	Sex	Age	Site	Immune status	*TP53*	CN-LOH	S-LOH	Copy gain
1.	M	76	scalp	+	**R248W**	2 (*2.4*), 14 (*0.1*), 17 (*24*)		9 (*0.7*), 11 (*1.4*)
2.	M	87	right ear	+	**E285K**	5 (*0.7*), 17 (*49*)		
3.	M	84	left dorsal hand	+	**E224** (splice-site)	6 (*1.6*), 13, 17 (*19*), 19		
4.	F	61	left cheek	+	**Y220N**	17 (*19*)	4, 5, 8, 15, 18	3, 8, 9 *(6.0)*, 15
5.	M	83	left cheek	+	H179Y, P278S	1, 9, 19, 22	2, 9, 21	
6.	M	85	right temple	+	P142N, H179Y	5, 6	9	
7.	M	58	left helix	-	E286K, T329I, E349*		3, 9, 10, 11	3, 11, 12
8.	M	63	lower lip	+	**none**[§]	1, 4, 9, 11		

Supplementary Table 2. Description of sample cases and genomic abnormalities. Chromosomal abnormalities that could be definitively called, by examination of both allele-specific SNP copy number and mutation frequencies, are listed by type (S-LOH denotes simple LOH, or deletion of one chromosome with no compensatory gain as seen in CN-LOH). In samples with CN-LOH at 17p (shaded), the ratio of heterozygous to homozygous mutations ρ, a measure of relative age of the duplication, is italicized in parentheses (for $n > 15$).

Bold-face notes loss of wild-type allele in *TP53* , for which all mutations are found in COSMIC; in samples 1-4 *TP53* mutation is duplicated through CN-LOH on chromosome 17p. [§]In sample 8, in which no mutation in *TP53* was seen, early, duplicated mutation was detected in *ATM* at G2024R, in the PI-kinase related domain.