Supporting Information

Shaw *et al.* 10.1073/pnas.0807735105

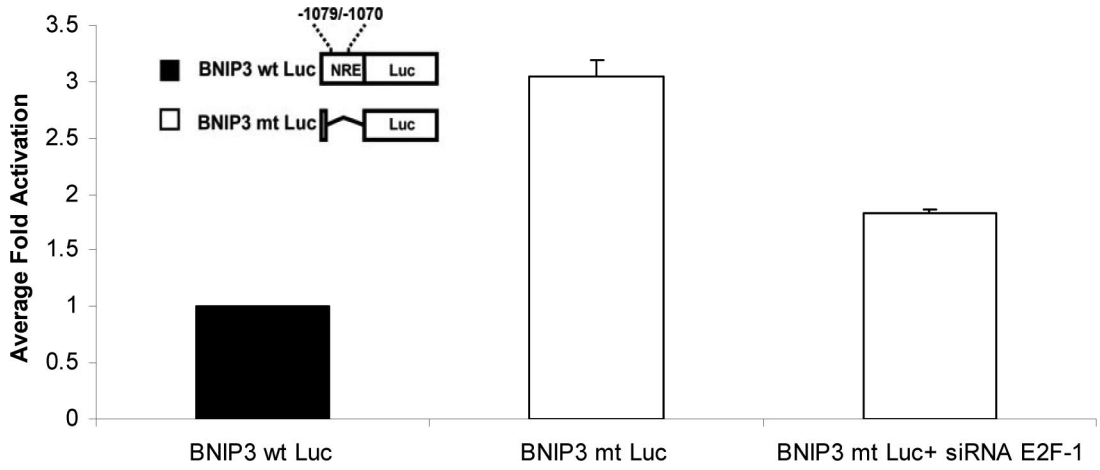

Fig. S1. The NF-κB elements (-1079/-1070) are required for inhibiting the Bnip3 promoter. Postnatal ventricular myocytes were transfected with Bnip3 promoter luciferase reporter plasmids containing wild Bnip3 promoter (BNIP3 wt) or Bnip3 promoter in which the NF-κB elements (-1079/-1070) adjacent to the E2F-1-binding site were deleted (Bnip3 mt) (1, 2); in the absence and presence of siRNA directed against E2F-1 (see *Materials and Methods* in the main text for details). Deletion of the NF-κB elements resulted in a 1.7-fold ($P < 0.01$) increase in Bnip3 luciferase reporter activity compared with the wild Bnip3 promoter. This finding is consistent with our earlier work demonstrating the importance of this NF-κB site for repressing of Bnip3 promoter activity (1, 2). siRNA directed against E2F-1 suppressed the basal increase in Bnip3 mt activity. Data are expressed as mean change ± SE (in fold) versus the respective control; $n = 3$ replicates for each condition tested. (*, Different from WT Bnip3 promoter; †Different from Bnip3 mt promoter.) 1. Baetz D, *et al.* (2005) Nuclear factor-κB-mediated cell survival involves transcriptional silencing of the mitochondrial death gene BNIP3 in ventricular myocytes. *Circulation* 112:3777–3785. 2. Shaw J, *et al.* (2006) Transcriptional silencing of the death gene BNIP3 by cooperative action of NF-κB and histone deacetylase 1 in ventricular myocytes. *Circ Res* 99:1347–1354.