 is tryptophan at the 3rd CDR3 position, which is encoded by the IGLV gene. Indeed 43% of all Genbank lambda chains have W at position 3 of CDR3. Thus, there is considerable evidence that the germlines of the CH103 lineage are relatively common by a variety of criteria.

Table 13. Localization of sites under positive selection using the fixed effects likelihood (FEL)¹ (p-value < 0.10) and the mixed effects model of evolution (MEME)² (q-value < 0.1).

Week after infection	Using FEL					Using MEME					
	n Neg ^a	n Pos ^b	CH103 n Neg ^c	Non-CH103 n Pos ^d	Fisher's p (n Pos) ^e	Fisher's p (n Neg) ^f	Substitution per site ^g	selected sites	Positively selected sites inside CH103 footprint	Positively selected sites outside CH103 footprint	Fisher's exact p value
4	0	6	0	0	na	1	0.0035	0	0	0	na
14	0	14	0	0	na	1	0.0095	0	0	0	na
20	5	20	3	2	0.009	0.71	0.022	3	2/92	1/830	0.05
30	8	32	4	4	0.005	0.36	0.04	5	3/92	2/830	0.009
160	36	88	11	25	0.0004	0.6	0.057	34	11/92	23/830	0.0002

^a Number of positively selected sites; ^b Number of negatively selected sites; ^c Number of positively selected sites among 92 sites inside CH103 binding regions (footprint); ^d Number of positively selected sites among 830 sites not in CH103 footprint; ^e P value from Fisher's exact test for positively selected sites inside vs. outside CH103 footprint; ^f P value from Fisher's exact test for negatively selected sites inside vs. outside CH103 footprint; and ^g Per-site substitution rate among 922 aligned sites.

The 922 codons in the CH505 alignment were considered as 2 sets: 92 codons (10%) were included in the candidate regions for CH103 selection (CH103, CD4, and VRC01 contact residues, as well as V1 and V5 hypervariable loops which border these contacts), and the 830 other codons remaining in the alignment. We used FEL¹ and MEME² methods to quantify selection in the CH505 codon-aligned sequences, implemented through the HyPhy package at the DATAMONKEY website. The full alignment was used for the initial analysis, and the codon sets defined above were used to see if positive selection was concentrated in the CH103 contact/CD4bs region. We used the strategy implemented at the DATAMONKEY web site to select optimal substitution models, with a p < 0.10 cutoff as evidence suggesting positive selection for the FEL model, and a q < 0.10 cutoff for the MEME model. Analysis by using both FEL and MEME methods showed that positive selection was enriched in CH103 binding regions by week 20, and this focus continued throughout the course of the study, through week 160. Fisher's exact test was used to test the null hypothesis that the positively selected sites are evenly distributed throughout Env; they are not, and are enriched in the CH103 region. In contrast, the number of sites under negative selection was evenly distributed between the two regions. The amino acids that are changing in the regions of interest for CH103 escape are shown in Fig 5. At week 4, using FEL¹ and MEME², there was no statistical evidence for positive selection anywhere in the CH505 codon-aligned sequences, though there was evidence for negative selection at 6 positions with p values below the cutoff. However, FEL and MEME will underestimate positive selection within a subject, as the frequencies of identical sequences are not considered, and thus changes in population frequency are not considered positive selection. Given this, it is of note that in the week-4 sample, a single mutation in the full alignment of 55 sequences occurred more than once, and it was a N279K change in Loop D, found in 5 of the 55 sequences. There was also one instance of a short (7 residue) in-frame deletion spanning this position. This would produce just one ancestral change in the phylogenetic tree, so it could not provide statistical evidence of selection, but still coincidence of facts makes it of interest: 279 is located in a key contact position for CH103 in Loop D, in a region under clear strong subsequent selective pressure. Neighboring positions are mutating by week 14, a further indication that local positive selection might be underway, leaving open the possibility that these sites may be targeted by the CH103 lineage very early in infection. Codon models also do not take into account insertions and deletions, an essential aspect of HIV env evolution, which is evident in CH505 in V1 by week 20.

1. Kosakovsky Pond, S.L. & Frost, S.D. Not so different after all: a comparison of methods for detecting amino acid sites under selection. *Molecular biology and evolution* 22, 1208-1222 (2005).

2. Murrell, B., et al. Detecting individual sites subject to episodic diversifying selection. *PLoS genetics* 8, e1002764 (2012).

Table 14. Autologous and heterologous neutralization activity of CH103 clonal lineage antibodies.

Antibody ID	Neutralization activity (IC50, ug/ml)								MuLV
	C.CH505 T/F	Heterologous viruses							
		B.SF162	B.JRFL	A.Q168	A.Q842	B.BG1168	C.ZM106		
UCA	> 50	> 50	> 50	> 50	> 50	> 50	> 50	> 50	
18	49.4	> 50	> 50	> 50	> 50	> 50	> 50	> 50	
17	14.7	> 50	> 50	> 50	> 50	> 50	> 50	> 50	
14	10.9	> 50	> 50	> 50	> 50	> 50	> 50	> 50	
1AZCET15	12.8	> 50	> 50	> 50	> 50	> 50	> 50	> 50	
1A102RI6		> 50	> 50	> 50	> 50	> 50	> 50	> 50	
1AH92U	> 50	22.0	> 50	> 50	> 50	> 50	> 50	> 50	
13	6.2			24.1	> 50	42.6	> 50	> 50	
12						14.7	17.0	> 50	
11						15.4	19.4	> 50	
CH103						17.7	12.8	> 50	
CH104						13.4	12.0	> 50	
CH105					5.5	40.0	28.5	> 50	
CH106						8.2	8.1	> 50	

>50	>20 - <50	>10 - <20	>5 - <10
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>50	>20 - <50	>10 - <20	>5 - <10
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Table 15. Alignment of gp160 Env sequences of CH505 transmitted/funder (T/F) and tested heterologous HIV-1 viruses.

C. CH0505T/F	MRVMGIQRNY	PQW	WIWSML	GFWMLMICNG	...MWTVVY	YGVFVWKEAK	TTLFCASDAK	
A. Q168	-K-R--K--L	...	-K-GTM	LLG---	TVSV	.AEQL---	-----D-E	
A. Q842	--A---M-C	QNL	-R-GTM	ILG-IIF-SA	.VDNL---	-----E		
B. BG1168	---K--MK-C	QHL	-R-GIM	LLGI---	SA	.TEKL---	-----T	
B. JRFL	---K--RK--	QHL	-RGGT-	LLGIIV--SA	.VEKL---	-----T		
B. SF162	---K--RK--	QHL	-RGGT-	LLG-----SA	.VEKL---	-----T		
C. ZM106	-K-RE-L--W	R--	---GI-	-----V	VVGNL---	-----		60
B. HXB2	---KEKYQHL	WR	-G-R-GTM	LLG-----SA	.TEKL---	-----T		
C. CH0505T/F	AYEKEVHNW	ATHACVPTDP	NPQEMVLKNV	TENFNWKNKD	MVDQMHEVDI	SLWDQSLKPC		
A. Q168	--ST-K--I-	-----	---IH-E-	--E-----N	-E--T-I-	-----R-		
A. Q842	---T-K---	-----	---IH-E-	--E-----N	-E--T-I-	-----		
B. BG1168	---T-----	-----	---VK-E-	--D--V---	-T-----I-	-----		
B. JRFL	--DT-----	-----	---V--G-	--K-----N	-E--Q--I-	-----		
B. SF162	--DT-----	-----	---I--E-	-----N	-E-----I-	-----		
C. ZM106	---R-----	-----	S-----E-	-----	-----I-	-----		120
B. HXB2	--DT-----	-----	---V--V-	-----	-E-----I-	-----		
C. CH0505T/F	VKLTPLCVTL	NCTNA	T...A	SNSSI...I.	EGM	KNCSPNITTE		
A. Q168	-----	---VN	...NNT	...N V-NNT...G.	WD-ER	-----		
A. Q842	-----	D-N-VT	...NNG-	...S DM.....R.	EI	-----M-		
B. BG1168	-----	H--DVNTTCI	TTNNS-MTNS	---TEGNCS	SYNYNGR-EL	R-----S		
B. JRFL	-----	--KDV	...NA-NTTN	GSEGTM....	ERGEI	-----S		
B. SF162	-----	H--L	...KNA-NTKS	--WKEM....	DRGEI	---KV--S		
C. ZM106	-----	K-V-V	...NA-SKSN	ASATNDG...	SGE-	---T-----		
B. HXB2	-----S-	K--DL	...KND-NTNS	-SGRMIM..E	KGEI	-----S-S		180
C. CH0505T/F	LRDKREKKNA	LFYKLDIVQL	...DGNSSQ.	.YRLINCNTS	VITOACPKVS	FDPIPIHYCA		
A. Q168	-----Q-VYS	-----V-I	DN-S.	-----A	-----T	-E-----		
A. Q842	-----Q-VYS	-----I	NEDQ-----NN	K-----T	A-----T	-E-----		
B. BG1168	IQ--VQ-DY-	I-----PI	KSDNSDNTS.	-----	-----I-	-E-----		
B. JRFL	I--EVQ-EY-	-----V-PI	...N-NTS.	-----S-D-	-----I-	-E-----		
B. SF162	I-N-MQ-EY-	-----V-PI	...NDNTS.	.-K-----	-----I-	-E-----		
C. ZM106	I---KRNES-	-----P	...TNDNNSG	E-----AM	-----	-----		
B. HXB2	I-G-VQ-EY-	F-----IPI	D...NDTTS.	.-K-TS---	-----	-E-----		240

C.CH0505T/F	PAGYAILKCN	NKTFTGTGPC	NNVSTVQCTH	GIKPVVSTQL	LINGSLAEGE	IIIRSENITN	300
A.Q168	---F----	K DEK-N----	K-----	-----	-----K-	VM-----F--	
A.Q842	---F----	K DEE-N-I----	K-----	-----	-----K-	VK--C-----	
B.BG1168	---F----	D-K-S-K-T----	-----	-R-----L.T	VV-----EG	VVL-----F--	
B.JRFL	---F----	D--N-K----	K-----	-R-----	-----E-	VV--D-F----	
B.SF162	---F----	D-K-N-S----	T-----	-R-----	-----EG	VV-----F-D	
C.ZM106	-----	---N-----	Y-----	-----	-----	-----L-D	
B.HXB2	---F----	---N-----	T-----	-R-----	-----E-	VV--V-F-D	
C.CH0505T/F	NVKTIIIVHLN	ESVKIECTRP	NNKTRTSIRI	..GPGQAFYA	TGQVIGDIRE	AYCNINESKW	360
A.Q168	-A-N-L-QFK	-P--N-----	D-N-----	..-----	..I-----Q	---TV-G-E-	
A.Q842	-A-----Q-V	NP--N-----	-N-K-H----	..-----	---DI-----Q	-H--V-RTE-	
B.BG1168	-A-----Q-K	DP--E-----	-N-IK--HL	..---R-WH-	---I-----K	-F-TL-STN-	
B.JRFL	-A-----Q-K	--E-N-----	-N-K-H----	..---R--T-	---EI-----Q	-H--SRA--	
B.SF162	-A-----Q-K	--E-N-----	-N-K-K-T-	..---R--T-	---DI-----Q	-H--SGE--	
C.ZM106	-----	--IH-T-----	-N-K-----	..---T-----	---EI-----K	-----S-E--	
B.HXB2	-A-----Q--	T--E-N-----	-N-KR----	QR---R--VT	I-K--NM-Q	-H--SRA--	
C.CH0505T/F	NETLQVSKK	LKEYFP.HKN	ITFQPSSGGD	LEITTHSFNC	GGEFFYCNTS	SLFNRTYMAN	420
A.Q168	-KA--K-VEQ	-RSS-E.N-T	-I-AN-----	-----	-----	G--DS-WNDT	
A.Q842	-N--HQ-VEQ	-RKH-.N-T	-N-AN-T----	-----	-----T	N--S-WNHT	
B.BG1168	TN--KQMEV-	-R-Q-E.N-T	-A-NQ-T----	P--VM-T----	-----T	Q--SIWYNT	
B.JRFL	-D--KQIVI-	-R-Q-E.N-T	-V-NH-----	P--VM-----	-----ST	Q--S-WNN-	
B.SF162	-N--KQIVT-	-QAQ-G.N-T	-V-KQ-----	P--VM-----	-----ST	Q--S-WNNT	
C.ZM106	-KA--E-G--	--H--N-T	-K-A-----	-----	R-----	K--S--H-	
B.HXB2	-N--KQIAS-	-R-Q-GNN-T	-I-KQ-----	P--V-----	-----ST	Q--S-WFNS	
C.CH0505T/F	STDMANSTET	NSTRTITIHC	RIKQIINMWQ	EVGRAMYAPP	IAGNITCISN	ITGLLLTRDG	480
A.Q168	DSR	QENG--LP-	-----	RT-Q-I----	-Q-A-R-V--	----I-----	
A.Q842	A.....SMNS	-E-ND--ILP-	-----	R-Q-----	-R-V-R-E-	----I-----	
B.BG1168	T.NSSWNNK-	-W-NN--LP-	-----	Q-K-I----	-K-K-K----	-----	
B.JRFL	T....EG-NN	-EGN--LP-	-----	--K-----	-R-Q-R-S-	-----	
B.SF162	IGPN	-TNG--LP-	-----R-	--K-----	-R-Q-R-S-	-----	
C.ZM106	A.....TSRN	-ATNA--LP-	--R-----	-----	-----V--	-----	
B.HXB2	TWSTEG-NN-	-EGSD--LP-	-----	K-K-----	-S-Q-R-S-	-----	
C.CH0505T/F	GK...NNTET	FRPGGNMKD	NWRSELYKYK	VVEVKPLGVA	PTNARRRVE	REKRAVGMGA	

A.Q168	-NN.NSTN--	-----D-R-	-----KIE-----	-----K-K-----G	-----I--	
A.Q842	-NT.NSTR--	-----D-R-	-----KIE-----	-----K-K-----	-----I--	
B.BG1168	-DT.N-G-I	-----D-R-	-----QIE-----	-----K-K-----Q	-----L--	
B.JRFL	-IN.E-G-I	-----D--	-----KIE-----	-----K-K-----Q	-----I--	
B.SF162	-EIS-T-I	-----D-R-	-----KIE-----	-----K-K-----Q	-----TL--	
C.ZM106	-NGDT-D--	-----D-N	-----I-----	-----E-K-----	-----I--	
B.HXB2	-NS.N-ES-I	-----D-R-	-----KIE-----	-----K-K-----Q	-----I--	540
C.CH0505T/F	VFLGFLGAAG	STMGAASITL	TVQARQLLSG	IVQQQSNNLK	AIEAQQHMLK	LTVWGIKQLQ
A.Q168	-----I--	-----	-----	-----L-R	-----	
A.Q842	M-----	-----	-----R	-----L--	-----	
B.BG1168	M-----	-----M-	-----N-N-R	-----L-Q	-----	
B.JRFL	M-----	-----L-	-----N-N-R	-----R-Q	-----	
B.SF162	M-----	-----L-	-----N-N-R	-----L-Q	-----	
C.ZM106	L-----	-----A--V-	-----R	-----L-Q	-----	
B.HXB2	L-----	-----M-	-----N-N-R	-----L-Q	-----	600
C.CH0505T/F	ARVLALERYL	KDQQLGMMWG	CSGKLICTTN	VYWNSSWSNK	TYGDIWDNMT	WMQWEREISN
A.Q168	-----V--	-----I-	-----P-----	-----SQSE-E--	-----L--K----	
A.Q842	-----V--	-----I-	-----S	-----SQNE----	-----L--DK----	
B.BG1168	-----V--	-----I-	-----A	-----SQEE--L-	-----K--N-	
B.JRFL	-----V--	G-----I-	-----A	-----SLDR-N--	-----E-----D-	
B.SF162	-----V--	-----I-	-----A	-----SLDQ-N--	-----E-----D-	
C.ZM106	T-----	-----L-	-----R	-----SLT-----	-----DK-V--	
B.HXB2	-I--V----	-----I-	-----A	-----P-A-----	-----SLEQ--NHT-	-----E-D--N-
C.CH0505T/F	YTEIYYELLE	ESQNOQEKNE	QDLLALDRWN	SLWNWFNITN	WLWYIKIFIM	IVGGLIGLRI
A.Q168	--Q--T-I-	-----	-----K-A	-----D-SK	-----R----	
A.Q842	--Q--D--	-----	-----K-A	-----D-S	-----	
B.BG1168	--SV--T--	Q-----	E-E-K-A	-----D-K	-----R----	
B.JRFL	--SE--T-I-	-----	E-E-K-A	-----D-K	-----	
B.SF162	--NL--T-I-	-----	E-E-K-A	-----D-SK	-----V----	
C.ZM106	--NT--R--	D-S-----	K-----S-K	N--T--D-S-	-----V-----	
B.HXB2	--SL-HS-I-	-----	E-E-K-A	-----	-----L----	720
C.CH0505T/F	IFAVLSLVNR	VRQGYSPSL	QTLIPSPRGP	DRPGIEEEG	GEQDRNRSTR	LVSQFLALVW
A.Q168	V-----V--	-----F	-----L-A----	-----D-----	-----G-G--RQ	-----N--ST-I-

A.Q842	V-----VI--	-----F	-----HT-N--L	-----ER--	-----K---I-	-----A-
B.BG1168	V---I-I---	-----	-----RF-A---	-----E-----	-----GR---I--	-----P-I-
B.JRFL	V-T---I---	-----F	-----L-A---	-----E-----	-----R-D-G-	-----N-----I-
B.SF162	V-T---I---	-----F	-----RF-A---	-----E-----	-----R-D-SP	-----H-L---I-
C.ZM106	-----I-I---	-----	-----TQ-G-	-----L-R-----	-----D-I-	-----N---T-A-
B.HXB2	V-----I---	-----F	-----HL-T---	-----E-----	-----R-D-I-	-----N-S---I-
						780
C.CH050ST/F	DDLRSCLFI	YHRLRDFILI	AARAGELLGR	SSLKGLRRGW	EALKYLGSLV	QYWGLELKR
A.Q168	-----N---S	-----L---	-----IV---		-----I---WN-L	-----IQ---N-
A.Q842	-----S	-----	V-TV-----H	-----L-	-----G-----N-L	S---R-RI-
B.BG1168	-----S	-----L---	V-IV-----		-----WN-L	-----SQ---N-
B.JRFL	V-----S	-----LL-T	VT-IV-----		-----V---WN-L	-----SQ---N-
B.SF162	-----S	-----L---	IV-----		-----W-N-L	-----IQ---N-
C.ZM106	-----S	-----	VV-----H	-----R-QK-	-----	-----C-----
B.HXB2	-----S	-----LL-	VT-IV-----		-----WN-L	-----SQ---N-
						840
C.CH050ST/F	AISLDTLAI	AVCEGTDRIL	EFVLGICRAI	RNIPTRIRQG	PETALL	
A.Q168	-----N-T--	-----A-	IIQRAIT-V	L-----	-----R---	(SEQ ID NO: 15)
A.Q842	-TN-----I-	VIAGW---VI	IGQRL---F	L---R---	-----R---	(SEQ ID NO: 16)
B.BG1168	-V---N-T--	V-A-----I	ALQOR---	LH-----	-----R---	(SEQ ID NO: 17)
B.JRFL	-V---NAT--	-----A-	ALQRTY---	LH-----	-----L-R---	(SEQ ID NO: 18)
B.SF162	-V---F-AI--	-----A-	VAQR-G--F	LH-R---	-----R---	(SEQ ID NO: 19)
C.ZM106	-----SI-M	-----A-	L-QR---G-	YH-R---	-----A---	(SEQ ID NO: 20)
B.HXB2	-V---NAT--	-----A-	V-Q-A---	H-R---	-----L-RI--	(SEQ ID NO: 21)
					886	(SEQ ID NO: 22)

EXAMPLE 2

Shown in Fig. 19 are CH505 Env sequences for a multivalent-valent vaccine that can be made both with RNAs (Geall et al, Proc. Natl. Acad. Sci. 109: 14604-14609 (2012)) and DNAs (Ledgerwood, et al. Clin Vaccine Immunol. 19:1792-7 (2012)) as gp160s for genetic immunization and as well made as gp160s and gp140s (Liao et al, Nature 201: 469-76 (2013)) for poxvirus vector immunizations in ALVAC (canary pox) vectors such as was used in RV144 (Rerks-Ngarm et al. NEJM 361:2209-2220 (2009)) and NYVAC that either are replicating (such as NYVAC-KC, Kibler et al, PLoS One 6: e25674, Epub 2011, Nov 9) or non-replicating (such as NYVAC-C, Perreau et al, J. Virol. 85: 9854-62 (2011)). Criteria for choosing the Envs was based on the following criteria: (i) the expressed Envs optimally bound to members of the CH103 BnAb lineage, or (ii) the viruses with these Envs escaped the CH103 lineage and, therefore, were involved in its early stimulation, or (iii) the viruses with these Envs did not escape from the CH103 lineage and, therefore, were able to continue to stimulate the later stages of the CH103 lineage, or (iv) the viruses with these Envs were hypersensitive to neutralization by the CH103 lineage and, therefore, were able to optimally drive the CH103 lineage.

EXAMPLE 3

The HIV-1 arms race in patient CH0505, in which CD4 binding site BnAbs develop over time (clonal lineage under “antibody”) in response to HIV-1 virus evolution (virus evolution tree under “HIV-1”), is shown in Fig. 20. In the clonal lineage shown, env binding to the heterologous 63521 clade B transmitted founder Env increased 4 logs over the time of clonal lineage development.

Figure 21 shows the same virus clonal lineage tree of CH0505 and shows at the stars on the right panel examples of sequential envs chosen for

immunogens. The stars on the tree in the left panel are env sequences shown in Fig. 17

The contact regions for CD4, VRC01, and b12, and the signature sites that impact VRC01 and b12 neutralization, are under intense selective pressure in CH0505. Fig. 22 illustrates several points: i) the 110 positions that are in or near the CD4bs are under far more intense selective pressure than the 846 positions that are not in the CD4bs region (see: "in or near CD4b" vs "excluding CD4bs"), ii) using the 10 CHAVI 17 samples that had a 24 week time point (blue), it can be seen that the diversification in or near the CD4bs is strikingly high in CH0505 very early on, already at 24 weeks, iii) using the 9 CHAVI 17 samples that had a sample between 1-2 years (range: 60-96 weeks), it can be seen that the pressure on the CD4bs region is unrelenting, and remarkable compared to other subjects, and iv) population breadth is first apparent at week 92, this autologous pressure first drove extensive diversification in the CD4 region, and then breadth developed in the presence of these diverse forms.

Fig. 23, like Fig. 22, shows how sites within the CD4 binding site of the CH0505 virus sequences are highly mutated in response to the antibodies generated in this patient.

Fig. 24 shows that the single transmitted/founder virus Envelope gp140C of CH0505 binds remarkably well to the unmutated common ancestor of the CH103, 104 and 105 CD4 binding site bnAbs isolated from CH0505, and this env should be able to drive this clonal lineage.

Fig. 25 shows that neutralization arises early on in the clonal lineage at I4 antibody and there are relatively few mutations from the UCA to I4 that an immunogen must induce.

Fig. 27 shows viral evolution during BnAb development in the HIV-1 infected individual CH505.

EXAMPLE 4

Figure 26 shows an Env designed to focus induction of CD4 binding site antibodies by deletion of V1, V2 and V3 loop sequences that were highlighted in red font (underlined) (as example by CH0505_CON gp120) resulting in a core Env as example shown in example as CH0505_DV123core. This strategy can also be applied to the other HIV-1 Envs in the list of evolved CH0505 Envs (evolved CH0505 Envs are Env sequences obtained in sequential times after transmission) and well as the other heterologous HIV-1 Envs.

EXAMPLE 5

BALB/c mice were immunized IM with 25 µg per dose of either the CH505 Transmitted/founder (T/F) Env delta 7 gp120 X4, the week 53.e16 CH505 variant X4, the week 78.33 CH505 variant X4, or the week 100B6 CH505 variant. In addition, BALB/c mice were also immunized IM with sequential Envs T/F, then week 53.e16 Env gp120, then week 78.33 Env gp120, then week 100B6 CH505 gp120 Env. A significant level of CD4 binding site antibodies occurs when a plasma titer of >1:200 to the resurfaced core 3 (RSC3) is present that is > 2.8 times over plasma binding to the RSC3 with an isoleucine deletion at position 371 (Lynch RM et al. J. Virol. 86: 7588-95, 2012). Each group represents the mean of 3-4 mice per group. Data represent the ration of binding RSC# to RSCEDelta 371 proteins expressed as log Area Under the Curve (AUC) RSC3/log AUC RSC3Delta 371. Each animal end point binding titers were >200. Fig. 30 demonstrates that immunization with each individual gp120 alone X4 did not induce antibodies with a ration above 2 except for the week 533nv where the ratio went to ~3. However, the sequential immunization induced RSC3/RSC3D371 ratio of RSC3-binding antibodies of >4 demonstrating the

superiority of this particular combination of antibodies of inducing the desired type of CD4 binding site antibodies over individual Env immunizations.

5

* * *

References:

- Wei et al, Nature 422: 307-12 (2003); McMichael et al, Nature Rev. Immunol. 10:11-23 (2010) Epub 2009 Dec 11; Cohen et al, New Eng. J. Med. 364:1943-54 (2011), Bar et al, PLoS Pathog. 8: e1002721, Epub 2012 May 31; Goonetilleke et al, J. Exp. Med. 206:1253-72 (2009); Keele et al, Proc. Natl. Acad. Sci. 105:7552-7 (2008), Gray et al, J. Virol. 85:4828-40 (2011); Moore et al, PLoS Pathogens 5:e1000598, Epub 2009, Sept 18; Gray et al, J. Virol. 83:11265-74 (2009); Morris et al, PLoS One 6:e23532 (2011) Sept 30; McElrath and Haynes, Immunity 33: 542-54 (2010) and Haynes et al, Nature Biotech. 30:423-33 (2012)).

WHAT IS CLAIMED IS:

1. A composition comprising:

(a) a recombinant HIV-1 envelope protein comprising:

all the consecutive amino acids immediately after the signal peptide
MRVMGIQRNYPQWWIWSMLGFWMLMICNG in the sequence defined by SEQ
ID NO: 879, 861, 884 or 866;

all the consecutive amino acids immediately after the signal peptide
MRVTGIQRNYPQWWIWSMLGLWMLMICNG in the sequence defined by SEQ
ID NO: 889 or 871; or

all the consecutive amino acids immediately after the signal peptide
MKVRGIQRNYPQWWIWSMLGLWMLMICNG in the sequence defined by SEQ
ID NO: 893 or 875; and

(b) a carrier.

2. The composition according to claim 1, wherein the recombinant HIV-1
envelope protein comprises all the consecutive amino acids immediately after the
signal peptide in the sequence defined by SEQ ID NO: 879.

3. The composition according to claim 1, wherein the recombinant HIV-1
envelope protein comprises all the consecutive amino acids immediately after the
signal peptide in the sequence defined by SEQ ID NO: 861.

4. The composition according to claim 1, wherein said recombinant HIV-
1 envelope protein is a purified gp120 HIV-1 envelope protein consisting of all the
consecutive amino acids immediately after the signal peptide in the sequence defined
by SEQ ID NO: 879, 861, 884, 866, 889, 871, 893, or 875.

5. The composition according to claim 4, wherein the purified gp120
HIV-1 envelope protein consists of all of the consecutive amino acids immediately
after the signal peptide in the sequence defined by SEQ ID NO: 879.

6. The composition according to claim 4, wherein the purified gp120 HIV-1 envelope protein consists of all of the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 861.

7. The composition according to claim 4, wherein the purified gp120 HIV-1 envelope protein consists of all of the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 884 or 866.

8. A composition comprising:

(a) a recombinant gp140 HIV-1 envelope protein comprising:

all the consecutive amino acids immediately after the signal peptide MRVMGIQRNYPQWWIWSMLGFWMLMICNG that correspond to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 844 or to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 852,

all the consecutive amino acids immediately after the signal peptide MRVTGIQRNYPQWWIWSMLGLWMLMICNG that correspond to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:857, or

all the consecutive amino acids immediately after the signal peptide MKVRGIQRNYPQWWIWSMLGLWMLMICNG that correspond to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:860; and

(b) a carrier.

9. The composition according to claim 8, wherein the recombinant gp140 HIV-1 envelope protein is an uncleaved gp140 or a cleaved gp140.

10. The composition according to any one of claims 1 to 9, wherein said composition further comprises an adjuvant.

11. A recombinant HIV-1 envelope protein comprising all the consecutive amino acids immediately after the signal peptide MRVMGIQRNYPQWWIWSMLGFWMLMICNG in the sequence defined by SEQ ID NO: 879 or in the sequence defined by SEQ ID NO: 861.

12. A nucleic acid comprising a nucleotide sequence encoding the HIV-1 envelope protein of the composition set forth in claim 1 or 9, wherein said nucleotide sequence is present in a vector.

13. A nucleic acid comprising a nucleotide sequence encoding the HIV-1 envelope protein according to claim 11, wherein said nucleotide sequence is present in a vector.

14. The nucleic acid according to claim 12 or 13, wherein said vector is a viral vector or mycobacterial vector.

15. The nucleic acid according to claim 14, wherein said viral vector is an adenoviral vector or a pox virus vector.

16. A composition comprising the nucleic acid according to any one of claims 12 to 15 and a carrier.

17. The composition according to claim 16, further comprising an adjuvant.

18. The nucleic acid according to claim 12, wherein the nucleotide sequence comprises the sequence defined by SEQ ID NO: 446, 721, 740, or 784.

19. Use of the composition according to any one of claims 2 to 3 and 5 to 10 for inducing an immune response to HIV-1 in a mammal.

20. Use of the composition according to any one of claims 2 to 3 and 5 to 10 for the manufacture of a medicament for inducing an immune response to HIV-1 in a mammal.

21. Use of the composition according to claim 1 or 4 for inducing an immune response to HIV in a mammal.

22. Use of the composition according to claim 1 or 4 for the manufacture of a medicament for inducing an immune response to HIV in a mammal.

23. The use according to claim 21 or 22, wherein the composition comprises the recombinant HIV-1 envelope protein comprising all the consecutive

amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 879 or in the sequence defined by SEQ ID NO: 861 and is for administration as a prime in a prime boost regimen.

24. The use according to claim 23, wherein the composition is for use in combination with a second composition, wherein the second composition comprises at least one second recombinant HIV-1 envelope protein comprising all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 884, 866, 889, 871, 893, or 875, and wherein the second recombinant HIV-1 envelope protein is for administration as a boost in the prime boost regimen.

25. The use according to claim 23, wherein the composition is for use in combination with a second composition, wherein the second composition comprises a second recombinant HIV-1 envelope protein comprising all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 884 or in the sequence defined by SEQ ID NO: 866, and wherein the second recombinant HIV-1 envelope protein is for administration as a boost in the prime boost regimen.

26. The use according to claim 25, wherein the composition is for use in combination with a third composition, wherein the third composition comprises a third recombinant HIV-1 envelope protein comprising all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 889 or in the sequence defined by SEQ ID NO: 871, and wherein the third recombinant HIV-1 envelope protein is for administration as a further boost in the prime boost regimen.

27. The use according to any one of claims 19 to 26, wherein said composition is for administration by injection.

28. The use according to any one of claims 19 to 26, wherein said composition is for intrarectal or vaginal administration.

29. Use of the composition according to claim 16 or 17 for inducing an immune response to HIV-1 in a mammal.

30. Use of the composition according to claim 16 or 17 for the manufacture of a medicament for inducing an immune response to HIV-1 in a mammal.
31. The use according to any one of claims 19 to 30, wherein said mammal is a human.
32. The use according to claim 29 or 30, wherein said mammal is a human.
33. The use according to any one of claims 19 to 22, wherein the composition is for use in combination with the nucleic acid according to any one of claims 12-15 and 18 and, wherein the nucleic acid is for administration as a prime in a prime boost regimen.
34. The use according to any one of claims 29, 30 or 32, wherein the composition further comprises a second nucleic acid encoding a CH505 TF gp140, gp145, or gp160 HIV-1 envelope protein, wherein the second nucleic acid is for use as a prime in a prime boost regimen, and wherein the encoded CH505 TF gp160 HIV-1 envelope protein comprises the sequence defined by SEQ ID NO:844, the encoded CH505 TF gp140 HIV-1 envelope protein corresponds to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:844, or the encoded CH505 TF gp145 HIV-1 envelope protein corresponds to a gp145 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 844.
35. The use according to claim 31, wherein the composition further comprises a nucleic acid encoding a second HIV-1 envelope protein, wherein the nucleic acid is for use as a prime in a prime boost regimen, and wherein the encoded second HIV-1 envelope protein is a CH505 TF gp160 comprising the sequence defined by SEQ ID NO:844, a CH505 TF gp140 corresponding to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:844, or a CH505 TF gp145 corresponding to a gp145 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 844.
36. The use of claim 33, wherein the composition further comprises a second nucleic acid encoding a CH505 TF gp140, gp145, or gp160 HIV-1 envelope protein and wherein the encoded CH505TF gp160 HIV-1 envelope protein comprises

the sequence defined by SEQ ID NO:844, the encoded CH505 TF gp140 corresponds to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:844, or the encoded CH505 TF gp145 corresponds to a gp145 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 844.

37. The use of claim 34 or 36, wherein the second nucleic acid encodes the CH505 TF gp160 HIV-1 envelope protein, and wherein the second nucleic acid comprises the sequence defined by SEQ ID NO: 446;

wherein the second nucleic acid encodes the CH505 TF gp140 HIV-1 envelope protein, and wherein the second nucleic acid corresponds to a nucleic acid encoding a gp140 form of a gp160 HIV-1 envelope sequence encoded by SEQ ID NO:466; or

wherein the second nucleic acid encodes the CH505 TF gp145 HIV-1 envelope protein, and wherein the second nucleic acid corresponds to a gp145 form of a gp160 HIV-1 envelope encoded by SEQ ID NO: 446.

38. The composition according to claim 10, wherein said composition comprises the purified gp120 HIV-1 envelope protein defined in claim 5.

39. Use of the composition according to claim 38 for inducing an immune response to HIV-1 in a mammal.

40. Use of the composition according to claim 38 for the manufacture of a medicament for inducing an immune response to HIV-1 in a mammal.

41. The use of claim 39 or 40, wherein the composition is for use in combination with a second recombinant HIV-1 envelope protein, said second recombinant HIV-1 envelope protein comprising all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 884 or in the sequence defined by SEQ ID NO: 866 and a third recombinant HIV-1 envelope protein, said third recombinant HIV-1 envelope protein comprising all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 889 or in the sequence defined by SEQ ID NO: 871.

42. The use of claim 39 or 40, wherein the composition is for use in combination with a nucleic acid encoding a CH505 TF gp140, gp145, or gp160 HIV-1 envelope protein, wherein the nucleic acid is for use as a prime in a prime boost regimen, and wherein the encoded CH505 TF gp160 HIV-1 envelope protein comprises the sequence defined by SEQ ID NO:844, the encoded CH505 TF gp140 corresponds to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:844, or the encoded CH505 TF gp145 corresponds to a gp145 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 844.

43. The composition according to claim 10, wherein said composition comprises the purified gp120 HIV-1 envelope protein defined in claim 6.

44. Use of the composition according to claim 43 for inducing an immune response to HIV-1 in a mammal.

45. Use of the composition according to claim 43 for the manufacture of a medicament for inducing an immune response to HIV-1 in a mammal.

46. The use of claim 44 or 45, wherein the composition is for use in combination with a second recombinant HIV-1 envelope protein and a third recombinant HIV-1 envelope protein, wherein said second recombinant HIV-1 envelope protein comprises all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 884 or in the sequence defined by SEQ ID NO: 866, and wherein said third recombinant HIV-1 envelope protein comprises all the consecutive amino acids immediately after the signal peptide in the sequence defined by SEQ ID NO: 889 or in the sequence defined by SEQ ID NO: 871.

47. The use of claim 44 or 45, wherein the composition is for use in combination with a nucleic acid encoding a CH505 TF gp140, gp145, or gp160 HIV-1 envelope protein, wherein the nucleic acid is for use as a prime, and wherein the encoded CH505 TF gp160 HIV-1 envelope protein comprises the sequence defined by SEQ ID NO:844, the encoded CH505 TF gp140 corresponds to a gp140 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO:844, or the encoded CH505 TF gp145 corresponds to a gp145 form of a gp160 HIV-1 envelope sequence defined by SEQ ID NO: 844.

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JUMBO APPLICATIONS/PATENTS

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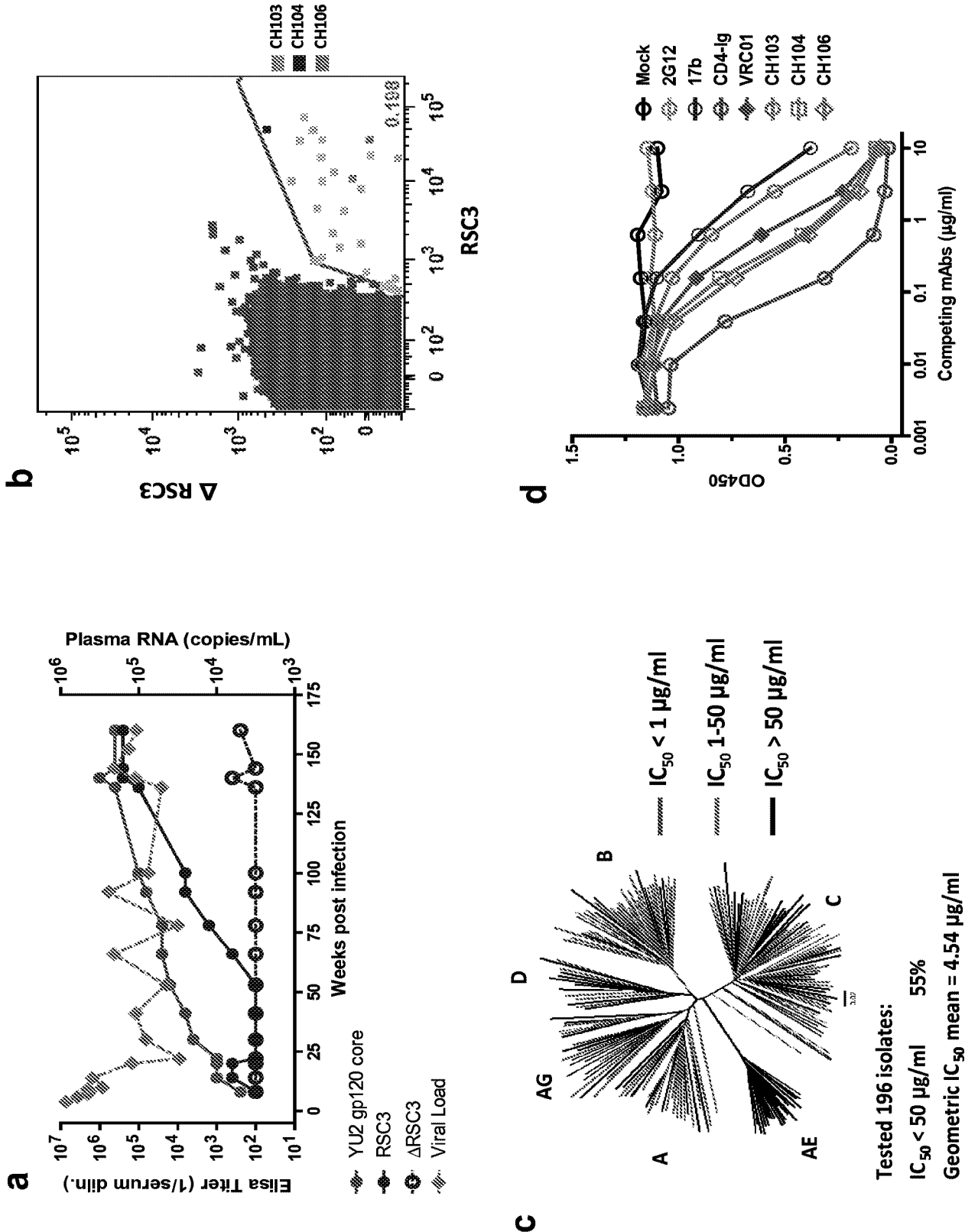
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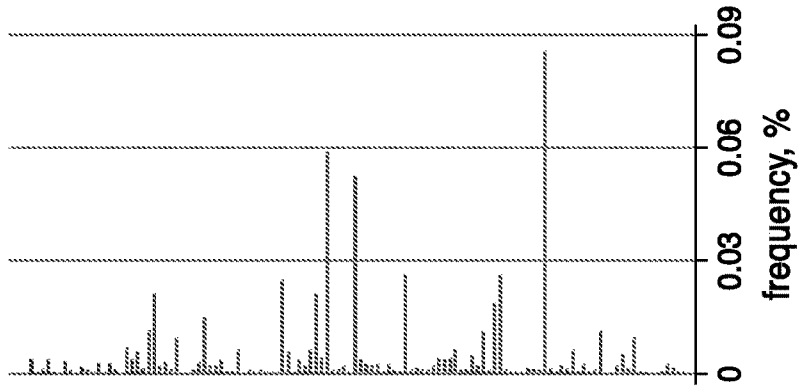
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FIG. 1

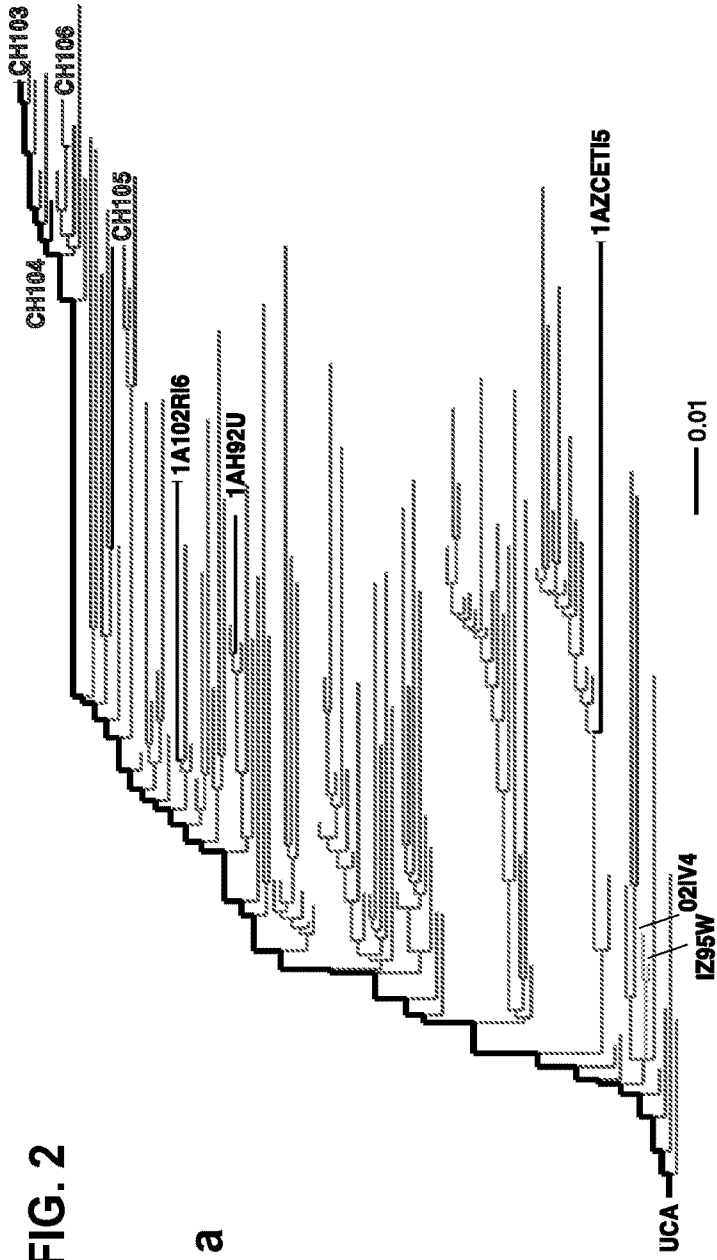




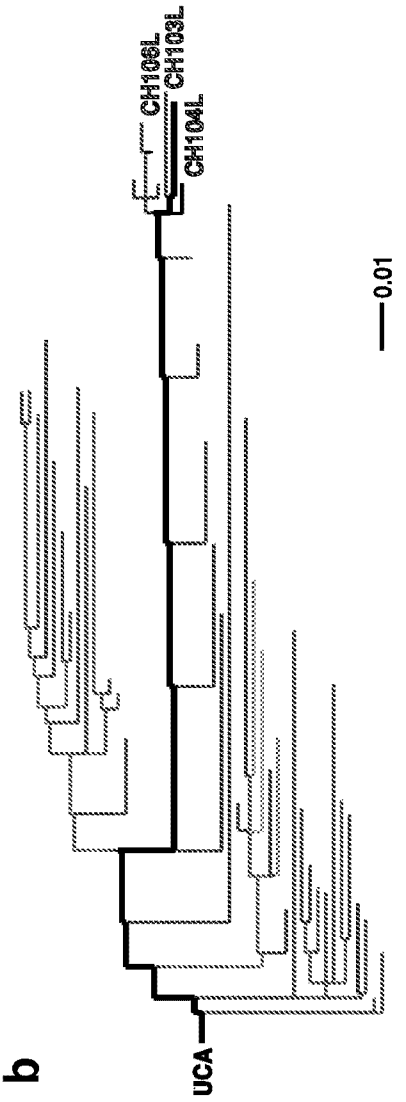
Week 14
Week 53
Week 92
Week 144

FIG. 2

a



b



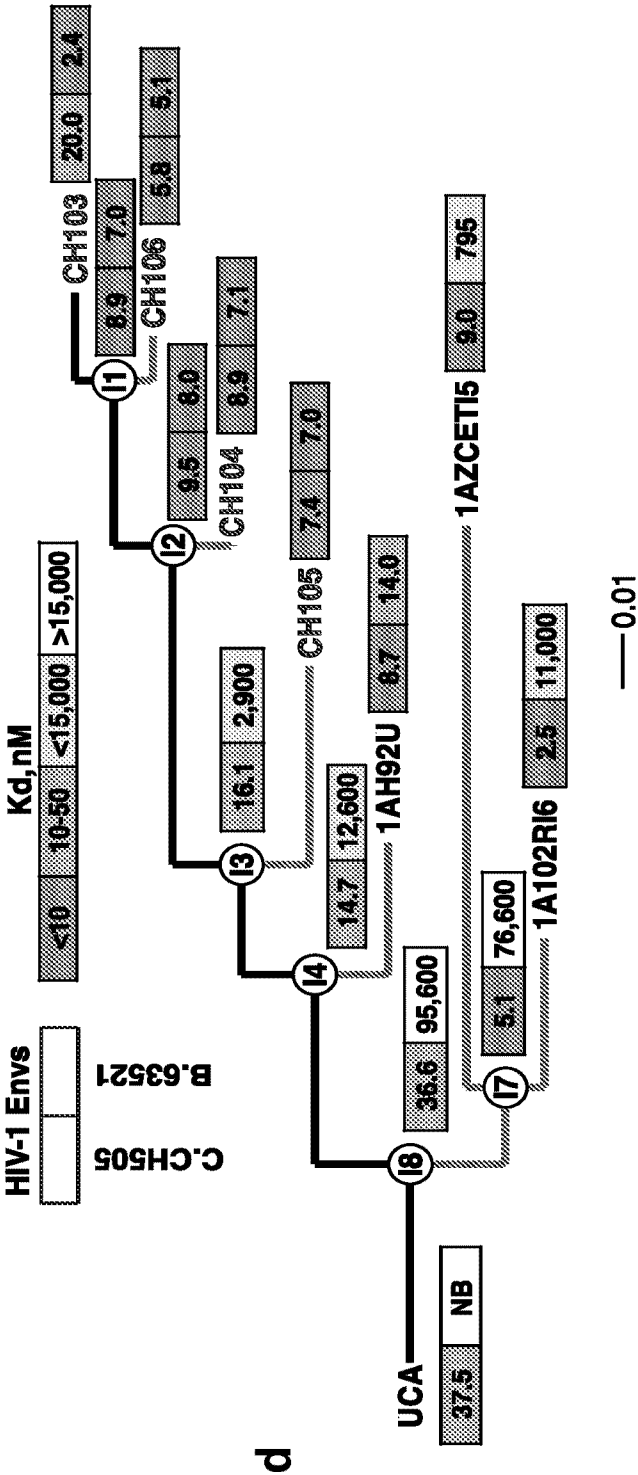
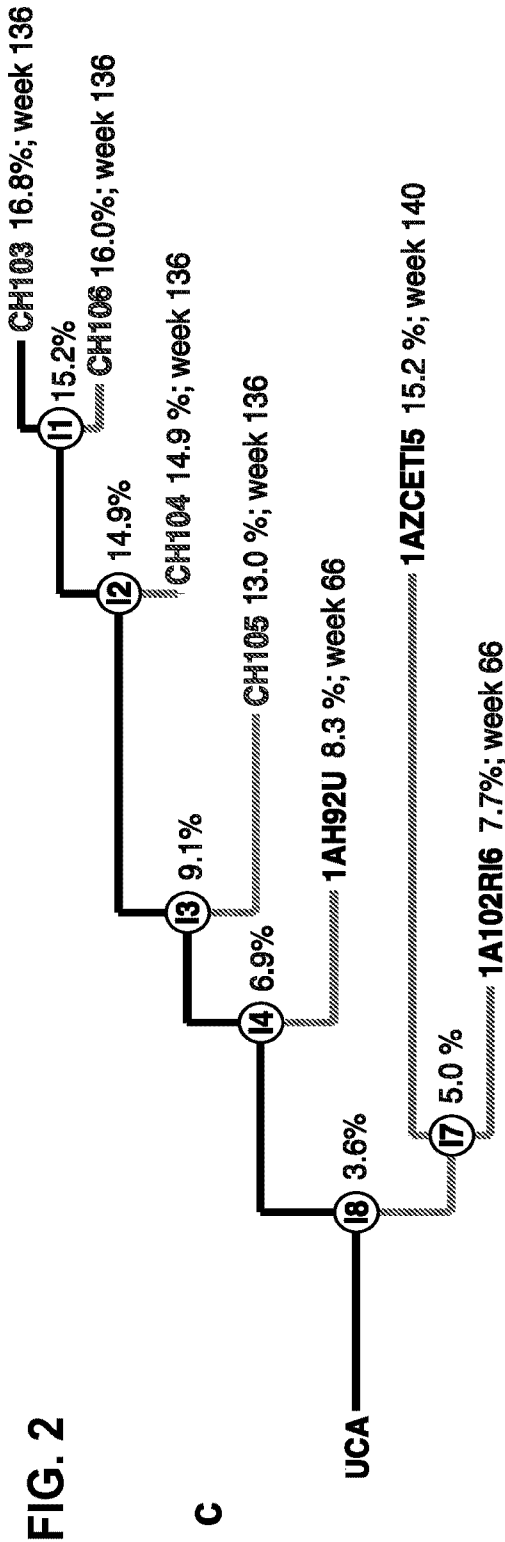


FIG. 3

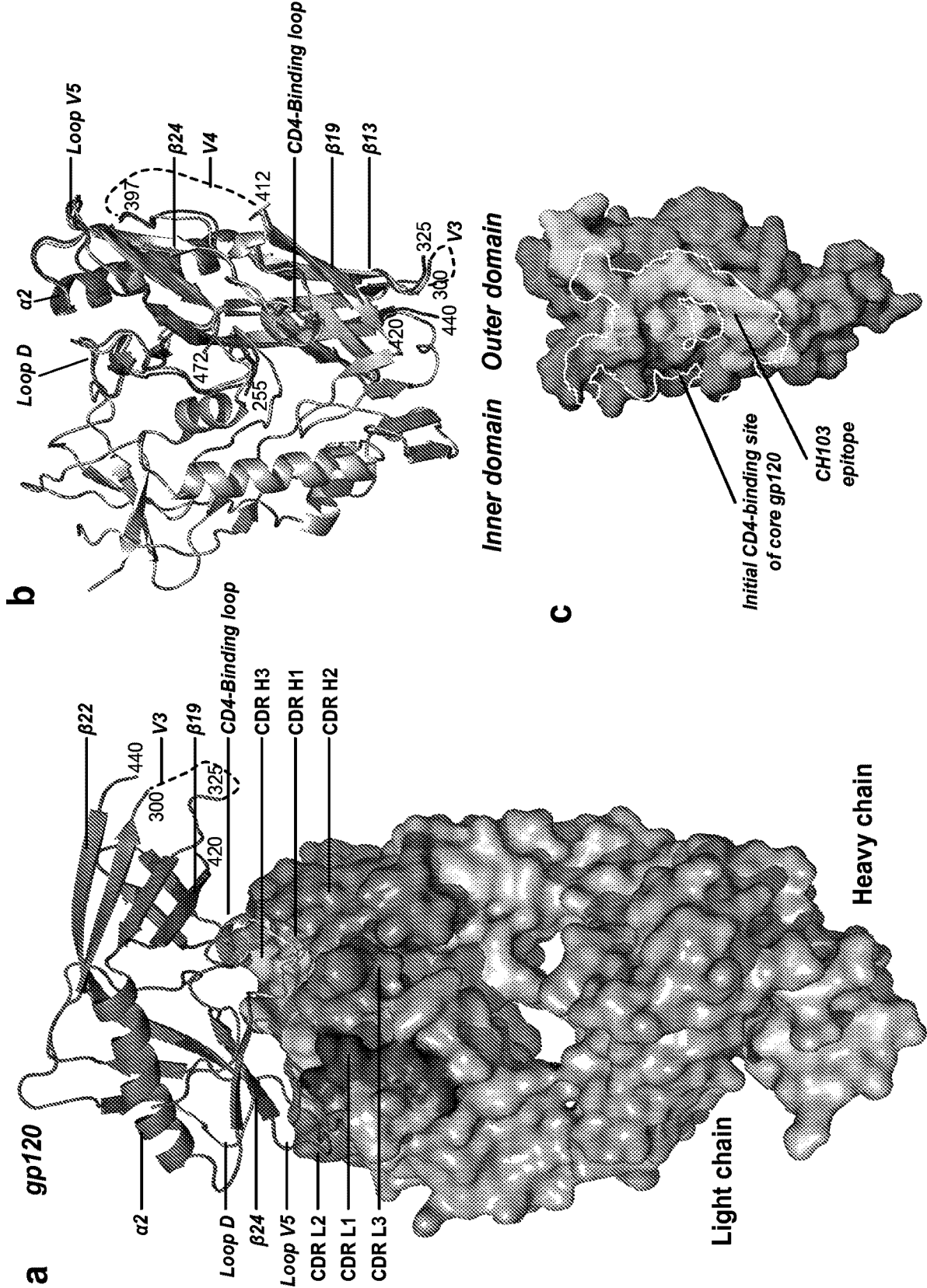


FIG. 3



FIG. 4

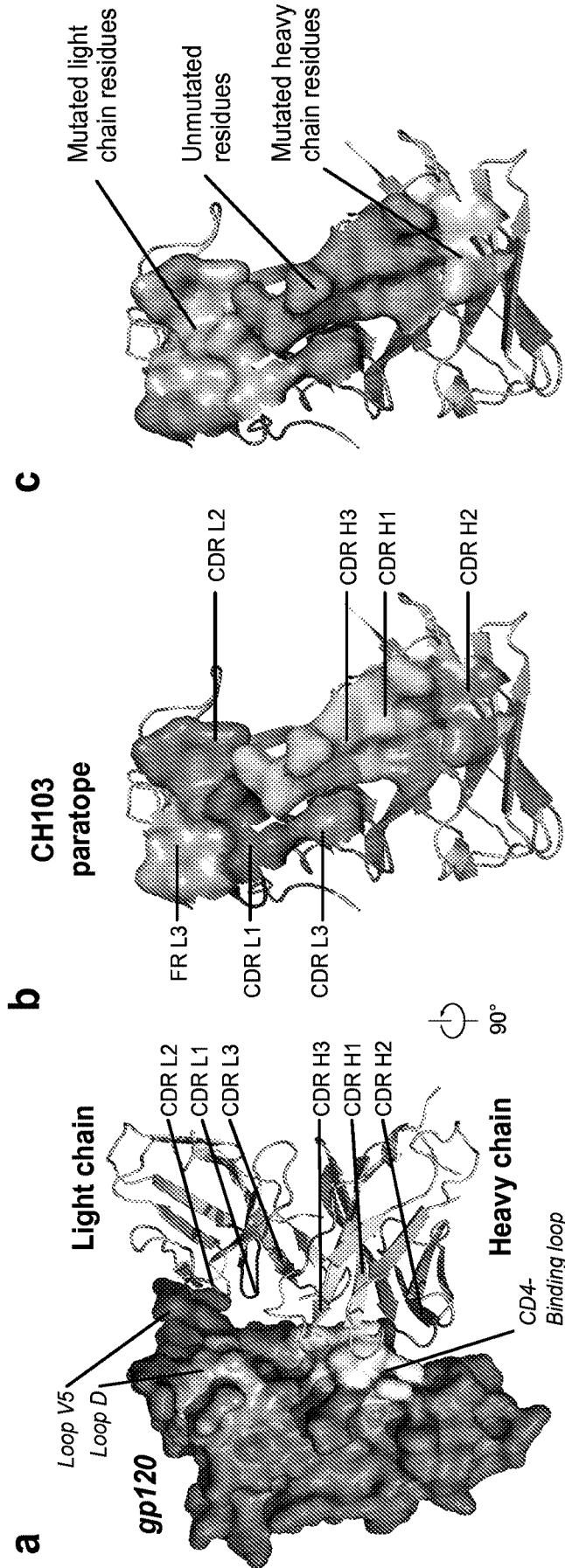
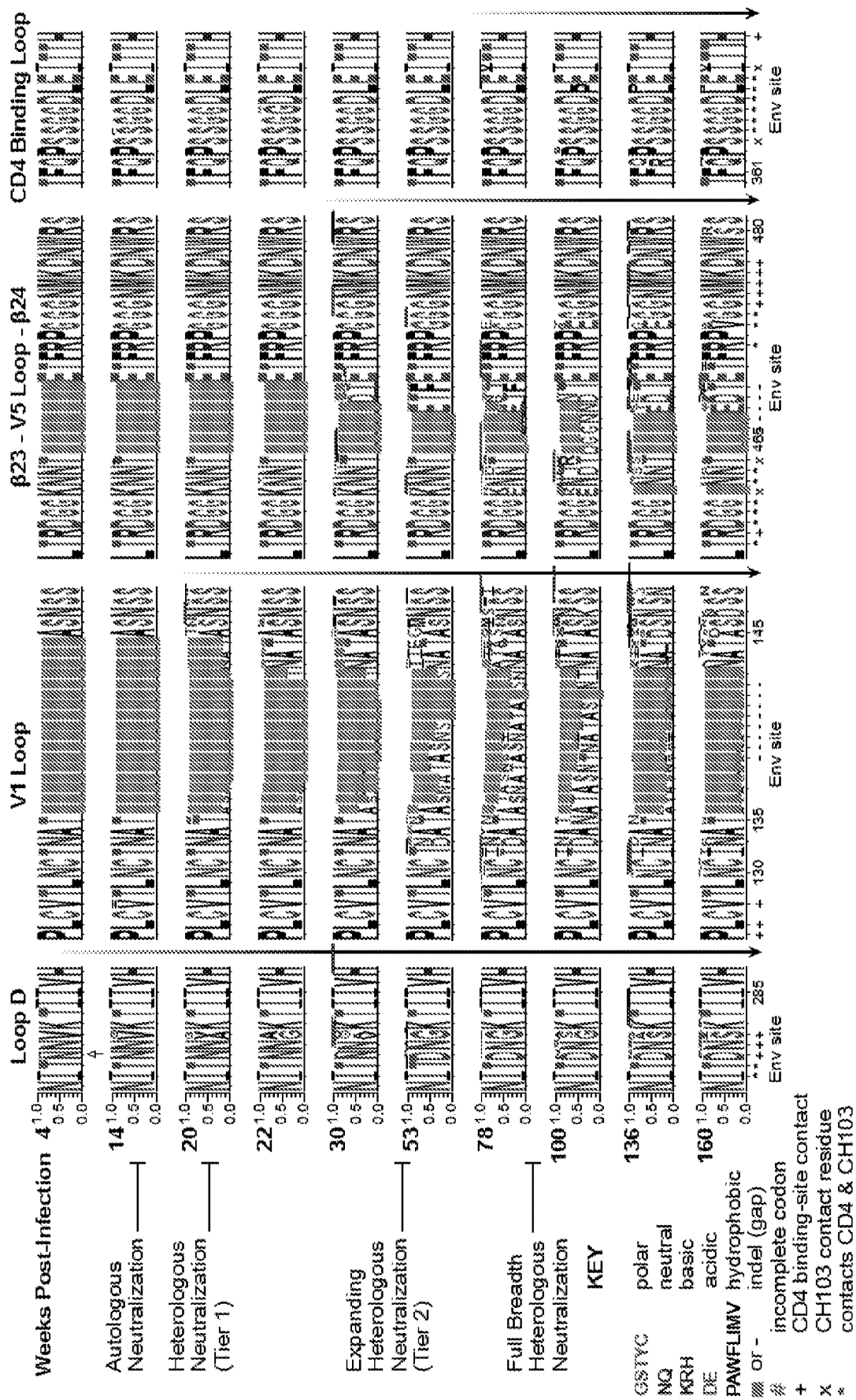


FIG. 5



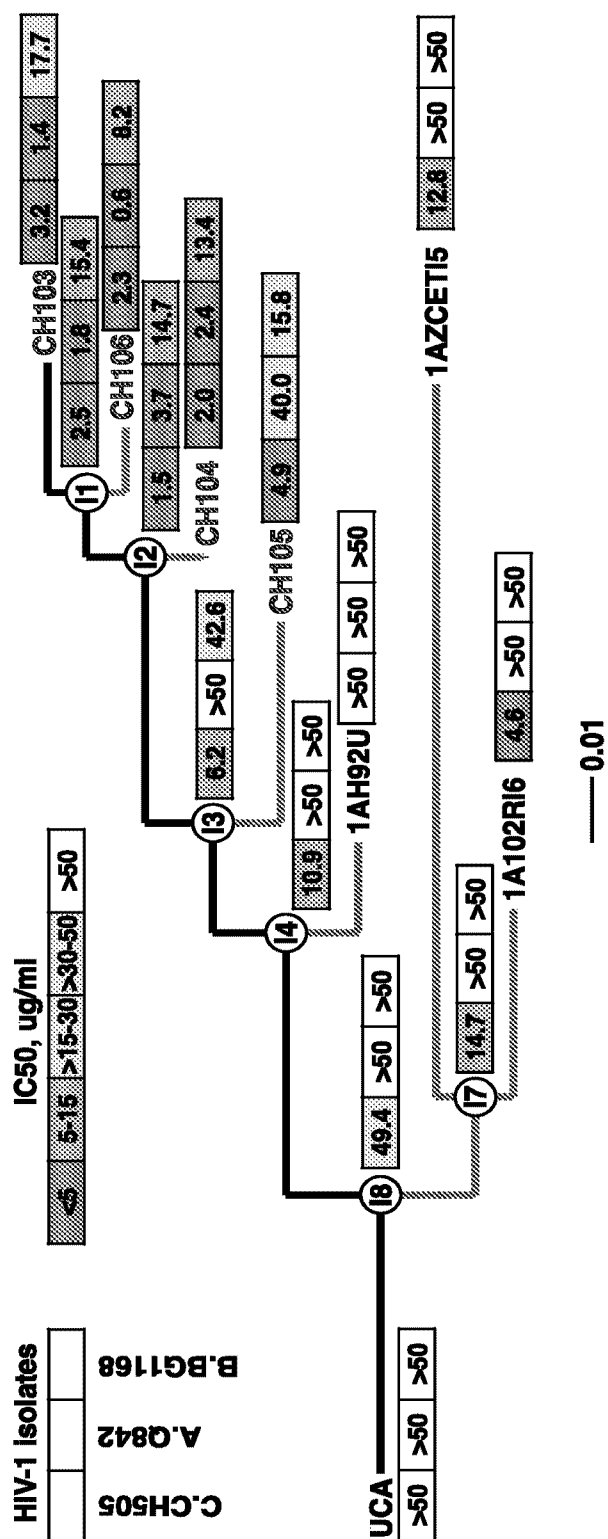
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FIG. 6

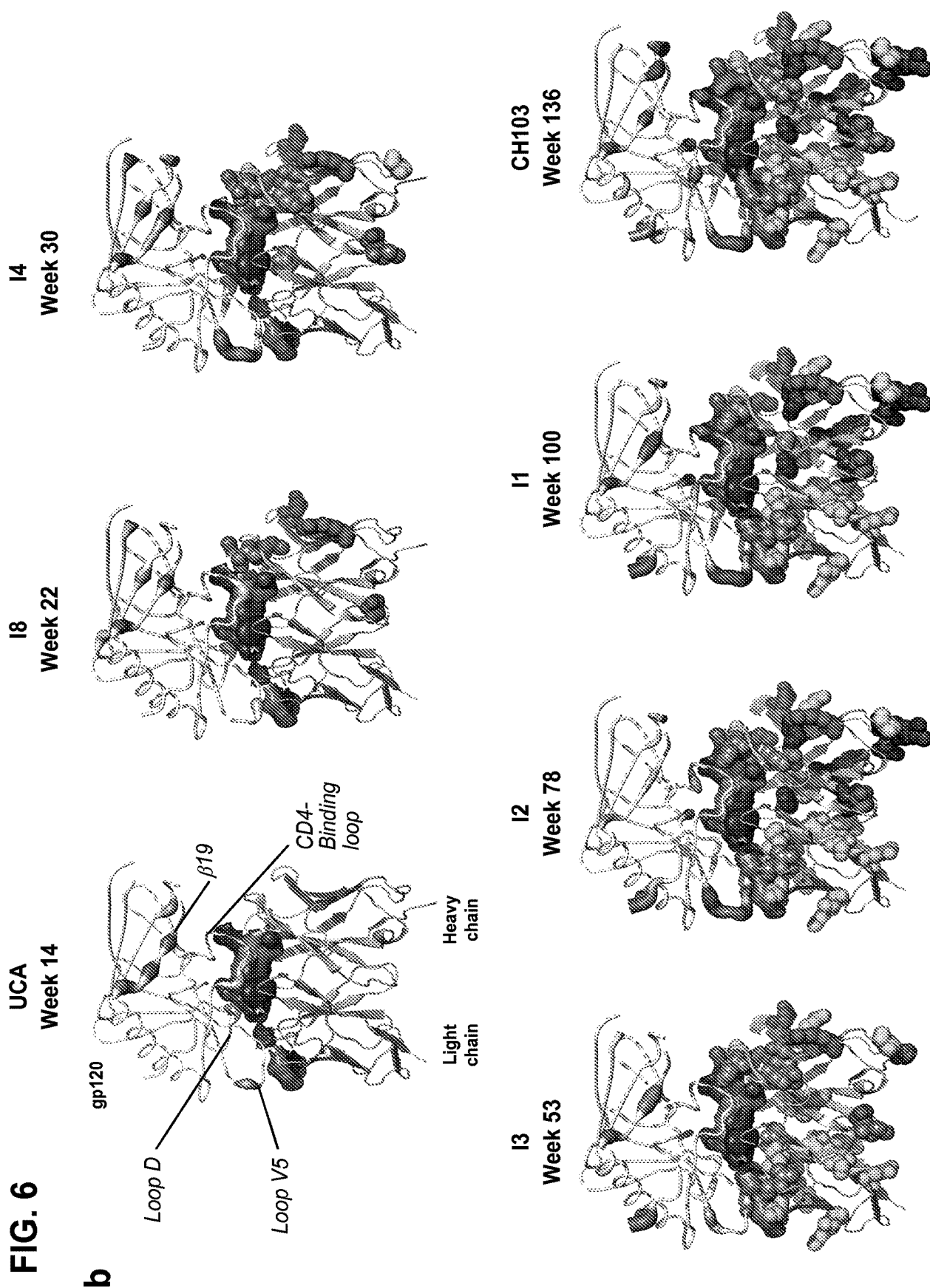


FIG. 7

Hamming distance frequency distributions of sequences at (a) week 4 and (b) week 14. A model of the best fit Poisson distribution is shown as a red line. Analysis of the sequence diversity in the first available sample (a) from subject CH505 using the Poisson Fitter tool (ref below) indicates that the sequences were a consistent with a star phylogeny and that the mutations were accumulating according to a Poisson distribution (goodness of fit $p = 0.11$). This is consistent with a single founder virus establishing the infection, with random accumulation of mutations prior to selection. The lambda parameter was 1.325, and assuming the mutation rate of 2.16×10^{-5} , the estimated time from the most recent common ancestor was 22 days (95% CI, 18-27). Given that the outer bound of this confidence interval is 27 days, it is highly like this sample was taken within 4 weeks of infection, thus we are calling this sampling time "week 4" as a conservative estimate. This timing estimate is further supported by Feibig staging at time of enrollment. By week 14 (b), the tree was no longer consistent with a star phylogeny or a Poisson distribution ($p < 10^{-10}$), indicating selection was well underway. Of note, although the mutation data at week 4 (a) is statistically consistent with a Poisson distribution, the observed number of pairwise sequence identities was somewhat reduced relative to expectation, and the observed number of Hamming distances of 1 and 2 are slightly more than expected. This is of interest as this shift is the a result of a single mutation in loop D, in a CH103 contact residue (N279K) -- so although the deviation from the Poisson was not significant, given its location it is possible that the site is a very early indicator of selection.

Reference: Giorgi EE, Funkhouser B, Athreya G, Perelson AS, Korber BT, Bhattacharya T. Estimating time since infection in early homogeneous HIV-1 samples using a Poisson model. BMC Bioinformatics 2010 Oct 25;11:532. PMID: 20973976 http://www.hiv.lanl.gov/content/sequence/POISSON_FITTER/poiss_fitter.html

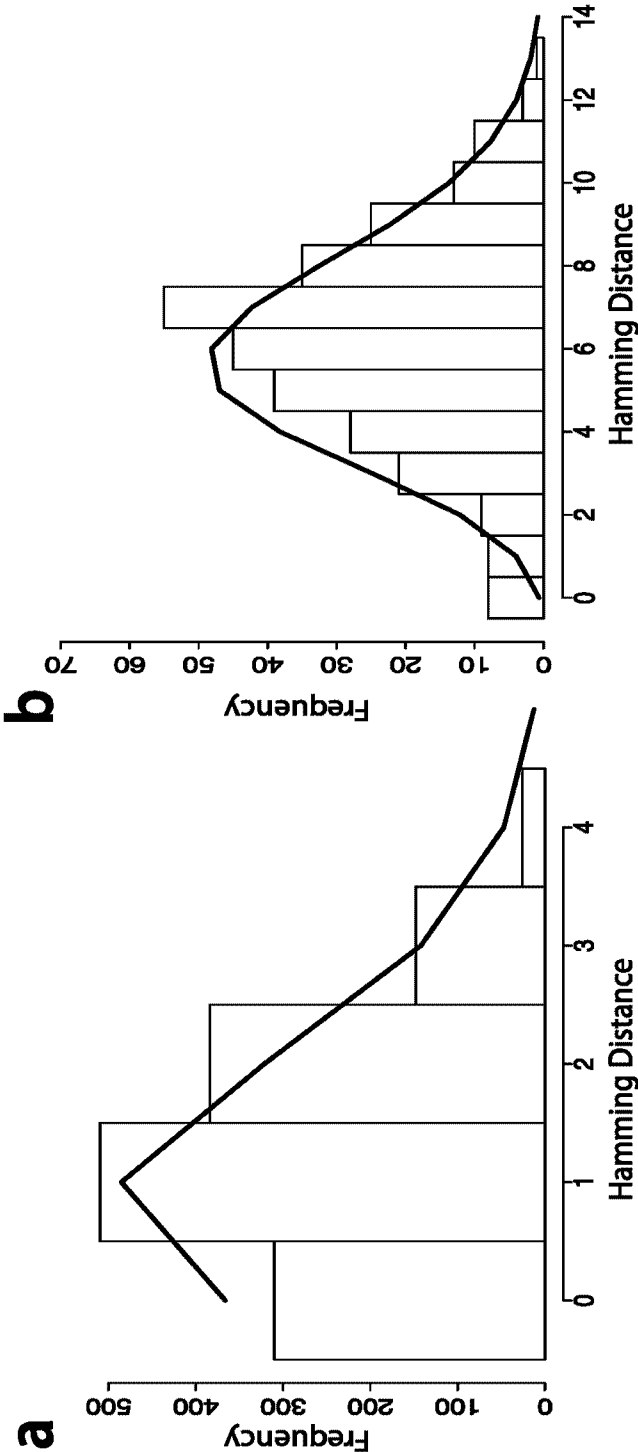


FIG. 8

Binding of plasma antibodies of CH505 patient over time to autologous transmitted/founder (T/F) and heterologous HIV-1 Env proteins. Plasma samples were longitudinally collected from HIV-1 patient CH505 starting from time of infection(in x axis) and tested for neutralization activity against the autologous transmitted/founder (T/F) virus and heterologous HIV-1 Env pseudoviruses including subtype B (B) SF162, JRFL and BG1168) and subtype A in TZM-bl cell-based neutralization assays. Results were expressed as IC50 (reciprocal plasma dilution) (in y axis).

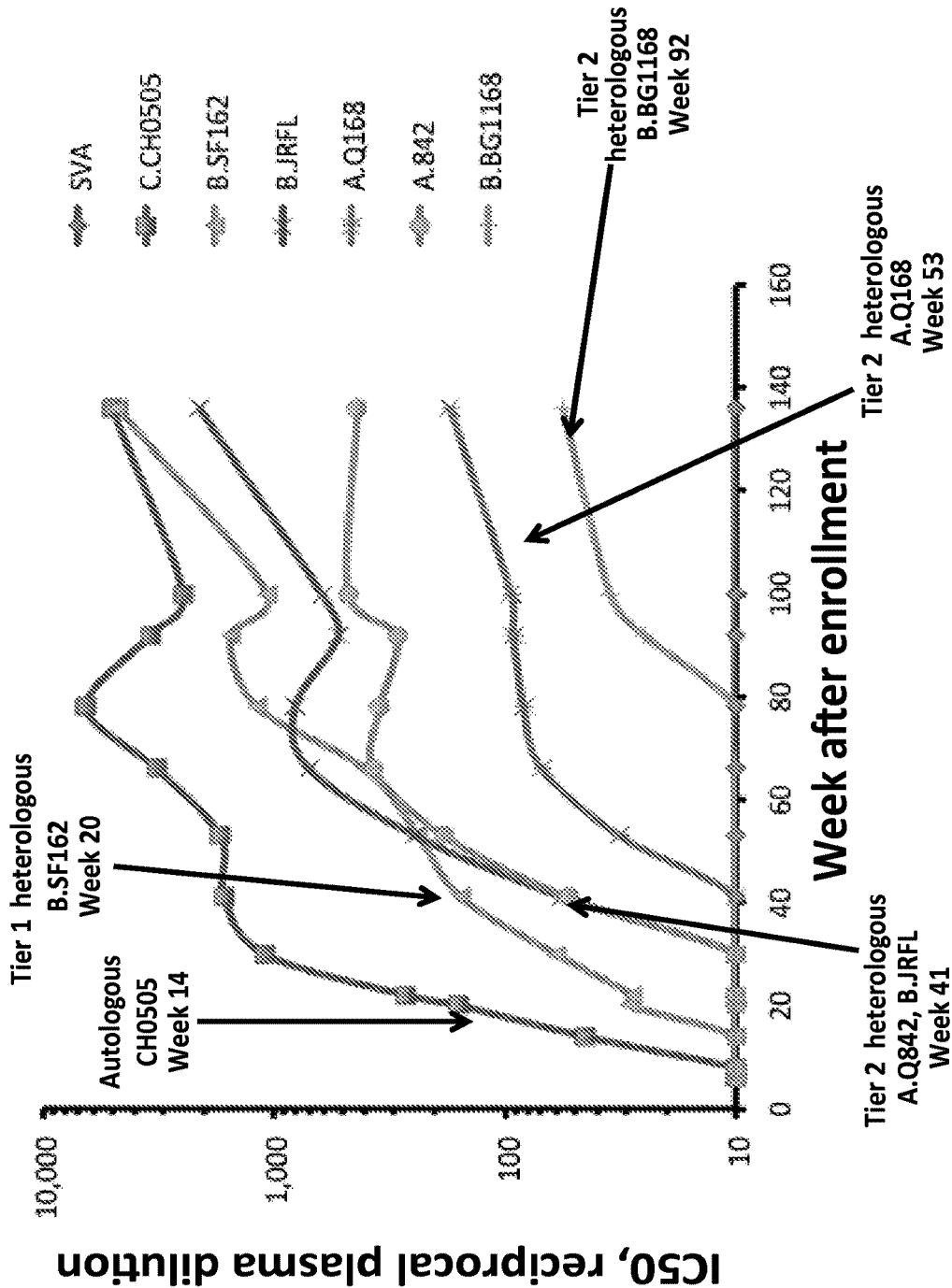


FIG. 9

Reactivity of antibodies in CH103 clonal lineage to HIV-1 Env resurfaced core3 (RSC3) and RSC3 mutant. Antibodies in CH103 clonal lineage were tested in dose range from 100 μ g to 0.0005 μ g/ml for binding to (a) HIV-1 Env RSC and (b) RSC3 with P363N and Δ 371I mutations in ELISA. Results are expressed as EC50 (μ g/ml) and are indicated next to the individual antibodies. NB = no detectable binding.

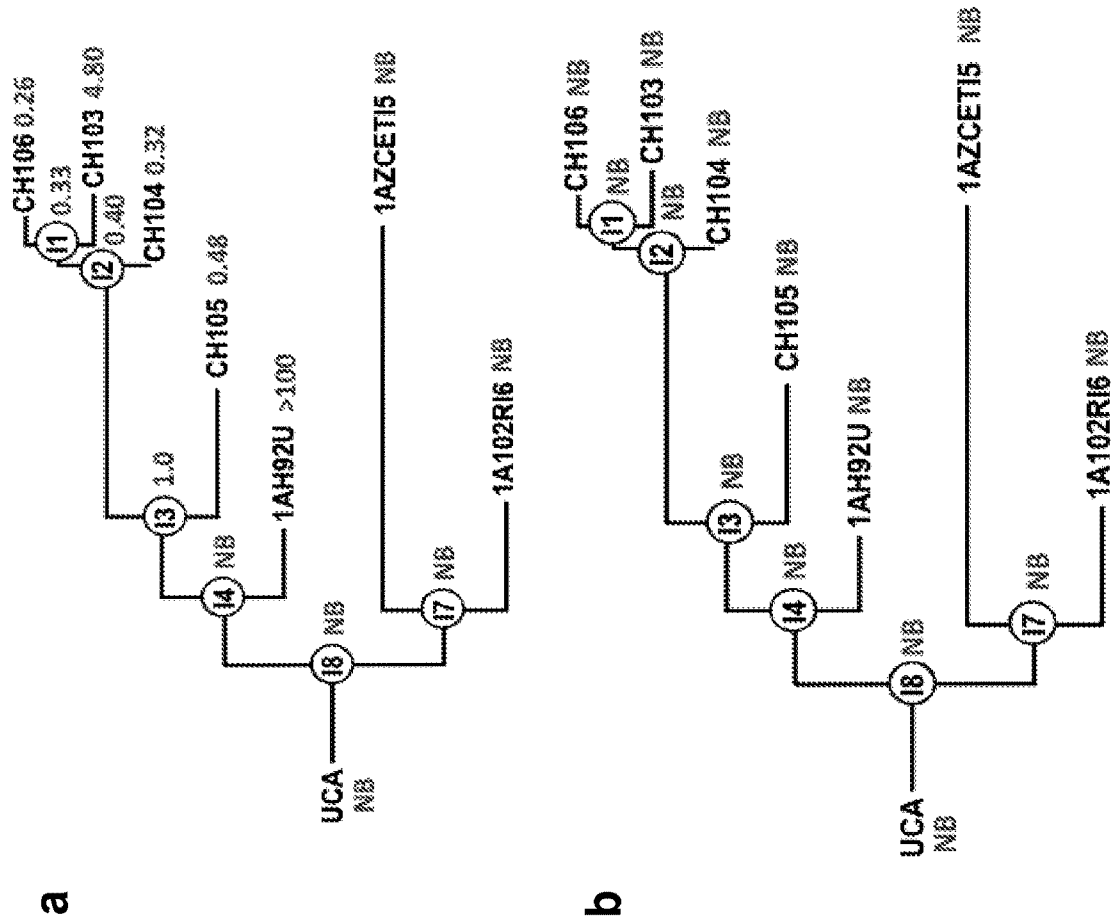


FIG. 10

SDS-PAGE analysis of recombinant HIV-1 Env gp140 and gp120 proteins*. HIV-1 Env gp120 and gp140 proteins were analyzed on SDS-PAGE under reducing condition (a) and gp140 proteins were analyzed on blue native PAGE for (b). Individual HIV-1 Env proteins are identified on the top of gels. a, The HIV-1 gp120 and gp140 used in the study had no degradation under reducing condition in SDS-PAGE. b, Most heterologous HIV-1 Env gp140 Envs and all autologous CH505 gp140 Envs migrated predominantly as trimers and also contain dimer and monomer forms.

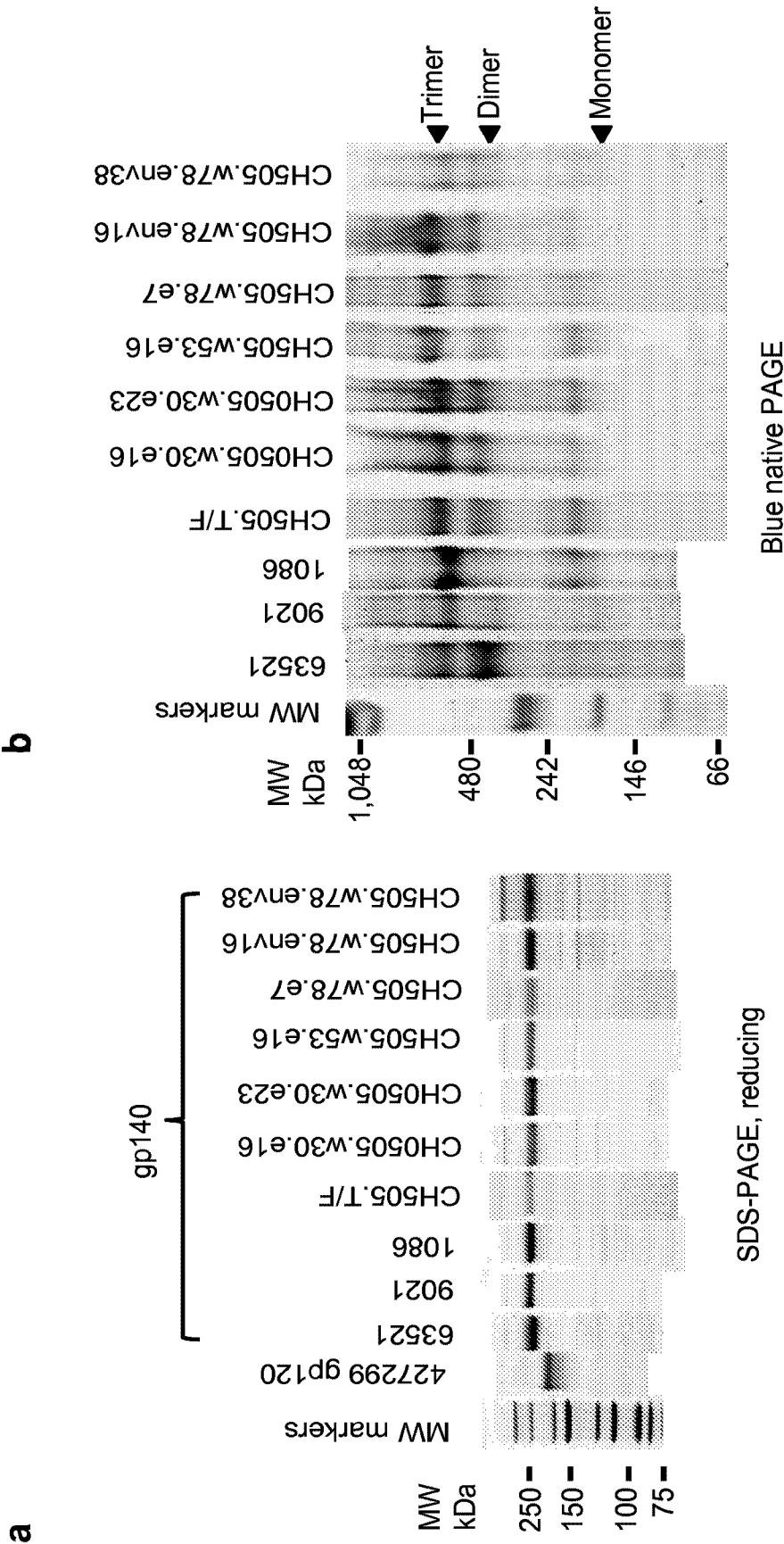
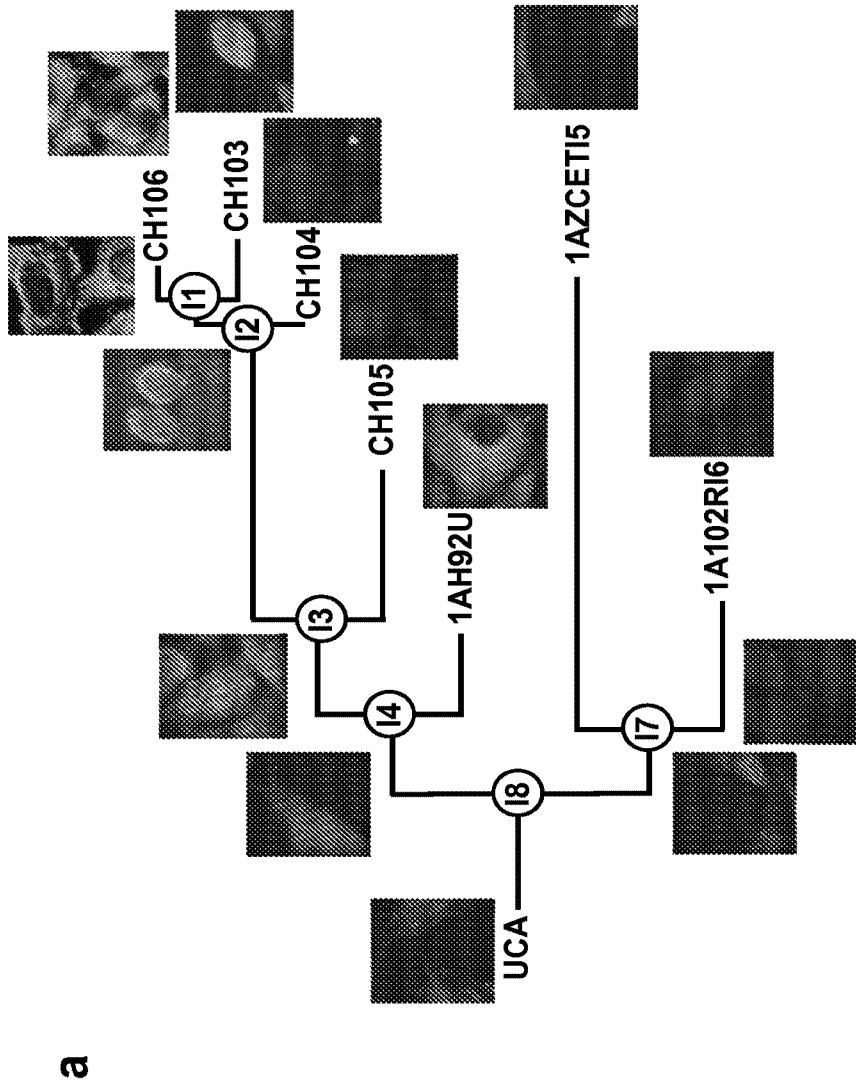
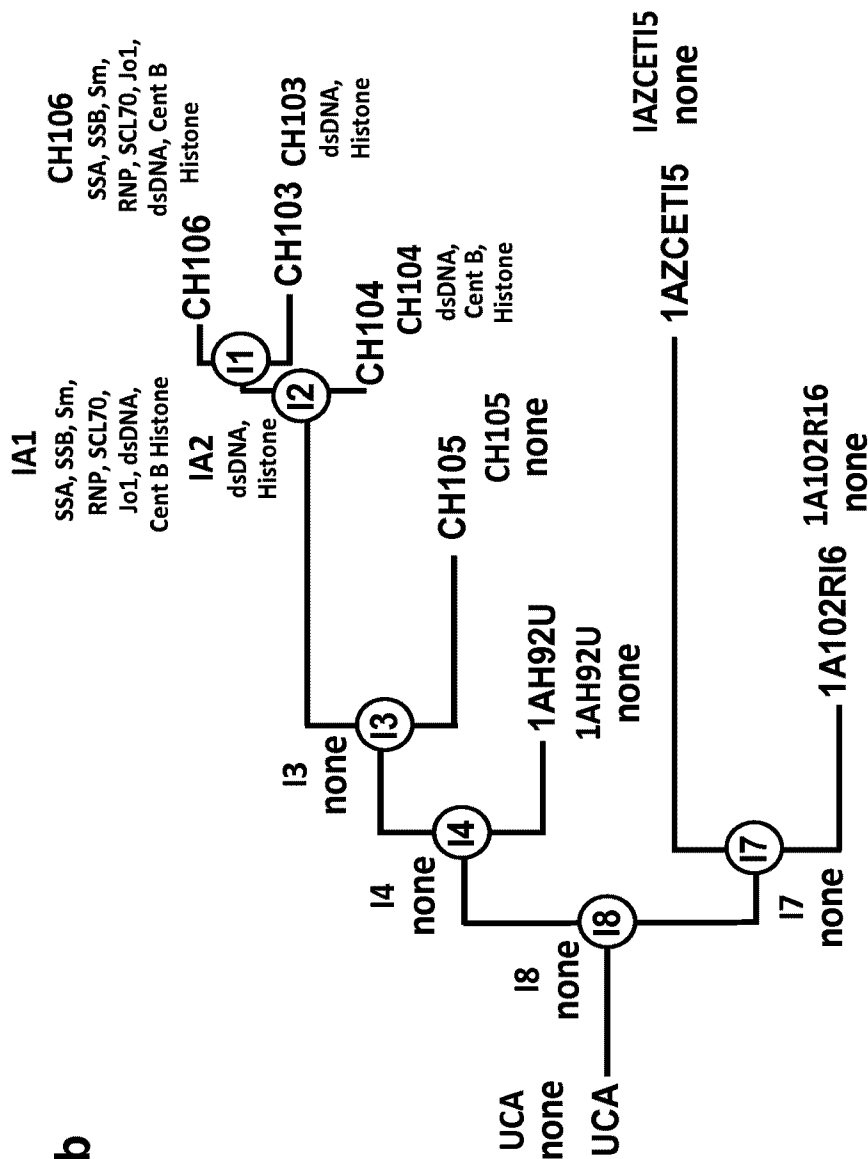


FIG. 11



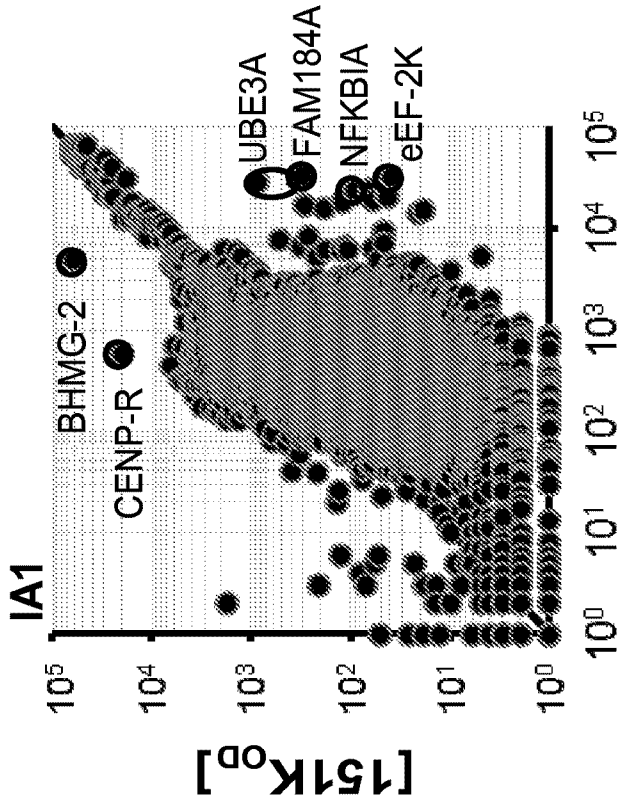
Polyreactivity analysis of CH103 clonal lineage antibodies by HEp-2 staining, ANA assays and protein array microchip analysis. Reactivity of antibodies in CH103 clonal lineage was assayed by indirect immunofluorescence staining (a) and by ANA assays (b). Pictures at magnification x 200 of immunofluorescence staining for individual antibodies are presented next to the antibody ID. Results of the reactivity of individual antibodies with panel of autoantigens assayed by ANA is indicated (b). The intermediate antibody (I1) and CH106 were identified as reactive with HEp-2 cells and then selected for further testing for reactivity with human host cellular antigens (c and d) using Invitrogen ProtoArrays™. It was found that I1 (c) and CH106 (d) exhibit specific autoreactivity and robust polyreactivity. Bound antibody was determined by immunofluorescence and relative fluorescence intensities for 9,400 recombinant human proteins in the 151K array (y-axis) is plotted against (x-axis) the homologous intensities in IA1 (c) and CH106 (d) arrays. All proteins are printed in duplicate on each array and each data point represents one fluorescence measurement. The diagonals in each graph represent equal fluorescence intensities (equivalent binding) by the I1, CH106 and 151K control Ab. Self-antigens bound by the I1 and CH106 are identified by high fluorescence intensity versus 151K and are indicated by circles. Polyreactivity is indicated by significant and general skewing from the diagonal. Autoantigens identified: BHTM2 (betaine-homocysteine methyltransferase 2); CENP-R (centromere protein R) [151K]; eEF-2K (eukaryotic elongation factor-2 kinase); UBE3A (ubiquitin-protein ligase E3A) [IA1 and CH106]; TGM2 (transglutaminase 2) [CH106]; NFKBIA (nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha); FAM184A (family with sequence similarity 184, member A) [I1].



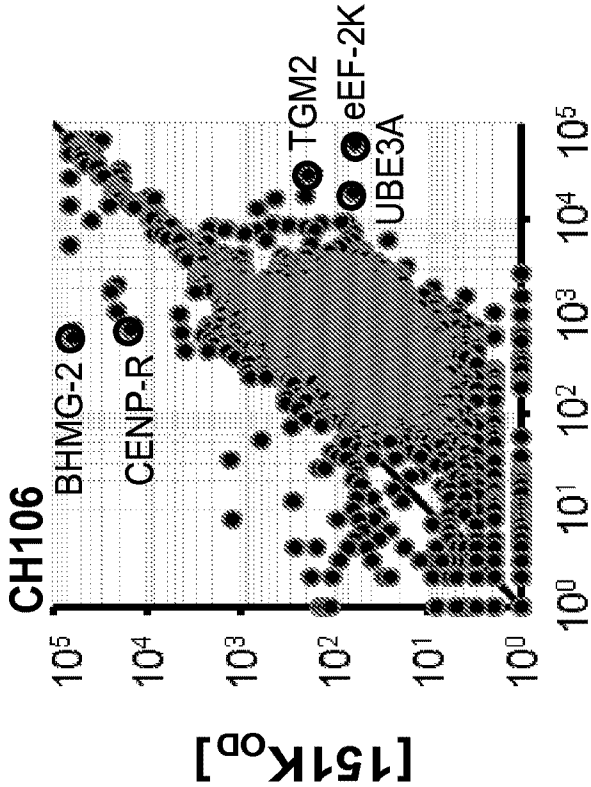
Polyreactivity analysis of CH103 clonal lineage antibodies by HEp-2 staining, ANA assays and protein array microchip analysis . Reactivity of antibodies in CH103 clonal lineage was assayed by indirect immunofluorescence staining (a) and by ANA assays (b). Pictures at magnification x 200 of immunofluorescence staining for individual antibodies are presented next to the antibody ID. Results of the reactivity of individual antibodies with panel of autoantigens assayed by ANA is indicated (b). The intermediate antibody (I1) and CH106 were identified as reactive with HEp-2 cells and then selected for further testing for reactivity with human host cellular antigens (c and d) using Invitrogen ProtoArrays™. It was found that I1 (c) and CH106 (d) exhibit specific autoreactivity and robust polyreactivity. Bound antibody was determined by immunofluorescence and relative fluorescence intensities for 9,400 recombinant human proteins in the 151K array (y-axis) is plotted against (x-axis) the homologous intensities in IA1 (c) and CH106 (d) arrays. All proteins are printed in duplicate on each array and each data point represents one fluorescence measurement. The diagonals in each graph represent equal fluorescence intensities (equivalent binding) by the I1, CH106 and 151K control Ab. Self-antigens bound by the I1 and CH106 are identified by high fluorescence intensity versus 151K and are indicated by circles. Polyreactivity is indicated by significant and general skewing from the diagonal. Autoantigens identified: BHMT2 (betaine-homocysteine methyltransferase 2); CENP-R (centromere protein R) [151K]; eEF-2K (eukaryotic elongation factor-2 kinase); UBE3A (ubiquitin-protein ligase E3A) [IA1 and CH106]; TGM2 (transglutaminase 2) [CH106]; NFKBIA (nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha); FAM184A (family with sequence similarity 184, member A) [I1].

FIG. 11

c



d



Polyreactivity analysis of CH103 clonal lineage antibodies by HEp-2 staining, ANA assays and protein array microchip analysis . Reactivity of antibodies in CH103 clonal lineage was assayed by indirect immunofluorescence staining (a) and by ANA assays (b). Pictures at magnification x 200 of immunofluorescence staining for individual antibodies are presented next to the antibody ID. Results of the reactivity of individual antibodies with panel of autoantigens assayed by ANA is indicated (b). The intermediate antibody (I1) and CH106 were identified as reactive with HEp-2 cells and then selected for further testing for reactivity with human host cellular antigens (c and d) using Invitrogen ProtoArrays™. It was found that I1 (c) and CH106 (d) exhibit specific autoreactivity and robust polyreactivity. Bound antibody was determined by immunofluorescence and relative fluorescence intensities for 9,400 recombinant human proteins in the 151K array (y-axis) is plotted against (x-axis) the homologous intensities in IA1 (c) and CH106 (d) arrays. All proteins are printed in duplicate on each array and each data point represents one fluorescence measurement. The diagonals in each graph represent equal fluorescence intensities (equivalent binding) by the I1, CH106 and 151K control Ab. Self-antigens bound by the I1 and CH106 are identified by high fluorescence intensity versus 151K and are indicated by circles. Polyreactivity is indicated by significant and general skewing from the diagonal. Autoantigens identified: BHM2 (betaine-homocysteine methyltransferase 2); CENP-R (centromere protein R) [151K]; eEF-2K (eukaryotic elongation factor-2 kinase); UBE3A (ubiquitin-protein ligase E3A) [IA1 and CH106]; TGM2 (transglutaminase 2) [CH106]; NFKBIA (nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha); FAM184A (family with sequence similarity 184, member A) [I1].

FIG. 12

Crystal packing of the CH103-gp120 complex in P21 space group. **a**, A view of the crystal lattice. The two complexes in each asymmetric unit are marked with red and blue dashed lines and are shown in cartoon diagrams with gp120 in red and salmon, CH103 heavy chain in green and palegreen, and CH103 light chain in light blue and cyan. **b**, A close-up view of the lattice between two neighboring complexes. When extended core gp120 of clade C ZM176.66 from the VRC01 complex is superposed with its ordered corresponding portions in the CH103 complex, the inner domain shown in magenta clashes with the neighboring complex, indicating inner domain of gp120 is not present in the CH103-gp120 crystal due to proteolytic degradation during crystal growth.

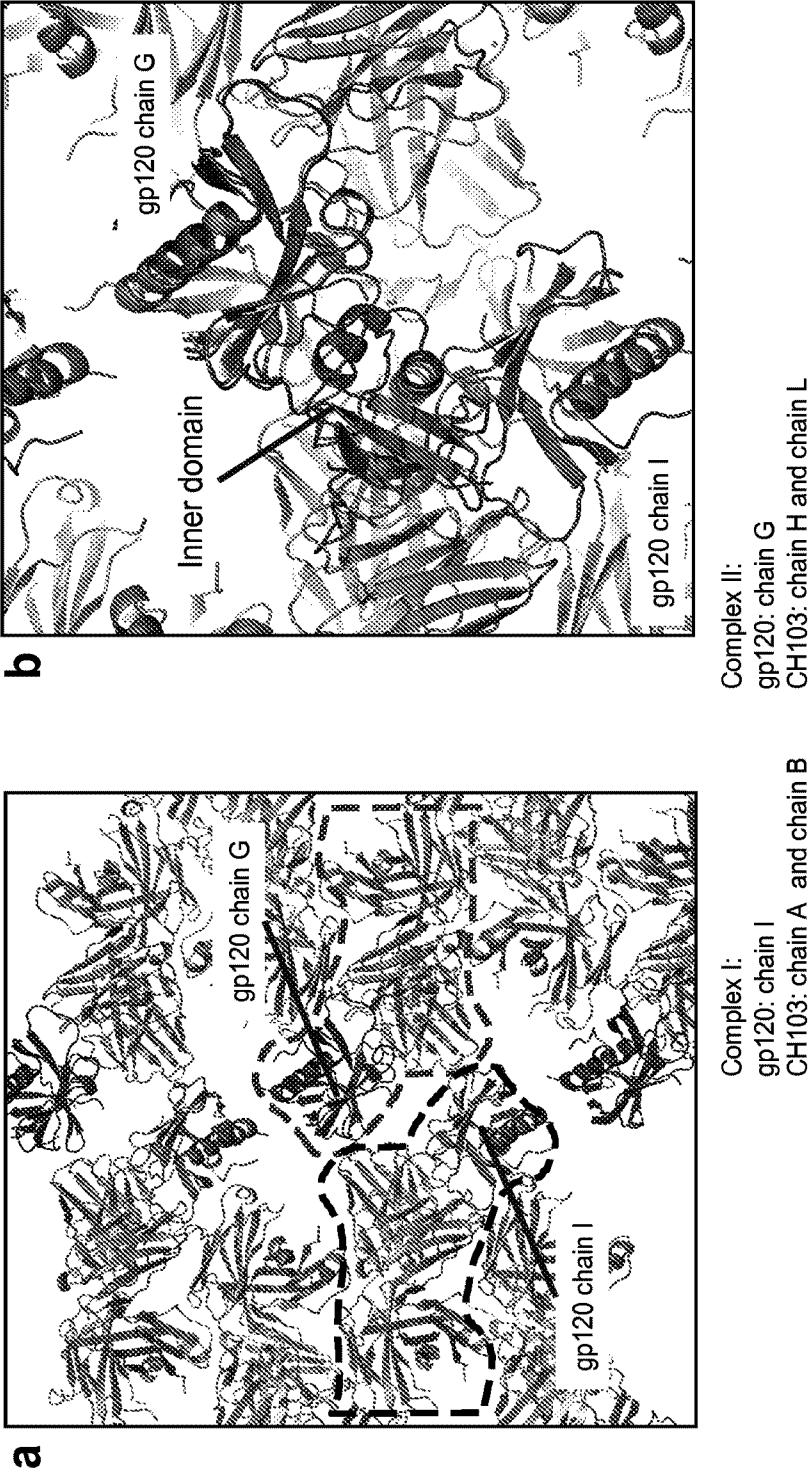
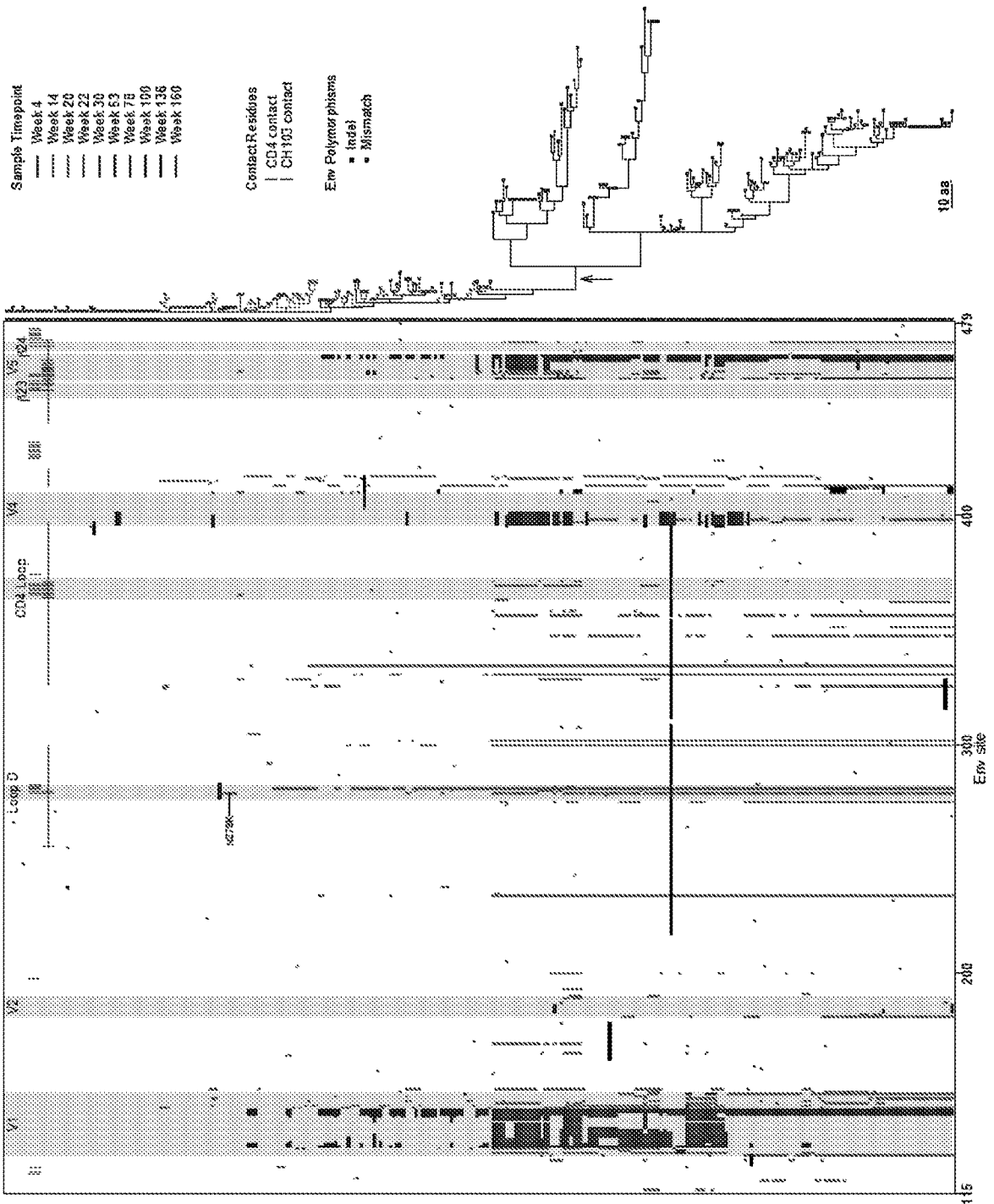


FIG. 13

Pixel map and phylogenetic tree of HIV evolution over time in CH505. The pixel tool (<http://www.hiv.lanl.gov/content/sequence/pixel/pixel.html>) was used to illustrate the amino changes in the V1 to V5 region of the envelope; we focused on this region as it most critical CD4bs antibody susceptibility, and includes of all known CD4 binding contacts, which are indicated as black tic marks along the top of the figure. Blue tic marks indicated CH103 contact residues, and the horizontal blue line indicates that part of gp120 that was used for the CH103 crystal structure (although the contact surface is mostly there, still quite a bit is missing that is important for CD4 and VRC01, which is why we use CD4 contacts to help define bits that may be important for CH103 binding in those missing regions). Each row is a sequence, and they are ordered according to the phylogeny. Red bits indicate amino acid change relative to the TF virus, and black bits indicate either an insertion or deletion. The phylogenetic tree on the right was made with PhyML v2 [1] and the JTT substitution model [2] from the translated Env sequences. The tree was configured as a ladder and the T/F virus was reconstructed from the first time point sequences obtained at week 4 after transmission. Colors indicated the estimated number of weeks from infection. The tree was rendered with APE v3.0-6 [3] and both used R v2.15.1 [4]. The arrow indicates the week 30-53 selective bottleneck.

1. Guindon S, Gascuel O. 2003. A simple, fast and accurate algorithm to estimate large phylogenies by maximum likelihood. *Syst. Biol.* 52:696-704.
2. Jones DT, Taylor WR, Thornton JM. 1992. The rapid generation of mutation data matrices from protein sequences. *Comput Appl Biosci* 8: 275-282.
3. Paradis E, Claude J, Strimmer K. 2004. APE: analyses of phylogenetics and evolution in R language. *Bioinformatics* 20: 289-290.
4. R Core Team. 2012. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL <http://www.R-project.org/>.

FIG. 14

Entropy map illustrating the per site diversity within each time point sampled in CH505. Full gp160 is shown, and CD4 binding-site contacts and CH103 contacts residues are highlighted. This figure shows the Shannon entropy of each position in the alignment, where the observed frequency of all in a position characters is considered, and a gap is treated as a character (Korber J Virol. 1994;68(11):7467-81). This provides a map of regional within-time point diversity spanning Env, and illustrates where mutations are concentrated and the relative diversity of key regions over time.

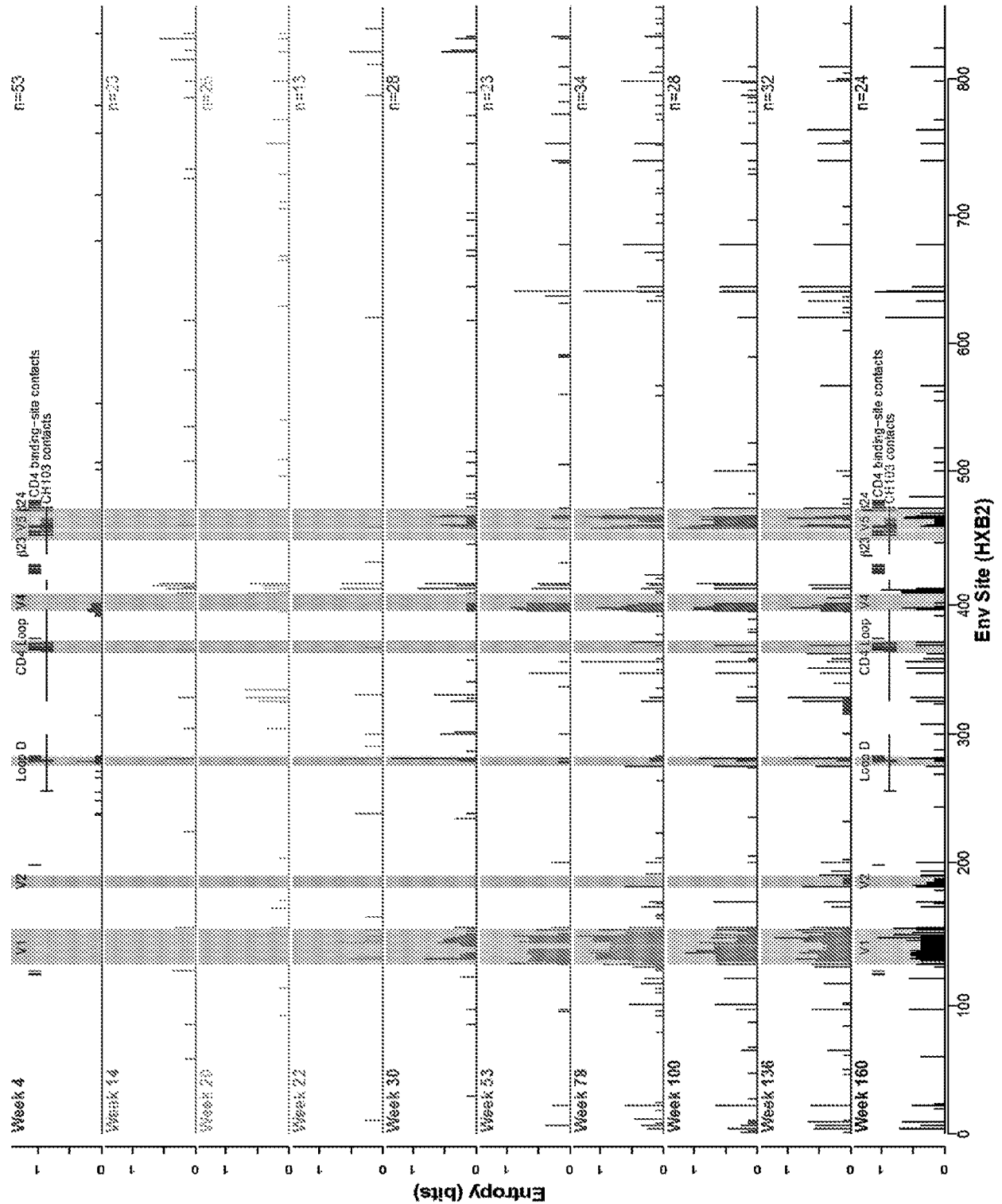
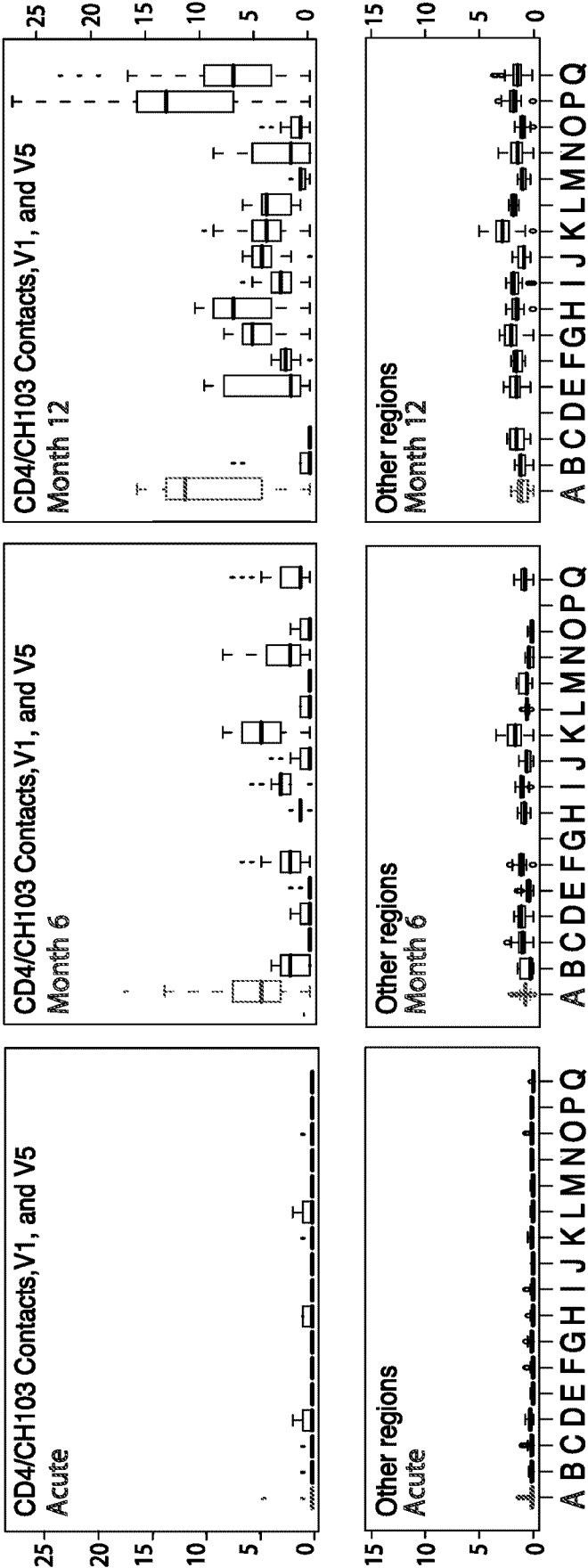
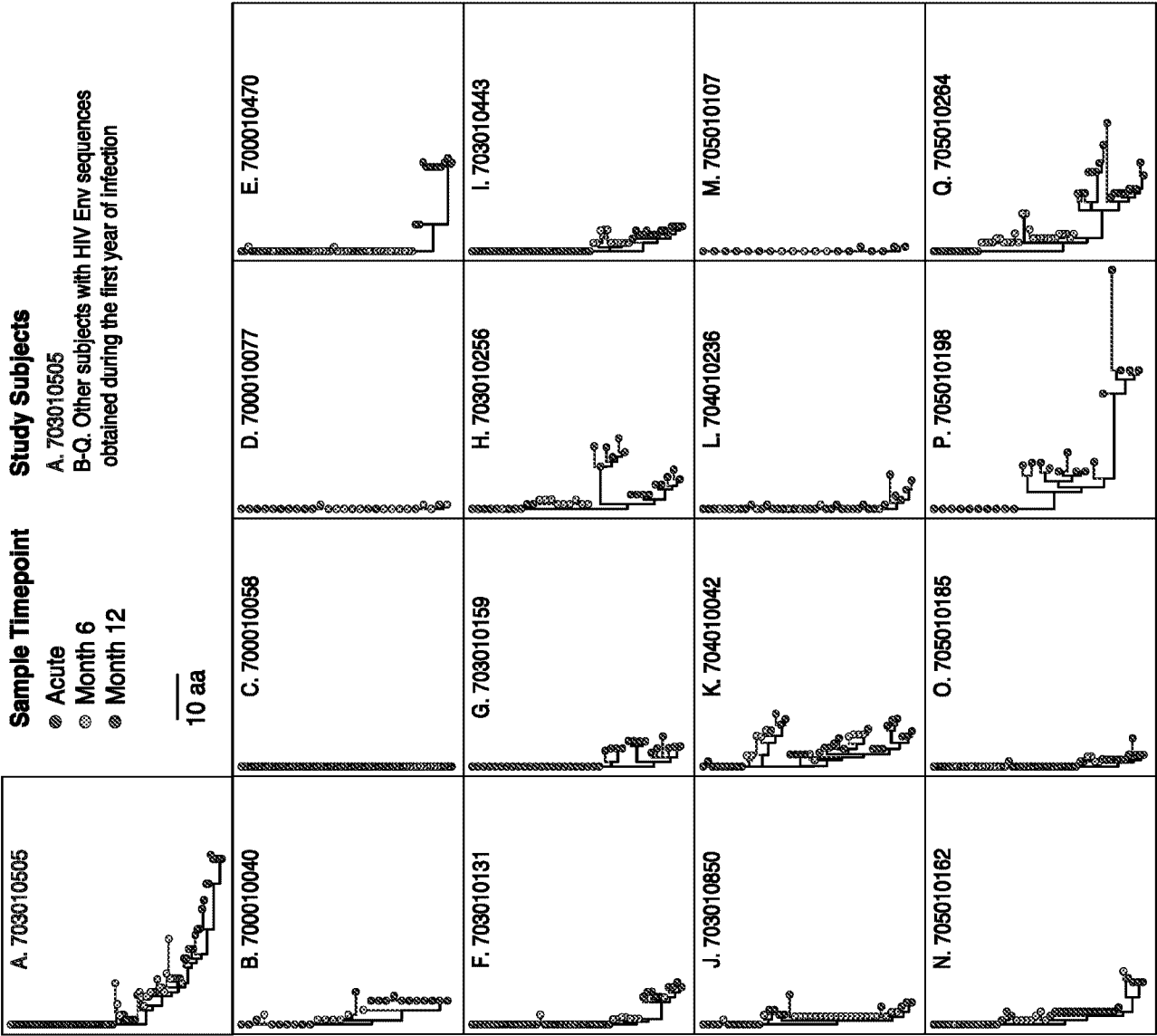


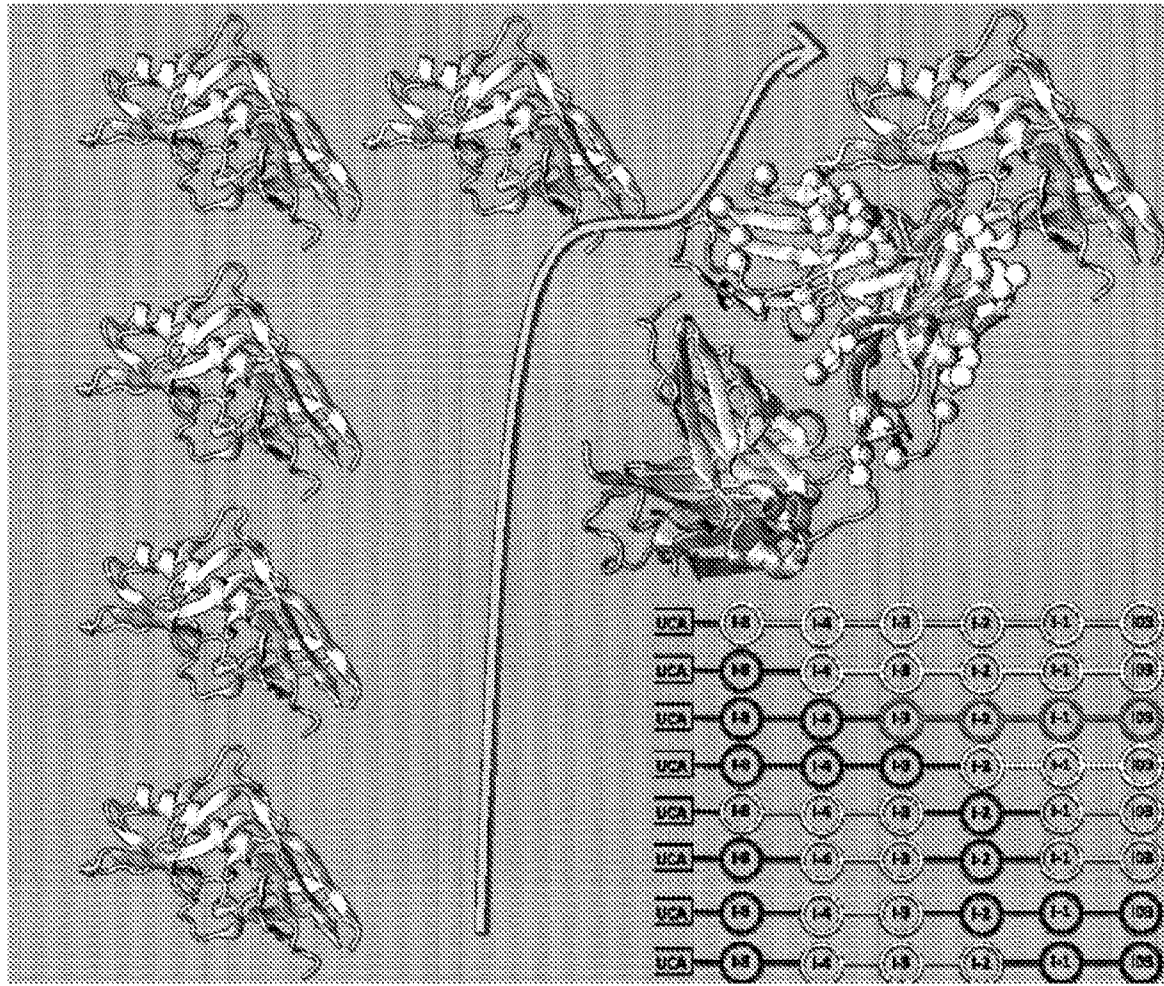
FIG. 15

A comparison of the speed of viral sequence evolution in CH505 in regions relevant to the CH103 epitope to other subjects. a, The distribution of sequence distances expressed as the percentage of amino acids that are different between two sequences, resulting from in a pair-wise comparison of all sequences sampled in a given time point. These are all homogeneous infection cases, so in acute infection there is very little mutation in the CH103 relevant regions, or elsewhere in the virus (left hand panels). By 24 weeks after enrollment (week 30 from infection in CH505, labeled month 6 here as it is approximate), extensive mutations have begun to accrue, focused in CH103 relevant regions (top middle panel), but not in other regions of Env (bottom middle panel). CH103 has the highest ranked diversity among 15 subject sampled in this time frame ($p=0.067$), indicating a focused selective pressure began unusually early in this subject. By 1 year (month 12 indicates samples taken between 10-14 months from enrollment), this region has begun to evolve in many individuals, possibly due to autologous NAb responses that come up later in infection.

a





**FIG. 16**

Co-evolution of virus and antibody - interplay between maturation of antibody CH103 and sequence variability epitope in gp120. The sequence variability (within sample) at each time point is mapped on a gp120 structure that tracks the viral evolution over time from 14 weeks thru 100 weeks post-transmission. Entropy at each residue is color-coded as green to white to red to indicate no sequence variation to slight variation to high sequence variation. This extensive virus within-time point diversity coincides with maturing antibody lineages that ultimately develop breadth. Here we capture the somatic mutations along the CH103 clonal lineage beginning with unmutated common ancestor (UCA) to I-8 to I-4 to I-3 to I-2 to I-1 and to mature CH103. The color balls in heavy (violet) and light (cyan) chains of antibody indicate the appearance/disappearance of somatic mutations during the evolutionary path according to the following scheme. Red balls: mutations appeared in I-8 and remained all the way thru maturation to CH103. Orange balls: mutations appeared in I-4 and remained thru maturation to CH103. Blue balls: mutations that appeared early but are lost before maturing to CH103, and Gray balls: mutations that appeared very late in maturation. Structure of Fab CH103-gp120 from ZM176 66 complex determined in this work is used to map these mutations. The sequence entropy at week 100 is used to pair with CH103 to simply illustrate the relative spatial locations of somatic mutations and sequence variability in gp120. As discussed in the text, viral evolution with time tracks with neutralization breadth and this simple mapping supports that (i) T/F virus began to diversify very early in regions in or proximal to the epitope, and (ii) Somatic mutations that occur early in evolution and remain fixed in heavy chain tend to cluster near the gp120 contact region unlike those mutations that appear later.

FIG. 17A

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FIG. 17A cont'd

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FIG. 17A cont'd

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DMANSTETNSTRTITITHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
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KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVLSLWDQSLKPCVKLTPLCVTLNCTNATASNSSIIEGMKNCFSNITTELRLD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
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EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST

FIG. 17A cont'd

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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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 IGDIREAYCNINESKWNETLQRVSKKLKEYFFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 IGDIREAYCNINESKWNETLQRVSKKLKEYFFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNMKDNW
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLLNGLSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKKLKEYFFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNMKDNW
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST

FIG. 17A cont'd

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 EISNYTEIYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGIIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLITTHSFNCGGEFFYCNTSSLFNRTYMANST
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 GYSPLSLQTLIPSPRGPDRPGGIIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGIIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNNVKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNNVKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNNVKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNNVKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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FIG. 17A cont'd

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 DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
 GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGKIPVVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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 DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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FIG. 17A cont'd

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GYSPLSLQTLIPSPRGPDPRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
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SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIVAGEGTDRIILEFVLGICRAIRNIPTRIRQGFETALL*

>703010505.w4.36

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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIVAGEGTDRIILEFVLGICRAIRNIPTRIRQGFETALL*

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QEMVLKNVTENFNMWKNMDMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIIEGMKNCFSNITTELRLD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST

FIG. 17A cont'd

DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
 SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQGFETALL*
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGKIPVVSTQLLLNGLSLAEGEIIIRSENI TNKVXTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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 IGDIREAYCNINESKWNETLQRVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
 SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQGFETALL*
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGKIPVVSTQLLLNGLSLAEGEIIIRSENI TNKVXTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 GDIREAYCNINESKWNETLQRVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTTRDGGKNNTEFPRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST

FIG. 17A cont'd

VQCTHGKIPVVSTQLLLLNGSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTISRIGPGQAFYATGQV
IGDIRAYCNINESKWNETLQRVSKKLKEYFPKHNTTFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNSTDMA NST
ETNSTRTITIHCRKIQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGGNMKDNWRSELYK
YKVVVKPLGVAPTNAARRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQSNLLKATEAQ
QHMLKLTWVGKIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWEREISNYT
EIIYELLEESQNOQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQGYSPLS
LQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLKGL
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGKIPVVSTQLLLLNGSLAEGEIIIRSENIIVHLNESVKIECTRPNNKTRTISRIGPGQAFYATGQVIGDIRA
YCNINESKWNETLQRVSKKLKEYFPKHNTTFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANSTDMANSTE
TNSTRTITIHCRKIQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGGNMKDNWRSELYK
YKVVVKPLGVAPTNAARRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQSNLLKATEAQ
QHMLKLTWVGKIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWEREISNYT
EIIYELLEESQNOQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQGYSPLS
LQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLKGL
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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
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DREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
TVQCTHGKIPVVSTQLLLLNGSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTISRIGPGQAFYATGQ
VIGDIRAYCNINESKWNETLQRVSKKLKEYFPKHNTTFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIHCRKIQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGGNMKDN
WRSELYKYKVVVKPLGVAPTNAARRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQSNLL
KATEAQQHMLKLTWVGKIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWE
REISNYTEIYELLEESQNOQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGR
SSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLEDTLAIAGGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL
*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVISLWDQSLKPCVKLTPLCVTLNCTNATASNSSIIEGMKNCFSNITTEL RD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGKIPVVSTQLLLLNGSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTISRIGPGQAFYATGQV
IGDIRAYCNINESKWNETLQRVSKKLKEYFPKHNTTFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIHCRKIQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGGNMKDN
WRSELYKYKVVVKPLGVAPTNAARRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQSNLL
KATEAQQHMLKLTWVGKIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWE
REISNYTEIYELLEESQNOQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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KATEAQQHMLKLTWVGKIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWE
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QGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGIKPVVSTQQLLNGSLAEGEIIIRSENITKNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
DMANSTETNSARTITITHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTDRDGGKNNTEFRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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RREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
DMANSTETNSARTITITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTDRDGGKNNTEFRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIIEGMKNCSFNITTELDRD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGIKPVVSTQQLLNGSLAEGEIIIRSENITKNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
DMANSTETNSARTITITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTDRDGGKNNTEFRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
DMANSTETNSARTITITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTDRDGGKNNTEFRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIIEGMKNCSFNITTELDRD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGIKPVVSTQQLLNGSLAEGEIIIRSENITKNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
IGDIRAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
DMANSTETNSRTITITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTDRDGGKNNTEFRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ

FIG. 17A cont'd

GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
 SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIAVGEGTDRIILEFVLGICRAIRNIPTRIRQGFETALL*
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGKIPVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKLKEYFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNQOEKNEQDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQ
 GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNQOEKNEQDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQ
 GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 VQCTHGKIPVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNQOEKNEQDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQ
 GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGKIPVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKLKEYFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNQOEKNEQDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQ
 GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGKIPVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKLKEYFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNQOEKNEQDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQ
 GYSPLSLQTLTIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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 IGDIREAYCNINESKWNETLQRVSKLKEYFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITITHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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FIG. 17A cont'd

KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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QEMVLKNVTENFNMWKNMDMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSII EGMKNCSFNITTEL RD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNAKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITITHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
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RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITITHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIPAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITITHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
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KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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GYSPLSLQTLIPSPRGPDPPGGIEEGGEGQDRNRSTRVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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IGDI REAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITITHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTRPGGGNMKDNW
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST

FIG. 17A cont'd

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 RSELYKYKVVEVKPLGVAPTNNRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDPRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 RREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGFCNNVST
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FIG. 17A cont'd

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FIG. 17A cont'd

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 DMANSTETNSTRTITIHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 GYSPLSLQTLIPSPRGPDPRPGGIIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

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FIG. 17A cont'd

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FIG. 17A cont'd

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 EISNYTEI IYELLEESQNQQEKNQDILLALDRWNSLWNWFNITNWLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDPRGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

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 KAIEAQQHMLKLTVWGTIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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FIG. 17A cont'd

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GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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IGDI REAYCNINESKWNETLQRVSKLKEYFPKHNTTFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
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FIG. 17A cont'd

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 GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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FIG. 17A cont'd

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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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FIG. 17A cont'd

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FIG. 17A cont'd

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FIG. 17A cont'd

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FIG. 17A cont'd

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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKFTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQVSKKLKEYFPFKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNNFNTNWLWYIRIFIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
 SSLKGLRRGWEALKYLGSLVQYWGLELKRSAI SLEDTLAI AVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*
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 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNNL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNNFNTNWLWYIRIFIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 IGDIREAYCNINESKWNETLQVSKKLKEYFPFKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNNFNTNWLWYIRIFIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

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VQCTHGKIPVVSTQLLNGSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
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GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIAVGEGTDRILKFLVLGICRAIRNIPTRIRQGFETALL*
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIAVGEGTDRILKFLVLGICRAIRNIPTRIRQGFETALL*
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QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSFNITTEL RD
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IGDI REAYCNINESKWNETLQRVSKLKEYFPFKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTIKIHCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGGNMKDNW
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KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
EISNYTEIYELLEESQNNQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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IGDI REAYCNINESKWNETLQRVSKLKEYFPFKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
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EISNYTEIYELLEESQNNQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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IGDI REAYCNINESKWNETLQRVSKLKEYFPFKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
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KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
EISNYTEIYELLEESQNNQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNNVKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
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 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEIYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGIEEGGEGQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
 SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIVAGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNNVKTTIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGOV
 IGDIREAYCNINESKWNETLQVSKKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
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 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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 DMANSTETNSTRTTIKIHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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FIG. 17A cont'd

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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
 VQCTHGKIPVVSTQQLLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
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 DMANSTETNSTRTITIHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
 SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLSETLAIAVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
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 IGDIREAYCNINESKWNETLQRVSKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
 DMANSTETNSTRTITIHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
 RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQOQMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
 EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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 IGDIREAYCNINESKWNETLQRVSKLKEYFPHKNITFPQSSGGDLBITTHSFNCGGEFFYCNTSSLPNRTYMANST
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FIG. 17A cont'd

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EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNSTSSLFNRTYMANST
DMANSTETNTRTITITICRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
RSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
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EISNYTEIIYELLEESQNOQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLSLVNVRVQ
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GYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAAYCNINESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNSTSSLFNRTYMANST
DMANSTETNTRTITITICRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGNNMKDNW
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GYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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VQCTHGKIPVVSTQLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
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KAIEAQQHMLKLTWVGIKQLQAEVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDNMTWMQWER
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GYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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FIG. 17A cont'd

QDRNRSTRLVSGFLALAWDDLRLSLCLFIYHRLRDFILIAARAGELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKR
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KAEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTETIIYELLEESQNQQEKNEDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGKIPVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQRVSKLKEYFPKHNTTFQSSGGDLBITTHSFNCGGEFFYCNTSSSLFNRTYMANST
DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTETFRPGGNNMKDNW
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IGDI REAYCNINESKWNETLQRVSKLKEYFPKHNTTFQSSGGDLBITTHSFNCGGEFFYCNTSSSLFNRTYMANST
DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTETFRPGGNNMKDNW
RSELYKYKVVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQSNNLL
KAEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTETIIYELLEESQNQQEKNEDLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQ
GYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
SSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLEDTLATAVGEGETDRILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATANNSSIIEEMKNCSFNITTELDR
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
VQCTHGKIPVSTQQLLLNGSLAEGEIIIRSENITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNINESKWNETLQRVSKLKEYFPKHNTTFQSSGGDLBITTHSFNCGGEFFYCNTSSSLFNRTYMANST
DMANSTETNSTRTITIHCRICKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTETFRPGGNNMKDNW
RSELYKYKVVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQSNNLL

FIG. 17A cont'd

KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIVAGEGTDRIILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
QEMVLKNVTENFNMWKNMDMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATANNSSIIEGMKNCFSNITTELRLD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAYCNISESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITILHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNNMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIVAGEGTDRIILEFVLGICRAIRNIPTRIRQGFETALL*

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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGNIREAYCNISESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITILHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNNMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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DMANSTETNSTRTITILHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNNMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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QEMVLKNVTENFNMWKNMDMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATANNSSIIEGMKNCFSNITTELRLD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAYCNISESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITILHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNNMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLETLAIVAGEGTDRIILEFVLGICRAIRNIPTRIRQGFETALL*

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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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IGDIRAYCNISESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITILHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFPRPGGNNMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
KAIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLGWMGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWER
EISNYTEIIYELLEESQNQOEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNVRVQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRLSLCLFTYHRLRDFILIAARAGELLGR
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KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIRAYCNISESKWNETLQVSKKLKEYFPHKNITFQPSGGDLBITTHSFNCGGEFFYCNTSSLFNRTYMANST

FIG. 17A cont'd

DMANSTETNNTRTITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
 QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATTSSNSSIIEGMKNCSFNITTEL RD
 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLLNGLSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKKLKEYFFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNNTRTITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNLWYIKI FIMIVGGLIGLRI IFAVLSLVNVRVQ
 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLLNGLSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGNIREAYCNINESKWNETLQRVSKKLKEYFFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNNTRTITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
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 RSELYKYKVVEVKPLGVAPTNNRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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 QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATTSSNSSIIEGMKNCSFNITTEL RD
 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLLNGLSLAEGEIIIRSENIITNNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNINESKWNETLQRVSKKLKEYFFPHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
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 RSELYKYKVVEVKPLGVAPTNNRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAIEAQQHMLKLTVWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTGYDIWDMNTWMQWER
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 GYSPLSLQTLIPSPRGPDRPGGI EEEGGEQDRNRSTRVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
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 REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVSTV

FIG. 17A cont'd

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 GDIRKAYCNINESKWNETLQRVSKKLKEYFPKKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANSTD
 MANSTETNNTRTITTHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETFRPGGGNMKDNWR
 SELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQSNLLK
 AIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDNMTWMQWERE
 ISNYTEIIYELLEESQNQOEKNEQDILLALDRWNSLWNNFNTNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQG
 YSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALAWDDLRSLCLFIYHRLRDFILIAARAGELLGRS
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 DKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPCNNVS
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 VIGDIREAYCNISESKWNETLQRVSKKLKEYFPKKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
 TDMANSTETNRTITTHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETFRPGGGNMKDNWR
 SELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQSNLLK
 AIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDNMTWMQWERE
 ISNYTEIIYELLEESQNQOEKNEQDILLALDRWNSLWNNFNTNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQG
 YSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGELLGRS
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 TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNVKTIIVHLNESVKIECTRPNNKTRKTSIRIGPGQAFY
 ATGQVIGDIREAYCNISESKWNETLQRVSKKLKEYFPKKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRITTHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETFRPGGGN
 MKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQ
 QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDNMTW
 MQWEREISNYTEIIYELLEESQNQOEKNEQDILLALDRWNSLWNNFNTNWLWYIKIFIMIVGGLIGLRIIFAVLSLV
 NRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
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 >703010505.w20.25
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 TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNVKTIIVHLNESVKIECTRPNNKTRKTSIRIGPGQAFY
 ATGQVIGDIREAYCNINESKWNETLQRVSKKLKEYFPKKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRITTHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETFRPGGGN
 MKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQ
 QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDNMTW
 MQWEREISNYTEIIYELLEESQNQOEKNEQDILLALDRWNSLWNNFNTNWLWYIKIFIMIVGGLIGLRIIFAVLSLV
 NRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
 ELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAI AVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*
 >703010505.w20.13
 MRVMGIQRNYPQWWIWSMLGFWMMLM CNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
 QEMVLKNVTENFNMWKNDMVDQMHDVLSLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIEGMKNCSFNIT
 TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNVKTIIVHLNESVKIECTRPNNKTRKTSIRIGPGQAFY
 ATGQVIGDIREAYCNINESKWNETLQRVSKKLKEYFPKKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRITTHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETFRPGGGN
 MKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQ
 QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTYGDIWDNMTW
 MQWEREISNYTEIIYELLEESQNQOEKNEQDILLALDRWNSLWNNFNTNWLWYIKIFIMIVGGLIGLRIIFAVLSLV
 NRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG

FIG. 17A cont'd

ELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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QEMVLKNVTENFNMWKNDMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFNIT
TELDRREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPGC
NNVSTVQCTHGIKPVVSTQLLNGSLAEGETIIRSENIINNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
ATGQVIGDIRAAYCNINESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGGEFFYCNTSSLFNRTY
MANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGON
MKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQ
QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNMTW
MQWEREISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLSLV
NRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
ELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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ITTELDRREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTG
PCNNVSTVQCTHGIKPVVSTQLLNGSLAEGETIIRSENIINNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQA
FYATGQVIGDIRAAYCNINESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGGEFFYCNTSSLFNR
TYMANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGG
GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
QQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM
TWMQWEREISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLS
LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
AGELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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QEMVLKNVTENFNMWKNDMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFN
ITTELDRREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTG
PCNNVSTVQCTHGIKPVVSTQLLNGSLAEGETIIRSENIINNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQA
FYATGQVIGDIRAAYCNINESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGGEFFYCNTSSLFNR
TYMANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGG
GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
QQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM
TWI QWEREISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLS
LANRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
AGELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAVGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNDMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFN
ITTELDRREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTG
PCNNVSTVQCTHGIKPVVSTQLLNGSLAEGETIIRSENIINNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQA
FYATGQVIGDIRAAYCNINESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGGEFFYCNTSSLFNR
TYMANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGG
GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
QQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM
TWMQWEREISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLS
LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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TELDRREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPGC
NNVSTVQCTHGIKPVVSTQLLNGSLAEGETIIRSENIINNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
ATGQVIGDIRAAYCNINESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGGEFFYCNTSSLFNRTY
MANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGON
MKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQ
QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNMTW
MQWEREISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLSLV
NRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNDMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFNIT

FIG. 17A cont'd

TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPC
 NNVTQVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
 ATGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFPEKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRIITIHCRITKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNN
 MKDNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQ
 QSNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTW
 MWEREISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVL
 SLVNRVRQGYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
 ELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGGEGTDRILEFVLGICRAIRNIPTRIRQGFET
 ALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCN
 NVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFPEKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYM
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 KDNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQ
 SNNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTW
 QWEREISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVL
 SLVNRVRQGYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
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 ALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGSCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAYCNISESKWNETLQRVSKKLKEYFPEKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMA
 NSTDMANSTETNSTRIITIHCRITKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNM
 DNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQS
 NLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTW
 QWEREISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVL
 SLVNRVRQGYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGSCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAYCNISESKWNETLQRVSKKLKEYFPEKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMA
 NSTDMANSTETNSTRIITIHCRITKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNM
 DNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQS
 NLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTW
 QWEREISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVL
 SLVNRVRQGYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
 ELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGGEGTDRILEFVLGICRAIRNIPTRIRQGFET
 ALL*

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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAYCNISESKWNETLQRVSKKLKEYFPEKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYMA
 NSTDMANSTETNSTRIITIHCRITKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNM
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 NLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTW
 QWEREISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVL
 SLVNRVRQGYSPLSLQTLIPSPRGPDPRGGIEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAG
 ELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGGEGTDRILEFVLGICRAIRNIPTRIRQGFET
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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKNTFTGTGPCN
 NVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFPEKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRTYM
 ANSTDMANSTETNSTRIITIHCRITKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNM
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FIG. 17A cont'd

SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLG MWGCSGKLI CTN VYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNQQEKNEQD LLDALDRWNSLW NWFNITNWLWYIKI FIMIVGGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPD RGGIEEEGGEQDENRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
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ALL*

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QEMVLKNVTENFNMWKNDMVDQM HEDVLSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSI IEGMKNCSPNVT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTE SVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPCN
NVSTVQCTHGKIPVSTQ LLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIR EAYCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIRCR IKTINMWQEVGRAMYAPPIAGNITCISNITG LLLTRDGGKNTETFRPGGGM
KDNWRSELYKYKVVEVKPLGVAPT NARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQ
SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLG MWGCSGKLI CTN VYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNQQEKNEQD LLDALDRWNSLW NWFNITNWLWYIKI FIMIVGGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPD RGGIEEEGGEQDENRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
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ALL*

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ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTE SVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPCN
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TGQVIGDIR EAYCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIRCR IKTINMWQEVGRAMYAPPIAGNITCISNITG LLLTRDGGKNTETFRPGGGM
KDNWRSELYKYKVVEVKPLGVAPT NARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQ
SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLG MWGCSGKLI CTN VYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNQQEKNEQD LLDALDRWNSLW NWFNITNWLWYIKI FIMIVGGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPD RGGIEEEGGEQDENRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
LLGRSSLKGLRRGW EALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRI LEFVLGICRAIRNI PTRIRQGFET
ALL*

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MRVMGIQRNYPQWWIWSMLGFWMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVWATHACVPTDPNP
QEMVLKNVTENFNMWKNDMVDQM HEDVLSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSI IEGMKNCSPNVT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTE SVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPCN
NVSTVQCTHGKIPVSTQ LLLNGSLAEGEIIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIR EAYCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIRCR IKTINMWQEVGRAMYAPPIAGNITCISNITG LLLTRDGGKNTETFRPGGGM
KDNWRSELYKYKVVEVKPLGVAPT NARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQ
SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLG MWGCSGKLI CTN VYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNQQEKNEQD LLDALDRWNSLW NWFNITNWLWYIKI FIMIVGGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPD RGGIEEEGGEQDENRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
LLGRSSLKGLRRGW EALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRI LEFVLGICRAIRNI PTRIRQGFET
ALL*

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QEMVLKNVTENFNMWKNDMVDQM HEDVLSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSI IEGMKNCSPNVT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTE SVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPCN
NVSTVQCTHGKIPVSTQ LLLNGSLAEGEIIIRSENITNNGKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIR EAYCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSG#DLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIRCR IKTINMWQEVGRAMYAPPIAGNITCISNITG LLLTRDGGKNTETFRPGGGM
KDNWRSELYKYKVVEVKPLGVAPT NARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQ
SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLG MWGCSGKLI CTN VYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNQQEKNEQD LLDALDRWNSLW NWFNITNWLWYIKI FIMIVGGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPD RGGIEEEGGEQDENRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
LLGRSSLKGLRRGW EALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRI LEFVLGICRAIRNI PTRIRQGFET
ALL*

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QEMVLKNVTENFNMWKNDMVDQM HEDVLSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSI IEGMKNCSPNVT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTE SVITQACPKVSFDPIPIHYCAPAGYAILKCNKFTTGTGPCN
NVSTVQCTHGKIPVSTQ LLLNGSLAEGEIIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIR EAYCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIRCR IKTINMWQEVGRAMYAPPIAGNITYI SNITG LLLTRDGGKNTETFRPGGGMKDNW
RSELYKYKVVEVKPLGVAPT NARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQOQSNLL
KAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLG MWGCSGKLI CTN VYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNQQEKNEQD LLDALDRWNSLW NWFNITNWLWYIKI FIMIVGGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPD RGGIEEEGGEQDENRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
LLGRSSLKGLRRGW EALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRI LEFVLGICRAIRNI PTRIRQGFET
ALL*

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FIG. 17A cont'd

MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVFVWKEAKTTFLFCASDAKAYEKEVHNVWATHACVPTDPNP
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REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVSTV
QCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNKTI IIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQVI
GDI REAYCNI SESKWNETLQRVSKKLKEYFPFKNITFPQSSGGDL EITTHSFNCGGEFFYCNTSSLFNRTYMANST
MANSTETNSTRIITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNMKDNWR
SELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQQQSNLLK
AIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWERE
ISNYTEIIYELLEESQNQKEKNEQDILLALDRWNSLWNNFNI TNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQG
YSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGRS
SLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGGEGTDRILEFVLGICRAIRNIPTIRIQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVFVWKEAKTTFLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSFNITTELRD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNKTI IIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQVI
IGDI REAYCNI SESKWNETLQRVSKKLKEYFPFKNITFPQSSGGDL EITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRIITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNMKDNWR
RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQQQSNLLK
KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWERE
ISNYTEIIYELLEESQNQKEKNEQDILLALDRWNSLWNNFNI TNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQG
GYSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGGEGTDRILEFVLGICRAIRNIPTIRIQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVFVWKEAKTTFLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSFNITTE
LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGSCNN
VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNKTI IIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
GQVIGDI REAYCNI SESKWNETLQRVSKKLKEYFPFKNITFPQSSGGDL EITTHSFNCGGEFFYCNTSSLFNRTYMA
NSTDMANSTETNSTRIITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNMK
DNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQQQSNLLK
KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQ
WEREISNYTEIIYELLEESQNQKEKNEQDILLALDRWNSLWNNFNI TNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQG
GYSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGE
LLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGGEGTDRILEFVLGICRAIRNIPTIRIQGFET
ALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVFVWKEAKTTFLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSFNITTELRD
REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVSTV
QCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNKTI IIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQVI
GDI REAYCNI SESKWNETLQRVSKKLKEYFPFKNITFPQSSGGDL EITTHSFNCGGEFFYCNTSSLFNRTYMANST
MANSTETNSTRIITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNMKDNWR
SELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQQQSNLLK
AIEAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWERE
ISNYTEIIYELLEESQNQKEKNEQDILLALDRWNSLWNNFNI TNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNRVRQG
YSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAGE
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ALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVFVWKEAKTTFLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSFNITTELRD
REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVSTV
QCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNKTI IIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
ATGQVIGDI REAYCNI SESKWNETLQRVSKKLKEYFPFKNITFPQSSGGDL EITTHSFNCGGEFFYCNTSSLFNRTY
MANSTDMANSTETNSTRIITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNN
MKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQQ
QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQ
QWEREISNYTEIIYELLEESQNQKEKNEQDILLALDRWNSLWNNFNI TNWLWYIKIFIMIVGGLIGLRI IFAVLSLV
NRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRLVSGFLALVWDDLRLSLCLFIYHRLRDFILIAARAG
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TALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVFVWKEAKTTFLFCASDAKAYEKEVHNVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMDVDMHEDVI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSFNITTE
LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNN
VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNKTI IIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
GQVIGDI REAYCNI SESKWNETLQRVSKKLKEYFPFKNITFPQSSGGDL EITTHSFNCGGEFFYCNTSSLFNRTYMA
NSTDMANSTETNSTRIITIHCRIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNETETFRPGGNNMKDNWR
SELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQQQSNLLKAI
EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWEREISNYTEIIY

FIG. 17A cont'd

LLEESQNQQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQGYSPLSLQTLI
PSRPGPDRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLKGLRRGWE
ALKYLGSLVQYWGLELKRSAISLLDTLAIAGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATAS#ISVIEEMKNCSFNITTELDRD
KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGKIPVYSTQLLNGSLAEGEIIIRSENITDNVKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDI REAYCNISESKWNETLQVRSKKLKEYFFHKNITFPQSSGGDLITTHSFNCGGEFFYCNTSSLFNRITYMANST
ETNSTRTITIRCRKIQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTETFRPGGNNMKDNWRSEL
YKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQSNLLKAI
AQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQWEREISN
YTEI IYELLEESQNQQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVRQGYSP
LSLQTLIPSRPGPDRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLK
GLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWVWATHACVPTDPNP
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ELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCN
NVSTVQCTHGKIPVYSTQLLNGSLAEGEIIIRSENITNNAKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDI REAYCNISESKWNETLQVRSKKLKEYFFHKNITFPQSSGGDLITTHSFNCGGEFFYCNTSSLFNRITYM
ANSTDMANSTETNSTRTITIRCRKIQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTETFRPGGNNMK
DNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQSNLLKAI
SGIVQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQ
WEREISNYTEI IYELLEESQNQQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNR
VRQGYSPLSLQTLIPSRPGPDRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLK
GLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNN
VSTVQCTHGKIPVYSTQLLNGSLAEGEIIIRSENITNNDKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
GQVIGDI REAYCNISESKWNETLQVRSKKLKEYFFHKNITFPQSSGGDLITTHSFNCGGEFFYCNTSSLFNRITYM
NSTDMANSTETNSTRTITIRCRKIQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTETFRPGGNNMK
DNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQSNLLKAI
NLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQ
WEREISNYTEI IYELLEESQNQQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNR
VRQGYSPLSLQTLIPSRPGPDRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLK
GLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWVWATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATNATASNSSIIEGMKNCSFNITTE
LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGSCNN
VSTVQCTHGKIPVYSTQLLNGSLAEGEIIIRSENITDNKGTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYAT
GQVIGDI REAYCNISESKWNETLQVRSKKLKEYFFHKNITFPQSSGGDLITTHSFNCGGEFFYCNTSSLFNRITYM
NSTDMANSTETNSTRTITIRCRKIQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTETFRPGGNNMK
DNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQSNLLKAI
QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQ
WEREISNYTEI IYELLEESQNQQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNR
VRQGYSPLSLQTLIPSRPGPDRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLK
GLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAIAGEGTDRILEFVLGICRAIRNIPTRIRQGFETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWVWATHACVPTDPNP
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ELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNN
NVSTVQCTHGKIPVYSTQLLNGSLAEGEIIIRSENITNNAKTIIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDI REAYCNISESKWNETLQVRSKKLKEYFFHKNITFPQSSGGDLITTHSFNCGGEFFYCNTSSLFNRITYM
ANSTDMANSTETNSTRTITIRCRKIQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTETFRPGGNNMK
DNWRSELYKYKVVEVKPLGVAPTNNRRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQSNLLKAI
QSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGC SGKLICTTNVYWNSSWSNKTYGDIWDMNTWMQ
WEREISNYTEI IYELLEESQNQQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLSLVNR
VRQGYSPLSLQTLIPSRPGPDRPGGIIEEGGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLGRSSSLK
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QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATNATASNSSIIEGMKNCSFNITTE

FIG. 17A cont'd

ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGFCN
 NVSTVQCTHGKIPVVSTQLLNGSLAEGETIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFFHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRITYM
 ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGG
 NMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQ
 QQSNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDNMT
 WMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRIIFAVLSL
 VNRVRQGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARA
 GELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
 ETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNVTENFNMWKNMVDQMHEVDVSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSIIEGMKNCSFNITT
 ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGFCN
 NVSTVQCTHGKIPVVSTQLLNGSLAEGETIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFFHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRITYM
 ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGG
 NMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQ
 QQSNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDNMT
 WMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRIIFAVLSL
 VNRVRQGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARA
 GELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
 ETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNVTENFNMWKNMVDQMHEVDVSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSIIEGMKNCSFNITT
 ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGFCN
 NVSTVQCTHGKIPVVSTQLLNGSLAEGETIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFFHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRITYM
 ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGG
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 QQSNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDNMT
 WMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRIIFAVLSL
 VNRVRQGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARA
 GELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
 ETALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNVTENFNMWKNMVDQMHEVDVSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSIIEGMKNCSFNITT
 ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGFCN
 NVSTVQCTHGKIPVVSTQLLNGSLAEGETIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFFHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRITYM
 ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGG
 NMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQ
 QQSNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDNMT
 WMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRIIFAVLSL
 VNRVRQGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARA
 GELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
 ETALL*

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 QEMVLKNVTENFNMWKNMVDQMHEVDVSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSIIEGMKNCSFNITT
 ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGFCN
 NVSTVQCTHGKIPVVSTQLLNGSLAEGETIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFFHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRITYM
 ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGGNM
 KDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQ
 QQSNLLKAI EAQOHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDNMT
 WMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRIIFAVLSL
 VNRVRQGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARA
 GELLGRSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
 ALL*

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MRVMGIQRNYPQWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNVTENFNMWKNMVDQMHEVDVSLWDQSLKPCVKLTPLCVTLNCTNATTNATASNSSIIEGMKNCSFNITT
 ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGFCN
 NVSTVQCTHGKIPVVSTQLLNGSLAEGETIIRSENITNNAKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
 TGQVIGDIRAYCNISESKWNETLQRVSKKLKEYFFHKNITFQPSGGGLEITTHSFNCGGEFFYCNTSSLFNRITYM
 ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNPETFRPGGGNM
 KDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGLGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQ

FIG. 17A cont'd

SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
LLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGFET
ALL*

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MRVMGIQRNY PQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATANATASNSSI IBOGKNCSFNIT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPCN
NVSTVQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIRAEXCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIXHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGNN
KDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQ
SNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGE
LLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGFET
ALL*

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MRVMGIQRNY PQWWIWSMLGFWMMLMICNGMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATANATASNSSI IBOGKNCSFNIT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPCN
NVSTVQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIRAHCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGG
NMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQ
QSSNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARA
GELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
ETALL*

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QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATANATASNSSI IBOGKNCSFNIT
ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPCN
NVSTVQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIRAHCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIKHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGG
NMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQ
QSSNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARA
GELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
ETALL*

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ELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPCN
NVSTVQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYA
TGQVIGDIRAHCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
ANSTDMANSTETNSTRTITIHCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGGG
NMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIVQ
QSSNLLKAI EAQOHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKTYGDIWDNM TW
QWEREISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARA
GELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
ETALL*

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QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATANATASNSSI IBOGKNCSFNIT
TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPCN
NVSTVQCTHGKIPVSTQLLNGSLAEGEIIIRSENITNNVKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
ATGQVIGNIREAYCNISESKWNETLQRVSKKLKEYFFHKNITFPQSSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYM
MANSTDMANSTETNSTRTITIHCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTTETFRPGG
GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVOARQLLSGIV
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QWEREISNYTEI IYELLEESQNOQEKNEQDLLALDRWNSLWNWPNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVN
RVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARA
GELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVGEGTDRILEFVLGICRAIRNIPTRIRQGF
ETALL*

FIG. 17A cont'd

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MRVMGIQRNYPOWWIWSMLGFWMMLMCNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 TELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPGC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIINNVTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
 ATGQVIGNIREAYCNISESKNWETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRTITITHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTDTETFRPGG
 GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
 QQQSLLKAI EAQQHMLKLTWVGKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDM
 TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLS
 LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
 AGEILLGRSSSLKGLRRGWREALKYLGSLVQYWGLELKRSAISLDDTLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQG
 FETALL*

>703010505.w30.34

MRVMGIQRNYPOWWIWSMLGFWMMLMCNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 MANSTDMANSTETNSTRTITIPCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTDTETFRPGG
 GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
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 TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLS
 LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
 AGEILLGRSSSLKGLRRGWREALKYLGSLVQYWGLELKRSAISLDDTLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQG
 FETALL*

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MRVMGIQRNYPOWWIWSMLGFWMMLMCNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNTNFNMWKNMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFNIT
 TELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPGC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIINNVTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
 ATGQVIGNIREAHNCNISESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRTITILHCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTDTETFRPGG
 GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
 QQQSLLKAI EAQQHMLKLTWVGKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDM
 TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLS
 LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
 AGEILLGRSSSLKGLRRGWREALKYLGSLVQYWGLELKRSAISLDDTLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQG
 FETALL*

>703010505.w30.15

MRVMGIQRNYPOWWIWSMLGFWMMLMCNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNTNFNMWKNMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFNIT
 TELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPGC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIINNVTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
 ATGQVIGNIREAHNCNISESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTDTETFRPGG
 GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
 QQQSLLKAI EAQQHMLKLTWVGKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDM
 TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLS
 LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
 AGEILLGRSSSLKGLRRGWREALKYLGSLVQYWGLELKRSAISLDDTLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQG
 FETALL*

>703010505.w30.30

MRVMGIQRNYPOWWIWSMLGFWMMLMCNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNTNFNMWKNMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATASNATASNSSIIEGMKNCSFNIT
 TELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPGC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIINNVTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY
 ATGQVIGNIREAHNCNISESKWNETLQRVSKKLKEYFPHKNIITFQPSGGDLEITTHSFNCGGEFFYCNTSSLFNRTY
 MANSTDMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNNTDTETFRPGG
 GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
 QQQSLLKAI EAQQHMLKLTWVGKQLQARVLALERYLKDQQLLGMWGCSCGKLICTTNVYWNSSWSNKTYGDIWDM
 TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRIIFAVLS
 LVNRVRQGYSPLSLQTLIPSPRGPDPRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAAR
 AGEILLGRSSSLKGLRRGWREALKYLGSLVQYWGLELKRSAISLDDTLAIAVGEGETDRILEFVLGICRAIRNIPTRIRQG
 FETALL*

>703010505.w30.33

MRVMGIQRNYPOWWIWSMLGFWMMLMCNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 TELRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPGC
 NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIINNVTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFY

FIG. 17A cont'd

ATGQVIGDIREAYCNISESKWNETLQRVSKKLKEYFPHKNITFQPSGGDELEITTHSFNCGGEFFYCNTSSLFNRTY
MANSTDMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGG
GNMKDNWRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIV
QQQSNLLKAI EAQQHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKT YGDIWDM
TWMQWEREISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLS
LVNRVRQGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAAR
AGELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLEDTLAI AVGEGTDRI LEFVLGICRAIRNIPTRIRQG
FETALL*

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MRVMGIQRNYPQWWIWSMLGFWMIMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHDVIVSLWDQSLKPCVKLTPLCVTLNCTNATSNSSII EGMKNCSFNITTELDRD
REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
QCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNDKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIREAYCNISESKWNETLQRVSKKLKEYFPHKNITFQPSGGDELEITTHSFNCGGEFFYCNTSSLFNRTYMANST
MANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGGMKDN
WRSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNL
LKAIEAQHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKT YGDIWDMTWMQW
REISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLG
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*

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REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNDKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIREAYCNISESKWNETLQRVSKKLKEYFPHKNITFQPSGGDELEITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGGMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNL
LKAIEAQHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKT YGDIWDMTWMQW
REISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLG
RSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLEDTLAI AVGEGTDRI LEFVLGICRAIRNIPTRIRQGFETALL*

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REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNDKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIREAYCNISESKWNETLQRVSKKLKEYFPHKNITFQPSGGDELEITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGGMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNL
LKAIEAQHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKT YGDIWDMTWMQW
REISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLG
RSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLEDTLAI AVGEGTDRI LEFVLGICRAIRNIPTRIRQGFETALL*

>703010505.w30.13

MRVMGIQRNYPQWWIWSMLGFWMIMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNWATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHDVIVSLWDQSLKPCVKMTPLCVTLNCTNATSNSSII EGMKNCSFNITTELDRD
REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNDKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIREAYCNISESKWNETLQRVSKKLKEYFPHKNITFQPSGGDELEITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGGMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNL
LKAIEAQHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKT YGDIWDMTWMQW
REISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLG
RSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLEDTLAI AVGEGTDRI LEFVLGICRAIRNIPTRIRQGFETALL*

>703010505.w30.19

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QEMVLKNVTENFNMWKNMVDQMHDVIVSLWDQSLKPCVKMTPLCVTLNCTNATSNSSII EGMKNCSFNITTELDRD
REKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKKTFTGTGPCNNVST
VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TNNDKTIIVHLNESVKIECTRPNNKTRTSIRIGPGQAFYATGQV
IGDIREAYCNISESKWNETLQRVSKKLKEYFPHKNITFQPSGGDELEITTHSFNCGGEFFYCNTSSLFNRTYMANST
DMANSTETNSTRTITIRCRICKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTDTEFRPGGGMKDNW
RSELYKYKVVEVKPLGVAPTNAARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNL
LKAIEAQHMLKLT VWGIKQLQARVLALERYLKDQQLLGMWGCSGKLICTTNVYWNSSWSNKT YGDIWDMTWMQW
REISNYTEI IYELLEESQNQQEKNEQDLLALDRWNSLWNWFNITNLWYIKIFIMIVGGLIGLRIIFAVLSLVNRVR
QGYSPLSLQTLIPSPRGPDRPGGIEEGEGEQDRNRSTRLVSGFLALVWDDLRSCLCFIYHRLRDFILIAARAGELLG
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FIG. 17A cont'd

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 NCSFNITTELRLDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNT
 FTGTGPCNNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITNGKTTIVHLNESVKIECTRFNNKTRTSIRI
 GPGQAFYATGQVIGDIRAYCNISESKNWNETLQRVSKKLKEYFPFKNITFQPSGGGLEIITHSFNCGGEFFYCNTS
 SLFNRTYMANSTDMANSTETNSTRTITIRCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNTDET
 FRPGGGNMKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQL
 LSGIVQQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYD
 IWDNMTWMQWEREISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI I
 FAVLSLVNVRVROGYSPLSLQTLIPSPRGPDRPGGLEEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFI
 LIAARAGELLGRSSSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVAGEGTDRILEFVLGICRAIRNIPT
 RIRQGFETALL*

>703010505.w30.14

MRVMGIQRNYPOWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
 QEMVLKNVTENFNMWKNMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEEMKNCSFNITTELRLD
 KREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTGTGPCNNVST
 VQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITDNVKTIVHLNESVKIECTRFNNKTRTSIRIGPGQAFYATGQV
 IGDIREAYCNISESKNWNETLQRVSKKLKEYFPFKNITFQPSGGGLEIITHSFNCGGEFFYCNTSSLFNRTYMANST
 DMANSTETNSTRTITIRCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNTDETFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 QLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDNMTWMQWEREISNYTEI IYELLEESQ
 NOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNVRVROGYSPLSLQTLIPSPRGP
 DRPGGLEEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGELLGRSSSLKGLRRGWEALKYLG
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>703010505.w53.15

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 QEMVLKNVTENFNMWKNMVDQMHEDEVISLWDQSLKPCVKLTPLCVTLNCTNATASNSSILEGMKNCSFNITTE
 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTNGTGPCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITDNGKTTIVHLNESVKIECTRFNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAHACNISESKWNETLQRVSKKLKEYFPFKNITFQPSGGGLEIITHSFNCGGEFFYCNTSSLFNRTYMA
 NSTETNSTRTITILHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNTDETFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDNMTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGLEEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGELLGR
 SSKGLRRGWEALKYLGSLVQYWGLELKRSAISLDDTLAIVAGEGTDRILEFVLGICRAIRNIPTIRQGFETALL*

>703010505.w53.27

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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTNGTGPCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITDNGKTTIVHLNESVKIECTRFNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAHACNISESKWNETLQRVSKKLKEYFPFKNITFQPSGGGLEIITHSFNCGGEFFYCNTSSLFNRTYMA
 NSTETNSTRTITILHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNTDETFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDNMTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGLEEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGELLGR
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>703010505.w53.3

MRVMGIQRNYPOWWIWSMLGFWMMLMICNGMWVTVYYGVVWKEAKTTLFCASDAKAYEKEVHNVWATHACVPTDPNP
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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTNGTGPCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITDNGKTTIVHLNESVKIECTRFNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAHACNISESKWNETLQRVSKKLKEYFPFKNITFQPSGGGLEIITHSFNCGGEFFYCNTSSLFNRTYMA
 NSTETNSTRTITILHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNTDETFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDNMTWMQWER
 EISNYTEI IYELLEESQNOQEKNEQDILLALDRWNSLWNWFNITNWLWYIKIFIMIVGGLIGLRI IFAVLSLVNVRVRO
 GYSPLSLQTLIPSPRGPDRPGGLEEEGGEQDRNRSTRLVSGFLALVWDDLRSLCLFIYHRLRDFILIAARAGELLGR
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 LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPPIPIHYCAPAGYAILKCNKNTFTNGTGPCNN
 VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENIITDNGKTTIVHLNESVKIECTRFNNKTRTSIRIGPGQAFYAT
 GQVIGDIRAHACNISESKWNETLQRVSKKLKEYFPFKNITFQPSGGGLEIITHSFNCGGEFFYCNTSSLFNRTYMA
 NSTETNSTRTITILHCRIKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNTDETFRPGGGNMKDNW
 RSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLL
 KAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSCGLICTTNVYWNSSWSNKTGYDIWDNMTWMQWER

FIG. 17A cont'd

ETISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVLSLVNVRVRQ
GYSPLSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRVSGFLALAWDDLRSLCLFIYHRLRDFILIAARAGELLGR
SSLKGLRRGWEALKYLGSLVQYWGLELKRSAISLLDTLAI AVGEGTDRI LEFVLGICRAIRNIPTRIROGFETALL*
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MRVMGIQRNYPOWWIWSMLGFWMMLMNCNMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTDATASNATAINISIEEMKNCSPNIT
TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPC
NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TDNGKTIIVHLNESVKLECTRPSNNTRTSIRIGPGQAFY
ATGQVIGDIREAHCNISESKWNETLQRVSKKLKEYFPKHNI TFQSSGGGLEITTHSFNCGGEFFYCNTSSLFNRTY
MVNSTDMANSTETNSTRIITIRCRKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTETFRPGG
GGNMKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGI
VQQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDN
MTWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVL
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SPNITTELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIH#MANSTDNSTRIITIR
RIKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTETFRPGGGNMKDNWRSELYKYKVVEVKPL
GVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGIVQQQSNLLKAI EAQQHMLKLTW
GIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDNMTWMQWEREISNYTEIIYELLE
SQNOQEKNEQDILLALDRWNSLWNNWFNITNWL*YIKIFIMIVGGGLIGLRIIFAVLSLVNVRVRQGYSPSLQTLIPSP
RGPDPRPGGIEEGGGEQDRNRSTRVSGFLALAWDDLRSLCLFIYHRLRDFILTAARAGELLGRSSLKGLRRGWEALKY
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>703010505.w53.10

MRVMGIQRNYPOWWIWSMLGFWMMLMNCNMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
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SPNITTELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFN
GTGPCNNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TDNGKTIIVHLNESVKLECTRPSNNTRTSIRIGP
GQAFYATGQVIGDIREAHCNISESKWNETLQRVSKKLKEYFPKHNI TFQSSGGGLEITTHSFNCGGEFFYCNTSS
FNRTYMANSTETNSTRIITIRCRKQI INMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGKNNTEFTETFRPGG
GNMKDNWRSELYKYKVVEVKPLGVAPTNNARRRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTVQARQLLSGI
VQQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDN
TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVL
SLVNRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRVSGFLALAWDDLRSLCLFIYHRLRDFILIAA
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GFETALL*

>703010505.w53.29

MRVMGIQRNYPOWWIWSMLGFWMMLMNCNMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
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TELDRKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPC
NNVSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TDNGKTIIVHLNESVKLECTRPSNNTRTSIRIGPGQAFY
ATGQVIGDIREAHCNISESKWNETLQRVSKKLKEYFPKHNI TFQSSGGGLEITTHSFNCGGEFFYCNTSSLFNRTY
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VQQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDN
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SLVNRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRVSGFLALAWDDLRSLCLFIYHRLRDFILIAA
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GFETALL*

>703010505.w53.14

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VQQQSNLLKAI EAQQHMLKLTWVGIKQLQARVLALERYLKDQQLLGMWGCSSGKLICTTNVYWNSSWSNKTYGDIWDN
TWMQWEREISNYTEIIYELLEESQNOQEKNEQDILLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGGLIGLRIIFAVL
SLVNRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGGEQDRNRSTRVSGFLALAWDDLRSLCLFIYHRLRDFILIAA
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MRVMGIQRNYPOWWIWSMLGFWMMLMNCNMWVTVYYGVPVWKEAKTTLFCASDAKAYEKEVHNWVATHACVPTDPNP
QEMVLKNVTENFNMWKNMVDQMHEVDI SLWDQSLKPCVKLTPLCVTLNCTNATASNSSIEGMKNCSPNITTEL
LRDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSVITQACPKVSFDPIPIHYCAPAGYAILKCNKTFNGTGPCNN
VSTVQCTHGIKPVVSTQLLNGSLAEGEIIIRSENI TDNGKTIIVHLNESVKLECTRPSNNTRTSIRIGPGQAFYAT

FIG. 17A cont'd

GQVIGDIREAHCNISESKWNETLQRVSKKLKEYFPFKNITFQPSGGDLEITTHSFNCGGEFFYCNTSSLFNRTYMA
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FNRTYMANSTDMANSTETNSTRITITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGNNNTETFE
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DIWDMNTWQWEREISNYTEIYELLEESQNOQKEQDQLLALDRWNSLWNNWFNITNWLWYIKIFIMIVGGGLIGLRI
IFAVLSLVNRVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGEQDRKRSTLVSGFLALVWDDLRLSLCLFIYHRLRDF
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SFNITTELRLDKREKKNALFYKLDIVQLDGNSSQYRLINCNTSAITQACPKVSFDPIPIHYCAPAGYAILKCNKTFN
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GQAFYATGQVIGDIREAHCNISESKWNETLQRVSKKLKEYFPFKNITFQPSGGDLEITTHSFNCGGEFFYCNTSSL
FNRTYMANSTETNSTRITITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGNNNTETFEFTRPGGGM
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ALL*

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FNRTYMANSTETNSTRITITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGNNNTETFEFTRPGGGM
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RVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALAWDDLRLSLCLFIYHRLRDFILIAARAGE
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ALL*

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FNRTYMANSTETNSTRITITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGNNNTETFEFTRPGGGM
KDNWRSELYKYKVVEVKPLGVAPTNAARRVVEREKRAVGMGAVFLGFLGAAGSTMGAASITLTQARQLLSGIVQQQ
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ALL*

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FNRTYMANSTETNSTRITITIRCRKQIINMWQEVGRAMYAPPIAGNITCISNITGLLLTRDGGNNNTETFEFTRPGGGM
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RVRQGYSPSLQTLIPSPRGPDPRPGGIEEGGEQDNRSTRLVSGFLALAWDDLRLSLCLFIYHRLRDFILIAARAG
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ALL*

DEMANDE OU BREVET VOLUMINEUX

LA PRÉSENTE PARTIE DE CETTE DEMANDE OU CE BREVET COMPREND PLUS D'UN TOME.

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CONTENANT LES PAGES 1 À 70

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