ABSTRACT

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Stroke is one of the leading causes of death worldwide and its effective protective treatment is still missing.
Nicotinic acid has been a widely used substance to modify lipid profiles for many years and it successfully prevents clinical cardiovascular diseases. Recently explored receptor, GPR109A, causes its effect and it was found to be expressed in spleen, adipose, and immune cells including macrophages. The aim of this work is to investigate the effect of nicotinic acid on the infarct size during cerebral ischemia and to determine the role of macrophages in its effect.
We compared two groups in our experiments, transgenic CD11bDTR mice and control wild type (WT) mice. Both groups were treated with diphtheria toxin (DT), then a middle cerebral artery occlusion was performed and afterwards mice were treated with either nicotinic acid or vehicle. We have found depletion of macrophages in CD11bDTR group induced by DT treatment and depletion of macrophages in WT group treated with nicotinic acid. We also found significant difference in infarct volume in the wild type mice that are treated with nicotinic acid than their corresponding control.
According to our results, nicotinic acid seems to have a protective effect on cerebral ischemia.