Regulation of PTEN gene transcription


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

The contents of this document may be freely copied and distributed in any media, provided the authors, plus the institutions, are credited, as stated under the terms of Creative Commons Attribution 4.0 International (CC BY 4.0) License. For more information see our license.

12/09/2020
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


Reactome database release: 73

This document contains 1 pathway and 15 reactions (see Table of Contents)
Regulation of PTEN gene transcription

Stable identifier: R-HSA-8943724

Transcription of the PTEN gene is regulated at multiple levels. Epigenetic repression involves the recruitment of Mi-2/NuRD upon SALL4 binding to the PTEN promoter (Yang et al. 2008, Lu et al. 2009) or EVI1-mediated recruitment of the polycomb repressor complex (PRC) to the PTEN promoter (Song et al. 2009, Yoshimi et al. 2011). Transcriptional regulation is also elicited by negative regulators, including NR2E1:ATN1 (atrophin-1) complex, JUN (c-Jun), SNAIL and SLUG (Zhang et al. 2006, Vasudevan et al. 2007, Escriva et al. 2008, Uygur et al. 2015) and positive regulators such as TP53 (p53), MAF1, ATF2, EGR1 or PPARG (Stambolic et al. 2001, Virolle et al. 2001, Patel et al. 2001, Shen et al. 2006, Li et al. 2016).

Literature references


### Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2015-10-29</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salm, L.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
TP53 binds the PTEN promoter

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-5632939

**Type:** binding

**Compartments:** nucleoplasm

PTEN (phosphatase and tensin homolog deleted in chromosome 10) is a tumor suppressor gene that is deleted or mutated in a variety of human cancers. TP53 (p53) binds to the p53-binding site at the PTEN promoter level (Stambolic et al. 2001).

**Followed by:** PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2014-12-23</td>
<td>Authored, Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2014-12-30</td>
<td>Reviewed</td>
<td>Hwang, PM., Kang, JG., Wang, PY.</td>
</tr>
<tr>
<td>2016-02-04</td>
<td>Reviewed</td>
<td>Inga, A., Zaccara, S.</td>
</tr>
<tr>
<td>2016-08-11</td>
<td>Reviewed</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-09-30</td>
<td>Reviewed</td>
<td>Leslie, N., Kriplani, N.</td>
</tr>
</tbody>
</table>
EGR1 binds the PTEN gene promoter

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944078

**Type:** binding

**Compartments:** nucleoplasm

In response to UV-induced DNA damage, expression levels of both EGR1 and PTEN increase. EGR1 binds directly to the EGR1 binding site GCGGCGGCG in the promoter region of PTEN to stimulate PTEN transcription (Virolle et al. 2001).

**Followed by:** PTEN gene transcription is stimulated by TP53, EGR1, PPARg, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2015-10-29</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
Activated PPARG binds PTEN gene promoter

Location: Regulation of PTEN gene transcription

Stable identifier: R-HSA-8944099

Type: binding

Compartments: nucleoplasm

The nuclear receptor PPARG (PPARgamma), activated by ligand binding, binds to peroxisome proliferator response elements (PPREs) in the promoter of the PTEN gene to activate PTEN transcription. It has not been tested whether nuclear receptors that heterodimerize with PPARG are involved in transcriptional regulation of PTEN (Patel et al. 2001).

Followed by: PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-09-30</td>
<td>Reviewed</td>
<td>Leslie, N., Kriplani, N.</td>
</tr>
<tr>
<td>2016-11-02</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
NR2E1 associated with transcription repressors binds PTEN promoter

Location: Regulation of PTEN gene transcription

Stable identifier: R-HSA-6807077

Type: binding

Compartments: nucleoplasm

Inferred from: Nr2e1 binds Pten promoter (Mus musculus)

NR2E1 (TLX) associated with transcription repressors binds the evolutionarily conserved TLX consensus site in the PTEN promoter. NR2E1 inhibits PTEN transcription by associating with various transcriptional repressors, probably in a cell type or tissue specific manner. PTEN transcription is inhibited when NR2E1 forms a complex with ATN1 (atrophin-1) (Zhang et al. 2006, Yokoyama et al. 2008), KDM1A (LSD1) histone methyltransferase containing CoREST complex (Yokoyama et al. 2008), or histone deacetylases HDAC3, HDAC5 or HDAC7 (Sun et al. 2007).

Followed by: PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Author/Reviewer</th>
<th>Author/Reviewer Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>2015-10-29</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-09-30</td>
<td>Reviewed</td>
<td>Leslie, N., Kriplani, N.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
SALL4 binds the PTEN gene promoter

Location: Regulation of PTEN gene transcription

Stable identifier: R-HSA-8943728

Type: binding

Compartments: nucleoplasm

The transcription factor SALL4 binds the promoter of the PTEN gene (Yang et al. 2008, Lu et al. 2009).

Followed by: SALL4 recruits NuRD to PTEN gene

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
SALL4 recruits NuRD to PTEN gene

Location: Regulation of PTEN gene transcription

Stable identifier: R-HSA-8943780

Type: binding

Compartments: nucleoplasm

SALL4 recruits the transcriptional repressor complex NuRD, containing histone deacetylases HDAC1 and HDAC2, to the PTEN gene promoter (Lu et al 2009, Gao et al. 2013). SALL4 may also recruit DNA methyltransferases (DNMTs) to the PTEN promoter (Yang et al. 2012).

Preceded by: SALL4 binds the PTEN gene promoter

Followed by: PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-10-29</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
MECOM (EVII) binds the PTEN gene promoter

Location: Regulation of PTEN gene transcription

Stable identifier: R-HSA-8943811

Type: binding

Compartments: nucleoplasm

The transcription factor MECOM (EVII) binds the promoter of the PTEN gene (Yoshimi et al. 2011).

Followed by: MECOM (EVII) recruits polycomb repressor complexes (PRCs) to the PTEN gene promoter

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-10-30</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
MECOM (EVI1) recruits polycomb repressor complexes (PRCs) to the PTEN gene promoter

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8943817

**Type:** binding

**Compartments:** nucleoplasm

The transcription factor MECOM (EVI1) can associate with the polycomb repressor complexes (PRCs) and recruit them to the promoter of the PTEN gene (Song et al. 2009). Both the BMI1-containing PRC, supposedly PRC1.4, and the EZH2-containing PRC2 complex are recruited to the PTEN promoter, resulting in transcriptional silencing of the PTEN gene (Song et al. 2009, Yoshimi et al. 2011). Since the exact composition of the EZH2-containing PRC2 at the PTEN promoter is not known, the core EZH2-PRC2 complex is shown.

**Preceded by:** MECOM (EVI1) binds the PTEN gene promoter

**Followed by:** PTEN gene transcription is stimulated by TP53, EGR1, PPARγ, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Edition</th>
<th>Authored/Edited by</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-10-30</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
SNAI1,SNAI2 bind the PTEN gene promoter

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944026

**Type:** binding

**Compartments:** nucleoplasm

The transcriptional repressor SNAI1 (Snail1) binds the promoter of the PTEN gene. Binding of SNAI1 to the PTEN promoter increases in response to ionizing radiation and interferes with binding of TP53 to the PTEN promoter. Phosphorylation of SNAI1 at serine residue S246 may be required for SNAI1-mediated repression of PTEN transcription (Escriva et al. 2008). Another Slug/Snail family member SNAI2 (SLUG) can also bind to the PTEN gene promoter to repress PTEN transcription (Uygur et al. 2015).

**Followed by:** PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
<th>Author/Editor</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-11-01</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>

https://reactome.org
JUN binds the PTEN gene promoter

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944047

**Type:** binding

**Compartments:** nucleoplasm

The transcription factor JUN binds the AP-1 element in the PTEN gene promoter (Hettinger et al. 2007) and represses PTEN gene transcription. RAS/RAF/MAPK signaling positively affects JUN-mediated inhibition of PTEN transcription, but the mechanism is not known. The JUN partner FOS is not needed for JUN-mediated downregulation of PTEN (Vasudevan et al. 2007).

**Followed by:** PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-11-01</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
p-T69,T71-ATF2 binds PTEN gene promoter

Location: Regulation of PTEN gene transcription

Stable identifier: R-HSA-8944397

Type: binding

Compartments: nucleoplasm

The transcription factor ATF2, activated downstream of stress signaling by p38 MAPKs, binds to ATF response elements in the PTEN gene promoter to activate PTEN transcription. It has not been examined whether ATF2 heterodimerization partners are involved in ATF2-mediated up-regulation of PTEN (Shen et al. 2006).

Followed by: PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-11-03</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
mTORC1 phosphorylates MAF1

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944454

**Type:** transition

**Compartments:** cytosol

Activated mTORC1 complex phosphorylates the transcription factor MAF1 on serine residues S60, S68 and S75 (Shor et al. 2010, Michels et al. 2010). mTORC1-mediated phosphorylation of MAF1 inhibits translocation of MAF1 to the nucleus (Shor et al. 2010).

**Followed by:** MAF1 translocates to the nucleus

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-11-03</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
MAF1 translocates to the nucleus

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944457

**Type:** omitted

**Compartments:** nuclear envelope

Phosphorylation of MAF1 by the activated mTORC1 complex inhibits translocation of MAF1 to the nucleus, and hence its transcriptional activity, but the mechanism has not been elucidated (Shor et al. 2010).

**Preceded by:** mTORC1 phosphorylates MAF1

**Followed by:** MAF1 binds the PTEN gene promoter

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-11-03</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
MAF1 binds the PTEN gene promoter

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944420

**Type:** binding

**Compartments:** nucleoplasm

The transcription factor MAF1 binds to the promoter region of the PTEN gene to stimulate PTEN transcription (Li et al. 2016). MAF1 is known as a transcriptional repressor of RNA polymerase III-dependent genes, such as genes encoding transport RNAs (tRNAs). Phosphorylation of MAF1 by the mTORC1 complex inhibits MAF1 translocation to the nucleus and transcriptional activity of MAF1 (Shor et al. 2010, Michels et al. 2010).

**Preceded by:** MAF1 translocates to the nucleus

**Followed by:** PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-08-11</td>
<td>Authored</td>
<td>Carracedo, A., Salmena, L.</td>
</tr>
<tr>
<td>2016-11-03</td>
<td>Authored</td>
<td>Orlic-Milacic, M.</td>
</tr>
<tr>
<td>2017-05-09</td>
<td>Edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>
PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN

**Location:** Regulation of PTEN gene transcription

**Stable identifier:** R-HSA-8944104

**Type:** omitted

**Compartments:** nucleoplasm, cytosol

**Inferred from:** Nr2e1 represses Pten transcription (Mus musculus)

PTEN (phosphatase and tensin homolog deleted in chromosome 10) is a tumor suppressor gene that is deleted or mutated in a variety of human cancers. TP53 (p53) stimulates PTEN transcription (Stambolic et al. 2000, Singh et al. 2002). PTEN, acting as a negative regulator of PI3K/AKT signaling, affects cell survival, cell cycling, proliferation and migration. PTEN regulates TP53 stability by inhibiting AKT-mediated activation of TP53 ubiquitin ligase MDM2, and thus enhances TP53 transcriptional activity and its own transcriptional activation by TP53. Beside their cross-regulation, PTEN and TP53 can interact and cooperate to regulate survival or apoptotic phenomena (Stambolic et al. 2000, Singh et al. 2002, Nakanishi et al. 2014).

In response to UV induced DNA damage, PTEN transcription is stimulated by binding of the transcription factor EGR1 to the promoter region of PTEN (Virolle et al. 2001).

PTEN transcription is also stimulated by binding of the activated nuclear receptor PPARG (PPARgamma) to peroxisome proliferator response elements (PPREs) in the promoter of the PTEN gene (Patel et al. 2001), binding of the ATF2 transcription factor, activated by stress kinases of the p38 MAPK family, to ATF response elements in the PTEN gene promoter (Shen et al. 2006) and by the transcription factor MAF1 (Li et al. 2016).

NR2E1 (TLX) associated with transcription repressors binds the evolutionarily conserved TLX consensus site in the PTEN promoter. NR2E1 inhibits PTEN transcription by associating with various transcriptional repressors, probably in a cell type or tissue specific manner. PTEN transcription is inhibited when NR2E1 forms a complex with ATN1 (atrophin-1) (Zhang et al. 2006, Yokoyama et al. 2008), KDM1A (LSD1) histone methyltransferase containing CoREST complex (Yokoyama et al. 2008), or histone deacetylases HDAC3, HDAC5 or HDAC7 (Sun et al. 2007).

Binding of the transcriptional repressor SNAI1 (Snail1) to the PTEN promoter represses PTEN transcription. SNAI1-mediated repression of PTEN transcription may require phosphorylation of SNAI1 on serine residue S246. Binding of SNAI1 to the PTEN promoter increases in response to ionizing radiation and is implicated in SNAI1-mediated resistance to gamma-radiation induced apoptosis (Escriva et al. 2008). Binding of another Slug/Snail family member SNAI2 (SLUG) to the PTEN gene promoter also represses PTEN transcription (Uygur et al. 2015).

Binding of JUN to the AP-1 element in the PTEN gene promoter (Hettinger et al. 2007) inhibits PTEN tran-
scription. JUN partner FOS is not needed for JUN-mediated downregulation of PTEN (Vasudevan et al. 2007).

Binding of the transcription factor SALL4 to the PTEN gene promoter (Yang et al. 2008) and SALL4-mediated recruitment of the transcriptional repressor complex NuRD (Lu et al. 2009, Gao et al. 2013), containing histone deacetylases HDAC1 and HDAC2, inhibits the PTEN gene transcription. SALL4-mediated recruitment of DNA methyltransferases (DNMTs) is also implicated in transcriptional repression of PTEN (Yang et al. 2012).

Binding of the transcription factor MECOM (EVII) to the PTEN gene promoter and MECOM-mediated recruitment of polycomb repressor complexes containing BMI1 (supposedly PRC1.4), and EZH2 (PRC2) leads to repression of PTEN transcription (Song et al. 2009, Yoshimi et al. 2011).

Preceded by: TP53 binds the PTEN promoter, EGR1 binds the PTEN gene promoter, Activated PPARG binds PTEN gene promoter, NR2E1 associated with transcription repressors binds PTEN promoter, SALL4 recruits NuRD to PTEN gene, MECOM (EVII) recruits polycomb repressor complexes (PRCs) to the PTEN gene promoter, SNAI1, SNAI2 bind the PTEN gene promoter, JUN binds the PTEN gene promoter, p-T69,T71-ATF2 binds PTEN gene promoter, MAF1 binds the PTEN gene promoter

Literature references


Table of Contents

Introduction

- Regulation of PTEN gene transcription
  - TP53 binds the PTEN promoter
  - EGR1 binds the PTEN gene promoter
  - Activated PPARG binds PTEN gene promoter
  - NR2E1 associated with transcription repressors binds PTEN promoter
  - SALL4 binds the PTEN gene promoter
  - SALL4 recruits NuRD to PTEN gene
  - MECOM (EV11) binds the PTEN gene promoter
  - MECOM (EV11) recruits polycomb repressor complexes (PRCs) to the PTEN gene promoter
  - SNAI1,SNAI2 bind the PTEN gene promoter
  - JUN binds the PTEN gene promoter
  - p-T69,T71-ATF2 binds PTEN gene promoter
  - mTORC1 phosphorylates MAF1
  - MAF1 translocates to the nucleus
  - MAF1 binds the PTEN gene promoter
  - PTEN gene transcription is stimulated by TP53, EGR1, PPARG, ATF2, MAF1, and inhibited by NR2E1, SALL4, MECOM, SNAI1, SNAI2, JUN