Methods and Results:
sites may have a role in adrenergic stimulation of the CMs, whether direct interaction is needed for SNs directly interact with cardiomyocytes (CMs). Although it has been proposed that these contact domains exist (neuro-cardiac junction, NCJ). In addition, silencing of NGF expression by CMs in co-cultures led to 66% decrease of neuronal density, supporting that NGF released by CMs sustains SN viability.

We tested whether SN/CM interactions are required for NGF-mediated pro-survival signalling to the neuron and correct myocardial innervation. Cultured neurons in contact with CMs showed fast TrkA activation, NGF uptake, bigger synaptic boutons and survived NGF withdrawal, whereas CM-conditioned medium did not sustain neuronal viability because of the very low NGF concentration (16.1 pg/mL). Altogether, these results support that the NCJ is essential for intercellular neurotrophin signalling. Consistently, NGF concentration at the contact site was estimated by using the TrkA inhibitor K252a and resulted about 1000-fold higher (1.75 ng/mL) when compared to that in CM conditioned medium.

Dystrophin accumulation on CM membrane contacted by SNs was observed in mouse cardiac slices, neuron and correct myocardial innervation. Cultured neurons in contact with CMs showed fast TrkA domains exist (neuro-cardiac junction, NCJ). In addition, silencing of NGF expression by CMs in co-cultures to be immune cell regulated - is unknown.

Methods and Results: Electron microscopy and immunofluorescence on mouse heart slices and SN/CM co-cultures showed close association between neurons and CMs and enrichment of the NGF receptor (TrkA) at the contact site, suggesting that specialized and locally organized signalling is required for efficient neurotrophin signalling and correct innervation pattern is not known.


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