1

AUTOTRON

Automatic annotate the current document.

Documents having a pubmed ID will have the mentions and the concepts extracted with **PUBTATOR** mesh/omim diseases ncbi gene, otherwise they will have the mentions and the concepts annotated with **METAMAP** umls cui concepts.

Select your task.

Task

Select a a task

Gene Cancer Association

Gene Disease Association

test / pubmed_24662820 < > DELETE + ASSERTION ANNOTATION

year

2015

PKA signaling drives mammary tumorigenesis through Src.

authors

Beristain AG, Molyneux SD, Joshi PA, Pomroy NC, Di Grappa MA, Chang MC, Kirschner LS, Privé GG, Pujana MA, Khokha R

journal

Oncogene; 2015 Feb 26; 34(9) 1160-73. doi:10.1038/onc.2014.41

abstract

Protein kinase A (PKA) hyperactivation causes hereditary endocrine neoplasias; however, its role in sporadic epithelial cancers is unknown. Here, we show that heightened PKA activity in the mammary epithelium generates tumors. Mammary-restricted biallelic ablation of Prkar1a, which encodes for the critical type-I PKA regulatory subunit, induced spontaneous breast tumors characterized by enhanced type-II PKA activity. Downstream of this, Src phosphorylation occurs at residues serine-17 and tyrosine-416 and mammary cell transformation is driven through a mechanism involving Src signaling. The phenotypic consequences of these alterations consisted of increased cell proliferation and, accordingly, expansion of both luminal and basal epithelial cell populations. In human breast cancer, low PRKAR1A/high SRC expression defines basal-like and HER2 breast tumors associated with poor clinical outcome. Together, the results of this study define a novel molecular mechanism altered in breast carcinogenesis and highlight the potential strategy of inhibiting SRC signaling in treating this cancer subtype in humans.

provenance

pubmed

document_id

pubmed_24662820

2

DELETE + ASSERTION

3

est / pubmed_24662820 < > Press Esc to exit from relationship annotation ANNOTATION

ear

015

KA signaling drives mammary tumorigenesis through Src .

uthors

eristain AG, Molyneux SD, Joshi PA, Pomroy NC, Di Grappa MA, Chang MC, Kirschner LS, Privé GG, Pujana MA, Khokha R

ournal

ncogene; 2015 Feb 26; 34(9) 1160-73. doi:10.1038/onc.2014.41

bstract

rotein kinase A (PKA) hyperactivation causes hereditary endocrine neoplasias; however, its role in sporadic epithelial cancers is unknown. Here, we show that heightened

KA activity in the mammary epithelium generates tumors. Mammary-restricted biallelic ablation of Prkar1a, which encodes for the critical type-I PKA regulatory

ubunit, induced spontaneous breast tumors characterized by enhanced type-II PKA activity. Downstream of this, Src phosphorylation occurs at residues serine-17 and

yrosine-416 and mammary cell transformation is driven through a mechanism involving Src signaling. The phenotypic consequences of these alterations consisted of

ncreased cell proliferation and, accordingly, expansion of both luminal and basal epithelial cell populations. In human breast cancer, low PRKAR1A/high SRC expression

defines basal-like and HER2 breast tumors associated with poor clinical outcome. Together, the results of this study define a novel molecular mechanism altered in

BIOMARKER

Relationships

Subject

Gene (6)

PRKAR1A

PRKAR1A

Insertion datetime: 2023-05-23 15:09:26

Annotators: 1

SRC

SRC

ERBB2

ERBB2

Predicate

Object

Other