

# Joining of adjacent Okazaki fragments of the C-strand

Blackburn, EH., Hayashi, MT., Orlic-Milacic, M., Price, C., Seidel, J.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

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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

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Reactome database release: 88

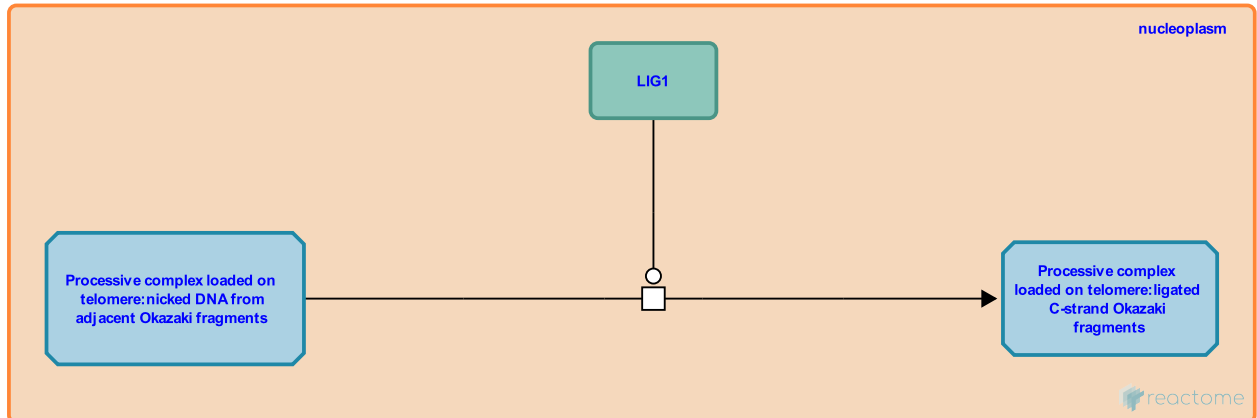
This document contains 1 reaction ([see Table of Contents](#))

## Joining of adjacent Okazaki fragments of the C-strand ↗

**Stable identifier:** R-HSA-174456

**Type:** transition

**Compartments:** nucleoplasm



Removal of the flap by FEN1 leads to the generation of a nick between the 3'-end of the upstream Okazaki fragment and the 5'-end of the downstream Okazaki fragment. DNA ligase I (LIG1) then seals the nicks between adjacent processed Okazaki fragments to generate intact double-stranded DNA (Turchi and Bambara 1993, Bambara et al. 1997, Waga and Stillman 1998, Levin et al. 2000). LIG1 is necessary for ligation of Okazaki fragments at the lagging telomere DNA strand. LIG1 deficiency results in telomere instability, manifested through telomere sister fusions, which is a consequence of DNA breaks in the lagging strand (C-strand) (Le Chalony et al. 2012).

### Literature references

Le Chalony, C., Gross, J., Gauthier, LR., Boussin, FD., Hoffschir, F., Biard, DS. et al. (2012). Partial complementation of a DNA ligase I deficiency by DNA ligase III and its impact on cell survival and telomere stability in mammalian cells. *Cell. Mol. Life Sci.*, 69, 2933-49. ↗

### Editions

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