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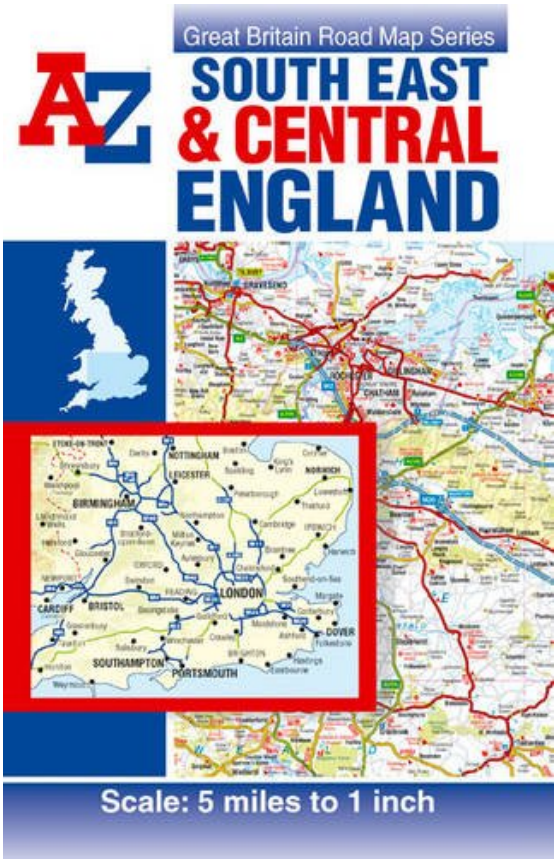

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SMAD4 and activating mutations in the oncogene KRAS, these abnormalities are not found in early PanIN lesions. We have shown that within the PanIN lesions the earliest sign of a mutator phenotype is inactivation of the ataxia telangiectasia mutated (ATM) gene, a DNA damage checkpoint kinase essential for the phosphorylation of histone H2A.3 at serine 139 (gammaH2A.3), a well-known biomarker of DNA damage. Interestingly, gammaH2A.3 is also required for the maintenance of cell cycle checkpoints and apoptosis. Further, we have shown that downregulation of gammaH2A.3 by RNA interference induces a PanIN-like phenotype in normal human pancreatic ductal epithelial (HPDE) cells. Recently, ATM was shown to be required for the maintenance of a DNA damage-resistant phenotype in PDAC cells. The long-term goal of this proposal is to develop a mouse model of human PDAC, which can be used to study the role of ATM in pancreatic tumorigenesis. To this end we propose the following Specific Aims: 1) To establish a mouse model of human PDAC in which ATM is inactivated by deletion or by over-expression of an inhibitor of ATM, and 2) to assess the relevance of ATM inactivation in the pathogenesis of human PDAC in a large panel of human PCs. These studies will employ a Cre-loxP system combined with a conditional knockout mouse for ATM or the use of small molecule inhibitors of ATM. We will also use transgenic mice expressing the gene for Cre recombinase under the control of the human hepatocyte growth factor/scatter factor (HGF/SF) promoter. Our specific hypothesis is that inactivation of ATM will accelerate, rather than impede, the 520fdb1ae7