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APPENDIX 1

Members of the IDEAL investigators: Ira Jacobson, Weill Cornell Medical College, New York, NY, USA; Fred Poordad, Cedars-Sinai Medical Center, Los Angeles, CA, USA; Eric Lawitz, Alamo Medical Research, San Antonio, TX, USA; Jonathan McCone, Mt. Vernon Endoscopy Center, Alexandria, VA, USA; Mitchell L. Shiffman, Virginia Commonwealth University, Richmond,

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Fig. S1 Quartiles of LDL_C by HCV RNA level during treatment at week 2, week 4 and week 12 timepoints.

Fig. S2 LDL_C (</>130 mg/dL) by HCV RNA level during treatment at week 2, week 4 and week 12 time-points.

Table S1: Measures of linkage dis-equilibrium between genome wide sig-nificant SNPs and rs12980275 by race.

Table S2: Multiple regression modelfor baseline LDL in Caucasians.

Table S3: Top GWAS associationpolymorphisms in association withtotal cholesterol in Caucasians.

with differential expression of intrahepatic interferon-stimulated genes in patients with chronic hepatitis *C*. *Hepatology* 2010; 52(6): 1888– 1896.

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VA, USA; Greg W. Galler, Kelsey Research Foundation, Houston, TX, USA; William M. Lee, University of Texas Southwestern Medical Center, Dallas, TX, USA, Robert Reindollar, Piedmont Healthcare, Statesville, NC, USA; John King, Louisiana State University, Shreveport, LA, USA; Reem Ghalib, The Liver Institute at Methodist Dallas Medical Center, Dallas, TX, USA; is a stronger predictor of treatment response than IL28B genotype in patients with hepatitis *C. Gastroenterology* 2011; 140(3); 1021–1031.

Bradley Freilich, Kansas City Gastroenterology and Hepatology, Kansas City, MO, USA; Lisa M. Nyberg, Kaiser Permanente, San Diego, CA, USA; Zachary Goodman, Armed Forces Institute of Pathology, Washington, DC, USA; Navdeep Boparai, Kenneth Koury, Clifford A. Brass, Schering-Plough Corporation, now Merck & Co., Inc., Whitehouse Station, NJ, USA.

Table S4:Top GWAS associationpolymorphisms in association withserum HDL-cholesterol in Caucasians.

Table S5: Top GWAS associationpolymorphisms in association withserum triglycerides in Caucasisans.

Table S6: Multiple regression modelfor LDL at 24 weeks post treatment fornon-SVR patients.

Table S7: Multiple regression model for LDL at 24 weeks post treatment for patients who attained SVR.

Table S8: Comparison between treat-ment response at the timepoint LDLwas measured.

Table S9: Comparison between geno-types within treatment response levels atthe timepoint LDL was measured.

Table S10:Multiple logistic regressionsion model for SVR.

Table S11: Multiple logistic regression model results testing interactionbetween LDL and IL28B genotype(rs12980275) for SVR.

Table S12: Univariate comparison of SVR rates by each *IL28B* genotype (rs12980275).

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