Supporting Information

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Fig. S1. Effect of neonatal intermittent hypoxia (IH) on carotid body and adrenal chromaffin cell responses in adult rats. (A) Isolated superfused carotid body (CB) sensory responses to CO₂ (PCO₂ = 65 mmHg) in postnatal day (P) 40 rats exposed to normoxia (P40; Left), neonatal IH (P40_{IH}; Center), or treated with decitabine during neonatal IH (P40_{IH} + decitabine; Right). Black bar represents the duration of the CO₂ challenge. Integrated CB sensory activity presented as impulses per second (imp/s). Superimposed action potential from the "single" fiber from which the data are derived is presented in the inset. (B) Mean CB responses to CO2 from P40, P40_{IH}, and P40_{IH} + decitabine-treated rats shown as the difference in response between baseline and hypoxia (Δ imp/s). Data presented are mean ± SEM of n = 12 (P40), 14 (P40_{IH}), and 18 (P40_{IH} + decitabine) fibers from 10 to 11 rats in each group. (C) Analysis of CB morphology. Number of glomus cells (glomic volume) and total CB volume were analyzed in control (P40), neonatal IH (P40_{IH}) and neonatal IH + decitabine-treated (P40_{IH} + decitabine) rats by morphometric analysis. Data are expressed as mean ± SEM from n = 4 rats in each group. (D) CBs from rats exposed to neonatal normoxia (P40) or neonatal normoxia + decitabine (P40 + decitabine) were isolated, exposed to graded hypoxia, and the difference in response between baseline and hypoxia (Δ imp/s) was determined. Data are presented are mean \pm SEM of n = 14 (P40) and 18 (P40 + decitabine) fibers from 10 rats each. (E) K⁺-evoked catecholamine secretion from chromaffin cells that were isolated from P40, P40_{IH}, P40_{IH} + decitabine rats. (F) Total catecholamine (CA) secreted during K⁺ challenge (number of secretory events × CA molecules secreted per event). Data shown are mean \pm SEM from n = 12 cells from P40_{IH} and 10 cells from P40_{IH} + decitabine-treated rats from three different litters in each group; n.s., not significant (P > 0.05). (G). Absence of effects of decitabine on cytosolic and mito chondrial aconitase activities in adrenal medulla from control P40 rats exposed to normoxia in the neonatal period. Data shown are mean \pm SEM from n = 4independent experiments each. (H). Analysis of Sod2 mRNA in CB and adrenal medulla (AM) in control P40 rats treated with (+) or without (-) decitabine in the neonatal period. Data are expressed relative to 18S rRNA and are presented as mean ± SEM from n = 4 independent experiments. n.s., P > 0.05 for P40 vs. $P40_{IH}$ + decitabine or P40 + decitabine.

Table S1.	Nucleotide sequence of primers used for the analysis of
cytosine n	nethylation in the Sod2 gene

Primer	Sequence of forward (For) and reverse (Rev) primers
1	For: 5'-AATATGTTTTGGGGATGAAGTTT-3'
	Rev: 5'-TCCAAAATTTTCAAAAAACTCA-3'
2	For: 5'-GGGGTTTTTTAGAATTAGGAAT-3'
	Rev: 5'-ACAACTAAACRCTAACCCTAC-3'
3	For: 5'-TYGTTTTGTTATAAGTTGGGT-3'
	Rev: 5'-CTTCTACCCRCRCTAAAAA-3'
4	For: 5'-AAGTGGTAGGGTTYGGGG-3'
	Rev: 5'-ACACRAAAAACCTCCAATACA-3'
5	For: 5'-TATTAAYGYGTAGATTATGTAGTTG-3'
	Rev: 5'-AACTCACAATAATAAAAATACATCAA-3'