Supplemental Methods:

Hemodynamic and Blood Pressure measurements

For hemodynamics measurements, adult mice were anesthetized with ketamine/xylazine and cardiac contractile and relaxation parameters were assessed, as previously described (Arber et al., 1997). Blood pressure measurements were performed in trained awake mice using the tail-cuff method as previously described (Chen et al., 2008).

***In vitro* cardiac muscle model of load-induced hypertrophy**

Right ventricular papillary muscles were isolated and mounted in a cardiac tissue culture chamber containing oxygenated modified Hepes buffered solution containing (in mM): 137.2 NaCl, 15.0 KCl, 1.2 $MgCl_2$, 2.8 Na-Acetate, 10 Taurine, 1.0 $CaCl_2$, 10.0 mM Glucose, and 10 mM Hepes in equilibrium with 100% O_2. Calcium concentration was increased in the solution to 1.80mM, and subsequently replaced with a modified M199 cell culture media containing (in mM): 2.0 L-carnitine, 5.0 creatine, 5.0 taurine, 2.0 L-glutamine, 0.2% albumin, 100 IU/ml penicillin, 0.1 mg/ml streptomyocin, and 10mM Hepes in equilibrium with 5% CO_2 and 95% O_2, at 34^{O}C and pH ~7.4. Muscles were initially left at slack length and stimulated at 0.2 Hz at a voltage 15% above threshold for a period of one hour. When active forces stabilized, the stimulation frequency was increased to 1 Hz. Systolic and diastolic forces were measured to monitor muscle viability for the experimental duration. To obtain stress-strain data, titanium dioxide markers were arrayed on the surface of muscles. During continuous stretch of the muscle, local uniaxial muscle deformations were recorded using a CCD camera, and correlated with the acquired force data, as previously described (Knoll et al., 2002; Lorenzen-Schmidt et al., 2005).

Supplemental References:

1. Arber, S., et al. 1997. MLP-deficient mice exhibit a disruption of cardiac cytoarchitectural organization, dilated cardiomyopathy, and heart failure. *Cell* **88**: 393-403.
2. Chen, Y., et al. 2008. Common genetic variants in the chromogranin A promoter alter autonomic activity and blood pressure. *Kidney Int.* **74:**115-125.

3. Knoll, R., et al. 2002. The cardiac mechanical stretch sensor machinery involves a Z disc complex that is defective in a subset of human dilated cardiomyopathy. *Cell* **111**: 943-955.
4. Lorenzen-Schmidt, I., McCulloch, A.D. and Omens, J.H. 2005. Deficiency of actinin-associated LIM protein alters regional right ventricular function and hypertrophic remodeling. *Ann. Biomed. Eng.* **33:** 888-896.

	WT (n=3)	**KO** (n=5)
Systolic BP (mmHg)	130 ± 4.1	134 ± 8.8
Diastolic BP (mmHg)	97 ± 4.0	107 ± 8.1
Pulse rate (bpm)	664 ± 32	672 ± 21

Supplemental Table 1. Blood pressure measurement assessment in wild type and *Fhl1* deficient mice. Blood pressure (BP) and heart rate were determined in trained awake mice using the tail-cuff method as previously described (Chen et al., 2008). No significant differences in systolic BP, diastolic BP and pulse rate were observed between wild type (WT) and *Fhl1*$^{-/-}$ (KO) mice.

	WT SHAM n=4	**WT TAC-5w** n=7	**KO SHAM** n=4	**KO TAC-5w** n=8
LV +dP/dt (mmHg/s)	9285 ± 1068	6592 ± 821*	9569 ± 317	8534 ± 1152
LV -dP/dt (mmHg/s)	8371 ± 1313	8413 ± 1002	7865 ± 395	10885 ± 914*
EXP τ	11.0 ± 0.4	10.7 ± 1.3	11.1 ± 0.8	8.1 ± 0.8*
LVEDP (mmHg)	11.1 ± 1.3	16.7 ± 2.5*	11.7 ± 2.3	11.4 ± 2.0
PG (mmHg)		95.3 ± 40		98.2 ± 33
LV/BW ratio (mg/g)	0.37 ± 0.0001	0.53 ± 0.0002*	0.37 ± 0.0003	0.42 ± 0.0003

Supplemental Table 2. Hemodynamic measurement of contractile parameters within wild type (WT) and *Fhl1* deficient (KO) mouse hearts after 5 weeks of TAC. LV dP/dt, left ventricular maximum (+dP/dt) and minimum (-dP/dt) first derivatives of LV pressure; LVEDP, left ventricular end diastolic pressure; τ, time constant of relaxation (using an exponential function), PG, trans-aortic pressure gradient; LV/BW ratio, left ventricular to body weight ratio. * $p<0.05$.

	Primer sequences and Template
FHL1 (4.5 LIM)	F (EcoRI): ATGTCGGAGAAGTTCGACT R (BamHI): TTACAGCTTTTTGGCACAGTCAG Template RefSeq acc. no.: NM_010211
FHL1 (3.5 LIM)	F (EcoRI): ATGTCGGAGAAGTTCGACT R2 (BamHI): AGTGATGGGGTTCTTGCATCCA Template RefSeq acc. no.: NM_010211
FHL1 (2 LIM)	F (EcoRI): GCTACTCGGGAGGACTCCC R (BamHI): AGTGATGGGGTTCTTGCATCCA Template RefSeq acc. nos.: NM_010211 and NM_001077361
FHL1 (1.5 LIM)	F (EcoRI): ATGTCGGAGAAGTTCGACT R (BamHI): GCCCTTGTACTCCACGTT Template RefSeq acc. nos.: NM_010211 and NM_001077361
FHL1 (KyoT2)	F (EcoRI): ATGTCGGAGAAGTTCGACT R (BamHI): TCACGGAGCATTTTTTGCAGTGGAAG Template RefSeq acc. no.: NM_001077361
Raf1	F (EcoRI): ATGGAGCACATACAGGGA R (SacI): CTAGAAGACTGGTAGCCTTGG Template RefSeq acc. no.: NM_029780.3
MEK1	F (BamHI): ATGCCCAAGAAGAAGCCGACG R (SacI): TCAGATGCTGGCAGCGTGGGT Template RefSeq acc. no.: NM_008927.3
MEK2	F (BamHI): ATGCTGGCCCGGAGGAAGCCG R (SacI): TCACACTGCAGTCCGCGTGGG Template RefSeq acc. no.: NM_023138.3
ERK2	F (BamHI): ATGGCGGCGGCGGCGGCGGCG R (XhoI): CTGTGATGGAGATCCAAGAAT Template RefSeq acc. nos.: NM_011949.3 and NM_001038663.1
ERK2 (TYDD)	GATCACACAGGGTTCTTGgacGAGgACGTAGCCACACGTTGGTACAG (lower case letters denote the mutations) Template: pACT2-ERK2

Supplemental Table 3. Primer sequences and reference templates used to generate FHL1, Raf1, MEK1/2 and ERK2 hybrid genes for yeast-two hybrid assays. Primer sequences are shown from 5'->3' and designed as follows. Forward (F) primers were designed to incorporate two random nucleotides, a linker restriction site and one random nucleotide to be in frame with vector, as well as the Kozak sequence: gccacc and a START codon (if not present within cDNA). Reverse (R) primers were designed to incorporate two random nucleotides, a linker restriction site and STOP codon if not present within cDNA.