

Glycoproteomic Approach Identifies KRAS as a Positive Regulator of CREG1 in Non-small Cell Lung Cancer Cells

David J. Clark^{1,2}, Yuping Mei¹, Shisheng Sun³, Hui Zhang³, Austin J. Yang², Li Mao^{*1,2}

¹ Department of Oncology and Diagnostic Sciences, University of Maryland School of Dentistry, Baltimore, MD

² Marlene and Stewart Greenebaum Cancer Center, University of Maryland, Baltimore, MD

³ Department of Pathology, John Hopkins University, Baltimore, MD

* Corresponding Author

Department of Oncology and Diagnostic Sciences

University of Maryland School of Dentistry

650 W. Baltimore St., Baltimore, MD 21201, USA

E-mail: Lmao@umaryland.edu

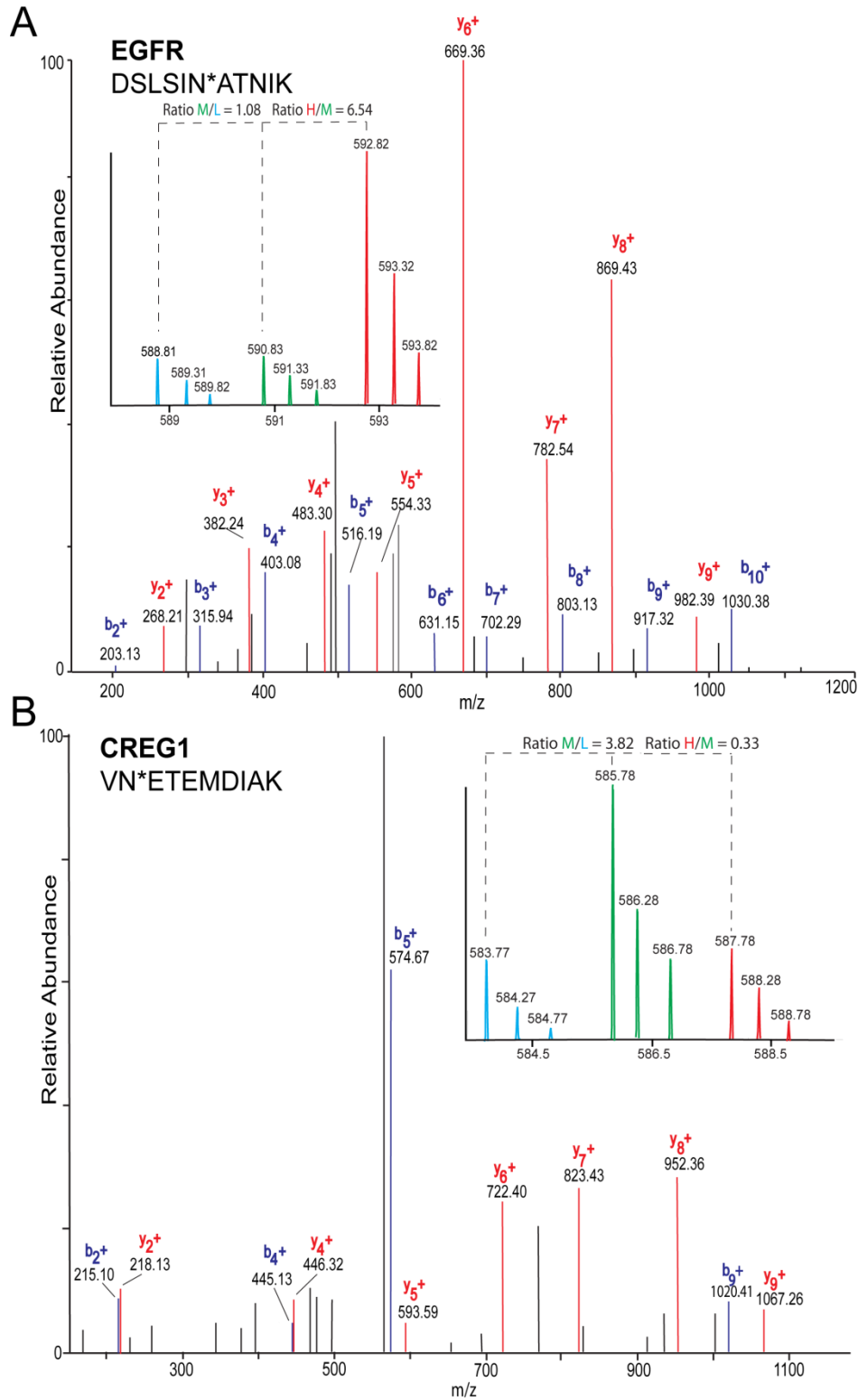


Figure S1. MS/MS mass spectrum of the identified glycopeptide. b- (blue) and y- (red) fragments allow for the mapping of the site of glycosylation as indicated by *. Inset is representative MS1 spectrum of the quantified glycopeptide derived from each cell line: HBE4-L (light blue), A549-M (green), and HCC827-H (red). (A) DSLSINATNIK derived from EGFR, and (B) VN*ETEMDIK derived from CREG1.

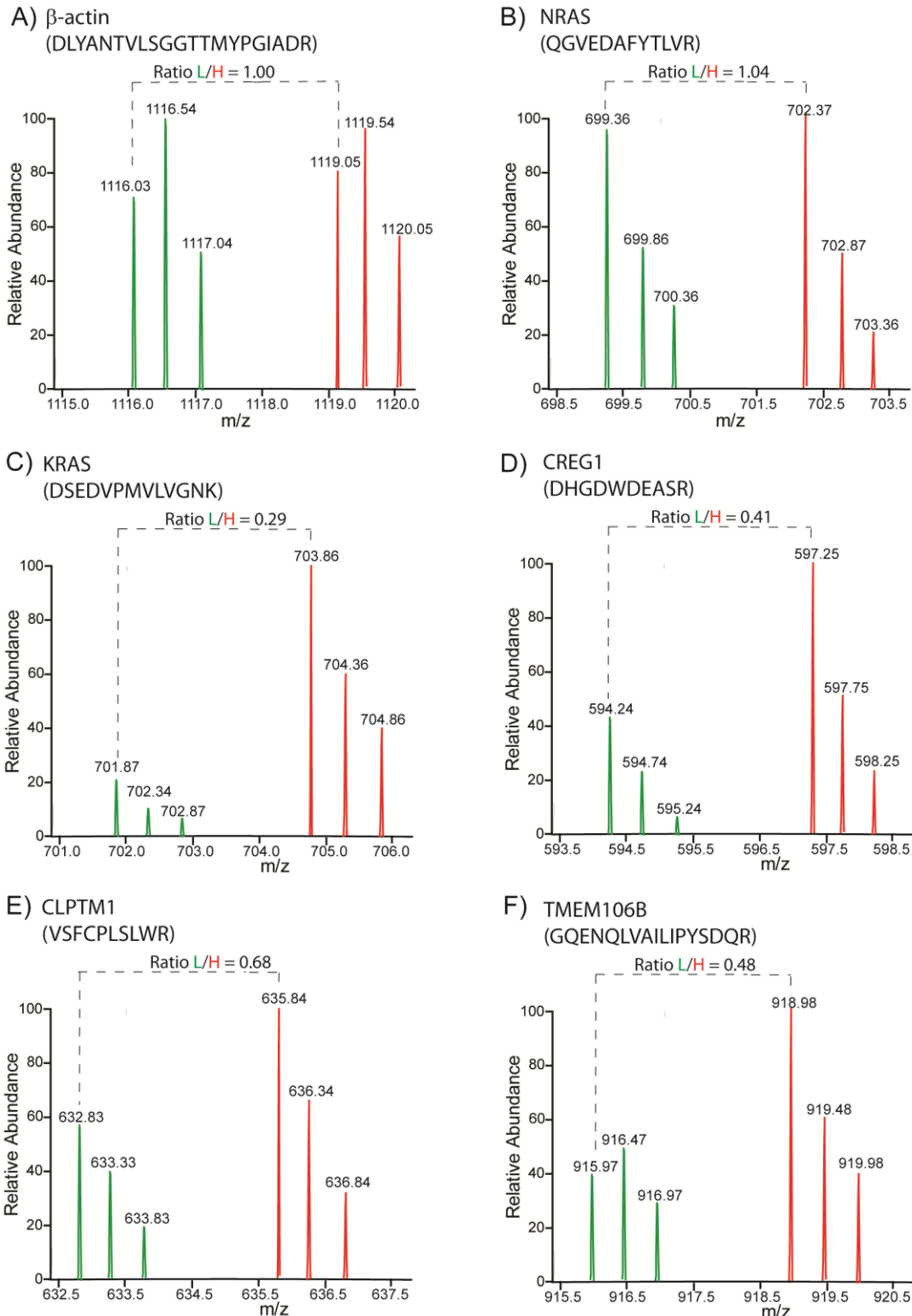


Figure S2. Representative MS1 spectra from Double SILAC analysis of isotopically labeled light (L) ^{siRNA-KRAS}A549 -K0R0 (green) cells compared to labeled heavy (H) A549-K4R6 (red) cells. β -actin and NRAS peptide levels remain in 1:1 ratio (A&B), whereas reduced peptide levels were observed in KRAS, CREG1, CLPTM1, and TMEM106B as a result of siRNA targeting KRAS (C-F).