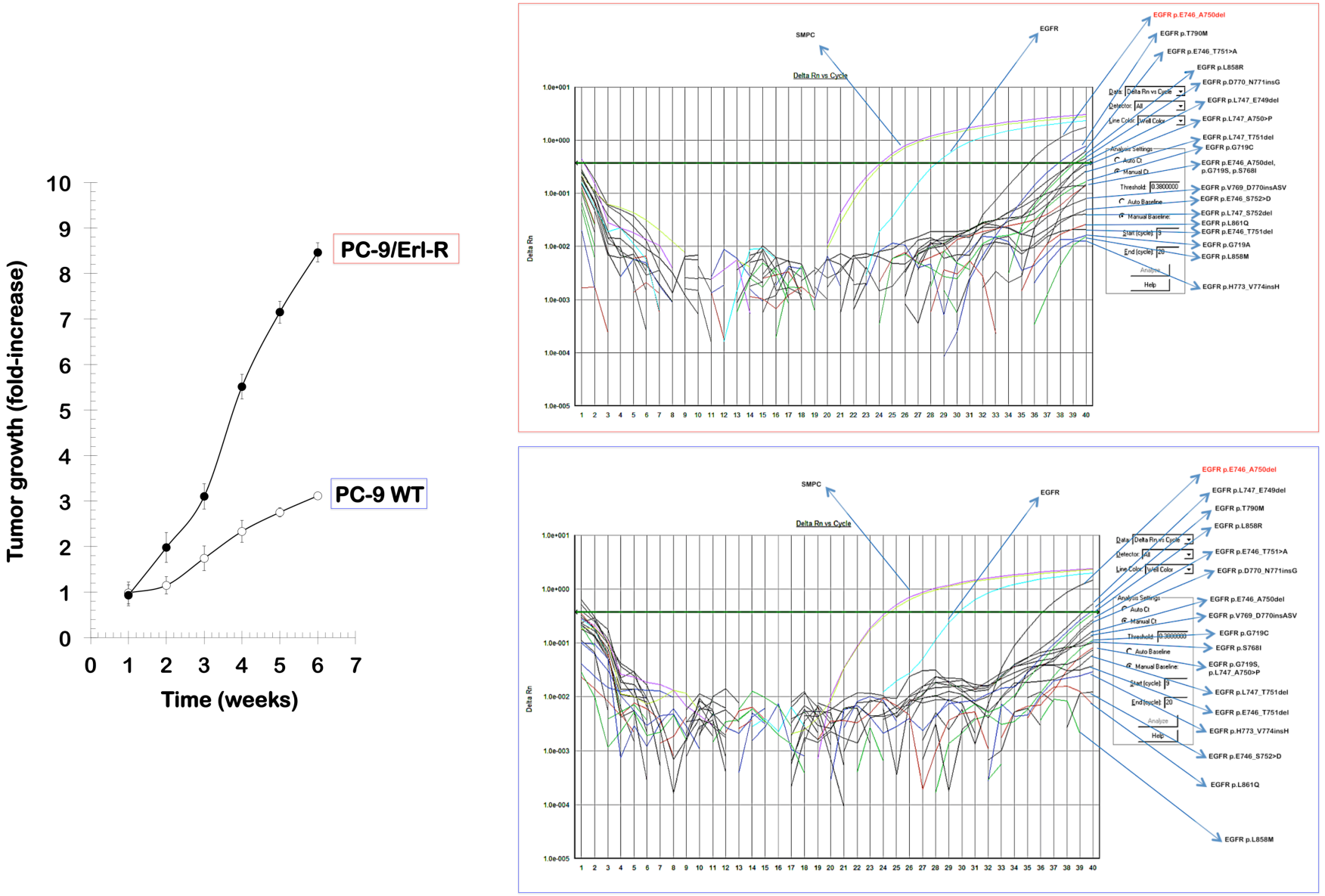
Silibinin suppresses EMT-driven erlotinib resistance by reversing the high *miR-21*/low *miR-200c* signature *in vivo*

Sílvia Cufí[1,2a], Rosa Bonavia[3a], Alejandro Vazquez-Martin[1,2a], Cristina Oliveras-Ferraros[1,2], Bruna Corominas-Faja[1,2], Elisabet Cuyàs[1,2], Begoña Martin-Castillo[2,5], Enrique Barrajón-Catalán[6,7], Joana Visa[4], ç Antonio Segura-Carretero[8,9], Jorge Joven[10], Joaquim Bosch-Barrera[2,4*], Vicente Micol[6,7*], Javier A. Menendez[1,2*]

[a]These authors contributed equally to this work

[*]Co-senior authors

[1]Metabolism & Cancer Group, Translational Research Laboratory, Catalan Institute of Oncology, Girona, Catalonia (Spain); [2]Girona Biomedical Research Institute (IDIBGi), Girona, Catalonia (Spain); [3]Animal Care Facility, IDIBELL, L'Hospitalet de Llobregat, Barcelona, Catalonia (Spain); [4]Medical Oncology, Catalan Institute of Oncology, Girona, Catalonia (Spain); [5]Unit of Clinical Research, Catalan Institute of Oncology, Girona, Catalonia (Spain); [6]Molecular and Cellular Biology Institute (IBMC), Miguel Hernández University, Elche, Alicante (Spain); [7]Monteloeder, Inc., Elche, Alicante (Spain); [9]Unitat de Recerca Biomèdica (URB-CRB), Institut d'Investigació Sanitaria Pere i Virgili (IISPV), Universitat Rovira i Virgili, Reus, Catalonia (Spain); [10]Department of Analytical Chemistry, Faculty of Sciences, University of Granada, Granada, Spain.



Supplementary Figure 1. Tumor growth (*left*) and mutational analysis of the *EGFR* gene (*right*) in erlotinib-responsive PC-9 (WT) parental cells and erlotinib-refractoryPC-9/Erl-R derivatives.