Research Article

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2026.65.010193

Analyzing the Gene Expressions of the Olfactory System in Kidney Diseases

Aibing Rao

Shenzhen Luwei (Biomanifold) Biotechnology Limited, Shenzhen, PR China

***Corresponding author:** Aibing Rao, Shenzhen Luwei (Biomanifold) Biotechnology Limited, Shenzhen, PR China

ARTICLE INFO

Received: April 06, 2026

Published: April 29, 2026

Citation: Aibing Rao. Analyzing the Gene Expressions of the Olfactory System in Kidney Diseases. Biomed J Sci & Tech Res 65(3)-2026. BJSTR. MS.ID.010193.

ABSTRACT

Background: Early studies have shown that the kidney organ can also breathe through olfactory receptors, but the roles that the olfactory system may play are unclear. This study was aimed to analyze the gene expression profiles in the olfactory system as to various kidney diseases.

Methods: A public data set of eight types of kidney diseases and healthy controls (HC) was used for gene expression comparison in the olfactory system. Pairwise t-tests were performed and receiver operating characteristic (ROC) curves were plotted. The summary statistics concerning statistic significance were collected.

Results: Most of the olfactory receptor (OR) genes were down-regulated universally and four genes were down with statistic significance. Seven of ten adenylate cyclase genes were down- regulated while ADCY7 and ADCY3 were up-regulated for most disease types. In the selected G-protein α units, GNA11, GNAO1 were down-regulated while GNAI3 and GNAI1 were up- regulated. Moreover, OR1F2P, OR2W1 and OR7C1 were down mainly in systemic lupus erythematosus and OR2B2, OR2B6 and OR6A2 were down in rapidly progressive glomerulonephritis. OR12D3, OR3A2, OR3A3 and OR7C2 were down- regulated for both SLE and RPGN. The linear regression models gave rise to AUCs in between 0.75 and 1 with average 0.88.

Conclusion: The genes of the olfactory system showed very different expression profiles in kidney diseases compared to HC. Most genes were uniformly down expressed. Some genes were more related to specific diseases such as SLE and RPGN. The olfactory system had the expression abnormality in the diseases.

Abbreviations: ROC: Receiver Operating Characteristic; OR: Olfactory Receptor; HC: Healthy Controls; GPCRs: G Protein-Coupled Receptors; HC: Healthy Controls; SLE: Systemic Lupus Erythematosus; IGAN: IgA Nephropathy; RPGN: Rapidly Progressive Glomerulonephritis; HN: Hypertensive Nephropathy; MGN: Membranous Glomerulonephritis; DN: Diabetic Nephropathy; MCD: Minimal Change Disease; FSGS: Focal Segmental Glomerulosclerosis

Introduction

Olfactory receptors (ORs) are sensory G protein-coupled receptors (GPCRs) and have emerged as key contributors to kidney physiology. ORs have largely been understudied for their roles in kidney [1] and they were expressed in different areas of the nephron, regulating blood pressure, fibrosis, and filtration [2,3]. In the ordorant sensory signaling cascading, first ordorants bind with ORs, and then the trimeric olfactory G protein Golf dissociates into active α and βγ subunits where the α subunit triggers adenylate cyclase activation [3]. The cascading in kidney might be involved with pathogenesis. For example, loss of AC3 in mouse kidney resulted in abnormal glomerular filtration rate and plasma renin [4]. OR51E2, activated by short-chain fatty acids produced by gut microbiota, modulates renin release and blood pressure [5] and the intestinal response to colitis [6]. OR51E1 modulates sex-dependent renin alternation, vascular reactivity and arterial stiffness [7]. OR2Y1 participates kidney glucose handling via SGLT1[8] and contributes to the progression of type 2 diabetes [9]. The expressions of OR10AA1P, OR10A2, OR2Y1, OR1F1, and OR52P1 changed along with the progression of kidney fibrosis [10]. In addition, ectopic ORs have biological effects in cardiovascular cells [2]. In this research, a systemic analysis was performed for the gene expression of selected genes in the olfactory system.

Materials and Methods

Microarray Data Preparation and Description

A microarray data set, GSE104954 [11], was downloaded from GEO. It included 195 kidney biopsy tissues from ten different kidney diseases and healthy controls (HC). Eight kidney disease groups with at least 13 samples were selected for the analysis: systemic lupus erythematosus (SLE), IgA nephropathy (IGAN), rapidly progressive glomerulonephritis (RPGN), hypertensive nephropathy (HN), membranous glomerulonephritis (MGN), diabetic nephropathy (DN), minimal change disease (MCD), and focal segmental glomerulosclerosis (FSGS). The group sizes range from 13 to 25. The data was normalized by mapping the 25th and the 75th percentiles to 0 and 1 in both sample and gene dimensions. It included 37 OR genes, 10 ADCY genes, and 15 GNA genes listed in Table 1. The validation sets only contained IGAN, HN and HC by combining GSE37460 and GSE93798.

T-tests and Receiver Operating Characteristic Concerning Eight Dis- Eases with HC

For each gene in Table 1, pairwise t-tests were performed for a given disease and HC. The test results included HC mean $\mu 0$, disease mean $\mu 1$, and p value. A gene is up-regulated if $\mu 0 < \mu 1$ and down-regulated otherwise. It is statistically significant (denoted as s.s.) if $p \leq 0.05$. Furthermore, a receiver operating characteristic (ROC) curve was plotted and the area under the curve (AUC) was collected [12] for each disease and HC.

Statistic Summary

Each gene was summarized by first counting the number of ups or downs across the diseases and then only counting for cases with $p \leq 0.05$. The average AUC was also collected for up or down groups.

Linear Regression Models and Associated Rocs

Genes which were s.s. in either direction for at least five disease were selected to build linear regression models and ROC curves were plotted.

Data Analysis and Software

The statistics and the plots were implemented in R scripts developed in house. The student t-test used t.test in R package stats. The ROC used R package ROCR.

Results

T-test and ROC Summary

In Table 2, AUC averages were taken for up and down respectively. Most of OR genes have a "Down" count of 8 or 7 (Column 4) with OR11A1, OR2J3 and OR2S2 as exceptions. OR10H3, OR7A5, OR1D2, and OR2F1 were s.s. for most diseases (Column 2) and their AUCs were 0.79, 0.75, 0.77 and 0.74. There are 13 genes (from OR10H1 to OR7A10) which were down s.s. mainly for RPGN and SLE. Moreover, OR1F2P, OR2W1 and OR7C1 were down s.s. only for SLE and OR2B2, OR2B6 and OR6A2 were down s.s. only for RPGN. OR12D3, OR3A2, OR3A3 and OR7C2 were down s.s. for both SLE and RPGN. OR11A1 was the only up-regulated OR gene for most diseases but none was s.s.. In Table 3, most genes were universally down, but ADCY7 and ADCY3 were universally up and ADCY8 was in between. ADCY6, ADCYAP1, ADCY1 and ADCY7 were s.s. In Table 4, the top 6 genes were down (Column 4) and the bottom 9 genes were up (Column 8) universally. GNA11, GNAO1, GNAI3 and GNAI1 were s.s. with average AUCs greater than 0.74. GNA12, GNAQ, GNAS and GNAZ were up s.s. while GNA14 was down s.s. only for DN and RPGN. GNAT2 was down-regulated s.s. solely for SLE.

Linear Regression Models and ROC

Based on the above t-test results, OR10H3, OR7A5, OR1D2, and OR2F1 in OR, and ADCY6, ADCYAP1, ADCY1 and ADCY7 in ADCY, and GNA11, GNAO1, GNAI3 and GNAI1 in GNA, were s.s. universally and were selected for linear models. Supplemental Tables 1,2 & 3 in the supplemental list the model results. Based on the linear models, ROCs were plotted and the model performance is listed in Table 4. It showed that AUCs ranged from 0.75 to 1 with average 0.88, FPRs ranged from 0.0% to 24% with average 12%, and TPRs ranged from 60% to 100% with average 80%, indicating that each model separated the disease from HC very well.

Validation

Limited validation due to data availability was performed only for IGAN and HN. Most of the genes showed the same expression status except occasional exceptions which might be due to the data capture variations as shown in Supplemental Tables 2,3 & 4.

Summary

In summary, 4 genes in each group, namely OR10H3, OR7A5, OR1D2, OR2F1; ADCY6, AD- CYAP1, ADCY1, ADCY7; GNA11, GNAO1, GNAI3 and GNAI1 were universally down or up across all kidney diseases, hence they might be related to the common pathogenesis of most kidney diseases. Only 3 of them were up: ADCY7, GNAI3 and GNAI1. For disease specific genes, OR1F2P, OR2W1, and OR7C1 might be related to SLE, while OR2B2, OR2B6 and OR6A2 might be related to RPGN. For disease-pair specific genes, OR12D3, OR3A2, OR3A3, OR7C2 and GNAT2 might be specifically related to SLE and RPGN, while GNA12, GNAQ, GNAS and GNAZ might be specifically related to DN and RPGN.

Limitations

These results need to be further investigated and validated due to the group size. Since there are more than 400 human ORs, a lot of other ORs, ADCYs and GNAs were not considered here due to data availability.

Discussion

The genes in the olfactory system have been understudied especially for kidney diseases but there were some results in general. OR7A5 was shown to be up in glioma and inhibit lipid metabolism and proliferation for glioma cells [13], so down-regulation of OR7A5 in kidney tissue might disturb lipid metabolism. The activation of OR1D2 by specific agonists such as bourgeonal triggered Ca2+ increases in human airway smooth muscle cells via a cAMP-dependent signal transduction cascade and induced the secretion of IL-8 and CSF2 for airway inflammation [14]. Since undecanal is a potent antagonist to OR1D2 [15] and it is widely used in cosmetics and food industry, we now suspect that undecanal might be a risk factor by affecting OR1D2 expression. ADCY7 and ADCY6 were elevated under hypoxia, leading to the elevation of cAMP levels and enhanced PKA activity [16]. In chronic kidney diseases, hypoxia is associated with renal inflammation and fibrosis [17] and hence ADCY7 in the context of kidney pathogenesis should be investigated. GNA11 participates calcium signaling and mutation analysis demonstrated its involvement in hypercalcemia or hypocalcemia [18] and uveal melanoma [19], and GNA11/GNAQ mosaicism was demonstrated to induce hyperactivated calcium signaling [20], hence, GNA11 down-regulation in kidneys might reduce intracellular calcium influx to disturb multiple signaling pathways.

Declarations

Ethical Approval

Not applicable.

Informed Consent to Participate

Not applicable.

Competing Interests

A. Rao is a co-founder and full-time employee of Shenzhen Luwei (Biomanifold) Biotechnology Limited.

Data Availability Statement

The original data sets GSE104954, GSE37460 and GSE93798 are available via GEO data portal.

Funding Acknowledgement

Research reported in this paper was supported by President Foundation of the Third Affiliated Hospital of Southern Medical University under award number YQ202205.

Tables and Figures

(Tables 1-4) (**Supplemental Text**).

Table 1: Lists of OR, ADCY, and GNA genes contained in GSE104954. OR: olfactory receptor; ADCY: adenylate cyclase; GNA: G-protein α unit. Note that ADCYAP1 and ADCYAP1R1 are not ADCY enzymes but included in the ADCY group for convenience.

Category	Genes
ORs	OR10C1, OR10H1, OR10H2, OR10H3, OR10J1, OR11A1, OR12D2, OR12D3, OR1A1, OR1A2, OR1D2, OR1F1, OR1F2P, OR1G1, OR2B2, OR2B6, OR2C1, OR2F1, OR2F2, OR2H1, OR2H2, OR2J2, OR2J3, OR2S2, OR2W1, OR3A1, OR3A2, OR3A3, OR51E2, OR52A1, OR5I1, OR6A2, OR7A10, OR7A17, OR7A5, OR7C1, OR7C2
GNAs	GNA11, GNA12, GNA13, GNA14, GNA15, GNAI1, GNAI2, GNAI3, GNAL, GNAO1, GNAQ, GNAS, GNAT1, GNAT2, GNAZ
ADCYs	ADCY1, ADCY10, ADCY2, ADCY3, ADCY6, ADCY7, ADCY8, ADCY9, ADCYAP1, ADCYAP1R1

Table 2: T-test and AUC summary for ORs. N: count; s.s.: statistically significant; Up/Down: expression up/down-regulated for a disease type; AVG: average; AUC: area under the curve. Empty cells: not applicable.

Gene	Down s.s. (N)	Disease Down s.s.	Down All (N)	AVG AUC Down	Up s.s. (N)	Disease Up s.s.	Up All (N)	AVG AUC Up
OR10H3	8	DN, FSGS, HN, IGAN, MCD, MGN, RPGN, SLE	8	0.79	0		0	
OR7A5	8	DN, FSGS, HN, IGAN, MCD, MGN, RPGN, SLE	8	0.75	0		0	
OR1D2	7	DN, FSGS, HN, IGAN, MCD, RPGN, SLE	8	0.77	0		0	
OR2F1	5	DN, FSGS, MGN, RPGN, SLE	7	0.74	0		1	0.54
OR10H1	4	IGAN, MCD, RPGN, SLE	8	0.65	0		0	
OR10H2	4	DN, FSGS, RPGN, SLE	8	0.64	0		0	
OR10J1	4	DN, MGN, RPGN, SLE	8	0.67	0		0	
OR12D2	4	DN, IGAN, RPGN, SLE	7	0.69	0		1	0.53
OR1F1	4	FSGS, MCD, RPGN, SLE	8	0.67	0		0	
OR1G1	4	FSGS, IGAN, RPGN, SLE	8	0.68	0		0	
OR2C1	4	IGAN, MCD, RPGN, SLE	8	0.69	0		0	
OR3A1	4	FSGS, MCD, RPGN, SLE	8	0.66	0		0	
OR52A1	4	IGAN, MCD, RPGN, SLE	8	0.65	0		0	
OR1A1	3	MCD, RPGN, SLE	8	0.65	0		0	
OR1A2	3	DN, FSGS, MCD	8	0.69	0		0	
OR2F2	3	DN, RPGN, SLE	8	0.65	0		0	
OR7A10	3	DN, RPGN, SLE	8	0.66	0		0	
OR12D3	2	RPGN, SLE	8	0.62	0		0	
OR3A2	2	RPGN, SLE	8	0.66	0		0	
OR3A3	2	RPGN, SLE	8	0.66	0		0	
OR51E2	2	FSGS, RPGN	8	0.59	0		0	
OR5I1	2	FSGS, SLE	8	0.66	0		0	
OR7C2	2	RPGN, SLE	8	0.67	0		0	
OR1F2P	1	SLE	7	0.6	0		1	0.58
OR2B2	1	RPGN	7	0.62	0		1	0.52
OR2B6	1	RPGN	8	0.64	0		0	
OR2H1	1	MGN	8	0.65	0		0	
OR2W1	1	SLE	8	0.61	0		0	
OR6A2	1	RPGN	7	0.64	0		1	0.55
OR7C1	1	SLE	8	0.62	0		0	
OR10C1	0		8	0.59	0		0	
OR11A1	0		3	0.55	0		5	0.58
OR2H2	0		7	0.59	0		1	0.53
OR2J2	0		8	0.59	0		0	
OR2J3	0		5	0.56	0		3	0.53
OR2S2	0		6	0.59	0		2	0.54
OR7A17	0		8	0.57	0		0	

Table 3: T-test and AUC summary for ADCYs. N: count; s.s.: statistically significant; Up/Down: expression up/down-regulated for a disease type; AVG: average; AUC: area under the curve. Empty cells: not applicable.

Gene	Down s.s. (N)	Disease Down s.s.	Down All (N)	AVG AUC Down	Up s.s. (N)	Disease Up s.s.	Up All (N)	AVG AUC Up
ADCY6	7	DN, FSGS, HN, IGAN, MGN, RPGN, SLE	8	0.81	0		0	
ADCY-AP1	6	FSGS, HN, IGAN, MCD, RPGN, SLE	8	0.71	0		0	
ADCY1	5	DN, IGAN, MCD, RPGN, SLE	8	0.68	0		0	
ADCY-AP1R1	4	FSGS, MCD, RPGN, SLE	8	0.64	0		0	
ADCY9	1	DN	7	0.6	0		1	0.62
ADCY7	0		0		5	DN, HN, IGAN, RPGN, SLE	8	0.73
ADCY10	0		7	0.59	0		1	0.53
ADCY2	0		7	0.54	0		1	0.53
ADCY3	0		1	0.59	0		7	0.6
ADCY8	0		5	0.54	0		3	0.58

Table 4: T-test and AUC summary for GNAs. N: count; s.s.: statistically significant; Up/Down: expression up/down-regulated for a disease type; AVG: average; AUC: area under the curve. Empty cells: not applicable.

Gene	Down s.s. (N)	Disease Down s.s.	Down All (N)	AVG AUC Down	Up s.s. (N)	Disease Up s.s.	Up All (N)	AVG AUC Up
GNA11	8	DN, FSGS, HN, IGAN, MCD, MGN, RPGN, SLE	8	0.79	0		0	
GNAO1	7	DN, FSGS, HN, IGAN, MCD, RPGN, SLE	8	0.75	0		0	
GNAT1	3	MCD, RPGN, SLE	8	0.66	0		0	
GNA13	2	MCD, MGN	7	0.61	0		1	0.56
GNA14	2	DN, RPGN	7	0.66	0		1	0.56
GNAT2	2	RPGN, SLE	8	0.6	0		0	
GNAI2	1	MCD	2	0.63	3	DN, FSGS, RPGN	6	0.65
GNAI3	0		0		8	DN, FSGS, HN, IGAN, MCD, MGN, RPGN, SLE	8	0.87
GNAI1	0		0		7	DN, FSGS, HN, IGAN, MGN, RPGN, SLE	8	0.76
GNA15	0		0		4	FSGS, MGN, RPGN, SLE	8	0.72
GNA12	0		0		2	DN, RPGN	8	0.62
GNAL	0		0		2	MCD, MGN	8	0.65
GNAQ	0		1	0.55	2	DN, RPGN	7	0.64
GNAS	0		1	0.55	2	DN, RPGN	7	0.66
GNAZ	0		1	0.52	2	DN, RPGN	7	0.63

References

1. Zaidman NA, Pluznick JL (2022) Understudied G Protein-Coupled Receptors in the Kidney. Nephron 146(3): 278-281.
2. Beito MR, Ashraf S, Odogwu D, Romain Harmancey (2024) Role of ectopic olfactory receptors in the regulation of the cardiovascular– kidney–metabolic axis. Life 14: 548.
3. Shepard BD (2021) The sniffing kidney: roles for renal olfactory receptors in health and disease. KIDNEY360 2: 1056-1062.
4. Pluznick JL, Zou DJ, Zhang X, Qingshang Yan, Diego J Rodriguez-Gil, et al. (2009) Functional expression of the olfactory signaling system in the kidney. Proc Natl Acad Sci USA 106: 2059-2064.
5. Pluznick JL, Protzko RJ, Gevorgyan H, Zita Peterlin, Arnold Sipos, et al. (2013) Olfactory receptor responding to gut microbiota- derived signals plays a role in renin secretion and blood pressure regulation. Proc Natl Acad Sci USA 110(11): 4410-4415.
6. Kotlo K, Anbazhagan AN, Priyamvada S, Dulari Jayawardena, Anoop Kumar, et al. (2020) The olfactory G protein-coupled receptor (Olfr78/OR51E2) modulates the intestinal response to colitis. Am J Physiol Cell Physiol 318(3): C502-C513.
7. Xu J, Choi R, Gupta K, Helen R Warren, Lakshmi Santhanam, et al. (2024) An evolutionarily conserved olfactory receptor is required for sex differences in blood pressure. Sci Adv 10(12): eadk1487.
8. Shepard BD, Cheval L, Peterlin Z, Stuart Firestein, Hermann Koepsell, et al. (2016) A renal olfactory receptor aids in kidney glucose handling Sci Rep 6: 35215.
9. Shepard BD, Koepsell H, Pluznick JL (2019) Renal olfactory receptor 1393 contributes to the pro- gression of type 2 diabetes in a diet-induced obesity model. Am J Physiol Ren Physiol 316: F372-F381.
10. Motahharynia A, Moein S, Kiyanpour F, Kobra Moradzadeh, Moein Yaqubi, et al. (2022) Olfactory receptors contribute to progression of kidney fibrosis. NPJ Syst Biol Appl 8: 8.
11. Grayson PC, Eddy S, Taroni JN, et al. (2018) Vasculitis Clinical Research Consortium, the European Renal cDNA Bank cohort, and the Nephrotic Syndrome Study Network. Metabolic pathways and immunometabolism in rare kidney diseases. Ann Rheum Dis 77(8): 1226-1233.
12. Hajian-Tilaki K (2013) Receiver Operating Characteristic (ROC) Curve Analysis for Medical Di- agnostic Test Evaluation. Caspian J Intern Med 4(2): 627-635.
13. Bao Y, Tang Z, Chen R, Xuebin Yu, Xuchen Qi, et al. (2024) Pan-cancer analysis identifies olfactory receptor fam- ily 7 subfamily A member 5 as a potential biomarker for glioma. Peer J 12: e17631.
14. Kalbe B, Knobloch J, Schulz VM, Christine Wecker, Marian Schlimm, et al. (2016) Olfactory Receptors Modulate Physiological Processes in Human Airway Smooth Muscle Cells. Front Physiol 7: 339.
15. Harini K, Sowdhamini R (2015) Computational Approaches for Decoding Select Odorant- Olfactory Receptor Interactions Using Mini- Virtual Screening. PLoS ONE 10(7): e0131077.
16. Simko V, Iuliano F, Sevcikova A, Martina Labudova, Monika Barathova, et al. (2017) Hypoxia induces cancer-associated cAMP/PKA sig- naling through HIF-mediated transcriptional control of adenylyl cyclases VI and VII. Sci Rep 7: 10121.
17. Wang B, Li ZL, Zhang YL, Yi Wen, Yue-Ming Gao, et al. (2022) Hypoxia and chronic kidney disease. EBioMedicine 77: 103942.
18. Howles SA, Gorvin CM, Cranston T, Angela Rogers, Anna K Gluck, et al. (2023) GNA11 Variants Identified in Patients with Hypercalcemia or Hypocalcemia. J Bone Miner Res 38: 907-917.
19. Silva-Rodrıguez P, Fernandez-Dıaz D, Bande M, Lourdes Loidi, María José Blanco-Teijeiro, et al. (2022) GNAQ and GNA11 Genes: A Comprehensive Review on Oncogenesis, Prognosis and Therapeutic Opportunities in Uveal Melanoma. Cancers 14: 3066.
20. Zecchin D, Kn¨opfel N, Gluck AK, et al. (2024) GNAQ/GNA11 Mosaicism Causes Aberrant Calcium Signaling Susceptible to Targeted Therapeutics. J Invest Dermatol 144(4): 811-819.e4
21. Li W, Sang M, Hao X, Li Jia, Yong Wang, et al. (2020) Gene expression and DNA methylation analyses suggest that immune process-related ADCY6 is a prognostic factor of luminal-like breast cancer. J Cell Biochem 121(7): 3537-3546.
22. Ilanges A, Shiao R, Shaked J, Ji-Dung Luo, Xiaofei Yu, et al. (2022) Brainstem ADCYAP1+ neurons control multiple aspects of sickness behaviour. Nature 609: 761-771.
23. Chen Q, Zhang XY, Wang YP, Yun-Jie Fu, Feng Cao, et al. (2023) Unveiling adcyap1 as a protective factor linking pain and nerve regeneration through single-cell RNA sequencing of rat dorsal root ganglion neurons. BMC Biol 21(1): 235.
24. Kakeshita K, Koike T, Imamura T, Hayato Fujioka, Hidenori Yamazaki, et al. (2022) Prognostic impact of urine cyclic AMP levels in patients with chronic kidney disease. Clin Exp Nephrol 26: 1194-1199.
25. Zhang Y, Yang J, Wang X, Xinchang Li (2021) GNG7 and ADCY1 as diagnostic and prognostic biomarkers for pancreatic adenocarcinoma through bioinformatic-based analyses. Sci Rep, pp. 20441.
26. Zou T, Liu J, She L, Juan Chen, Tao Zhu, et al. (2019) A perspective profile of ADCY1 in cAMP signaling with drug- resistance in lung cancer. J Cancer 10(27): 6848-6857.
27. Danti FR, Galosi S, Romani M, Martino Montomoli, Keren J Carss, et al. (2017) GNAO1 encephalopathy: Broadening the phenotype and evaluating treatment and outcome. Neurol Genet 3(2): e143.
28. Briere L, Thiel M, Sweetser DA, Adam MP, Feldman J, et al. GNAO1-Related Disorder. In: (2023) (Edt.,), GeneReviews® [Inter- net]. Nov 9. Seattle (WA): University of Washington, Seattle; 1993-2024.
29. Taira R, Akamine S, Okuzono S, Fumihiko Fujii, Eriko Hatai, et al. (2024) Gnao1 is a molecular switch that regulates the Rho signaling pathway in differentiating neurons. Sci Rep 14: 17097.
30. Sethna F, Feng W, Ding Q, Alfred J Robison, Yue Feng, et al. (2017) Enhanced expression of ADCY1 underlies aberrant neuronal signalling and behaviour in a syndromic autism model. Nat Commun 8: 14359.
31. Li X, Wang D, Chen Z, Ermei Lu, Zhuo Wang, et al. (2015) Gαi1 and Gαi3 regulate macrophage polarization by forming a complex containing CD14 and Gab1. Proc Natl Acad Sci USA 112(15): 4731-4736.
32. Li ZW, Sun B, Gong T, Sheng Guo, Jianhua Zhang, et al. (2019) GNAI1 and GNAI3 Reduce Colitis-Associated Tumorigenesis in Mice by Blocking IL6 Signaling and Down-regulating Expression of GNAI2. Gastroenterology 156(8): 2297-2312.
33. Cao C, Huang X, Han Y, Yinsheng Wan, Lutz Birnbaumer, et al. (2009) Galpha(i1) and Galpha(i3) are required for epidermal growth factor-mediated activation of the Akt-mTORC1 pathway. Sci Signal 2(68): ra17.
34. Kim H, Park SH, Oh SW, Kitae Kwon, Se Jung Park, et al. (2021) Olfactory Receptor OR7A17 Expression Correlates with All-Trans Retinoic Acid (ATRA)-Induced Suppression of Proliferation in Human Keratinocyte Cells. Int J Mol Sci 22(22):12304

35. Haag F, Di Pizio A, Krautwurst D (2022) The key food odorant receptive range of broadly tuned receptor OR2W1. Food Chem 375: 131680.

36. Morita R, Hirohashi Y, Torigoe T, Satoko Ito-Inoda, Akari Takahashi, et al. (2016) Olfactory Receptor Family 7 Subfamily C Member 1 Is a Novel Marker of Colon Cancer-Initiating Cells and Is a Potent Target of Immunotherapy. Clin Cancer Res 22(13): 3298-3309.

37. Miyamoto S, Hirohashi Y, Morita R, Akihiro Miyazaki, Kazuhiro Ogi, et al. (2023) Exploring olfactory receptor family 7 subfamily C member 1 as a novel oral cancer stem cell target for immunotherapy. Cancer Sci 114(9): 3496-3508.

38. Morrell CN, Mix D, Aggarwal A, Rohan Bhandari, Matthew Godwin, et al. (2022) Platelet olfactory receptor activation limits platelet reactivity and growth of aortic aneurysms. J Clin Invest 132(9): e152373.

39. Weber L, Maßberg D, Becker C, Altmuller J, Ubrig B, et al. (2018) Olfactory Receptors as Biomarkers in Human Breast Carcinoma Tissues. Front Oncol 8: 33.

40. Masjedi S, Zwiebel LJ, Giorgio TD (2019) Olfactory receptor gene abundance in invasive breast carcinoma. Sci Rep 9: 13736.

41. He Z, Wang DW (2022) Olfactory receptor 2 activation in macrophages: novel mediator of atherosclerosis progression. Sig Transduct Target Ther 7: 247.

42. Cheng J, Yang Z, Ge XY, Ming-Xin Gao, Ran Meng, et al. (2022) Autonomous sensing of the insulin peptide by an olfactory G protein-coupled receptor modulates glucose metabolism. Cell Metabolism 34(2): 240-255.

43. Georgiou M, Singh N, Kane T, Anthony G Robson, Angelos Kalitzeos, et al. (2020) Photoreceptor Structure in GNAT2-Associated Achro- matopsia. Invest Ophthalmol Vis Sci 61(3) :40.

44. Tutunea-Fatan E, Lee JC, Denker BM, Lakshman Gunaratnam (2020) Heterotrimeric Gα12/13 proteins in kidney injury and disease. Am J Physiol Renal Physiol 318(3): F660-F672.

45. Turan S, Bastepe M (2013) The GNAS complex locus and human diseases associated with loss-of-function mutations or epimutations within this imprinted gene. Horm Res Paediatr 80(4): 229-241.

46. Vancura P, Abdelhadi S, Csicsely E, Gianluca Tosini, P Michael Iuvone, et al. (2017) Gnaz couples the circadian and dopaminergic system to G protein-mediated signaling in mouse photoreceptors. PLoS ONE 12(10): e0187411.

47. Son B, Kang W, Park S, Dabin Choi, Taesun Park, et al. (2021) Dermal olfactory receptor OR51B5 is essential for survival and col- lagen synthesis in human dermal fibroblast (hs68 cells). Int J Mol Sci 22(17): 9273.

48. Kang N, Bahk YY, Lee N, YoonGyu Jae, Yoon Hee Cho, et al. (2015) Olfactory receptor Olfr544 responding to azelaic acid regulates glucagon secretion in α-cells of mouse pancreatic islets. Biochem Biophys Res Commun 460(3): 616-621.

49. Arakawa H, Akkentli F, Erzurumlu RS (2014) Region-Specific Disruption of Adenylate Cyclase Type 1 Gene Differentially Affects Somatosensorimotor Behaviors in Mice. eNeuro 1(1).

ISSN: 2574-1241

DOI: 10.26717/BJSTR.2026.65.010193

Aibing Rao. Biomed J Sci & Tech Res

Submission Link: https://biomedres.us/submit-manuscript.php