

# miR-24 binds p16INK4A and p14ARF mRNAs

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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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- 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. [↗](#)

Reactome database release: 74

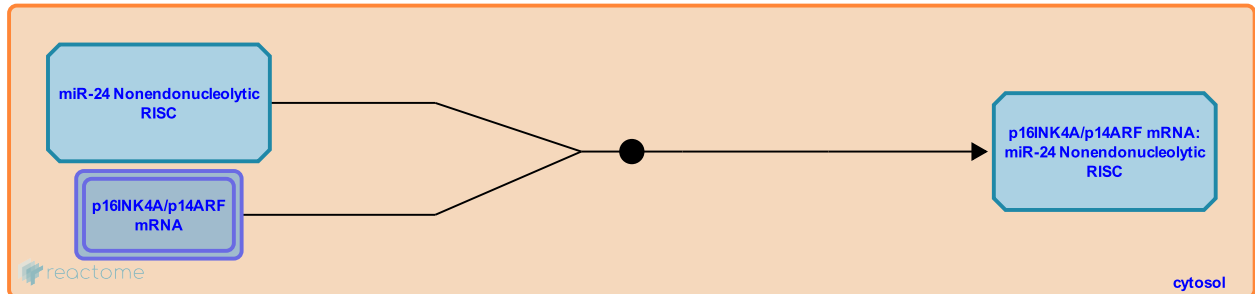
This document contains 1 reaction ([see Table of Contents](#))

## miR-24 binds p16INK4A and p14ARF mRNAs ↗

**Stable identifier:** R-HSA-3209151

**Type:** binding

**Compartments:** cytosol



MicroRNA miR-24 is able to bind both p16INK4A mRNA (Lal et al. 2008) and p14ARF mRNA (To et al. 2012) through their shared 3'UTR. miR-24 inhibits translation of p16INK4A and p14ARF mRNAs, but does not induce mRNA degradation, resulting in expression of high levels of p16INK4A and p14ARF transcripts, while protein levels of p16INK4A and p14ARF are low (Lal et al. 2008, To et al. 2012).

### Literature references

Lal, A., Kim, HH., Abdelmohsen, K., Kuwano, Y., Pullmann, R., Srikantan, S. et al. (2008). p16(INK4a) translation suppressed by miR-24. *PLoS ONE*, 3, e1864. ↗

To, KH., Pajovic, S., Gallie, BL., Thériault, BL. (2012). Regulation of p14ARF expression by miR-24: a potential mechanism compromising the p53 response during retinoblastoma development. *BMC Cancer*, 12, 69. ↗

### Editions

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