Defective OGG1 mutants do not excise FapyG

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references


Defective OGG1 mutants do not excise FapyG

**Stable identifier:** R-HSA-9656252

**Type:** transition

**Compartments:** nucleoplasm

**Diseases:** cancer

OGG1 R46Q mutant, reported in renal carcinoma, and OGG1 R154H mutant, reported in stomach cancer cell line MKN4 (Bruner et al. 2000), show decreased excision of FapyG from dsDNA (Audebert, Radicella et al. 2000), with the function of OGG1 R154H being more severely impaired. It is uncertain whether binding to FapyG in dsDNA substrate is affected in OGG1 R46Q and OGG1 R154H.

OGG1 R46L has not been functionally studied but has been reported in cancer and predicted to be pathogenic. It is annotated as a candidate disease variant based on its similarity with OGG1 R46L.

OGG1 S326C is a frequent genetic polymorphism in people of European and Asian descent. OGG1 S326C variant is susceptible to oxidative modifications, leading to diminished catalytic activity (Yamane et al. 2004, Moritz et al. 2014) under conditions of oxidative stress (Kershaw and Hodges 2012). This may be due to decreased specificity of OGG1 S326C for FapyG (Dherin et al. 1999).

**Literature references**


**Editions**

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