CORRECTION



Correction to: Activation of PKC δ and p38 δ MAPK during okadaic acid dependent keratinocyte apoptosis

Catherine A. Kraft¹ · Tatiana Efimova⁶ · Richard L. Eckert^{1,2,3,4,5}

Published online: 3 August 2023 © The Author(s), under exclusive licence to Springer-Verlag GmbH Germany, part of Springer Nature 2023

Correction to: Arch Dermatol Res (2007) 299:71–83 https://doi.org/10.1007/s00403-006-0727-4 now included in Fig. 3C; the figure should have appeared as shown below.

In this article, the beta-actin bands in Figure 3C were incorrect. The correct beta-actin bands from original data are

The original article can be found online at https://doi.org/10.1007/ s00403-006-0727-4.

Richard L. Eckert rle2@po.cwru.edu

- ¹ Department of Physiology and Biophysics, Case Western Reserve University School of Medicine, 2109 Adelbert Road, Cleveland, OH 44106-4970, USA
- ² Department of Biochemistry, Case Western Reserve University School of Medicine, 2109 Adelbert Road, Cleveland, OH 44106-4970, USA
- ³ Department of Reproductive Biology, Case Western Reserve University School of Medicine, 2109 Adelbert Road, Cleveland, OH 44106-4970, USA
- ⁴ Department of Oncology, Case Western Reserve University School of Medicine, 2109 Adelbert Road, Cleveland, OH 44106-4970, USA
- ⁵ Department of Dermatology, Case Western Reserve University School of Medicine, 2109 Adelbert Road, Cleveland, OH 44106-4970, USA
- ⁶ Division of Dermatology, Washington University School of Medicine, St. Louis, MO, USA

Fig.3 OA treatment activates keratinocyte apoptotic responses. ► a Keratinocytes, grown on coverslips, were treated with or without 100 nM OA. At 24 h post-infection the cells were stained with MitoSensor reagent and dye uptake was examined by fluorescence microscopy. The arrows indicate the transparent spherical structures. b Keratinocytes were treated with or without 100 nM OA for 24 h. The cells were then fractionated and cytochrome c level, in cytosol and mitochondrial fractions, was measured by immunoblot. Successful separation of the mitochondrial and cytosolic fractions was confirmed by detection of COX4 (a mitochondrial marker) and β -actin (a cytosol marker). c OA treatment results in procaspase-3 and PARP cleavage. Keratinocytes were treated for 48 h in the presence of absence of 100 nM OA and total cell extracts were fractionated on a 10% gel for immunoblot with anti-caspase 3, anti-PARP and anti- β -actin. The *asterisks* indicate the position of the procaspase 3 and PARP cleavage products



Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.