## **Supplementary Figures**

Fig S1. The age and gender distribution of ACP and PCP group.

Fig S2. The correlation between somatic mutation number and age.

Fig S3. Structure alterations called in each patient.

Fig S4. Kataegis analysis of somatic mutations (SNVs and InDels) in each patient.

**Fig S5.** The proportion of 96 different extending possible base-pair substitutions of all SNVs identified in ACPs and 10 PCPs.

Fig S6. Schematics for genes listed in Fig. 3a indicating the location of the identified mutations.

Fig S7. IGV graphic report for the alignment of deletion in CTNNB1 CP111 tumor and normal blood sample.

**Fig S8.** IGV graphic report for the alignment of nonsense mutation of W425X in FBXW7 of CP22 tumor and normal blood sample.

Fig S9. CTNNB1 mRNA stability in CTNNB1-WT/Mut 293T/HCT116 cells.



Fig S1. The age and gender distribution of ACP and PCP group.



Fig S2. The correlation between somatic mutation number and age.



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![](_page_4_Figure_0.jpeg)

Fig S3. Structure alterations called in each patient.

![](_page_4_Figure_2.jpeg)

![](_page_5_Figure_1.jpeg)

![](_page_5_Figure_3.jpeg)

![](_page_6_Figure_1.jpeg)

Total somatic mutation - CP15

![](_page_6_Figure_3.jpeg)

![](_page_7_Figure_0.jpeg)

![](_page_7_Figure_1.jpeg)

![](_page_7_Figure_3.jpeg)

![](_page_8_Figure_1.jpeg)

![](_page_8_Figure_3.jpeg)

![](_page_9_Figure_0.jpeg)

![](_page_9_Figure_1.jpeg)

![](_page_9_Figure_3.jpeg)

![](_page_10_Figure_1.jpeg)

![](_page_10_Figure_3.jpeg)

![](_page_11_Figure_1.jpeg)

![](_page_11_Figure_3.jpeg)

![](_page_12_Figure_1.jpeg)

![](_page_12_Figure_3.jpeg)

![](_page_13_Figure_1.jpeg)

![](_page_13_Figure_3.jpeg)

![](_page_14_Figure_0.jpeg)

Total somatic mutation - CP34

![](_page_14_Figure_2.jpeg)

Total somatic mutation - CP111

![](_page_15_Figure_1.jpeg)

![](_page_15_Figure_3.jpeg)

![](_page_16_Figure_1.jpeg)

![](_page_16_Figure_3.jpeg)

![](_page_17_Figure_1.jpeg)

![](_page_17_Figure_2.jpeg)

![](_page_17_Figure_3.jpeg)

**Fig S5.** The proportion of 96 different extending possible base-pair substitutions of all SNVs identified in 16 ACPs and 10 PCPs.

![](_page_18_Figure_0.jpeg)

Fig S6. Schematics for genes listed in Fig. 3a indicating the location of the identified mutations.

![](_page_19_Figure_0.jpeg)

Fig S7. IGV graphic report for the alignment of deletion in CTNNB1 CP111 tumor and normal blood sample.

	chr4														^			
	p16.2	p15.33	p15.31 p15.2	p15.1	p14 p13 p1	2 q11 q12	q13.1 q13.	2 p13.3 q21.	21 q21.3	q22.2 q2	23 q24	q25	q26 q27 q28.1	q28.3 q31.1	q31.22	q31.3 q32.1 q	32.2 q33 q34.2	q35.1
		153,249,490 bp			р		1	153,249,500 bp		1			153,249,510 bp				153,249,520 bp	
CP33T_coverage	[0 - 42]	1 1								-								^
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RefSeq Genes										FBXW7				*				

**Fig S8.** IGV graphic report for the alignment of nonsense mutation of W425X in FBXW7 of CP22 tumor and normal blood sample.

![](_page_20_Figure_0.jpeg)

**Fig S9. CTNNB1 mRNA stability in CTNNB1-WT/Mut 293T/HCT116 cells. a-b** CTNNB1 mRNA stability in CTNNB1-WT/Mut 293T/HCT116 treated with 5 μg/mL actinomycin D at different time points. Actin mRNA was used as a negative control.