ERRATUM



Erratum to: Co-regulation of mitochondrial respiration by proline dehydrogenase/oxidase and succinate

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In the original publication, the legend of Fig. 2d was wrongly published. The correct Fig. 2d is given below.

It should read:

(Yellow symbol) POX + NAC 5 mM(Blue symbol) POX + NAC 10 mM

The online version of the original article can be found under doi:10.1007/s00726-015-2134-7.

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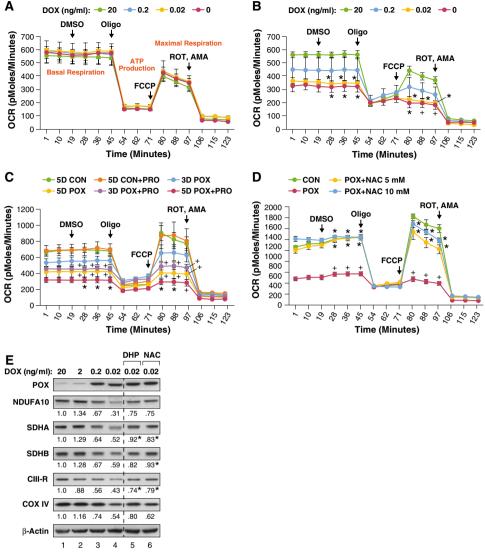


Fig. 2 Extended PRODH/POX ROS production decreases total respiratory fitness. Data for each time point represents mean \pm SEM (n = 3) for all panels. a DOX concentration does not affect respiration in DLD-VEC control cells. Cells grown for 48 h in the indicated concentration of DOX. Oxygen consumption rate (OCR) was measured and a cellular respiratory profile was established by the addition of 2.5 µM DMSO, OLIGO, FCCP, and ROT/AMA at the indicated timepoints. b Increasing PRODH/POX expression correlates with lower cellular respiration. DLD-POX cells were grown in the indicated amount of DOX and oxygen consumption rate was analyzed as in (a). Values with DOX = 0.02 and 0.0 were compared to DOX = 20; * $p \le 0.01$; ^+p < 0.02. c Prolonged PRODH/POX expression and addition of proline correlates with greater decreases in respiration. DLD-POX cells were grown in 0.2 ng/ml DOX to allow PRODH/POX expression for 3 days (3D POX) or 5 days (5D POX) alone or in media supplemented with 5 mM proline (3D POX + PRO and 5D POX + PRO). Respiration by these cells was compared to DLD-POX cells that had been grown for 5 days in 20 ng/ml DOX to suppress PRODH/POX expression, alone or in combination with 5 mM proline (5D CON and 5D CON + PRO, respectively). Oxygen consumption rate was measured and a respiratory profile established as described in (a). Values for 3D POX + PRO, 5D POX, and 5D POX + PRO were compared to 5D CON. $p \le 0.05$; *p < 0.01. **d** Inhibition of PRODH/POX-mediated ROS decreases effects on respiration. DLD-POX cells were grown for

48 h in either 20 ng/ml DOX (CON), or 0.2 ng/ml DOX alone (POX) or in combination with 5 or 10 mM of N-acetyl-L-cysteine (NAC). Oxygen consumption rate was measured and a respiratory profile established as described in (a). Compared to control, *p < 0.001; compared to POX, p < 0.01. e PRODH/POX expression down-regulated ETC component proteins. DLD-POX cells were grown in the indicated ng/ ml concentration of doxycycline (DOX) for 48 h, alone or in combination with 10 mM DHP or NAC. Whole cell lysates were harvested, and protein expression of subunits of Complex I (NDUFA10), Complex II (SDHA and SDHB), Complex III (CIII-R, Reiske Fe-S subunit), and Complex IV (COX IV) were analyzed by Western blotting. β-Actin was used as a protein loading control. The band intensities shown below each panel were quantified using Image Studio, normalized to β-actin control, and expressed as the level relative to untreated control (lane 1 20 ng/ml DOX). Values for DHP and NAC were compared to the mean \pm STD of the ratios for 0.2 ng/mL DOX (lane 3 e and lane 2, Fig. 5a) combined with those for 0.02 ng/ml DOX (lane 4 e) Although the DOX concentration for DHP and NAC treatment was 0.02 ng/mL DOX, the values at 0.02 ng/ml DOX were consistently lower than those at 0.2 ng/ml DOX. Thus, the values used represent a higher distribution for the PRODH/POX-mediated effect on ETC proteins. DHP and NAC increased the values of the ETC proteins, i.e., decreased the effect of PRODH/POX. *Value greater than 2 standard deviations of aforementioned distribution, denoting 95 % confidence limits

