# SUPPLEMENTAL MATERIAL

**Table S1. Detailed information of human heart sample donors.**

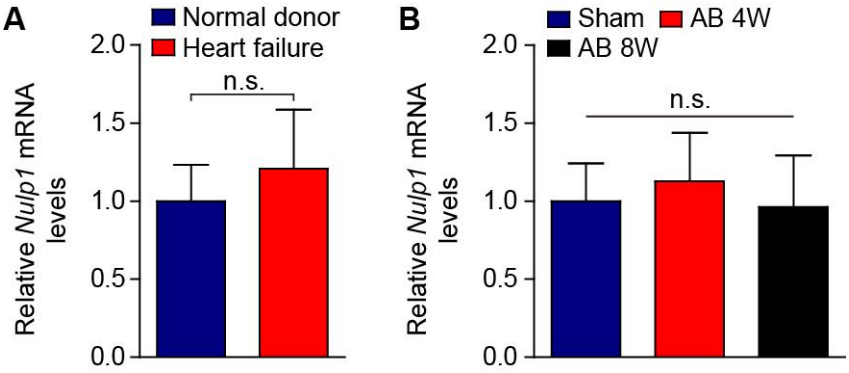
| Number | Group | Sex | Age (years) | Diagnosis | LVEDd (mm) | LVEF (%) |
|---|---|---|---|---|---|---|
| 1 | ND | Female | 44 | / | NA | NA |
| 2 | ND | Male | 55 | / | NA | NA |
| 3 | ND | Male | 52 | / | NA | NA |
| 4 | ND | Male | 39 | / | NA | NA |
| 5 | HF | Male | 30 | DCM | 73 | 25 |
| 6 | HF | Male | 63 | DCM | NA | NA |
| 7 | HF | Male | 57 | DCM | 66 | 34 |
| 8 | HF | Male | 63 | DCM | 72 | 28 |
| 9 | HF | Female | 46 | DCM | NA | 26 |
| 10 | HF | Male | 44 | DCM | 71 | 12 |
| 11 | HF | Female | 58 | DCM | 80 | 21 |
| 12 | HF | Male | 57 | DCM | 65 | 39 |

ND, normal donor; HF, heart failure; DCM, dilated cardiomyopathy; NA, not available

**Table S2. List of primers used for quantitative real-time PCR.**

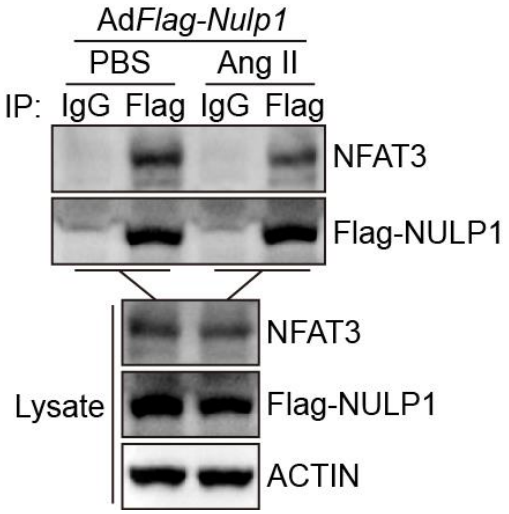
| Gene | Forward Primer | Reverse Primer |
|---|---|---|
| *Anp* Rat | AAAGCAAACTGAGGGCTCTGCTCG | TTCGGTACCGGAAGCTGTTGCA |
| *Myh7* Rat | CAAGGCTAACCTGGAGAAGATG | TCTGGACAGCTCCCCATTCT |
| *Gapdh* Rat | GACATGCCGCCTGGAGAAAC | AGCCCAGGATGCCCTTTAGT |
| *Anp* Mouse | ACCACCTGGAGGAGAAGA | TTCAAGAGGGCAGATCTATC |
| *Bnp* Mouse | GAGGTCACTCCTATCCTCTGG | GCCATTTCCTCCGACTTTTCTC |
| *Myh7* Mouse | TGTCCAGCAGGTGTCATACG | TTGCATTGATGCGTGTCACC |
| *Collagen I* Mouse | TGGTACATCAGCCCGAAC | GTCAGCTGGATAGCGACA |
| *Collagen III* Mouse | CCCAACCCAGAGATCCCATT | GAAGCACAGGAGCAGGTGTAGA |
| *Ctgf* Mouse | AGAACTGTGTACGGAGCGTG | GTGCACCATCTTTGGCAGTG |
| *Gapdh* Mouse | ACTCCACTCACGGCAAATTC | TCTCCATGGTGGTGAAGACA |

**Figure S1. mRNA levels of *Nulp1* were not changed in response to hypertrophic stimuli.**



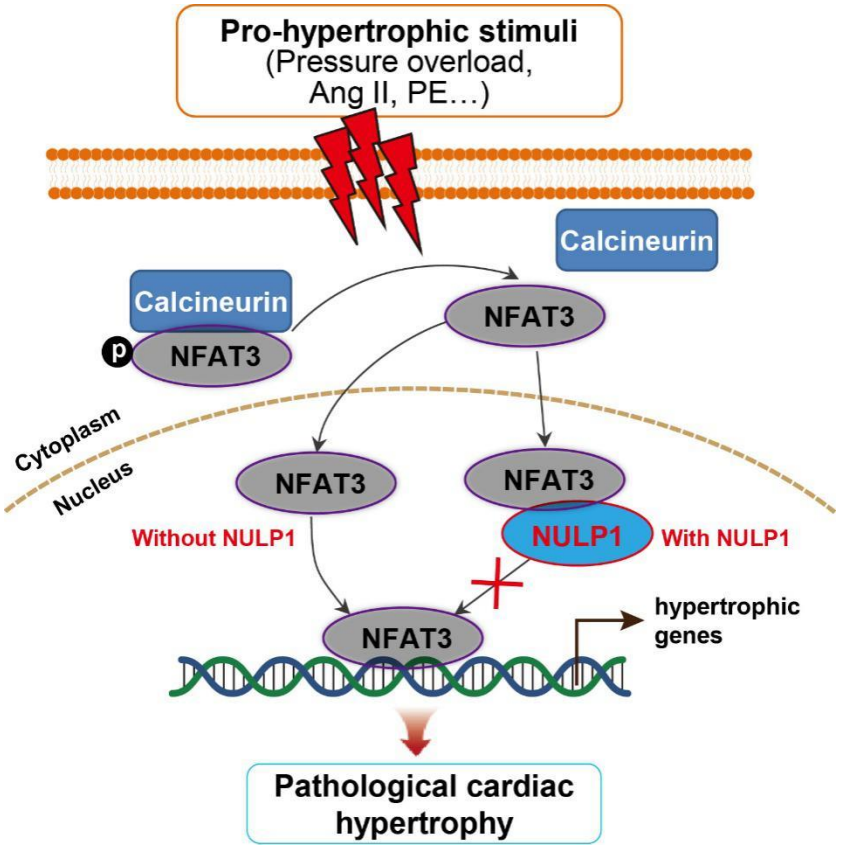

**A.** qPCR analyses of the mRNA levels of *Nulp1* in human heart samples from normal control donors (NF, n=4) and patients with heart failure (HF, n=4). **B.** Relative mRNA levels of *Nulp1* in mouse heart samples subjected to sham or aortic banding (AB) surgery for 4/8 weeks. n=4 mice per group. For all statistical plots, the data are presented as the mean ± s.d.; in **A**, the statistical analysis was performed using a Student's *t*-test; in **B**, the statistical analysis was performed using a one-way ANOVA or or two-tailed Student's t-test. n.s. indicates no significance.

**Figure S2. NULP1 interacts with endogenous NFAT3 in neonatal rat ventricular myocytes (NRVMs).**




Flag-tagged NULP1 was overexpressed in NRVMs by adenovirus infection, followed by PBS (phosphate buffer saline) or Ang II (angiotensin II )challenge for 6h. Western blots were then performed with NFAT3 or Flag antibody after co-immunoprecipitation (CO-IP) from whole-cell lysate using Flag or IgG antibody.

**Figure S3. Schematic image for the regulatory mechanism of NULP1 on pathological cardiac hypertrophy.**




Upon pro-hypertrophic stimuli, calcineurin dephosphorylates NFAT3, which then translocates into the nucleus and activates downstream hypertrophic genes. NULP1 predominantly located in nucleus, can directly interact with NFAT3, suppress its translational activity, and alleviate pathological cardiac hypertrophy.