

**DNA REPAIR IN DRUG RESISTANCE INDUCED BY FUSION  
TYROSINE KINASES**

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Fusion tyrosine kinases (FTKs) can be responsible for the pathogenesis of many leukaemias and lymphomas. We examined the role of DNA repair in therapeutic drug resistance to anticancer drugs in the murine pro-B myeloid cell line BaF3 and its clones, transformed with the following fusion oncogenic kinases: BCR/ABL, TEL/ABL, TEL/JAK2, TEL/PDGFR and NPM/ALK. These cells can be used as models of human leukaemias. Transformed cells, unlike their non-transformed counterparts, displayed resistance to idarubicin, mitomycin C and cisplatin, but they were not protected from drug-induced DNA damage, implying the activation of the mechanisms preventing DNA damage-induced apoptosis. The drugs induced DNA damage, which, directly or indirectly, was detected by the alkaline version of the single cell gel electrophoresis (the comet assay). The FTK-transformed cells required a shorter period of time to remove damage to their DNA than the control cells. We hypothesize that the observed increase in the efficacy of DNA repair may be stimulated by FTKs, and may contribute to drug resistance.

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