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# Protein tyrosine kinase 6 signaling in the regenerating epithelial linings of the intestine

**Angela L. Tyner**
University of Illinois College of Medicine, USA

Tyrosine kinases play important roles in normal tissue homeostasis and also contribute to a number of diseases, including cancer. Protein tyrosine kinase 6 (PTK6; often also referred to as BRK for breast tumor kinase) belongs to a family of intracellular tyrosine kinases related to, but distinct from the Src family. We cloned Ptk6 from normal mouse intestinal epithelial cell RNA, and subsequently generated the Ptk6 null mouse model. In the normal intestine, PTK6 is expressed primarily in differentiated epithelial cells. Disruption of Ptk6 led to increased growth and impaired differentiation in the small intestine, which was accompanied by increased activation of AKT, a serine threonine kinase that regulates growth and cell survival, and increased levels of nuclear β-catenin. Following DNA-damage, PTK6 expression is induced in undifferentiated intestinal crypt progenitor cells, where it promotes apoptosis by negatively regulating AKT and other phosphorylated prosurvival signaling proteins such as ERK1/2. Although functions of PTK6 in normal intestine suggested that it might have tumor suppressor functions, we found that disruption of the mouse Ptk6 gene made mice resistant to the carcinogen azoxymethane (AOM) alone and AOM plus dextran sodium sulfate (DSS). Fewer tumors formed in AOM and AOM/DSS treated Ptk6 null mice. PTK6 is induced shortly after AOM administration and plays an essential role in activation of the STAT3 transcription factor. PTK6 plays multiple roles in intestinal epithelial cells and has context specific functions that are dependent on expression levels, intracellular localization, and cell type in the intestine.

## Biography

Angela Tyner received her Ph.D. in Developmental Biology from the University of Chicago, and then pursued postdoctoral studies in Molecular Biology at Princeton University. She is a Professor in the Department of Biochemistry and Molecular Genetics at the University of Illinois at Chicago, and leader of the Cancer Cell Signaling Program within the University of Illinois Cancer Center.

atyner@uic.edu