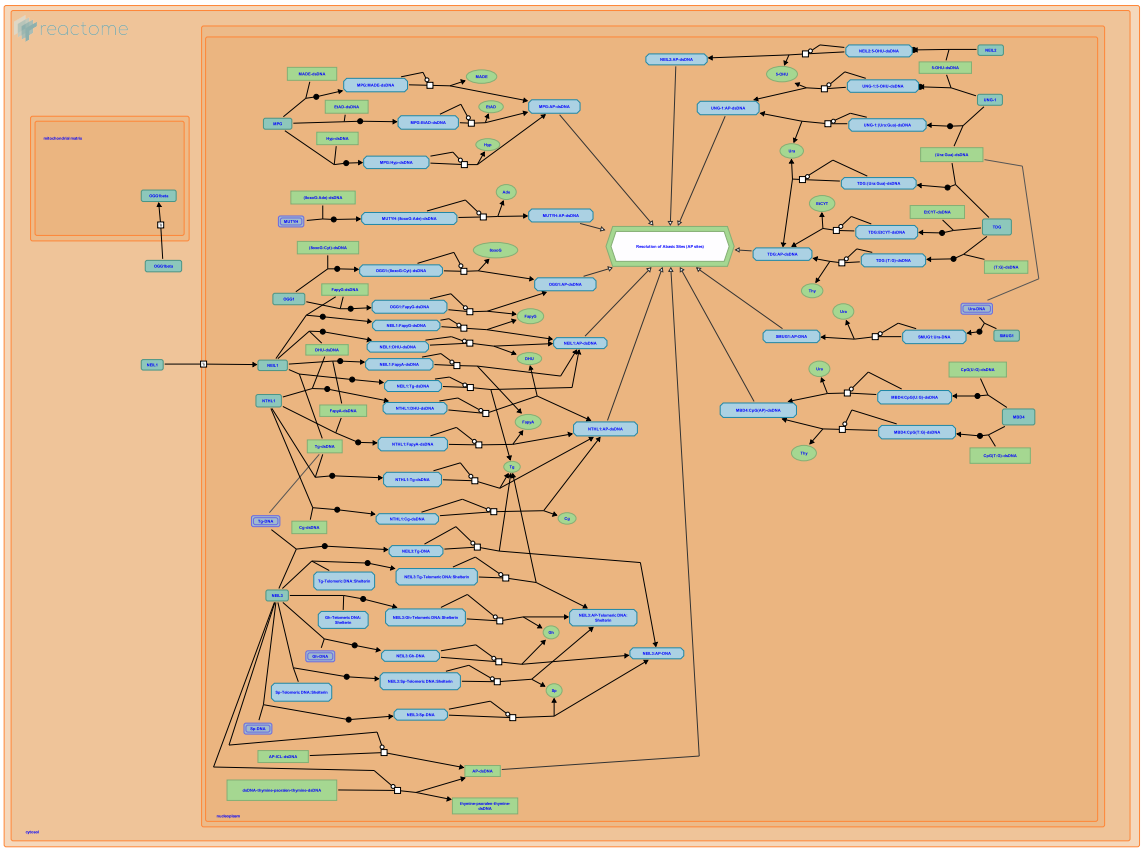
Base-Excision Repair, AP Site Formation



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15/12/2019

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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

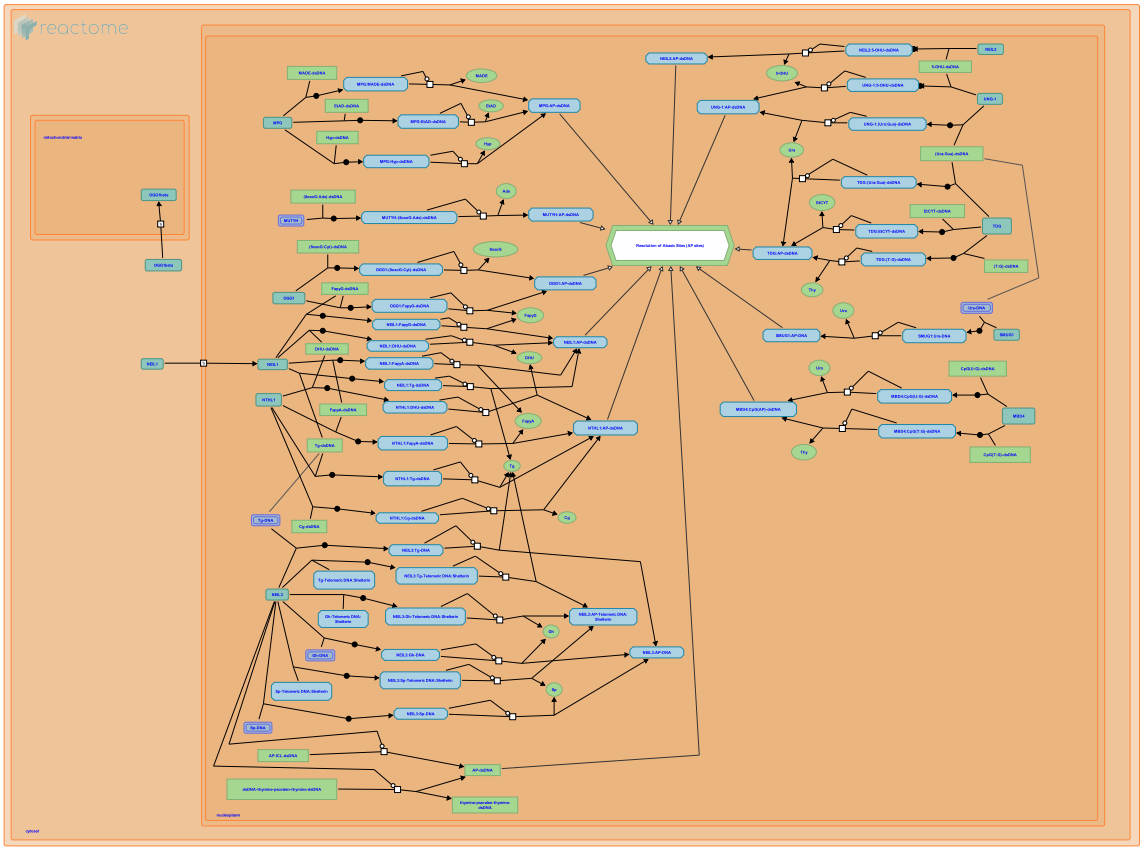
Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Base-Excision Repair, AP Site Formation ↗

Stable identifier: R-HSA-73929

Compartments: nucleoplasm



Base excision repair is initiated by DNA glycosylases that hydrolytically cleave the base-deoxyribose glycosyl bond of a damaged nucleotide residue, releasing the damaged base (Lindahl and Wood 1999, Sokhansanj et al. 2002).

Literature references

Sokhansanj, BA., Rodrigue, GR., Fitch, JP., Wilson DM, 3rd. (2002). A quantitative model of human DNA base excision repair. I. Mechanistic insights. *Nucleic Acids Res, 30*, 1817-25. ↗

Lindahl, T., Wood, RD. (1999). Quality control by DNA repair. *Science, 286*, 1897-905. ↗

Editions

2004-02-03	Authored, Edited	Matthews, L.
2014-12-04	Edited, Revised	Orlic-Milacic, M.
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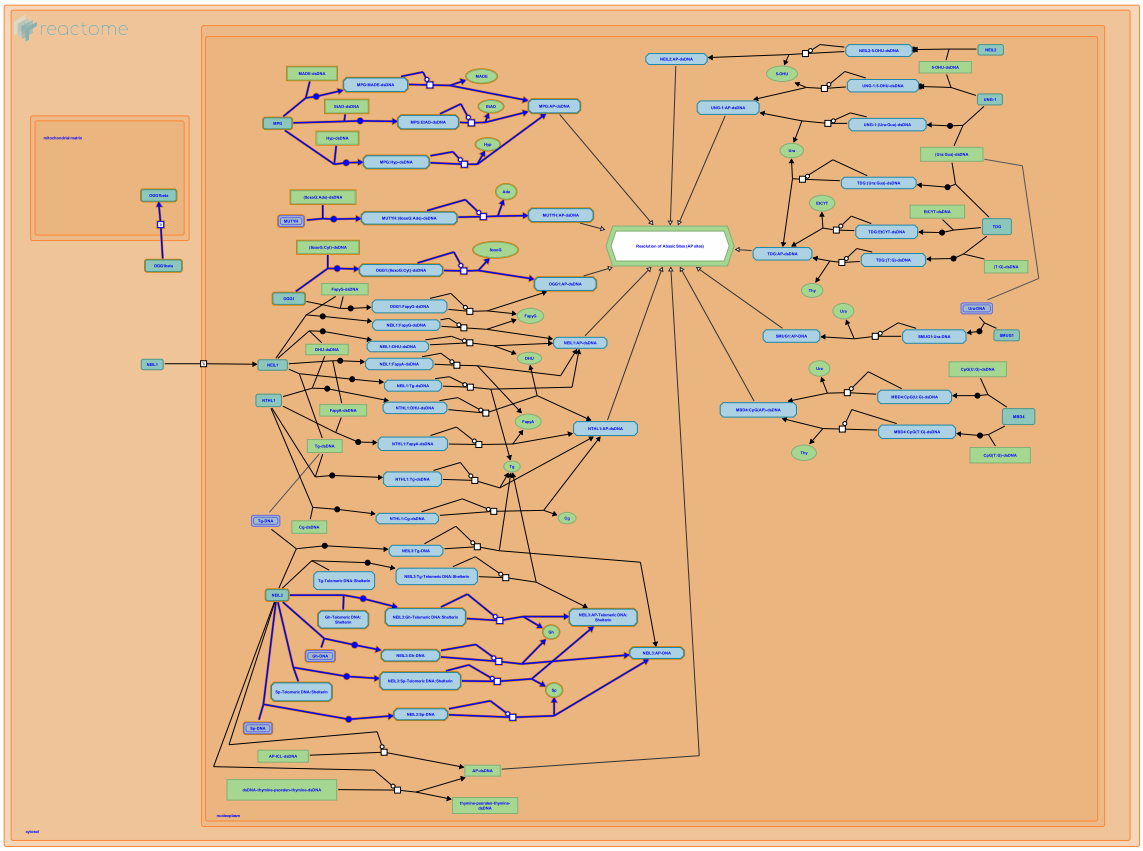
Depurination ↗

Location: Base-Excision Repair, AP Site Formation

Stable identifier: R-HSA-73927

Compartments: nucleoplasm



Depurination of a damaged nucleotide is mediated by a purine-specific DNA glycosylase. The glycosylase cleaves the N-C1' glycosidic bond between the damaged DNA base and the deoxyribose sugar, generating a free base and an abasic i.e. apurinic/apyrimidinic (AP) site (Slupphaug et al. 1996, Parikh et al. 1998).

Literature references

Slupphaug, G., Mol, CD., Kavli, B., Arvai, AS., Krokan, HE., Tainer, JA. (1996). A nucleotide-flipping mechanism from the structure of human uracil-DNA glycosylase bound to DNA. *Nature, 384*, 87-92. ↗

Parikh, SS., Mol, CD., Slupphaug, G., Bharati, S., Krokan, HE., Tainer, JA. (1998). Base excision repair initiation revealed by crystal structures and binding kinetics of human uracil-DNA glycosylase with DNA. *EMBO J, 17*, 5214-26. ↗

Editions

2004-02-03	Edited	Matthews, L.
2004-02-09	Authored	Matthews, L.
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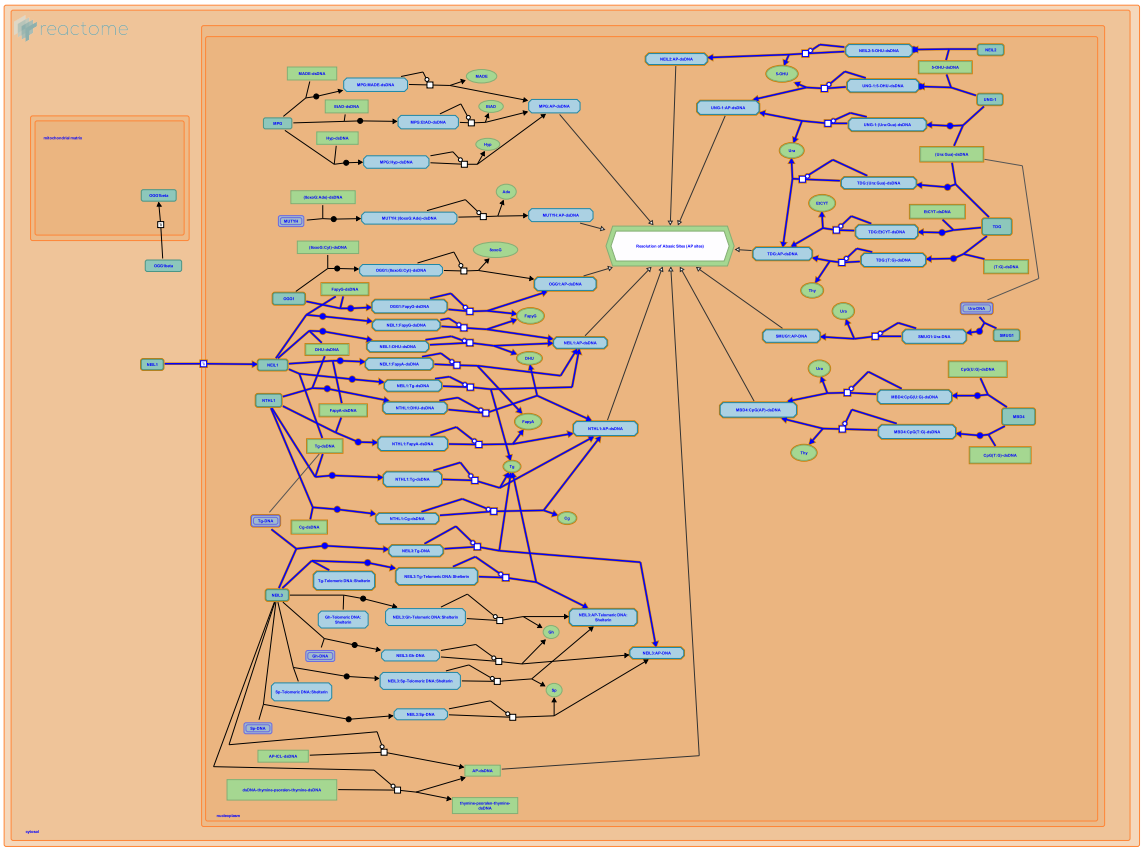
Depyrimidination ↗

Location: Base-Excision Repair, AP Site Formation

Stable identifier: R-HSA-73928

Compartments: nucleoplasm



Depyrimidination of a damaged nucleotide in DNA is mediated by a pyrimidine-specific DNA glycosylase. The glycosylase cleaves the N-C1' glycosidic bond between the damaged DNA base and the deoxyribose sugar generating a free base and an abasic i.e. apurinic/apyrimidinic (AP) site (Lindahl and Wood 1999).

Literature references

Lindahl, T., Wood, RD. (1999). Quality control by DNA repair. *Science, 286*, 1897-905. ↗

Editions

2004-02-03	Edited	Matthews, L.
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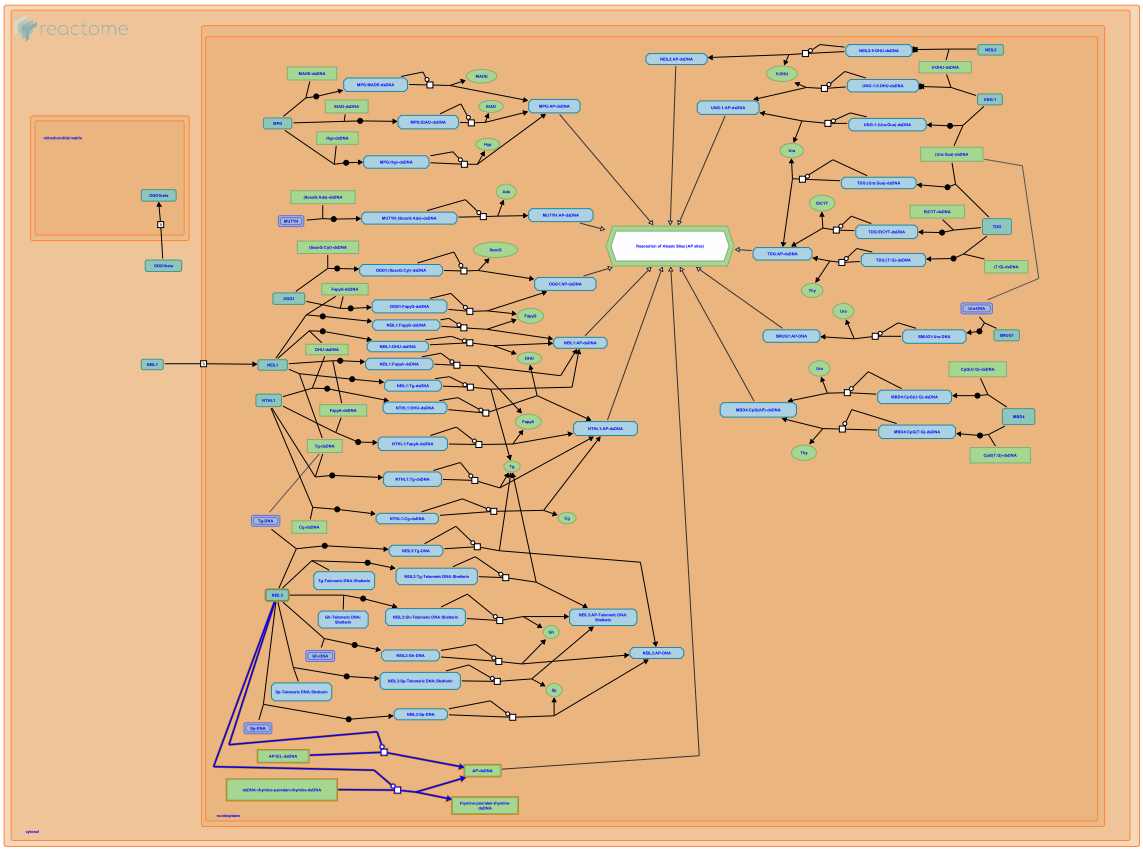
NEIL3-mediated resolution of ICLs ↗

Location: Base-Excision Repair, AP Site Formation

Stable identifier: R-HSA-9636003

Compartments: nucleoplasm



DNA glycosylase activity of NEIL3 is involved in resolution (unhooking) of psolaren-induced interstrand crosslinks (ICLs), as well as abasic site-induced ICLs (AP-ICLs) in a Fanconia anemia (FA) pathway-independent fashion (Semlow et al. 2016, Martin et al. 2017).

Literature references

Semlow, DR., Zhang, J., Budzowska, M., Drohat, AC., Walter, JC. (2016). Replication-Dependent Unhooking of DNA Interstrand Cross-Links by the NEIL3 Glycosylase. *Cell, 167*, 498-511.e14. ↗

Martin, PR., Couvé, S., Zutterling, C., Albelazi, MS., Groisman, R., Matkarimov, BT. et al. (2017). The Human DNA glycosylases NEIL1 and NEIL3 Excise Psoralen-Induced DNA-DNA Cross-Links in a Four-Stranded DNA Structure . *Sci Rep, 7*, 17438. ↗

Editions

2019-02-11	Reviewed	Zhou, J.

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