Additional files

Supplementary Figure 1 – Schematic representation of human Myc mutants, isoforms, and family members

Attached – Supplementary Figure 1

Supplementary Figure 2 – Representative images of colony formation in Myc transformed MCF10A, SH-EP, and LF1/3T cells

Attached – Supplementary Figure 2

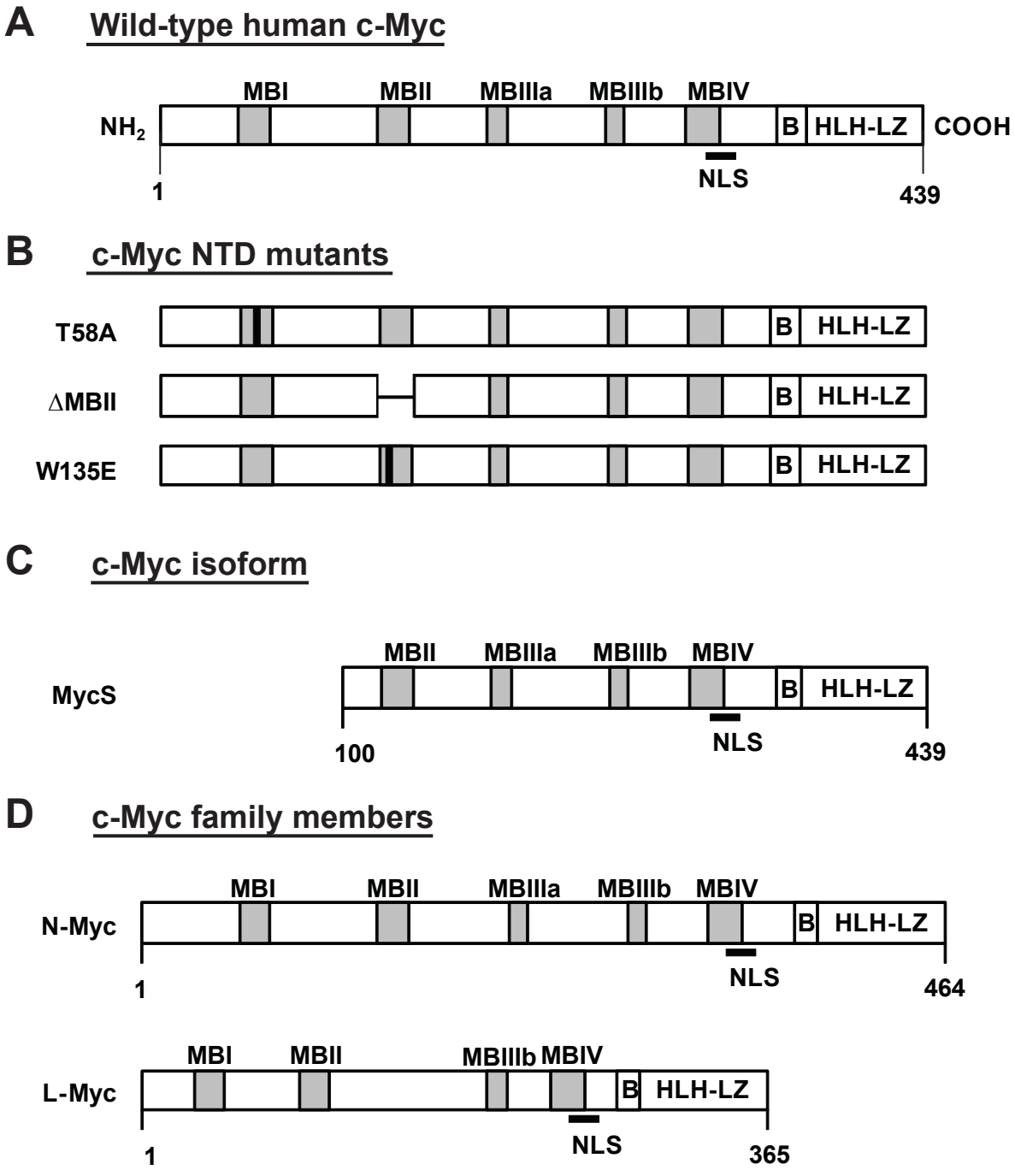
Supplementary Figure 3 – Myc dependent cell death in MCF10A cells

Attached – Supplementary Figure 3

Supplementary Table 1 - Review of structure-function data for Myc-dependent transformation

Attached – Supplementary Table 1

Supplementary Figure 1: Schematic representation of human Myc mutants, isoforms, and family members



Supplementary Figure 2: Representative images of colony formation in Myc-transformed MCF10A, SH-EP and LF1/3T cells

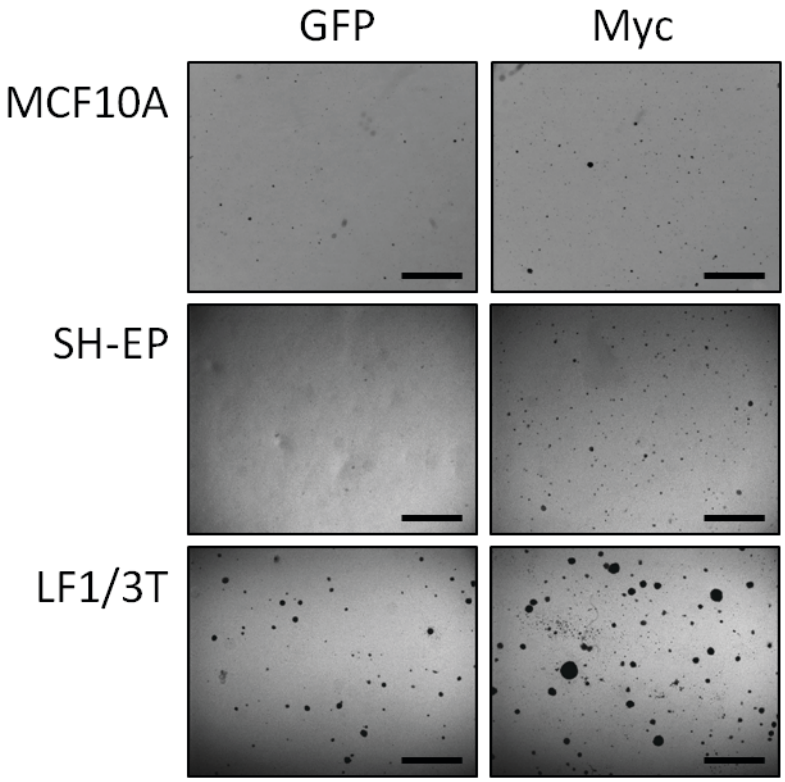
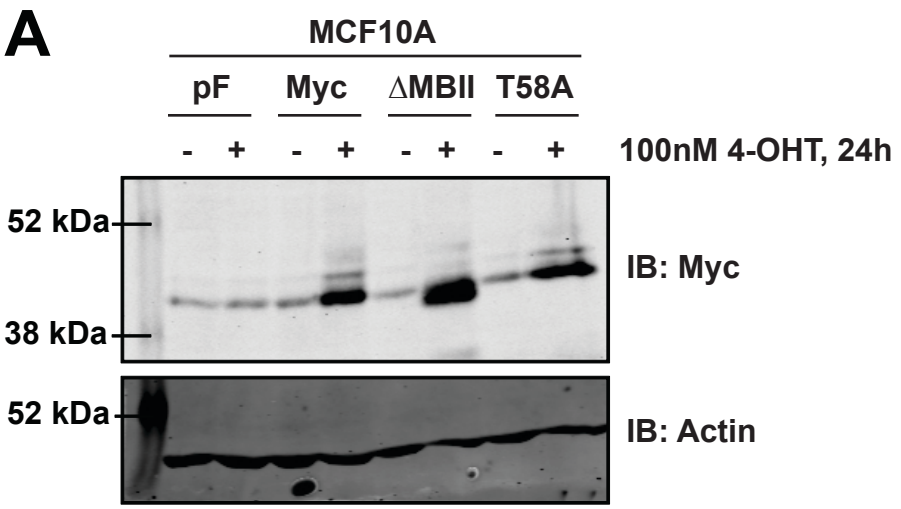


Figure Legend: Soft agar colony formation was imaged on a Leica MZ FLIII Stereomicroscope at 1.6x magnification. Scale bar, 2 mm.

Supplementary Figure 3: Myc-induced cell death in MCF10A cells



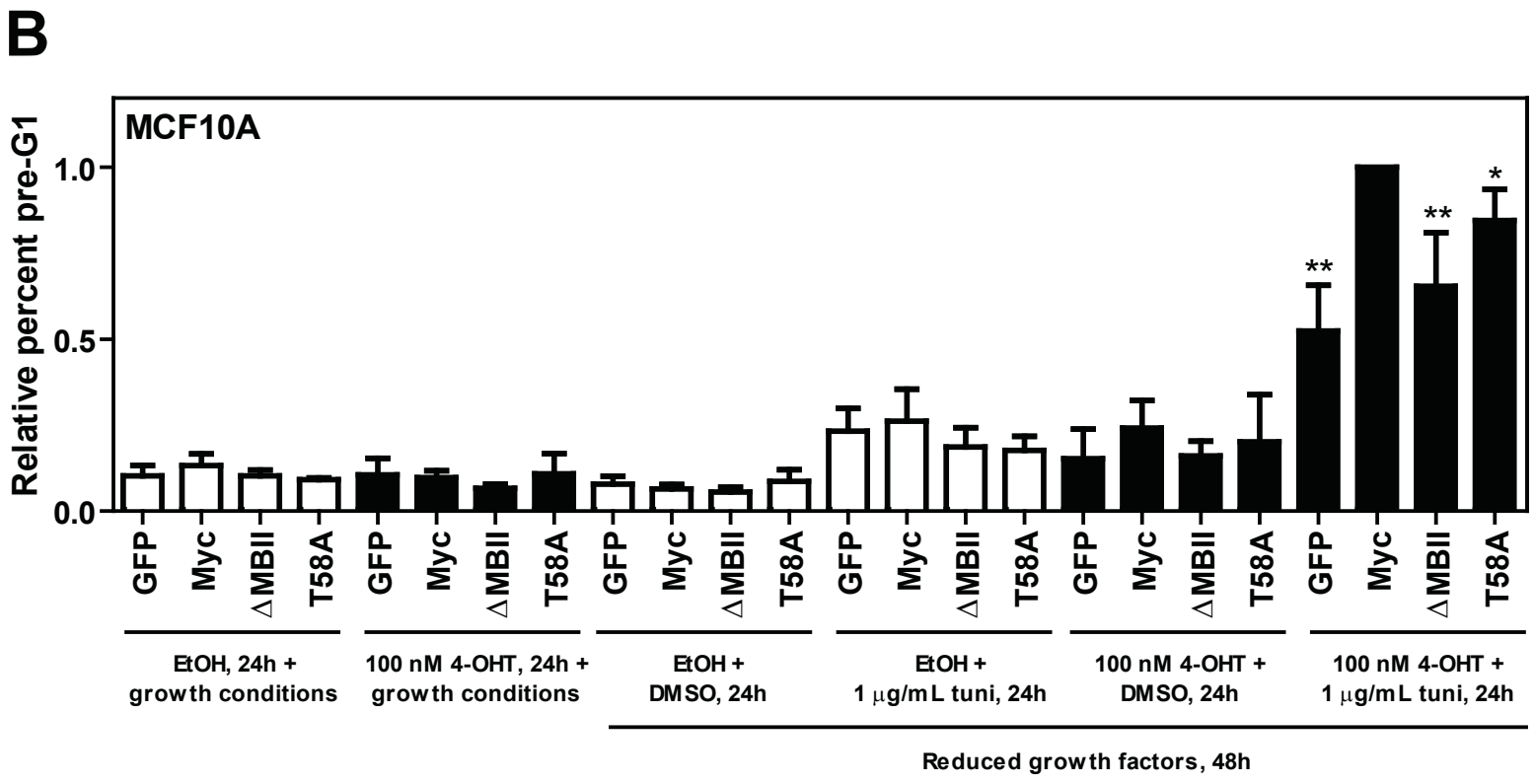


Figure Legend: A) MCF10A cells were stably infected to exress 4-hydroxytamoxifen (4-OHT) inducible Myc and Myc mutants (Callus *et al*, 2008). Inducble expression was evaluated by western bloitting. Briefly, cells were treated with ethanol contraol or 100 nM 4-OHT for 24 hours in reduced growth factor media (0.05% horse serum and supplemented with only 10 μL/mL insulin). B) Myc-dependent cell death was evaluated by treating MCF10A cells with 1 μg/mL tunicamycin (tuni) with concurrent induction of Myc and Myc mutants. Briefly, MCF10A cells were treated with growth factor reduced media for 24 hours and then subsequently treated with 4-OHT (to induce Myc expression) and tunicmycin for an additional 24 hours. Cell death was measured by fixed propidium iodide cell cycle analysis and the percent pre-G1 population. Data is presented relative willd-type Myc, treated with treated with tunicamycin, with mean ± standard deviation of 5 independent experiments. *$p<0.05$, **$p<0.01$, paired t-test. Only low levels of cell death were observed in all control samples.

Supplementary Table 1: Summary of structure-function data for Myc-dependent transformation

Model System	Control	Myc	ΔMBII	T58A	W135E	MycS	L-Myc	N-Myc	Ref.
REF Co-transformation[1]	-	+	-	++	*nd*	-[2]	- or +/-	+	Land, 1983; Stone, 1987; Birrer, 1988; Barrett, 1992; McMahon, 1998; Chang, 2000; Nikiforov, 2002; Pulverer, 2004
Rat-1A	-	+	-	+[3]	-	+	+	+	Small, 1987; Xiao, 1998; Conzen, 2000; Prescott, 2001; Oster, 2003; Huang, 2005; O'Donnell, 2006
Bone Marrow Transplant	-	+	-	++	*nd*	*nd*	*nd*	*nd*	Hemann, 2005; Herbst, 2005
HMEC	-	+	-	++	*nd*	*nd*	*nd*	*nd*	Cowling, 2007; Thibodeaux, 2009

[1] Myc-depdendent transformation was evaluated by focus formation in rat embryo fibroblasts (REFs) co-transfected with activated H-Ras

[2] Evaluated through a deletion mutant spanning amino acids 1-110

[3] Data summarized here represents experiments conducted in 10% FBS. The T58A point mutant is able to promote transformation to a greater extent than wild-type Myc when experiments are conducted in 2% FBS.

-, does not promote transformation
+/-, promotes transformation to a lesser extent than wild-type Myc
+, promotes transformation to a similar extent as wild-type Myc
++, promotes transformation to a greater extent than wild-type Myc
nd, not determined