GenCLiP 2.0 使用手册

简介

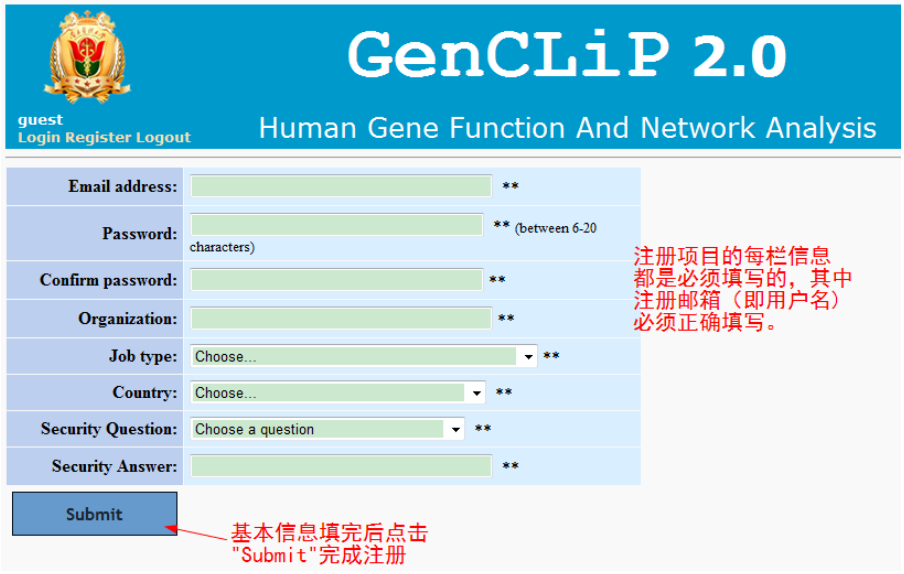
GenCLiP 2.0（http://ci.smu.edu.cn.）是一个基于文献挖掘的网络服务平台，主要有 4 大功能模块，包括基因功能注释、分子网络构建、GO 和 Pathway 分析和关键词相关基因搜索。

网络平台面向广大的生物医学工作者，包括国内外生物医药企业、科研机构、本专科院校、医疗机构、医药科研机构工作者和医疗服务工作者等，用户需适应全英文网页环境，具备良好的生物医学背景知识。

GenCLiP 2.0 兼容 IE、Safari、Chrome 等常用的浏览器，如浏览器不能显示基因网络图（由 Cytoscape Web 绘制），需从 http://get.adobe.com/flashplayer/下载最新的 Flash 插件安装。

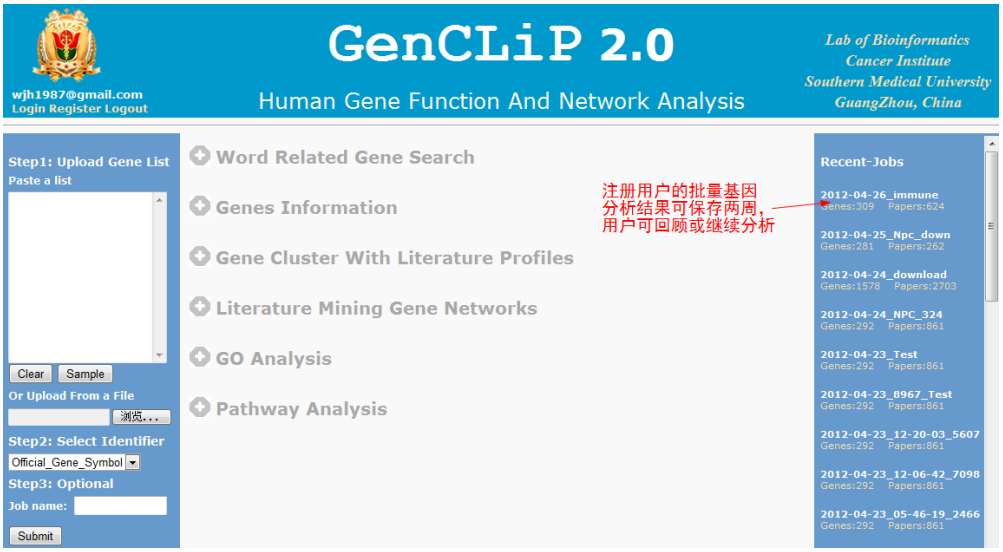
用户注册

进入填写注册基本信息的界面：



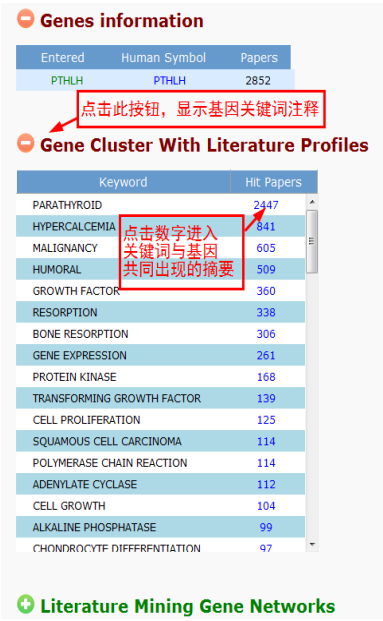
点击“Login”进入注册用户登陆界面：

注册用户分析界面：

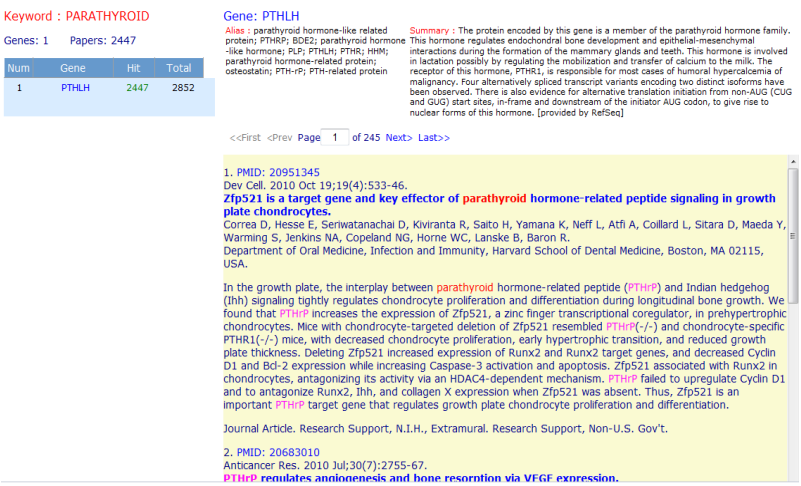


单个基因信息检索

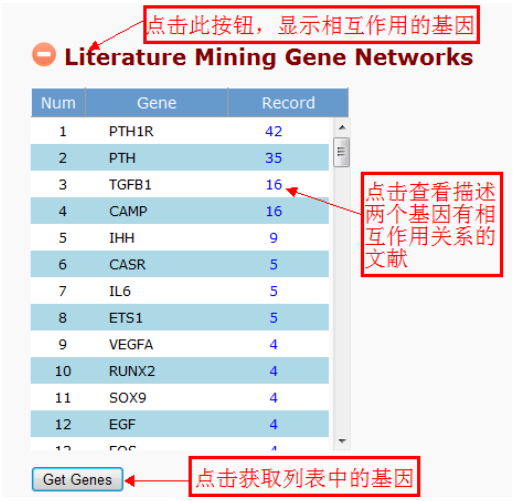
关键词：



查阅文献：



相互作用的基因：



点击 PTH1R 的记录数“42”,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-rP; PTH-related protein

Gene: **PTH1R**
Alias : parathyroid hormone/parathyroid hormone-related peptide receptor; parathyroid hormone/parathyroid hormone-related protein receptor; parathyroid hormone receptor 1; PTH/PTHrP type I receptor; PTH1 receptor; PTH1R; seven transmembrane helix receptor; PTHR1; PTH/PTHr receptor; parathyroid hormone 1 receptor; PTHR; PFE

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

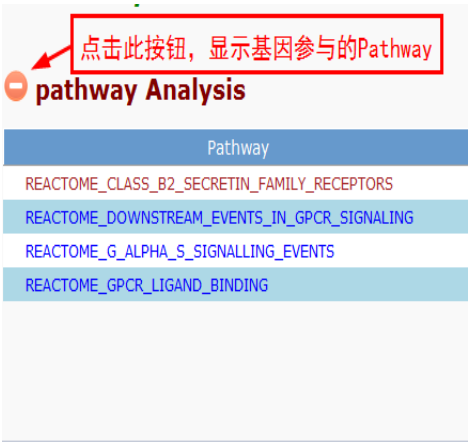
Parathyroid hormone-related peptide (PTHrP), which frequently causes the humoral hypercalcemia of malignancy syndrome, is an autocrine/paracrine regulator of chondrocyte proliferation and differentiation that acts through the PTH/PTHrP receptor (PTH1R). PTHrP is generated in response to Indian hedgehog (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 endochondral 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 PTHrP or the PTH1R, it appears plausible that impaired expression of PTHrP 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 growth plates and impaired growth, but it remains uncertain whether this contributes to altered chondrocyte growth and differentiation.

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

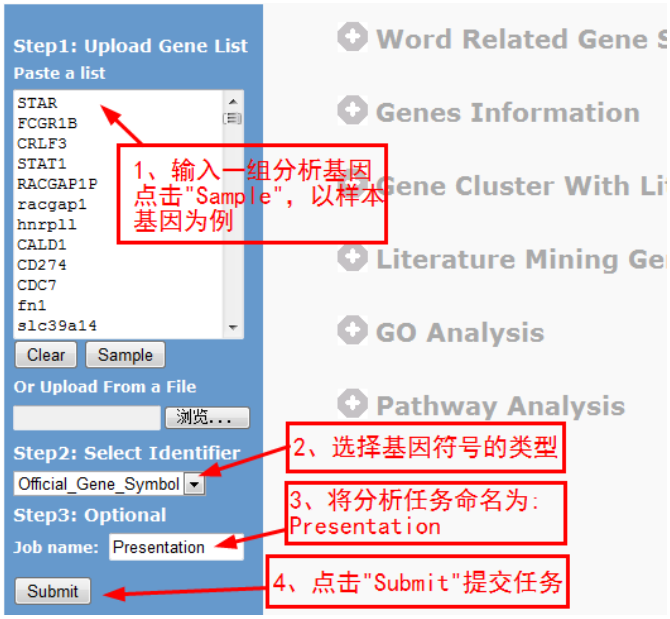
GO 和 Pathway 注释：



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。

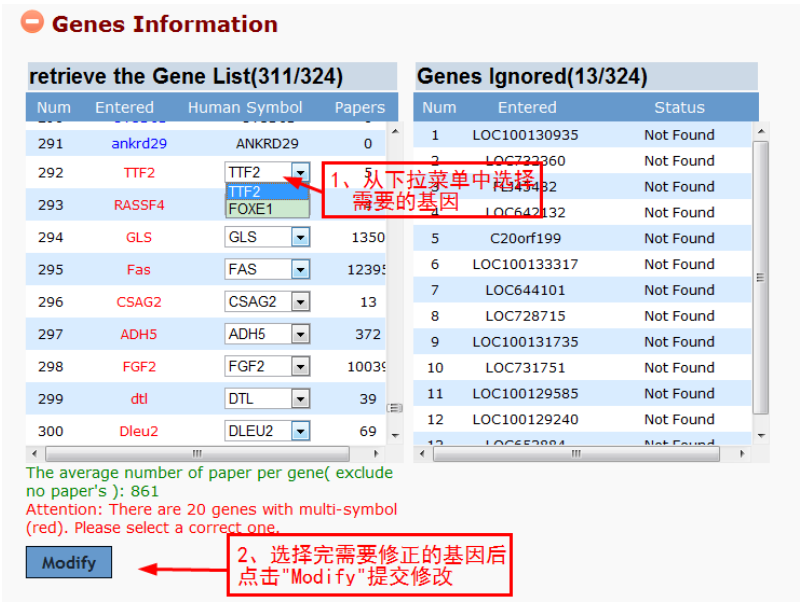
无论是在输入框输入还是以文本文件（txt 格式）上传一组分析基因，格式都为一个基因一行。



提交后显示基因信息，其它按钮为灰色，表示尚未分析

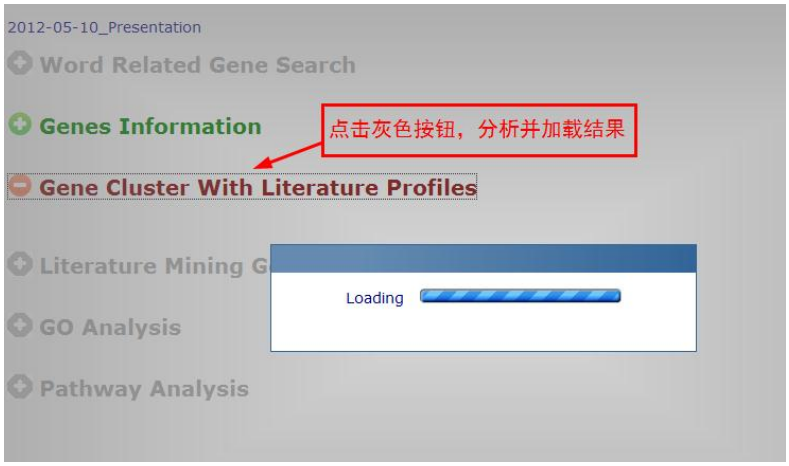
基因信息

如有红字“Attention”提示有基因符号对应了多个基因，可进行修正：

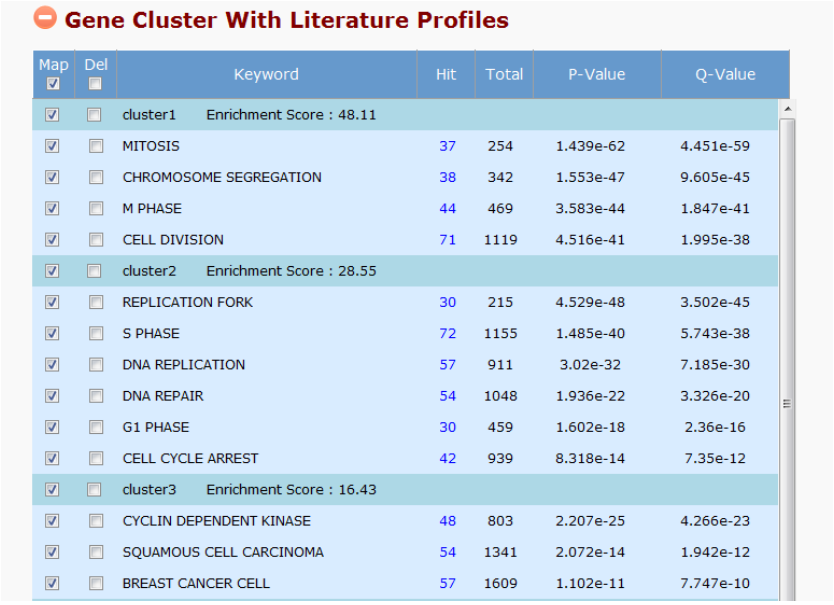


基因功能注释和聚类

点击“Gene Cluster With Literature Profiles”按钮，等待数秒后弹出聚类结果。



加载完后弹出基因关键词注释结果：



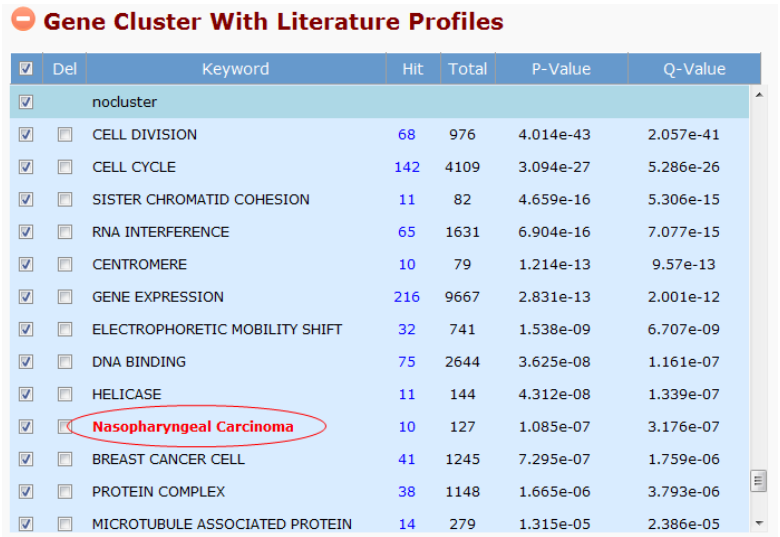
Gene Cluster With Literature Profiles

Map	Del	Keyword	Hit	Total	P-Value	Q-Value
☑	☐	cluster1 Enrichment Score : 48.11				
☑	☐	MITOSIS	37	254	1.439e-62	4.451e-59
☑	☐	CHROMOSOME SEGREGATION	38	342	1.553e-47	9.605e-45
☑	☐	M PHASE	44	469	3.583e-44	1.847e-41
☑	☐	CELL DIVISION	71	1119	4.516e-41	1.995e-38
☑	☐	cluster2 Enrichment Score : 28.55				
☑	☐	REPLICATION FORK	30	215	4.529e-48	3.502e-45
☑	☐	S PHASE	72	1155	1.485e-40	5.743e-38
☑	☐	DNA REPLICATION	57	911	3.02e-32	7.185e-30
☑	☐	DNA REPAIR	54	1048	1.936e-22	3.326e-20
☑	☐	G1 PHASE	30	459	1.602e-18	2.36e-16
☑	☐	CELL CYCLE ARREST	42	939	8.318e-14	7.35e-12
☑	☐	cluster3 Enrichment Score : 16.43				
☑	☐	CYCLIN DEPENDENT KINASE	48	803	2.207e-25	4.266e-23
☑	☐	SQUAMOUS CELL CARCINOMA	54	1341	2.072e-14	1.942e-12
☑	☐	BREAST CANCER CELL	57	1609	1.102e-11	7.747e-10

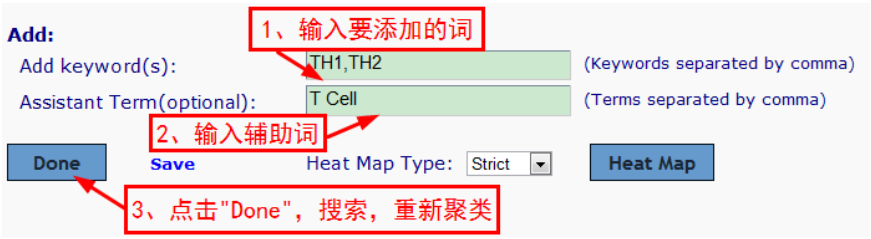
可更改默认参数对关键词结果筛选再聚类：

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：

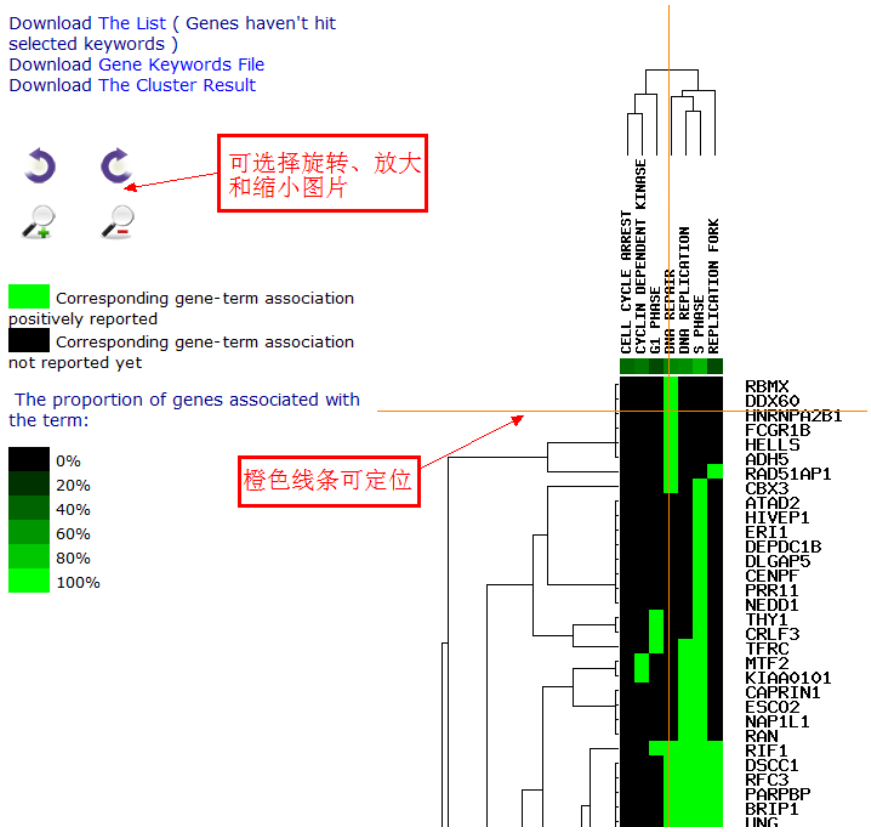


重新搜索、返回聚类结果：

去除不需要的关键词注释：

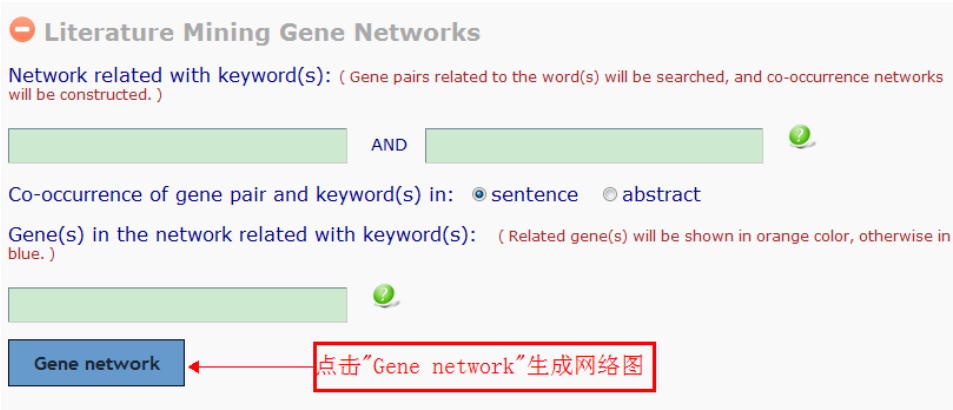
选择关键词注释产生聚类分析热图：

聚类分析结果：

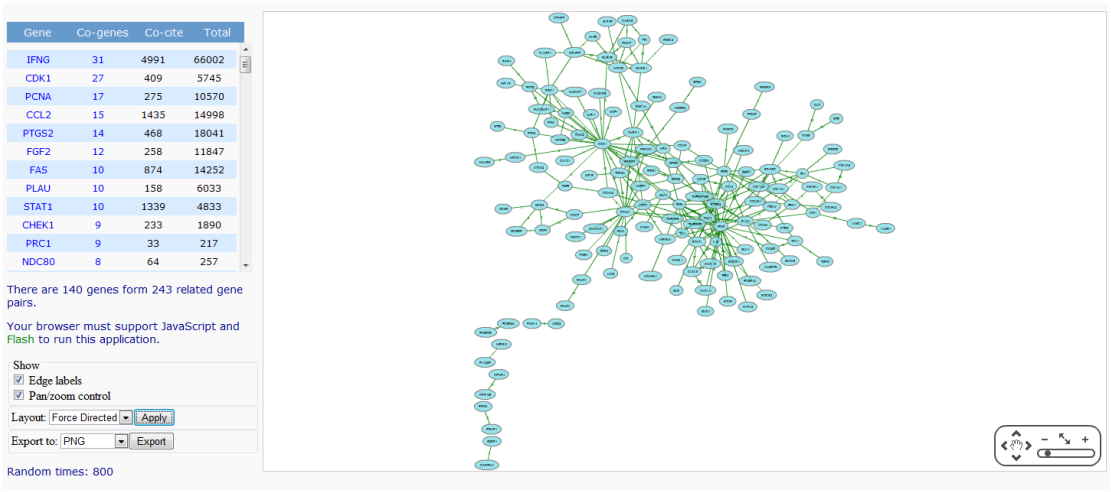


构建基因网络

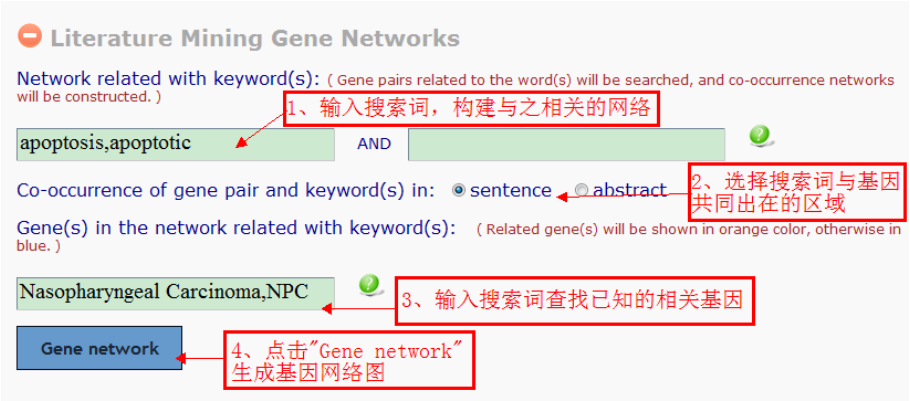
点击“Literature Mining Gene Network”，打开基因网络构建的界面：



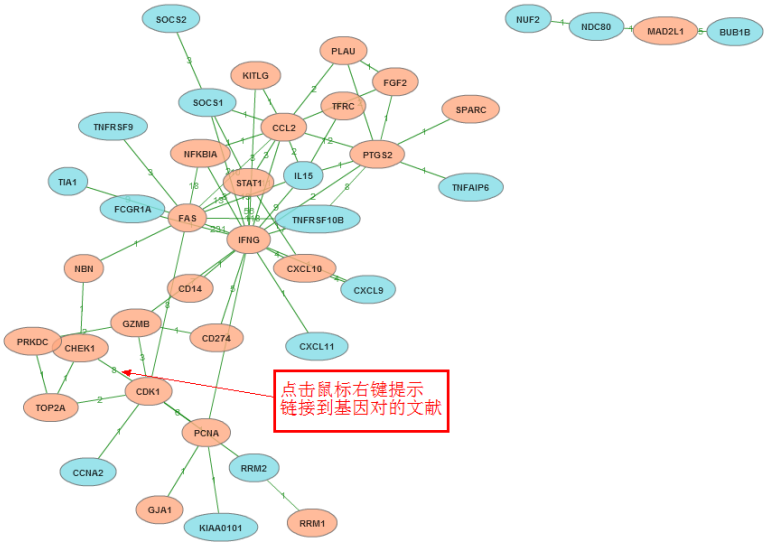
不加任何条件生成的网络图：



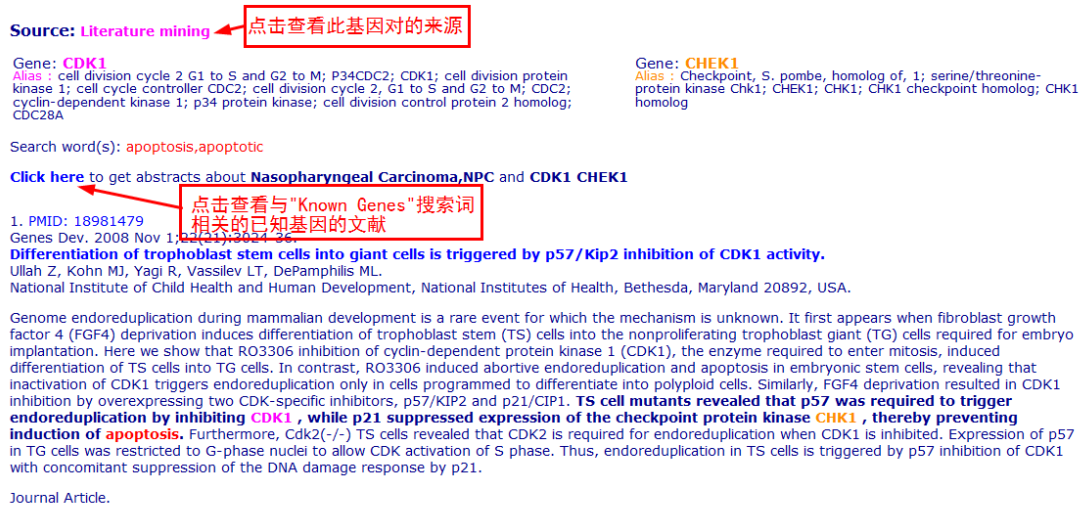
构建与自由词相关的基因网络，并且在网络中显示已知的基因，如果想用某个主题的多个词构建网络，以逗号将词隔开。



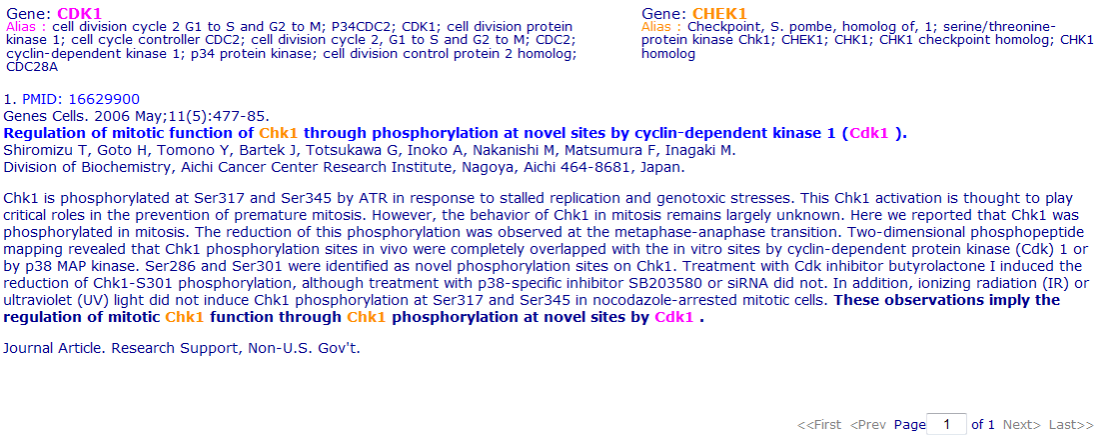
生成与“apoptosis,apoptotic”（凋亡）相关的基因网络图，并在网络中显示与"Nasopharyngeal Carcinoma,NPC"（鼻咽癌）有关的基因（橙色）：



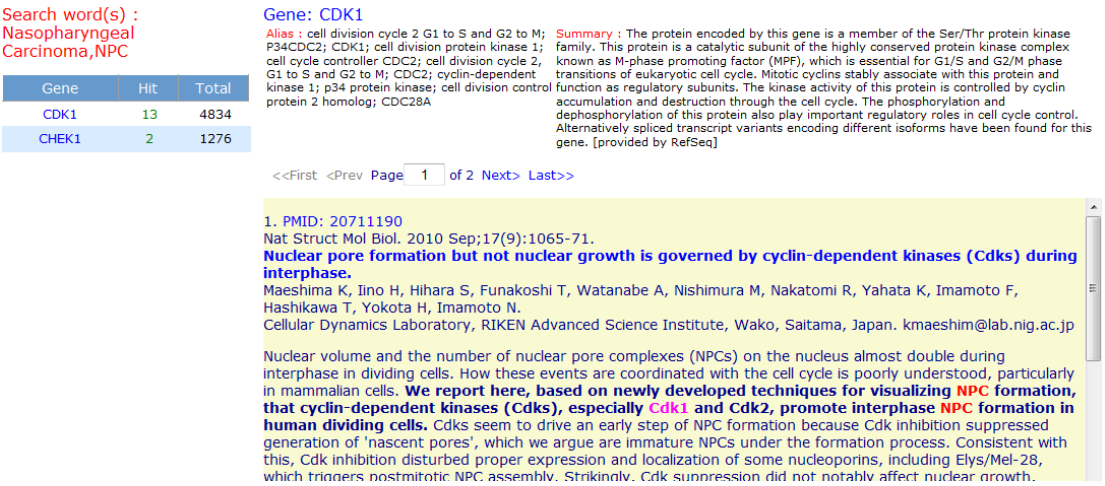
CDK1 和 CHEK1 相互作用，并且搜索词与它们共同出现在一个句子的文献：



点击“Literature mining”的结果：

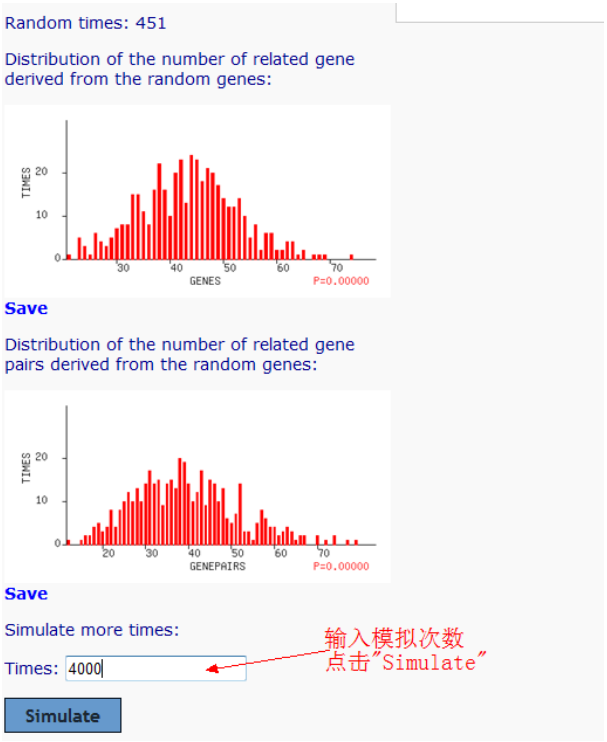


点击“Click here”的结果，这两个基因已知与鼻咽癌相关：

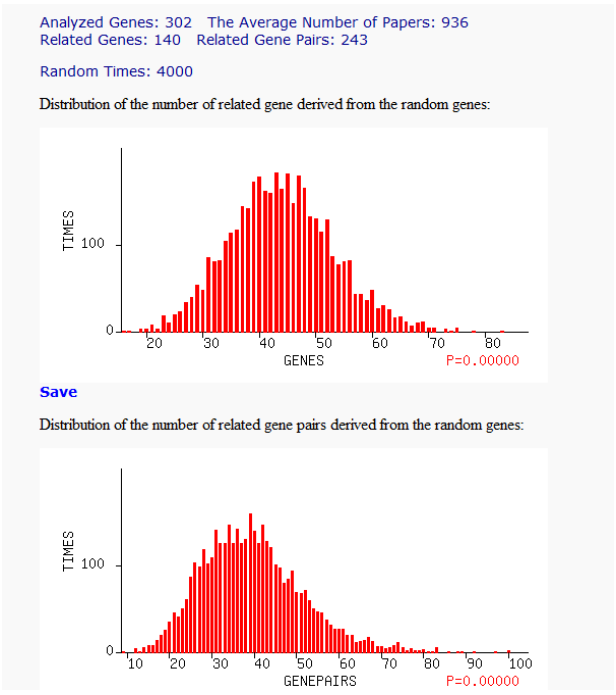


随机模拟

首次随机模拟结果与基因网络的结果同时给出，模拟次数限制在 1000 次以内，用户可以输入模拟次数（不超过首次次数的 10 倍）重新模拟，计算可能比较耗时，具体与基因数量和模拟次数有关，一般不超过 5 分钟。



结果在新的页面：

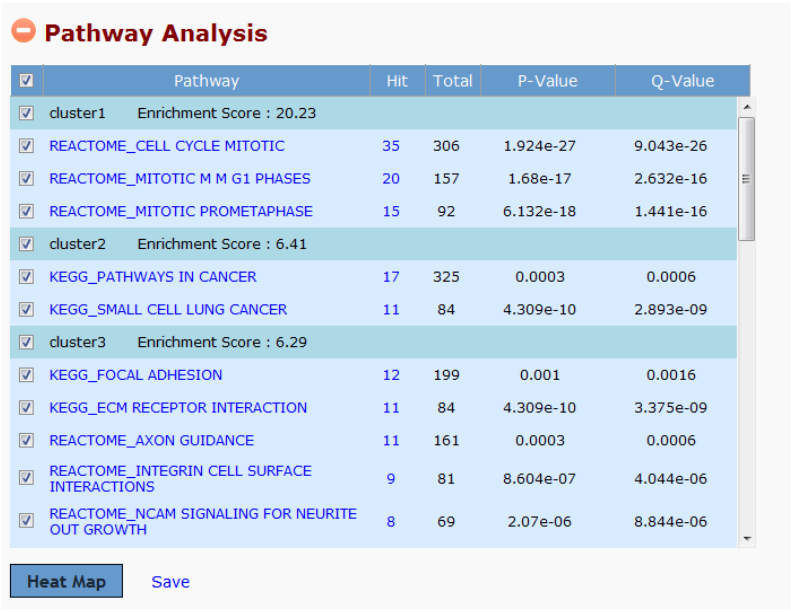


GO 和 Pathway 分析

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

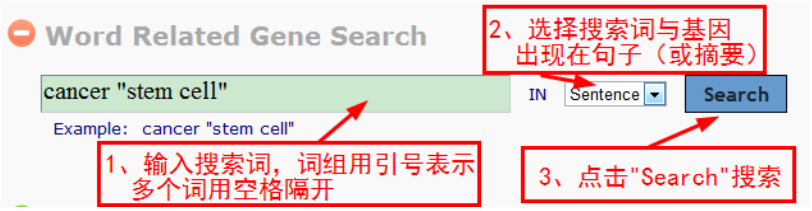
GO 分析结果，选择注释产生聚类分析热图与关键词注释中的操作一致。

Pathway Analysis 结果，解释和其他操作与 GO 的一致。

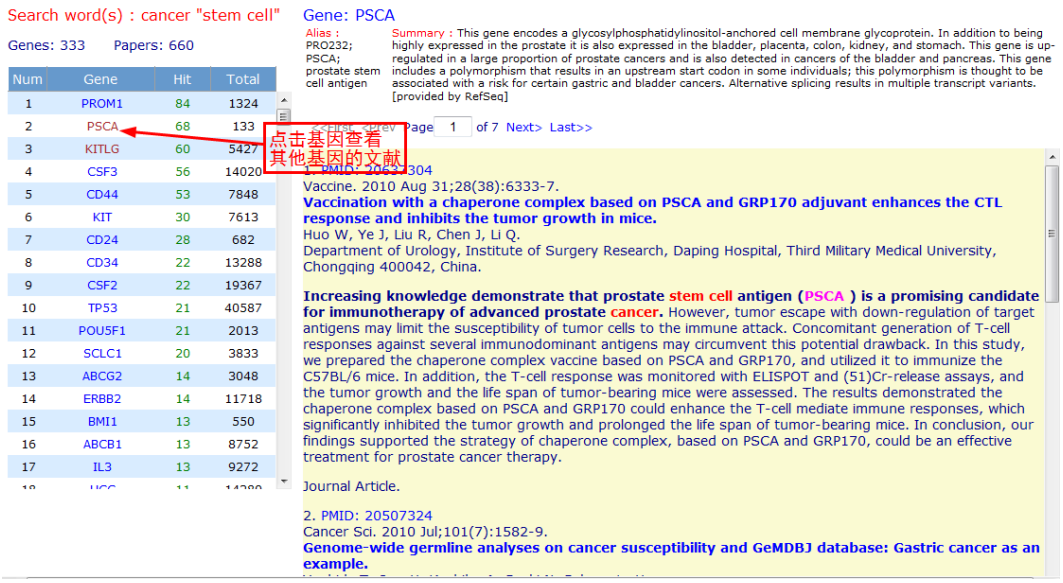


词相关基因检索功能

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。



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

注册用户可以查看近期的分析，或继续分析，分析档案保留两周。

回顾构建好的基因网络：