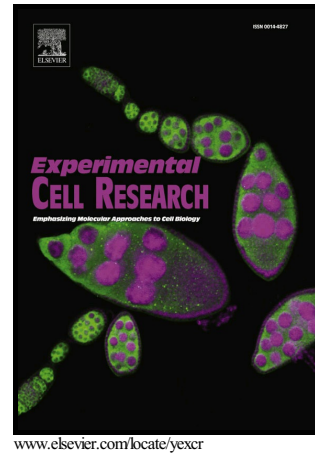


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Calcium influx differentially regulates migration velocity and directedness in response to electric field application

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Calcium influx differentially regulates migration velocity and directedness in response to electric field application.

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Abstract

Neural precursor cells (NPCs) respond to externally applied direct current electrical fields (DCEFs) by undergoing rapid and directed migration toward the cathode in a process known as galvanotaxis. It is unknown if the underlying mechanisms of galvanotactic migration is common to non-electrosensitive cells and if so, how NPCs and other galvanotactic cells sense and transduce electrical fields into cellular motility. In this study, we show that distinct aspects of NPC galvanotactic migration: motility (quantified through |velocity|) and directedness, are differentially regulated by calcium. We use low-Ca²⁺ culture conditions; an intracellular Ca²⁺ chelator; and voltage gated calcium channel (VGCC) inhibitors to specific channels expressed on NPCs, to demonstrate the role of Ca²⁺ influx in DCEF-induced NPC migration. Consistent with existing literature, we show Ca²⁺ is involved in F-actin polymerization that lengthens NPC membrane protrusions necessary for cellular motility. However, inhibiting Ca²⁺ results in reduced velocity but has no effect on DCEF-induced directedness. This dissociation between velocity and directedness reveal that these migration parameters can be independently regulated, thus suggesting a parallel process of sensing DCEFs by NPCs.

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