Figure5ApossiblerolefortheH2O2induceddegradationof

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migration of T cells during the phosphorylation-induced degradation of CD40 and HO-1a. A. Western blot of MDA-MB-231 cells treated with H2O2 for 24 hr shows that phosphorylation of CD40, HO-1a, and CD40, and protein expression of HO-1a and CD40 was correlated with decreased migration of T cells. B. Western blot of cells treated with H2O2 for 24 hr shows that H2O2 significantly reduced migration of MDA-MB-231 cells compared to control cells. C. Western blot of MDA-MB-231 cells treated with H2O2 for 24 hr shows that phosphorylation of CD40, HO-1a, and CD40, and protein expression of HO-1a and CD40, together with phosphorylation of HO-1a and CD40, was correlated with decreased migration of T cells. D. Immunostaining of CD40, HO-1a, and CD40 with H2O2 using antibodies against the CD40 and HO-1a ligand and anti-CD40, anti-CD40A, anti-CD40B, and anti-CD40C ligand. Immunostaining was carried out for CD40, HO-1a, and HO-1a and specifically antibodies against the TIMP-1 chromatin (C) and phosphor-PIP1 (D) ligands. H. Liu et al. / Cytokine 60 (2012) 360–368 361 nloaded from niversity O f Southern C alifor, W iley O nline L ibrary on [06/08/2023]. See the T erm s and C /term s-and-conditions on W iley O nline L ibrary for rules of use; O articles are governed by the applicable C reative C ons L icense

New Paper Disruption of P-SIRT1 Promotes Breast Cancer Gopalakrishnan1,2, V.P. K. Ramesh1,2, S.P. K. Gupta1,2, D.B. W. Greig1,2, and S.S. Gopalan1* 1 Department of Medicine, Cancer Biology and Molecular Biology Division, Dr. Bhartiya University, Hyderabad, India 2 Department of Internal Medicine, Dr. S.T. S. Ambady2, S.R. A. Ambady4, S. P. Gopalan1,2, D.B. W. Greig1,2 and S.S. Gopalan1* 3 Department of Surgery-U.S. National Cancer Institute, Cleveland, Ohio, USA 4 Department of Reproductive and Endocrinology, University of Michigan, Detroit, Michigan, USA 5 Department of Pathology, University of Michigan, Detroit, Michigan, USA 6 Department of Molecular and Cellular Medicine, University of Michigan, Detroit, Michigan, USA 7 Department of Internal Medicine, U.S. National Cancer Institute, Detroit, Michigan, USA 8 Department of Pathology, U.S. National Cancer Institute, Detroit, Michigan, USA 9 Department of Pharmacology, University of Michigan, Detroit, Michigan, USA 10 V.P. K. Ramesh1,2, S.P. K. Gupta1,2, S.S. Gopalan1,2, S.P. K. Gupta1,2, S.P. K. Gupta1,2, D.B. W. Greig1,2 and S.S. Gopalan1* 1 Department of Medicine, Cancer Biology and Molecular Biology Division, Dr. Dr. Bhartiya University, Hyderabad, India 2 Department of Internal Medicine, U.S. National Cancer Institute, Cleveland, Ohio, USA 3 Department of Reproductive and Endocrinology, University of Michigan, Detroit, Michigan, USA 4 Department of Pharmacology, University of Michigan, Detroit, Michigan, USA 5 Department of Internal Medicine, U.S. National Cancer Institute, Detroit, Michigan, USA 6 Department of Pathology, U.S. National Cancer Institute, Detroit, Michigan, USA 7 Department of Pharmacology, University of Michigan, Detroit, Michigan, USA 8 Department of Internal Medicine, U.S. National Cancer Institute, Detroit, Michigan, USA 9 Department of Pharmacology, University of Michigan, Detroit, Michigan, USA 10 V.P. K. Ramesh1