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Methamphetamine downregulates peroxiredoxins in rat pheochromocytoma cells

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Abstract

Methamphetamine (METH) is an abusive psychostimulant that induces neuronal cell death/degeneration in experimental animals and humans. METH-induced apoptosis in rat pheochromocytoma cells was utilized to study the neurotoxic mechanism. During METH intoxication, we found that peroxiredoxins and thioredoxins/thioredoxin reductases (peroxiredoxin reducing systems) which are known to prevent oxidative stress and apoptosis were differentially downregulated and upregulated, respectively. We also found not only the free radicals but also the oxidative forms of peroxiredoxin and thioredoxin were increased, indicating the dysfunction of these enzymes. Thus, METH-induced differential regulation and oxidation of peroxiredoxins and thioredoxin may be an important mechanism for apoptosis.

Keywords: Methamphetamine; Peroxiredoxin; Thioredoxin; Thioredoxin reductase

Methamphetamine (METH) and its analogs are known to cause long-term depletion of dopamine (DA) [1], to decrease the number of DA transporters [2], to decrease the activity of tyrosine hydroxylase [3], and to cause the degeneration of DA terminals in the caudate nucleus of rats [4]. They also induce apoptotic death in different neuronal cells *in vitro* [5,6]. The neurotoxic mechanisms have been attributed to oxidative stress, excitotoxicity, and energy failure [7,8]. Recently, gene and protein expression profiles have intensively been used to further elucidate the possible protein(s) underlying the neurotoxic mechanisms of METH [9,10]. Proteomic data from our laboratory (unpublished data) and from Iwazaki et al. [10] simultaneously found that METH downregulates the expression of peroxiredoxin (Prx)-2 in pheochromocytoma (PC12) cells and the brain of the rat.

Prxs, synonymous with thioredoxin (Trx) peroxidases, are a family of multi-functional and Trx-dependent peroxidases that are divided into three major subclasses: typical 2-cysteine (Cys) Prx-1–4, atypical 2-Cys Prx-5, and 1-Cys Prx-6 [11]. Prx-1, -2, and -6 are mainly localized in the cytosol, while Prx-3 is exclusively localized in the mitochondria. Prx-4 is expressed in the endoplasmic reticulum and can be exported into the extracellular space. Prx-5 is found in mitochondria as a long form and in peroxisomes as a short variant. They have been suggested to play roles in several cellular functions including protection of proteins and lipids against oxidative injury, cell proliferation, differentiation, and apoptosis regulation [12]. The aberrant expressions of Prxs in neurodegenerative disorders have also been identified [13], suggesting an important role of Prxs in mediating neurotoxicity.

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Alternatively, in contrast to glutathione, which is thought to be largely responsible for the low redox potential and free thiol levels inside cells [14], the Trx system, which is comprised of NADPH, Trxs, and thioredoxin reductases (TRs), may play a critical role in the redox regulation of protein thiols, including Prxs [15], signal transduction, and gene regulation [16]. TR reduces oxidized Trx by consuming NAPDH. Since Trx and TR in conjunction with Prx constitute cyclic enzymes in detoxifying various peroxides [17], oxidative stress contributes to the neurotoxicity mechanisms of METH [7], and METH downregulates Prx-2 expression [10], the expressions of Prxs, Trxs, and TRs during the neurotoxicity of METH were studied and their possible functions are discussed.

Materials and methods

Reagents and cell culture. All reagents were purchased from Sigma-Chemical (St. Louis, MO, USA) except where otherwise specified. METH was purchased from the National Bureau of Controlled Drugs, Department of Health (Taipei, Taiwan). The antibodies of Prxs, Trxs, and TRs were purchased from LabFrontier (Seoul, Korea), Abcam (Cambridge, MA, USA), and Upstate (Charlottesville, VA, USA), respectively. The anti-Prx-SO_3 antibody which recognizes both sulfinic ($-SO_2$) and sulfonic ($-SO_3$) forms of overoxidized Prx-1–4 was purchased from LabFrontier. Anti-caspase-3 and PARP antibodies were purchased from CellSignaling (Beverly, MA, USA). Dulbecco's modified Eagle's medium (DMEM), fetal bovine serum, and horse serum were purchased from HyClone (Logan, UT). Rat PC12 cells purchased from American Type Culture Collection (ATCC, Manassas, VA, USA) were maintained in DMEM supplemented with 10% horse serum and 5% fetal bovine serum and incubated in a CO_2 incubator (5%) at 37 °C.

MTT assay. The plating number is $3–5 \times 10^4$ cells/cm^2 throughout the following experiments. After treatment, 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazolium bromide (MTT) was added (0.5 mg/ml) and incubated at 37 °C for another 3 h. After discarding the medium, 100 μl DMSO was applied to the well to dissolve the formazan crystals and the absorbances at 570/630 nm were measured on a micro-ELISA reader.

Apoptosis detection. An annexin V (FITC-conjugated) apoptosis kit (BioVision, Mountain View, CA, USA) was used to analyze the apoptotic cells. The experimental protocol followed the manufacturer's instructions. In brief, after METH treatment for certain periods, cells growing on 12-well plates were loaded with 0.5 ml binding buffer and 5 μl annexin V-FITC. After incubation for 5 min in the dark, cells were washed once with 1 ml culture medium for making the fluorescent micrographs (Axiovert-200 M, Carl Zeiss, Göttingen, Germany) or resuspended for the flow-cytometric analysis (Beckton–Dickinson, Franklin Lakes, NJ, USA). The mean values of the fluorescent intensities of FITC were collected using an FL-1 channel ($488/530^{Ex/Em}$ nm). Five thousand live cells were analyzed per sample.

Western blot analysis. Cells were collected by centrifugation (1000*g*, 5 min, 4 °C) and lysed in a PRO-PREP™ protein extraction solution (iNtRON Biotechnology, Kyungki-Do, Korea). After resuspension and incubation on ice for 20 min, cell debris was removed by centrifugation at 12,000*g* for 5 min (4 °C) and the supernatant was utilized for Western blot analysis. Equal amounts of sample were separated using a NuPAGE® 4–12% Bis–Tris Gel (Invitrogen, Carlsbad, CA, USA). The resolved proteins (30 μg/lane) were then electroblotted onto Immobilon PVDF membranes (Millipore, Bedford, MA, USA). Membranes were blocked with 5% skim milk and then incubated with the first antibody (1:2000) overnight (at 4 °C) and then the second antibody (1:5000) for 1 h (at room temperature). After washing, the blots were processed for visualization using an enhanced chemiluminescence system (Pierce, Rockford, IL, USA). Blots were then exposed to Kodak XAR-5 film to obtain the fluorographic images. The images were scanned and quantified using an image analyzer (GeneTools, Synoptics, England).

Detection of intracellular superoxide and hydrogen peroxide. Once inside the cell, 2′,7′-dichlorofluorescein diacetate (DCFDA) is cleaved by endogenous esterases and can no longer pass out of the cell. The de-esterified product becomes the fluorescent compound, DCF, upon oxidation by ROS, mainly H_2O_2. Consequently, the intensities of DCF were used to represent intracellular H_2O_2 formation. On the other hand, dihydroethidine (DHE) can be directly oxidized to fluorescent ethidium bromide (EtBr) by the superoxide ($\cdot O_2^-$) anion. Thus, 20 μM DCFH-DA or 10 μM DHE was added to cells and incubated at 37 °C for 30 or 20 min, respectively. Cells were resuspended for the flow-cytometric analysis. The mean values of the fluorescent intensities of DCF and EtBr were, respectively, collected using the FL-1 and FL-2 channels. Ten thousand live cells were analyzed per sample.

Assaying redox state of Trx. The redox assaying of Trx was followed as described by Bersani et al. [18] with little modification. In brief, cells were carboxymethylated by 200 μl of 8 M urea, 100 mM Tris (pH 8.2), 1 mM EDTA, and 30 mM iodoacetate and incubated at 37 °C for 15 min. Samples were precipitated by nine volumes of acetone/1N HCl (98/2, v/v). Pellets were incubated with 196 μl of 8 M urea, 100 mM Tris (pH 8.2), 1 mM EDTA, and 3.5 mM dithiothreitol for 30 min at 37 °C for 30 min. Then, 4 μl of 500 mM iodoacetamide was added and incubated for other 15 min. Equal amounts of sample were separated using urea–polyacrylamide gel electrophoresis.

Statistical analysis. Results were analyzed by one- or two-way analysis of variance according to the suitability. Differences between means were assessed by the Student–Newman–Keuls method and were considered significant at $p < 0.05$.

Results and discussion

METH-induced apoptosis in neuronal-like PC12 cells

After METH treatment for different time periods, PC12 cells underwent significant cell death as revealed by the MTT assay (Fig. 1a). The sub-maximum dose of 2 mM METH treatment for 2d was adopted to study its neurotoxic effects for the following experiments. The fluorescent intensity of annexin V-FITC was significantly increased in METH-treated cells as visualized by microscopy (Fig. 1b, left panels) and analyzed by flow-cytometry (Fig. 1b, upper right panels), demonstrating the presence of apoptosis in METH-treated PC12 cells, which is consistent with the findings of Cadet et al., who also utilized 1–3 mM METH to induce apoptosis in immortalized neural cells [5]. Such concentration has also been found in human plasma [19]. Furthermore, the apoptotic event was further strengthened by the finding of increased cleavages of poly(ADP-ribose)polymerases (PARP) and caspase 3 (Fig. 1b, lower right panels) consisting with the finding of Jayanthi et al. [20].

METH differentially regulated Prx, Trx, and TR expressions

As described previously, Prxs are a family of multi-functional antioxidant and thioredoxin-dependent peroxidases that provide cellular protection against oxidative stress and apoptosis, modulation of intracellular signaling cascades, and regulation of cell proliferation [21]. Taking protection and signaling modulation for example,

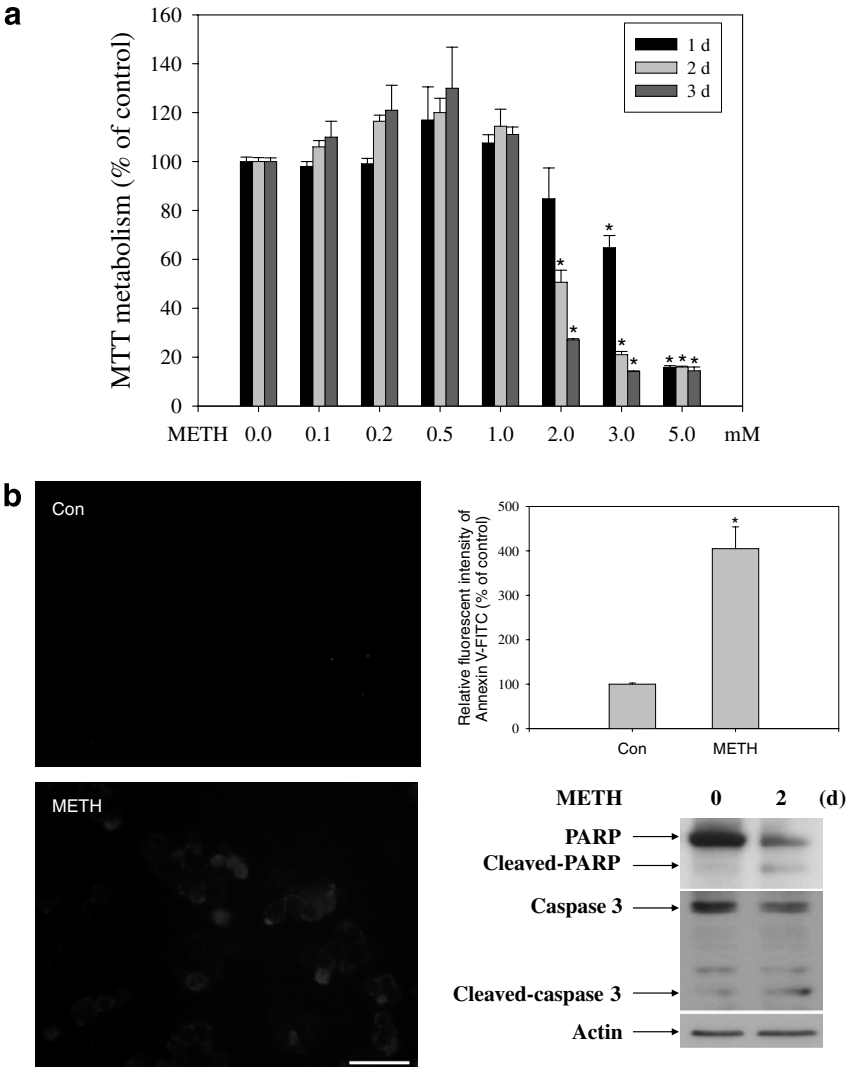

Fig. 1. Effect of METH-induced apoptosis in neuronal-like PC12 cells. (a) PC12 cells were treated with METH (0–5 mM) for 1, 2, and 3d, and MTT was added for the survival assay. Cell viability is expressed as a percentage of the results of the MTT assay measured in the control (Con) group without METH. (b) Cells with or without METH (2 mM) treatment for 2d were stained with annexin V-FITC or harvested for Western blot analysis (right lower panel). Stained cells were either taken photographs (left panels) or analyzed by flow cytometry (right upper panel). The fluorescent intensities of the control group are expressed as a percentage of the mean values of FL-1 (left lower panel). Data points represent means ± SEM of at least three independent experiments ($n = 3$–6). *$p < 0.05$ compared to the control group.

increased expressions of Prx-1, -2, -3, and -5, respectively, protect lymphoma, thyroid, thymoma, and tendon cells against H_2O_2-dependent apoptosis [22–24]. Prx-4 reduces EGF- and p53-induced reactive oxygen species [25]. Disruption of the Prx-6 gene renders the heart vulnerable to ischemic reperfusion injury [26]. Currently, we found that METH downregulated the expressions of Prx-1, -2, -4, -5, and -6 (Fig. 2a). Normally, the expressions of Prxs can be upregulated by free radicals through activation of transcription factors, such as Nrf-2 or AP-1 [21]. Our unpublished data also found that exogenous H_2O_2 upregulated the expressions of Prxs (Supplement 1) in PC12 cells. However, others have shown that silica not only induces oxidative stress but also results in degradation of Prx-1 and -2 [27]. Calpain activation may partially contribute to this proteolytic effect. The ubiquitin–proteasome system tends not to involve this effect, since METH has been shown to induce ubiquitinated neuronal inclusions in the nigrostriatal system and PC12 cells [28] and bears the same toxicity effect as shown by proteasome inhibition [29]. Thus, whether oxidative stress induces up- or downregulation of Prxs and how stress induces Prx proteolytic mechanisms still remain to be studied. Besides, overexpression of each Prx by transient transfection or stable expression might be helpful to reveal their significances during METH intoxication.

Alternatively, Trxs and TRs can be classified into cytosolic (Trx-1 and TR-1) and mitochondrial (Trx-2 and TR-2) forms [16]. Presently, METH upregulated the expressions of Trx-1 (Fig. 2b, left panel) and TR-1 (Fig. 2b, right panel) but not the expressions of Trx-2 or TR-2. Although the significance of these findings is

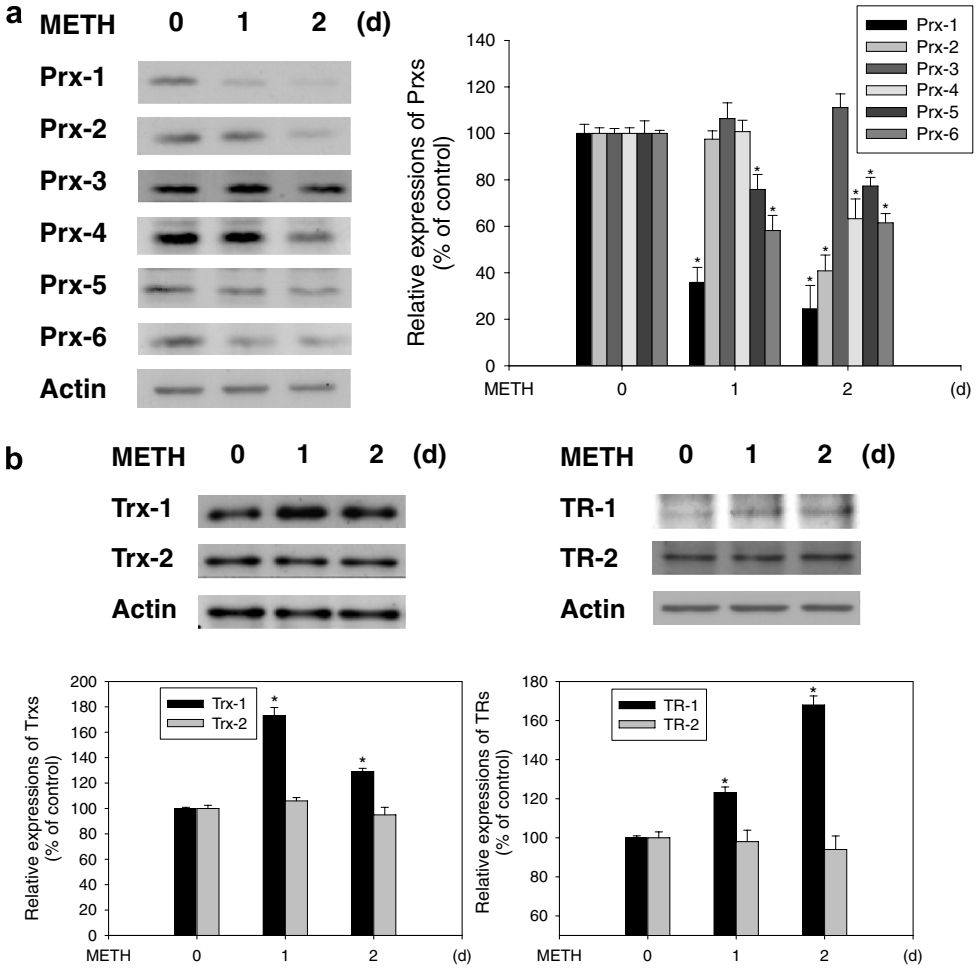

Fig. 2. Effects of METH on Prx, Trx, and TR expressions. Cells treated with or without METH were harvested and analyzed by Western blotting. The transferred membranes were probed with different kinds of antibodies for (a) Prxs, (b) Trxs, or TRs. Data points represent the normalized intensity of Prxs, Trxs, or TRs versus actin and are means ± SEM of at least three independent experiments ($n = 3$–6). *$p < 0.05$ compared to the control group.

currently unknown, these phenomena were similarly found in the mouse during the embryonic stage, where elevated transcripts of Trx-1 and TR-1 rather than Trx-2 and TR-2 were observed as well [30]. Increased levels of Trx-1 and TR-1 have also been detected in primate lungs [31] and PC12 cells (Supplement 2) in response to oxidative stress. Thus, for the diverse properties of the Trx system in reducing oxidized Prxs or modulating signal transductions, it is possible that METH-upregulated Trx-1 and TR-1 expressions could be a compensatory effect owing to decreased Prxs. However, further studies were required to dissect these questions.

Besides, other antioxidant defense system, such as superoxide dismutase, catalase, glutathione peroxidase, or glutathione, could also be important and have already been intensively studied during intoxication of METH or its analogs in human, animal, or cell line [32–35]. For instance, the decreased level of striatal glutathione has been demonstrated in METH-treated rats [33]. Besides, overexpression of glutathione peroxidase in rat pheochromocytoma cells resulting in protection against METH has also been done [35]. We also found the alterations of these anti-oxidative defense systems (data not shown). Recently, since the crosstalk between glutathione and Trx (or Prx) has been intensively studied [36], revealing the interactions between glutathione and Trx (or Prx) during METH intoxication could be another interesting topic.

METH increased overoxidation of Prxs and oxidative state of Trx-1

Consistent with others' findings [37], METH significantly increased intracellular H_2O_2 and $\cdot O_2^-$ levels (Fig. 3a). On the other hand, Prx-1–5 have two conserved Cys residues (as exemplified by Cys^{51} and Cys^{172} of Prx-1) [38]. The active site of Cys^{51} is oxidized to cysteine sulfenic acid (Cys-SOH) when Prx reduces peroxide. The unstable Cys^{51}-SOH reacts with Cys^{172}-SH to form a disulfide which is then reduced back to the active form by the Trx system. However, under oxidative stress conditions, the sulfenic intermediate (Cys-SOH) can easily be overoxidized to cysteine sulfinic acid (Cys-SO_2H) or cysteine sulfonic acid

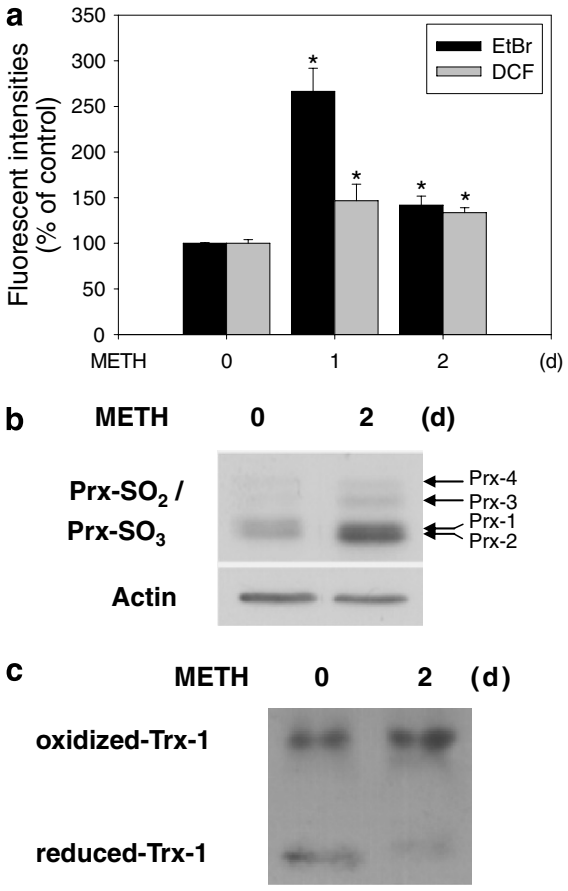

Fig. 3. Effects of METH on free radical formation and oxidation of Prx and Trx. (a) Cells treated with or without METH for 1 and 2d were stained with DCFDA or DHE. After flow-cytometric analysis, the fluorescent intensities of the control group were expressed as a percentage of the mean values of FL-1 (DCF) or FL-2 (EtBr). Data points represent at least three independent experiments ($n = 3$–6). *$p < 0.05$ compared to the control group. (b) Cells treated with or without METH were harvested and analyzed by Western blotting for visualizing overoxidation of Prxs. The predicted sizes of Prxs were indicated. (c) Cells treated with or without METH were harvested and carboxymethylated for visualizing oxidized/reduced state of Trx.

(Cys-SO_3H) before disulfide bond formation. Overoxidation impairs the functions of Prxs. We thus further characterized if overoxidized Prxs would occur after METH treatment. Our data indicated that METH significantly increased the overoxidized forms of Prxs (Fig. 3b), suggesting the occurrence of METH-induced oxidative stress and the inability of Prxs to cope with them. Immunoprecipitation of each Prx will be required to study the extent of oxidative insult.

On the other hand, Trx is a small protein with two redox-active cysteine residues in an active center (Cys-Gly-Pro-Cys-). After oxidative insult or reducing process, disulfide bond will form between these two reduced cysteine residues that result in dysfunctional oxidized Trx. TR in coupling with NADPH regenerates the oxidized form of Trx into reduced form. Consistent with the occurrence of METH-induced oxidative stress, although METH-upregulated Trx-1 expression (Fig. 2b, left panel), most of the Trx-1 was in oxidative status (Fig. 3c), this has further exacerbated the apoptotic events. In addition to the redox function, Trxs and TRs also exhibit a wide range of cellular functions [15,17]. For instance, Trx binds to a variety of cellular proteins, such as ASK1, Ref-1, PKC, and NF-κB [39]. Among them, ASK1 is an apoptosis signal-regulating kinase 1 and an activator of the c-Jun N-terminal kinase (JNK) pathway which is required for TNF-α-induced apoptosis [40]. In addition, METH has been shown to activate JNK and c-Jun [41] and c-Jun-deficient mice show attenuation of METH-induced neuronal apoptosis [42], suggesting a role of JNK in mediating METH neurotoxicity. Indeed, a JNK inhibitor, dicumarol, did significantly prevent METH-induced apoptosis supporting this viewpoint (Supplement 3). Since Trx-1 in reduced state binds and inactivates ASK1 signaling [43], it is possible that the elevation of Trx-1 might quench METH-induced ASK1/JNK activation as a self-protective mechanism. However, the simultaneous increase of oxidized Trx-1 retarded this mechanism and finally led to apoptosis.

Taken together, METH-induced downregulation of Prxs and upregulation of Trx-1/TR-1 have been documented. For the importance of Prxs and Trx in regulating apoptosis and oxidative stress, METH-induced altered regulation and oxidation of Prxs and Trx could contribute to a novel neurotoxic mechanism.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.bbrc.2006.12.138.

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