melatonintreatment, which were similar/increased in comparison to those of COPD model. Co-treatmentof melatonin and SIRT1 inhibitor was slightly observed the reduction inpathophysiological factors in comparison to only melatonin treatment.

Conclusions: Therefore, our results indicate that melatonin attenuates inflammatoryresponse in CS and LPS exposure via the reduction in NF-κB acetylation by elevation of SIRT1expression.

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Upregulation of Hypoxia-inducible factor-1a in Calbindin-D_{9k} Knockout mice

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Introduction: Environment in military actionsuch as underwater or airborne, soldiers are frequently exposed to hypoxicatmosphere. Hypoxia is involved with various human disease from inflammation tocancers that has fascinated medical researchers as well as military engineers.Hypoxia-inducible factor (HIF) is the key controller in hypoxia that triggersmore than 1,000 related genes. Recently, many studies suggest that HIF systemsupervise the intracellular calcium control. In our previous study we identified significant changes in geneexpression focused on calcium channel-related proteins, between hypoxia andnormoxic group. Calbindin-d9k is a cytosolic calcium-binding protein thatparticipates in intracellular calcium absorption. In this study, we investigateinteraction between HIF1a and calbindin-D_{9k}.

Materials and Methods: 8 weeks old C57BL/6 mice and Calbindin-D9K Knockout mice were exposed to hypoxia for 3 weeks. Hypoxiccondition was created in polycarbonate chamber with nitrogen supply to removeoxygen. Oxygen concentration were measured and maintained thoroughly about 12±2% partial pressure of O2.Expression of HIF1a in kidney were analyzed by western blotting.

Results: HIF1a protein expression was increased in calbindin-D_{9k} knockout mice compared to that of wild type mouse innormal oxygen atmosphere. However, in hypoxia, knockout mice showed decreasedprotein level.

Conclusions: Calbindin-D_{9k} knockout miceshow the enhanced HIF1a expression when normal atmosphere. Our finding suggestthat calbindin-D_{9k} might be involve in HIF systems.

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Platelet rich plasma improves dermal therapeutic effect of mesenchymal stem cells via enhancing angiogenesis

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Introduction: Skin injury is a main complication that could be occurred after radiotherapy. Moreover, vascular dysfunction and impaired wound healing after irradiation are presented as a major clinical challenge. Stem cells have been emerged as a promising therapeutic agent in regenerative medicine. However, they alone have no satisfactory effects on irradiated wound healing. In this context, we investigated therapeutic effect of umbilical cord blood (UCB)-derived mesenchymal stem cell (MSC) and platelet rich plasma (PRP) on dermal wound with impaired healing by irradiation.

Materials and Methods: PRP obtained from UCB was activated via newly developed method using bead mill homogenizer (Myung *et al*. 2017). Then, various factors in MSC and PRP were analyzed using cytokine array and real-time PCR. For assessing therapeutic effects of UCB-MSCs and PRP, gross examination, histologic analysis and quantification of expressed growth factors were performed in mouse model of radiation-impaired traumatic wound.

Results: PRP induced up-regulation in expression of angiogenic factors and gene of MSCs such as VEGF and endothelin-1. The result suggested that PRP improve the therapeutic effect of MSCs by enhancing angiogenesis in stem cell therapy. For confirming this, the wound closure, epidermal regeneration, angiogenesis and growth factors expression were evaluated in irradiated wound model. The results revealed that co-treatment of UCB-MSCs and PRP promoted regeneration of epidermis and granulation tissue. In addition, significantly improved angiogenesis was also identified by CD31 IHC and expression of growth factors.

Conclusions: PRP stimulates angiogenic paracrine release

of UCB-MSCs, and the combination therapy of UCB-MSCs and PRP improves regeneration efficacy of radiation-trauma complex injury via enhancing angiogenesis.

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4-Hydroxycinnamic acid protects mice from cigarette smoke-induced pulmonary inflammation via MAPK pathways

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Introduction: Cigarette smoke (CS) is the main etiological cause of chronic obstructive pulmonary disease, the prevalence of which has continuously increased in recent years. 4-Hydroxycinnamic acid (HA) is a plant phenolic acid that has anti-inflammatory activities. In this study, we explored the therapeutic effects of HA on airway inflammation caused by CS and lipopolysaccharide (LPS) in mice.

Materials and Methods: The animals received 1 h of CS exposure for 7 days and intranasal instillation of LPS on day 4. HA (10 and 20 mg/kg) was administered to animals via oral gavage 1 h before CS exposure.

Results: HA treatment significantly decreased the accumulation of inflammatory cells and production of cytokines, including tumornecrosis factor-α, interleukin (IL)-6, and IL-1β, caused by CS and LPS exposure. After histological examination, we observed that HA treatment significantly reduced the infiltration of inflammatory cells into lung tissue caused by CS and LPS exposure. Furthermore, HA-treated groups showed significantly decreased phosphorylation of extracellular signal-regulated kinase, c-Jun N-terminal kinase, p38, and nuclear factor-κB, and activity of cytochrome c oxidase subunit-2 caused by CS and LPS.

Conclusions: In conclusion, HA effectively suppresses the airway inflammatory response induced by CS and LPS exposure, and is closely associated with the downregulation of mitogen-activated protein kinases signaling.

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Effects of exhaustive swimming on magnesium redistribution and transporters

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Introduction: Magnesium (Mg) plays a central rolein energy production, neuronal activity, cardiacexcitability, neuromuscular transmission, muscular contraction,vasomotor tone, and blood pressure, all of which aresignificantly related to physical performance. Blood Mg levels have been shown to increase during exhaustiveswimming exercise. However, Mg redistribution and transporter expression duringthe exercise remains to be determined.

Materials and Methods: The maleSprague-Dawley rats (n=20, 220-250 g) were subjected to 30 min forced swimmingexercise until exhaustion. Afterswimming, inductively coupled plasma-mass spectrometry was applicable to the determinationof Mg in serum, red blood cell (RBC), quadriceps muscle, heart, kidney, liver,lung and brain. The levels of glycogen, adenosine triphosphate (ATP), hexokinase (HE), citrate synthase (CS), malondialdehyde (MDA) and superoxidedismutase (SOD) in quadriceps muscle were measured by spectrophotometry. Also,the Transient receptor potential melastatin-7 (TRPM7) channels and solutecarrier family 41 member A1 (SLC41A1) gene expressions were measured byreal-time PCR.

Results: After exercise, Mg in serum, heart, kidney, liver, lungand brain were increased, while Mg in RBC and quadriceps muscle were decreased. Also, HK, CS, MDA and SOD in quadriceps muscle wereincreased, but glycogen and ATP in the muscle were decreased. The geneexpression levels of TRPM, the Mg influx as aMg channel, were not changed but the levels of SLC42A1, the Mg efflux as a Na^+/Mg^{2+}exchanger, were upregulated.

Conclusions: These results suggested that the exhaustiveswimming exercise could produce Mgredistribution from quadriceps muscle to serum of circulatory system. Thedecreased Mg levels in quadricepsmuscle were related to the increased metabolic demands andthe stimulation of Mg efflux.

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Anti-fatigueactivities of Korean Red Ginseng and Antler Velvet response to exhaustiveswimming exercise

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