**Supplemental data 1**

**Effect of aldosterone on the amount of $Ca_v3$. mRNA**. Representative ethidium bromide-stained gel of RT-PCR products. Left, $Ca_v3.2$ primers have been described elsewhere.[1] Right, primers used for cyclophilin (Cyc) and the internal *cacna1g* ($Ca_v3.1$) primer have been described elsewhere.[1,2]

For *cacna1g* 3'end analysis, the primers used were forward (nuc 6673) AAGTCTCCAAGCACATCCGCC and two reverse primers : either T1 (nuc 7214) ACCAGAGAGACTCAGCGTGTC (GENBANK AF027984), that corresponding to the primer (forward) used in[3] or T2 (nuc 7279) TGGAGAAAGGTGATGGGGGAG, described elsewhere.[4]

In aldosterone-treated cells compared to control, $Ca_v3.1$ mRNA level increased by factors of 2.4 (n=3, $P$<0.05) and $Ca_v3.2$ mRNA level increased by factors of 1.4 (n=3, $P$<0.05). This finding contrasts with recent data from Lalevée *et al.,*[3] who found that aldosterone -induced $I_{CaT}$ was associated with an increase in the amount of $Ca_v3.2$ mRNA (up to 410%) and a non significant increase in $Ca_v3.1$ mRNA levels. There may be two reasons for this discrepancy. Firstly, Lalevée *et al.* used cyclophilin gene expression to normalize the amount of channel mRNA. However, we have previously shown that cyclophilin gene expression is strongly repressed in a rat model mimicking renin-independent hyperaldosteronism.[2] We have confirmed that cyclophilin is also downregulated in aldosterone - treated cultures of neonatal cardiomyocytes (decrease of 42 %) and this is probably the reason for the larger increase in $Ca_v3.2$ mRNA levels in the study by Lalevee *et al*.. Secondly, Lalevée *et al.* used $Ca_v3.1$ mRNA primers binding to the 3' end of the $Ca_v3.1$ cDNA. However, this region is known to generate splice variants.[4-6] Indeed, we not found significant different between control and aldosterone conditions using T1 primer (see figure). Consistently to $Ca_v3.1$ primers, T2 primer, located 65 bp dowtream from T1, revealed an increase in mRNA in aldosterone treated cell vs control (n=3, $P$<0.05). Then, T1 and T2 revealed two $Ca_v3.1$ variants that were differently regulated by aldosterone.

1. Ferron, L., Capuano, V., Deroubaix, E., Coulombe, A., and Renaud, J. F. (2002) Functional and molecular characterization of a T-type Ca(2+) channel during fetal and postnatal rat heart development. *J Mol Cell Cardiol* **34**, 533-546

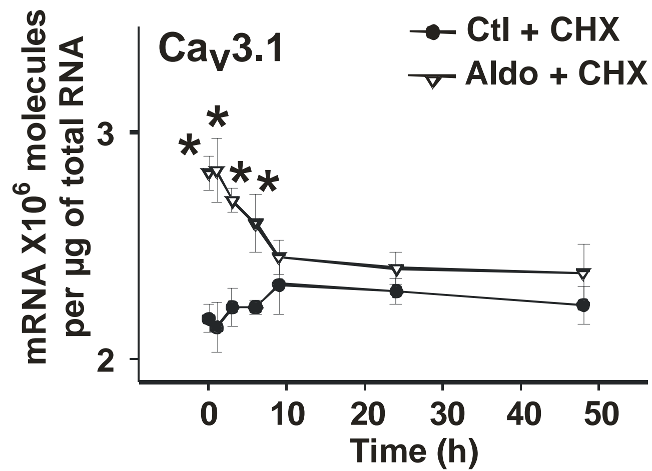
2. Capuano, V., Ruchon, Y., Antoine, S., Sant, M. C., and Renaud, J. F. (2002) Ventricular hypertrophy induced by mineralocorticoid treatment or aortic stenosis differentially regulates the expression of cardiac K+ channels in the rat. *Mol Cell Biochem* **237**, 1-10

3. Lalevee, N., Rebsamen, M. C., Barrere-Lemaire, S., Perrier, E., Nargeot, J., Benitah, J. P *et al*. (2005) Aldosterone increases T-type calcium channel expression and in vitro beating frequency in neonatal rat cardiomyocytes. *Cardiovasc Res* **67**, 216-224

4. Emerick, M. C., Stein, R., Kunze, R., McNulty, M. M., Regan, M. R., Hanck, D. A *et al*. (2006) Profiling the array of Ca(v)3.1 variants from the human T-type calcium channel gene CACNA1G: Alternative structures, developmental expression, and biophysical variations. *Proteins* **64**, 320-342

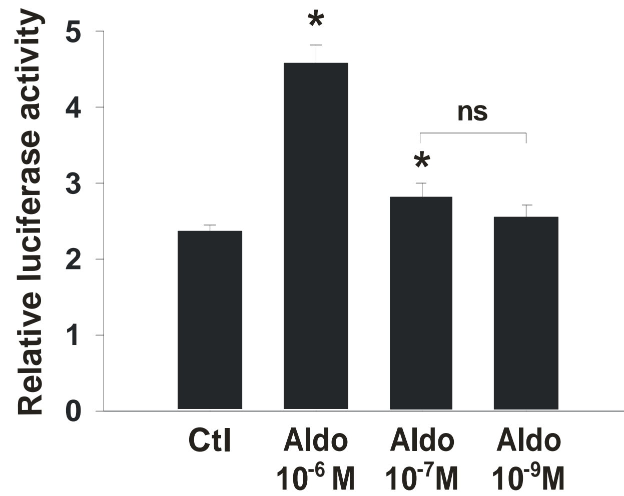
5. Mittman, S., Guo, J., and Agnew, W. S. (1999) Structure and alternative splicing of the gene encoding alpha1G, a human brain T calcium channel alpha1 subunit. *Neurosci Lett* **274**, 143-146

6. Ernst, W. L., and Noebels, J. L. (2009) Expanded alternative splice isoform profiling of the mouse Cav3.1/alpha1G T-type calcium channel. *BMC Mol Biol* **10**, 53



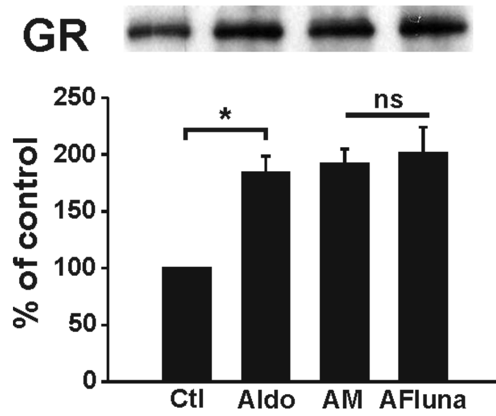

**Supplemental data 2**

**Decrease in Ca$_v$3.1 mRNA levels over time after cycloheximide treatment.** Twenty four hours after plating, cells are treated with or without aldosterone in presence of 10 µM cycloheximide (CHX). The amount of Ca$_v$3.1 mRNA is quantified by RT-PCR at 0, 1, 3, 6, 9, 24 and 48 hours after the beginning of treatment. *$P$<0.05 *vs* control at corresponding timing, n=4 for each point.



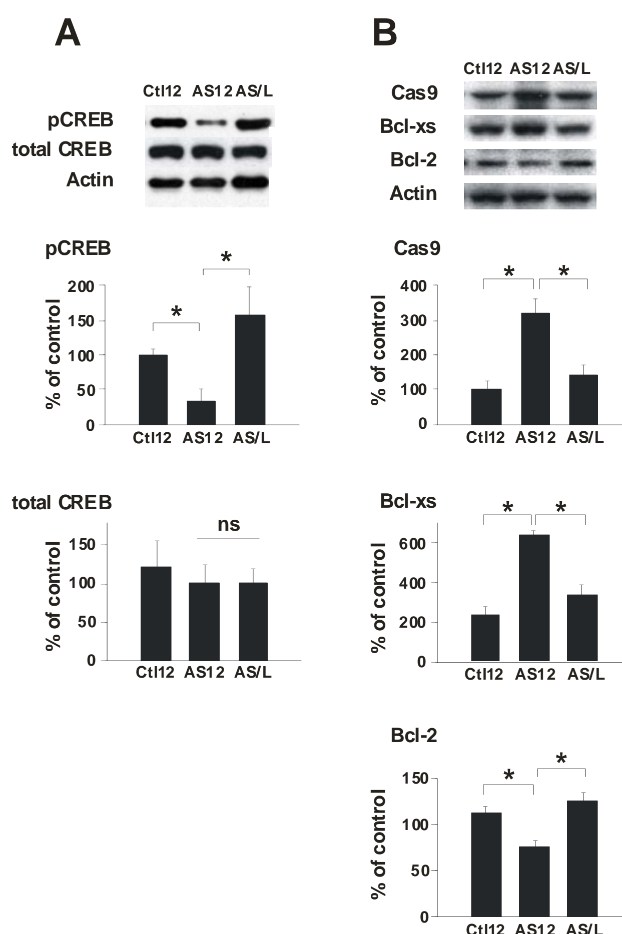

**Supplemental data 3**

**Dose-dependent effect of aldosterone on *cacna1g* promoter activity**. Cells were transfected with pGL4400 and treated with aldosterone for 24 hours. n=4 experiments performed in triplicate, *$P<0.05$ *vs* control.




**Supplemental data 4**

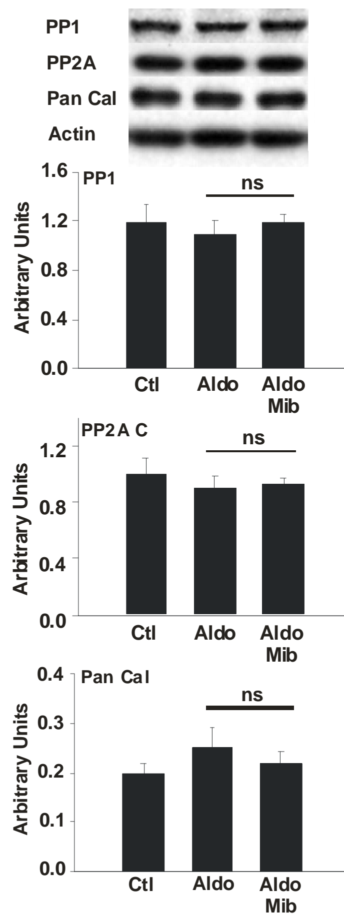
**$I_{CaT}$ blockade does not alter the nuclear translocation of GRs**. Western blots were performed with GR antibodies from nuclear extracts of cells treated for 30 min with 1 µM aldosterone plus 1 µM mibefradil (AM) or 10 µM flunarizine (AFluna). n=3, **P*<0.05, ns vs aldosterone.




**Supplemental data 5**

**Inverse pattern of expression between $I_{CaT}$ and pCREB and apoptotic markers in adult hypertrophied cardiomyocytes.** Stenosed rat model was detailed elsewhere (1). Briefly, total proteins were extracted from the left ventricles of rats subjected to 12 weeks of stenosis (AS12, n=6) paired with sham-operated rats (Ctl12, n=5). AS12 rats were treated with losartan (AS/L, n=5), an angiotensin II receptor (AT1) antagonist, for 2 weeks (12 mg/kg per day) *via* Alzet miniosmotic pumps inserted into the intraperitoneal cavity. From this model, we have previously described that $I_{CaT}$ is re-expressed in AS12 and $I_{CaT}$ density was reduced by losartan treatment in AS/L (1). Western blots were performed with total CREB and pCREB antibodies [A] or with pro apoptotic markers Cas 9 and Bcl-$x_s$ or with anti apoptotic marker Bcl-2 [B]. CREB activation that was reduced in AS12 (high $I_{CaT}$ density), re-increased in condition in which $I_{CaT}$ was reduced (AS/L). The increases of Cas 9 and Bcl-Xs and the decrease in Bcl-2 observed in AS12 simultaneously to $I_{CaT}$ re emergence, was reverse in AS/L. ns *vs* Ctl, **P*<0.05.

1. Ferron, L., Capuano, V., Ruchon, Y., Deroubaix, E., Coulombe, A., and Renaud, J. F. (2003) Angiotensin II signaling pathways mediate expression of cardiac T-type calcium channels. *Circ Res* **93**, 1241-1248




**Supplemental data 6**

**Aldosterone-induced $I_{CaT}$ did not modify the amounts of catalytic subunits of phosphatase.**
Western blots were performed with antibodies against the catalytic subunit of PP1, PP2A (PP2AC) and PP2B (Pan-calcineurin, Pan Cal) from total protein extracts. Cells were treated with 1µM aldosterone (Aldo, n=5) plus 1µM mibefradil (AM, n=5). No significant difference was found between control (n=4) and Aldo or Aldo mib.