Understanding The Role Of *Achyranthes aspera* Extract On Pancreatic Cancer Through Differential Gene Expression Analysis

A PROJECT REPORT

SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE AWARD OF THE DEGREE

OF

MASTER OF TECHNOLOGY
IN
BIOINFORMATICS

Submitted by:

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(2K18/BIO/01)

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JUNE, 2020

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CANDIDATE'S DECLARATION

I, Amit Negi, 2K18/BIO/01 of M.Tech (Bioinformatics), hereby declare that the project Dissertation titled "**Understanding The Role *Achyranthes aspera* Extract On Pancreatic Cancer Through Differential Gene Expression Analysis** " which is submitted by me to the Department of Biotechnology, Delhi Technological University, Delhi in partial fulfilment of the requirements for the award of the degree of Master of Technology, is original and not copied from any source with proper citation. This work has not previously formed the basis for the award of the Degree, Diploma Associateship, Fellowship or other similar title or recognition.

Place: Delhi **AMIT NEGI**

Date: (2K18/BIO/01)

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CERTIFICATE

I hereby certify that the Project Dissertation titled **"Understanding The Role *Achyranthes aspera* Extract On Pancreatic Cancer Through Differential Gene Expression Analysis"** which is submitted by **Amit Negi(2K18/BIO/01)**, Department of Biotechnology, Delhi Technological University, Delhi in partial fulfillment of the requirement for the award of the degree of Master of Technology, is a record of the project work carried out by the student under my supervision. To the best of my knowledge this work has not been submitted in part or full for any Degree or Diploma to this University or elsewhere.

Place: Delhi **Prof. Jaigopal Sharma**

Date: Professor

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I, Amit Negi wish to express my profound gratitude and indebtedness to **Prof. Jaigopal Sharma,** Department of Bio-Technology, Delhi Technological University, Delhi for introducing the present topic and for her inspiring guidance, constructive criticism and valuable suggestion throughout the project work. And my sincere thanks to all our friends who have patiently extended all sorts of help for accomplishing this undertaking.

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Understanding The Role *Achyranthes aspera* Extract On Pancreatic Cancer Through Differential Gene Expression Analysis

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ABSTRACT

Herbal drugs are broadly utilized since ancient time demonstrating that herbs are a developing segment of contemporary, advancely generated medication. India has an old legacy of conventional herbal medication. The medicinal plants are utilized for treatment of different ailments on account of their security and viability. The issue of microbial aversion is developing and the standpoint for the utilization of antimicrobial medications later on is as yet undetermine. So, measures must be made to control the utilization of anti-infection, to create examination to all the more likely comprehend the hereditary systems of obstruction, and to proceed with studies to grow either man-made or innate noval medications. Various researches have been done on herbals affirming their possible antimicrobial property against microbes. One of the methodologies towards accomplishing this goal is the sound restriction of bioactive phytoconstituents. *Achyranthes aspera* (Amaranthaceae) is a significant therapeutic herb found as a weed all through India. Despite the fact that practically the entire of its parts are utilized in conventional processes of medications, seeds, roots and shoots are the most significant parts which are utilized therapeutically. Using the bioinformatics tools, bioinformatics-analysis has been done when plant extract is used on human cancer cells to find the most significant genes and the level of their expression.

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1. INTRODUCTION

It has been realized that information with respect to herbs passed on starting with one age then onto the next for a large number of years. As we realize that herbal medications have a powerful conventional base and possibly valuable as a medications regarding well being and viability which leads for medicating various ailments .Plants have an unprecedented capacity to blend aromatic substances which are normally phenols. The therapeutically dynamic plant mixes are generally their secondary metabolites which may be flavonoids, terpenoids, tannins, and so forth that are accountable for defending the plants from microbes, pest and small insects. In the ongoing past there has been a huge increment in the utilization of plant based well being items in creating just as created nations bringing about a fast development of herbal items across the world.

Achyranthes aspera is one of the plant which is used as a herbal plant. Amaranthaceae is the family which A. aspera linn. belongs , is a yearly, hardened erect, enduring herb, 1-2m tall, having a woody base, usually found as a weed on side of the road. *Achyranthes aspera* is a notable plant medicate in Ayurved Allopathic, Siddha, homeopathic. It is also used in the home remedies. It is the yearly bush discovered dispersed all through the tropical and subtropical districts. It is normally found in various part of the world like India, Sri Lanka, Baluchistan, Australia, Tropical Asia and America. This tropical plant is referred to by various names in different languages across India, for example, in hindi it is known as Chirchita, likewise in Aghedi (Gujarati), Apamarga (Sanskrit), Apang (Bengali). The plant is utilized in indigenous arrangement of medication as antiarthritic, antifertility, diuretic, anti-helminthic, antiviral, antihypertensive, . It is additionally valuable to treat renal dropsy, skin surge, nasal, inflammation, asthma, malaria and snake nibbles.

This plant juice is used in treating of dysentery, boils, diarrhea. It also help to treat haemorrhoids, itches and skin related problems too. The secondary metabolites which are present in the plant are saponins, alkaloids, steroids and terpenoids. The secondary metabolite flavonoids have appeared to slow down the progession of tumor cells and mainly act as an anti-inflammatory factors

According to the conventational healers if the *Achyranthes aspera* is added then it enhances the viability of most of the medication of plant origin. So the current study ia an attempt to analyse the more prominent investigation of the pharmacological utlilization of the *Achyranthes aspera* through the bioinformatics tools.

2. MATERIALS AND METHODS

2.1 Microarray data processing and screening for DEGs

In the study the expression profile GSE44290 from the GEO database was used to identify DEGs. The series GSE44290 provides the microarray data which contains the differentially expressed genes. To determine the DEGs GEO2R tool is used. Then excel file was produced which having the DEGs.

2.2 Pathway and GO enrichment analysis of DEGs

To understand the functional changes of the DEGs, their biological processes (BP), molecular function (MF) and cellular components (CC) were analysed by iDEP.91.

Enrichment of pathways was also performed by using iDEP.91.

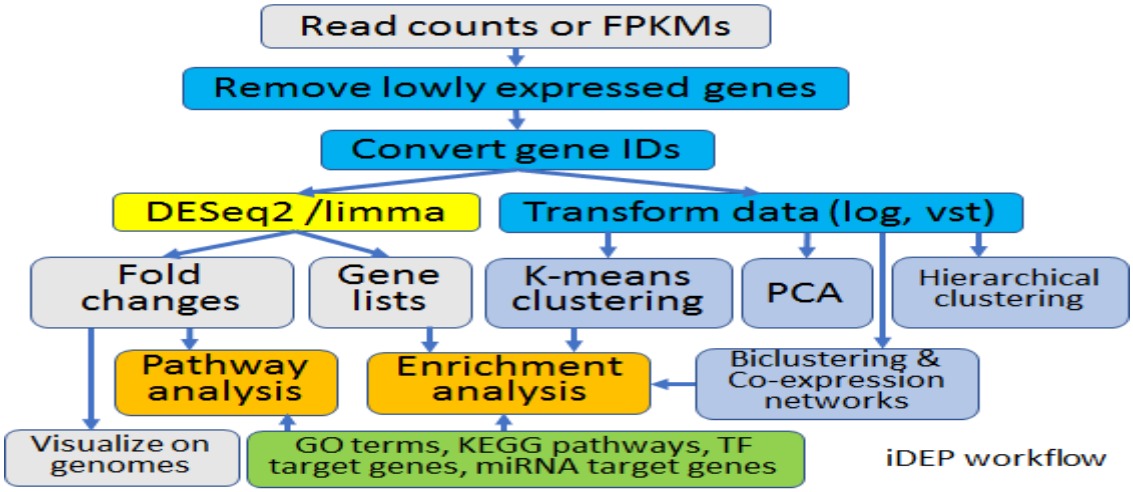


Fig1. Workflow of the iDEP.91 tool

2.3 PPI network construction

A PPI network was constructed using STRING database on iDEP.91 for the proteins. In which how proteins are interacted with each other is shown.

2.4 k-Means clustering and heat map analysis

k-Means clustering method includes clustering genes into groups based on their expression pattern across all samples and heat map was used to find out the level of expression of the most significant genes.

3. RESULTS

3.1 Identification of DEGs

The series GSE44290 was to identify the DEGs between the treated and untreated cell with the *Achyranthes aspera* using the tool GEO2R. Fold change of >|1.5| and p-value < 0.05 were used to identify the DEGs. The total genes of 1671 were identified in which 720 genes were downregulated and 951 genes are upregulated.

3.2 Functional enrichment analysis of DEGs

To understand the function of the DEGs, their biological processes (BP), molecular function (MF) and cellular components (CC) were analysed by iDEP.91.

For the biological process:

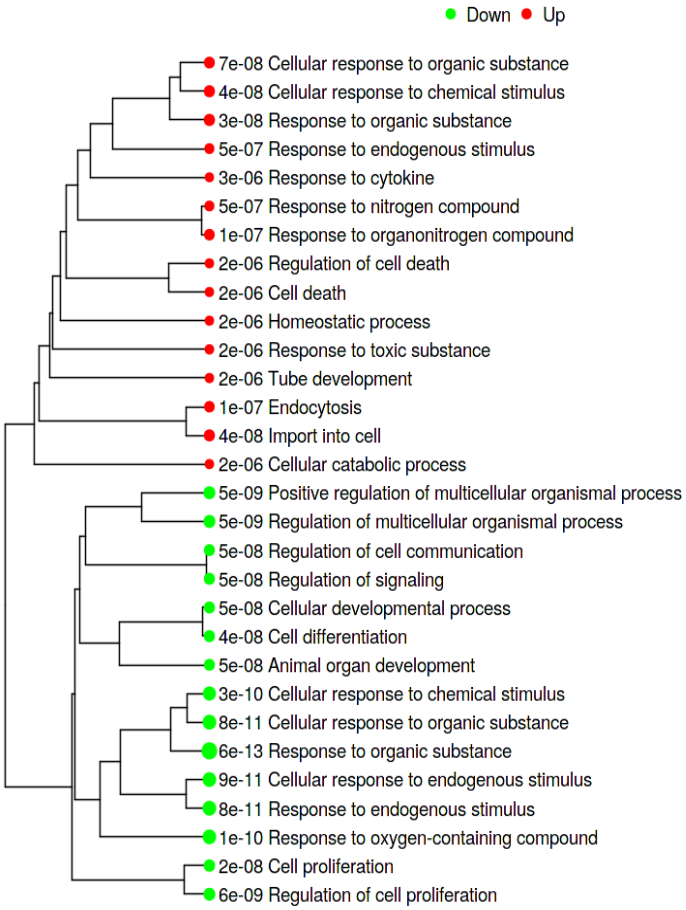


Fig 2. Enrichment tree of DEGs in biological process

	Direction	adj.Pval	nGenes	Pathways
1	Down regulated	5.51E-13	170	Response to organic substance
2	Down regulated	8.08E-11	98	Response to endogenous stimulus
3	Down regulated	8.08E-11	142	Cellular response to organic substance
4	Down regulated	9.35E-11	87	Cellular response to endogenous stimulus
5	Down regulated	9.82E-11	98	Response to oxygen-containing compound
6	Down regulated	3.45E-10	159	Cellular response to chemical stimulus
7	Down regulated	4.56E-09	150	Regulation of multicellular organismal process
8	Down regulated	4.56E-09	100	Positive regulation of multicellular organismal process
9	Down regulated	5.56E-09	94	Regulation of cell proliferation
10	Down regulated	1.86E-08	107	Cell proliferation
11	Down regulated	4.05E-08	180	Cell differentiation
12	Down regulated	4.50E-08	164	Regulation of signaling
13	Down regulated	4.50E-08	186	Cellular developmental process
14	Down regulated	5.37E-08	162	Regulation of cell communication
15	Down regulated	5.42E-08	158	Animal organ development

16	Up regulated	2.88E-08	187	Response to organic substance
17	Up regulated	3.81E-08	185	Cellular response to chemical stimulus
18	Up regulated	4.08E-08	69	Import into cell
19	Up regulated	6.69E-08	159	Cellular response to organic substance
20	Up regulated	1.10E-07	74	Response to organonitrogen compound
21	Up regulated	1.42E-07	61	Endocytosis
22	Up regulated	5.17E-07	77	Response to nitrogen compound
23	Up regulated	5.35E-07	103	Response to endogenous stimulus
24	Up regulated	1.61E-06	47	Response to toxic substance
25	Up regulated	1.92E-06	72	Tube development
26	Up regulated	2.15E-06	134	Cellular catabolic process
27	Up regulated	2.33E-06	130	Cell death
28	Up regulated	2.33E-06	106	Regulation of cell death
29	Up regulated	2.33E-06	113	Homeostatic process
30	Up regulated	3.08E-06	85	Response to cytokine

Table 1. GO enrichment analysis of DEGs in biological process

For the molecular function:

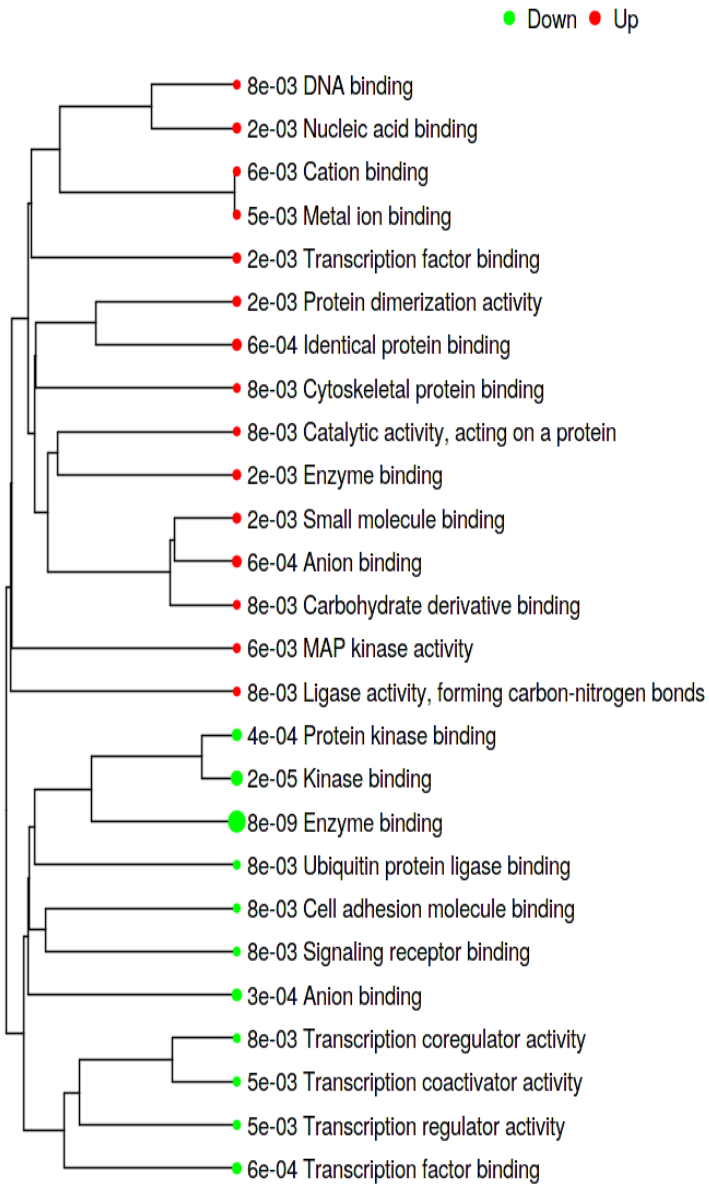


Fig 3. Enrichment tree of DEGs in molecular function

	Direction	adj.Pval	nGenes	Pathways
1	Down regulated	8.28E-09	115	Enzyme binding
2	Down regulated	2.42E-05	46	Kinase binding
3	Down regulated	0.00028489	120	Anion binding
4	Down regulated	0.00041848	39	Protein kinase binding
5	Down regulated	0.00058318	39	Transcription factor binding

6	Down regulated	0.00472788	23	Transcription coactivator activity
7	Down regulated	0.00485576	86	Transcription regulator activity
8	Down regulated	0.00830411	73	Signaling receptor binding
9	Down regulated	0.00830411	29	Cell adhesion molecule binding
10	Down regulated	0.00847612	32	Transcription coregulator activity
11	Down regulated	0.00847612	21	Ubiquitin protein ligase binding
12	Up regulated	0.00058123	99	Identical protein binding
13	Up regulated	0.00058123	145	Anion binding
14	Up regulated	0.00190059	194	Nucleic acid binding
15	Up regulated	0.00190059	45	Transcription factor binding
16	Up regulated	0.00190059	130	Small molecule binding
17	Up regulated	0.00190059	76	Protein dimerization activity
18	Up regulated	0.00193541	113	Enzyme binding
19	Up regulated	0.00465616	192	Metal ion binding
20	Up regulated	0.00598895	5	MAP kinase activity
21	Up regulated	0.00598895	194	Cation binding
22	Up regulated	0.00815049	122	DNA binding
23	Up regulated	0.00815049	55	Cytoskeletal protein binding
24	Up regulated	0.00815049	8	Ligase activity, forming carbon-nitrogen bonds
25	Up regulated	0.00815049	112	Carbohydrate derivative binding
26	Up regulated	0.00815049	111	Catalytic activity, acting on a protein

Table 2. GO Enrichment analysis of the DEGs for the molecular function

For the cellular component:

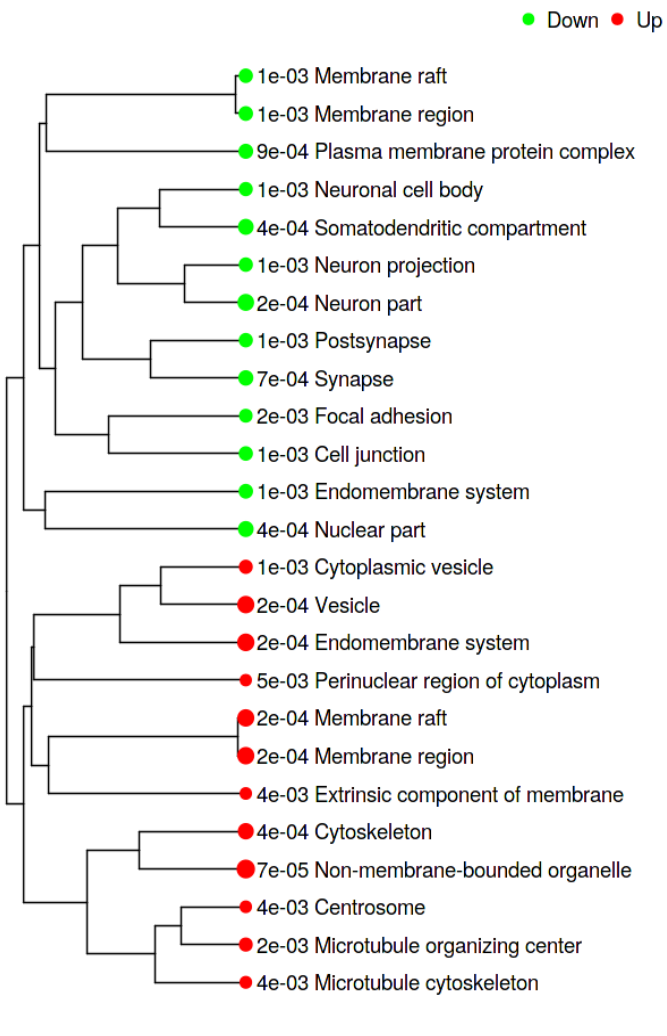


Fig 4. Enrichment tree of DEGs in cellular component

	Direction	adj.Pval	nGenes	Pathways
1	Down regulated	0.000236	82	Neuron part
2	Down regulated	0.00045	47	Somatodendritic compartment
3	Down regulated	0.00045	176	Nuclear part
4	Down regulated	0.000655	60	Synapse
5	Down regulated	0.000864	40	Plasma membrane protein complex

6	Down regulated	0.001037	173	Endomembrane system
7	Down regulated	0.001037	24	Membrane region
8	Down regulated	0.001223	63	Cell junction
9	Down regulated	0.001223	23	Membrane raft
10	Down regulated	0.001275	61	Neuron projection
11	Down regulated	0.001314	36	Postsynapse
12	Down regulated	0.001496	30	Neuronal cell body
13	Down regulated	0.001985	27	Focal adhesion
14	Up regulated	6.91E-05	209	Non-membrane-bounded organelle
15	Up regulated	0.00015	219	Endomembrane system
16	Up regulated	0.000169	191	Vesicle
17	Up regulated	0.000169	30	Membrane region
18	Up regulated	0.000175	29	Membrane raft
19	Up regulated	0.000351	116	Cytoskeleton
20	Up regulated	0.001499	123	Cytoplasmic vesicle
21	Up regulated	0.001661	48	Microtubule organizing center
22	Up regulated	0.003836	67	Microtubule cytoskeleton
23	Up regulated	0.004408	35	Centrosome
24	Up regulated	0.004408	24	Extrinsic component of membrane
25	Up regulated	0.005049	44	Perinuclear region of cytoplasm

Table 3. GO enrichment analysis of DEGS in cellular component

For the KEGG:

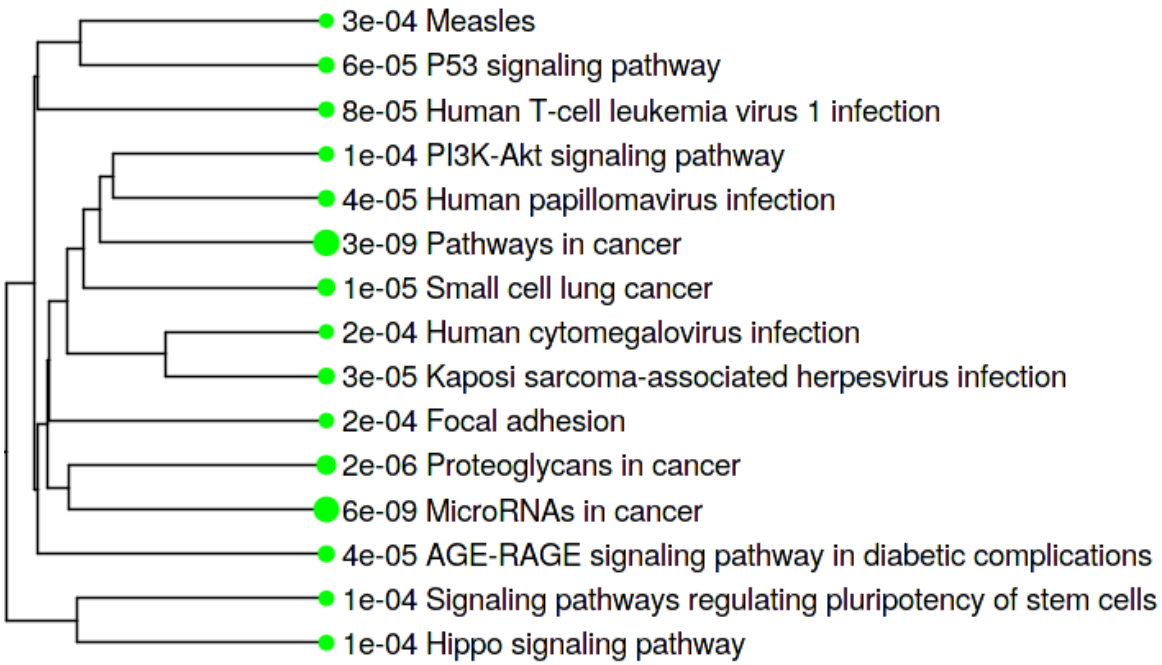


Fig 4. GO enrichment analysis of DEGs in KEGG

	Direction	adj.Pval	nGenes	Pathways
1	Down regulated	3.18E-09	76	Pathways in cancer
2	Down regulated	6.43E-09	34	MicroRNAs in cancer
3	Down regulated	2.46E-06	35	Proteoglycans in cancer
4	Down regulated	1.41E-05	21	Small cell lung cancer
5	Down regulated	3.48E-05	31	Kaposi sarcoma-associated herpesvirus infection
6	Down regulated	3.53E-05	21	AGE-RAGE signaling pathway in diabetic complications
7	Down regulated	3.53E-05	45	Human papillomavirus infection

8	Down regulated	6.09E-05	17	P53 signaling pathway
9	Down regulated	7.86E-05	33	Human T-cell leukemia virus 1 infection
10	Down regulated	0.000107	26	Hippo signaling pathway
11	Down regulated	0.000136	45	PI3K-Akt signaling pathway
12	Down regulated	0.00014	24	Signaling pathways regulating pluripotency of stem cells
13	Down regulated	0.000166	30	Focal adhesion
14	Down regulated	0.000244	32	Human cytomegalovirus infection
15	Down regulated	0.0003	23	Measles

Table 4. GO enrichment analysis of DEGs in KEGG

3.3 Construction of PPI network

For the PPI network construction, STRING database was used in top 100 proteins were taken.

PPI network for proteins:

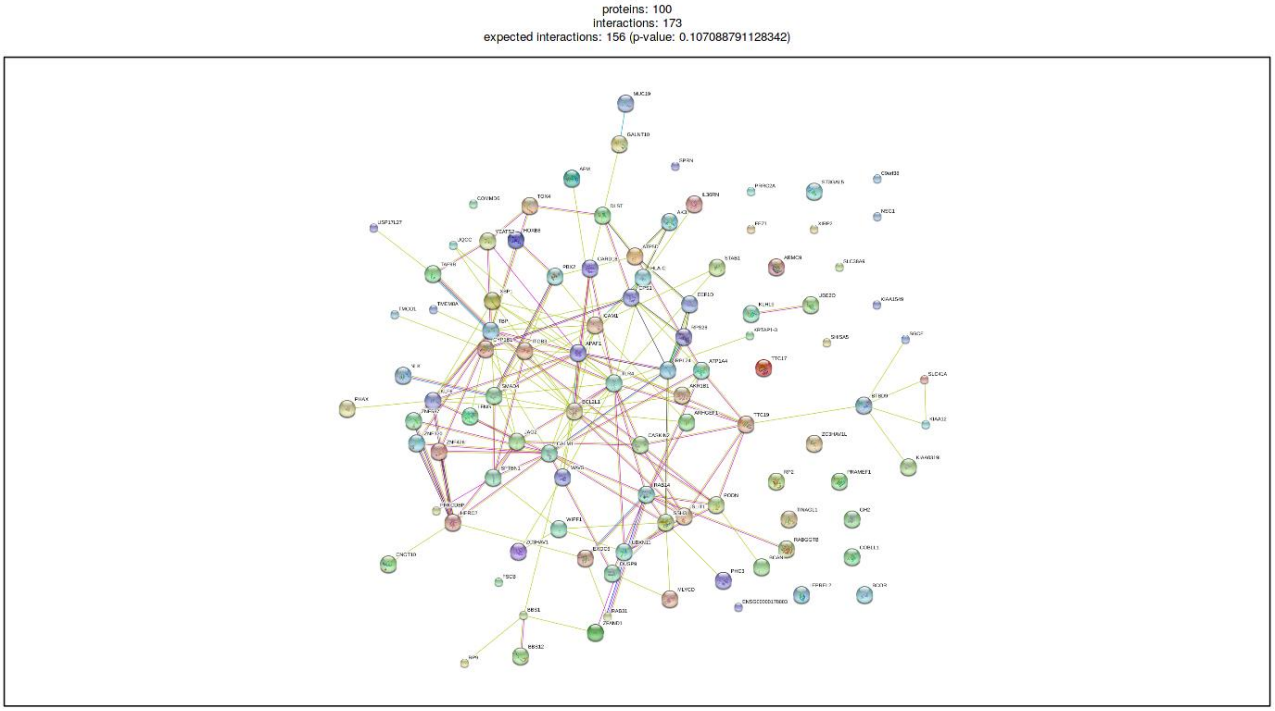


Fig 6. Protein protein interaction network of top 100 proteins

3.4 Pathways analysis

In this basically different pathways were analysed, red and blue indicates activated and suppressed pathways, respectively.

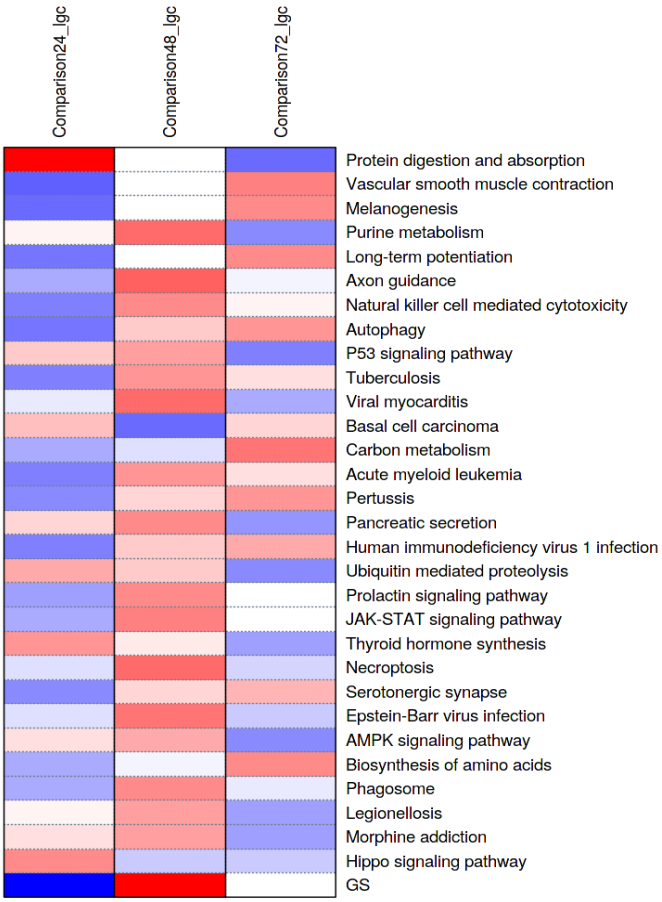


fig 7. Pathways analysis in which red and blue indicates the activated and suppressed pathways

3.5 k-means clustering analysis

In this analysis four clusters of genes were made using the iDEP.91 tool.

Cluster	adj.Pval	nGenes	Pathways	Genes
A	1.29E-06	19	MicroRNAs in cancer	CDCA5, EGFR, APC, IKBKB, MDM2, MDM4, MMP16, NFKB1, PIM1, PLCG1, DDIT4, PRKCA, FSCN1, BRCA1, TP53, UBE2I, VEGFA, CD44, HDAC4
A	4.86E-06	36	Pathways in cancer	ADCY2, DVL1, EGFR, FGF2, FGFR1, GNAS, GSK3B, BIRC5, IGF1, IKBKB, IL2, IL3RA, IL4, IL6ST, ITGA2, JUN, LAMC1, LAMC2, SMAD2, SMAD4, MDM2, NFKB1, NFKB2, PIM1, PLCG1, PMAIP1, CYCS, PRKCA, RALGDS, CXCL12, STAT1, TP53, VEGFA, CASP7, CBL
A	0.00015	25	PI3K-Akt signaling pathway	CREB3, EGFR, FGF2, FGFR1, GH2, GSK3B, IGF1, IKBKB, IL2, IL3RA, IL4, ITGA2, LAMC1, LAMC2, MDM2, NFKB1, PDPK1, DDIT4, PRKCA, SPP1, BRCA1, TP53, VEGFA, VWF, YWHAZ
A	0.00015	13	Th17 cell differentiation	HLA-DRB1, IKBKB, IL1B, IL2, IL4, IL6ST, JUN ,LCK, SMAD2, SMAD4, NFKB1, PLCG1, STAT1
A	0.000295	12	T cell receptor signaling pathway	FYN, GSK3B, IKBKB, IL2, IL4, JUN, LCK, NFKB1, PDPK1, PLCG1, PTPRC, BCL10
A	0.000295	17	Proteoglycans in cancer	EGFR, FGF2, FGFR1, FLNA, GAB1, ANK1, IGF1, ITGA2, SMAD2, MDM2, PDPK1, PLCG1, PRKCA, TP53, VEGFA, CBL, CD44
A	0.000295	11	Colorectal cancer	EGFR, GSK3B, APC, BIRC5, JUN, SMAD2, SMAD4, PMAIP1, CYCS, RALGDS, TP53
A	0.000304	10	P53 signaling pathway	IGF1, IGFBP3, MDM2, MDM4, GTSE1, PMAIP1, CYCS, TP53, CCNB2, EI24
A	0.000517	14	Non-alcoholic fatty liver disease (NAFLD)	CYC1, GSK3B, IKBKB, IL1B, JUN, NDUFS7, NDUFC2, NFKB1, NDUFA13, CYCS, PRKAB2, NDUFA4L2, CASP7, ADIPOQ
A	0.00061	9	Inflammatory bowel disease (IBD)	HLA-DRB1, IL1B, IL2, IL4, IL18, JUN, SMAD2, NFKB1, STAT1
A	0.000704	11	NF-kappa B signaling pathway	CYLD, IKBKB, IL1B, LCK, NFKB1, NFKB2, PLCG1, CXCL12, UBE2I, BCL10, CD40
A	0.000704	11	AGE-RAGE signaling pathway in diabetic complications	IL1B, JUN, SMAD2, SMAD4, NFKB1, PIM1, PLCG1, PRKCA, CCL2, STAT1, VEGFA
A	0.001389	21	Human papillomavirus infection	CREB3, DLG2, DLG3, DVL1, EGFR, GNAS, GSK3B, APC, IKBKB, ITGA2, LAMC1, LAMC2, MDM2, NFKB1, PARD3, PSEN1, SPP1, STAT1, TP53, VEGFA, VWF

A	0.001566	9	Pancreatic cancer	EGFR, IKBKB, SMAD2, SMAD4, NFKB1, RALGDS, STAT1, TP53, VEGFA
A	0.001919	12	Apoptosis	CTSB, BIRC5, IKBKB, IL3RA, JUN, NFKB1, PDPK1, PMAIP1, CYCS, TP53, TUBA4A, CASP7
B	2.10E-07	38	Pathways in cancer	CDKN1A, CDKN1B, TXNRD2, CREBBP, E2F1, EDN1, EPOR, ERBB2, FGF5, FH, GNA11, GRB2, HMOX1, HRAS, KLK3, ITGB1, KIT, KRAS, MET, PLCB2, PML, WNT4, PPARG, MAPK1, MAPK8, BAK1, RAD51, RALA, RELA, BCL2L1, STAT3, TRAF2, WNT10B, BIRC7, CALM3, RASSF5, CASP8, CDC42
B	1.21E-06	15	HIF-1 signaling pathway	CDKN1A, CDKN1B, CREBBP, DN1, EIF4E, ERBB2, GAPDH, HK2, HMOX1, MAPK1, RELA ,STAT3, TEK, TLR4, HKDC1
B	1.14E-05	12	Pancreatic cancer	CDKN1A, E2F1, ERBB2, KRAS, MAPK1, MAPK8, BAK1, RAD51, RALA, RELA, BCL2L1, STAT3
B	1.44E-05	17	Hepatitis B	CDKN1A, TIRAP, CREBBP, E2F1, GRB2, HRAS, HSPG2, KRAS, MAPK1, MAPK8, RELA, SLC10A1, STAT3, TLR2, TLR4, CASP8, CASP10
B	6.33E-05	11	Chronic myeloid leukemia	CDKN1A, CDKN1B, E2F1, GRB2, HRAS, KRAS, MAPK1, BAK1, PTPN11, RELA, BCL2L1
B	6.73E-05	17	Kaposi sarcoma-associated herpesvirus infection	CDKN1A, CREBBP, E2F1, HRAS, ICAM1, KRAS, LYN, PPP3CA, MAPK1, MAPK8, BAK1, RELA, STAT3, SYK, TRAF2, CALM3, CASP8
C	6.89E-05	15	MicroRNAs in cancer	CDKN1A, CDKN1B, CREBBP, E2F1, ERBB2, GRB2, HMOX1, HRAS, ITGB3, KRAS, MET, MAPK1, BAK1, STAT3, CDC25A
C	6.89E-05	12	Prostate cancer	CDKN1A, CDKN1B, CREBBP, E2F1, ERBB2, GRB2, HRAS, KLK3, KRAS, MAPK1, RELA, TMPRSS2
C	6.89E-05	10	Non-small cell lung cancer	CDKN1A, E2F1, ERBB2, GRB2, HRAS, KRAS, MAPK1, BAK1, STAT3, RASSF5
C	9.32E-05	16	Tuberculosis	CEBPB, TIRAP, CREBBP, IL1A, IL10, IL10RB, PPP3CA, MAPK1, MAPK8, RELA, SYK, TLR2, TLR4, CALM3, CASP8, CASP10
C	0.000187	18	Ras signaling pathway	CSF1, FGF5, KSR2, GRB2, HRAS, KIT, KRAS, MET, MAPK1, MAPK8, PTPN11, RALA, RELA, BCL2L1, TEK, CALM3, RASSF5, RIN1
C	0.000188	15	Alzheimer disease	COX8A, GAPDH, APOE, APP, NDUFA10, NDUFAB1, PLCB2, PPP3CA, MAPK1, SDHD, ADAM17, CACNA1S, CALM3, CASP8, COX5A
C	0.000188	16	Huntington disease	CLTC, COX8A, CREBBP, DNAH5, DNAI1, NDUFA10, NDUFAB1, PLCB2, POLR2H, POLR2L, PPARG, SDHD, SOD1, TBP, CASP8, COX5A

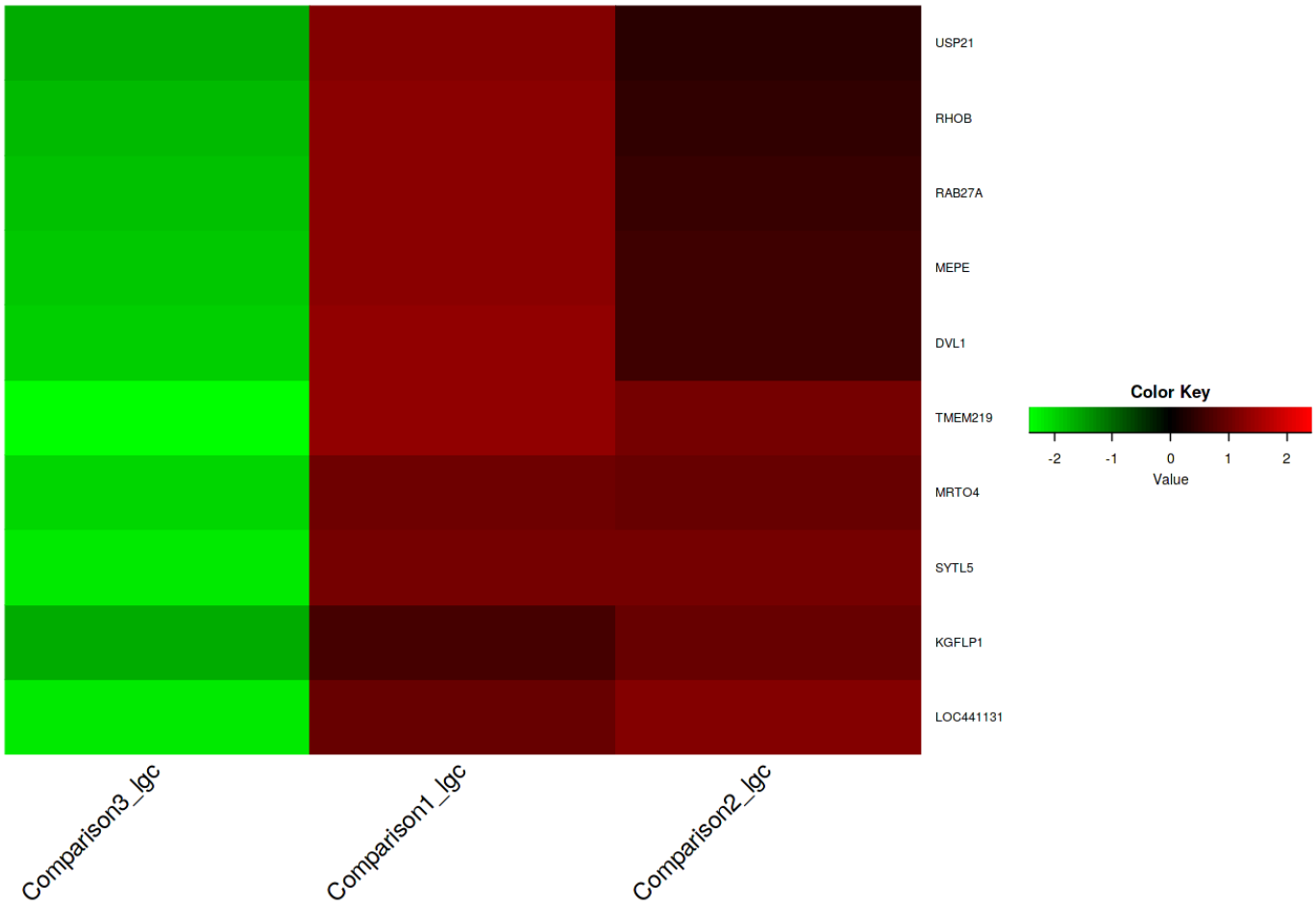
C	0.000188	12	Toxoplasmosis	IL10, IL10RB, ITGB1, MAPK1, MAPK8, RELA, BCL2L1, STAT3, TLR2, TLR4, BIRC7, CASP8
D	0.000257	16	Proteoglycans in cancer	CDKN1A, ERBB2, GRB2, HRAS, HSPG2, ITGB1, ITGB3, KRAS, MET, WNT4, MAPK1, PTPN11, STAT3, TLR2, TLR4, WNT10B

Table 5. k-Means clustering of the genes into four cluster

3.6 Heat map analysis

Through the heat map analysis we found the most significant genes and their level of expression at different time point:

Fig 8. Heat map analysis in which the most significant genes and their level of expression at different time point



3.7 Role of some significant genes in other cancer and diseases

- **USP21 :-** This gene, when it is overly expressed which leads to the formation of the non small cell lung cancer(NSCLC). This gene also supports the NSCLC cell proliferation.
- **RAB27A :-** when this gene is over expressed which increase the malignancy in breast cancer cells through the secretion of IGF-II(Insulin Like Growth Factor-II).
- **MEPE :-** When the over expression this gene is achieved which leads to decrease in bone mass and ultimately refers to osteomalacia. It causes bone related problem too.
- **DVL1 :-** When the overexpression of this gene take place which promotes the prostrate cancer by the beta-catenin signalling. It is also upregulated in the cervical cancer.
- **SYTL5 :-** Basically this gene is a vesicle trafficking gene, so when this gene gets up-regulated which promotes the expression of protein in breast tumours.

4. CONCLUSION

In India the *Achyranthes aspera* is a significant herbal plant which is found as a weed. In spite of the fact that practically the entirety of its parts are utilized in conventional frameworks of drugs, seeds, shoots and roots. These are the main significant parts that are utilized therapeutically. Using the bioinformatics tools, bioinformatics-analysis has been done on the plant to find the most significant genes which are downregulated so as to treat the cancer cells. Many cancer relatead genes were downregulated by the use of the plant extract over the different period of time which show the pharmacological behaviour of *Achyranthes aspera*. Now a days situation is that we are utilizing this weed in different sort of ailments and disease. Its pharmacological qualities have the incredible capability of healing up sicknesses. The herbal plants are utilized for treatment of different illnesses on account of their well being and viability. This study shows that the plant is having medicinal properties by which it is able to downregulate some genes which are upregulated in the cancer cells.

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