

## Appendix 1

	<i>JAK/STAT</i>	<i>Related factor</i>	<i>Drug</i>	<i>Signaling factor</i>	<i>Complications</i>	<i>Regeneration</i>	<i>Other signaling</i>	<i>Test</i>
<i>Elizaveta Fasler-Kan (2024)</i>	STAT1 STAT3 STAT 6		-	TNF/IGf-STAT1 IFN- $\alpha$ /IL-6 - stat3 IL-4- Stat 6		IFN- $\alpha$ and IFN- $\gamma$ suppressed proliferation/IL-4 and IL-6 stimulated proliferation.	nuclear factor kappa B (NF- $\kappa$ B)	FC- IF-WB
<i>Tao Zeng (2023)</i>	STAT 2	T cells (CD3D, CD3E, and CD2), B cells (CD19 and CD79A), monocytes (CD86 and CD11) M2 macrophages (CD163, VSIG4, and MS4A4A), and neutrophils (CD66b, CD11b, and CCR7), Immune biomarkers of Th1(T-bet, STAT4, STAT1, IFN-g, and TNF-a), Th2 (GATA3, STAT6, STAT5A, and IL13), Tfh (BCL6 and IL21), and Treg (FOXP3, CCR8,	-	STAT2 expression of KIRC in decreased B cells, Mesenchymal stem cells, Natural killer T-cells cohort		STAT2 known as a potential diagnostic biomarker for KIRC. High expression of STAT2 related to poor OS, PFS and DFS. STAT2 was highly significant in immune escape in the KIRC microenvironment, and immune inhibitor for KIRC therapy.		

	STAT5B, and TGFb)							
<i>Renate Pichler (2023)</i>	JAK2/STAT1	IFN $\gamma$	-	IFNG-JAK2/STAT1	-	IFN signaling is strongly counteracted by multiple redundant immune checkpoint and T cell exhaustion processes	-	-
<i>Kun Meng (2023)</i>	STAT2 STAT3	IFN- $\gamma$	Ruxolitinib	GBP2-STAT2/STAT3	-	GBP2, HLA-DRA, ISG15, ISG20 and ITGAX are be closely correlated with both pathological grade and clinical stage of renal carcinoma, GBP2 promotes cell metastasis in ccRCC via the activation of JAK/STAT pathway		WB
<i>J Guo (2023)</i>	STAT 3 AKT1	CCND1, CASP3, JUN, VEGFA, , EGFR		wogonin, baicalein, acacetin, oroxylin A, moslosooflavone, salvigenin, neobaicalein		up-regulating expressio and activity of p53 by wogonin or baicalein, crippling tumor cell proliferation and benefiting apoptosis. Blocking the expression and activation of STAT3 and AKT1		

						is a therapeutic choice.		
Hao Deng (2023)	STAT 3 JAK 2	FCSN1 TIMP1	AG490(JAK inhibitor)	KIF2C-STAT3/JAK2	-	KIF2C positively regulates JAK2/STAT3 signaling in ccRCC cells JAK2/STAT3 signaling inhibitor impair the tumor-promoting effects of KIF2C in ccRCC	-	WB, TIME R web
Changjiu Li (2021)	STAT3, STAT6	SOCSs family Stat3-SOCS1, SOCS3, CISH STAT 6 OCS1, CISH IRF1 - SOCS1 , SOCS2	-	-	-	SOCS3 one of the most key negative regulating factors of the JAK/STAT signaling pathway that downregulated cancer progression	IRF1	-
Wei Li (2014)	JAK 1 STAT1	-	-	IFN-pSTAT1	-	expression rates of JAK1, STAT1 in the renal cell carcinomas were significantly lower, No significant differences were in expression levels of STAT2	-	-
Jau-Shyang Huang (2015)	JAK 2 STAT 1 STAT 3	-	Cinnamaldehyde AG490	AGE -STAT 1, STAT3, JAK2 and not STAT5		cinnamaldehyde, suppress AGE-induced biological responses that mediated by inactivating the JAK2-		

						STAT1/STAT3 cascade		
<i>Feiguo Liang (2020)</i>	STAT4 JAK3 STAT4, STAT1 STAT6, STAT5	CD8+ T cell (CD8A and CD8B), T cell (CD3D, CD3E and CD2), B cell (CD19 and CD79A), Monocyte (CD86 and CD11), M2 Macrophage (CD163, VSIG4, MS4A4A),  T-bet (TBX21), , IFN-g (IFNG), TNF-a (TNF) of Th1, and GATA3, IL13 of Th2		JAK3- Tregs (FOXP3, CCR8, STAT5B, and TGFb) JAK3- T exhaustion cell (PD-1, CTLA4, LAG3, TIM-3 and GZMB)		JAK3 is known as immunotherapeutic target for renal carcinoma therapy,	P13K-Akt and Ras signaling pathway	
<i>Wenbin Song (2020)</i>	STAT 3 JAK	-	-	SOCS4-STAT3	-	SOCS4 protein have nothing to do with protein levels of non-phosphorylated STAT and JAK, this signaling promotes cell proliferation and invasion, and inhibits apoptosis	-	-
<i>Hui Liu (2021)</i>	STAT3 JAK	VEGFA CCND1	-	-	-	apoptosis induced by gypenosides of RCC cells through regulating PI3K/Akt/mTOR signaling	MAPK3, PIK3CA	-

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(2023)

STAT 3

IL-15  
IL-12p70

STAT3 levels in  
tumors was  
significantly  
lower than in  
the kidney and  
related to  
pathological  
stage of the  
disease  
And not nuclear  
grade

multi  
plex  
assay  
s and  
ELISA