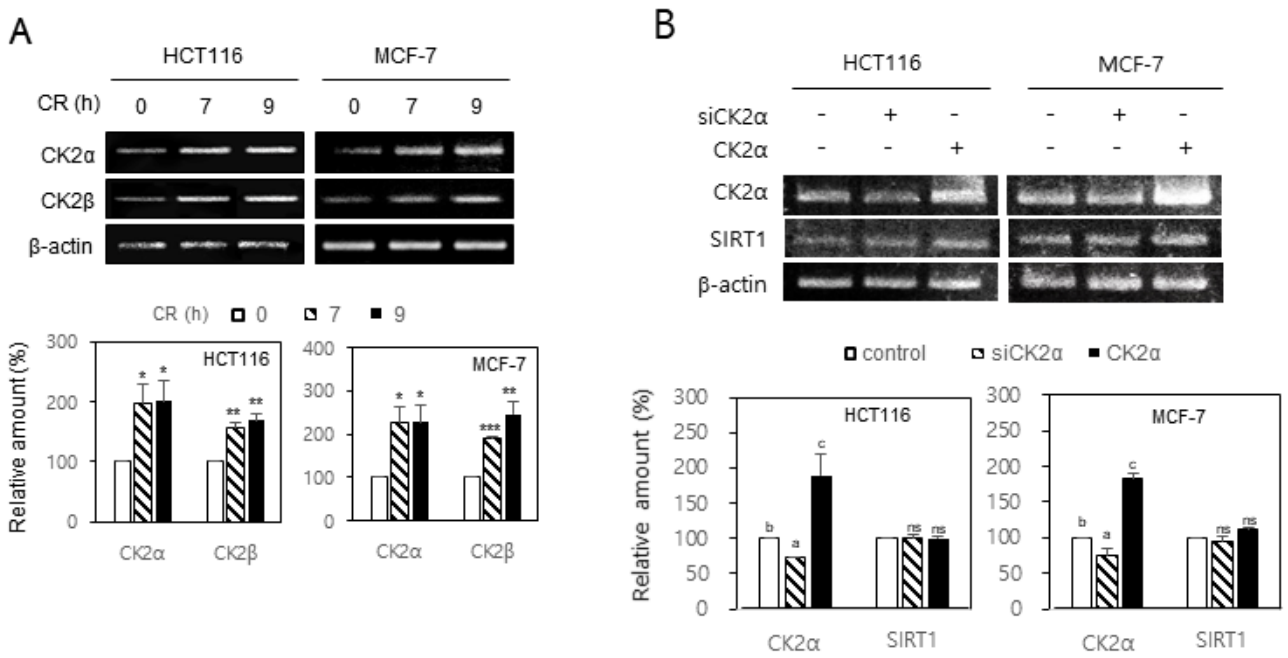
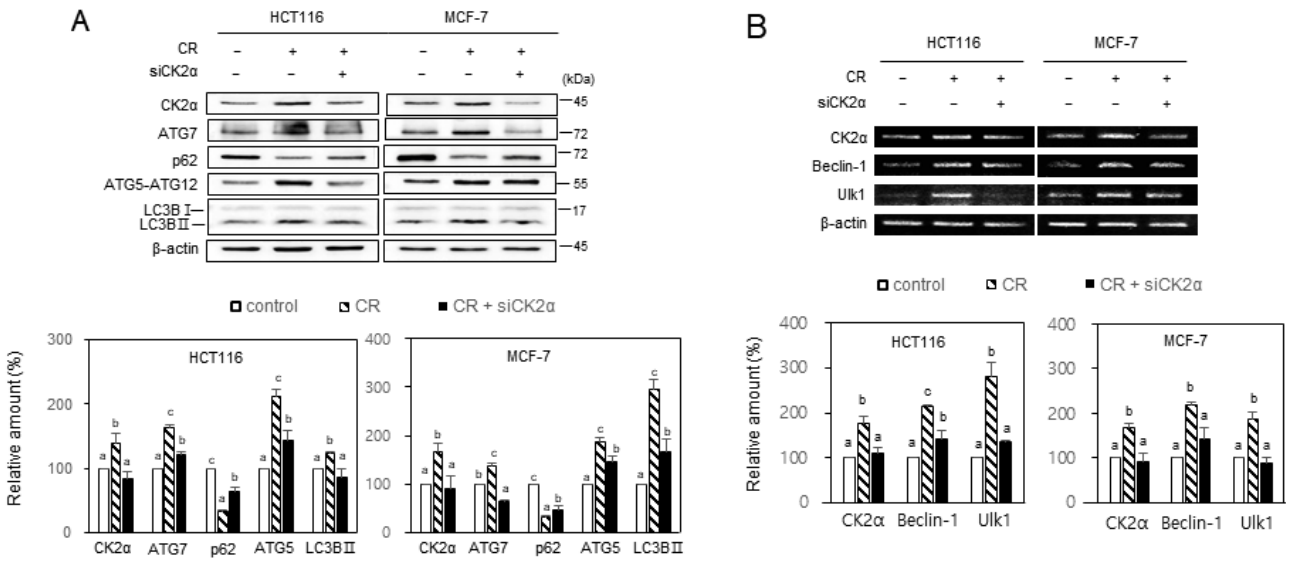
Supplementary Table S1. Sequences of primers used for RT-PCR

Gene		Primer sequence
CK2α (h)	Forward	5′-AAGACCCTGTGTCACGAACCC-3′
	Reverse	5′-GGCTCCTCCCGAAAGATCATAC-3′
SIRT1 (h)	Forward	5′-CAGTGGCTGGAACAGTGAGA-3′
	Reverse	5′-TCTGGCATGTCCCACTATCA-3′
Beclin-1 (h)	Forward	5′-CTCTCGCAGATTCATCCCCC-3′
	Reverse	5′-TGGGCATAACGCATCTGGTT-3′
Ulk1 (h)	Forward	5′-CCTCATGGAGCAAGGCACA-3′
	Reverse	5′-CTCACGGTGCTGGAACATCT-3′
β-actin (h)	Forward	5′-TCCCTGGAGAAGAGCTACGA-3′
	Reverse	5′-AGCACTGTGTTGGCGTACAG-3′
bec-1 (n)	Forward	5′-CAAAGAAGGCCAGATTCAGC-3′
	Reverse	5′-CGTTGTCGGATGGTTTTCTT-3
unc-51 (n)	Forward	5′-GCTGCTCTTGACGAGCTTTT-3′
	Reverse	5′-ACAAATCCCTGTCGTTCCAG-3
act-2 (n)	Forward	5′-CCCAATTGAGCATGGTATCG-3′
	Reverse	5′-AGCGTTCGTTTCCAACAGTG-3′

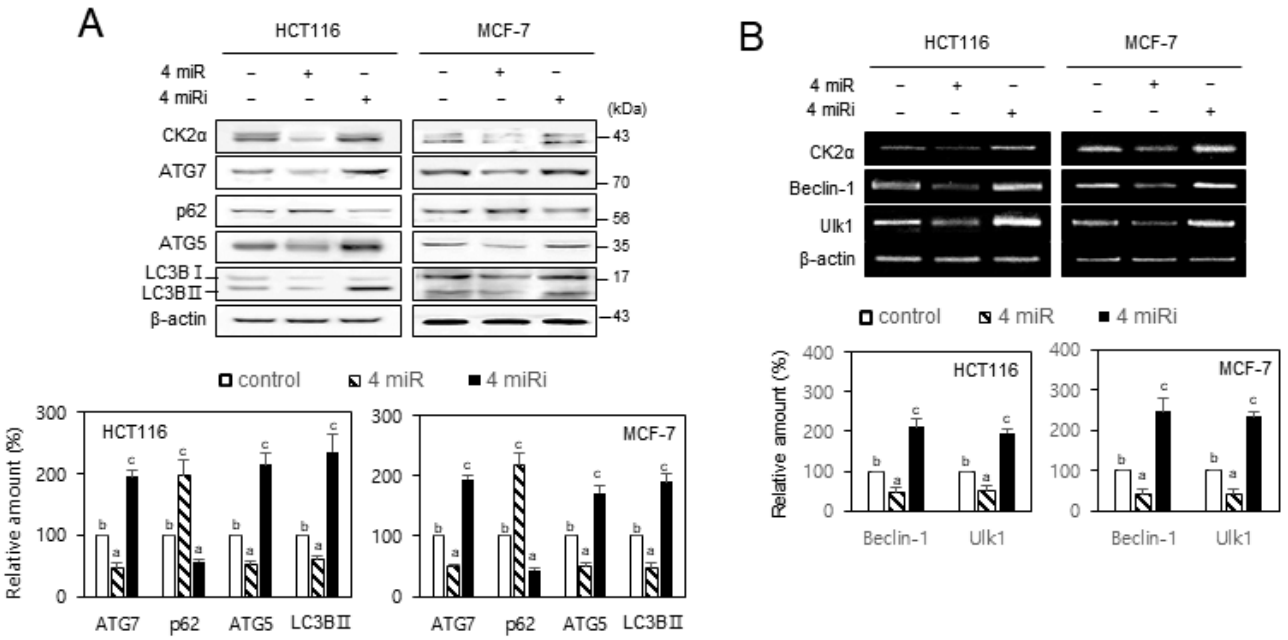
h, human; n, nematode.



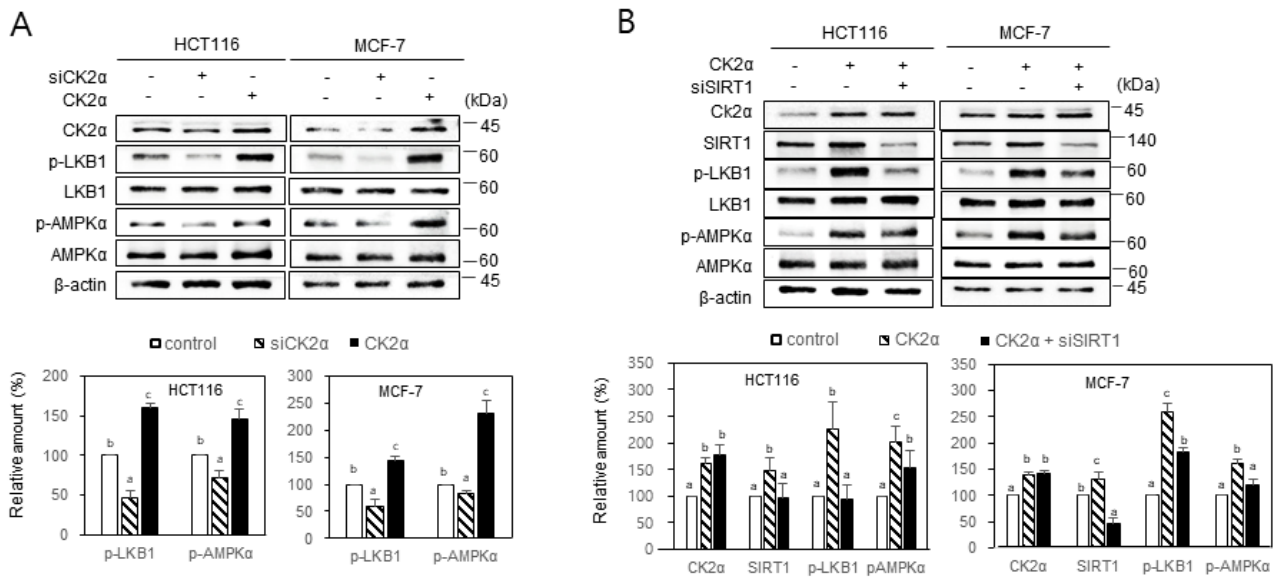
Supplementary Fig. S1. CR upregulated CK2α and SIRT1 at the transcription level, whereas CK2 upregulated SIRT1 at the post-translation level. (A) HCT116 and MCF-7 cells were transfected with CK2α siRNA for 1.5 days, then incubated under CR condition for 7 h. (B) Cells were transfected with CK2α siRNA or pcDNA3.1-HA-CK2α for 2 days. Total RNA was then extracted from cells, after which the level of each mRNA was determined by RT-PCR using specific primers and reverse transcriptase (top). Representative data from three independent experiments are shown. β-Actin was used as a control. The graphs represent the quantitation of each mRNA relative to β-actin (bottom). Data were reported as mean ± SEM. $*P < 0.05$; $**P < 0.01$; $***P < 0.001$; ns, not significant. Bars that do not have the same letter (a, b, c) were significantly different among groups at $P < 0.05$.



Supplementary Fig. S2. CK2α downregulation abrogated autophagy induction, caused by CR. HCT116 and MCF-7 cells were transfected with CK2α siRNA for 1.5 days, then incubated under CR condition for 7 h. The level of each protein (A) and mRNA (B) was determined by immunoblotting using specific antibodies and RT-PCR using specific primers, respectively (top). Representative data from three independent experiments are shown. β-Actin was used as a control. The graphs represent the quantitation of each protein and mRNA relative to β-actin (bottom). Data were reported as mean ± SEM. Bars that do not have the same letter (a, b, c) were significantly different among groups at $P < 0.05$.



Supplementary Fig. S3. Antisense inhibitors of miR-186, miR-216b, miR-337-3p, and miR-760 induced autophagy in human cancer cells. MCF-7 and HCT116 cells were transfected with four miRs (miR-186, miR-216b, miR-337-3p, and miR-760) or antisense inhibitors of four miRs (4 miRi). The level of each protein (A) and mRNA (B) was determined by immunoblotting using specific antibodies and RT-PCR using specific primers, respectively (top). Representative data from three independent experiments are shown. β-Actin was used as a control. The graphs represent the quantitation of each protein and mRNA relative to β-actin (bottom). Data were reported as mean ± SEM. Bars that do not have the same letter (a, b, c) were significantly different among groups at $P < 0.05$.



Supplementary Fig. S4. CK2 activated AMPK through the SIRT1-LKB1 pathway in human cancer cells. (A) HCT116 and MCF-7 cells were transfected with pcDNA3.1-HA-CK2α or CK2α siRNA for 2 days. (B) Cells were transfected with pcDNA3.1-HA-CK2α for 2 days in the absence or presence of SIRT1 siRNA. The level of each protein was determined by immunoblotting using specific antibodies (top). Representative data from three independent experiments are shown. β-Actin was used as a control. The graphs represent the quantitation of p-AMPK (T172) and p-LKB1 (S428) relative to the unphosphorylated AMPK and LKB1 levels, respectively. The graphs represent the quantitation of CK2α and SIRT1 relative to β-actin (bottom). Data were reported as mean ± SEM. Bars that do not have the same letter (a, b, c) were significantly different among groups at $P < 0.05$.