Supplemental Figure 1: Data acquisition and analysis. The flow diagram presented above depicts all data acquisition, filtering and analysis choices employed to identify targets of the dominant negative cJun (TAM67).

Supplemental Figure 2: Epidermal expression of COX-2 and OPN proteins in response to TPA and TAM67. Epidermal protein lysates were run on 10% Bis/Tris NuPAGE gels, and transferred membranes were probed with anti-COX2 or OPN antibody to determine the expression levels of these proteins in response to TPA and TAM67. Densitometry values (normalized to Eif4a1 protein expression) are shown below each western blot. The results shown are representative of 3 experiments. Primary keratinocytes exhibit similar results.

Supplemental Figure 3: TAM67 specifically inhibits TPA-induced 4X AP-1 promoter reporter activity. Promotion sensitive (P+) JB6 cells were transiently transfected with 20 ηg pcDNA-TAM67 (or vector control) and 200 ηg luciferase reporter. 48 hours after transfection, the cells were treated with TPA (or DMSO) for 24 hours. The relative TPA-induced luciferase activities of 4X AP-1 and SRE promoter reporter constructs were measured in the presence or absence of TAM67. Panel A depicts the TPA response in the absence of TAM67. TAM67 inhibits TPA-induced (B) trancription from the 4X AP-1 promoter reporter without affecting SRE driven transcription (B). The results shown are averages of 6 wells and representative of 3 experiments. Primary keratinocytes exhibit similar results.

Supplemental Table 1: The tumor promoter induced genes most down-regulated by TAM67 whose induction was completely blocked. Microarray analysis of DMBA-initiated, TPA-

induced K14-HPV16-E7 transgenic or wild type epidermal tissue in the presence or absence of transgenic K14-TAM67 expression was carried out in triplicate. Changes in gene expression were analyzed by co-hybridization of differentially labeled test and control cDNAs derived from these RNAs onto 20,000 oligonucleotide microarrays. Genes of interest were required to be induced by the tumor promoter and inhibited by TAM67 in the presence of tumor promoter such that induction was completely blocked. The *TPA-induced* genes down-regulated more than 4 fold by TAM67 are shown in panel A. Several *E7-induced* genes are also inhibited more than 4 fold by TAM67 expression (B). However, only 3 *E7+TPA-induced* genes were inhibited more than 2 fold (C). To be included in 1A-1C, TAM67 expression was required to eliminate at least 90% of the upregulation by tumor promoter. Table 1D shows all genes in the triple overlap group (greater than 1.5-fold inhibited and not completely blocked). Statistical significance was determined using a pooled t-test.

Supplemental Table 2: Published AP-1/c-Jun target genes oppositely regulated by tumor promoter and TAM67 in mouse epidermis. A review of the literature reveals 17 published AP-1/c-Jun target genes (34-38) to be oppositely regulated by tumor promoter and TAM67 *in vivo* as measured by our analysis. All values are presented as fold change. Red highlights increased gene expression, while green depicts decreased gene expression. The blue boxes indicate opposite regulation by tumor promoter and TAM67 ($\geq$ 2 fold). For these reported cJun targets complete or nearly complete blockade by TAM67 of TPA induction occurred for three of seven inhibited genes and of E7 induction for two of three inhibited genes (a different set).