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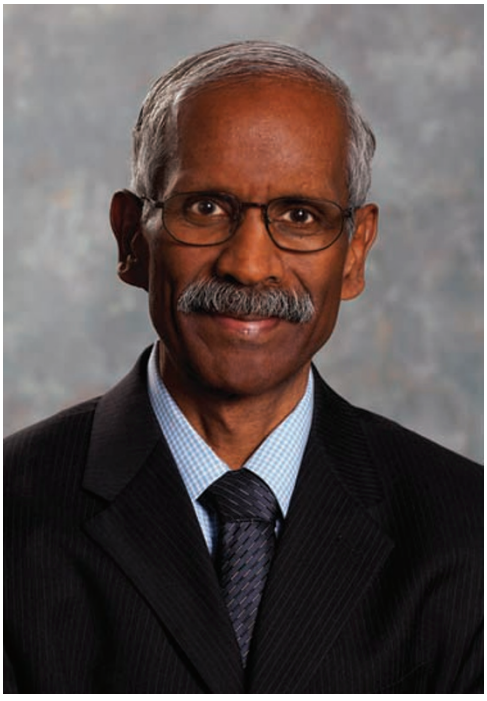

Dr G Chinnadurai

G Chinnadurai obtained his PhD in Molecular Biology from the University of Texas in Dallas in 1974. He conducted his Post-Doctoral research at the Institute for Molecular Virology, Saint Louis University School of Medicine, with Dr Maurice Green, continued in the same institution as a faculty member and was appointed as Professor of Molecular Virology in 1983. Dr Chinnadurai's research focuses on two areas: apoptosis regulation by viral and cellular genes, and mechanisms of oncogenic transformation. The research on these projects centers around two adenoviral early proteins, E1A and E1B-19K. His work on apoptosis regulation in adenovirus-infected cells started with the isolation and mapping of large plaque (*lp*) mutants of adenovirus type 2 to the E1B-19K-coding region. This work associated E1B-19K with a large plaque phenotype and facilitated the linking of an earlier known class of cytopathic (*cyt*) mutants to the *19K* locus. He then established the functional similarity between E1B-19K and BCL-2 on the basis of genetic complementation analyses, in which the *19K* mutant phenotype was shown to be suppressed in cells that overexpressed BCL-2 and in cells infected with a recombinant adenovirus that expressed BCL-2 from the E1B region. An important outcome was the identification and characterization of four cellular proteins that interact with E1B-19K and BCL-2. One of these proteins is the founding member of the BH3-only family of proteins, BIK. This class of proteins functions as apical effector of apoptosis in animal cells. The other 19K/BCL-2-interacting proteins are two other BH3-only members, BNIP3 and BNIP1, and BNIP2, a pro-apoptotic regulator of Cdc42. During mapping of the *lp* locus, Dr Chinnadurai also developed the overlap recombination method to construct adenovirus recombinants by transfection of overlapping DNA fragments. This method has been used widely in adenovirology and adenovirus-based gene therapy approaches. Of considerable note, his work on E1A identified a transformation suppression and tumor-restraining activity encoded by the C-terminal region of E1A. This work led to the identification of a novel transcriptional co-repressor, CtBP (C-terminal Binding Protein), which is essential for animal development and represses multiple tumor suppressors, pro-apoptotic genes and epithelial differentiation genes. His group also identified the pivotal cellular regulators, the acetyl transferase, Tip60, and the DNA repair protein, CtIP.

Conflict of interest

The author is currently receiving grant support from the National Cancer Institute (NCI, Rockville, MD, USA).