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MEDICINE
PATHOLOGY

The Genetic Profiles of Pancreatic Cancter

Some of the most commonly used cell lines of pancreatic cancer are listed below with their profile of mutations:

Cell Line	K-Ras	AKT2 19q	9p LOH	p16 HD	p16 Mut	13q LOH	BRCA2 Mut	17p LOH	p53 Mut	MKK4 HD	MKK4 Mut	18q LOH	DPC4 HD	DPC4 Mut	DCC HD
AsPc1	12 Asp	Ampl.	LOH	–	2 bp del.	LOH	–	LOH	1 bp del.	HD	–	LOH	–	100 Thr	–
BxPc3	–	Poss. Ampl.	LOH	HD		LOH	–	LOH	220 Cys	–	–	LOH	HD		–
CAPAN1	12 Val	–	LOH	HD		LOH	1 bp del.	LOH	159 Val	–	221 Stop	LOH	–	343 Stop	–
CAPAN2	12 Val	–	LOH	–	6 bp ins.	–	–		–	–	–	LOH	–	–	–
CFPAC1	12 Val	–		–	Meth	LOH	–	LOH	242 Arg	–	–	LOH	HD		–
COLO 357				–	Meth				–			LOH	HD		–
HS766T	61 His	–	LOH	–	Splicing	–	–	–	rearr.	–	–	LOH	HD		–
MiaPaCa2	12 Cys	–	LOH	HD		–	–	LOH	248 Trp	–	–	LOH	–	–	HD
Panc1	12 Asp	Ampl.	LOH	HD		LOH	–	LOH	273 His	–	–	LOH	–	–	–
Su86.86	12 Asp	Ampl.	LOH	HD		LOH	–	LOH	245 Ser	–	–	LOH	–	–	–

"-" refers to absence of the genetic alteration
Blank spaces refer to undetermined data points.
In tumors with homozygous deletions ("HD"), the gene is not present, and mutations ("Mut") cannot be assayed.
For small intragenic deletions ("del.") and insertions ("ins."), the number of basepairs ("bp") involved is given.
"Splicing" refers to a splice site mutation predicted to interfere with correct splicing of the precursor RNA to form the mature mRNA.
For K-ras, p53, and DPC4, the codon and amino acid change are shown.
AsPc1 is reported to have constitutive TCF4 activity, but no mutation of beta-catenin.
Hs766T is reported to also have constitutive TCF activity along with a mutation of the beta-catenin gene, but newly purchased samples of this cell line have shown no mutation in beta-catenin.
The amplicon on 19q13 contains the AKT2 gene in three lines, but there is a report of an amplicon of 19q13 in BxPc3 that lacks AKT2 amplification .
COLO 357 refers to the cell line available from ECACC.
Methylation of the p16 promoter ("Meth.") is associated with absence of RNA expression.
Hs766T is reported to have a rearrangement ("rearr.") of one allele of p53, and the other allele appears to have a deletion involving exons 2 through 4, resulting in an inactive p53 gene.
No alterations of the LKB1/STK11 gene are reported in these cell lines.
Additional abnormalities are known, such as the homozygous deletions of 8p22 and 18q22 in MiaPaCa2, but it is not clear that these affect any key genes for cancer development.