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Protective potentials of far-infrared ray against neuropsychotoxic conditions

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ABSTRACT

Compelling evidence suggests that far-infrared ray (FIR) possesses beneficial effects on emotional disorders. However, the underlying mechanism conveyed by FIR remains unclear. Recently, we demonstrated that exposure to FIR induces antioxidant potentials via up-regulation of glutathione peroxidase (GPx)-1 gene. The antioxidant potentials might be important for the modulation on the neuropsychotoxic conditions. Exposure to FIR protects from methamphetamine (MA)-induced memory impairments via phosphorylation of ERK $_{1/2}$ signaling by positive modulation of protein kinase C δ (PKCδ), M1 muscarinic acetylcholine receptor (M1 mAChR), and nuclear factor E2-related factor 2 (Nrf2) transcription factor. In addition, exposure to FIR positively modulates MA-induced behavioral sensitization via attenuating mitochondrial dysfunction by down-regulation of dopamine D1 receptor. In this mini-review, we have discussed with the protective potentials mediated by FIR against MA-induced psychotoxic burdens.

1. Introduction

Infrared radiation is an invisible electromagnetic wave with longer wavelengths than visible light. The International Commission on Illumination subdivided infrared rays into three broad categories based on wavelength: (1) near-infrared ray (0.7–1.4 μm), (2) middle infrared ray (1.4–3 μm), and (3) far-infrared ray (FIR) (3–1000 μm) (Toyokawa et al., 2003; Vatansever and Hamblin, 2012). Among them only FIR transfer energy in the form of heat (Plaghki et al., 2010). According to the clinical applications, FIR treatment has been divided into two categories; 1) FIR emitter composed of electrified ceramic plates, which is placed 20 cm above a patient and steadily increases the skin temperature (Karstens et al., 2006), and 2) FIR dry sauna therapy (Teraoka et al., 2004); which unlike traditional saunas, transfer heat directly without using air as a medium (Ishi et al., 1991). In terms of standard experimental conditions for animals, FIR panel was positioned at the height of 40 cm above the mice. This panel emits FIR ranging from 5 to 20 μm. The panel surface temperature was controlled at 40 °C. (Mai et al., 2018a, 2018b, 2018c; Nagasawa et al., 1999; Shui et al., 2015; Tran et al., 2016; Udagawa and Nagasawa, 2000).

Although precise mechanism on the cerebral thermoregulation mediated by FIR remains to be further elucidated, neuroimaging and magnetic resonance studies have revealed that FIR exposure eventually increases the temperature of body tissues, resulting in high motility of water molecules due to a decrease in the size of water clusters (Inoue and Kabaya, 1989). Therefore, it might be speculated that FIR stimulates the penetration of water molecules into various sites inside of the body tissue and also modulates dynamic functions of humoral factors in the body fluid (Imamura et al., 2001; Vatansever and Hamblin, 2012).

Health benefits of FIR have been reported in diverse morbid

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conditions (Lin et al., 2008; Nagasawa et al., 1999; Shui et al., 2015; Vatansever and Hamblin, 2012). It is also well-known that exposure to FIR exhibits antioxidant and anti-inflammatory potentials (Chang et al., 2016; Mai et al., 2018a, 2018b, 2018c; Tran et al., 2016; Vatansever and Hamblin, 2012). In addition, FIR treatment facilitates the anti-depressive efficacy (Tsai et al., 2007). Similarly, we demonstrated that exposure to FIR protects against acute restraint stress-induced oxidative damage via induction of GPx-1 gene (Tran et al., 2016).

Interestingly, it was reported that constant exposure to stress enhances abusive potentials (Deroche et al., 1992; Hahn et al., 1986; Leao et al., 2009; Pacchioni et al., 2002). Although pharmacodynamic of MA is similar to those of amphetamines, MA is highly addictive because of high CNS penetration rate and long lasting action. MA drug abuse also can be allied with a high risk of stressful conditions, i.e., homelessness, unemployment, and antisocial behavior (Glasner-Edwards and Mooney, 2014).

MA drug abuse leads to schizophrenia-like symptoms, such as hallucination and delusion (Yui et al., 1997, 2002). Moreover, clinical reports suggested that chronic MA use causes cognitive impairment (Scott et al., 2007). We demonstrated that repeated MA treatment induces recognition memory impairments and can be used as an animal model for schizophrenia and MA psychosis (Kamei et al., 2006; Tran et al., 2018).

Up to now, efficient pharmacological modulators against MA-induced disorder have not been developed yet (Forray and Sofuoglu, 2014). This mini-review highlighted the therapeutic potentials mediated by FIR against MA-induced psychotoxicity (i.e., memory impairments and behavioral sensitization).

2. Exposure to FIR positively modulates protein kinase Cδ (PKCδ) in the prefrontal cortex (PFC) of the mice

PKC, a well-known cognitive kinase, plays an important regulatory role in cellular signaling (Talman et al., 2016). In particular, PKCδ is a major proinflammatory kinase, which regulates oxidative stress, apoptosis, and dopaminergic system (Dang et al., 2015; Kanthasamy et al., 2003; Nam et al., 2015; Nguyen et al., 2015). MA treatment selectively and specifically induces PKCδ out of other isoenzymes (Shin et al., 2011), and causes memory impairments (Tran et al., 2018). Exposure to FIR inhibits induction of PKCδ, and subsequently protects against MA-induced memory impairments (Mai et al., 2018b). Importantly, we recently demonstrated that PKC activator, bryostatin-1, significantly activates PKCδ, and counteracts FIR-mediated protective effects against MA-induced memory impairments (Mai et al., 2018c). Moreover, the memory-enhancing effect of FIR is comparable to that of genetic depletion of PKCδ (Mai et al., 2018b, 2018c; Tran et al., 2018). Therefore, we propose that PKCδ might be a molecular target for FIR against MA-induced memory dysfunction.

It has been demonstrated that FIR increases endothelial nitric oxide synthase (eNOS) gene expression (Akasaki et al., 2006; Park et al., 2013a), which might lead to the normalization of autonomic nervous activity (Kuwahata et al., 2011). Furthermore, inhibition of PKCδ modulates eNOS (Iwase et al., 2000; Monti et al., 2010) and glutathione system (Ghigo et al., 1996; Hofmann and Schmidt, 1995). Therefore, it is plausible that FIR-mediated up-regulation of eNOS and GSH might down-regulate PKCδ activation, which might be important for FIR-mediated neuroprotective potentials (Mai et al., 2018a, 2018b, 2018c).

3. Exposure to FIR facilitates nuclear factor E2-related factor 2 (Nrf2)-dependent glutathione system in the prefrontal cortex (PFC)

MA treatment decreases reduced glutathione (GSH) levels, and it facilitates oxidation-dependent memory impairments (Mai et al., 2018b, 2018c; Tran et al., 2018). GSH plays a critical role in preventing oxidative stress induced by MA (Kim et al., 1999; Mai et al., 2018b; Shin et al., 2014). MA-induced endoplasmic reticulum stress might be responsible for the GSH depletion (Krasnova and Cadet, 2009; Yang et al., 2018), and might cause up-regulation of the enzymes, responsible for GSH biosynthesis through the Nrf2/antioxidant responsive element (ARE) signaling (Jayanthi et al., 2009). Activation of Nrf2 also requires induction of c-glutamylcysteine ligase modifier subunit (GCLm), c-glutamylcysteine ligase catalytic subunit (GCLc), and glutathione peroxidase (GPx) (Mai et al., 2018c; Singh et al., 2008). Exposure to FIR positively modulates Nrf2-related system, and protects against MA-induced impaired recognition memory (Mai et al., 2018c). This positive modulation of the Nrf2 system might be essential for FIR-mediated antioxidative potential in response to MA.

4. Exposure to FIR positively modulates interaction between M1 muscarinic acetylcholine receptor (M1 mAChR) and ERK $_{1/2}$ in the prefrontal cortex (PFC)

mAChRs are divided into five subunits (i.e., M1-M5) (Nathan et al., 2013). It is well-known that a selective M1 antagonist (i.e., dicyclomine) causes memory dysfunction (Kim et al., 2013; Nathan et al., 2013). Consistently, M1 mAChR activation is critical for enhancing memory function via JAK2/STAT3 signaling pathway (Kim et al., 2013; Park et al., 2013b). Repeated MA treatment significantly down-regulates M1 mAChR, and results in recognition memory impairment (Mai et al., 2018c). A selective agonist of M1 mAChR (i.e., GSK1034702) enhances cognitive function, and M1/M4 agonists (i.e., xanomeline) improve psychosis-related symptoms (Foster et al., 2014; Nathan et al., 2013). Similarly, exposure to FIR rescues MA-induced memory impairments via up-regulation of M1 mAChR (Mai et al., 2018c). Therefore, it is plausible that FIR-mediated memory enhancing effects require M1 mAChR against MA (Mai et al., 2018c).

$ERK_{1/2}$ signaling plays a critical role in psychostimulant-induced neural plasticity (Nestler, 2001; Valjent et al., 2001), in association with memory impairments (Zhu et al., 2012). ERK $_{1/2}$ activation in PFC protects against MA-induced memory impairments (Kamei et al., 2006; Tran et al., 2018), whereas inactivation of $ERK_{1/2}$ impairs memory function in rodents (Schafe et al., 2000; Tran et al., 2018). Exposure to FIR attenuates MA-induced memory dysfunction via activation of $ERK_{1/2}$ signaling by the interactive modulation between M1 mAChR and Nrf2 (Mai et al., 2018c). Importantly, we demonstrated that pharmacological inhibitor of ERK, i.e., U0126, or M1 mAChR antagonist, i.e., dicyclomine, counteracts against FIR-mediated memory enhancing potentials (Mai et al., 2018c). More importantly, these protective potentials of FIR are comparable to those by clozapine, or genetic depletion of PKCδ in mice (Kamei et al., 2006; Mai et al., 2018b, 2018c; Tran et al., 2018), suggesting that FIR application can be a potential therapeutic tool in response to MA-induced memory impairments.

5. Exposure to FIR positively modulates dopamine D1 receptor and c-Fos immunoreactivity (c-Fos-IR) in the striatum

It has been well-known that dopamine receptors mediate MA-induced behavioral sensitization. Earlier studies have demonstrated that dopamine D1 or D2 receptor antagonists attenuate MA-induced behavioral sensitization (Kuribara, 1995; Kuribara and Uchihashi, 1994). In contrast, Shuto et al. (2006) reported that activation of D1 receptor reversed MA-induced behavioral sensitization. However, we and others have demonstrated that D1 receptor antagonist, SCH23390, attenuates MA-induced behavioral sensitization (Kelly et al., 2008; Mai et al., 2018a). Moreover, recently we reported that FIR attenuates MA-induced behavioral sensitization via down-regulation of dopamine D1 receptor. We also demonstrated that the protective potentials by dopamine D1 receptor antagonist, i.e., SCH23390, are comparable to those by FIR (Mai et al., 2018a). Therefore, the role of D1 receptor remains to be further explored against MA-mediated drug dependence. Previously, we showed that exposure to FIR attenuates c-Fos-IR and

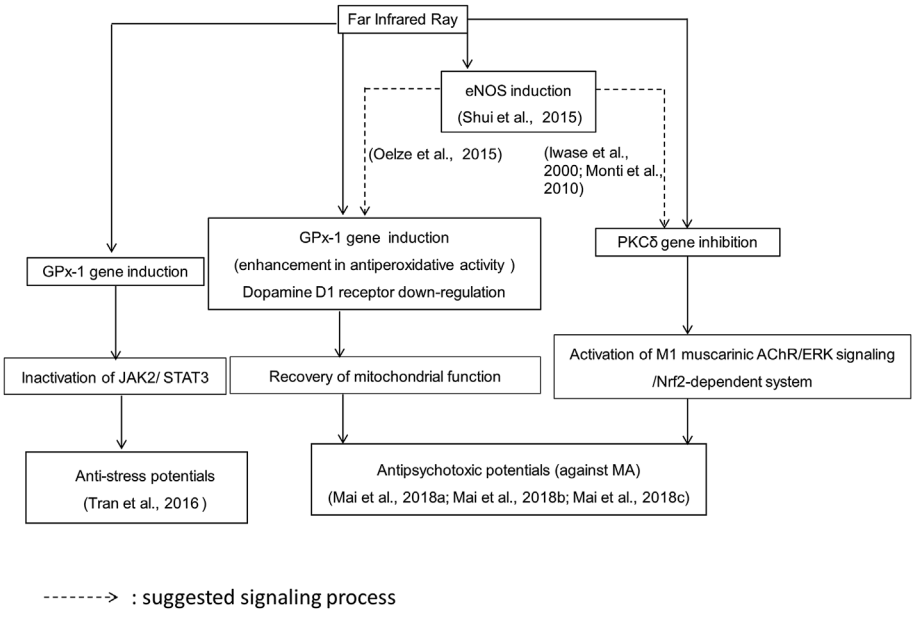

Fig. 1. Schematic diagram on the protective potential mediated by far-infrared ray (FIR) against neuropsychotoxicity. Exposure to FIR induces glutathione peroxidase-1 (GPx-1) gene and GPx-1 gene attenuates acute restraint stress via inactivation of JAK2/STAT3 signaling pathway (Tran et al., 2016). In addition, FIR-mediated GPx-1 gene induction modulates dopamine D1 receptor, followed by recovering mitochondrial dysfunction. This phenomenon may be helpful for protecting drug dependence (Mai et al., 2018a). In contrast, FIR-mediated eNOS induction might modulate GPx-1 gene and PKCδ gene (Iwase et al., 2000; Monti et al., 2010; Oelze et al., 2014; Shui et al., 2015). FIR-mediated PKCδ inhibition activates muscarinic M1 acetylcholine receptor (M1 mAChR) as well as ERK signaling, and further facilitates nuclear factor E2-related factor 2 (Nrf2)- dependent system. This signaling process may be important for FIR-mediated memory enhancing potentials against MA exposure (Mai et al., 2018b, 2018c).

oxidative burdens induced by acute restraint stress (Tran et al., 2012). Similarly, exposure to FIR attenuates MA-induced increase in c-Fos-IR in the nucleus accumbens, and consequently alleviates oxidative damage (Mai et al., 2018a; Tran et al., 2016).

6. Exposure to FIR restores homeostasis of enzymatic antioxidants in the striatum

We demonstrated that MA treatment significantly decreases GPx-1 expression (Kim et al., 1999; Tran et al., 2018), and that depletion of GPx-1 potentiates oxidative stress-responsible neurotoxicity (Shin et al., 2008, 2014; Tran et al., 2016). Consistently, genetic overexpression of GPx-1 inhibits against oxidative stress-responsible neurotoxicity (Lubos et al., 2011). Importantly, it is plausible that the loss of homeostasis in enzymatic antioxidants (i.e., between GPx and superoxide dismutase) might cause behavioral sensitization in rodents (Tran et al., 2017). It has been reported that FIR induces antioxidant potentials via the homeostasis in response to behavioral sensitization-evoked oxidative burdens (Mai et al., 2018a, 2018c; Tran et al., 2016).

7. Exposure to FIR attenuates mitochondrial dysfunction in the striatum

We have reported that MA induces mitochondrial dysfunction with behavioral sensitization (Mai et al., 2018a; Shin et al., 2016, 2017). Repeated MA treatment caused mitochondrial dysfunction (i.e., mitochondrial transmembrane potential, and accumulation of intramitochondrial Ca^{2+}), decreased mitochondrial complex I activity, and mitochondrial oxidative burdens (Mai et al., 2018a; Shin et al., 2016, 2017). Importantly, drug abuse can alter mitochondrial-related genes (Sokolov et al., 2003). In addition, mitochondrial alteration in drug dependence has been recognized (Feng et al., 2013; Liou et al., 2014; Mai et al., 2018a). Recently we reported that recovery of mitochondrial function may be important for alleviating behavioral sensitization induced by MA (Mai et al., 2018a). Similarly, one report indicated that FIR provides protective effects on a neurodegenerative cell model of spinocerebellar ataxia type 3 via improvement in mitochondrial respiratory function (Chang et al., 2016). Our findings also suggested that exposure to FIR ameliorates mitochondrial dysfunction possibly via inhibition of dopamine D1 receptor, and induction of mitochondrial antioxidant enzyme (Mai et al., 2018a).

8. Conclusion

It is recognized that there is no feasible therapeutic approach available against psychotoxic effects induced by MA. Importantly, here we suggest that exposure to FIR protects from MA-induced psychotoxicity. Memory enhancing activity of FIR against MA requires up-regulation of M1 mAChR, Nrf2-dependent system, and $ERK_{1/2}$ by inhibiting PKCδ phosphorylation. Exposure to FIR also protects against behavioral sensitization induced by MA via attenuating mitochondrial dysfunction by up-regulation of GPx-1. Recovery of mitochondrial function may be helpful for FIR-mediated psychoprotective activity. The antipsychotic potentials by FIR may be, at least in part, comparable to those by clozapine. Combined, we propose that application of FIR might constitute a potential therapeutic tool against neuropsychotoxic conditions induced by MA or possibly other psychostimulants (Fig. 1).

Conflicts of interest

Authors reported no potential conflicts of interest to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neuint.2018.11.019.

References

Akasaki, Y., Miyata, M., Eto, H., Shirasawa, T., Hamada, N., Ikeda, Y., Biro, S., Otsuji, Y., Tei, C., 2006. Repeated thermal therapy up-regulates endothelial nitric oxide synthase and augments angiogenesis in a mouse model of hindlimb ischemia. Circ. J. 70, 463–470.

Chang, J.C., Wu, S.L., Hoel, F., Cheng, Y.S., Liu, K.H., Hsieh, M., Hoel, A., Tronstad, K.J.,

Yan, K.C., Hsieh, C.L., Lin, W.Y., Kuo, S.J., Su, S.L., Liu, C.S., 2016. Far-infrared radiation protects viability in a cell model of Spinocerebellar Ataxia by preventing polyQ protein accumulation and improving mitochondrial function. Sci. Rep. 6, 30436.

Dang, D.K., Duong, C.X., Nam, Y., Shin, E.J., Lim, Y.K., Jeong, J.H., Jang, C.G., Nah, S.Y., Nabeshima, T., Kim, H.C., 2015. Inhibition of protein kinase (PK) Cdelta attenuates methamphetamine-induced dopaminergic toxicity via upregulation of phosphorylation of tyrosine hydroxylase at Ser40 by modulation of protein phosphatase 2A and PKA. Clin. Exp. Pharmacol. Physiol. 42, 192–201.

Deroche, V., Piazza, P.V., Casolini, P., Maccari, S., Le Moal, M., Simon, H., 1992. Stress-induced sensitization to amphetamine and morphine psychomotor effects depend on stress-induced corticosterone secretion. Brain Res. 598, 343–348.

Feng, Y.M., Jia, Y.F., Su, L.Y., Wang, D., Lv, L., Xu, L., Yao, Y.G., 2013. Decreased mitochondrial DNA copy number in the hippocampus and peripheral blood during opiate addiction is mediated by autophagy and can be salvaged by melatonin. Autophagy 9, 1395–1406.

Forray, A., Sofuoglu, M., 2014. Future pharmacological treatments for substance use disorders. Br. J. Clin. Pharmacol. 77, 382–400.

Foster, D.J., Choi, D.L., Conn, P.J., Rook, J.M., 2014. Activation of M1 and M4 muscarinic receptors as potential treatments for Alzheimer's disease and schizophrenia. Neuropsychiatric Dis. Treat. 10, 183–191.

Ghigo, D., Geromin, D., Franchino, C., Todde, R., Priotto, C., Costamagna, C., Arese, M., Garbarino, G., Pescarmona, G.P., Bosia, A., 1996. Correlation between nitric oxide synthase activity and reduced glutathione level in human and murine endothelial cells. Amino Acids 10, 277–281.

Glasner-Edwards, S., Mooney, L.J., 2014. Methamphetamine psychosis: epidemiology and management. CNS Drugs 28, 1115–1126.

Hahn, B., Zacharko, R.M., Anisman, H., 1986. Alterations of amphetamine elicited perseveration and locomotor excitation following acute and repeated stressor application. Pharmacol. Biochem. Behav. 25, 29–33.

Hofmann, H., Schmidt, H.H., 1995. Thiol dependence of nitric oxide synthase. Biochemistry 34, 13443–13452.

Imamura, M., Biro, S., Kihara, T., Yoshifuku, S., Takasaki, K., Otsuji, Y., Minagoe, S., Toyama, Y., Tei, C., 2001. Repeated thermal therapy improves impaired vascular endothelial function in patients with coronary risk factors. J. Am. Coll. Cardiol. 38, 1083–1088.

Inoue, S., Kabaya, M., 1989. Biological activities caused by far-infrared radiation. Int. J. Biometeorol. 33, 145–150.

Ishi, K., Shibata, Y., Takahashi, T., Mishiro, H., Ohsaka, T., Ikezawa, M., Kondo, Y., Nakazato, T., Urasawa, S., Niimura, N., Kato, R., Shibasaki, Y., Oyamada, M., 1991. Spectrum of coherent synchrotron radiation in the far-infrared region. Phys. Rev. A. 43, 5597–5604.

Iwase, K., Miyanaka, K., Shimizu, A., Nagasaki, A., Gotoh, T., Mori, M., Takiguchi, M., 2000. Induction of endothelial nitric-oxide synthase in rat brain astrocytes by systemic lipopolysaccharide treatment. J. Biol. Chem. 275, 11929–11933.

Jayanthi, S., McCoy, M.T., Beauvais, G., Ladenheim, B., Gilmore, K., Wood 3rd, W., Becker, K., Cadet, J.L., 2009. Methamphetamine induces dopamine D1 receptor-dependent endoplasmic reticulum stress-related molecular events in the rat striatum. PLoS One 4 e6092.

Kamei, H., Nagai, T., Nakano, H., Togan, Y., Takayanagi, M., Takahashi, K., Kobayashi, K., Yoshida, S., Maeda, K., Takuma, K., Nabeshima, T., Yamada, K., 2006. Repeated methamphetamine treatment impairs recognition memory through a failure of novelty-induced ERK1/2 activation in the prefrontal cortex of mice. Biol. Psychiatry 59, 75–84.

Kanthasamy, A.G., Kitazawa, M., Kanthasamy, A., Anantharam, V., 2003. Role of proteolytic activation of protein kinase Cdelta in oxidative stress-induced apoptosis. Antioxidants Redox Signal. 5, 609–620.

Karstens, W., Bobela, D.C., Smith, D.Y., 2006. Impurity and free-Carrier effects on the far-infrared dispersion spectrum of silicon. J. Opt. Soc. Am. A. Opt. Image Sci. Vis. 23, 723–729.

Kelly, M.A., Low, M.J., Rubinstein, M., Phillips, T.J., 2008. Role of dopamine D1-like receptors in methamphetamine locomotor responses of D2 receptor knockout mice. Gene Brain Behav. 7, 568–577.

Kim, B.K., Tran, H.Y., Shin, E.J., Lee, C., Chung, Y.H., Jeong, J.H., Bach, J.H., Kim, W.K., Park, D.H., Saito, K., Nabeshima, T., Kim, H.C., 2013. IL-6 attenuates trimethyltin-induced cognitive dysfunction via activation of JAK2/STAT3, M1 mAChR and ERK signaling network. Cell. Signal. 25, 1348–1360.

Kim, H.C., Jhoo, W.K., Choi, D.Y., Im, D.H., Shin, E.J., Suh, J.H., Floyd, R.A., Bing, G., 1999. Protection of methamphetamine nigrostriatal toxicity by dietary selenium. Brain Res. 851, 76–86.

Krasnova, I.N., Cadet, J.L., 2009. Methamphetamine toxicity and messengers of death. Brain Res. Rev. 60, 379–407.

Kuribara, H., 1995. Dopamine D1 receptor antagonist SCH 23390 retards methamphetamine sensitization in both combined administration and early posttreatment schedules in mice. Pharmacol. Biochem. Behav. 52, 759–763.

Kuribara, H., Uchihashi, Y., 1994. Effects of dopamine antagonism on methamphetamine sensitization: evaluation by ambulatory activity in mice. Pharmacol. Biochem. Behav. 47, 101–106.

Kuwahata, S., Miyata, M., Fujita, S., Kubozono, T., Shinsato, T., Ikeda, Y., Hamasaki, S., Kuwaki, T., Tei, C., 2011. Improvement of autonomic nervous activity by Waon therapy in patients with chronic heart failure. J. Cardiol. 57, 100–106.

Leao, R.M., Cruz, F.C., Planeta, C.S., 2009. Exposure to acute restraint stress reinstates nicotine-induced place preference in rats. Behav. Pharmacol. 20, 109–113.

Lin, C.C., Liu, X.M., Peyton, K., Wang, H., Yang, W.C., Lin, S.J., Durante, W., 2008. Far infrared therapy inhibits vascular endothelial inflammation via the induction of heme oxygenase-1. Arterioscler. Thromb. Vasc. Biol. 28, 739–745.

Liou, C.M., Tsai, S.C., Kuo, C.H., Williams, T., Ting, H., Lee, S.D., 2014. Chronic methamphetamine exposure induces cardiac fas-dependent and mitochondria-dependent apoptosis. Cardiovasc. Toxicol. 14, 134–144.

Lubos, E., Loscalzo, J., Handy, D.E., 2011. Glutathione peroxidase-1 in health and disease: from molecular mechanisms to therapeutic opportunities. Antioxidants Redox Signal. 15, 1957–1997.

Mai, H.N., Sharma, N., Shin, E.J., Nguyen, B.T., Jeong, J.H., Jang, C.G., Cho, E.H., Nah, S.Y., Kim, N.H., Nabeshima, T., Kim, H.C., 2018a. Exposure to far infrared ray protects methamphetamine-induced behavioral sensitization in glutathione peroxidase-1 knockout mice via attenuating mitochondrial burdens and dopamine D1 receptor activation. Neurochem. Res. 43, 1118–1135.

Mai, H.N., Sharma, N., Shin, E.J., Nguyen, B.T., Nguyen, P.T., Jeong, J.H., Cho, E.H., Lee, Y.J., Kim, N.H., Jang, C.G., Nabeshima, T., Kim, H.C., 2018b. Exposure to far-infrared ray attenuates methamphetamine-induced impairment in recognition memory through inhibition of protein kinase C delta in male mice: comparison with the antipsychotic clozapine. J. Neurosci. Res. 96, 1294–1310.

Mai, H.N., Sharma, N., Shin, E.J., Nguyen, B.T., Nguyen, P.T., Jeong, J.H., Jang, C.G., Cho, E.H., Nah, S.Y., Kim, N.H., Nabeshima, T., Kim, H.C., 2018c. Exposure to far-infrared rays attenuates methamphetamine-induced recognition memory impairment via modulation of the muscarinic M1 receptor, Nrf2, and PKC. Neurochem. Int. 116, 63–76.

Monti, M., Donnini, S., Giachetti, A., Mochly-Rosen, D., Ziche, M., 2010. deltaPKC inhibition or varepsilonPKC activation repairs endothelial vascular dysfunction by regulating eNOS post-translational modification. J. Mol. Cell. Cardiol. 48, 746–756.

Nagasawa, H., Udagawa, Y., Kiyokawa, S., 1999. Evidence that irradiation of far-infrared rays inhibits mammary tumour growth in SHN mice. Anticancer Res. 19, 1797–1800.

Nam, Y., Wie, M.B., Shin, E.J., Nguyen, T.T., Nah, S.Y., Ko, S.K., Jeong, J.H., Jang, C.G., Kim, H.C., 2015. Ginsenoside Re protects methamphetamine-induced mitochondrial burdens and proapoptosis via genetic inhibition of protein kinase C delta in human neuroblastoma dopaminergic SH-SY5Y cell lines. J. Appl. Toxicol. 35, 927–944.

Nathan, P.J., Watson, J., Lund, J., Davies, C.H., Peters, G., Dodds, C.M., Swirski, B., Lawrence, P., Bentley, G.D., O'Neill, B.V., Robertson, J., Watson, S., Jones, G.A., Maruff, P., Croft, R.J., Laruelle, M., Bullmore, E.T., 2013. The potent M1 receptor allosteric agonist GSK1034702 improves episodic memory in humans in the nicotine abstinence model of cognitive dysfunction. Int. J. Neuropsychopharmacol. 16, 721–731.

Nestler, E.J., 2001. Molecular basis of long-term plasticity underlying addiction. Nat. Rev. Neurosci. 2, 119–128.

Nguyen, X.K., Lee, J., Shin, E.J., Dang, D.K., Jeong, J.H., Nguyen, T.T., Nam, Y., Cho, H.J., Lee, J.C., Park, D.H., Jang, C.G., Hong, J.S., Nabeshima, T., Kim, H.C., 2015. Liposomal melatonin rescues methamphetamine-elicited mitochondrial burdens, proapoptosis, and dopaminergic degeneration through the inhibition PKCdelta gene. J. Pineal Res. 58, 86–106.

Oelze, M., Kroller-Schon, S., Steven, S., Lubos, E., Doppler, C., Hausding, M., Tobias, S., Brochhausen, C., Li, H., Torzewski, M., Wenzel, P., Bachschmid, M., Lackner, K.J., Schulz, E., Munzel, T., Daiber, A., 2014. Glutathione peroxidase-1 deficiency potentiates dysregulatory modifications of endothelial nitric oxide synthase and vascular dysfunction in aging. Hypertension 63, 390–396.

Pacchioni, A.M., Gioino, G., Assis, A., Cancela, L.M., 2002. A single exposure to restraint stress induces behavioral and neurochemical sensitization to stimulating effects of amphetamine: involvement of NMDA receptors. Ann. N. Y. Acad. Sci. 965, 233–246.

Park, J.H., Lee, S., Cho, D.H., Park, Y.M., Kang, D.H., Jo, I., 2013a. Far-infrared radiation acutely increases nitric oxide production by increasing Ca(2+) mobilization and Ca(2+)/calmodulin-dependent protein kinase II-mediated phosphorylation of endothelial nitric oxide synthase at serine 1179. Biochem. Biophys. Res. Commun. 436, 601–606.

Park, S.J., Shin, E.J., Min, S.S., An, J., Li, Z., Hee Chung, Y., Hoon Jeong, J., Bach, J.H., Nah, S.Y., Kim, W.K., Jang, C.G., Kim, Y.S., Nabeshima, Y., Nabeshima, T., Kim, H.C., 2013b. Inactivation of JAK2/STAT3 signaling axis and downregulation of M1 mAChR cause cognitive impairment in klotho mutant mice, a genetic model of aging. Neuropsychopharmacology 38, 1426–1437.

Plaghki, L., Decruynaere, C., Van Dooren, P., Le Bars, D., 2010. The fine tuning of pain thresholds: a sophisticated double alarm system. PLoS One 5, e10269.

Schafe, G.E., Atkins, C.M., Swank, M.W., Bauer, E.P., Sweatt, J.D., LeDoux, J.E., 2000. Activation of ERK/MAP kinase in the amygdala is required for memory consolidation of pavlovian fear conditioning. J. Neurosci. 20, 8177–8187.

Scott, J.C., Woods, S.P., Matt, G.E., Meyer, R.A., Heaton, R.K., Atkinson, J.H., Grant, I., 2007. Neurocognitive effects of methamphetamine: a critical review and meta-analysis. Neuropsychol. Rev. 17, 275–297.

Shin, E.J., Dang, D.K., Tran, H.Q., Nam, Y., Jeong, J.H., Lee, Y.H., Park, K.T., Lee, Y.S., Jang, C.G., Hong, J.S., Nabeshima, T., Kim, H.C., 2016. PKCdelta knockout mice are protected from para-methoxymethamphetamine-induced mitochondrial stress and associated neurotoxicity in the striatum of mice. Neurochem. Int. 100, 146–158.

Shin, E.J., Dang, D.K., Tran, T.V., Tran, H.Q., Jeong, J.H., Nah, S.Y., Jang, C.G., Yamada, K., Nabeshima, T., Kim, H.C., 2017. Current understanding of methamphetamine-associated dopaminergic neurodegeneration and psychotoxic behaviors. Arch. Pharm. Res. 40, 403–428.

Shin, E.J., Duong, C.X., Nguyen, X.T., Bing, G., Bach, J.H., Park, D.H., Nakayama, K., Ali, S.F., Kanthasamy, A.G., Cadet, J.L., Nabeshima, T., Kim, H.C., 2011. PKCdelta inhibition enhances tyrosine hydroxylase phosphorylation in mice after methamphetamine treatment. Neurochem. Int. 59, 39–50.

Shin, E.J., Ko, K.H., Kim, W.K., Chae, J.S., Yen, T.P., Kim, H.J., Wie, M.B., Kim, H.C., 2008. Role of glutathione peroxidase in the ontogeny of hippocampal oxidative stress and kainate seizure sensitivity in the genetically epilepsy-prone rats. Neurochem. Int. 52, 1134–1147.

Shin, E.J., Shin, S.W., Nguyen, T.T., Park, D.H., Wie, M.B., Jang, C.G., Nah, S.Y., Yang,

B.W., Ko, S.K., Nabeshima, T., Kim, H.C., 2014. Ginsenoside Re rescues methamphetamine-induced oxidative damage, mitochondrial dysfunction, microglial activation, and dopaminergic degeneration by inhibiting the protein kinase Cdelta gene. Mol. Neurobiol. 49, 1400–1421.

Shui, S., Wang, X., Chiang, J.Y., Zheng, L., 2015. Far-infrared therapy for cardiovascular, autoimmune, and other chronic health problems: a systematic review. Exp. Biol. Med. (Maywood) 240, 1257–1265.

Shuto, T., Kuroiwa, M., Hamamura, M., Yabuuchi, K., Shimazoe, T., Watanabe, S., Nishi, A., Yamamoto, T., 2006. Reversal of methamphetamine-induced behavioral sensitization by repeated administration of a dopamine D1 receptor agonist. Neuropharmacology 50, 991–997.

Singh, A., Boldin-Adamsky, S., Thimmulappa, R.K., Rath, S.K., Ashush, H., Coulter, J., Blackford, A., Goodman, S.N., Bunz, F., Watson, W.H., Gabrielson, E., Feinstein, E., Biswal, S., 2008. RNAi-mediated silencing of nuclear factor erythroid-2-related factor 2 gene expression in non-small cell lung cancer inhibits tumor growth and increases efficacy of chemotherapy. Cancer Res. 68, 7975–7984.

Sokolov, B.P., Jiang, L., Trivedi, N.S., Aston, C., 2003. Transcription profiling reveals mitochondrial, ubiquitin and signaling systems abnormalities in postmortem brains from subjects with a history of alcohol abuse or dependence. J. Neurosci. Res. 72, 756–767.

Talman, V., Pascale, A., Jantti, M., Amadio, M., Tuominen, R.K., 2016. Protein kinase C activation as a potential therapeutic strategy in Alzheimer's Disease: is there a role for embryonic lethal abnormal vision-like proteins? Basic Clin. Pharmacol. Toxicol. 119, 149–160.

Teraoka, F., Hamada, Y., Takahashi, J., 2004. Bamboo charcoal inhibits growth of HeLa cells in vitro. Dent. Mater. J. 23, 633–637.

Toyokawa, H., Matsui, Y., Uhara, J., Tsuchiya, H., Teshima, S., Nakanishi, H., Kwon, A.H., Azuma, Y., Nagaoka, T., Ogawa, T., Kamiyama, Y., 2003. Promotive effects of far-infrared ray on full-thickness skin wound healing in rats. Exp. Biol. Med. (Maywood) 228, 724–729.

Tran, H.Y., Shin, E.J., Saito, K., Nguyen, X.K., Chung, Y.H., Jeong, J.H., Bach, J.H., Park, D.H., Yamada, K., Nabeshima, T., Yoneda, Y., Kim, H.C., 2012. Protective potential of IL-6 against trimethyltin-induced neurotoxicity in vivo. Free Radic. Biol. Med. 52, 1159–1174.

Tran, T.H., Mai, H.N., Shin, E.J., Nam, Y., Nguyen, B.T., Lee, Y.J., Jeong, J.H., Tran, H.Y., Cho, E.H., Nah, S.Y., Lei, X.G., Nabeshima, T., Kim, N.H., Kim, H.C., 2016. Repeated exposure to far infrared ray attenuates acute restraint stress in mice via inhibition of JAK2/STAT3 signaling pathway by induction of glutathione peroxidase-1. Neurochem. Int. 94, 9–22.

Tran, T.V., Shin, E.J., Jeong, J.H., Lee, J.W., Lee, Y., Jang, C.G., Nah, S.Y., Lei, X.G., Toriumi, K., Yamada, K., Nabeshima, T., Kim, H.C., 2017. Protective potential of the glutathione peroxidase-1 gene in abnormal behaviors induced by phencyclidine in mice. Mol. Neurobiol. 54, 7042–7062.

Tran, T.V., Shin, E.J., Nguyen, L.T.T., Lee, Y., Kim, D.J., Jeong, J.H., Jang, C.G., Nah, S.Y., Toriumi, K., Nabeshima, T., Yamada, K., Kim, H.C., 2018. Protein kinase Cdelta gene depletion protects against methamphetamine-induced impairments in recognition memory and $ERK_{1/2}$ signaling via upregulation of glutathione peroxidase-1 gene. Mol. Neurobiol. 55, 4136–4159.

Tsai, J.F., Hsiao, S., Wang, S.Y., 2007. Infrared irradiation has potential antidepressant effect. Prog. Neuro-Psychopharmacol. Biol. Psychiatry 31, 1397–1400.

Udagawa, Y., Nagasawa, H., 2000. Effects of far-infrared ray on reproduction, growth, behaviour and some physiological parameters in mice. In Vivo 14, 321–326.

Valjent, E., Caboche, J., Vanhoutte, P., 2001. Mitogen-activated protein kinase/extracellular signal-regulated kinase induced gene regulation in brain: a molecular substrate for learning and memory? Mol. Neurobiol. 23, 83–99.

Vatansever, F., Hamblin, M.R., 2012. Far infrared radiation (FIR): its biological effects and medical applications. Photon. Laser Med. 4, 255–266.

Yang, X., Wang, Y., Li, Q., Zhong, Y., Chen, L., Du, Y., He, J., Liao, L., Xiong, K., Yi, C.X., Yan, J., 2018. The main molecular mechanisms underlying methamphetamine- induced neurotoxicity and implications for pharmacological treatment. Front. Mol. Neurosci. 11, 186.

Yui, K., Goto, K., Ikemoto, S., Ishiguro, T., 1997. Methamphetamine psychosis: spontaneous recurrence of paranoid-hallucinatory states and monoamine neurotransmitter function. J. Clin. Psychopharmacol. 17, 34–43.

Yui, K., Ikemoto, S., Goto, K., Nishijima, K., Yoshino, T., Ishiguro, T., 2002. Spontaneous recurrence of methamphetamine-induced paranoid-hallucinatory states in female subjects: susceptibility to psychotic states and implications for relapse of schizophrenia. Pharmacopsychiatry 35, 62–71.

Zhu, J., Chen, Y., Zhao, N., Cao, G., Dang, Y., Han, W., Xu, M., Chen, T., 2012. Distinct roles of dopamine D3 receptors in modulating methamphetamine-induced behavioral sensitization and ultrastructural plasticity in the shell of the nucleus accumbens. J. Neurosci. Res. 90, 895–904.