Supplemental Figure 1

A) Hematoxilin and Eosin (H&E) staining of backskin, ear and tail from epidermal *c-fos*-deficient mice and controls shows no differences in differentiation. Scale bar represents 100μm. 10X magnification.

B) Ki67 staining of backskin, ear and tail from epidermal *c-fos*-deficient mice and controls shows no differences in proliferation. Scale bar represents 100μm. 10X magnification.

Supplemental Figure 2

A) Quantification of number of papillomas per mouse upon DMBA/TPA 2-step skin carcinogenesis protocol in *c-fos* epidermal-deficient mice (red; n=10) compared to littermate controls (blue; n=10). *$p < 0.05$ is considered as significant.

B) CAA to CTA H-Ras gene mutation at codon 61 analyses in mice lacking epidermal *c-fos* and controls. *c-fos* deficiency does not alter mutation frequency. Representative image. n=10

C) Quantification of percentages of proliferative and apoptotic keratinocytes in 4-week-old K5-SOS$^+$ *c-fos* epidermal-deficient mice compared to littermate controls after Ki67 immunohistochemistry (left panel) or TUNEL assay (right panel) shows no differences in proliferation or apoptosis. n=7 per genotype.

D) Immunohistochemistry analyses show c-fos expression in c-fos-deficient K5-SOS$^+$ papillomas of 6-week-old mice compared to controls. *c-fos*-deficient mice show background signal although no positive c-fos nuclei. Similar Keratin 5 (basal cell marker) expression pattern was observed in both cases. Scale bar represents 100μm. 10X magnification.

E) RT-qPCR analyses show lack of significant changes in *p16* and *p19* expression of K5-SOS$^+$ papillomas when comparing *c-fos*-deficient epidermis and control mice.

F) RT-qPCR analyses show lack of significant changes in *Presenilin 1* and *2* and *ADAM10* expression of K5-SOS$^+$ papillomas when comparing *c-fos*-deficient epidermis and control mice.

G) Western blot upon topical TPA treatment of *c-fos* epidermal-deficient mice and controls in the absence/presence of DAPT. Enhanced differentiation upon TPA treatment is observed, which is impaired by inhibiting Notch activation. Representative image. n=4

Supplemental Figure 3

A) RT-qPCR analyses of primary keratinocytes lacking *c-fos* infected with pBabe retroviruses over-expressing *H-RasV12 (Ras)* or empty vector (Co). Lack of *c-fos* induces increased expression of genes involved in differentiation and growth arrest like *Keratin 10* and *p21* compared to controls. *TIMP-3* levels are reduced upon *Ras* over-expression but not affected by *c-fos* deficiency. Senescence associated markers *p16* and *p19* are not induced in *c-fos*-deficient cells. *$p < 0.05$ is considered as significant.

B) TACE activity analyses of primary keratinocytes lacking *c-fos* infected with pLPC retroviruses over-expressing *H-RasV12* (pRas), pBabe retroviruses over-expressing *H-RasV12* (pRas) or *GFP* (GFP) for 6 days. Lack of *c-fos* induces precocious TACE activation upon *Ras* over-expression compared to controls. *$p < 0.05$ is considered as significant. Experiment performed 2 times.

C) Western blot analyses of proliferation markers such as pSer10-HistoneH3 and PCNA after 3 days of *H-RasV12*-over-expression in *c-fos*-deficient keratinocytes showing no significant changes in expression levels compared to control cells.

Supplemental Figure 4

A) Phospho-c-fos western blot analysis of wild-type keratinocytes upon increasing concentrations of Ca2+ for 24 hours shows dose-dependent increased c-fos phosphorylation.

B) RT-qPCR analyses of primary keratinocytes lacking *c-fos* treated with 2mM Ca2+ at different time points. Lack of *c-fos* induces enhanced expression of *p53, TACE, Notch1, Hes1* and of early differentiation markers like *Keratin 1* and *p21*. *p < 0.05 is considered as significant.

C) RT-qPCR analyses of primary keratinocytes lacking *c-fos* treated with 2mM Ca2+ at different time points. Lack of *c-fos* does not alter the expression of basal cell markers like *Keratin 5* and *Keratin 14* as well as levels of *ADAM10*. Lack of *c-fos* induces increased expression of early differentiation markers such as *Keratin 10* and increased levels of *TIMP-1*. Conversely, *TIMP-3 expression* is impaired in keratinocytes lacking *c-fos*. *p < 0.05 is considered as significant.

D) Increased TACE activity upon Ca2+-induced differentiation in *c-fos*-deficient keratinocytes. 24h of 1mM and 2mM Ca2+ treatment. *p < 0.05 is considered as significant.

Supplemental Figure 5

RT-qPCR analyses of primary keratinocytes lacking *c-fos* treated with 10ng/ml of TPA at different time points. Lack of *c-fos* induces increased expression of *p53, TACE, Notch1* and of early differentiation and growth arrest markers like *Keratin 1, Keratin 10, p21* as well as of *TIMP-1* compared to controls. *ADAM10* levels are reduced upon TPA in *c-fos*-deficient keratinocytes and *TIMP-3* levels are not significantly changed. *p < 0.05 is considered as significant.

Supplemental Figure 6

A) RT-qPCR analyses of primary keratinocytes concomitantly lacking *c-fos* and *p53* treated with 2mM Ca2+ at different time points. Concomitant *c-fos*- and *p53*-deficiency impairs expression of Notch1, TACE, *Keratin 1, p21, Hes1* and *Keratin 10* compared to controls, which express both *c-fos* and *p53.*

B) CyclinD1 RT-qPCR analyses of primary keratinocytes concomitantly lacking *c-fos* and *p53* infected with empty vector, H-RasV12 (Ras), shTACE and Ras and shTACE viruses.

C) Western blot analyses of *c-fos*-deficient primary keratinocytes upon TACE inhibition with TAPI-1 (broad ADAM and MMP inhibitor) in 2mM Ca2+-inducing differentiation conditions for 24h shows impaired expression of differentiation markers such as NICD and Keratin 1 indicating TACE-dependent precocious differentiation.

C) TACE activity in Ca2+-induced keratinocyte differentiation conditions upon *TACE* siRNA-mediated knock-down. 24h of 2mM Ca2+ treatment. *p < 0.05 is considered as significant.

E) Western blot analyses of wild type keratinocytes upon *ADAM10* siRNA-mediated knock-down shows that upon ADAM10 downregulation, keratinocyte differentiation

measured by expression of Keratin 1, Keratin 10 and NICD is not altered. The results indicate that ADAM10 is dispensable for Ca2+-induced keratinocyte differentiation.

Supplemental Figure 7

A) RT-qPCR analyses show increased *Notch1* mRNA levels upon NICD over-expression in wild-type primary keratinocytes with AdNICD, which is abolished in the presence of MAM51 peptide inhibitor. *p < 0.05 is considered as significant. Experiment performed 2 times.

B) Western blot analyses show increased levels of Notch1 full length upon NICD over-expression in wild-type primary keratinocytes with AdNICD, by using two different antibodies from BD Pharmingen and Santa Cruz Biotechnology. Experiment performed 2 times.

C) RT-qPCR shows unchanged *TACE* expression levels upon NICD over-expression expression in wild-type primary keratinocytes with AdNICD in the absence or presence of MAM51 peptide inhibitor. Experiment performed 2 times.

D) Western blot shows unchanged TACE protein levels upon NICD over-expression with AdNICD in wild-type primary keratinocytes. Experiment performed 2 times.

E) Impaired *Notch1* mRNA expression analyzed by RT-qPCR upon γ-secretase inhibition with DAPT in Ca2+-induced differentiation conditions in primary wild type keratinocytes. *p < 0.05 is considered as significant.

Supplemental Figure 8

A) RT-qPCR analyses of primary keratinocytes lacking *p53* treated with 2mM Ca2+ at different time points. Lack of *p53* impairs expression of *TACE, Notch1, Hes1, p21, Keratin 1, Keratin 10* as well as *TIMP-3*, and no changes in *ADAM10* levels. *p < 0.05 is considered as significant.

B) RT-qPCR analyses of primary keratinocytes lacking *p53* treated with 10ng/ml of TPA at different time points. Lack of *p53* impairs the expression of *TACE, Notch1, Hes1, Keratin 1, Keratin 10* and *p21*. *p < 0.05 is considered as significant.

Supplemental Figure 9

A) RT-qPCR analyses reveal that wild-type p53 (wt p53) over-expression and not mutant p53 (mut p53), induce the expression of *TACE, Notch1, Hes 1, Keratin 10, p21* and does not affect the expression of *Loricrin, ADAM10* and *TIMP-3* compared to controls. TACE over-expression induces expression of *Notch1, Hes 1, Keratin 10, p21* and does not alter the levels of *Loricrin, ADAM10* and *TIMP-3* compared to controls. Infection of wild-type cells with adenovirus over-expressing NICD (AdNICD) induces the expression of *Notch1, Hes 1, Keratin 10, p21* and *TIMP-3* while no changes in *TACE, Loricrin* and *ADAM10*. *p < 0.05 is considered as significant. Experiment performed 2 times.

B) Western blot reveals that wt p53 over-expression, and not mut p53, induces Pro-TACE, TACE, Notch1 and NICD expression in wild type keratinocytes upon transient transfection. Experiment performed 2 times.

C) Western blot analyses of wild-type keratinocytes upon treatment with Nutlin-3 for 24 and 48 hours to induce p53 stabilization. Increased protein levels of Pro-TACE, TACE, Notch1 and NICD are detected when p53 levels are increased by Nutlin-3 treatment.

D) RT-qPCR analyses of primary keratinocytes lacking *p53* infected with retroviruses over-expressing *H-RasV12* (Ras) or empty vector (Co) after 6 days. Lack of *p53* does not affect *ADAM10, PSN1/2* expression, but impairs expression *Keratin 10, Involucrin, p16* and *p19*. *p < 0.05 is considered as significant.

E) Western blot analyses of primary keratinocytes lacking *p53* infected with retroviruses over-expressing *H-RasV12* (Ras) or empty vector (Co) after 6 days. Lack of *p53* impairs expression of senescence-associated genes (p16, p19) compared to controls.

Supplemental Figure 10

A) Quantification of number of papillomas and papilloma size average per mouse upon DMBA/TPA 2-step skin carcinogenesis protocol in $p53^{KI/KI}$ mice (red; n=7) compared to littermate $p53^{+/+}$ controls (blue; n=7). Similar number and size of tumors are developed.

B) Analyses on proliferation by Ki67 as well as apoptosis by TUNEL assay in the papillomas of $p53^{KI/KI}$ mice treated with DMBA/TPA for 15 weeks and, when indicated, 15 days of intraperitoneal treatment with 3mg/mouse/day of Tamoxifen. Quantification of these assays reveals no significant differences in both proliferation and apoptosis upon *p53* restoration. n=7 mice per genotype.

C) Histological analyses of papillomas derived from $p53^{+/+}$ and $p53^{KI/KI}$ mice treated with DMBA/TPA for 15 weeks and 15 days of intraperitoneal treatment with 3mg/mouse/day of Tamoxifen. SA-β-gal staining (upper panel) does not reveal differences in senescence, TUNEL assay (middle panel) does not reveal differences in apoptosis; however, increased Keratin 10 expression (lower panel) is detected upon p53 restoration. Scale bar represents 100μm. 10X magnification.

D) p53 western blot analyses in $p53^{+/+}$and $p53^{KI/KI}$ cells over-expressing *H-Ras-V12* before and after restoration of *p53* by using 4-OH-Tamoxifen for 48h.

E) RT-qPCR analyses of wild-type and $p53^{KI/KI}$ keratinocytes over-expressing *H-RasV12* 4 or 6 days after retroviral infection (Ras) or empty vector. All keratinocytes were treated with 1μM 4-OH Tamoxifen for 48 hours to restore p53 expression. *p53* restoration induces expression of genes involved in differentiation and growth arrest such as *TACE, Notch1, Hes1, Keratin 1, Keratin 10* and *p21*, but impaired expression of senescence-associated genes (*p16, p19*) compared to controls. *p < 0.05 is considered as significant.

Supplemental Figure 11

A) Immunohistochemistry of Notch1 showing and overview of the tumors and a delineated area enlarged in part B. Scale bar represents 200μm. 5X magnification.

B) Immunohistochemistry of c-Fos, TACE, Notch1, Keratin 1 and Loricrin, as well as Hematoxilin & Eosin staining in poorly-differentiated and well-differentiated human skin SCC showing different expression pattern and correlation of lack of c-Fos expression with reduced total Notch1 - likely due to activation, and increased membranous TACE, Keratin 1 and Loricrin expression. Representative images; see Supplemental Table 2 for complete analyses. Scale bar represents 50μm. 20X magnification.

Supplemental Figure 12

A) H&E and TACE staining in the absence or presence of a TACE antibody blocking peptide in normal skin, which has been sun exposed. Enlarged image shows

membranous TACE staining (arroheads). Representative images. 10X and 20X magnifications. Scale bar represents 100μm.

B) H&E and TACE actinic keratosis staining. Minimal atypia shows membranous TACE staining, pronounced atypia shows cytosolic TACE staining. Scale bar represents 100μm. 10X magnification.

Supplemental Figure 13

A) RT-qPCR analyses show c-Fos mRNA levels in a panel of SCC cell lines, which are increased in almost 10/11 cases compared to human primary keratinocytes. *p < 0.05 is considered as significant.

B) c-Fos activity assay shows c-Fos activity in the panel of SCC cell lines. c-Fos activity is increased in all SCC cell lines compared to human primary keratinocytes. *p < 0.05 is considered as significant.

C) RT-qPCR analyses show reduced *c-Fos* levels upon shRNA-mediated *c-Fos* knock-down in HEK and in the panel of SCC cell lines. *p < 0.05 is considered as significant.

D) FACS analysis of EdU proliferation assay performed after *c-Fos* shRNA-mediated knock-down in cells with *P53+/+* (HEK) or SCC cell lines with *P53 +/m* alleles (dark/light grey bars) compared to SCC cell lines with mutant *P53 m/m* alleles (black/white bars). Results show that inhibition of *c-Fos* in cells with mutant *P53* alleles has no effect on proliferation, as well as in human primary keratinocytes. However, impaired proliferation is observed in SCC cell lines that contain a functional *P53* allele. *p < 0.05 is considered as significant. Experiment performed 2 times.

Supplemental Figure 14

A) FACS analysis of EdU proliferation assay performed after c-Fos inhibition (10μM c-Fos inhibitor T-5224, 48h treatment) in cells with *P53+/+* (HEK), a SCC cell line (SCCO12) with *P53 +/m* alleles (dark/light grey bars) compared to a SCC cell line (SCCO9) with mutant *P53 m/m* alleles (black/white bars). Results show that inhibition of c-Fos in cells with mutant *P53* alleles has no effect on proliferation, as well as in human primary keratinocytes. However, impaired proliferation is observed in SCC cell lines that contain a functional *P53* allele. *p < 0.05 is considered as significant. Experiment performed 2 times.

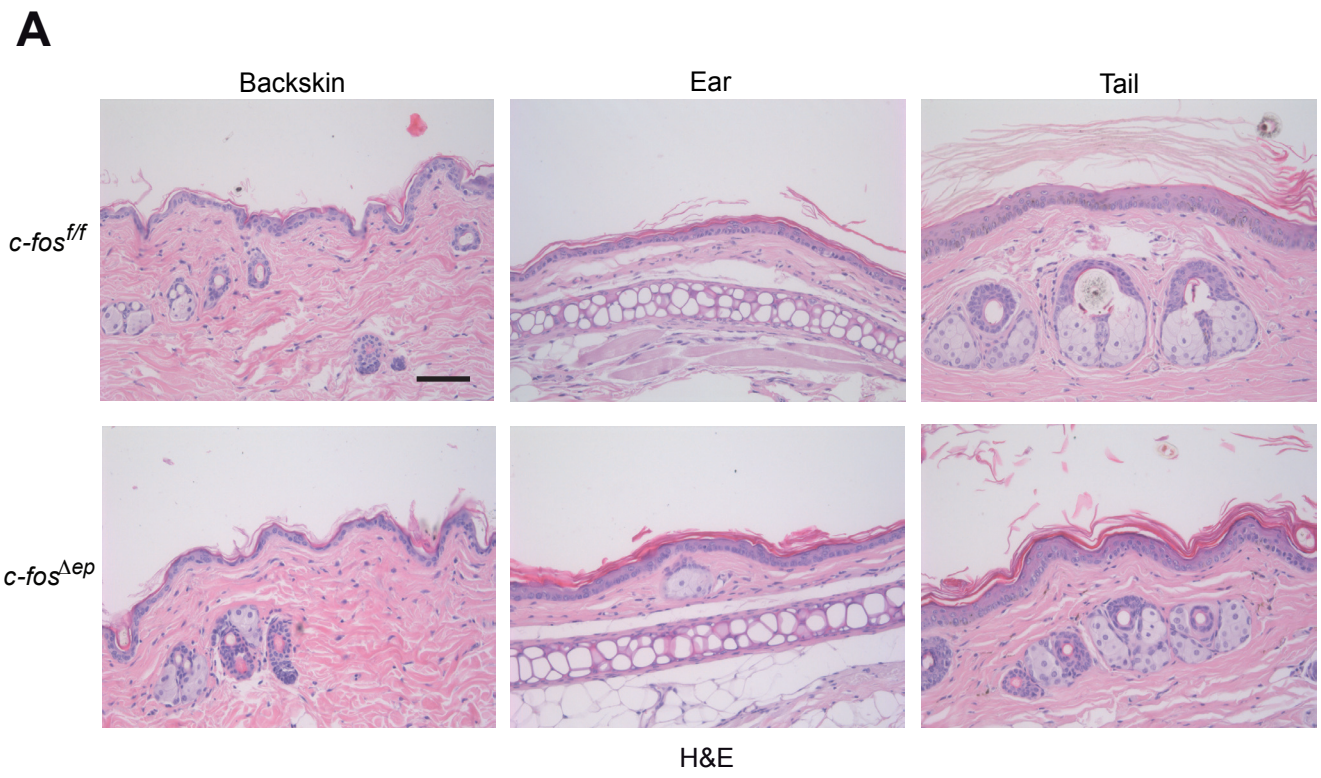
B) FACS analysis of EdU proliferation assay performed after c-Fos inhibition (10μM c-Fos inhibitor T-5224, 48h treatment) in a SCC cell line (SCCO12) with mutant *P53 m/m* alleles (dark/light grey bars) where *P53* was knocked-down by using shRNA (black/white bars). Results show that *P53* knock-down impairs the observed proliferation arrest observed in SCC cell lines that contain a functional *P53* allele upon c-Fos inhibition. *p < 0.05 is considered as significant. Experiment performed 2 times.

Supplemental Figure 15

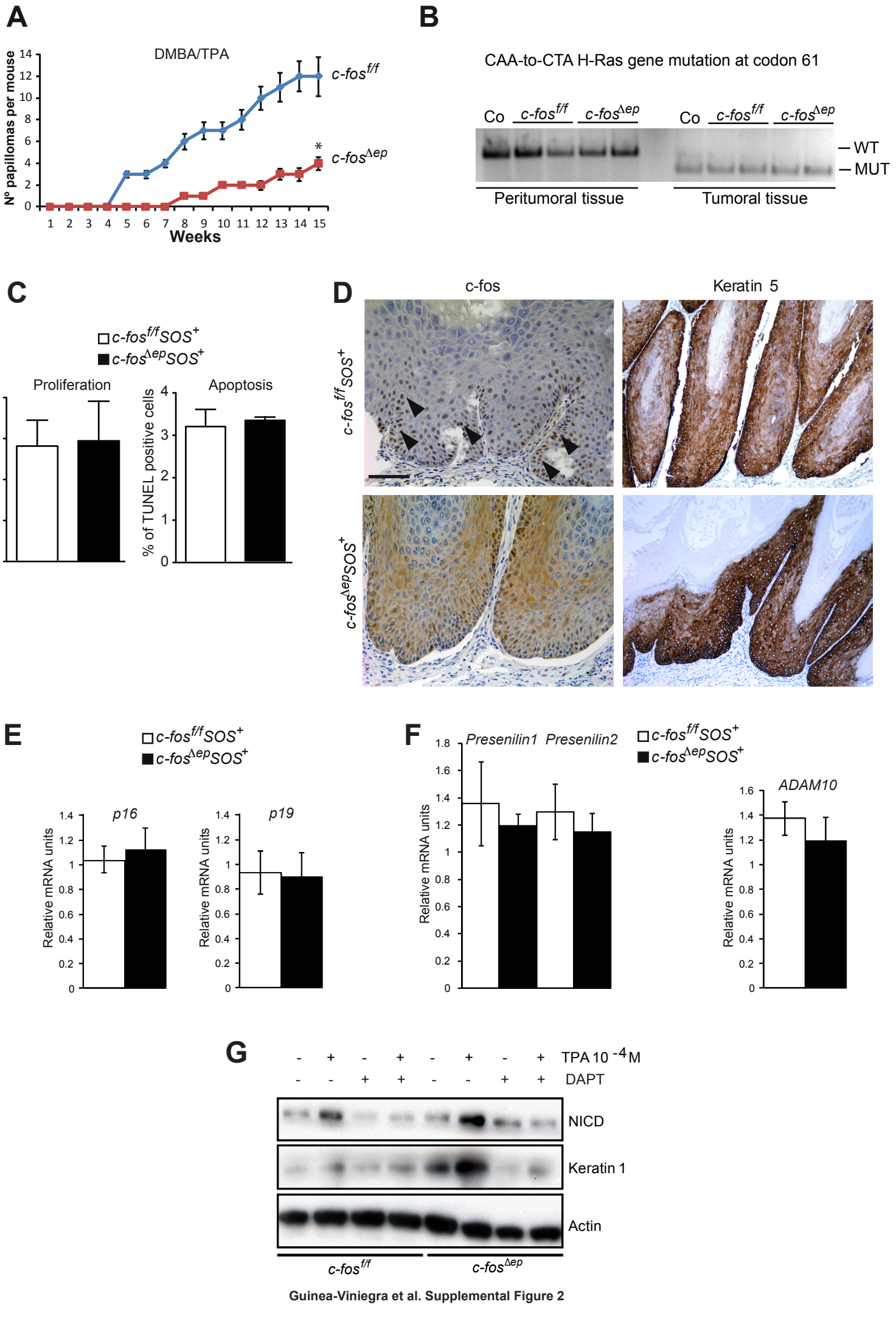
A) RT-qPCR analyses upon wild-type *P53* ectopic expression (light grey) in SCC cell lines with non-functional mutant *P53* (m/m) compared to control cells (black) shown increased expression of keratinocyte differentiation markers like *TACE, Notch1* and *Keratin 1*. *p < 0.05 is considered as significant. Experiment performed 2 times.

B) RT-qPCR analyses demonstrate induction of *Notch1, Keratin 1* and *Keratin 10* differentiation marker expression upon *TACE* over-expression (light grey) through transient transfection in SCC cell lines with non-functional *p53* alleles. *p < 0.05 is considered as significant. Experiment performed 2 times.

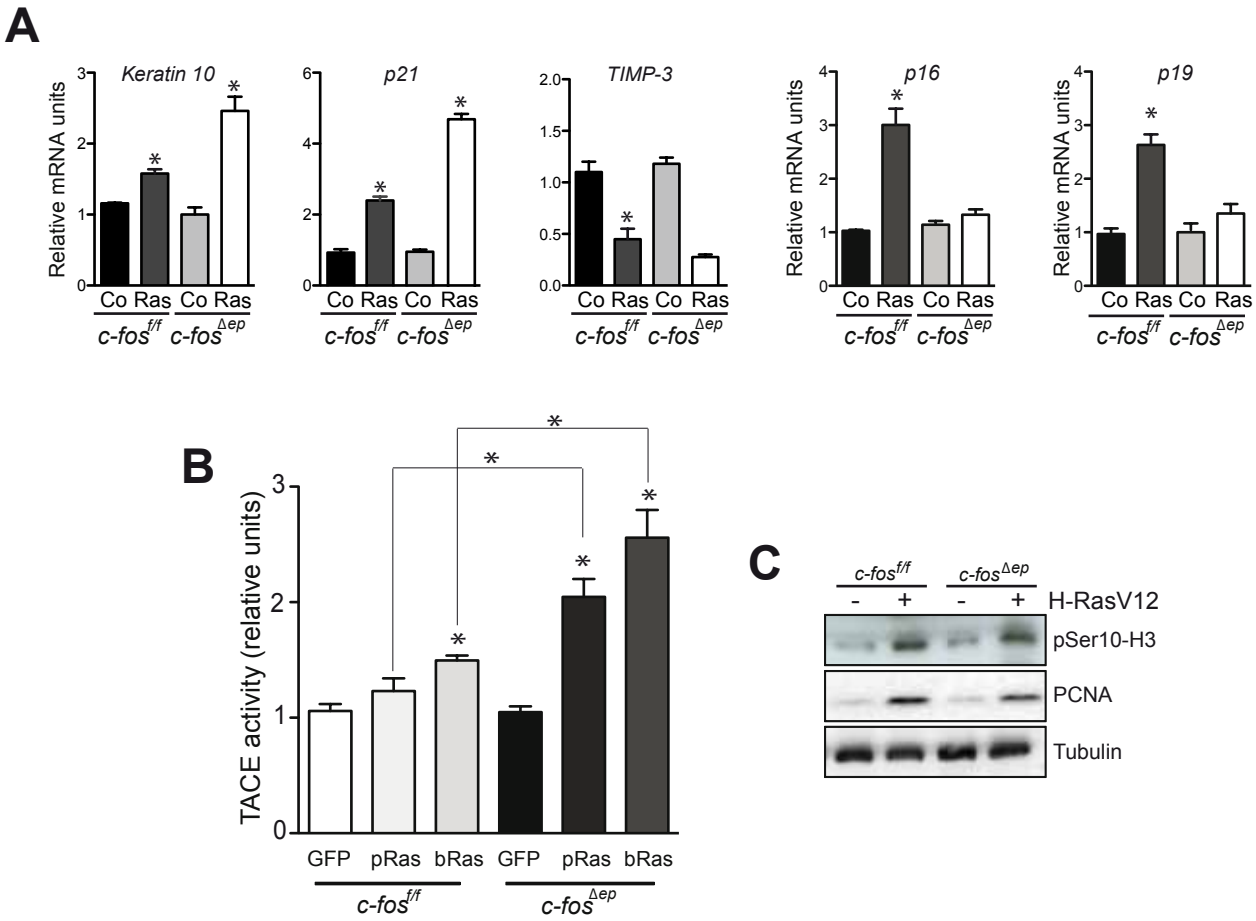
C) *TACE* over-expression, as quantified by Western blot against TACE, in the panel of SCC cell lines with non-functional *P53* alleles. Experiment performed 2 times.

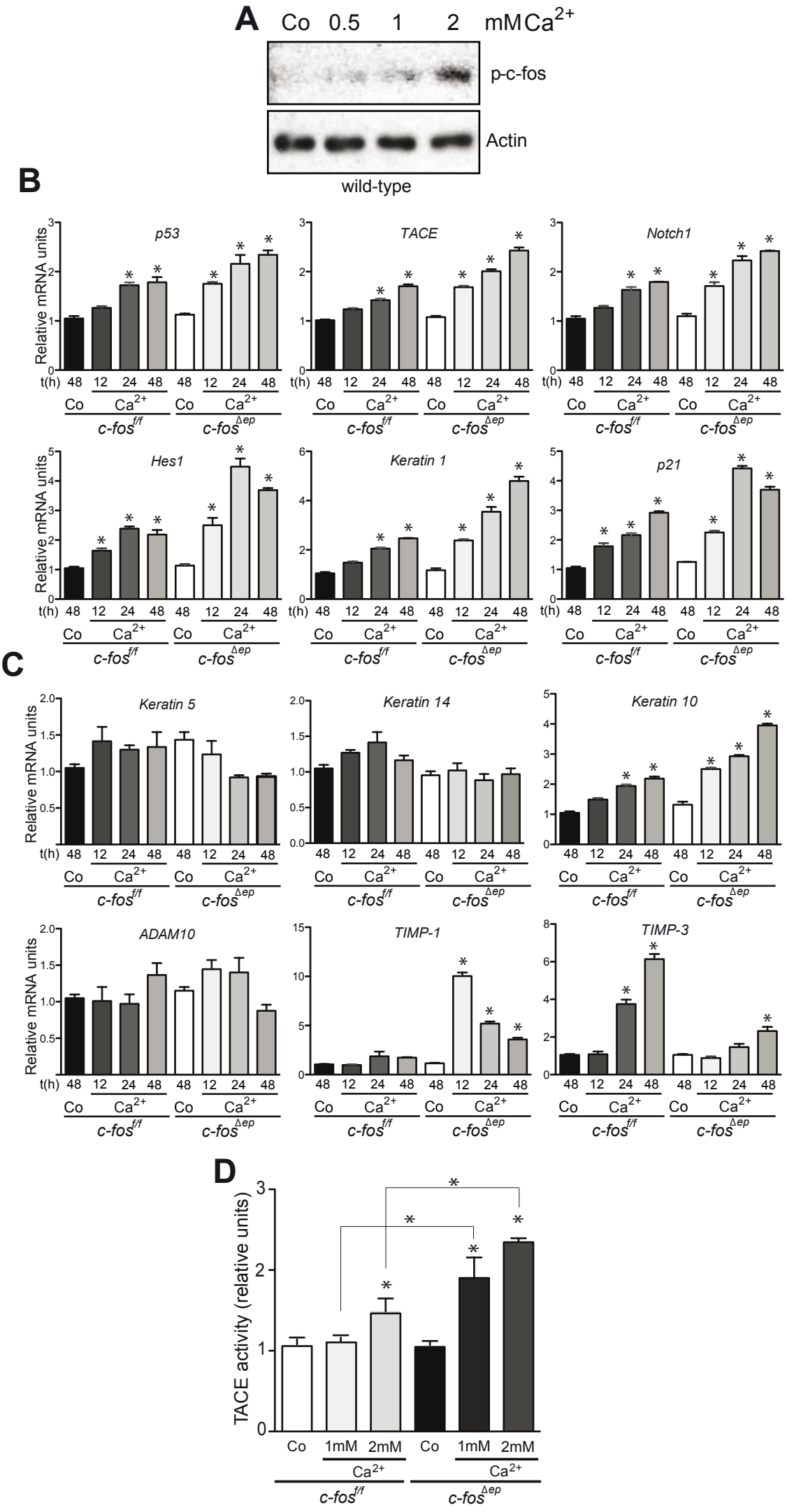


Guinea-Viniegra et al. Supplemental Figure 1

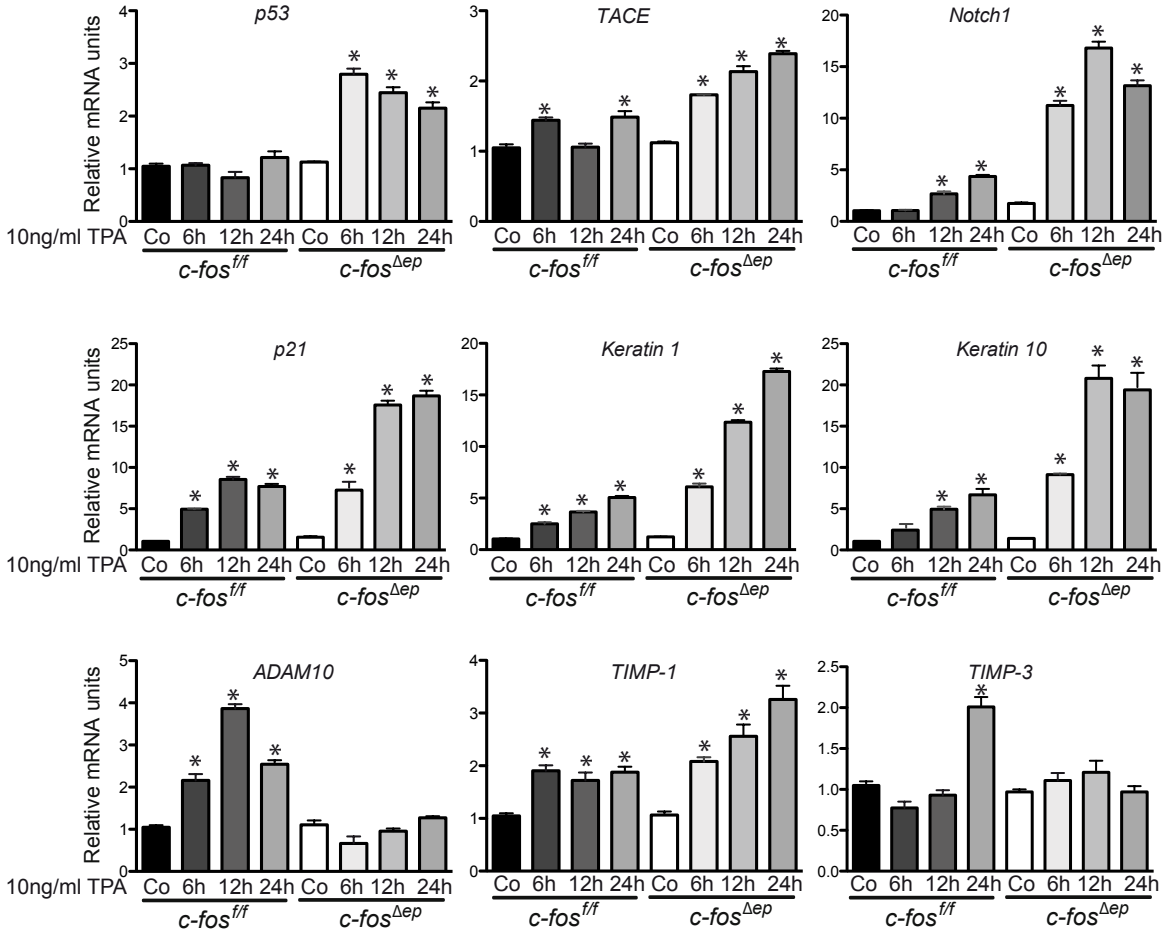


Guinea-Viniegra et al. Supplemental Figure 2

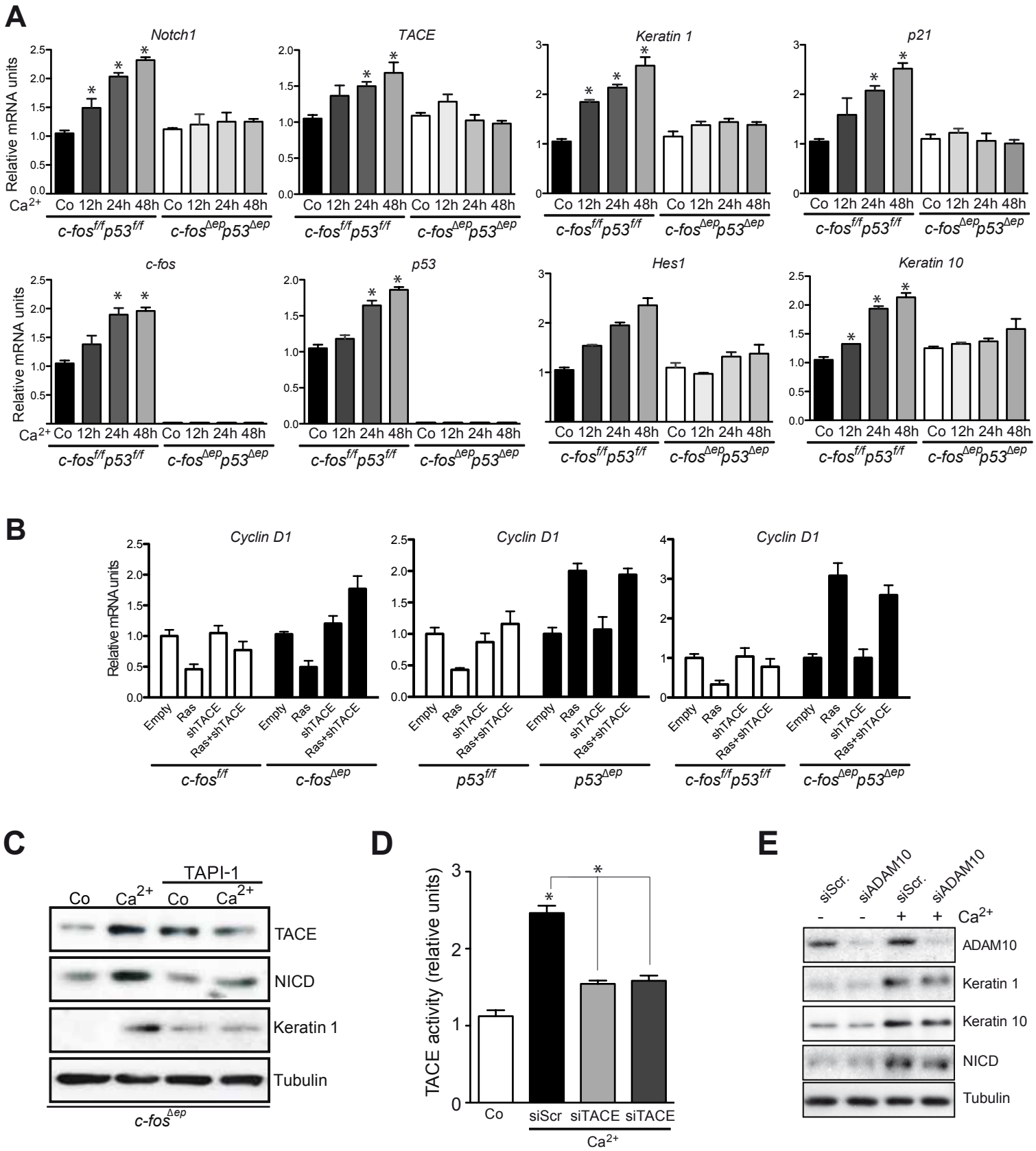

A
Keratin 10
p21
TIMP-3
p16
p19
Relative mRNA units
Co Ras Co Ras
c-fos f/f
c-fos Δep
B
TACE activity (relative units)
GFP pRas bRas GFP pRas bRas
c-fos f/f
c-fos Δep
C
c-fos f/f
c-fos Δep
- + - +
H-RasV12
pSer10-H3
PCNA
Tubulin



Guinea-Viniegra et al. Supplemental Figure 4



Guinea-Viniegra et al. Supplemental Figure 5


A
Notch1
TACE
Keratin 1
p21
c-fos
p53
Hes1
Keratin 10
Relative mRNA units
Ca2+ Co 12h 24h 48h Co 12h 24h 48h
c-fosf/fp53f/f
c-fosΔepp53Δep
B
Cyclin D1
Relative mRNA units
Empty
Ras
shTACE
Ras+shTACE
c-fosf/f
c-fosΔep
p53f/f
p53Δep
c-fosf/fp53f/f
c-fosΔepp53Δep
C
TAPI-1
Co
Ca2+
TACE
NICD
Keratin 1
Tubulin
c-fosΔep
D
TACE activity (relative units)
Co
siScr
siTACE
siTACE
Ca2+
E
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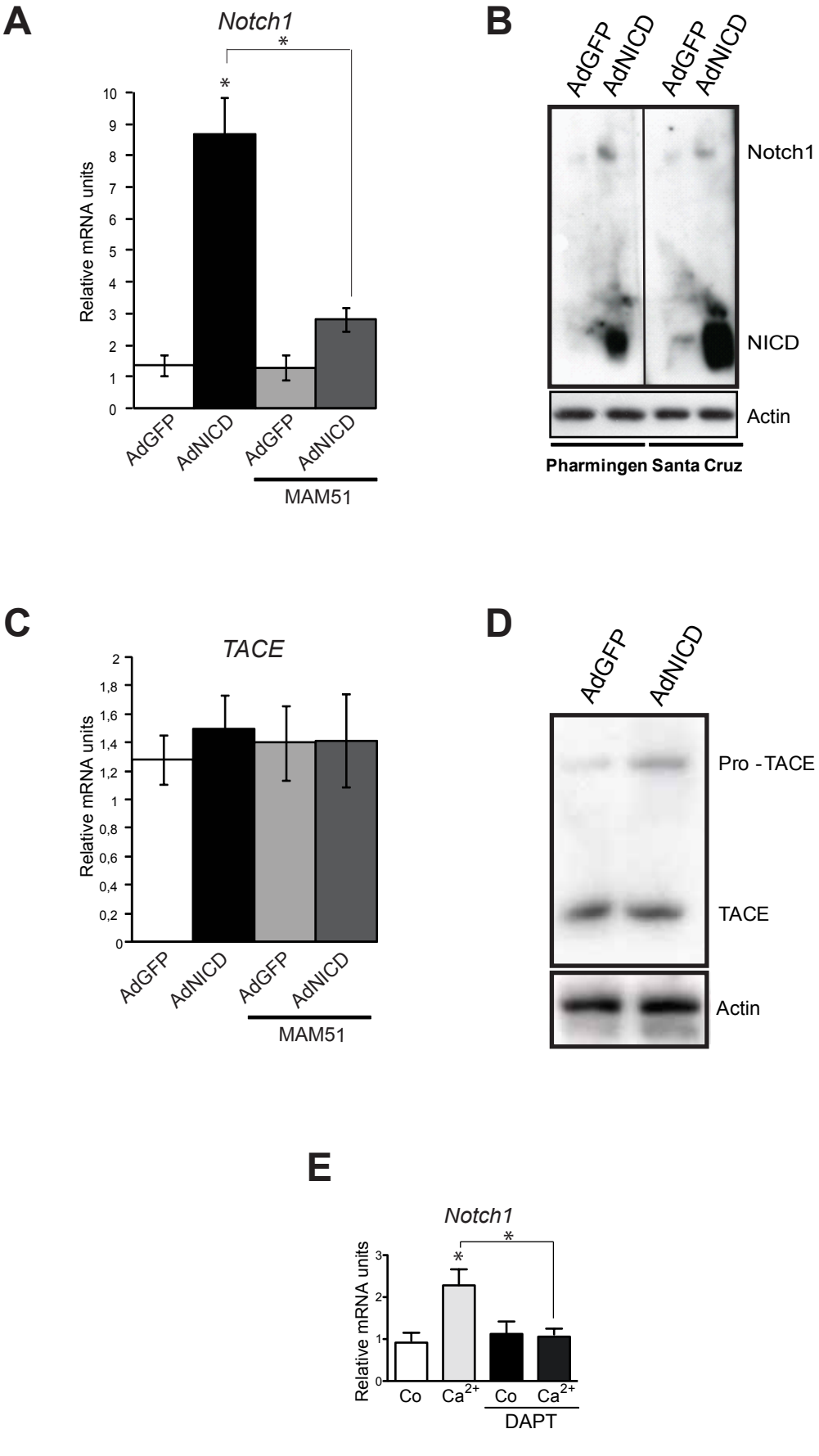
Keratin 1
Keratin 10
NICD
Tubulin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Notch1
Relative mRNA units
AdGFP
AdNICD
AdGFP
AdNICD
MAM51
B
AdGFP
AdNICD
AdGFP
AdNICD
Notch1
NICD
Actin
Pharmingen Santa Cruz
C
TACE
Relative mRNA units
AdGFP
AdNICD
AdGFP
AdNICD
MAM51
D
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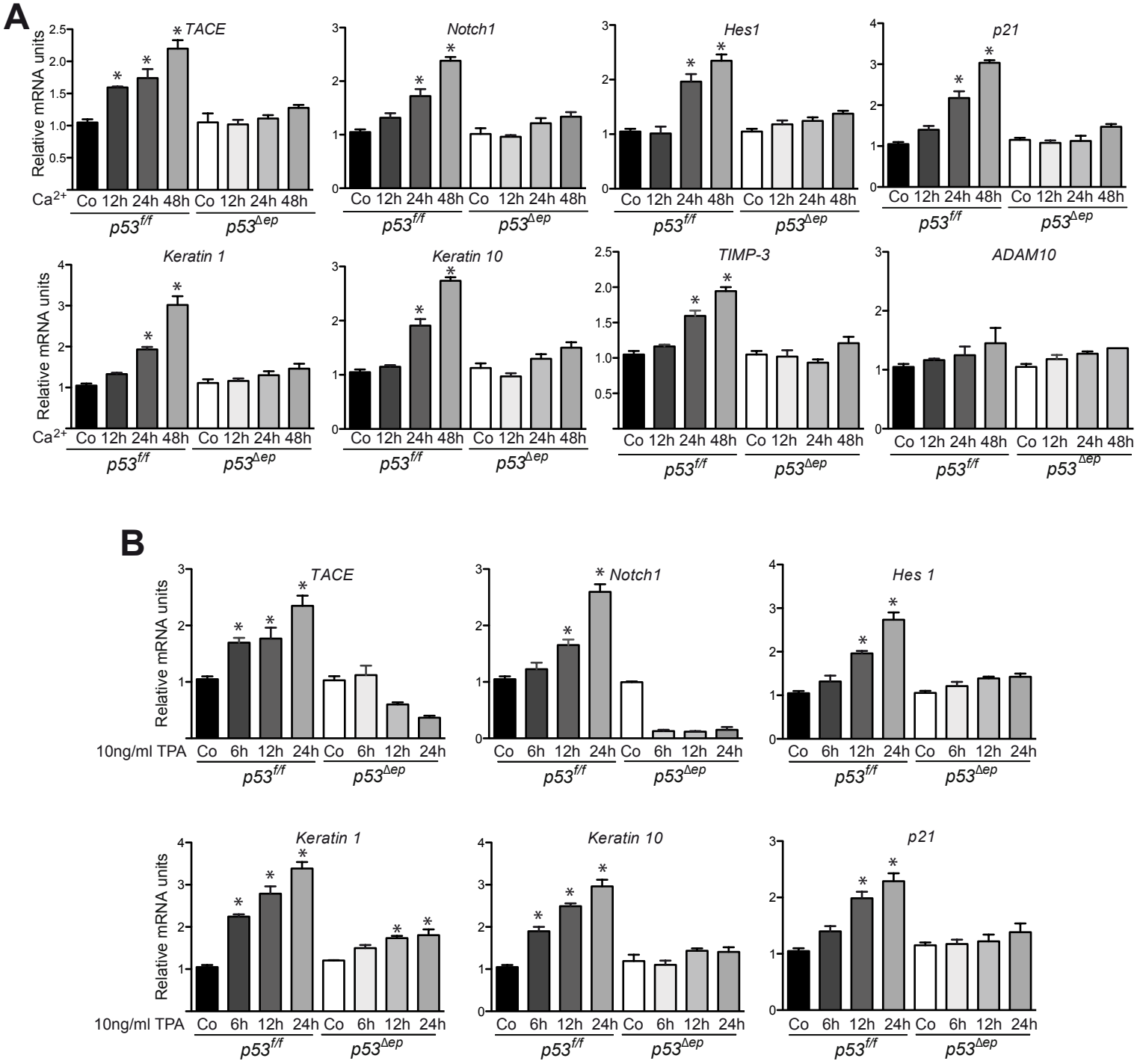
DAPT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ADAM10
Relative mRNA units
Ca2+
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p53f/f
p53Δep
B
TACE
Notch1
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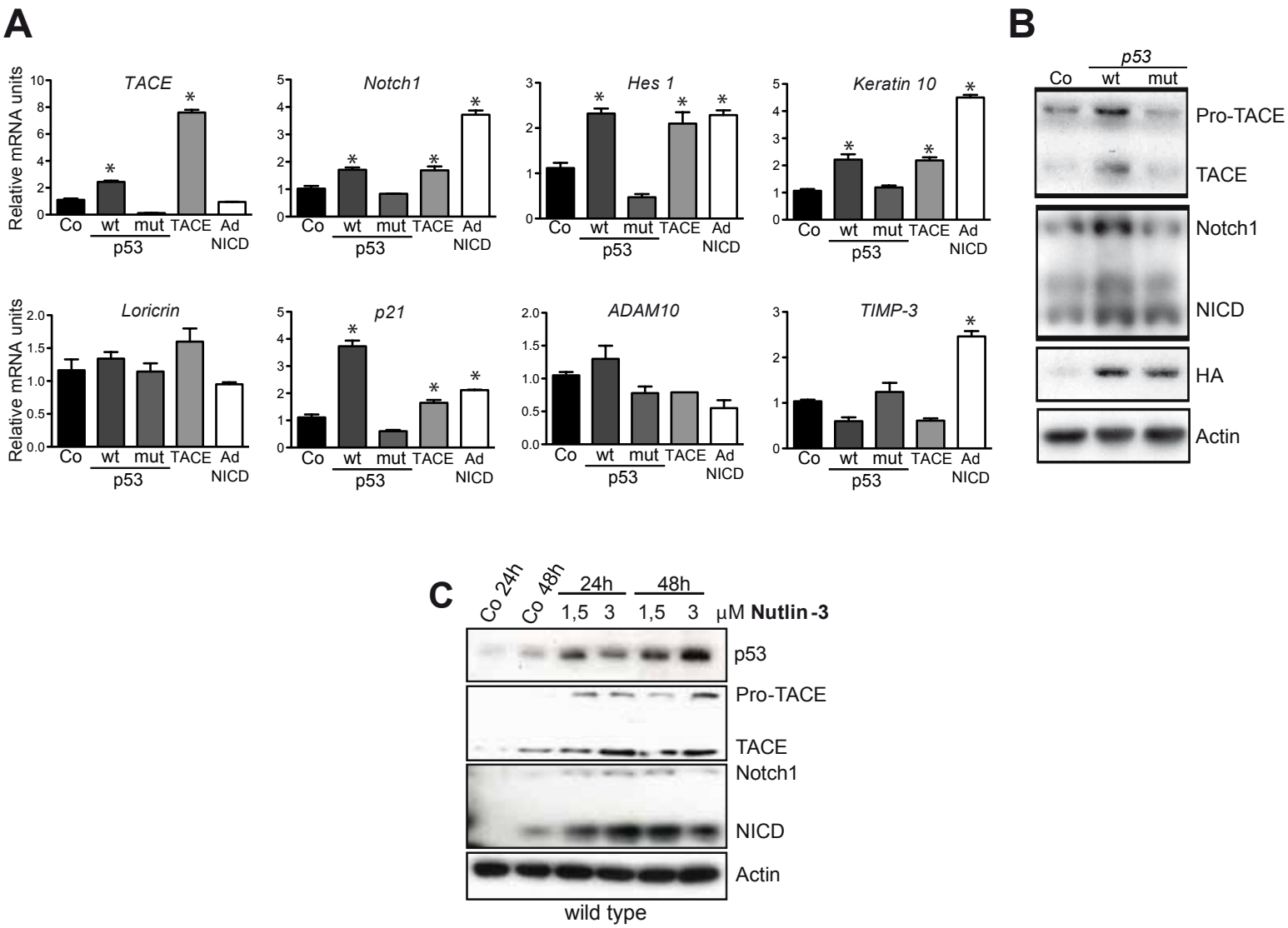
p53Δep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Keratin 10
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Relative mRNA units
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wt
mut
TACE
Ad NICD
p53
B
p53
Co
wt
mut
Pro-TACE
TACE
Notch1
NICD
HA
Actin
C
Co 24h
Co 48h
24h
48h
1,5
3
1,5
3
μM Nutlin-3
p53
Pro-TACE
TACE
Notch1
NICD
Actin
wild type
D
ADAM10
PSN1
PSN2
Relative mRNA units
Co
Ras
p53f/f
p53Δep
Keratin10
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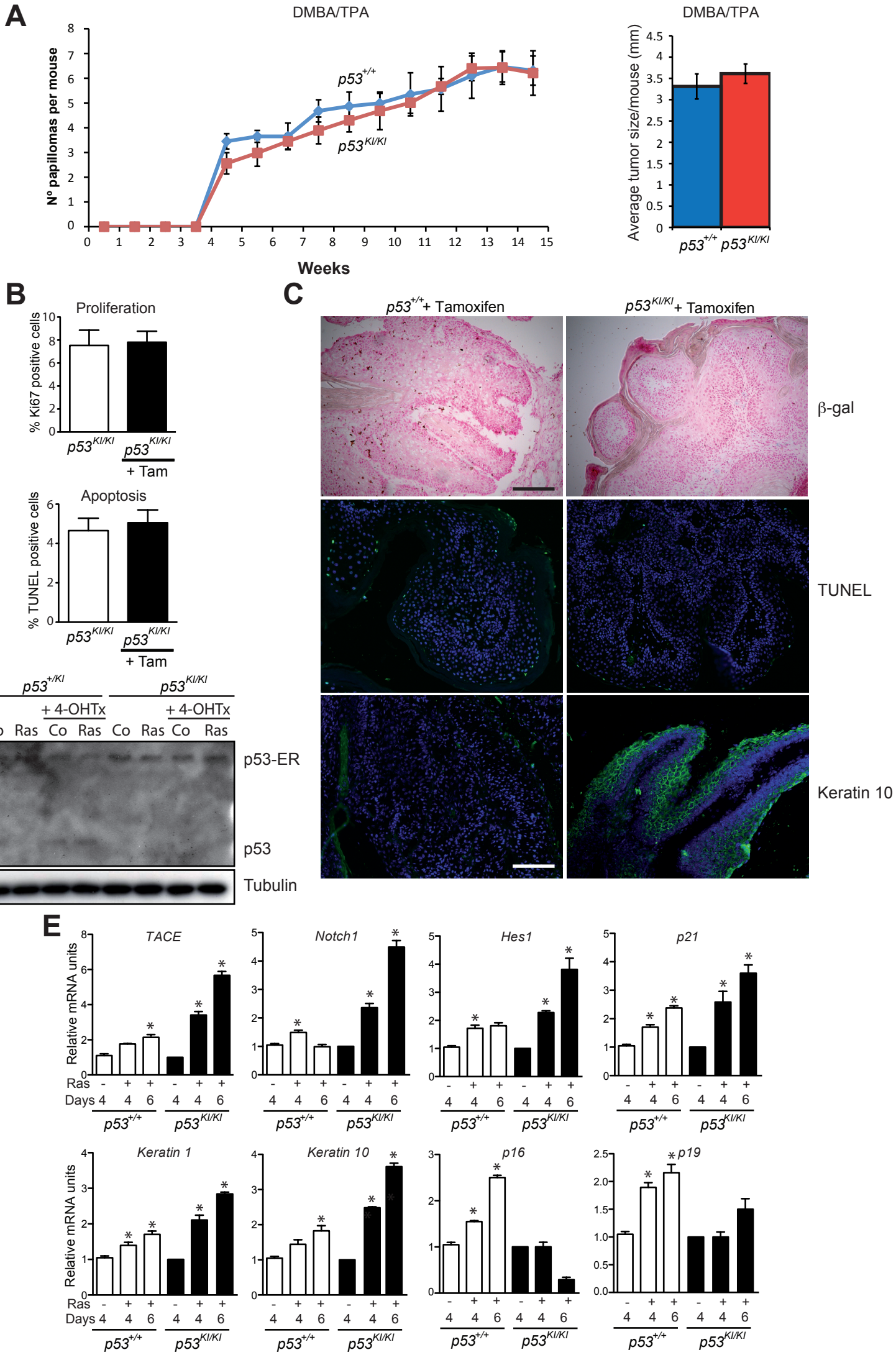
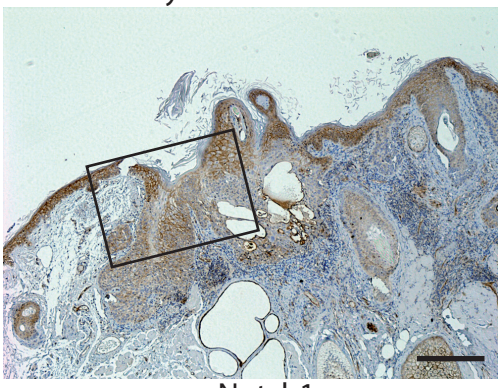


Guinea-Viniegra et al. Supplemental Figure 10

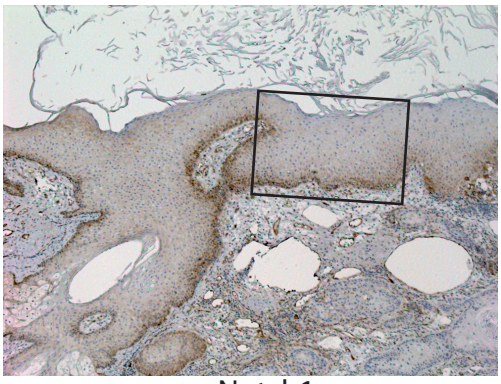
A

Poorly-differentiated SCC



Notch1

Well-differentiated SCC

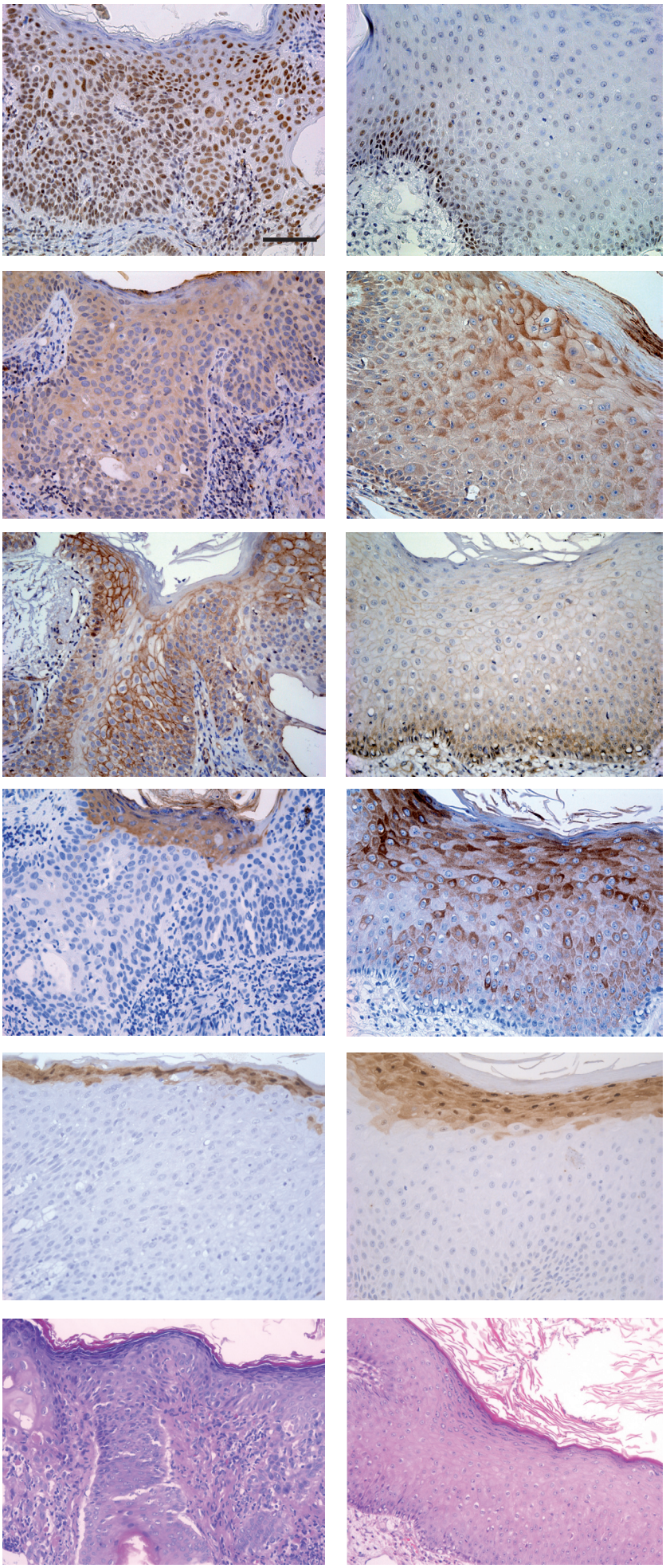


Notch1

B

Poorly-differentiated SCC

Well-differentiated SCC



c-Fos

TACE

Notch1

Keratin 1

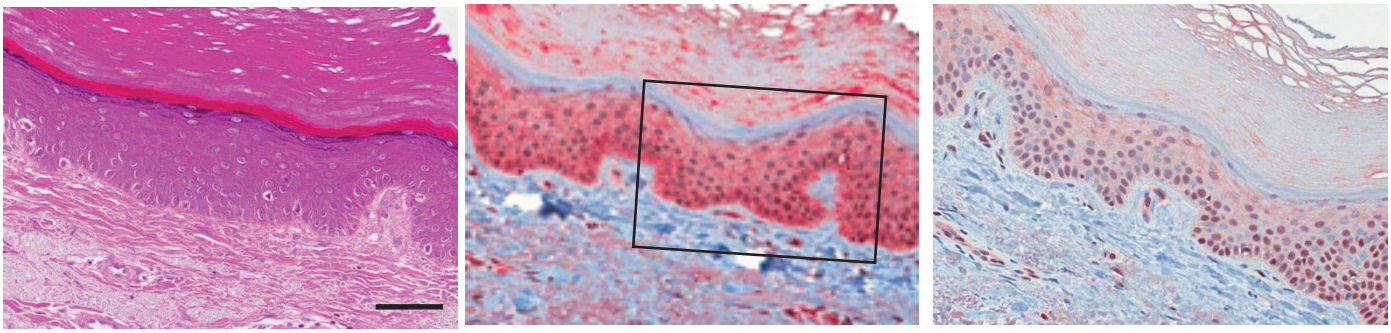
Loricrin

H&E

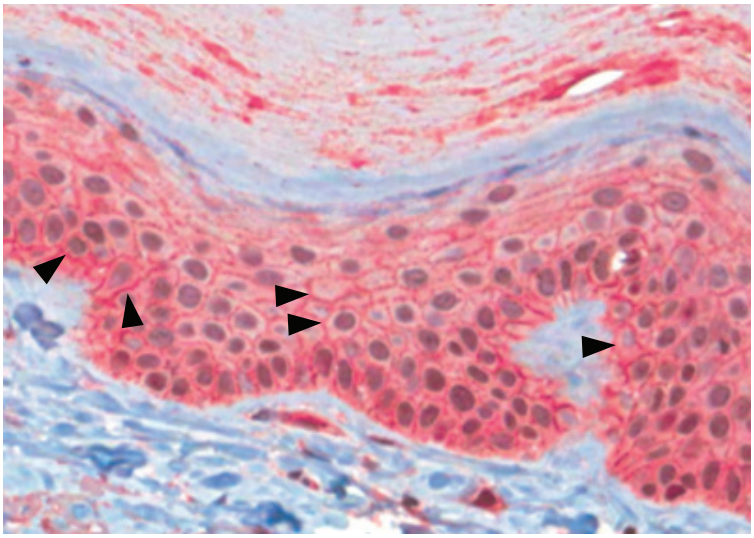
Guinea-Viniegra et al. Supplemental Figure 11

A

Normal skin - sun exposed



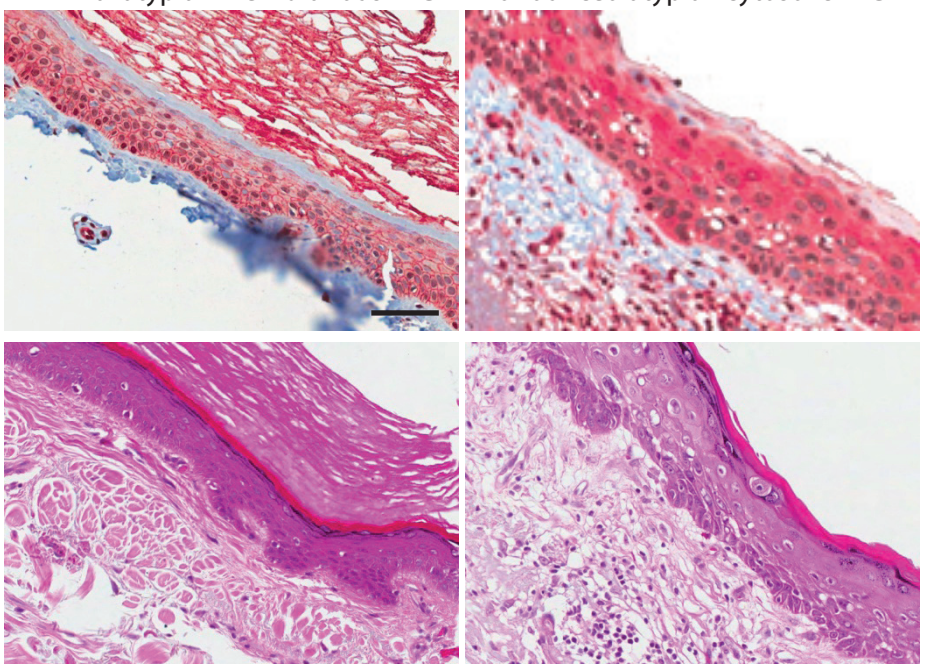
H&E TACE TACE + blocking peptide



TACE

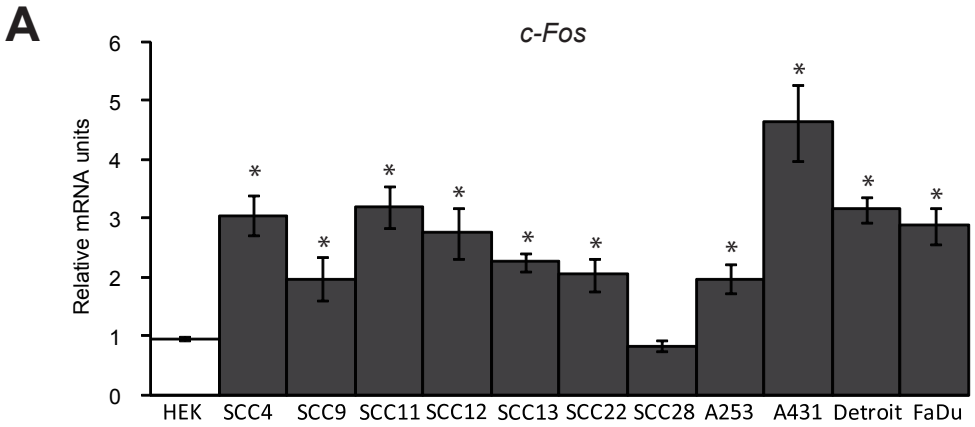
B

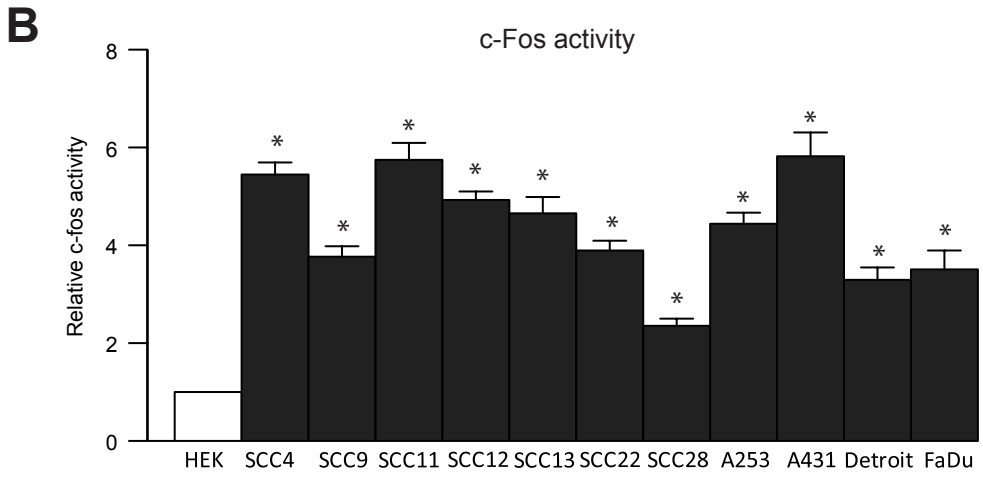
Minimal atypia - membranous TACE Pronounced atypia - cytosolic TACE

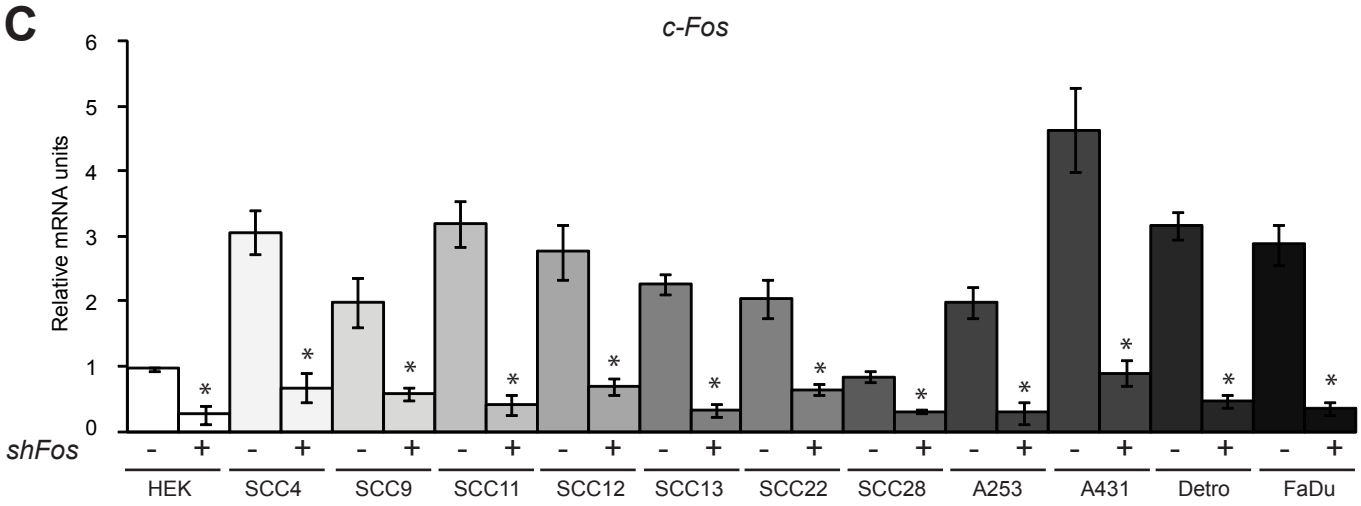


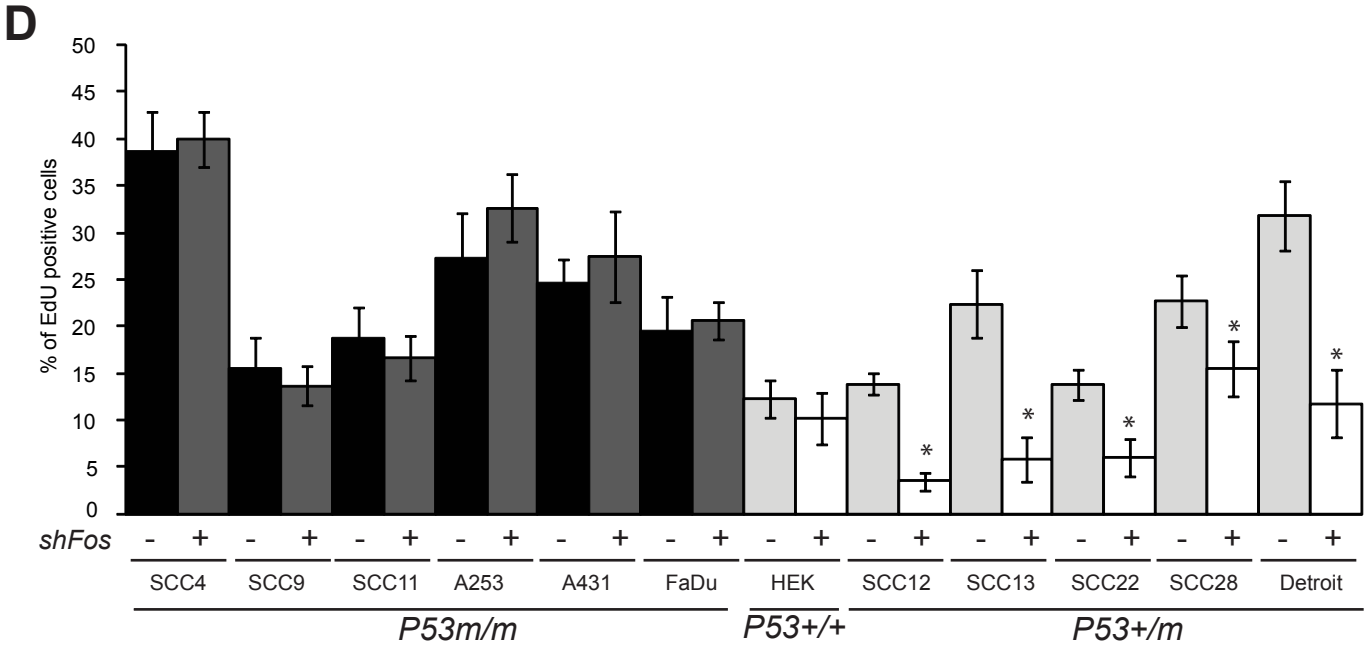
TACE

H&E

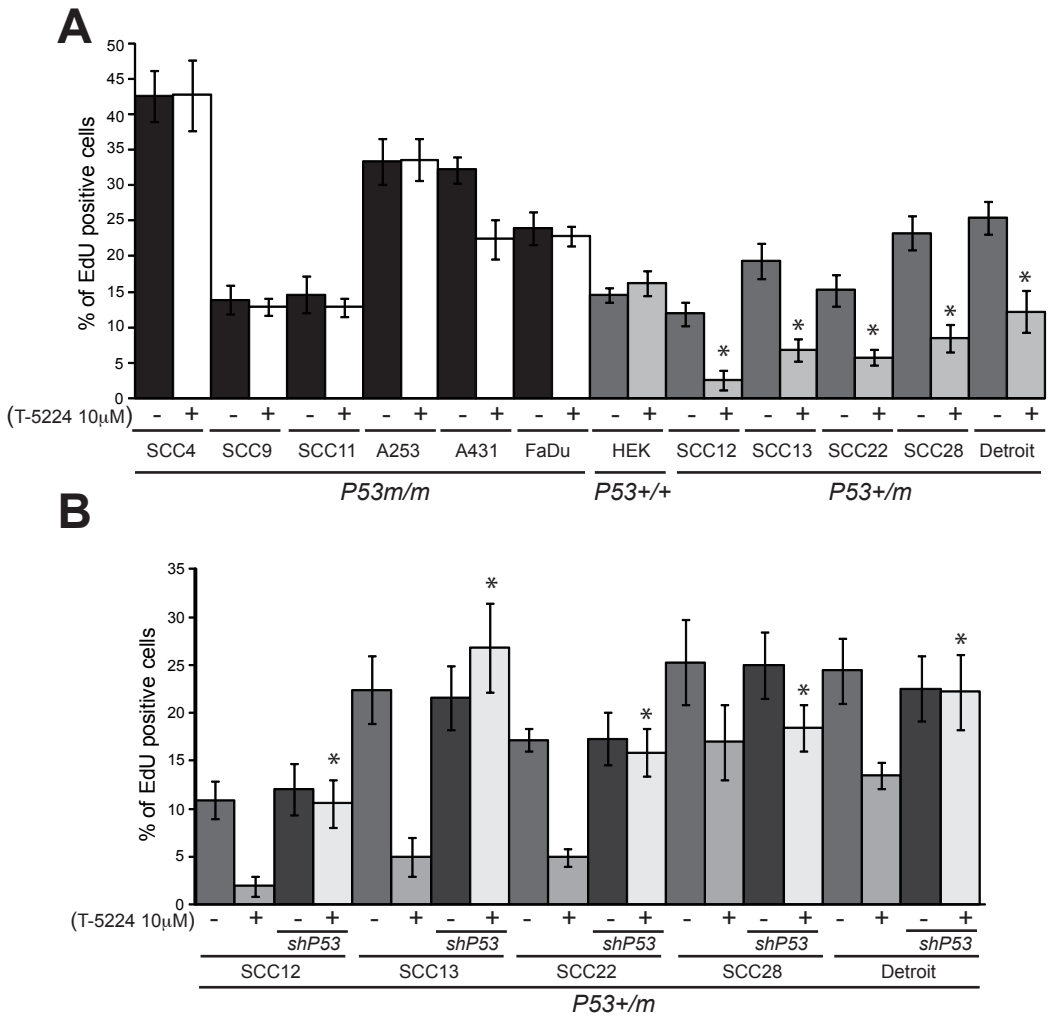


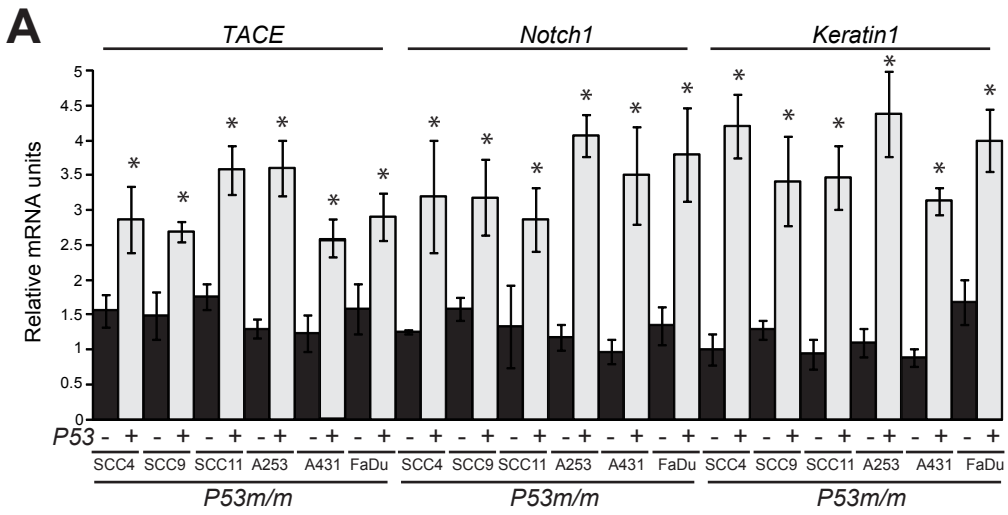


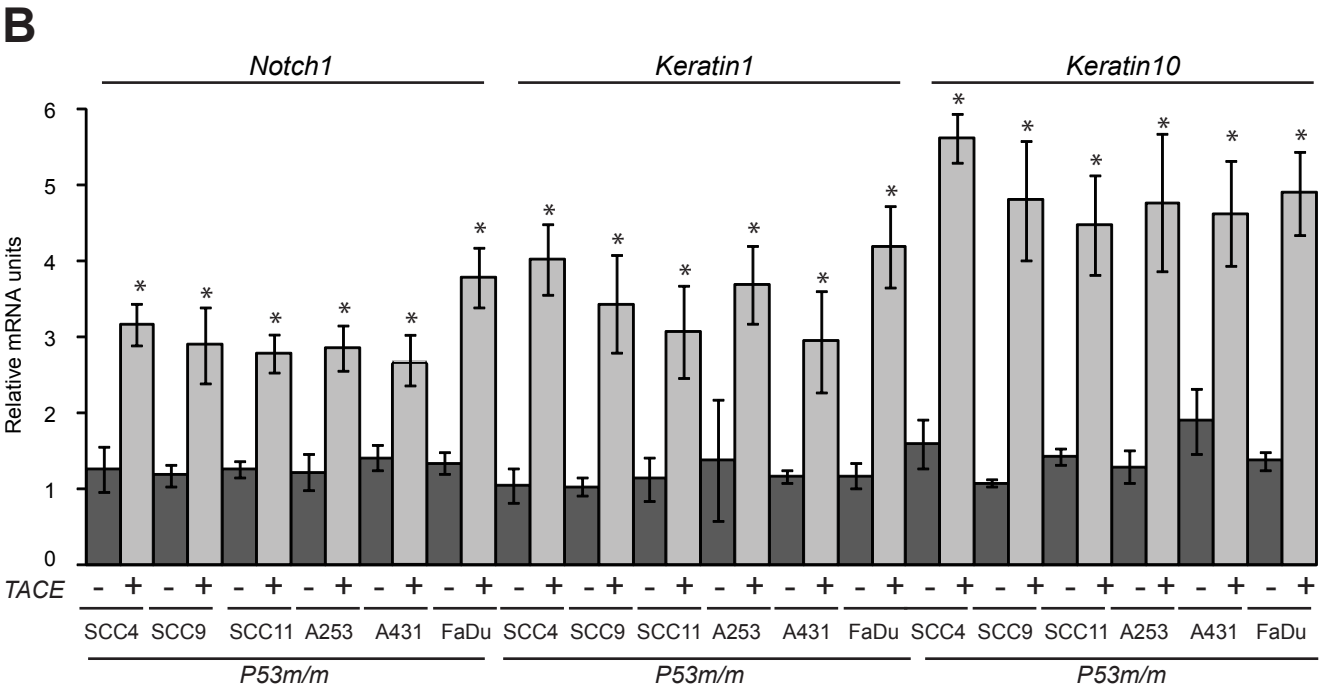


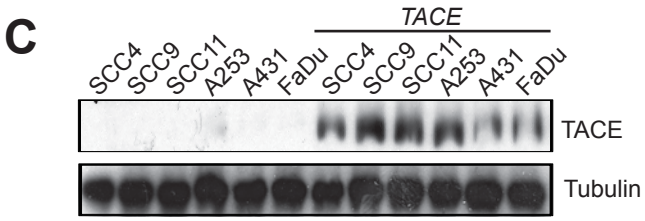


Guinea-Viniegra et al. Supplemental Figure 13


A
% of EdU positive cells
50
45
40
35
30
25
20
15
10
5
0
(T-5224 10μM)
- + - + - + - + - + - + - + - + - + - + - + - + - +
SCC4
SCC9
SCC11
A253
A431
FaDu
HEK
SCC12
SCC13
SCC22
SCC28
Detroit
P53m/m
P53+/+
P53+/m
B
% of EdU positive cells
35
30
25
20
15
10
5
0
(T-5224 10μM)
- + - + - + - + - + - + - + - + - + - + - + - + - +
shP53
shP53
shP53
shP53
shP53
SCC12
SCC13
SCC22
SCC28
Detroit
P53+/m


A
TACE
Notch1
Keratin1
Relative mRNA units
P53
SCC4 SCC9 SCC11 A253 A431 FaDu
P53m/m


B
Notch1
Keratin1
Keratin10
Relative mRNA units
TACE
SCC4 SCC9 SCC11 A253 A431 FaDu
P53m/m


C
TACE
SCC4 SCC9 SCC11 A253 A431 FaDu
TACE
Tubulin

Supplemental Table 1: Primary human SCCs: c-Fos and TACE expression

		Nuclear c-Fos		
Skin SCC subtype	**Nº Samples**	**Undifferentiated cells**	**Differentiated cells**	No expression
Well-differentiated tumor	28	25	0	3
Poorly-differentiated tumor	54	47	0	7
		Membranous TACE		
Skin SCC subtype	**Samples**	**Undifferentiated cells**	**Differentiated cells**	No expression
Well-differentiated tumor	28	2	24	2
Poorly-differentiated tumor	54	47	0	7

Results obtained from immunohistochemistry for c-Fos and TACE in well-differentiated and poorly-differentiated human SCCs.

Supplemental Table 2: Skin lesions: TACE expression and localization

		TACE localization	
Skin Lesion	**Nº Samples**	**Membranous TACE**	**Cytoplasmic TACE**
Sun-exposed skin	29	26	3
Actinic keratosis	7	6	1

Results obtained from immunohistochemistry for TACE in normal sun-exposed skin and actinic keratosis lesions.

Supplemental Table 3: Origin of keratinocyte-derived SCC cell lines, P53 status/mutation and qPCR analyses upon c-Fos shRNA knock-down

				RNA expression levels			
Cell line	**Tissue of origin**	**P53 status**	**P53 mutation**	**P53**	**TACE**	**Notch1**	**Keratin1**
HEK	Foreskin	+/+	None	1.12	1.03	1.22	1.09
SCCO12	Larynx	+/m	Substitution - c.1024C>T	4.25	3.88	4.36	5.3
SCCO13	Facial	+/m	Substitution - c.772G>A	3.54	2.98	4.03	3.69
SCCO22	Hypopharynx	+/m	Substitution - c.659A>G	5.35	4.78	4.84	5.06
SCCO28	Head & Neck	+/m	N. D.	2.41	2.5	2.11	2.31
Detroit 562	Pharynx	+/m	Substitution - c.524G>A	3.68	4.76	4.03	4.24
A431	Vulva	m/m	Substitution - c.818G>A	N.D.	1.34	1.25	1.16
SCCO4	Base of tongue	m/m	Substitution - c.451C>T	N.D.	1.12	1.32	1.08
SCCO9	Anterior tongue	m/m	Deletion - c.822_853del32	N.D.	1.38	1.18	1.31
SCCO11	Epiglotis	m/m	Substitution - c.725G>C	N.D.	1.18	0.98	1.14
FaDu	Pharynx	m/m	Substitution - c.743G>T	N.D.	0.98	0.99	1.21

Relative expression values from qPCR analyses of P53, TACE, Notch1 and Keratin 1 mRNA levels in SCC cell lines upon shRNA-mediated c-Fos knock-down. Relative expression to GAPDH. "+" corresponds to P53 wild-type allele, "m" corresponds to inactivating mutant P53 allele. N. D. indicates not determined. P53 mutation data from: www-p53.iarc.fr, p53.free.fr and www.sanger.ac.uk/genetics/CGP/CellLines (60).

Supplemental Methods

Mouse RT-qPCR primer sequences

ADAM10
Right primer sequence: ACGCTGGTGTTTTTGGTGTA
Left primer sequence: AATTCTGCTCCTCTCCTGGG
c-fos
Right primer sequence: CTGTCACCGTGGGGATAAAG
Left primer sequence: CCTACTACCATTCCCCAGCC
CyclinD1
Right primer sequence: GGGTGGGTTGGAAATGAACT
Left primer sequence: CTTCCTCTCCAAAATGCCAG
Hes1
Right primer sequence: GTCACCTCGTTCATGCACTC
Left primer sequence: TCTGGAAATGACTGTGAAGCA
Involucrin
Right primer sequence: ACTCCTGGTGCTGCTGTTTT
Left primer sequence: GATATGGCAGGGGATCAGAA
Keratin 1
Right primer sequence: CTAAGTTTTGGGTCCGGGTT
Left primer sequence: AGTTTGCCTCCTTCATCGAC
Keratin 10
Right primer sequence: ATCTGCCCCTTAAGGTCCTC
Left primer sequence: CGAGCTGGAGGGTAAAATCA
Loricrin
Right primer sequence: GAGGTCTTTCCACAACCCAC
Left primer sequence: TCCCTCACTCATCTTCCCTG
Notch1
Right primer sequence: TCTTACACGGTGTGCTGAGG
Left primer sequence: GAATGGAGGTAGGTGCGAAG
p16
Right primer sequence: TCGAATCTGCACCGTAGTTG
Left primer sequence: CGTGAACATGTTGTTGAGGC
p19
Right primer sequence: TAGTACCGGAGGCATCTTGG
Left primer sequence: ATCCTGACGCCCTGAACC
p21
Right primer sequence: ACGGGACCGAAGAGACAAC
Left primer sequence: CAGATCCACAGCGATATCCA
p53
Right primer sequence: AATGTCTCCTGGCTCAGAGG
Left primer sequence: CTAGCATTCAGGCCCTCATC
Presenilin1
Right primer sequence: ATTGGCTCAGGGTTGTCAAG
Left primer sequence: ACTTCCAGAATGCCCAGATG
Presenilin2
Right primer sequence: TCCATCTCTGGGTCATAGGG
Left primer sequence: AATGAGCCCATATTTCCTGC
TACE
Right primer sequence: ACCAACCACATGACCACTGA
Left primer sequence: TGACATCAAGTACCGAACGC
TIMP-1
Right primer sequence: TGGGGAACCCATGAATTTAG
Left primer sequence: ATCTGGCATCCTCTTGTTGC
TIMP-3
Right primer sequence: GCTTCTTTCCCACCACTTTG
Left primer sequence: GTGCTCCTGAGCTGTTGGA

Human RT-qPCR primer sequences

c-Fos
Right primer sequence: GTGACCGTGGGAATGAAGTT
Left primer sequence: CCGGGGATAGCCTCTCTTAC
Keratin 1
Right primer sequence: GTACCTGGTTCTGCTGCTCC
Left primer sequence: TGACCCTGAGATCCAAAAGG
Keratin10
Right primer sequence: TGTCGATCTGAAGCAGGATG
Left primer sequence: GAAAAGCATGGCAACTCACA
Notch1
Right primer sequence: GTTCTTGCAGGGGGTGC
Left primer sequence: GGTGAGACCTGCCTGAATG
P21
Right primer sequence: CATGGGTTCTGACGGACATC
Left primer sequence: TGCCGAAGTCAGTTCCTTGT
P53
Right primer sequence: TGTTTCCTGACTCAGAGGGG
Left primer sequence: GAGCGTGCTTTCCACGAC
TACE
Right primer sequence: GTCTGAGAGCAAAGAATCAAGC
Left primer sequence: TCTCCTATTCCTGACCAGCG