GenCLiP 2.0 使用手册

■ 用户注册



进入填写注册基本信息的界面:

guest Login Register Logou	GenCLi Human Gene Function Ar	
Email address:	**	
Password:	** (between 6-20 characters)	注册项目的每栏信息
Confirm password:	**	都是必须填写的,其中 注册邮箱(即用户名)
Organization:	**	必须正确填写。
Job type:	Choose ▼ **	
Country:	Choose ▼ **	
Security Question:	Choose a question ▼ **	
Security Answer:	**	
Submit	──基本信息填完后点击 "Submit"完成注册	

点击"Login"进入注册用户登陆界面:



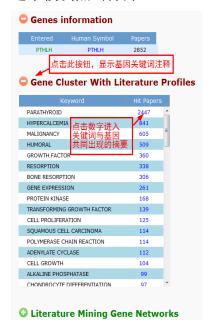
注册用户分析界面:



■ 单个基因信息检索



逐个模块点击弹开:



Keyword: PARATHYROID

Genes: 1 Papers: 2447

Num			Total
1	PTHLH	2447	2852

Gene: PTHLH

Alias: parathyroid hormone-like related protein; PTHRP; BDE2; parathyroid hormone -like hormone; PLP; PTHLH; PTHR; HHM; parathyroid hormone-related protein; osteostatin; PTH-rP; PTH-related protein

Summary: The protein encoded by this gene is a member of the parathyroid hormone family. This hormone regulates endochondral bone development and epithelial-mesenchymal interactions during the formstoin of the mammary glands and teath. This hormone is involved in lactation possibly by regulating the mobilization and transfer of calcium to the milk. The receptor of this hormone, PTRL, is responsible for most cases of humoral hypercalcemia of malignancy. Four alternatively spliced transcript variants encoding two distinct isoforms have been observed. There is also evidence for alternative translation minition from non-AUG CGU and CGU start attex, in-rime and downstream the initiator AUG codon, to give rise to nuclear forms of this hormone. [provided by RefSeq]

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1. PMID: 20951345
Dev Cell. 2010 Oct 19;19(4):533-46.
Zfp521 is a target gene and key effector of parathyroid hormone-related peptide signaling in growth plate chondrocytes.
Correa D, Hesse E, Seriwatanachai D, Kiviranta R, Saito H, Yamana K, Neff L, Atfi A, Coillard L, Sitara D, Maeda Y, Warming S, Jenkins NA, Copeland NG, Horne WC, Lanske B, Baron R.
Department of Oral Medicine, Infection and Immunity, Harvard School of Dental Medicine, Boston, MA 02115, USA.

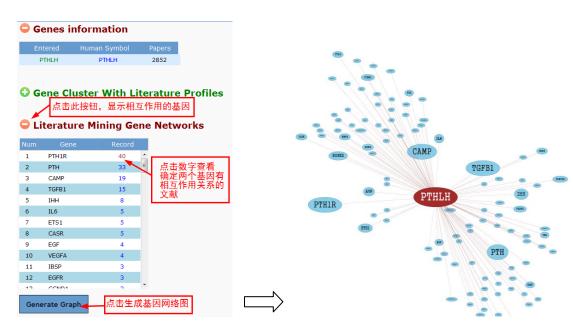
In the growth plate, the interplay between parathyroid hormone-related peptide (PTHrP) and Indian hedgehog (Ihh) signaling tightly regulates chondrocyte proliferation and differentiation during longitudinal bone growth. We found that PTHrP increases the expression of Zfp521, a zinc finger transcriptional coregulator, in prehypertrophic chondrocytes. Mice with chondrocyte-targeted deletion of Zfp521 resembled PTHrP(-/-) and chondrocyte-specific PTHR1(-/-) mice, with decreased chondrocyte proliferation, early hypertrophic transition, and reduced growth plate thickness. Deleting Zfp521 increased expression of Runx2 and Runx2 target genes, and decreased Cyclin D1 and 8d-2 expression while increasing Caspase-3 activation and apoptosis. Zfp521 associated with Runx2 in chondrocytes, antagonizing its activity via an HDAC4-dependent mechanism. PTHrP failed to upregulate Cyclin D1 and to antagonize Runx2, thh, and collagen X expression who and spoptosis. Zfp521 as an important PTHrP target gene that regulates growth plate chondrocyte proliferation and differentiation.

Journal Article. Research Support, N.I.H., Extramural. Research Support, Non-U.S. Gov't.

2. PMID: 20683010

Anticancer Res. 2010 Jul;30(7):2755-67.

PTHrP regulates andiogenesis and hone resorption via VEGE expression.



点击 PTH1R 的记录数 "40",链接基因对的文献出处:

Gene: PTHLH
Alias: parathyroid hormone-like related protein; PTHRP;
BDE2: parathyroid hormone-like hormone; PLP; PTHLH;
PTHR; HHM; parathyroid hormone-related protein;
osteostatin; PTH-P; PTH-related protein

Gene: PTH1R

Alias: parathyroid hormone/parathyroid hormone-related peptide receptor; parathyroid hormone/parathyroid hormone/parathyroid hormone-related protein receptor; parathyroid hormone receptor 1; PTH/PTHIP type I receptor; PTH1 receptor; PTHIR; seven transmembrane helix receptor; PTHR1; PTH/PTHr receptor; parathyroid hormone 1 receptor; PTHR; PFE

31. Pmil): 10912527 Pediatr Nephrol. 2000 Jul;14(7):606-11. Role of parathyroid hormone-related peptide and Indian hedgehog in skeletal development. 17?ppper H. Department of Pediatrics, Massachusetts General Hospital and Harvard Medical School, Boston 02114, USA.

Parathyroid hormone-related peptide (PTHrP), which frequently causes the humoral hypercalcemia of malignancy syndrome, is an autocrine/paracrine regulator of chondrocyte proliferation and differentiation that acts through the PTH/PTHrP receptor (PTHIR). PTHrP is generated in response to Indian hedgehog (Ihh), which mediates its actions through the membrane receptor patched, but interacts also with hedgehog-interacting protein (Hip). Mice lacking PTHrP show accelerated chondrocyte differentiation, and thus premature ossification of those bones that are formed through an endochodral process, and similar but more-severe abnormalities are observed in PTH1R-ablated animals. The mirror image of these skeletal findings, i.e., a severe delay in chondrocyte differentiation and endochondral ossification, is observed in transgenic mice that overexpress PTHrP under the control of the alpha1(II) procollagen promoter. Severe abnormalities in chondrocyte proliferation and differentiation are also observed in two genetic disorders in humans that are most likely caused by mutations in the PTH1R. Heterozygous PTH1R mutations that lead to constitutively activity were identified in Jansen metaphyseal chondrodysplasia, and homozygous or compound heterozygous mutations that lead to less-active or completely inactive receptors were identified in patients with Blomstrand lethal chondrodysplasia. Based on the growth plate abnormalities observed in these human disorders and in mice with abnormal expression of either PTHP or the PTH1R. It appears plausible that impaired expression of PTHP and/or its receptor contributes to the growth abnormalities in children with end-stage renal disease. In fact, mild-to-moderate renal failure leads in animals to a reduction in PTH1R expression in PTH1R and/or its receptor contributes

Journal Article. Review

32. PMID: 10875241 Endocrinology. 2000 Jul;141(7):2410-21.

Dissection of differentially regulated (G+C)-rich promoters of the human parathyroid hormone (PTH)/PTH-related peptide receptor gene. Minagawa M, Kwan MY, Bettoun JD, Mansour FW, Dassa J, Hendy GN, Goltzman D, White JH.

Department of Medicine, McGill University, Montr??al, Qu??bec, Canada.

The PTH/PTH-related peptide (PTHrP) receptor (PTHR) is required for normal skeletal development, and a wide array of physiological responses mediated by PTH and PTHrP. We have previously identified three promoters, P1-P3, which control human PTHR gene transcription. P2 and P3 are (G+C)-rich, function in a number of tissues, lie within the same CpG island, and display many hallmarks of housekeeping promoters. However, they are differentially regulated during development as P2, but not P3, functions in fetal tissues. Here, we have used both stably and transiently transfected

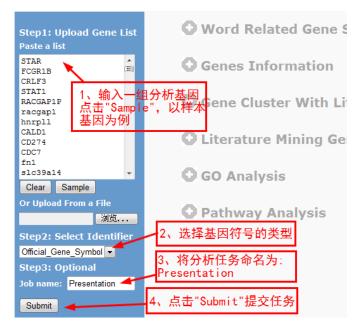




■ 批量基因的分析流程

以网页上提供的样本基因,324个鼻咽癌表达上调基因为例。

无论是在输入框输入还是以文本文件(txt 格式)上传一组分析基因,格式都为一个基因一行。



提交后显示基因信息, 其它按钮为灰色, 表示尚未分析

■ 基因信息



如有红字"Attention"提示有基因符号对应了多个基因,可进行修正:

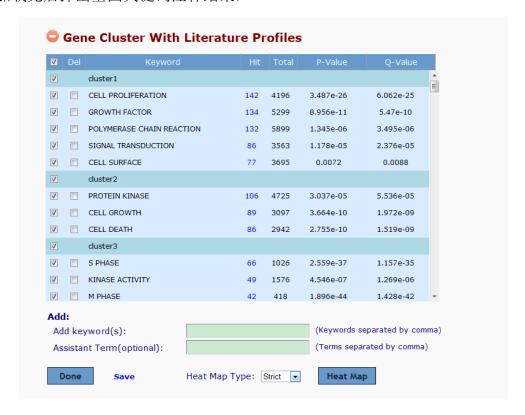


■ 基因功能注释和聚类

点击"Gene Cluster With Literature Profiles"按钮,短暂的等待后弹出聚类结果。



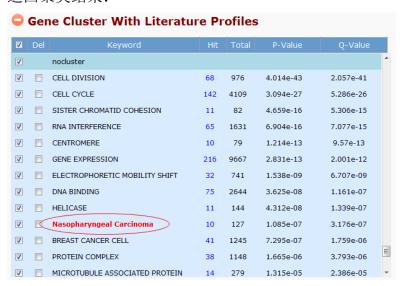
加载完后弹出基因关键词注释结果,



添加新的词进行注释,可同时输入多个词,以逗号隔开:



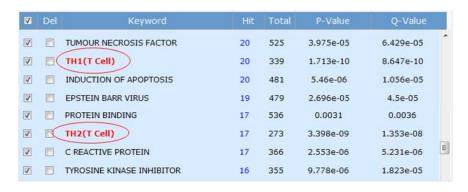
重新搜索、返回聚类结果:



添加新的词并且需要辅助词进行注释:



重新搜索、返回聚类结果:



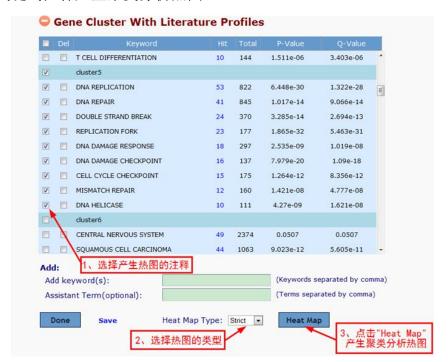
去除不需要的关键词注释:



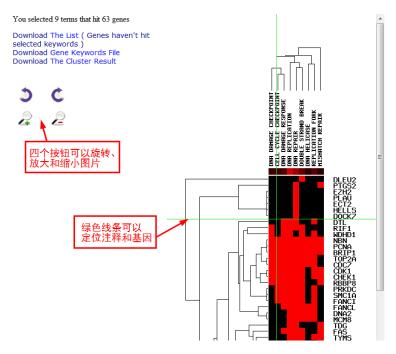
去除了所选注释, 聚类结果随之发生变化



选择关键词注释产生聚类分析热图:



聚类分析结果:

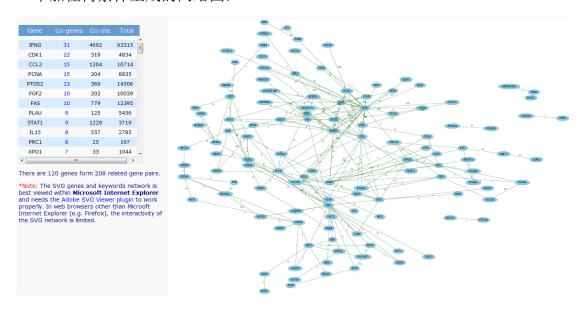


■ 构建文献基因网络

点击"Literature Mining Gene Network", 打开基因网络构建的界面:



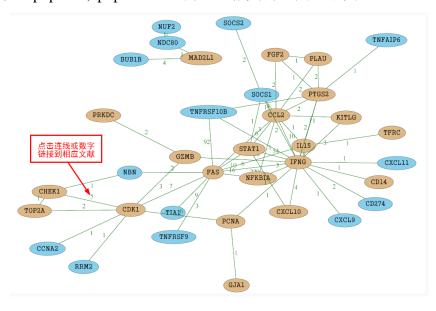
不加任何条件生成的网络图:



构建与自由词相关的基因网络,并且在网络中显示已知的基因,如果想用某个主题的多个词构建网络,以逗号将词隔开。



生成与"apoptosis,apoptotic" (凋亡) 相关的基因网络图:



CDK1 和 CHEK1 相互作用,并且搜索词与它们共同出现在一个句子的文献:

Source: Literature mining 🛧 点击查看此基因对的来源

Gene: CDK1
Alias : cell division cycle 2 G1 to S and G2 to M; P34CDC2; CDK1; cell division protein kinase 1; cell cell cycle controller CDC2; cell division cycle 2, G1 to S and G2 to M; CDC2; cyclin-dependent kinase 1; p34 protein kinase; cell division control protein 2 homolog; CDC28A

Gene: CHEK1

Alias: Checkpoint, S. pombe, homolog of, 1; serine/threonineprotein kinase Chk1; CHEK1; CHK1; CHK1 checkpoint homolog; CHK1

homolog

Search word(s): apoptosis,apoptotic

Click here to get abstracts about Nasopharyngeal Carcinoma, NPC and CDK1 CHEK1

点击查看与"Known Genes"搜索词 相关的已知基因的文献 1 PMID: 18981479

Genes Dev. 2008 Nov 1. 12(1) 35년 강조 전략 기소 때 Differentiation of trophoblast stem cells into giant cells is triggered by p57/Kip2 inhibition of CDK1 activity.

Ullah Z. Kohn MJ. Yaqi R. Vassilev LT. DePamphilis ML.

National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland 20892, USA.

Genome endoreduplication during mammalian development is a rare event for which the mechanism is unknown. It first appears when fibroblast growth factor 4 (FGF4) deprivation induces differentiation of trophoblast stem (TS) cells into the nonproliferating trophoblast giant (TG) cells required for embryo implantation. Here we show that R03306 inhibition of cyclin-dependent protein kinase 1 (CDK1), the enzyme required to enter mitosis, induced impariation. Here we show that RU330b inhibition of cyclin-dependent protein kinase 1 (CDK1), the enzyme required to enter mitosis, induced differentiation of TS cells into TG cells. In contrast, R0330b induced abortive endorequipication and apoptosis in embryonic stem cells, revealing that inactivation of CDK1 triggers endoreduplication only in cells programmed to differentiate into polyploid cells. Similarly, FGF4 deprivation resulted in CDK1 inhibition by overexpressing two CDK-specific inhibitors, p57/KIP2 and p21/CIP1. TS cell mutants revealed that p57 was required to trigger endoreduplication by inhibiting CDK1, while p21 suppressed expression of the checkpoint protein kinase CHK1, thereby preventing induction of apoptosis. Furthermore, Cdk2(-/-) TS cells revealed that CDK2 is required for endoreduplication when CDK1 is inhibited. Expression of p57 in TG cells was restricted to G-phase nuclei to allow CDK activation of S phase. Thus, endoreduplication in TS cells is triggered by p57 inhibition of CDK1 with province that the DNA damage response by p21. with concomitant suppression of the DNA damage response by p21.

Journal Article.

点击 "Literature mining"的结果:

Gene: CDK1
Alias: cell division cycle 2 G1 to S and G2 to M; P34CDC2; CDK1; cell division protein kinase 1; cell cycle controller CDC2; cell division cycle 2, G1 to S and G2 to M; CDC2; cyclin-dependent kinase 1; p34 protein kinase; cell division control protein 2 homolog; CDC28A

Gene: CHEK1 Alias: Checkpoint, S. pombe, homolog of, 1; serine/threonine-protein kinase Chk1; CHEK1; CHK1; CHK1 checkpoint homolog; CHK1 homolog

1 PMID: 16629900

Genes Cells. 2006 May;11(5):477-85.

Regulation of mitotic function of Chk1 through phosphorylation at novel sites by cyclin-dependent kinase 1 (Cdk1).

Shiromizu T, Goto H, Tomono Y, Bartek J, Totsukawa G, Inoko A, Nakanishi M, Matsumura F, Inagaki M.

Division of Biochemistry, Aichi Cancer Center Research Institute, Nagoya, Aichi 464-8681, Japan

Chk1 is phosphorylated at Ser317 and Ser345 by ATR in response to stalled replication and genotoxic stresses. This Chk1 activation is thought to play critical roles in the prevention of premature mitosis. However, the behavior of Chk1 in mitosis remains largely unknown. Here we reported that Chk1 was phosphorylated in mitosis. The reduction of this phosphorylation was observed at the metaphase-anaphase transition. Two-dimensional phosphopeptide mapping revealed that Clk1 phosphorylation sites in vivo were completely overlapped with the in vitro sites by cyclin-dependent protein kinase (Cdk) 1 or by p38 MAP kinase. Ser286 and Ser301 were identified as novel phosphorylation sites on Chk1. Treatment with Cdk inhibitor butyrolactone I induced the reduction of Chk1-S301 phosphorylation, although treatment with p38-specific inhibitor SB203580 or siRNA did not. In addition, ionizing radiation (IR) or ultraviolet (UV) light did not induce Chk1 phosphorylation at Ser317 and Ser345 in nocodazole-arrested mitotic cells. These observations imply the regulation of mitotic Chk1 function through Chk1 phosphorylation at novel sites by Cdk1.

Journal Article. Research Support, Non-U.S. Gov't.

<<First <Prev Page 1 of 1 Next> Last>>

点击 "Click here"的结果,这两个基因已知与鼻咽癌相关:

Search word(s): Nasopharyngeal Carcinoma, NPC

Gene		
CDK1	13	4834
CHEK1	2	1276

Gene: CDK1

Gene: CDK1

Alias: cell division cycle 2 G1 to S and G2 to M;

Basic Scale division cycle 2 G1 to S and G2 to M;

Basic Scale division cycle 2 G1 to S and G2 to M;

Basic Scale division cycle 2 G1 to S and G2 to M;

Basic Scale division cycle 2 G1 to S and G2 to M;

Basic Scale G2 to M

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1. PMID: 20711190 Nat Struct Mol Biol. 2010 Sep;17(9):1065-71.

Nuclear pore formation but not nuclear growth is governed by cyclin-dependent kinases (Cdks) during interphase. Maeshima K, Iino H, Hihara S, Funakoshi T, Watanabe A, Nishimura M, Nakatomi R, Yahata K, Imamoto F,

Hashikawa T, Yokota H, Imamoto N

Cellular Dynamics Laboratory, RIKEN Advanced Science Institute, Wako, Saitama, Japan. kmaeshim@lab.nig.ac.jp

Nuclear volume and the number of nuclear pore complexes (NPCs) on the nucleus almost double during interphase in dividing cells. How these events are coordinated with the cell cycle is poorly understood, particularly in mammalian cells. We report here, based on newly developed techniques for visualizing NPC formation, that cyclin-dependent kinases (Cdks), especially Cdk1 and Cdk2, promote interphase NPC formation in human dividing cells. Cdks seem to drive an early step of NPC formation because Cdk inhibition suppressed generation of 'nascent pores', which we argue are immature NPCs under the formation process. Consistent with this, Cdk inhibition disturbed proper expression and localization of some nucleoporins, including Elys/Mel-28, which tridgers postmitotic NPC assembly. Strikingly, Cdk suppression did not notably affect nuclear growth.

■ GO和 Pathway 分析

点击 "GO Analysis"和 "Pathway Analysis", 进行分析和自动加载结果:



GO 分析结果,选择注释产生聚类分析热图与关键词注释中的操作一致。



Pathway Analysis 结果,解释和其他操作与 GO 的一致。



■ 词相关基因检索功能

Word Related Gene Search

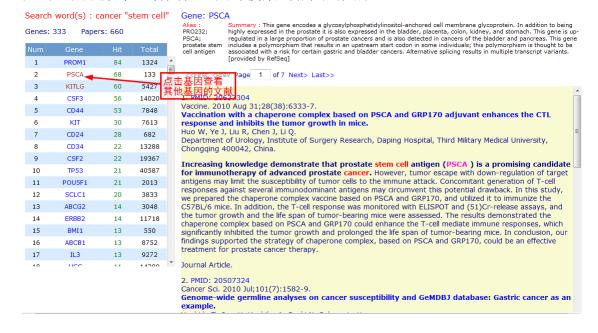
查找与检索词相关的基因,可以限定检索词与基因共同出现在同个句子或摘要,输入时不需要带除双引号外的其它标点符号,每个词之间用空格隔开,表示在同时出现这些词,词组用双引号表示。

选择搜索词与基因

出现在句子(或摘要)



点击基因或数字查看其他基因与搜索词相关的文献:



■ 回顾分析



AND

Co-occurence:

Sentence

Abstract

Gene network