Supplementary Figure Legends

Figure S1

Administration of TSA after TAC surgery attenuated the expression of hypertrophy associated gene expression in mice LV. *p < 0.002; **p < 0.01. n = 5

Figure S2

H3K9m3 histone enrichment at the intergenic bdP was examined from left ventricles of Sham, TAC and TSA by ChIP and assessed by qPCR.

Figure S3

Expression of Ezh2 and Hdac2 mRNA in Sham, TAC and TSA animals as determined by qRT-PCR.

Figure S4

RNA-ChIP using histone H3 antibody in left ventricles of control (Sham) animals show specific enrichment for AS β -MHC transcript in chromatin purified RNA samples.

Figure S5

shRNA-mediated silencing of Ezh2 enzyme is confirmed by immunoblot. nt=non-target control, Ezh2KD=Ezh2 knockdown cells.

Figure S6

HDAC inhibition by TSA is associated with increased H3K9/14ac enrichment at the intergenic bdP in Ezh2-KD cells. *p < 0.01. n = 4

Figure S7

TSA stimulation in Ezh2-KD cells increased H3K4m3 methylation at the intergenic bdP. *p < 0.01; *p < 0.02. n = 4

Figure S8

Loss of AS β -MHC transcript did not alter the chromatin binding of Ezh2 at the intergenic bdP. *p < 0.007. n = 3

Figure S9

Enrichment of H3K27m3 marks at the intergenic bdP remains unaffected in AS β -MHC deficient cells.

Figure S10

The stable chromatin association of *pri-miR-208b* transcript in *AS* β -*MHC* deficient cells was reduced when stimulated with TSA

Figure S11

Nuclear snoRNA is nearly depleted in cytosolic fraction serving as control. *p < 0.0007. n = 3

Figure S12

18S rRNA expression is predominant to cytoplasmic fraction isolated from Sca1+ cells. p < 0.0007. n = 3

Figure S13

Immunoblotting for nuclear Brm, Mecp2 proteins and cytosolic Gapdh serves as controls for nuclear and cytosolic fractionation

Figure S14

Gene expression profiles of Sca1+ vascular progenitor cells compared to mouse neonatal ventricular cardiomyocytes (NVCM) after TSA treatment. Relative mRNA levels quantified by qRT-PCR. *p < 0.03. n = 3

Oligonucleotide Sequences Amplimer sets used for mRNA

Amplir	mer sets used for mRNA gene expression		
Anp	ACAGCCAAGGAGGAAAAGGC	GAPDH	TGAAGCAGGCATCTGAGGG
	CCACAGTGGCAATGTGACCA		CGAAGGTGGAAGAGTGGGAG
Bnp	TCCAGAGCAATTCAAGATGCA	18S rRNA	TCGGAACTGAGGCCATGATT
	CTTTTGTGAGGCCTTGGTCC		CTTTCGCTCTGGTCCGTCTT
Sercal	2a CCCCCTGGGAGAATATCTGG	Med13	ATCCATCAAGTGCCTGCTTC
	GATCTGGAAAATGAGCGGCA		GTGCGGACTGAGGATCAACT
EZH2	CTAATTGGTACTTACTACGATAACTTT	Oct4	CTCCCGAGGAGTCCCAGGACAT
	ACTCTAAACTCATACACCTGTCTACAT		GATGGTGGTCTGGCTGAACACCT
α -MHC	C CCACCTGGGCAAGTCTAACAA	Sca1	TGCAGAAAGAGCTCAGGGACTGG
	TGTAGTCCACGGTGCCAGC		TCCATCAGGGTAGGGGCAGGT
β-ΜΗΟ	C GATGTTTTTGTGCCCGATGA	Sox2	AAGGAGAGAAGTTTGGAGCC
	ACCGTCTTGCCATTCTCCG		TCTGGCGGAGAATAGTTGG
Tgfb3	CCCAACCCCAGCTCCAAGCG	Six	TTAAGAACCGGAGGCAAAGA
	CCTCAACAGCCACTCGCGCA		GGGGGTGAGAACTCCTCTTC
Spp1	GCCTGTTTGGCATTGCCTCCTC		
	CACAGCATTCTGTGGCGCAAGG		
Sln	GAGGTGGAGAGACTGAGGTCCTTGG		
	GAAGCTCGGGGCACACAGCAG		
Nanog	CAAGGGTCTGCTACTGAGATGCTCTG		
-	TTTTGTTTGGGACTGGTAGAAGAATCA	G	

Amplimer sets used for ChIP Primer A (-3.3 kb α-MHC)

Primer A (-3.3 kb α -MHC)	CAAGAGAAAGCAGACAACAG
	CGGACTCACTCACTCTTTTT
Primer B (-2.7 kb α -MHC)	AGGGAGGATCACACTGGATG
	TGAGGCTCTACCACCAGTCC
<i>Primer C</i> (-2.2 kb α -MHC)	ATGGTCCTTCTCACCTGTGG
	GGTTTGCCCTCTTCTTCCTT
<i>Primer D</i> (-1.7 kb α -MHC)	GAGCCTCAAGTGACCTCCAG
	CTCCAAGGGACCTGATTCAA
<i>Primer E</i> (-1.2 kb α -MHC)	TCAGTCTGCAGAGCCCCTAT
	GGCTGAGGGAGAAAGGGTAT
<i>Primer F</i> (-0.8 kb α -MHC)	GCTGTGCAGCTGTTCAGTTC
	CAGGCCATCATCCAATCTCT
<i>Primer G</i> (-0.3 kb α -MHC)	TATTAAGCCTGGAAGAGAAG
	GCAGATAGAGGAGAGACAGG
<i>Primer H</i> (+0.7 kb α -MHC)	CAATCTTCCAGTGAGCCA CA
	CTGGACGGAGAGAGGAACAG
Antisense 3' end 1 (AS 3' end 1)	GCAACCACAATGGACTTTCC
	ACGATGGCGATGTTCTCTTT
Antisense 3' end 2 (AS 3' end 2)	GCATGCATTGGTTCAGAATG
	AGCCGCAGTAGGTTCTTCCT
Anp (+41 to +142)	GTGGGCAGAGACAGCAAACA
	AAGCCAAAAGGCCAAGACG
<i>Bnp</i> (-6 to +155)	AGCTCAGCCGGCAGGAAT
	CGTGTTCTCCCTTGTCTCGC

Supplementary Figures S1-S8

Figure S1



Figure S5



Figure S2



Figure S3



Figure S4



Figure S6







Figure S8



Supplementary Figures S9-S14





Figure S14

