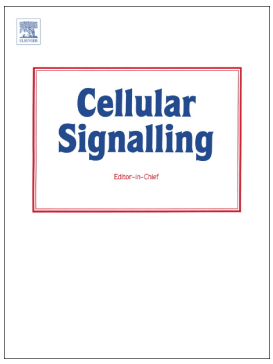

A cellular threshold for active ERK1/2 levels determines Raf/MEK/ERK-mediated growth arrest versus death responses

Seung-Keun Hong, Pui-Kei Wu, Jong-In Park

PII: S0898-6568(17)30260-7
DOI: doi:10.1016/j.cellsig.2017.10.001
Reference: CLS 9008

To appear in: *Cellular Signalling*

Received date: 30 June 2017
Revised date: 2 October 2017
Accepted date: 2 October 2017

Please cite this article as: Seung-Keun Hong, Pui-Kei Wu, Jong-In Park , A cellular threshold for active ERK1/2 levels determines Raf/MEK/ERK-mediated growth arrest versus death responses. The address for the corresponding author was captured as affiliation for all authors. Please check if appropriate. Cls(2017), doi:10.1016/j.cellsig.2017.10.001

A cellular threshold for active ERK1/2 levels determines Raf/MEK/ERK-mediated growth arrest versus death responses

Running Title: ERK1/2-induced cell death

Seung-Keun Hong, Pui-Kei Wu, and Jong-In Park*

Department of Biochemistry, Medical College of Wisconsin, Milwaukee, WI 53226, USA

*Corresponding author at:

Department of Biochemistry, Medical College of Wisconsin, 8701 Watertown Plank Road, Milwaukee, WI 53226, USA

Phone: +1 (414) 955-4098

Fax : +1 (414) 955-6510

E-mail: jipark@mcw.edu (J.-I. Park)

Abbreviations: 4-HT, 4-hydroxytamoxifen; CD, common docking; ERK, extracellular signal-regulated kinase; FRS, F-site recruitment site; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; MEK, mitogen-activated protein kinase kinase; MTT, 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyltetrazolium bromide; PARP, poly (ADP-ribose) polymerase