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Disruption of DNA repair capacity is associated with cancer susceptibility, but it remains unclear if the inherent tumor phenotypes of DNA repair deficiency syndromes can be regulated by manipulating DNA repair pathways. BLM is a structure-specific helicase which functions in many aspects of DNA homeostasis. Increasing *BLM* dosage *in vivo* in the pink-eyed unstable (p^{un}) mouse model lowers endogenous levels of homologous recombination (HR). Transgenic expression of BLM reduces pigmented eye-spots that spontaneously develop in mouse retinal pigment epithelial (RPE) cells. In p^{un} mice, eye-spots arise because of a characteristic intra-chromosomal, HR-dependent deletion within the mouse *p* gene which restores melanin production in the otherwise transparent cells of the RPE. Thus, absolute numbers of RPE eye-spots represent an *in vivo* read-out of HR levels. The cover illustration depicts a clone of (five) pigmented cells in a RPE whole mount, originating from a single reversion event. Brown melanosomes are restricted to the cytoplasm, defining cell nuclei as clear regions. For more information on the effects of transgenic BLM expression on the intestinal tumor burden and pathology of $Apc^{Min/+}$ mouse models of familial adenomatous polyposis coli, see the article by McIlhatton et al. (beginning on page 650).



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