

TFAP2A, TFAP2C homodimers bind the CGB gene promoter

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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: 77

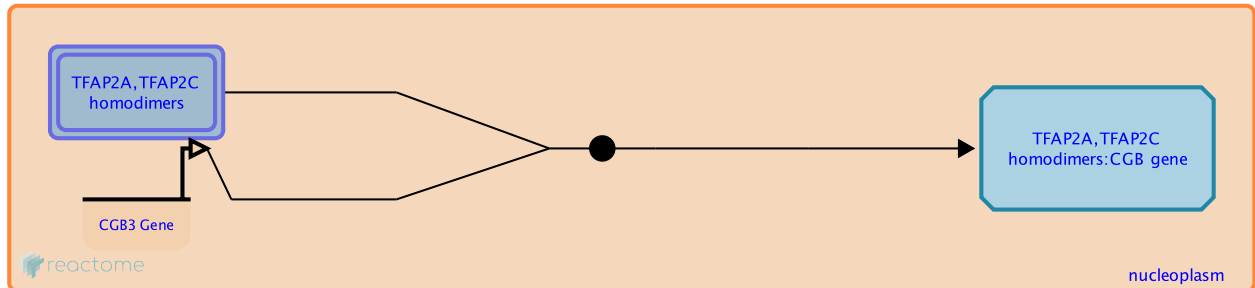
This document contains 1 reaction ([see Table of Contents](#))

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Stable identifier: R-HSA-8864426

Type: binding

Compartments: nucleoplasm



The CGB (chorionic gonadotropin beta) gene promoter, similar to CGA (chorionic gonadotropin alpha) gene promoter, contains several putative AP-2 transcription factor binding sites. TFAP2A (AP-2 alpha) and TFAP2C (AP-2 gamma), which are both expressed in the placenta, can both bind to the CGB promoter (Johnson et al. 1997, LiCalsi et al. 2000). It has not been examined whether TFAP2A and TFAP2C bind the CGB promoter as homodimers or heterodimers and if CITED family members are involved in TFAP2A/C-mediated transactivation of CGB transcription.

Literature references

Johnson, W., Albanese, C., Handwerger, S., Williams, T., Pestell, RG., Jameson, JL. (1997). Regulation of the human chorionic gonadotropin alpha- and beta-subunit promoters by AP-2. *J. Biol. Chem.*, 272, 15405-12. ↗

LiCalsi, C., Christophe, S., Steger, DJ., Buescher, M., Fischer, W., Mellon, PL. (2000). AP-2 family members regulate basal and cAMP-induced expression of human chorionic gonadotropin. *Nucleic Acids Res.*, 28, 1036-43. ↗

Editions

2016-03-14	Authored, Edited	Orlic-Milacic, M.
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