

Table 1. Mutations identified in viral RNA amplified from serum of the persistently infected tamarin T12025 between weeks 40 and 48 of infection

Nucleotide Position	pGBV-B/2 AY243572	T12025 Virus	Location	AA Residue	pGBV-B/2	T12025 Virus
1612	A	G	E2	389	Pro	-
1849	C	T	E2	468	Gly	-
2783	T	G	NS2	780	Phe	Glu
3001	T	C	NS2	852	Val	-
3793	A	C	NS3	1116	Gly	-
4396	C	A	NS3	1317	Asn	Lys
4435	T	C	NS3	1330	Tyr	-
5090	A	G	NS3	1549	Thr	Ala
5212	T	G	NS4A	1589	Arg	-
5225	G	A	NS4A	1594	Ala	Thr
5305	C	T	NS4B	1620	Pro	-
6061	T	C	NS5A	1872	Ile	-
6993	T	C	NS5A	2183	Met	Thr
7031	T	C	NS5A	2196	Phe	Leu
7152	T	C	NS5A	2236	Val	Ala
7248	G	A	NS5A	2268	Gly	Glu
7303	C	T	NS5B	2286	Phe	-
7357	C	T	NS5B	2304	Leu	-
7711	C	T	NS5B	2422	Thr	-
8402	T	C	NS5B	2653	Tyr	His
8942	A	G	NS5B	2833	Ile	Val
8969	T	G	NS5B	2842	Phe	Val
9299	G	A	3'NTR*	NA	NA	NA

Nonsilent mutations are bold. –, amino acids identical to parental GBV-B/2 residues. Mutations at nucleotides 5090, 6061, and 8942 were present at week 40 or 42, but partly or completely reverted to the pGBV-B/2 sequence at week 44 or 48. NA, not applicable. The two nonsilent mutations (Ala-1594 → Thr and Val-2236 → Ala in NS4A and NS5A, respectively) eliminate two of the three amino acid differences that distinguish the pGBV-B/2

sequence from that of the infectious molecular clone (GenBank accession no. AF179612) reported by Bukh *et al.* (1).

*3' NTR, 3' nontranslated RNA.

1. Bukh, J., Apgar, C. L. & Yanagi, M. (1999) *Virology* **262**, 470–478.