Abstract

AID is a member of APOBEC family of mutational enzymes. AID generates U:G mismatches in ssDNA by deaminating cytosine to uracil. In B cells error-prone repair of these mismatches induces a mutational burden in the process of somatic hypermutation of *Ig* locus during affinity maturation of immunoglobulins (Ig). AID also induces double-strand breaks during Ig class switch recombination or primary Ig diversification through templated gene conversion in some vertebrate species.

AID might gain tumorigenic potential in case of insufficient regulation of induction and repair processes, causing genomic instability and possibly leading to tumorigenesis. AID is induced in epithelial tissues by proinflammatory cytokines via canonical NF-κB pathway. Both exogenous factors (pathogens *Helicobacter pylori* or HCV), endogenous factors (bile acid) or even physiological state such as ovulation are the initiating factors. Thus, AID might be the link between inflammation and carcinogenesis.

AID is expressed in different stages of carcinomas, mostly during the initial oncogenic transformation. Mice with ectopic AID expression develop lung, gastric, oral and hepatic carcinomas as well as melanomas. AID also regulates epithelial-mesenchymal transition in other tumors. AID is responsible for treatment resistance in both CML and lung adenocarcinoma. Pharmacological inhibition of AID increases treatment efficacy and also decreases the disease progression.

Key words

AID, mutagenesis, mutational load, inflammation, carcinoma, tumorigenesis, genomic instability