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enes are located at sites of translocation breakpoints or deletions. These features led to this idea that they could be important factors in the development or progression of the tumors. In this study, we want to clarify cAMP-Epac signaling pathway effects on miR-21 expression in Glioblastoma cells. In this study, U87MG glioblastoma cell line were cultured in DMEM supplemented with $10 \%$ FBS. After $60-70 \%$ confluency, cells were treated by some related pharmalogical agents and 24 h later, miRNAs were extracted. miRNAs level quantified by real time-PCR. The results indicate that this signaling pathway can change aberrantly expressed mir-21 level in this glioblastoma cell line
Keywords: Glioblastoma, cAMP

## Regeneration of transected rat sciatic nerve using in vitro transdifferentiated BMSCs


#### Abstract

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Introduction: Peripheral nerve injury is a common lesion in clinical practice and cytotherapy is one of the approaches for treating this lesion. In this investigation, bone marrow stromal cells (BMSC) derive Schwann cells were tested as alternative source for cell therapy.

Methods: BMSC were collected from the long bones of rats and were in vitro transdifferentiated into Schwann-like cells. The preinducers were $\beta$-mercaptoethanol and retinoic acid, while the inducers were the basic-fibroblast growth actor, the platelet derive growth factor, forskelin and progesterone. The transdifferentiated Schwann like cells were rransplanted in a rat model of the sciatic nerve injury with 1 cm gaps. A sciatic function index was used in order to aluate the finctional outcome, while histological, immunohistochemical and ultrastructural studies were used for evaluating the structural improvement in the regenerating nerve.

Results: The results showed that there were functional and structural improvements in the experimental group compared with the untreated controls.

Conclusion: The conclusion of the study is that progesterone induced BMSCs into Schwann cells can be a feasible


 option for cell transplantation for restoring structural and functional activities in peripheral nerve injury.Keywords: Regeneration, BMSCs, Schwann cells, Transdifferentiation, Cell therapy

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The Effect of Intrathecal Administration of Ascorbic Acid on Thermal Pain Sensation

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Introduction: Ascorbic acid (AA) is a water-soluble antioxidant vitamin with acidic pH , which is highly concentrated in the brain. . It is considered that AA is not only a simple antioxidant but also a neuromodulator in the central nervous system. Our hypothesis was the decreasing effect for AA on thermal pain sensation regardless of its acidity; therefore we investigated it in this research.
Methods: Male Wistar rats (200-250g) were used. Groups included control, sham-saline, AA (OSVAH pharma Co. Tehran-Iran) $2 \mathrm{mg} / \mathrm{ml}$ and neutralized AA $2 \mathrm{mg} / \mathrm{ml}$ (all drugs were injected at $10 \mu \mathrm{l}$ volume intrathecaly).Thermal nociception threshold was measured prior and 5 minutes after treatments, using Tail Flick test.
Results: AA showed increasing effect on thermal pain sensation in $2 \mathrm{mg} / \mathrm{ml}$ ( $p<0.001$ ) interestingly and neutralized $\mathrm{AA} 2 \mathrm{mg} / \mathrm{ml}$ reduced thermal pain compared to $\mathrm{AA} 2 \mathrm{mg} / \mathrm{ml}$ ( $\mathrm{p}<0.05$ ) dose.

Conclusion: In spite of the hypothesis, AA increase thermal pain sensation in Tail Flick test. However neutralized AA can decrease thermal pain in comparison with AA. Probably acidic PH has role in pain sensation. Presumably AA has involved in pain transferring facilitation, by A $\delta$ fibers which have roles in thermal pain reflexes, or in facilitating Glutamate effects by non NMDA receptors such as AMPA; which needs more research

Key words: Ascorbic acid, thermal pain, intrathecal injection, Tail Flick test


# The Effect of Intrathecal Administration of Ascorbic Acid on Chemical Pain Sensation During Formalin Test in Rat 

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Introduction: Ascorbic acid (AA) is an essential micronutrient with acidic pH , it can block a variety of membrane bound receptor proteins including the NMDA site receptors which is important in pain signaling. The hypothesis was a decreasing effect of AA on chemical pain sensation regardless of its acidity; therefore we investigated it in this research.
Methods: Male Wistar rats ( $200-250 \mathrm{~g}$ ) were used. Groups included control, sham-saline, AA $2 \mathrm{mg} / 10 \mu \mathrm{l}$ and neutralized AA $2 \mathrm{mg} / 10 \mu \mathrm{l}$ (all drugs were injected intrathecaly). 5 minutes after treatment, 0.05 ml formalin $2.5 \%$ was injected in left paw and in one hour the pain responses of rats were recorded.

Results: AA has reducing effects on first, interphase and second stages of formalin test, in $2 \mathrm{mg} / 10 \mu \mathrm{l}$ dose ( $\mathrm{p}<0.001$ ) and neutralized AA $2 \mathrm{mg} / 10 \mu \mathrm{l}$ dose ( $\mathrm{p}<0.001$ ).

Conclusion: Effects of AA on chemical neurogenic and inflammatory pain is significant. Although AA has acidic pH regarding to the results it is reasonable to conclude, its effect is not due to its acidic property. Presumably AA with blockage of NMDA receptors, decreases the pain, which needs to clarify more.

Key words: Ascorbic acid, pain, intrathecal injection, formalin test.

