http://dx.doi.org/10.14400/JDC.2014.12.8.423

A review of the effects of environmental enrichment on stroke in animal experimental models

Kyoung-Hee Lee, Ki-Chul Hwang

Dept. of Occupational therapy, Baekseok University

뇌졸중 동물 실험 모델에서 환경 강화 효과에 대한 종설

이경희, 황기철

백석대학교 작업치료학과

Abstract The purpose of this article is to present the environmental enrichment(EE) method used to improve the functional recovery and change of brain plastic in animal experimental models of stroke.
In animal experimental models of stroke, the environmental enrichment is effective in altering the morphological, biochemical and behavioral characteristics of the brain and thereby improving the functional outcomes.
In this review article, we address the effects of EE in achieving a functional recovery in animal experimental models of stroke, thus attempting to describe them in patients with stroke from both occupational and rehabilitation perspectives.

Key Words : Stroke, Environmental enrichment, Behavioral, Animal experimental models, Rehabilitation therapy

요 약 본 연구의 목적은 뇌졸중 동물 실험 모델에서 환경 강화는 기능 회복을 향상 시키고 이로 인하여 뇌에 형태학적, 생화학과 행동에서 특징적인 변화와 그 효과들을 소개하고, 뇌졸중 환자에 환경 강화의 적용은 재활치료 및 작업치료를 포함한 다양 치료와 전 임상 실험의 중요성을 설명하고자 한다. 이 종설 논문에서는 주로 뇌졸중 동물 실험 모델에서 환경 강화로 인한 기능적 회복에 대한 효과와 신경 친화성 물질, 특정 단백질의 발현 및 임상 적용 사례 등의 연구 결과들을 소개하였다. 마지막으로 임상에서 뇌졸중 환자에게 환경 강화의 적용 가능성과 작업치료 및 재활치료의 관점에서 설명하였다.

주제어 : 뇌졸중, 환경 강화, 행동, 동물 실험 모델, 재활 치료

1. Introduction

Stroke is the third leading cause of death worldwide and it is also the major course of long-term disabilities in industrialized countries. Most of the surviving stroke patients achieve a substantial degree of spontaneous recovery, but they commonly have a persistent presence of sensorimotor and cognitive dysfunctions[1].

* 본 논문은 2014년도 백석대학교 대학연구비에 의하여 지원되었음
Received 20 June 2014, Revised 21 July 2014
Accepted 20 August 2014
Corresponding Author: Ki-Chul Hwang(Dept. of Occupational therapy, Baekseok University)
Email: otvision@bu.ac.kr

ISSN: 1738-1916

Ischemic stroke mainly occurs as a result of large-vessel atherosclerosis, embolisms, thrombosis and small-vessel occlusions. Some risk factors of stroke can be modified; these include high blood pressure, diabetes mellitus, smoking, alcohol abuse, diet or atrial fibrillation. But other risk factors, such as age, gender, race and genetic predisposition, cannot be modified[2].

A forced or robot-assisted training is effective in improving the degree of recovery of motor functions after stroke. To date, it has been proposed that the social and physical environment is associated with the improvement of recovery in patients with stroke[3-5].

Animal models of stroke are of great significance in not only examining the pathophysiologic mechanisms by which patients with stroke achieve a recovery of symptoms but also evaluating the efficacy of new therapeutic agents. Animal models of environmental enrichment (EE) are an experimental paradigm for conducting animal studies to examine the physical, social and intellectual activity and thereby to predict similar patterns in humans. Thus, experimental animals are subjected to physical exercise (e.g., wheel running) and exposure to a variety of passive visual stimuli (e.g., changes in the color of the background and object)[6].

In this article, we address the effects of EE in achieving a functional recovery in animal experimental models of stroke, thus attempting to describe them in patients with stroke from both occupational and rehabilitation therapy.

2. Environmental enrichment

The enriched environment(EE) is referred to as a group housing of healthy rats in a large cage with toys, for which changes are made daily in such a manner as to increase the neuronal plasticity and thereby to achieve a recovery of the experimental stroke in them[1]. In this experimental model, the training has a positive effect in achieving a functional recovery after brain damage. Such physical exercises as wheel running have been reported to stimulate the cell division as well as net neuronal survival in rats[7]. This has led to the speculation that the beneficial effects of EE might be totally or near totally based on the increased voluntary exercise. Thus, it can promote the activation, signaling and plasticity of neurons over the cerebral regions. This is followed by the activation of the somatosensory and visual cortex through the increased sensory stimulation due to the increased somatosensory and visual input[8].

It is known that intact rats become vulnerable to alterations in the morphological, biochemical and behavioral characteristics of the brain by exposure to EE. Moreover, the EE would be more effective on the behavior of rats; this would be notably effect on the learning and problem-solving activity. Furthermore, it has been known to be effective in improving the spatial memory and achieving a functional recovery[9-16]. With the accumulated experience of the EE, it has been shown to be effective in inducing numerous forms of brain plasticity and thereby improving the structure and function of brain[13,17]. Experimental studies have also shown that the EE caused substantial changes in the brain structure in rats; these changes include increases in the brain weight, the thickness of cerebral cortex, the size of neuronal body, the number and length of dendrites, dendritic arborization, the number and density of dendritic spines, the number of synapses, capillary diameter and the number of glial cells. Thus, the EE is mainly involved in the alteration of brain plasticity[6,18-22] .

Later studies have shown that the EE had more specific effects in altering various types of neurotransmitters; these include increases in cerebral levels of noradrenalin, the activity of acetylcholinesterase and levels of glutamate receptor subunits, GluR1, NR2A and NR2B, but not NR1, in the forebrain. In addition, the EE has also been reported to

be effective in inducing the up-regulation of neurotrophic factors, including GDNF, BDNF, NGF and NT-3, and altering the degree of the expression of various immediate early genes, including NGFI-A and Arc, in the hippocampus and cerebral cortex[1,23-29]. These reports indicate that the EE triggers the expression of many genes that are involved in the neuronal plasticity, thus having a great effect on the behavior of rats, including the learning and memory, in an experimental setting.

2.1 Environmental enrichment and stroke

To date, the effects of EE have been studied the most commonly using rats and mice. But its beneficial effects have been well documented in other mammalian species such as gerbils, ground squirrels, cats and monkeys. This has established the effects of EE in various experimental conditions. But there is a variability in the effects of EE depending on the species, age and gender[30-32]. Moreover, it has also been reported that the EE has positive effects on the recovery after various types of brain damage, including the hippocampal and cortical lesions[33].

2.2 Effect of environmental enrichment in animal models of stroke

Animal models of stroke have shown that the EE increased the neural stem/progenitor cell pool and stimulated the neurogenesis in the adult subventricular zone at 5 weeks after inducing the cortical stroke[34]. This is accompanied by the alterations in the density of dendritic spines in the pyramidal neurons that are located contralateral to the cortical infarct[35]. The aging process occurs in association with the temporary dysregulation of cellular response to ischemia and poor functional recovery. It has been shown that there was improvement in the motor functions with the continuous use of EE after the onset of ischemia although it increased the mortality. Moreover, after a 2-week exposure to the EE, followed by a 2-week group housing of mice in standard cages, there was no further improvement in the motor functions. By contrast, after a 4-week exposure to the EE, there was improvement in the motor functions accompanied by a better maintenance of the neurologic recovery. Animal studies have also shown that the degree of the expression of immediate-early genes, such as nerve growth factor-induced gene A, was decreased at 2-3 weeks after the experimental animals achieved a recovery following the EE. This suggests that such genes are involved in the recovery process. Moreover, experimental studies have also shown that the EE was effective in the functional outcomes after the onset of stroke in mice. Furthermore, further genomic studies in this series have been conducted using the experimental procedures that have been performed for the EE[1].

Following the onset of the permanent middle cerebral artery (MCA) occlusion using the EE in rats, there was a decrease in the degree of the expression of migration inhibitory factor (MIF) in the peri-infarct areas, which was accompanied by an increase in parvalbumin immunoreactive interneurons. This indicates not only that the MIF is part of a signaling network involved in the brain plasticity but also that it is also involved in the inhibition of the recovery of sensory and motor functions after the onset of stroke once its levels in the neurons or astrocytes are elevated. Thus, the MIF could contribute to promoting a recovery of the stroke as a potential therapeutic approach[28].

It has previously been suggested that the temporary modulation of the astrocytic proliferation, followed by the scar formation, might be effective in achieving a functional recovery after the onset of stroke in aged rats[33]. In addition, this is followed by the compensatory movement in the early phase of post-lesion period. Thus, the EE is involved in the functional improvement. The beneficial effects of EE are based on the increased motor activity that mainly

arise from the compensatory process[36]. It has also been reported that the EE is also effective in postoperatively improving the spatial memory deficits after the onset of other brain injuries such as traumatic brain injury and surgical hippocampal lesions. But it is less effective in improving the symptoms of focal cerebral ischemia[37,38].

The beneficial effects of EE in achieving a recovery of the symptoms after the onset of focal ischemia are not based on the decreased volume of infarct[35]. This indicates that the beneficial effects of EE are based on the increased plasticity of the cerebral areas that are adjacent to the lesions. In addition, it has also been reported that the EE is also effective in increasing the length and branching of dendrites in the contralateral cortex during the post-ischemic period when combined with the rehabilitative training[3,39]. Focal cortical ischemia is characterized by the increased neurogenesis in the ipsilateral dentate gyrus; this is comparable to the dentate gyrus of rats housed in standard cages, where there are no changes in the density of neurons, which can be explained by those in the morphology of neurons in an EE setting[20, 40].

To date, there are no extensive studies about the effects of EE on gene expression after the onset of focal ischemia. It has been proposed that there are same mechanisms by which the neuronal plasticity occurs in an EE setting in both healthy individuals and those with brain lesions. It is evident, however, that the ischemic events are mainly responsible for triggering the expression of genes that may be involved in the response to EE. This suggests that there is a variability in the expression of genes in an EE setting during the post-ischemic period. Thus, there may be delayed up-regulation of genes associated with the effects of EE in promoting the brain plasticity once down-regulated in response to the detrimental stimuli during the early phase after the onset of ischemia[1,32,41,42].

2.3 Effect of environmental enrichment in stroke patients

Clinical studies have shown that the cognitive cost of multitasking was decreased with the use of an adaptive version of NeuroRacer in a multitasking training mode in elderly people aged between 60 and 85 years as compared with both the active control group and the no-contact one. In addition, it has also been shown that the degree of efficacy of multitasking training was lower as compared with untrained 20-year-old subjects and it was sustained for six months. Furthermore, the multitasking training restored the neurological signs of cognitive control process due to aging, as demonstrated on electroencephalography. Thus, it was effective in increasing the sustained attention and working memory, accompanied by the increase in the midline frontal theta power, by which the effects of multitasking training in increasing the sustained attention are preserved six months later. This strongly indicates that the multitasking training is effective in promoting the plasticity of the prefrontal cognitive control system in the elderly, which has not been described in the literature according to our knowledge. From the same context, custom-designed video games are used to assess the degree of cognitive functions, to analyze the underlying mechanisms and to improve the cognitive functions in individuals at all ages[43].

3. Conclusions

As described here, the EE is effective in maintaining or increasing the independence and thereby improving the quality of life in individuals with neurological deficits with no respect to whether they present with brain damage or trauma. Such individuals deserve social, psychological and cognitive supports[44].

The EE may also be effective in improving the quality of life in patients with stroke, for which it is imperative that the patient-customized treatment

strategy be established from a rehabilitation perspective.

In animal models of stroke, the EE was effective in achieving a recovery of the sensorimotor functions after the onset of focal ischemia with no respect to the additional rehabilitation training[7,39,39,45].

The effects of rehabilitation training in achieving a functional recovery after the onset of brain injury are dependent on its timing and intensity. That is, earlier rehabilitation with or without higher intensity may lead to the aggravation of the brain damage [15]. The optimal timing of the EE combined with the rehabilitation training is 5 days after the onset of focal cerebral ischemia[39,46].

Thus, we propose that the EE combined with the rehabilitation training be performed in the field of occupational therapy and rehabilitation, both of which are based on the brain plasticity. This leads to the speculation that the external inputs are used to promote the cerebral reorganization. The treatment goals of occupational therapy are to restore the learning and working memory and to achieve a recovery of physical and cognitive functions. The rationale of rehabilitation therapy in the restoration of motor, sensory and cognitive functions is based on the efficient task-specific activities, for which the EE is effective in creating the optimal conditions for the successful outcomes of rehabilitation therapy.

Finally, there are several promising multi-modal approaches in the field of rehabilitation therapy, which are based on latest updates on neurophysiology and technological advances. Thus, considerable efforts have been made to restore the brain capacity and thereby to achieve a functional recovery in patients with stroke.

ACKNOWLEDGMENTS

This paper was supported by Baekseok University in 2014.

REFERENCES

[1] Nygren J, Wieloch T. Enriched environment enhances recovery of motor function after focal ischemia in mice, and downregulates the transcription factor NGFI-A. J Cereb Blood Flow Metab, Vol. 25, No. 12: 1625-1633, 2005.

[2] de Freitas GR, Bogousslavsky J. Primary stroke prevention. Eur J Neurol, Vol. 8, No. 1, pp. 1-15, 2001.

[3] Biernaskie J, Corbett D. Enriched rehabilitative training promotes improved forelimb motor function and enhanced dendritic growth after focal ischemic injury. J Neurosci, Vol. 21, No. 14, pp. 5272-5280, 2001.

[4] Calabro RS, Reitano S, Leo A, De Luca R, Melegari C, Bramanti P. Can robot-assisted movement training (Lokomat) improve functional recovery and psychological well-being in chronic stroke? Promising findings from a case study. Funct Neurol; Early view, Vol. 235 , No. 3, pp. 1-3, 2014.

[5] Zadravec M, Matjacic Z. Toward minimum effort reaching trajectories formation in robot-based rehabilitation after stroke: an innovative guidance scheme proposition. Int J Rehabil Res, Vol. 20, No. 5, pp. 523-542, 2014.

[6] Li L, Tang BL. Environmental enrichment and neurodegenerative diseases. Biochem Biophys Res Commun, Vol. 334, No. 2, pp. 293-7, 2005.

[7] Risedal A, Mattsson B, Dahlqvist P, Nordborg C, Olsson T, Johansson BB. Environmental influences on functional outcome after a cortical infarct in the rat. Brain Res Bull, Vol. 58, No. 3, pp. 315-321, 2002.

[8] Nithianantharajah J, Hannan AJ. Enriched environments, experience-dependent plasticity and disorders of the nervous system. Nat Rev Neurosci, Vol. 7, No. 9, pp. 697-709, 2006.

[9] Dahlqvist P, Ronnback A, Bergstrom SA, Soderstrom I, Olsson T. Environmental enrichment reverses learning impairment in the Morris water

maze after focal cerebral ischemia in rats. Eur J Neurosci, Vol. 19, No. 8, pp. 2288-2298, 2004.

[10] Duffy SN, Craddock KJ, Abel T, Nguyen PV. Environmental enrichment modifies the PKA-dependence of hippocampal LTP and improves hippocampus-dependent memory. Learn Mem, Vol. 8, No. 1, pp. 26-34, 2001.

[11] Bennett JC, McRae PA, Levy LJ, Frick KM. Long-term continuous, but not daily, environmental enrichment reduces spatial memory decline in aged male mice. Neurobiol Learn Mem, Vol. 85, No. 2, pp. 139-152, 2006.

[12] Frick KM, Fernandez SM. Enrichment enhances spatial memory and increases synaptophysin levels in aged female mice. Neurobiol Aging, Vol. 24, No. 4, pp. 615-626, 2003.

[13] Huang FL, Huang KP, Wu J, Boucheron C. Environmental enrichment enhances neurogranin expression and hippocampal learning and memory but fails to rescue the impairments of neurogranin null mutant mice. J Neurosci Vol. 26, No. 23, pp. 6230-6237, 2006.

[14] Tang AC. Neonatal exposure to novel environment enhances hippocampal-dependent memory function during infancy and adulthood. Learn Mem, Vol. 8, No. 5, pp. 257-264, 2001.

[15] Wagner AK, Kline AE, Sokoloski J, Zafonte RD, Capulong E, Dixon CE. Intervention with environmental enrichment after experimental brain trauma enhances cognitive recovery in male but not female rats. Neurosci Lett, Vol. 334, No. 3, pp. 165-168, 2002.

[16] Polito L, Chierchia A, Tunesi M, Kehoe PG, Albani D, Forloni G. Environmental Enrichment Lessens Cognitive Decline in APP23 Mice Without Affecting Brain Sirtuin Expression. J Alzheimers Dis, Vol. 254, No. 1, pp. 526-548, 2014.

[17] Hu YS, Xu P, Pigino G, Brady ST, Larson J, Lazarov O. Complex environment experience rescues impaired neurogenesis, enhances synaptic plasticity, and attenuates neuropathology in familial Alzheimer's disease-linked APPswe/PS1DeltaE9 mice. FASEB J, Vol. 24, No. 6, pp. 1667-1681, 2010.

[18] Baroncelli L, Braschi C, Spolidoro M, Begenisic T, Sale A, Maffei L. Nurturing brain plasticity: impact of environmental enrichment. Cell Death Differ, Vol. 17, No. 7, pp. 1092-1103, 2010.

[19] Lee MY, Yu JH, Kim JY, Seo JH, Park ES, Kim CH, Kim H, Cho SR. Alteration of synaptic activity-regulating genes underlying functional improvement by long-term exposure to an enriched environment in the adult brain. Neurorehabil Neural Repair, Vol. 27, No. 6, pp. 561-574, 2013.

[20] Lonetti G, Angelucci A, Morando L, Boggio EM, Giustetto M, Pizzorusso T. Early environmental enrichment moderates the behavioral and synaptic phenotype of MeCP2 null mice. Biol Psychiatry, Vol. 67, No. 7, pp. 657-665, 2010.

[21] Mainardi M, Landi S, Gianfranceschi L, Baldini S, De Pasquale R, Berardi N, Maffei L, Caleo M. Environmental enrichment potentiates thalamocortical transmission and plasticity in the adult rat visual cortex. J Neurosci Res, Vol. 88, No. 14, pp. 3048-3059, 2010.

[22] Zhang H, Cai R, Zhang J, Pan Y, Sun X. Environmental enrichment enhances directional selectivity of primary auditory cortical neurons in rats. Neurosci Lett, Vol. 463, No. 2, pp. 162-165, 2009.

[23] Zai L, Ferrari C, Dice C, Subbaiah S, Havton LA, Coppola G, Geschwind D, Irwin N, Huebner E, Strittmatter SM, Benowitz LI. Inosine augments the effects of a Nogo receptor blocker and of environmental enrichment to restore skilled forelimb use after stroke. J Neurosci, Vol. 31, No. 16, pp. 5977-5988, 2011.

[24] Ronnback A, Dahlqvist P, Svensson PA, Jernas M, Carlsson B, Carlsson LM, Olsson T. Gene expression profiling of the rat hippocampus one month after focal cerebral ischemia followed by enriched environment. Neurosci Lett, Vol. 385, No. 2, pp. 173-178, 2005.

[25] Sun H, Zhang J, Zhang L, Liu H, Zhu H, Yang Y. Environmental enrichment influences BDNF and NR1 levels in the hippocampus and restores cognitive impairment in chronic cerebral hypoperfused rats. Curr Neurovasc Res, Vol. 7, No. 4, pp. 268-280, 2010.

[26] Rossi C, Angelucci A, Costantin L, Braschi C, Mazzantini M, Babbini F, Fabbri ME, Tessarollo L, Maffei L, Berardi N, Caleo M. Brain-derived neurotrophic factor (BDNF) is required for the enhancement of hippocampal neurogenesis following environmental enrichment. Eur J Neurosci, Vol. 24, No. 7, pp. 1850-1856, 2006.

[27] Pham TM, Winblad B, Granholm AC, Mohammed AH. Environmental influences on brain neurotrophins in rats. Pharmacol Biochem Behav, Vol. 73, No. 1, pp. 167-175, 2002.

[28] Inacio AR, Ruscher K, Wieloch T. Enriched environment downregulates macrophage migration inhibitory factor and increases parvalbumin in the brain following experimental stroke. Neurobiol Dis, Vol. 41, No. 2, pp. 270-278, 2011.

[29] Pinaud R. Experience-dependent immediate early gene expression in the adult central nervous system: evidence from enriched-environment studies. Int J Neurosci, Vol. 114, No. 3, pp. 321-33, 2004.

[30] Rosenzweig MR. Effects of differential experience on the brain and behavior. Dev Neuropsychol, Vol. 24, No. 2-3, pp. 523-540, 2003.

[31] Rosenzweig MR, Bennett EL, Diamond MC. Effects of differential environments on brain anatomy and brain chemistry. Proc Annu Meet Am Psychopathol Assoc, Vol. 56, pp. 45-56, 1967.

[32] Rosenzweig MR, Bennett EL. Psychobiology of plasticity: effects of training and experience on brain and behavior. Behav Brain Res, Vol. 78, No. 1, pp. 57-65, 1996.

[33] Buchhold B, Mogoanta L, Suofu Y, Hamm A, Walker L, Kessler C, Popa-Wagner A. Environmental enrichment improves functional and neuropathological indices following stroke in young and aged rats. Restor Neurol Neurosci, Vol. 25, No. 5-6, pp. 467-484, 2007.

[34] Komitova M, Mattsson B, Johansson BB, Eriksson PS. Enriched environment increases neural stem/progenitor cell proliferation and neurogenesis in the subventricular zone of stroke-lesioned adult rats. Stroke, Vol. 36, No. 6, pp. 1278-1282, 2005.

[35] Johansson BB, Belichenko PV. Neuronal plasticity and dendritic spines: effect of environmental enrichment on intact and postischemic rat brain. J Cereb Blood Flow Metab, Vol. 22, No. 1, pp. 89-96, 2002.

[36] Knieling M, Metz GA, Antonow-Schlorke I, Witte OW. Enriched environment promotes efficiency of compensatory movements after cerebral ischemia in rats. Neuroscience, Vol. 163, No. 3, pp. 759-769, 2009.

[37] Passineau MJ, Green EJ, Dietrich WD. Therapeutic effects of environmental enrichment on cognitive function and tissue integrity following severe traumatic brain injury in rats. Exp Neurol, Vol. 168, No. 2, pp. 373-384, 2001.

[38] Puurunen K, Jolkkonen J, Sirvio J, Haapalinna A, Sivenius J. Selegiline combined with enriched-environment housing attenuates spatial learning deficits following focal cerebral ischemia in rats. Exp Neurol, Vol. 167, No. 2, pp. 348-355, 2001.

[39] Biernaskie J, Chernenko G, Corbett D. Efficacy of rehabilitative experience declines with time after focal ischemic brain injury. J Neurosci, Vol. 24, No. 5, pp. 1245-1254, 2004.

[40] Komitova M, Perfilieva E, Mattsson B, Eriksson PS, Johansson BB. Effects of cortical ischemia and postischemic environmental enrichment on hippocampal cell genesis and differentiation in the adult rat. J Cereb Blood Flow Metab, Vol. 22, No. 7, pp. 852-860, 2002.

[41] Diamond MC, Law F, Rhodes H, Lindner B, Rosenzweig MR, Krech D, Bennett EL. Increases in cortical depth and glia numbers in rats subjected to

enriched environment. J Comp Neurol, Vol. 128, No. 1, pp. 117-26, 1966.

[42] Zhao LR, Risedal A, Wojcik A, Hejzlar J, Johansson BB, Kokaia Z. Enriched environment influences brain-derived neurotrophic factor levels in rat forebrain after focal stroke. Neurosci Lett, Vol. 305, No. 3, pp. 169-172. 2001.

[43] Anguera JA, Boccanfuso J, Rintoul JL, Al-Hashimi O, Faraji F, Janowich J, Kong E, Larraburo Y, Rolle C, Johnston E, Gazzaley A. Video game training enhances cognitive control in older adults. Nature, Vol. 501, No. 7465, pp. 97-101, 2013.

[44] Kuipers P, Foster M, Smith S, Fleming J. Using ICF-Environment factors to enhance the continuum of outpatient ABI rehabilitation: an exploratory study. Disabil Rehabil, Vol. 31, No. 2, pp. 144-151, 2009.

[45] Risedal A, Zeng J, Johansson BB. Early training may exacerbate brain damage after focal brain ischemia in the rat. J Cereb Blood Flow Metab, Vol. 19, No. 9, pp. 997-1003, 1999.

[46] Lee KH, Kim JH, Choi DH, Lee J. Effect of task-specific training on functional recovery and corticospinal tract plasticity after stroke. Restor Neurol Neurosci, Vol. 31, No. 6, pp. 773-785, 2013.

이 경 희(Lee, Kyoung-Hee)

- 2005년 2월 : 포항대학교 작업치료과(체육학사)
- 2008년 8월 : 대구대학교 재활과학 대학원(이학석사)
- 2013년 8월 : 건국대학교 일반대학원 의학과(의학박사)
- 2013년 3월 : ~ 현재 : 백석대학교 작업치료학과 교수
- 관심분야 : 작업치료학, 재활의학, 신경과학
- E-Mail : otprin38@bu.ac.kr

황 기 철(Hwang, Gi-Chul)

- 2001년 2월 : 연세대학교 작업치료학과(보건학사)
- 2007년 8월 : 대구대학교 일반대학원 재활과학과(이학박사)
- 2012년 3월 ~ 현재 : 백석대학교 작업치료학과 교수
- 관심분야 : 재활과학, 작업치료학
- E-Mail : otvision@bu.ac.kr