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Data in Brief

Gene expression in rat striatum following carbon monoxide poisoning

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ABSTRACT

Carbon monoxide (CO) poisoning causes brain damage, which is attenuated by treatment with hydrogen [1,2], a scavenger selective to hydroxyl radical (•OH) [3]. This suggests a role of •OH in brain damage due to CO poisoning. Studies have shown strong enhancement of •OH production in rat striatum by severe CO poisoning with a blood carboxyhemoglobin (COHb) level >70% due to 3000 ppm CO, but not less severe CO poisoning with a blood COHb level at approximately 50% due to 1000 ppm CO [4]. Interestingly, 5% O_2 causes hypoxia comparable with that by 3000 ppm CO and produces much less •OH than 3000 ppm CO does [4]. In addition, cAMP production in parallel with •OH production [5] might contribute to •OH production [6]. It is likely that mechanisms other than hypoxia contribute to brain damage due to CO poisoning [7]. To search for the mechanisms, we examined the effects of 1000 ppm CO, 3000 ppm CO and 5% O_2 on gene expression in rat striatum. All array data have been deposited in the Gene Expression Omnibus (GEO) database under accession number GSE94780.

Specifications	
Organism/cell line/tissue	*Rattus norvegicus*, Sprague-Dawley strain, striatum
Sex	Male
Sequencer or array type	Agilent-014879 Whole Rat Genome Microarray 4x44K G4131F
Data format	Raw and analyzed
Experimental factors	Carbon monoxide (CO) poisoning and hypoxic hypoxia
Experimental features	The striatum was obtained 0 h, 2 h or 6 h after the 40 min-exposure to 1000 ppm CO, 3000 ppm CO, or 5% O_2.
Consent	All experiments were performed in accordance with the Fundamental Guidelines for the Proper Conduct of Animal Experiments and Related Activities in Academic Research Institutions under the jurisdiction of the Ministry of Education, Culture, Sports, Science, and Technology, Japan. The experimental protocol was approved by the Institutional Animal Care and Use Committee (IACUC) of Tokyo Medical University.
Sample source location	Male Sprague-Dawley rats were obtained from Charles River Laboratories (Kanagawa, Japan).

1. Direct link to deposited data

http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE94780

2. Experimental design, materials and methods

2.1. Exposure of rats to CO or 5% O_2

Male Sprague-Dawley rats (7 week-old, 235 to 265 g) were purchased from Charles River Laboratories (Kanagawa, Japan). The animals were acclimated with free access to food and water in a facility with controlled temperature (22 °C–24 °C) on a 12-h/12-h light/dark cycle (lights on between 08:00 and 20:00), for at least 1 week before the experiments.

The rat in the plastic chamber (26.5 cm in diameter, 28.5 cm in height) was exposed for 40 min to room air alone ($n = 1$), 1000 ppm CO ($n = 3$), 3000 ppm CO ($n = 3$), or 5% O_2 ($n = 3$). Room air containing pure CO or N_2 gas was introduced into the chamber at a flow rate of 8 L/min. The concentration of CO or O_2 in the chamber was adjusted using a Koflok gas flow regulator (Osaka, Japan) with monitoring each gas level using a Gastec CM-525HB or GOM-3A (Kanagawa, Japan), respectively. CO at 1000 ppm and 3000 ppm causes CO poisoning with approximately 50% COHb and over 70% COHb in the blood, respectively, and hypoxic conditions of the latter CO poisoning are comparable with those induced by 5% O_2 [4].

2.2. Preparation of the striatum and RNA extraction

The rat was anesthetized with sodium pentobarbital (50 mg/kg, i.p.) 0 h, 2 h or 6 h after exposure to room air alone, CO, or 5% O_2. The brain

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was removed after perfusion with chilled saline via a cannula inserted into the carotid artery through the left ventricle. Both striata were dissected on ice, weighed, immersed in the RNAlater solution (Ambion; Foster City, CA, USA) overnight at 4 °C, and stored at −80 °C until analyzed.

The total RNA of the striatum was obtained using an RNeasy Lipid Tissue Mini kit (Qiagen; Venlo, The Netherlands). The RNA concentration was measured by a NanoDrop spectrophotometer (Wilmington, DE, USA), and its purity was confirmed by an Agilent 2100 bioanalyzer (Agilent technologies; Santa Clara, CA, USA).

2.3. Microarray analysis

Microarray analysis was performed according to the One-Color Microarray-Based Gene Expression Microarrays Analysis Protocol (Agilent-014879 Whole Rat Genome Microarray 4x44K G4131F). The RNA (100 ng) was converted into cDNA using a Low Input Quick Amp Labeling Kit (Agilent), followed by in vitro transcription and incorporation of cyanine 3-CTP into cRNA for fluorescence labeling. The labeled cRNA was purified using an RNeasy Lipid Tissue Mini kit (Qiagen), fragmented, and then applied to the Whole Rat Genome Oligo Microarray (Agilent) for hybridization at 65 °C for 17 h. The microarray was scanned with an Agilent Microarray Scanner, and the fluorescence intensity for the 41,090 gene probes was calculated by Feature Extraction ver.10.7.3.1 (Agilent). Further analysis was performed using GeneSpring GX11 (Agilent). The GeneSpring flag setting was as follows: "present": feature signals were saturated; "absent": feature signals were below the background signal or not positive and significant; "marginal"; feature signals were not uniform or were outliers. The values of fluorescence intensity were normalized and corrected on the basis of those of the control animal (0 h after room air alone). Altered transcripts were identified using a comparative method on the log2 normalized intensity values between the probes of the control animal and each treatment. The gene, which corresponded to the probe having a change in the intensity exceeding a log ratio of three (namely, fold change >3 or <−3), was considered as that with a significantly differential expression pattern.

3. Results

Exposure for 40 min to 3000 ppm CO, but not 1000 ppm CO or 5% O_2, enhanced and suppressed expression of 36 genes and 16 genes in rat striatum, respectively, whose molecular functions are suggested, as follows: (A) enhanced (1) at 0 h, Ac2-248, alkaline phosphatase 1, dual oxidase 2, oncostatin M, tissue factor pathway inhibitor 2 and transformation related protein 63, (2) at 2 h, Ba1-651, a disintegrin and metalloproteinase domain 28, antigen identified by monoclonal antibody MRC OX-2 receptor, chondroadherin, diamine oxidase-like protein 2, GATA binding protein 6, glutamine/glutamic acid-rich protein A, lipocalin 6, nephrosis 2 homolog podocin (human), olfactory marker protein, outer dense fiber of sperm tails 1, S100 calcium binding protein A8 (calgranulin A), solute carrier family 17 (sodium-dependent inorganic phosphate cotransporter) member 7, solute carrier family 2 (facilitated glucose transporter) member 2, troponin I skeletal slow 1, type II keratin Kb35 and UDP glucuronosyltransferase 2 family polypeptide A1, (3) at 2 and 6 h, cytochrome P450, subfamily 11B polypeptide 1, (4) at 6 h, aminolevulinic acid synthase 2, beta-glo, CD52 antigen, desmocollin 2, free fatty acid receptor 1, gastric inhibitory polypeptide, globin alpha, hemoglobin beta chain complex, oncomodulin, phospholipase B, protein phosphatase with EF hand calcium-binding domain 1 and solute carrier family 5 (sodium/glucose cotransporter) member 1; (B) suppressed (1) at 0 h, 2 h and 6 h, calmodulin-like 3, (2) at 2 h, cyclic nucleotide gated channel alpha 1 and Rab38 member of RAS oncogene family, (3) at 2 h and 6 h, arginine vasopressin, calbindin 2, ectonucleotide pyrophosphatase/phosphodiesterase 2 and oxytocin, (4) at 6 h, 5-hydroxytryptamine (serotonin) receptor 7, kinesin light chain 3 and purinergic receptor P2X ligand-gated ion channel 2.

Hypoxia is reported to affect housekeeping gene expression in cultured cells [8,9]. However, hypoxic conditions induced by 1000 ppm CO, 3000 ppm and 5% O_2 in vivo had little or no effects on expression of 12, so-called, "housekeeping genes", in the rat, such as actin beta, ATP synthase H+ transporting mitochondrial F0 complex subunit b isoform 1, beta-2 microglobulin, glucuronidase beta, glyceraldehyde-3-phosphate dehydrogenase, hypoxanthine guanine phosphoribosyl transferase, peptidylprolyl isomerase A, phosphoglycerate kinase 1, ribosomal protein S18, ribosomal protein large P2, TATA box binding protein and tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation protein zeta polypeptide.

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References

[1] Q. Sun, J. Cai, J. Zhou, H. Tao, J.H. Zhang, W. Zhang, X.J. Sun, Hydrogen-rich saline reduces delayed neurologic sequelae in experimental carbon monoxide toxicity. Crit. Care Med. 39 (2011) 765–769, http://dx.doi.org/10.1097/CCM.0b013e318206bf44.

[2] I. Ohsawa, M. Ishikawa, K. Takahashi, M. Watanabe, K. Nishimaki, K. Yamagata, K. Katsura, Y. Katayama, S. Asoh, S. Ohta, Hydrogen acts as a therapeutic antioxidant by selectively reducing cytotoxic oxygen radicals. Nat. Med. 13 (2007) 688–694, http://dx.doi.org/10.1038/nm1577.

[4] S. Hara, H. Mizukami, K. Kurosaki, F. Kuriiwa, T. Mukai, Existence of a threshold for hydroxyl radical generation independent of hypoxia in rat striatum during carbon monoxide poisoning. Arch. Toxicol. 85 (2011) 1091–1099, http://dx.doi.org/10.1007/s00204-010-0637-2.

[5] S. Hara, H. Mizukami, F. Kuriiwa, T. Mukai, cAMP production mediated through $P2Y_{11}$-like receptors in rat striatum due to severe, but not moderate, carbon monoxide poisoning. Toxicology 288 (2011) 49–55, http://dx.doi.org/10.1016/j.tox.2011.07.001.

[6] S. Hara, M. Kobayashi, F. Kuriiwa, T. Mukai, H. Mizukami, Dual contradictory roles of cAMP signaling pathways in hydroxyl radical production in the rat striatum. Free Radic. Biol. Med. 52 (2012) 1086–1092, http://dx.doi.org/10.1016/j.freeradbiomed.2012.01.002.

[7] J.D. Roderique, C.S. Josef, M.J. Feldman, B.D. Spiess, A modern literature review of carbon monoxide poisoning theories, therapies, and potential targets for therapy advancement. Toxicology 334 (2015) 45–58, http://dx.doi.org/10.1016/j.tox.2015.05.004.

[8] R. Yamaji, K. Fujita, S. Takahashi, H. Yoneda, K. Nagao, W. Masuda, M. Naito, T. Tsuruo, K. Miyatake, H. Inui, Y. Nakano, Hypoxia up-regulates glyceraldehyde-3-phosphate dehydrogenase in mouse brain capillary endothelial cells: involvement of Na+/Ca2+ exchanger. Biochim. Biophys. Acta 17 (2003) 269–276, http://dx.doi.org/10.1016/S0167-4889(02)00397-X.

[9] H. Zhong, J.W. Simons, Direct comparison of GAPDH, beta-actin, cyclophilin, and 28S rRNA as internal standards for quantifying RNA levels under hypoxia. Biochem. Biophys. Res. Commun. 259 (1999) 523–526, http://dx.doi.org/10.1006/bbrc.1999.0815.