Effects of Selective Agonism of Angiotensin II AT1 and AT2 Receptors on Neural Differentiation and Proliferation in Human Neural Stem Cells

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Major components of the brain Renin-Angiotensin System (RAS) may provide new modes of therapy for impaired neurons. AngiotensinII via its receptors, AT1 and AT2, induces proliferation and differentiation of human neural stem cells (hNSC). Therefore, Selective agonism of AT1 and AT2 receptors could possibly induce proliferation and differentiation in damaged areas of the brain. Post-stroke, traumatic brain injury, and several neurodegenerative diseases, such as Alzheimer's and Parkinson's disease, are associated with the synaptic dysfunctions of actions mediated by the AT1 and AT2 receptor. Therefore, using Human Neural Stem Cells (hNSC), this study aims to enhance our understanding of how neural differentiation and proliferation are affected by selective agonism of both the AT1 and AT2 receptor in proliferating and differentiating conditions. It was hypothesized that stimulating the AT1 receptor would induce proliferation and stimulating the AT2 receptor would induce differentiation upon addition of selective agonists. The results supported the hypothesis with a 79% increase in proliferation conditions and a 58% increase in differentiation conditions with AT2 agonism, and a 5% increase in proliferation with AT1 agonism. These results indicate the attainability of the original aim. If the AT1 receptor could induce proliferation of damaged hNSC, and the AT2 receptor could induce differentiation of these hNSC rendering them functional, therapeutic treatments for non-proliferating and non-functioning neural stem cells associated with neurodegenerative diseases or traumatic brain injuries to proliferate and function again could become a reality.

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