Supporting Information

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SI Text

Dual Luciferase Reporter Assay. The p53-binding site within human *killin* promoter was amplified by PCR from the genomic DNA of human HEK293T cells using primers: 5′- GGTACCTCTGGGTGCGAGCGCAGAG-3′ and 5′-AGATCTCGTTATCCTCGCCTCGCGTTG-3′. The 140-bp killin promoter was cloned into the KpnI and BglII sites of the pGL3-basic vector (Promega), and reporter assays were carried out essentially as described (1). As a control, mutations (lowercase letters) at the key consensus bases within the p53-binding site (-GCAGGCTCTACTGAttATGCCCAGTGTAGCTGCCTGGGttTTGCTCGGGCCGG-) were also created (pGL3-Killin-mutant) by site-directed mutagenesis with the QuikChange kit (Stratagene).

Inducible GFP-Killin Expression. The entire coding region of Killin (amino acids 2–178) was amplified by PCR with primers B1: 5′-CGCGGATCCGATCGCCCGGGGCCAGGCTCC-3′ and B2: 5′-CGCGGATCCTCAGTCCTTTGGCTTGCTCTT-3′ and subcloned into the BamHI site of pEGFP-C1 (Clontech) to allow in-frame fusion of Killin to EGFP. The GFP-Killin fusion was then shuttled into the pTRE2 vector (Clontech) as a NheI-HindIII fragment to allow tetracycline-regulated expression stably transfected into the DLD-1 (TetR) cell line. GFP alone was also subcloned into pTRE2 from pEGFP-C1 as a NheI-DraI fragment. All fusion constructs were verified by DNA sequencing.

1. Stein S, Thomas EK, Herzog B, Westfall MD, Rocheleau JV, Jackson R, Wang M, Liang P (2004) *J Biol Chem* 279:48930–48940.