Figure S1. Identification of cardiac microvascular endothelial cells by immunofluorescence analysis. Magnification, (A) x100 and (B) x200. vWF, von Willebrand factor.

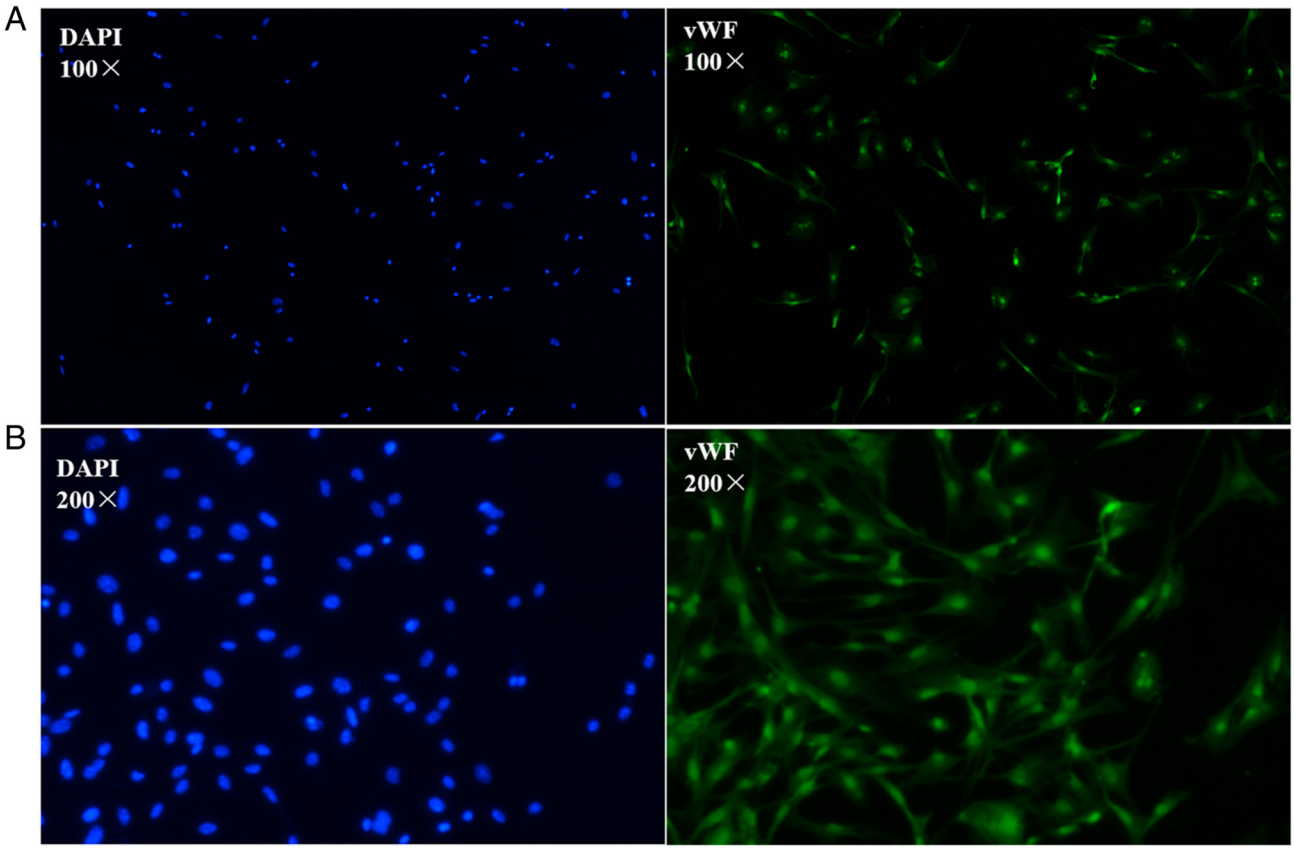

Figure S2. KLF2 expression after cell transfections. (A) KLF2 expression was increased after KLF2 overexpression vector transfection. Data are presented as the mean ± SD. *P<0.05, KLF2 Overexpression (n=6) vs. Control (n=6); #P<0.05, KLF2 Overexpression (n=6) vs. Control Overexpression (empty vector) (n=6). The control group was treated without vectors. (B) KLF2 expression was decreased after siKLF2 transfection. Data are presented as the mean ± SD. *P<0.05, siKLF2 (n=6) vs. Control (n=6); #P<0.05, siKLF2 (n=6) vs. NC (non-specific control siRNA) (n=6). The control group was treated without siRNAs. KLF2, Krüppel-like factor 2; NC, non-specific control; si, small interfering RNA.

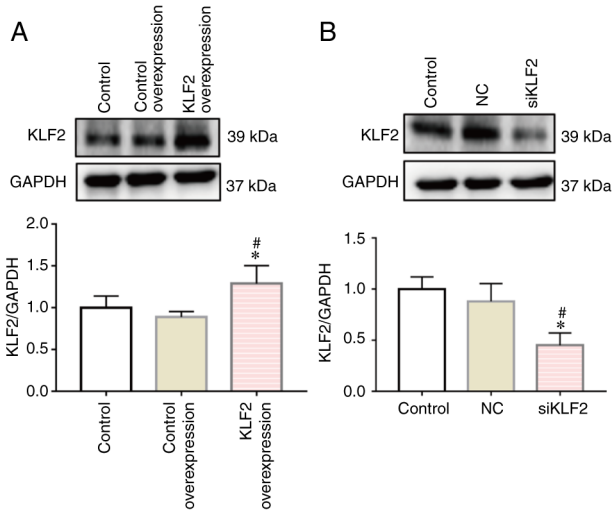

Figure S3. Palmitic acid decreased viability and increased ROS of CMECs. (A) Palmitic acid reduced CMEC viability (relative to 0 group). (B) Palmitic acid increased the intracellular ROS level (relative to 0 group). Data are presented as the mean ± SD. *P<0.05, PA, 400 μM (n=10) vs. PA, 0 μM (n=10); $^{\#\#}$P<0.05, PA, 800 μM (n=10) vs. PA, 0 μM (n=10). CMEC, cardiac microvascular endothelial cell; PA, palmitic acid; ROS, reactive oxygen species.

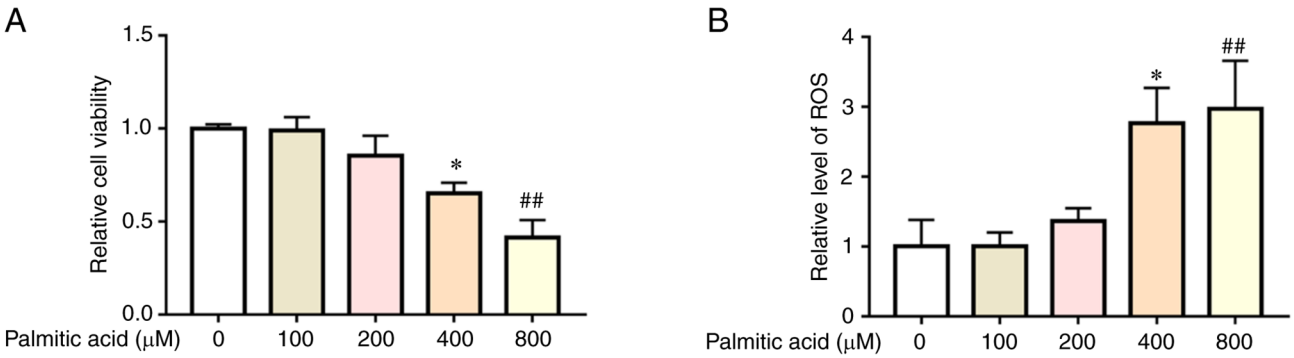

Figure S4. AICAR treatment exerts anti-inflammatory effects. Serum (A) TNF-α and (B) IL-6 levels. Data are presented as the mean ± SD. *P<0.05, Model (n=5) vs. Control (n=5); #P<0.05, Model + AICAR (n=5) vs. Model (n=5). AICAR, 5-aminoimidazole-4-carboxamide ribonucleotide.

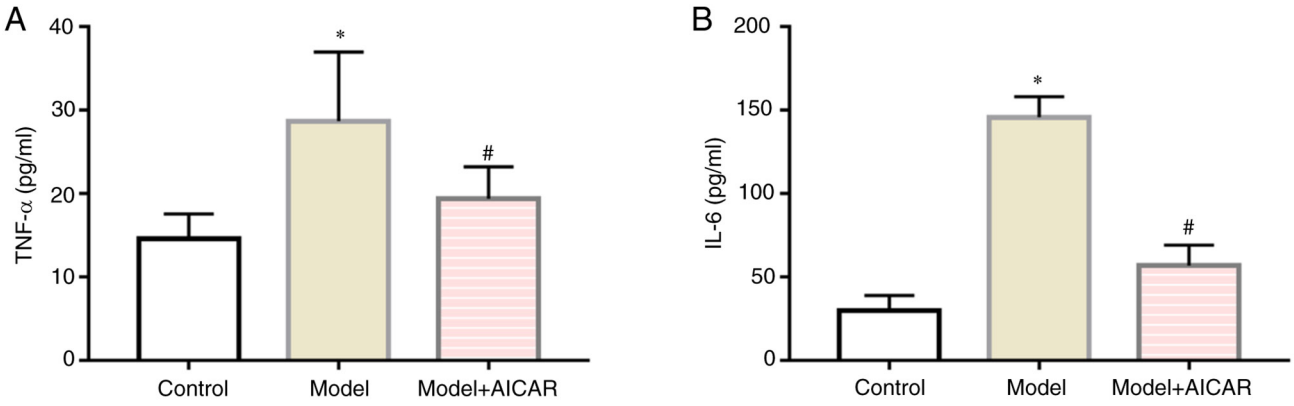

Figure S5. Nicorandil increased viability and decreased ROS of CMECs treated with palmitic acid. (A) Nicorandil restored the viability of cardiac microvascular endothelial cells (relative to control group). (B) Nico decreased the levels of intracellular ROS (relative to control group). Data are presented as the mean ± SD. *P<0.05, PA (n=6) vs. Control (n=6); #P<0.05, PA + Nico (n=6) vs. PA (n=6). Nico, nicorandil; PA, palmitic acid; ROS, reactive oxygen species.

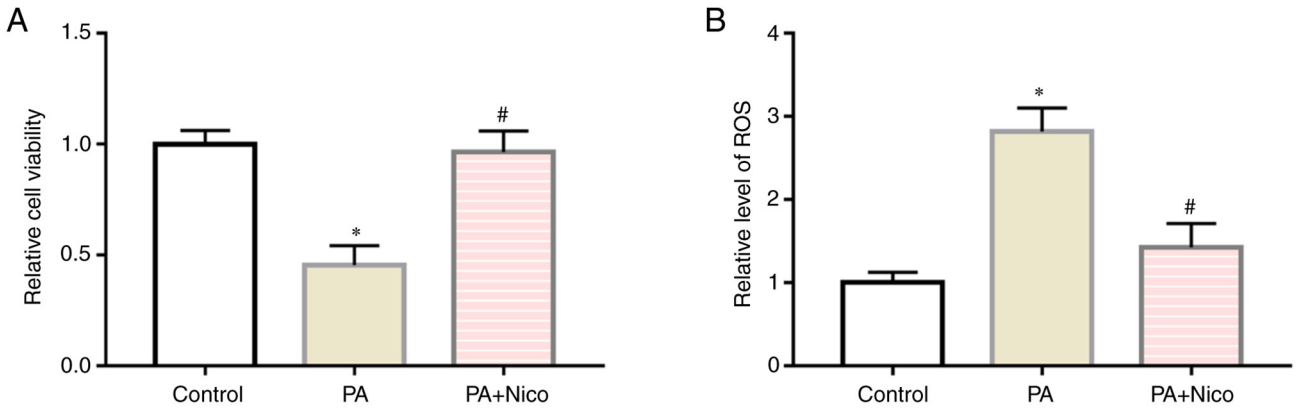

Figure S6. siKLF2 was used to suppress KLF2 expression in CMECs. AICAR did increased the ratio of p-AMPK/AMPK but did not increase the expression levels of KLF2 or the ratio of p-eNOS/eNOS. Data are presented as the mean ± SD. *P<0.05, PA (n=6) vs. Control (n=6); $^{\#}$P<0.05, PA + AICAR + NC (n=6) vs. PA (n=6); $^{\&}$P<0.05, PA + AICAR + siKLF2 (n=6) vs. PA (n=6); $^{\$}$P<0.05, PA + AICAR + NC (n=6) vs. PA + AICAR + siKLF2 group (n=6). AICAR, 5-aminoimidazole-4-carboxamide ribonucleotide; eNOS, endothelial nitric oxide synthase; KLF2, Krüppel-like factor 2; NC, non-specific control; p-, phosphorylated; PA, palmitic acid; si, small interfering RNA.