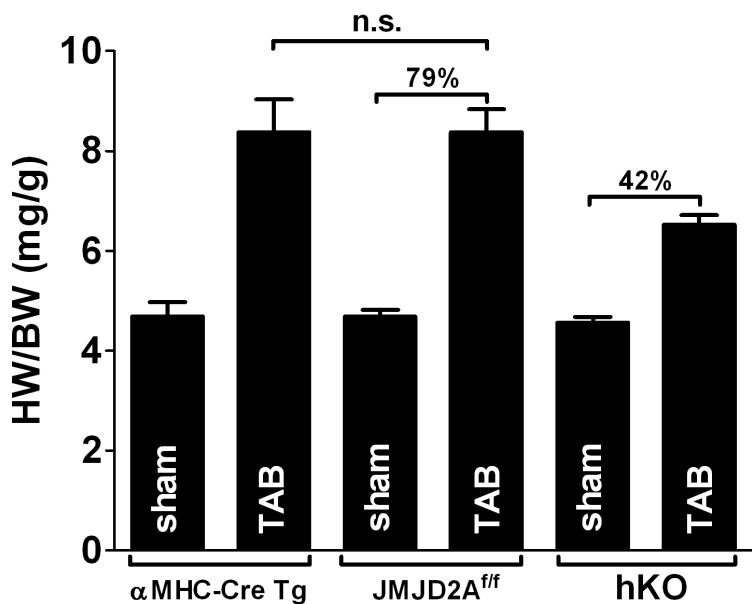
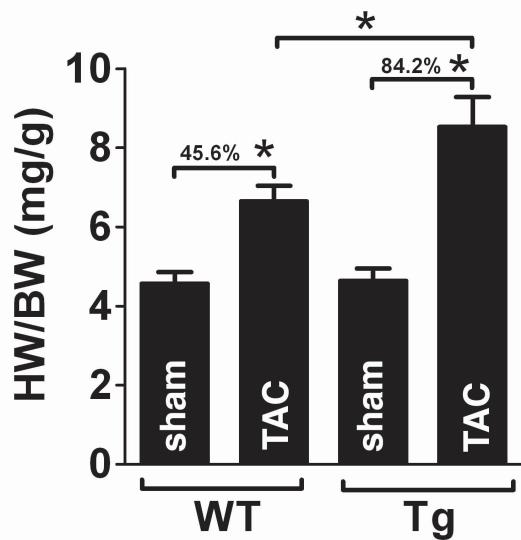


Supplemental Figure 1.

Tissue distribution of JMJD2A in mice. Various tissue homogenates from wild-type mice were subjected to Western blot analysis using anti-JMJD2A antibody. GAPDH was used as loading control. A higher molecular weight band of JMJD2A was detected in the skeletal muscle lysate. It remains to be determined whether this corresponds to the alternative variant form of JMJD2A (NM_172382) that has an extra 100 bp in the 3'-end of the coding sequence of JMJD2A.

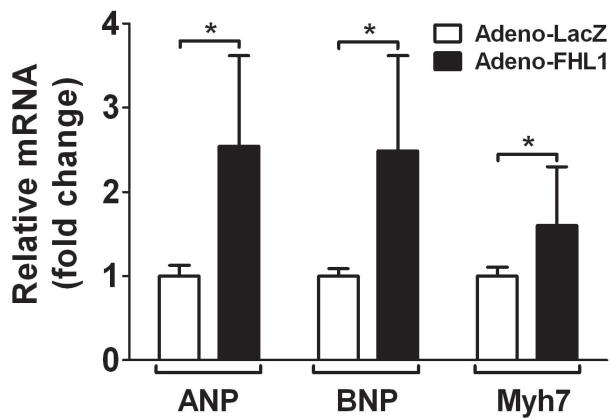


Supplemental Figure 2. Heart weight (HW) to body weight (BW) ratio of JMJD2A^{+/+} x α MHC-Cre transgenic (n=4), JMJD2A^{f/f}, and hKO (n=10-14) mice three weeks after TAC surgery.



Supplemental Figure 3.

JMJD2A promotes hypertrophy in response to pressure overload induced by TAC. Heart weight (HW)-to-body weight (BW) ratio (HW/BW) of wild type (WT) and JMJD2A-Tg line A (Tg) mouse hearts after three weeks of Sham and TAC operation. Values are means + SEM; n=5/group. *, p< 0.05.



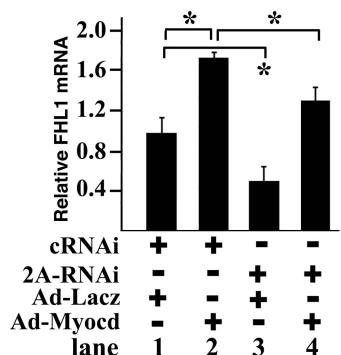
Supplemental Figure 4.

FHL1 promotes “fetal gene” expression in neonatal cardiomyocytes. Rat neonatal ventricular cardiomyocytes were transduced with adenoviruses expressing either LacZ or FHL1. 48 hrs after transduction, cells were harvested and relative mRNA of ANP, BNP, and •MHC were quantified with real-time qRT-PCR. The transcripts were normalized to internal GAPDH and expressed relative to those of control LacZ-transduced cells. N=3+SD. *, p<0.05.

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GTTGAGGGAAGACAGGTCTGGGTGCCGCTCCTGAACTTGGCCTTGAGCC**ATGG**CTCTCAAAGACACTCAG

Supplemental Figure 5.

5'-upstream sequence of the FHL1 promoter. A perfect consensus CArG box was highlighted in underline and bold. The primer pair used in the ChIP assay was highlighted in bold, underline, and italic. The translation start codon ATG in the first exon (bold, upper-case letter) was highlighted in bold.



Supplemental Figure 6

Down-regulation of JMJD2A attenuates expression of FHL1 in cardiomyocytes. Neonatal rat ventricular myocytes were transfected with non-specific control siRNA (cRNAi), or JMJD2A specific siRNAs (2A-RNAi). Twenty-four hours after transfection, cells were transduced with adenoviruses expressing β-galactosidase (ad-LacZ) or myocardin (ad-Myocd) at MOI of 50. Relative transcript levels of FHL1 were determined 48 hrs after transduction using qRT-PCR. Myocardin activated transcription of FHL1 and knockdown of JMJD2A attenuated this activation. N=3+SEM. *, p<0.05.

Table 1.

Primers/oligos	sequence (5' to 3')
Genotyping of knockout mice	p1: GGACTAGGTAGTCATGTTGG p2: AAGCAACGGAAGCAGAAAGTG
Genotyping of transgenic mice	p1: AGTGGTGGTAGGAAAGT
mouse ANP qPCR	p2: TCAATGTAGGCAATGTATCT
mouse BNP qPCR	p1: CAACACAGATCTGATGGATTCA p2: CCTCATCTTCTACCGGCATC
mouse Myh7 qPCR	p1: GTCAGTCGTTGGGCTGTAAC p2: AGACCCAGGCAGAGTCAGAA
mouse FHL1 qPCR	p1: CGCATCAAGGAGCTCACC p2: CTGCAGCCGCAGTAGGTT
mouse FHL2 qPCR	p1: GGCTTCTCAAAGACACTCAGG p2: TCGAACTTCTCCGACATGGT
mouse GAPDH qPCR	p1: AGAAAACCATCATGCCAGGT p2: ACAGGTGAAGCAGGTCTCGT
rat JMJD2A qPCR	p1: GGCACAGTCAAGGCTGAGAATG p2: ATGGTGGTGAAGACGCCAGTA
rat FHL1 qPCR	p1: AGCAGGAATCGAGCCTGAG p2: CTTCCATTCTTGGGAGGAAC
rat ANP qPCR	p1: GGCTTCTCAAAGACACTCAGG p2: GTCGAACCTCTCAGACATGGT
rat BNP qPCR	p1: CACAGATCTGATGGATTCAAGA p2: CCTCATCTTCTACCGGCATC
rat Myh7 qPCR	p1: GTCACTCGCTGGGCTGT p2: CAGAGCTGGGGAAAGAAGAG
rat GAPDH qPCR	p1: GAGGAGAGGGCGGACATT p2: ACTCTCATTCAAGGCCCTG
human BNP qPCR	p1: ATCACCATCTCCAGGAGCGA p2: AGCCTTCTCCATGGTGGTGAA
human JMJD2A qPCR	p1: ACTGGAAACGTCCGGGTTA p2: GGCTCCAGGGATGTCTGCT
mouse FHL1 ChIP	p1: AGAGTTCCGCAAGATAGCCAATA p2: GAGGGTACCATTCACATCTGCAC
mouse GAPDH ChIP	p1: GTGGGACATCTGCTCTGGT p2: CCAGACCTGTCTCCCTCAA
JMJD2A cloning	p1: CCAATGTGTCCATCGTGGATCT p2: GTTGAAGTCGCAGGAGACAACC
FHL1 promoter cloning	p1: AAAAAATCGATGCTCTGAGTCTGAAACTCTG p2: AAAAAGCGGCCGCCTACTCCATGATGGCCCGG
FHL1 promoter mutation	p1: AAAAAGGTACCATGGGACTGTGTTTGGAGG p2: AAAAACCTGAGCCACTCTGGCTTAGGCAG
Gel shift probes	p1: TTCTCCCTGGATCATATAAAATATTTAAGAATTCTAG p2: CTAGAAAATTCTAAATATTTATATGATCCAAGGGAGAA
Gel shift probes-Mutant	Sense GGATCACCTAAAATGGTAAAGAA Antisense GGTTCTAACCATTTAGGTGAT Sense GGATCATATAAAATATTTAAGAA Antisense GGTTCTAAATATTTATATGAT