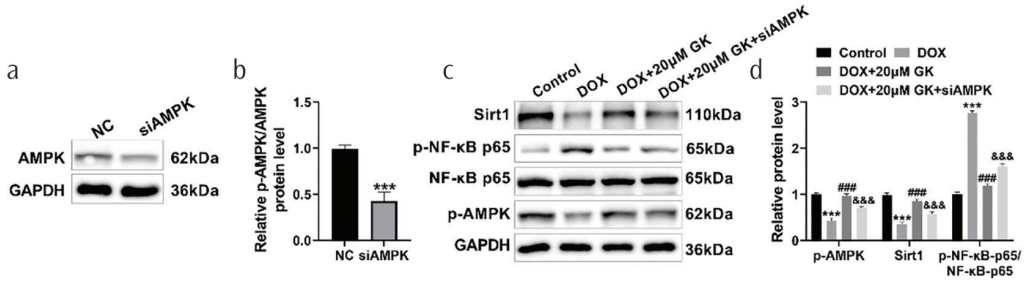

SUPPLEMENTRAY FIG. 1. Verification of the molecular mechanism by which AMPK mediates GK attenuation of DOX-induced myocardial injury via the AMPK/Sirt1/NF-κB pathway. (a, b) Western blot confirming the knockdown efficiency of siAMPK. $^{***}p < 0.001$ vs. NC. (c, d) Western blot results show that DOX treatment suppresses AMPK phosphorylation and Sirt1 protein expression while increasing phosphorylation of NF-κB p65. Co-treatment with DOX and 20 μM GK markedly promotes AMPK phosphorylation and Sirt1 expression and inhibits NF-κB p65 phosphorylation. In contrast, in the siAMPK cell line, combined treatment with DOX and 20 μM GK reverses these protein expression changes. $^{***}p < 0.001$ vs. Control; $^{\#\#\#}p < 0.001$ vs. DOX; $^{\&\&\&}p < 0.001$ vs. DOX + 20 μM GK. DOX, doxorubicin; AMPK, adenosine monophosphate–activated protein kinase; GK, ginkgetin; NF-κB, nuclear factor-κB.