**Supplementary Data**

**Supplementary Methods:**

**High-resolution-melting (HRM) mitochondrial-DNA analyses of microdissected p53$^+$tumor cells**

PCR was carried out on the Light-Cycler-480 (Roche-Diagnostics, Basel, Switzerland) on a total volume of 20µL containing 5µl of genomic DNA (20ng), 15µL of Light-Cycler-480 High-Resolution-Melting-Master1X (Roche-Diagnostics, Basel, Switzerland) and 0.4µM of each forward and reverse primer. The PCR was performed with an initial denaturing step at 94°C for 10min., followed by 50 cycles of denaturing (94°C for 2seconds), annealing (57°C for 6seconds), and extension (72°C for 6seconds). After PCR, a post-amplification melting curve program was initiated by heating to 94°C for 10seconds, cooling to 55°C for 10seconds, and continuously increasing the temperature by 0.1°C/second to finally reach 95°C. Each PCR run included a no-template control, and each sample was run in triplicate. Post-amplification fluorescent melting curves were analyzed with the LC480-Gene-Scanning-software v1.2.9 (Roche-Diagnostics, Basel, Switzerland). Analyses were performed on Roche-software.

**Polymorphic microsatellite marker analyses of microdissected p53$^+$ tumor cells**

PCR was performed using 4ng DNA and the final PCR volume was 20$\mu$L. The PCR mix contained 1U Taq-Gold (Applied-Biosystems, Foster-City, CA, USA), 4mM $MgCl_2$, 0.2mM dNTP, 0.2$\mu$M labelled forward primers FAM (6-carboxyfluorescein) and 0.2$\mu$M non-labelled reverse primers. Thirty-five PCR cycles were performed. After denaturing, the PCR products were run on ABI-PRISM®-310-Genetic-Analyzer. The analyses of the migration data were performed with Genescan 3.1-software (Applied-Biosystems, Foster-City, CA, USA). All these tests were performed on triplicate.

**_TP53_ gene screening and sequencing in p53$^+$tumor cells of one skin SCC with cells of donor origin**

PCR was carried out on the Light-Cycler-480 (Roche-Diagnostics, Basel, Switzerland) on a total volume of 20µL containing 5µl of genomic DNA (20ng), 15µL of Light-Cycler-480 High-Resolution-Melting-Master1X (Roche-Diagnostics, Basel, Switzerland) and 0.4µM of each forward and reverse primer. The PCR was performed with an initial denaturing step at 94°C for 10min., followed by 50 cycles of denaturing (94°C for 2seconds), annealing (57°C for 6seconds), and extension (72°C for 6seconds). After PCR, a post-amplification melting curve program was initiated by heating to 94°C for 10seconds, cooling to 55°C for 10seconds, and continuously increasing the temperature by 0.1°C/second to finally reach 95°C. Each PCR run included a no-template control, and each sample was run in triplicate. Post-amplification fluorescent melting curves were analyzed with the LC480-Gene-Scanning-software v1.2.9 (Roche-Diagnostics, Basel, Switzerland).

For sequencing, 20µL of PCR products were purified using affinity columns (Qiagen-SA/ Courtaboeuf/France). Labelling was performed using BigDye®-Terminator-v1.1-Sequencing-Kit (Applied-Biosystems/Foster-City-CA/USA) in both forward and reverse sens. The reaction was run according to the following protocol: an initial denaturing step at 94°C for 3min; 25 cycles at 94°C for 10seconds, annealing temperature at 60°C for 20seconds; BDX-terminator purified products were run on a 16-capillary automated sequencer (ABI-PRISM®-3130xl-Genetic-Analyzer, Applied-Biosystems, Foster-City, CA, USA). SeqScape-Software v 2.5 (Applied-Biosystems, Foster-City, CA, USA) enabled nucleotide change determination.

**Genotyping of skin SCC and _KRAS_ mutation detection**

DNA from laser-microdissected p53$^+$tumor cells in skin SCC was studied using the molecular

inversion probe-based (MIP) genotyping system : OncoScan-Express (Affymetrix-Inc/Santa Clara, CA/USA) which determines genotype of 330,000 SNPs.

MIP probes are circularizable oligonucleotides, the two ends of which carry two sequences complementary to two sequences on the genome separated by one nucleotide (exactly where the variant to be genotyped is). After hybridization to the genomic DNA, the reaction is split into four tubes where a single nucleotide is added to each tube. Upon the addition of the nucleotide, the MIP probe is ligated, this only occurs in the tube including the nucleotide complementary to the allele on the genome, and this ligation turns the probe into a circle. This structure is then selected using exonucleases, and enables minimal'cross talk' between probes, to obtain high quality data from highly multiplexed assays Ultimately, these products are amplified and hybridized onto an Affymetrix microarray for product identification.

The assay interrogates 541 somatic mutations specific for cancer, with coverage of over 200 tumor suppressor and oncogenes, with a median spacing of 1 probe per 0.5 kb for the top 10 “actionable” tumor suppressor genes, a median spacing of 1 probe per 2 kb for the top 190-plus actionable oncogenes, and a median backbone spacing of 1probe per 9 kb.

Data analyses for mutation detection were performed on a Nexus-7 software (BioDiscovery/ El Segundo, CA/USA).

To detect a possible shared *KRAS* mutation in the kidney biopsy we used *KRAS* TaqMan mutation detection assay (TMDA) (Hs00001325_mu, Life Technologies, Carlsbad CA, USA) for PCR analysis of DNA extracted from the kidney biopsy. PCR was carried out on the CFX96$^{TM}$ real time system (Bio-RAd, Hercule CA, USA) on a total volume of 20-µL containing 5µl of genomic DNA (20 ng), 10µL of the Bio-Rad IQ$^{TM}$ supermix (Bio-RAd, Hercule CA, USA) and 1µl of Taqman mutation detection assay.The PCR was performed with an initial denaturing step at 95°C for 10 minutes, followed by 50 cycles of denaturing (95°C for 15 seconds), annealing and extension (60°C for 1 minute).

**HPV typing**

In skin SCC and kidney biopsy, we used immunohistochemistry and *in situ* hybridization to assess $p16^{INK4a}$ oncoprotein expression and low and high-risks oncogenic HPV.

Five µm-thick tissue sections of SCC from the recipient were stained using an indirect immunoperoxydase automated method (Discovery/Roche/Basel/Switzerland). Anti-human $p16^{INK4a}$ (clone JC8, Biocare-Medical, dilution 1/50), mouse antibodies were used as primary antibodies. Inhibition of endogenous peroxidase and blocking of nonspecific binding sites were systematically performed. Systematic controls were use of an irrevelant isotypic antibody as primary antibody, and absence of primary antibody.

HPV *in situ* hybridization staining was performed on 5 µm-thick sections of skin SCC and kidney biopsies, on a Benchmark-Ultra device (Roche Diagnostics/Basel/Switzerland) using Inform HPV III Family 16 Probe (HPV genotypes: 16, 18, 31, 33, 35, -39, -45, -51, -52, -56, -58, and -66) and Inform HPV II Family 6 Probe (HPV genotypes 6, 11) (Roche-Diagnostics/Basel/Switzerland) with 90 min antigen retrieval (Cell Conditioning 1/Roche-Diagnostics/USA) and 4 min protease digestion (ISH protease 3, Roche-Diagnostics/USA).

**Double anti-p53 and anti-CD24 immunostaining on kidney biopsies**

Double immunofluorescent stainings were performed on 5 µm-thick sections of kidney biopsies on a Discovery device (Roche-Diagnostics/Basel/Switzerland). After 60 min antigen retrieval (Cell Conditioning 1/Roche-Diagnostics/USA), the slides were incubated with : mouse monoclonal anti-human p53 antibody (Clone DO-7/Dako/France dilution 1/50) labelled with Apex-Alexa-Fluor-488 Antibody Labelling Kit (Life technologies/NY 14072/USA), and goat polyclonal anti-human CD24 (sc-7034, Santa Cruz Biotechnology/California, dilution 1/200) labelled with Apex-Alexa-Fluor-555 Antibody Labelling Kit (Life technologies/NY

14072/USA). After incubation, sections were mounted in Vectashield medium (Vector Labs/Burlingame/CA/94010 U.S.A).

Systematic controls included absence of primary antibody and use of an irrevelant isotypic primary antibody

Tissue sections were analyzed by two different pathologists (AJ/CL) on a motorized Z-axis microscope (BX-61-Olympus/Tokyo/Japan), using epi-fluorescent light. Microscope pictures were obtained with a digital camera ColorView-III using Olympus-SIS Cell F software (Olympus/Tokyo/Japan).

**Supplementary Table:** primers used for exons 5 to 8 *TP53* gene sequencing

| Exon | Forward sequence<br>5' -> 3' | Reverse sequence<br>3' ->5' | Amplicon size bp |
|---|---|---|---|
| **5** | GCCCTGACTTTCAACTCTGTCT | AAGACCTGCCCTGTGCAGCTGT | 100 |
| | TGCCCTCAACAAGATGTTT | CACATGACGGAGGTTGTG | 136 |
| | GGCCATCTACAAGCAGTCA | AGACGACAGGGCTGGTTG | 118 |
| **6** | CCTCACTGATTGCTCTTAG | AGTATTTGGATGACAGAAACAC | 92 |
| | TCTTATCCGAGTGGAAG | GTCTGGTTTGCAACTGG | 111 |
| **7** | TGGGCCTGTGTTATCTCCTA | GTAACAGTTCCTGCATGGG | 80 |
| | TTAGTTGTAACAGTTCCTGCATGG | TCAGGAGCCACTTGCCAATCTAT | 88 |
| **8** | TGCCTCTTGCTTCTCTTTTC | GAAGAGAATCTCCGCAAGAAAG | 128 |
| | GTGTTTGTGCCTGTCCT | GACAAGAAGCGGTGGAG | 137 |

Sequence of M13 tail: GTAGCCGAGCGGCCAGT

**Supplementary Figures:**

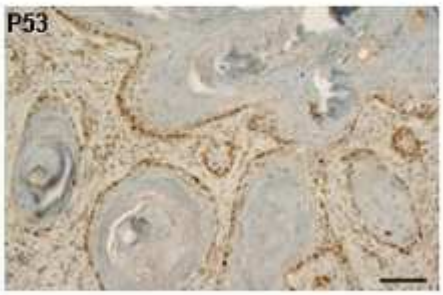
**Figure 1**: P53 positive immunostaining of nuclei from SCC basal layer and invasive areas. Indirect-immunoperoxidase, bar=300µm

**Figure 2**: Few p16 positive cells in SCC (A) compared with strong expression of p16 in positive control (B), bar=100µm

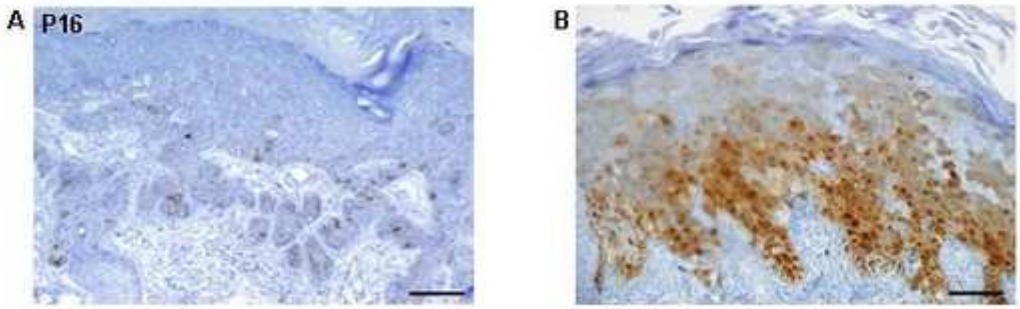
**Figure 3:** *KRAS* mutation in the skin SCC (OncoScan genotyping system)

**Supplementary Figures 1and 2**

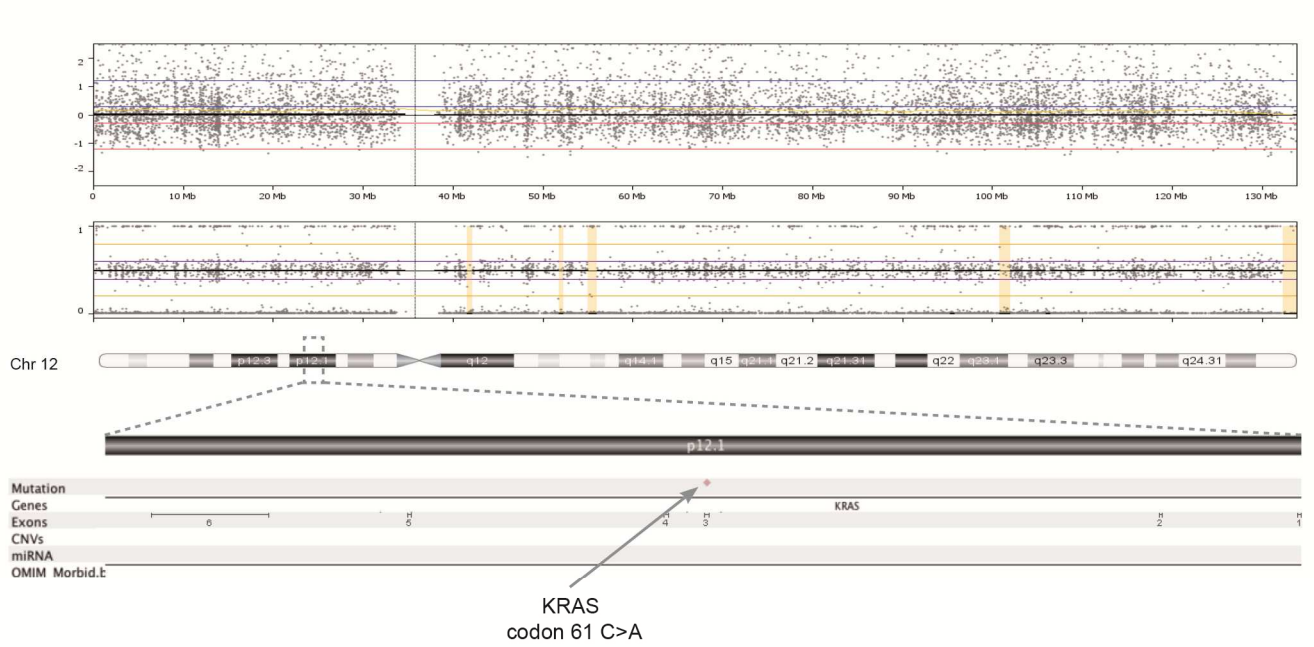
Supplementary Figure 1




Supplementary Figure 2



## Supplementary Figure 3




| Skin SCC point mutations (Oncoscan) | | | | Kidney tubules assay mutations | |
|---|---|---|---|---|---|
| Chromosome | Gene | Nucleotide mutation | Amino Acid Change | TMDA | Mutation statut |
| chr12 | KRAS | C>A | Q>K | Hs00001325_mu | WT |