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Data Article

Data on calcium increases depending on stretch in dystrophic cardiomyocytes

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ABSTRACT

In this data article, intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$) was measured in isolated ventricular Wild Type (WT) and *mdx* cardiomyocytes in two different conditions: at rest and during the application of an axial stretch. Using a carbon microfibers technique, axial stretch was applied to mimic effects of physiological conditions of ventricular filling. A study of cation entry with the same experimental model and the manganese quenching method reported (i) a constitutive cation entry in *mdx* cardiomyocytes and (ii) the involvement of TRPV2 channels in axial-stretch dependant cation entry, "Axial stretch-dependent cation entry in dystrophic cardiomyopathy: involvement of several TRPs channels" (Aguettaz et al., 2016) [1].

Here, the Ca^{2+} dye fluo-8 was used for $[Ca^{2+}]_i$ measurement, in both resting and stretching conditions, using a perfusion protocol starting initially with a calcium free Tyrode solution followed by the perfusion of 1.8 mM Ca^{2+} Tyrode solution. The variation of $[Ca^{2+}]_i$ was found higher in *mdx* cardiomyocytes.

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Specifications Table

Subject area	*Biology*
More specific subject area	*Calcium regulation in cardiomyopathy*
Type of data	*Figures*
How data was acquired	*Confocal microscopy, BIORAD 1024*
Data format	*Raw, analysed*
Experimental factors	*Isolated mouse cardiomyocytes were axial stretched*
Experimental features	*Intracellular calcium changes were recorded*
Data source location	*Poitiers, France*
Data accessibility	*Data is within this article*

Value of the data

- Protocol used in these experiments could activate Store-operated calcium channels (SOCs) and stretch-activated calcium channels (SACs).
- This protocol can be used to investigate stretch-dependent calcium increases in cardiomyocytes and other cells.
- The effects of SOCs and SACs inhibitors and activators may help to understand stretch-dependent calcium increases.

1. Data

These data mainly focus on describing intracellular calcium concentration ($[Ca^{2+}]_i$) measured in Wild Type (WT) and *mdx* cardiomyocytes in two different conditions: at rest and during the application of an axial stretch. Calcium measurement was performed using the Ca^{2+} dye fluo-8 and during a perfusion protocol starting initially with a calcium free Tyrode solution followed by the perfusion of 1.8 mM Ca^{2+} Tyrode solution. In these conditions, $[Ca^{2+}]_i$ kinetics are described (Fig. 1A) and amplitudes are compared in the presence of SACs inhibitors (Fig. 1B). The effect of TRPV2 inhibitors (Fig. 2) and Probenecid (Fig. 3) is also described.

2. Experimental design, materials and methods

2.1. Cells isolation, mechanical stimulation and Mn^{2+}-quenching experiments are described in [1]

2.1.1. Solutions and chemicals

Before experiments, cells were stored in culture medium containing Dulbecco's modified Eagle's medium (DMEM – Lonza: 12-604F), complemented with 10 μg/mL insulin, 10 μg/mL gentamycin, 4 mM $NaHCO_3$, 10 mM Hepes, 0.2% BSA and 12.5 μM blebbistatin (all from Sigma). During experiments, cardiomyocytes were superfused with Tyrode solution containing 140 mM NaCl, 5.4 mM KCl, 1.8 mM $CaCl_2$, 1.8 mM $MgCl_2$, 10 mM Hepes and 11 mM glucose or with calcium-free Tyrode solution containing 140 mM NaCl, 5.4 mM KCl, 1.8 mM $MgCl_2$, 10 mM Hepes, 11 mM glucose, 1 g/L BSA and 20 mM Taurine.

Tranilast (Trn) was purchased from Calbiochem (53902-12-8), GsMTx-4 from Abcam (ab141871), probenecid (Prb, P8761), Streptomycin sulfate (Strp, s9137) and 4-methyl-4′-[3.5-bis(trifluoromethyl)-1H-pyrazol-1-yl]-1.2.3-thiadiazole-5-carboxanilide (YM-58483, y4895) from Sigma, ryanodine from Merck (15662-33-6), fura-2-AM (108964-32-5) and fluo-8-AM (sc-362561) from Santa-Cruz.

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