Supplemental Table S1: Baseline polymorphisms and emergent resistance-associated variants in patient who experienced virologic failure

Patient	Virologic outcome	HCV target gene	Baseline polymorphisms at amino acid positions	Emergent resistance- associated variants
Patient 1 (Group 3 GT 1b)	Viral breakthrough at Week 6	NS3 NS5A NS5B	Sequence analysis ongoing	Sequence analysis ongoing
Patient 2 (Group 4 GT 1a)	Viral breakthrough at Week 8 ^a	NS3 NS5A NS5B	none L31M none	R155K Q30R, L31M P495L
Patient 3 (Group 4 GT 1a)	Relapse at follow- up Week 4	NS3 NS5A NS5B	V36M H56P none	V36M, R155K M28A, Q30R, H58P P495L

Note: Detected polymorphisms at amino acid positions associated with drug/class resistance are shown. NS3-V36M and NS5A-H58P by themselves conferred minimal (< 3-fold) resistance to asunaprevir and daclatasvir, respectively, when tested in cell-based HCV replication assays.

Supplemental Table S2: Hemoglobin (g/dL) levels overtime ^a

Group	1	2	3	4
BMS-791325 dose, mg	75 24	75 12	150 24	150 12
Treatment duration, weeks				
N	16	16	16	18
Baseline mean value	14.8 (1.21)	14.7 (1.02)	14.2 (1.12)	14.6 (1.29)
Mean change from baseline to:				
Treatment Week 4	-0.92 [0.14]	-0.75 [0.17]	-0.64 [0.18]	-0.42 [0.19]
Treatment Week 12	-0.48 [0.21]	-0.48 [0.24]	-0.01 [0.18]	-0.02 [0.21]
Treatment Week 24	-0.61 [0.21]	-	-0.01 [0.18]	-

^aData are baseline (standard deviation) or mean [standard error] change from baseline. Treatment consisted of daclatasvir (60 mg, orally, once daily), asunaprevir (200 mg, orally, twice daily) and BMS-791325 (75 or 150 mg orally, twice daily) for 24 or 12 weeks.

^a Resistance testing was performed at Week 10 since the viral load was < 1000 IU/mL (551 IU/mL) at Week 8.