Apoptosis induced DNA fragmentation

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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


Reactome database release: 72

This document contains 1 pathway and 12 reactions (see Table of Contents)
DNA fragmentation in response to apoptotic signals is achieved, in part, through the activity of apoptotic nucleases, termed DNA fragmentation factor (DFF) or caspase-activated DNase (CAD) (reviewed in Widlak and Garrard, 2005). In non-apoptotic cells, DFF is a nuclear heterodimer consisting of a 45 kD chaperone and inhibitor subunit (DFF45)/inhibitor of CAD (ICAD-L]) and a 40 kD nuclease subunit (DFF40/CAD) (Liu et al. 1997, 1998; Enari et al. 1998). During apoptosis, activated caspase-3 or -7 cleave DFF45/ICAD releasing active DFF40/CAD nuclease. The activity of DFF is tightly controlled at multiple stages. During translation, DFF45/ICAD, Hsp70, and Hsp40 proteins play a role in insuring the appropriate folding of DFF40 during translation(Sakahira and Nagata, 2002). The nuclease activity of DFF40 is enhanced by the chromosomal proteins histone H1, Topoisomerase II and HMGB1/2(Widlak et al., 2000). In addition, the inhibitors (DFF45/35; ICAD-S/L) are produced in stoichiometric excess (Widlak et al., 2003).

**Literature references**


**Editions**

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Association of DFF40 with DFF45

Location: Apoptosis induced DNA fragmentation

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