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## **GUEST EDITOR**

Oncogene (2007) 26, 6685; doi:10.1038/sj.onc.1210752



Dr AD Friedman

Alan Friedman earned his BS at the University of California at Berkeley and his MD from Harvard

Medical School and the Harvard-MIT Health Sciences and Technology program. He did his pediatric residency at Children's Hospital, Boston, followed by a fellowship in pediatric hematology/oncology at Johns Hopkins University, during which he conducted postdoctoral research with Steven McKnight at the Carnegie Institution of Washington. He then joined the faculty of the Johns Hopkins University School of Medicine where he is currently associate professor of Oncology and Pediatrics.

Dr Friedman's research focuses on the transcriptional control of myeloid cell differentiation and transformation. He found that C/EBPa is expressed specifically in myeloid cells within hematopoiesis, that C/EBPα contributes to both granulocyte and monocyte lineage specification and maturation, dependent on cellular context and in part via induction of PU.1 and interaction with AP-1, and that C/EBPa inhibits cell cycle progression to contribute to terminal differentiation. He, in addition, found that C/EBPa or its oncogenic variants inhibit apoptosis via induction of bcl-2 in cooperation with NF-κB p50. His group also found that RUNX1 stimulates myeloid differentiation while at the same time accelerating G1 to S progression and that RUNX1 levels themselves increase during the cell cycle, and he has developed murine models demonstrating cooperation between the dominant inhibitory TEL-AML1 and CBF\u00b3-SMMHC oncoproteins and mutations that accelerate G1. He has also mapped a critical myosin domain in CBFβ-SMMHC required for inhibition of RUNX1.