Abstract
**Abstract:**

DNA copy number alterations are expected to harbor deregulated oncogenes and tumor suppressor genes (TSGs) important for maintaining the tumor phenotype. For the current study, we selected two frequent homozygous deletions of pancreatic cancer (PaCa); one located at 6q25.3 and the other at 18q23. Analysis of genes located within the 6q25.3 deletion revealed single annotated gene *ARID1B*. *ARID1B* is part of the human SWI/SNF complex. Analysis of 18q23 deletion identified *PARD6G* which is part of par polarity complex.

Genomic analysis of the current study confirmed *ARID1B* gene deletion in MiaPaCa2 cells, absence of DNA sequence change in PaCa cell lines, no difference in between tumor and normal in terms of the presence of alternative transcript isoforms of *ARID1B*. Loss of *ARID1B* expression in PaCa cell lines were confirmed. Interstingly, this study identified a probable transcription repression of *ARID1B* in PaCa cell line which was confirmed by Bisulfite sequencing. Functional analysis of *ARID1B* in MiaPaCa2 cells confirmed no effect of *ARID1B* on growth rate, however colonogenicity was severely compromised in presence of *ARID1B* indicating that forced expression of *ARID1B* could alleviate tumor-related phenotype in the pancreatic cancer cell lines (Khursheed et.al, 2013).

Analysis of another recurrent deletion located at 18q23 revealed *PARD6G* gene which is a component of par polarity complex and is expected to regulate cell motility. Analysis of aCGH data (Bashyam et.al 2005) revealed possible single copy loss in PaCa cell lines. Mutation screening of *PARD6G* in PaCa cell line harboring probable single copy loss identified a six bp insertion in putative promoter of *PARD6G* in CFPAC-1 cell line. Bisulphite sequencing of *PARD6G* revealed partial methylation of CpG islands spanning 1st intron region. Given the importance of transcription regulation of TSG in cancer progression,
we studied *PARD6G* putative promoter region by construction of deletion constructs of *PARD6G* promoter. Luciferase reporter assay using these deletion constructs in MiaPaCa2 revealed a possible transcription activator and repressor region in putative promoter. Few transcription factor binding elements were observed in activator as well as repressor region of *PARD6G* promoter in bioinformatics prediction analysis which can be further characterized through DNA-protein interaction studies.