The Na+/glucose co-transporter inhibitor canagliflozin activates AMP-activated protein kinase by inhibiting mitochondrial function and increasing cellular AMP levels

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Supplementary Figure S1. Quantification of Western blots from Fig. 2. The Western blots were subject to densitometry on the Li-Cor Odyssey IR scanner and the results are presented as ratios (± SD, n = 2) of signals obtained with anti-pT172 to total AMPK-α, or with pACC to total ACC.
Supplementary Figure S2. Quantification of Western blots from Fig. 2. Results expressed as ratios (± SD, n = 2) of signals obtained with anti-pT172 to total AMPK-α, as in Fig. S1.

Supplementary Figure S3. Quantification of Western blots from Figs. 5 and 6. Results expressed as ratios (± SD, n = 2) of signals obtained with anti-pT172 to total AMPK-α, or with pACC to total ACC, as in Fig. S1.
Supplementary Figure S4. Representative Western blots from which the data in Fig. 7A were generated.
Supplementary Figure S5. Canagliflozin does not activate AMPK in mouse muscle, white adipose tissue or spleen in vivo. Samples were from the same experiment as in Fig. 7A but were from: (A) skeletal muscle (tibialis anterior, mean ± SEM, n = 5-11); (B) white adipose tissue (mean ± SEM, n = 4-12). (C) spleen (mean ± SEM, n = 7-10); ns, not significant.