Figure S1. Cardiac hypertrophy was induced by IS *in vitro*. Rat embryonic cardiomyoblast H9c2 cells were treated with (A) increasing concentrations of IS for 2 days or with (B) 500  $\mu$ M IS for indicated the periods. Total RNA was detected by reverse transcription-quantitative PCR to analyze the gene expression of cardiomyocyte hypertrophy markers, ANF, BNP and  $\beta$ -MHC. \*P<0.05, \*\*P<0.01, \*\*\*P<0.001 vs. control. IS, indoxyl sulfate; ANF, atrial natriuretic factor; BNP, brain natriuretic peptide;  $\beta$ -MHC,  $\beta$ -myosin heavy chain; ns, not significant.



Figure S2. Gene expression of AhR induced by IS. Rat embryonic cardiomyoblast H9c2 cells were treated with CKD serum for 2 days. Total protein samples were isolated for (A) western blotting with an antibody against AhR and (B) subsequent densitometry. (C) Total RNA was isolated for reverse transcription-quantitative PCR to analyze the mRNA level of AhR. AhR, aryl hydrocarbon receptor; IS, indoxyl sulfate; ns, not significant; CKD, chronic kidney disease.



Target name	Target sequence	
CYP1B1	5' GGAUGUGCCUGCCACUAUUTT	
	5' AAUAGUGGCAGGCACAUCCTT	
AhR	5' GCAACAAAGGAUCGGGAUATT	
	5' UAUCCCGAUCCUUUGUUGCTT	
NC SiRNA	5' UUCUCCGAACGUGUCACGUTT	
	5' ACGUGACACGUUCGGAGAATT	

Table SI. Sequences of siRNA against CYP1B1, AhR and negative control (NC).

Table SII. Primers for realtime PCR.

Target name	Sequence	Products (bp)
CYP1B1	F: ATGTGCCTGCCACTATTACAGA	187
	R: GGTATGGTAAGTTGGGTTGGTC	
AhR	F: AGAAAGGGAAAGACGGAGCG	232
	R: GCGGCGTGGATAAACTGATA	
ANF	F: ATGGGCTCCTTCTCCATCAC	204
	R: TTCATCGGTCTGCTCGCTCA	
BNF	F: TGGGAAGTCCTAGCCAGTCT	236
	R: GATCCGGTCTATCTTCTGCC	
β-ΜΗC	F: CCCTACGATTATGCGTTCTTCTCC	198
	R: CTGCTCCTCCCTCTGCTTCTGTT	
GAPDH mRNA	AAAGTGGAGATTGTTGCCATCA	106
	CCTTGACTGTGCCGTTGAATTT	

Table SIII. Primers for ChIP analysis.

Target name	Sequence	Products (bp)
CYP1B1 P1	F: CAGCGCCCAGGGAGATGACT	124
	R: GCTCTGTACGCCAGCAAACG	
CYP1B1 I2	F: TCAGAAATTGTAATTCGGTCAC R: AAGAGGACGGGAGGGATGAG	147