Supplementary Information for:

Biomarker Discovery for Immunotherapy of Pituitary Adenomas: Enhanced Robustness and Prediction Ability by Modern Computational Tools

Qingxia YANG[1,2], Yunxia WANG[2], Song ZHANG[2], Jing TANG[1,2], Fengcheng LI[2], Jiayi YIN[2], Yi LI[2], Jianbo FU[2], Bo LI[1], Yongchao LUO[2], Weiwei XUE[1,*] and Feng ZHU[1,2,*]

[1] Innovative Drug Research and Bioinformatics Group, School of Pharmaceutical Sciences, Chongqing University, Chongqing 401331, China

[2] Innovative Drug Research and Bioinformatics Group, College of Pharmaceutical Sciences, Zhejiang University, Hangzhou 310058, China

* Corresponding author: Prof. Feng ZHU (zhufeng@zju.edu.cn; prof.zhufeng@gmail.com)

Supplementary Table 1. Relevance between PA and the top-ranked (top-5 up & another top-5 down regulated) DEGs identified in this study confirmed by published literatures. N.A.: not available.

Gene Entrez	Gene Symbol	LogFC	*p*-value	Relevance between the Identified DEGs and PA Confirmed by the Comprehensive Literature Reviews
A: Strategy of Single Dataset (GSE51618)				
4435	CITED1	2.97	8.79E-03	N.A.
1191	CLU	2.91	1.13E-02	CLU expression in pituitary adenomas was found significantly higher than in non-neoplastic adenohypophyses[1]
1442	CSH1	-10.29	1.72E-06	N.A.
1443	CSH2	-11.09	4.12E-07	N.A.
1444	CSHL1	-10.39	4.13E-08	N.A.
8788	DLK1	-9.29	4.15E-05	DLK1 expression was reduced 2500-fold in nonfunctioning pituitary adenomas, compared with normal pituitary[2]
56884	FSTL5	3.25	1.23E-02	N.A.
2688	GH1	-11.17	1.71E-06	GH-immunoreactivity was detected in most adenoma cells in the cases of unusual chromophobic adenomas[3]
3006	HIST1H1C	2.87	4.06E-02	N.A.
140679	SLC32A1	3.20	3.59E-04	N.A.
B: Strategy of Single Dataset (GSE26966)				
158763	ARHGAP36	-7.60	2.21E-09	N.A.
339479	BRINP3	4.84	1.20E-05	BRINP3 was overexpressed in pituitary adenomas, compared with the normal people[4]
389073	C2orf80	4.81	2.12E-04	N.A.
1442	CSH1	-8.28	3.15E-25	N.A.

79695	GALNT12	4.08	3.59E-07	N.A.
2688	GH1	-9.71	1.46E-16	It was showed a faint GH1-immunoreactivity in pituitary adenomas[3]
5443	POMC	-8.00	1.00E-17	In silent subtype 2 and 3 pituitary adenomas, POMC had a diffuse low level or was absent, compared to the normal[5]
5617	PRL	-7.33	1.49E-07	PRL gene expression was rare pituitary adenomas[6]
201780	SLC10A4	4.17	7.53E-06	N.A.
91752	ZNF804A	4.91	3.36E-09	N.A.
C: DDI Strategy Integrating Five Datasets (GSE22812, GSE26966, GSE4237, GSE46311 and GSE51618)				
23305	ACSL6	0.91	3.51E-03	N.A.
1056	CEL	-2.22	9.21E-10	CEL expression was lacking in PRL cells pituitary adenomas, which is different from the normal pituitary[7]
1641	DCX	0.91	6.76E-04	DCX expression was positively correlated with N-cadherin, which was increased in pituitary adenomas[8,9]
51083	GAL	-2.69	4.22E-21	Different from normal pituitary, GAL was rarely expressed in somatotroph adenomas and prolactinomas[10]
29899	GPSM2	0.91	1.73E-02	PA pathogenesis is regulated by PI3K/AKT, which is the key pathway activated by GPSM2 during tumor growth[11,12]
3240	HP	-2.21	1.06E-09	The levels of five isoforms of HP were significantly decreased after GH substitution in PA patients[13]
5149	PDE6H	0.89	1.67E-02	N.A.
5443	POMC	-3.85	2.79E-18	In silent subtype 2 and 3 pituitary adenomas, POMC had a diffuse low level or was absent, compared to the normal[5]
55752	SEPT11	0.87	1.13E-02	N.A.
7252	TSHB	-3.26	6.65E-16	The mRNA expression of TSHB was absent in most PA patients[14]

Supplementary Table 2. Relevance between PA and the top-10 KEGG pathways identified here confirmed by published literatures. N.A.: not available.

KEGG Pathway	*p*-value	Relevance between SCZ and the Enriched Pathways Confirmed by the Comprehensive Literature Reviews
A: Strategy of Single Dataset (GSE51618)		
Alzheimer's disease	2.00E-12	N.A.
Oxidative phosphorylation	1.96E-09	Oxidative phosphorylation is revealed as the significant pathway in human pituitary adenoma by DEP data[15]
Huntington's disease	8.43E-09	N.A.
Phosphatidylinositol signaling system	1.96E-08	Phosphatidylinositol signaling system was found to be disturbed in pituitary tumors[16]
Oocyte meiosis	4.60E-08	Differentially expression genes in pituitary gonadotroph adenomas were enriched in oocyte meiosis pathway[17]
Parkinson's disease	9.81E-08	N.A.
Citrate cycle/TCA cycle	9.81E-08	N.A.
Pathways in cancer	1.54E-07	N.A.
Axon guidance	2.34E-07	N.A.
Focal adhesion	3.39E-07	N.A.
B: Strategy of Single Dataset (GSE26966)		
MAPK signaling pathway	2.57E-10	MAPK-signaling abnormality was demonstrated to be significantly associated with a pituitary adenoma[15]
Pathways in cancer	3.74E-10	N.A.
Alzheimer's disease	2.74E-08	N.A.
Axon guidance	3.69E-07	N.A.

DNA replication	5.26E-07	N.A.
Cardiac muscle contraction	5.26E-07	Cardiac muscle contraction is found to be significantly enriched pathway in pituitary adenoma[18]
Endocytosis	1.32E-06	Among pituitary adenoma, clathrin- and caveolar-mediated endocytosis were statistically significant pathways[19]
Huntington's disease	1.51E-06	N.A.
Cell cycle	2.22E-06	Pathogenetic mechanisms of pituitary tumorigenesis is genetic or epigenetic and result in cell cycle dysregulation[20]
Adipocytokine signaling pathway	2.22E-06	N.A.
C: DDI Strategy Integrating Five Datasets (GSE22812, GSE26966, GSE4237, GSE46311 and GSE51618)		
TGF-beta signaling pathway	2.83E-06	TGF-beta signaling pathway was significantly enriched for the genes of plurihormonal pituitary adenomas[21]
Ribosome	2.83E-06	Genes encoding ribosomal proteins were under-expressed in GH-secreting pituitary adenomas[22]
MAPK signaling pathway	2.83E-06	MAPK-signaling abnormality was significantly associated with pituitary adenoma[15]
Complement and coagulation cascades	4.15E-06	Genes in complement and coagulation cascades were upregulated in high MGMT expressing pituitary adenomas[23]
Pathways in cancer	2.08E-05	N.A.
Cell cycle	3.28E-05	Pathogenetic mechanisms of pituitary tumorigenesis is genetic or epigenetic and result in cell cycle dysregulation[20]
P53 signaling pathway	3.87E-05	P53 signaling pathway plays an important role in tumorigenesis and progression of plurihormonal pituitary adenomas[21]
Axon guidance	2.49E-04	N.A.
PPAR signaling pathway	4.23E-04	N.A.
Leukocyte transendothelial migration	9.45E-04	Leukocyte transendothelial migration was found to be an important pathway in pituitary adenomas[24]

Supplementary Table 3. Relevance between PA and the top-10 GO biological processes (BPs) identified here confirmed by literatures. N.A.: not available.

GO Biological Process	*p*-value	Relevance between PA and the Enriched GO BPs Confirmed by the Comprehensive Literature Reviews
A: Strategy of Single Dataset (GSE51618)		
Small molecule metabolic process	9.39E-41	N.A.
Neurogenesis	2.53E-37	N.A.
Intracellular signal transduction	4.72E-36	Raf/MEK/ERK signalling has been considered to be one of the major and central intracellular signal transduction pathways in pituitary adenomas aetiology[25]
Protein complex subunit organization	4.72E-36	Interleukin 4 receptor complex (including IL-4Ralpha, IL-13Ralpha1 and IL-2Rgammac chains) was overexpressed in invasive pituitary adenomas [26]
Regulation of protein modification process	4.72E-36	DNMT3b, which generated 5-methylcytosine by adding a methyl group directly to a cytosine base, was upregulated in PAs via histone modification[27]
Regulation of transport	2.71E-35	The miRNAs microarray results of GO analysis indicated positive regulation of sodium ion transport [28]
Phosphate containing compound metabolic process	2.47E-34	N.A.
Positive regulation of molecular function	3.23E-34	N.A.
Response to endogenous stimulus	6.87E-34	The cellular Ca2+ transport was associated with stimulus-secretion coupling in prolactin producing rat pituitary adenoma cells[29]
Regulation of phosphorus metabolic process	2.59E-33	N.A.
B: Strategy of Single Dataset (GSE26966)		
Regulation of transport	7.33E-74	The miRNAs microarray results of GO analysis indicated positive regulation of sodium ion transport[28]
Neurogenesis	7.9E-73	N.A.
Cellular response to organic substance	1.62E-71	The results of the GO analysis from normal pituitary and PA tissues indicated the functions of the downregulated genes were associated with the response to organic substance [30]
Response to oxygen containing compound	2.47E-70	N.A.

Regulation of multicellular organismal development	2.63E-69	N.A.
Regulation of cell differentiation	3.3E-69	Epigenetic chromatin structures were involved in regulation of the differentiation of pituitary adenomas [31]
Intracellular signal transduction	4.11E-69	The Raf/MEK/ERK signaling has been considered to be one of the major and central intracellular signal transduction pathways in pituitary adenomas aetiology [25]
Tissue development	6.09E-67	N.A.
Small molecule metabolic process	2.29E-66	N.A.
Response to endogenous stimulus	8.85E-66	The cellular Ca2+ transport was associated with stimulus-secretion coupling in prolactin producing rat pituitary adenoma cells [29]
C: DDI Strategy Integrating Five Datasets (GSE22812, GSE26966, GSE4237, GSE46311 and GSE51618)		
Regulation of cell proliferation	2.72E-38	Immunostaining assay indicated AIB1 participated in the regulation of proliferation of tumoral cells in prolactinomas[32]
Response to endogenous stimulus	2.62E-31	The cellular Ca2+ transport was associated with stimulus-secretion coupling in prolactin producing rat pituitary adenoma cells[29]
Response to external stimulus	5.24E-31	In vitro leptin stimulation of pituitary tumors caused stimulation of FSH and alpha-subunit secretion from a non-functioning adenoma and TSH secretion from a somatotroph adenoma [33]
Tissue development	1.2E-28	N.A.
Positive regulation of response to stimulus	3.64E-27	The results of the GO analysis from blood samples from nine patients with PAs and seven healthy controls indicated the positive regulation of response to stimulus [34]
Positive regulation of cell communication	6.54E-27	Interaction of TGF-beta and b-FGF facilitated the communication between lactotropes and folliculo-stellate cells in prolactinoma formation [35]
Regulation of cell death	2.57E-26	N.A.
Cellular response to organic substance	1.84E-25	The results of the GO analysis from normal pituitary and PA tissues indicated the functions of the downregulated genes were associated with the response to organic substance [30]
Response to oxygen containing compound	1.84E-25	N.A.
Regulation of cell differentiation	2.49E-25	Epigenetic chromatin structures were involved in regulation of the differentiation of pituitary adenomas[31]

Supplementary Table 4. Relevance between PA and the top-10 GO molecular functions (MFs) identified here confirmed by literatures. N.A.: not available.

GO Molecular Function	*p*-value	Relevance between PA and the Enriched GO MFs Confirmed by the Comprehensive Literature Reviews
A: Strategy of Single Dataset (GSE51618)		
Enzyme binding	2.26E-37	N.A.
Protein dimerization activity	1.48E-34	N.A.
Macromolecular complex binding	3.48E-34	It has been shown that L-triiodothyronine can be bound with a macromolecular complex in pituitary tumor cells[36]
Identical protein binding	3.67E-30	N.A.
Receptor binding	1.36E-29	The research of nonfunctioning pituitary adenomas showed epidepride was specifically bound with D2 receptor[37]
Ribonucleotide binding	1.06E-28	The significantly enriched GO term for molecular functions of DEGs between PAs and normal control tissues was guanyl ribonucleotide binding[18]
Transporter activity	2.90E-22	N.A.
Transmembrane transporter activity	4.62E-22	N.A.
Molecular function regulator	1.10E-21	mTOR pathway regulators was significantly correlated with the invasion, staging, and tumor growth of pituitary adenomas[38]
Adenyl nucleotide binding	2.78E-21	N.A.
B: Strategy of Single Dataset (GSE26966)		
Receptor binding	1.15E-53	The research of nonfunctioning pituitary adenomas showed epidepride was specifically bound with D2 receptor [37]
Enzyme binding	1.15E-53	N.A.
Transporter activity	6.52E-46	N.A.
Protein dimerization activity	4.58E-45	N.A.

Molecular function regulator	3.07E-42	mTOR pathway regulators was significantly correlated with the invasion, staging, and tumor growth of pituitary adenomas[38]
Macromolecular complex binding	7.40E-42	It has been shown that L-triiodothyronine can be bound with a macromolecular complex in pituitary tumor cells[36]
Ribonucleotide binding	8.78E-42	The significantly enriched GO term for molecular functions of DEGs between PAs and normal control tissues was guanyl ribonucleotide binding [18]
Transmembrane transporter activity	1.67E-40	N.A.
Identical protein binding	1.20E-38	N.A.
Lipid binding	1.17E-33	N.A.
C: DDI Strategy Integrating Five Datasets (GSE22812, GSE26966, GSE4237, GSE46311 and GSE51618)		
Receptor binding	9.55E-20	The research of nonfunctioning pituitary adenomas showed epidepride was specificly bound with D2 receptor [37]
Identical protein binding	4.58E-14	N.A.
Macromolecular complex binding	1.19E-12	It has been shown that L-triiodothyronine can be bound with a macromolecular complex in pituitary tumor cells[36]
Molecular function regulator	1.22E-12	mTOR pathway regulators was significantly correlated with the invasion, staging, and tumor growth of pituitary adenomas[38]
Ribonucleotide binding	1.22E-12	The significantly enriched GO term for molecular functions of DEGs between PAs and normal control tissues was guanyl ribonucleotide binding[18]
Sulfur compound binding	1.11E-11	The compounds with polar sulfur functions retained the high binding affinity for the calf uterine estrogen receptor in pituitary tumor cells[39,40]
Structural molecule activity	1.11E-11	N.A.
Nucleic acid binding transcription factor activity	2.14E-11	N.A.
Heparin binding	2.14E-11	Heparin-binding secretory transforming gene overexpression in rat pituitary cells mediated lactotroph tumor growth and stimulates PRL transcription [41].
Transcription factor binding	2.14E-11	N.A.

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