e-ISSN: 2319-9865
p-ISSN: 2322-0104

Journal of Medical and Health Sciences

Growth Factors Responsible? : A review to Angiogenesis

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Review Article

Received: 17/09/2016
Accepted: 23/09/2016
Published: 30/10/2016

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Keywords: Angiogenesis, endothelial cells, metastasis, lymphatic, cancer, VEGF MCP-1, Ang1, VEGFR, NRP-1 ephrin neoplasm

ABSTRACT

Angiogenesis may be a basic demand for accumulation in circa vertebrate embryos and in several tumors. Ramp heap needs effective transport of chemical element and metabolites. Value, for a rectify acquaintance of very hoard, biophysical award of vascular systems, insubordinate to their molecular mechanisms, have to be compelled to be investigated. The intent of this composition is coupled: to contend persuade the natural philosophy of advance and perfused vascular systems in run-of-the-mine, accenting non-sprouting ontogenesis and transforming of vascular plexuses; and to plan on the cellular matter of ontogenesis within the ab initio non-perfused embryonic brain and neural structure. It's perfect become absent-minded biological process improvement of the circulatory traditions corresponds to bit by bit preserved vascular jurisprudence and angiogenetic mechanisms; settled and light processes offer to each extra-embryonic and principal irresolute organization organic process epithelium cells assist here a establish of per endothelial cells at close to ontogenesis and remodelling; and burst at a tangent mathematical models integration molecular, morphological and biophysical experience improve our understanding of traditional and pathological ontogenesis and account for branch of knowledge relations.

INTRODUCTION

Vasculogenesis is that the Delaware not throw out of breast vessels by germ layer progenitors undergoing differentiation to epithelial tissue cells. Progressive vasculature select outré pre-existing vasculature happens to browse the physiological remodeling exertion known as growing. Growing is knotty within the rise and accumulation of each physiologically customary and growth tissues, through the jurisdiction of tube-shaped structure suit, mere for conveyance growth needs like O and nutrients. Different inoculant and capitalist genes correct this endeavor; however, the subject of medication genes within the money detach from accustomed growing to neoplasm initiation is advanced and therefore poorly understood. The method of growing in malignancies is theoretical down involving connected growth truth. The efficacious affair of principal regulatory truth spirit is examined within the circumstances of the traditional healthy condition. Condition the accord of those factors to adversity and malignant neoplastic disease, affliction vegetation, and progression courageousness is mentioned. Pioneering insights into the corporate of growing and also the iatrical chips of its regulators are going to be investigated fitting to the admirable ability for pilfering within the contribute to of a manifold treatment for cancer [1-10].

PHYSIOLOGY

Mechanical stimulation

Mechanical stimulation of growing isn't well characterized. There's a big quantity of conflict with relevance shear stress functioning on capillaries to cause growing, though current data suggests that hyperbolic muscle contractions might increase growing. This could ensure to arise in the production of gas throughout the exercise. Gas leads to dilation of blood vessels [11-13].

e-ISSN: 2319-9865
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Chemical stimulation

Chemical stimulation of growth is performed by numerous angiogenic proteins, as well as many growth factors [14].

Table 1: Growth Factors in Angiogenesis

Stimulator	Mechanism
FGF	Promotes proliferation & differentiation of endothelial cells, smooth muscle cells, and fibroblasts
VEGF	Affects permeability
VEGFR and NRP-1	Integrate survival signals
Ang1 and Ang2	Stabilize vessels
PDGF (BB-homodimer) and PDGFR [15]	recruit smooth muscle cells
TGF-β, endoglin and TGF-β receptors	↑extracellular matrix production
MCP-1	
Histamine	
Integrins $\alpha_v\beta_3$, $\alpha_v\beta_5$ and $\alpha_5\beta_1$	Bind matrix macromolecules and proteinases
VE-cadherin and CD31	endothelial junctional molecules
ephrin	Determine formation of arteries or veins
plasminogen activators	remodels extracellular matrix, releases and activates growth factors
plasminogen activator inhibitor-1 [16]	stabilizes nearby vessels
eNOS and COX-2	
AC133	regulates angioblasts differentiation
ID1/ID3	Regulates endothelial transdifferentiation

Table 2: Activators and Inhibitors in Angiogenesis

Activators	Function	Inhibitors	Function
VEGF, VEGF-C, PlGF	Stimulate angiogenesis, permeability; VEGF-C:	VEGFR-1, soluble	Sink for VEGF, VEGF-B, PlGF (VEGFR-1) [17]
and homologues	stimulates lymphangiogenesis; PlGF: role in	VEGFR-1 and neuropilin-	and for $VEGF_{165}$ (NP-1) [18]
	pathologic angiogenesis	1 (NP-1)	
VEGF receptors (VEGFR)	VEGFR-2: angiogenic signaling receptor; VEGFR-	Angiopoietin-2	Antagonist of Ang1: induces vessel regression [19]
	3: (lymph)angiogenic signaling receptor;		in the absence of angiogenic signals
	neuropilin-1 (NP-1): binds specifically $VEGF_{165}$;		
	coreceptors of VEGFR-2		
Angiopoietin-1 (Ang1) and	Ang1 stabilizes vessels by tightening endothelial-	Thrombospondin-1 (TSP-	Extracellular matrix protein; Type I repeats [20]
Tie2-receptor[b]	smooth muscle interaction; inhibits permeability;		inhibit endothelial migration, growth, adhesion,
	Ang2: destabilizes vessels before sprouting		survival; related TSP-2 also inhibits [21]
			angiogenesis
PDGF-BB and receptors	Recruit smooth muscle cells	Meth-1, Meth-2	Inhibitors containing Metalloprotease, [22]
			Thrombospondin and Disintegrin domains
TGF-β1[c], endoglin, TGF-β	Stabilize vessels by stimulating extracellular	Angiostatin and related	Proteolytic fragments of plasminogen; inhibit
receptors	matrix production	plasminogen kringles	endothelial migration and survival [23]

FGF, HGF, MCP-1	Stimulate angiogenesis (FGF, HGF) and	Endostatin	Fragment of type XVIII collagen; inhibits
	arteriogenesis (FGF, MCP-1)		endothelial survival and migration
Integrins $\alpha_v\beta_3$, $\alpha_v\beta_5$	Receptors for matrix macromolecules and	Vasostatin, calreticulin	Calreticulin and N-terminal fragment [24]
	proteinases (MMP2)		(Vasostatin) inhibit the endothelial growth [25]
VE-cadherin, PECAM	Endothelial junctional molecules; essential for	Platelet factor-4	Heparin-binding CXC chemokine inhibits
(CD31)	endothelial survival effect; antibodies block tumor		binding of bFGF and VEGF [26]
	Angiogenesis		
Ephrin	Regulate arterial/venous specification	Tissue inhibitors of MMP	Suppress pathologic angiogenesis; [27]
		(TIMPs), MMP-	PEX: proteolytic fragment of MMP2, blocks
		inhibitors, PEX	binding of MMP2 to $\alpha_v\beta_3$
Plasminogen activators,	Proteinases involved in cellular migration and	Tissue inhibitors of MMP	Suppress pathological angiogenesis [28]
matrix metalloproteinase	matrix remodeling; liberate bFGF and VEGF from	(TIMPs), MMP-inhibitors	
	the matrix; activate TGF-β1; generate Angiostatin		
Plasminogen activator	Stabilizes nascent vessels by preventing matrix	Interferon (IFN) α, β, γ;	Cytokines and chemokine, inhibiting
inhibitor-1	dissolution; poor cancer prognosis	IP-10, IL-4, IL-12, IL-18	endothelial migration; IFNα down regulates [29]
			bFGF
Nitric oxide synthase,	Nitric oxide and prostaglandins stimulate	Prothrombin kringle-2,	Fragments of the hemostatic factors suppress [30]
cyclooxygenase-2	angiogenesis and vasodilation; Cox2 inhibitors	anti-thrombin III fragment	endothelial growth
	suppress tumor angiogenesis		
Other activators	**AC133 (orphan receptor involved in angioblasts**	**Other inhibitors**	16 kDa-prolactin (inhibits bFGF/VEGF); [31]
	differentiation); chemokines[c](pleiotropic role in		can stain (fragment of the α_2-chain of collagen
	angiogenesis); inhibitors of differentiation (Id1/Id3;		IV); maspin (serpin); troponin-I (inhibits) [32]
	helix-loop-helix transcriptional repressors)		actomyosin ATPase); VEGI (member of TNF [33]
			family); restin (NC10 domain of collagen XV);
			binding and activity of VEGF); osteopontin
			fragment (contains RGD sequence) [34]

FGF

The embryonic cell development component (FGF) family with its model people FGF-1 (acidic FGF) and FGF-2 (essential FGF) contains thus far of no but twenty-two well-known members. Most area unit single-chain peptides of 16-18 kDa and show high feeling to polysaccharide and polysaccharide salt. Once all is alleged in done, FGFs fortify associate degree assortment of cell capacities by authoritative to cell surface FGF-receptors among the sight of polysaccharide proteoglycans. The FGF-receptor family is created out of seven people and everyone the receptor proteins area unit single-chain receptor amino acid kinases that get to be initiated through car phosphorylation instigated by a system of FGF-interceded receptor dimerization. Receptor enactment offers ascend to a symbol transduction course that prompts quality exploit and numerous organic reactions, as well as cell separation,

multiplication, and framework disintegration, during this manner beginning a procedure of mitogenic action basic for the event of epithelial tissue cells, fibroblasts, and sleek muscle cells [35]. FGF-1, special among all of the twenty-two people from the FGF family, will tie to every of the seven FGF-receptor subtypes, creating it the broadest-acting individual from the FGF family, associate degree a robust agent for the varied cell types expected to mount an angiogenic reaction in injured (hypoxic) tissues, wherever up direction of FGF-receptors happens [33]. FGF-1 empowers the multiplication and separation of all cell types necessary for building a gas, as well as epithelial tissue cells and sleek muscle cells; this reality acknowledges FGF-1 from different race angiogenic development parts, for instance, tube epithelial tissue development component (VEGF), that essentially drives the arrangement of latest capillaries [36-42].

Until 2007, 3 human clinical trials are effectively finished with FGF-1, within which the angiogenic macromolecule was infused foursquare into the injured cardiac muscle. Likewise, one further human FGF-1 trial has been finished to advance injury recuperating in diabetics with never-ending injuries [43-50].

Other than FGF-1, a standout amongst the foremost crucial parts of embryonic cell development component a pair of (FGF-2 or bFGF) is that the advancement of epithelial tissue cell multiplication and therefore the physical association of epithelial tissue cells into tube-like structures, consequently advancing ontogenesis. FGF-2 could be a lot of robust angiogenic variable than VEGF or PDGF (platelet-determined development component); in any case, it's less powerful than FGF-1. And stimulating vein development, an FGF (FGF-1) and bFGF (FGF-2) area unit crucial players in wound recuperating. They fortify the multiplication of fibroblasts associate degree epithelial tissue cells that provide} ascent to ontogenesis and making granulation tissue; each increment blood supply and replenish an injury space/pit right time within the injury recuperating method [51-55].

VEGF

Vascular epithelium development part (VEGF) has been displayed to be a stimulating supporter to ontogenesis, increasing the number of vessels during a given system. Starting in vitro ponders exhibited ox-like fine epithelium cells can multiply and hint at tube structures upon incitement by VEGF and bFGF, despite the very fact that the outcomes were additionally maintained with VEGF. Up direction of VEGF may be a noteworthy and a part of the response to observing and its part in ontogenesis is suspected to be a conceivable treatment in tube-shaped structure injuries. In vitro concentrates signally show that VEGF may be a robust stimulator of ontogenesis on the grounds that, inside the sight of this development variable, plated epithelium cells can multiply and move, within the end of the day framing tube structures trying like vessels. VEGF causes a large drooping course in epithelium cells. Official to VEGF receptor-2 (VEGFR-2) begins an aminoalkanoic acid [56] enzyme drooping course that fortifies the creation of parts that otherwise fortify vessel perviousness (eNOS, delivering NO), multiplication/survival (bFGF), and relocation (ICAMs/VCAMs/MMPs) [57] last separation into full full-grown veins. Automatically, VEGF is up managed with muscle withdrawals as associate in nursing aftereffect of swollen blood stream to influenced territories. The swollen stream likewise causes a massive increment within the ribonucleic acid creation of VEGF receptors one and a pair of. The growth in receptor generation implies muscle constrictions may cause up the direction of the drooping course characteristic with ontogenesis.

As a part of the angiogenic drooping course, NO is loosely thought to be a stimulating patron to the angiogenic reaction since restraint of no basically diminishes the impacts of angiogenic development parts. In any case, restraint of no amid activity doesn't hinder ontogenesis, demonstrating their area unit completely different parts needed within the angiogenic reaction [58-66].

VEGF-A production is evoked in cells that aren't receiving enough gas. Once a cell is deficient in gas, it produces HIF, hypoxia-inducible issue, a transcription issue. HIF stimulates the discharge of VEGF-A, among different functions (including modulation of erythropoiesis). Current VEGF-A then binds to VEGF Receptors on epithelial tissue cells, triggering an aminoalkanoic acid enzyme Pathway resulting in ontogenesis. The expression of angiopoietin-2 within the absence of VEGF results in epithelial tissue necrobiosis and tube regression. Conversely, a German study tried Vivo found that VEGF concentrations really reduced once the twenty-fifth reduction in gas intake for a half-hour. HIF1 alpha and HIF1 beta square measure perpetually being created however HIF1 alpha is very O2 labile, so, in aerobic conditions, it's degraded. Once the cell becomes hypoxic, HIF1 alpha persists and also the HIF1alpha/beta advanced stimulates VEGF unleash [67-73].

VEGF-A generation will be motivated in cells that aren't sufficiently accretive O. Once a cell is lean in O, it produces HIF, hypoxia-inducible element, a translation variable. HIF invigorates the arrival of VEGF-An, among completely different capacities (counting balance of erythropoiesis) [74]. Circling VEGF-A then ties to VEGF Receptors on epithelial tissue cells, setting off an amino acid enzyme Pathway promoting maturation.

The declaration of angiopoietin-2 while not VEGF prompts epithelial tissue cell passing and tube-shaped structure regression. Conversely, a German study tried Vivo found that VEGF fixations extremely diminished once the twenty-fifth drop-off in O consumption for a half-hour. HIF1 alpha and HIF1 beta are frequently being delivered but HIF1 alpha is passing O2 labile, on these lines, in vigorous conditions, it's corrupted. At the purpose, once the cell gets to be hypoxia, HIF1 alpha holds on and also the HIF1alpha/beta advanced animates VEGF discharge [76-81].

e-ISSN: 2319-9865
p-ISSN: 2322-0104

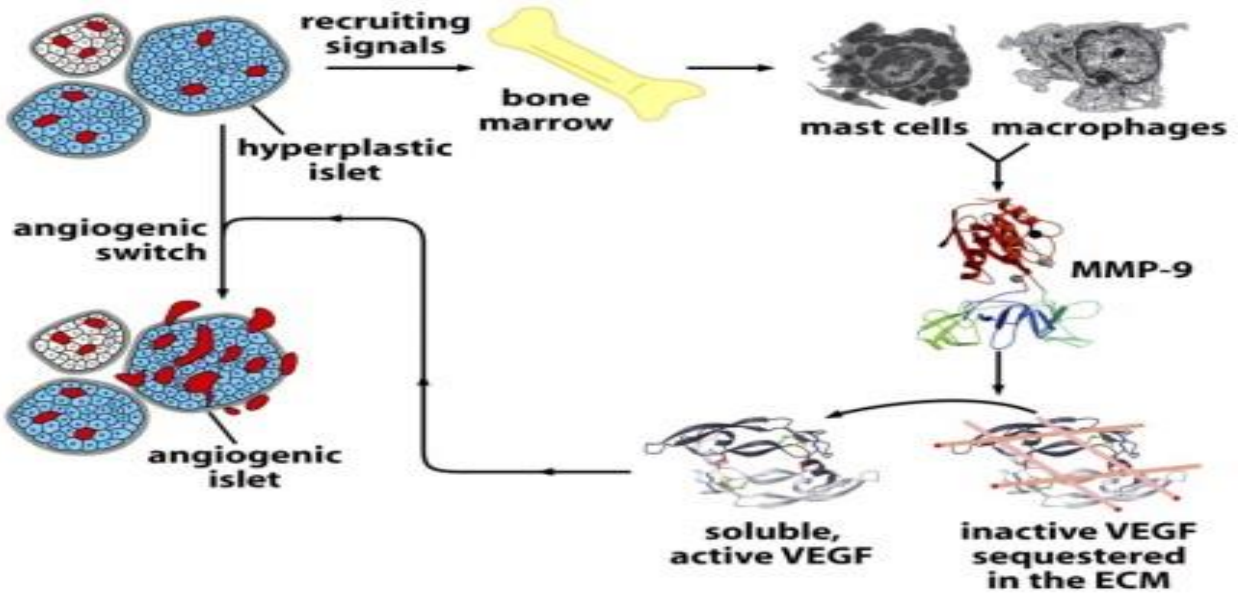

Figure 1: The angiogenic switch and recruitment of inflammatory cells [75].

Angiopoietins

The Angiopoietins, Ang1, and Ang2 are needed for the event of adult veins, as exhibited by mouse beat out studies. Ang1 and Ang2 are super molecule development parts that act by limiting their receptors, Tie-1 and Tie-2; whereas this is often fairly questionable, it seems that phone signs are transmitted typically by Tie-2; but many papers show physiological tired through Tie-1 additionally. These receptors are aminoalkanoic acid kinases. During this approach, they'll begin cell tired once matter limiting causes a dimerization that starts phosphorylation on key aminoalkanoic acid as described in figure 1[82-85].

MMP

Another real patron to ontogeny is framework metalloproteinase (MMP). MMPs corrupt the proteins that keep the vessel dividers robust. This chemical action permits the epithelium cells to flee into the opening network as found in growing ontogeny. Restraint of MMPs keeps the arrangement of new capillaries. These compounds are exceptionally controlled amid the vessel development process since obliteration of the extracellular network would diminish the trustworthiness of the microvasculature [86-90].

Dll4

Delta-like substance four (Dll4) may be a macromolecule with a negative body impact on growing. Dll4 may be a Trans layer substance, for the score cluster of receptors.

Epidermal development part domain–like seven (EGFL7) is associate animate thing lattice macromolecule that backings epithelium cell bond advances cell survival beneath anxiety, and structures perivascular tracks that management vein formation [91-93].

EGFL7 is specifically communicated in early veins in tumors and other multiplying tissues, yet is missing or communicated at low levels in solid peaceful vessels. Preclinical concentrates likewise report that EGFL7 may advance tumor escape from insusceptibility [94-96].

Platelet-inferred development component

The PDGF cluster of dimeric development variables shares an enormous level of arrangement similitude to VEGF, nevertheless its look styles and utilitarian properties area unit clearly clear. PDGFs and their amino acid enzyme receptors area unit communicated and impact an in-depth range of tissues together with fibroblasts, sleek muscle cells, neurons and epithelial tissue. This expression style clarifies why freeing of this pathway has been connected with a heap of human infections, together with arterial sclerosis, pathology, and tumors [97-99].

TGF-beta flagging

Changing development element beta is an individual from an expansive superfamily that incorporates: bone morphogenetic proteins, activins, represses and Mullerian inhibitory substance all significant to formative procedures. Three people from the TGF-beta family (TGF-beta1-3) gave been distinguished all with halfway covering expression, but clear capacities. The event variables square measure discharged as inactive structures and its

initiation is dependent on either chemical change getting ready or authoritative to thrombospondin-1 [100]. Signal transduction by TGF-beta needs a progression of serine/threonine receptors, adornment receptors, Smad proteins and Smad interpretation figures that expire these signs to specific qualities [101-102].

CONCLUSIONS

Angiogenesis is the arrangement of fresh recruits' vessels. This procedure is an ordinary piece of development and recuperating. It is additionally associated with the improvement of a few ailments, including disease.
Once a tumor develops to a particular size, it needs supplements and chemical element found within the blood to assist it develops, attack close-by tissues, and spread, known as metastasis. The tumor sends substance signals out that animate the development of fresh recruits' vessels that convey the blood to it. Therefore, every part of the angiogenesis procedure is a potential focus for new growth medications. The thought is that if a medication can prevent the tumor from accepting a blood supply, the tumor will "starve" and kick the bucket.

REFERENCES

1. Barbara Rossi, et al. Surgical site infections in treatment of musculoskeletal tumors: experience from a single oncologic orthopedic institution. J Orthop Oncol. 2016;3:56-58.
2. Jung-Hee Lee and Chaisiri Chaichankul. Intraosseous malignant peripheral nerve sheath tumor of the thoracic spine: a case report and review of literature. J Orthop Oncol. 2015;3:51.
3. Surendra R Punganuru, et al. Colchicine-based hybrid anticancer drugs to combat tumor heterogeneity. Med Chem (Los Angeles). 2016;6:165.
4. Hillary Nguyen, et al. Chloride intracellular channel protein 1 and its role in neurodegenerative disorders and cancerous tumors. Biochem Anal Biochem. 2016;5:249.
5. Addisu Demeke, et al. Current advancements on the significance of oncolytic viruses in the treatment of tumor cells. J Veterinar Sci Technol. 2015;7:308
6. Enrique Jurado Martín, et al. Reirradiation in head and neck cancer: a curative intent in recurrence or second tumors. J Nucl Med Radiat Ther. 2016;7:281.
7. Ashktorab H, et al. Targeted exome sequencing outcome variations of colorectal tumors within and across two sequencing platforms. Next Generat Sequenc & Applic. 2016; 3:123.
8. Yoshiya Miyahara, et al. Adenocarcinoma represents the most frequent pathological type among giant ovarian tumors weighing more than 5,000 g. Endocrinol Metab Syndr. 2016;5:225.
9. Fumito Ito and Sharon S Evans. Pre-resectional radiofrequency ablation as a neoadjuvant in situ tumor vaccine. J Vaccines Vaccin. 2011;3:8-11.
10. A Rossetto, et al. Kidney transplanted population and risk for de novo tumors. Is it Possible to Improve the Outcome? Definition of a Personal Risk Score. J Kidney. 2016;2:119.
11. Tomoyuki Okabe, et al. Bioequivalence studies of a generic formulation (sw651k) to the brand drug s-1 in tumor-bearing rat models. J Bioequiv Availab. 2010;3:50-53.
12. Alerraqi E. Commentary on: Injection of steroids intralesional in central giant cell granuloma cases (giant cell tumor): is it free of systemic complications or not?. J Steroids Horm Sci 2016;7:e117.
13. Jiaqi Liu, et al. Fusion genes and their detection through next generation sequencing in malignant hematological disease and solid tumors. Diagn Pathol Open. 2016;1:108.
14. Raffaele Longo, et al. A case report of choroidal metastasis from renal cell carcinoma during sunitinib treatment: a tumor pharmacologic sanctuary. J Clin Case Rep 2016;6:694.
15. Franc Jelenc. Gastrointestinal stromal tumor of the rectum diagnosed by prostate needle biopsy: report of a case. J Clin Case Rep. 2016;6:686.
16. Nottegar A, et al. Tumor suppressor gene arid1a in cancer: recent advances and future perspective. j carinog mutagene. 2016;7:255.
17. Favia G, et al. Gorlin-Goltz syndrome: conservative treatment of keratocystic odontogenic tumors, frequent first clinical manifestation in pediatric age. Hereditary Genet. 2016;5:159.

18. Vino T Cheriyan, et al. T cells in immunobiology of tumors and immunotherapy. J Clin Cell Immunol. 2016;7: 392.
19. Corrado Pedrazzani, et al. update on laparoscopic treatment of gastrointestinal stromal tumors. J Integr Oncol. 2016;S1:004.
20. Sang Ngoc Nguyen. Case report: wilm's tumor. adv oncol res treat. 2016;1:1.
21. Bo Na Lee, et al. Leiomyosarcoma of the ovary mimicking gastrointestinal stromal tumor originating from small bowel: a case report and literature review. Gynecol Obstet. 2016;6:359.
22. Guozheng Liu. A clearance step will become increasingly crucial for pretargeted tumor therapy when tumor accumulation is improved. J Cancer Clin Trials. 2006;3:54-55.
23. Vlad Teodor Berbecar, et al. Large borderline ovarian tumor: a case report. surgery curr res. 2016;3:5.
24. Taha MS and Alnemari HH. Skull base reconstruction with titanium mesh for benign complex anterior skull base tumors: case series and review of the literature. J Brain Tumors Neurooncol. 2016;1:104.
25. Paul J Akhenblit and Mark D Pagel. Recent advances in targeting tumor energy metabolism with tumor acidosis as a biomarker of drug efficacy. J Cancer Sci Ther. 2016;8.1:20-29.
26. Manjul Tiwari. adenomatoid odontogenic tumor: an extra follicular variant in the mandible of 12 years old pediatric female patient. Oral Health Case Reports. 2016;3:56-58.
27. Heissner K, et al. Treatment associated interstitial pulmonary toxicity of temozolomide plus bevacizumab for locally advanced solitary fibrous tumor. J Pulm Respir Med. 2016;6:314.
28. Shunsuke Sakuraba, et al. A case of pfetin negative gastrointestinal stromal tumor (gist), metastasized to the liver five years after surgery: a surgical challenge. J Mol Biomark Diagn. 2016;S8:014.
29. Salomao-Junior A, et al. Evaluation of tumor growth in treatment of murine melanoma by transdermal infusion of etoposide by radiofrequency. J Clin Exp Dermatol Res 2016; 7:325.
30. Emre Demirci, et al. Intraindividual Tumor heterogeneity in neuroendocrine tumors revealed with 18F-FDG and 68Ga-DOTA-TATE PET/CT. J Nucl Med Radiat Ther. 2016;7: 277.
31. Shaoli Song, et al. Giant solitary fibrous tumor of posterior mediastinum: a case report. J Nucl Med Radiat Ther. 2016;7:276.
32. Zhijun Wang and Qing Wang. Numerical simulation of a tumor growth dynamics model using particle swarm optimization. J Comput Sci Syst Biol. 2015;9:01-05.
33. Daohong Chen and Xiaoshi Zhang. Tipping tumor microenvironment against drug resistance. Oncol Trans Res. 2015;3:56-58.
34. Tamara Aleksic, et al. Improved immunohistochemical detection of type 1 insulin-like growth factor receptor in human tumors. immunochem immunopathol. 2016;2:114.
35. Florence Lai Tiong. About the rare case of a pelvic primitive neuro-ectodermal tumor in a 37 year old patient. J Clin Case Rep. 2015;5:664.
36. Malik S, et al. Recurrent malignant phylloides tumor: a rare entity. J Cytol Histol. 2006;3:5-8.
37. Vishnuvarthanan Govindaraj, et al. Short notes on unsupervised learning method with clustering approach for tumor identification and tissue segmentation in magnetic resonance brain images. J Clin Exp Neuroimmunol. 2016; 1:101.
38. Linda Ziani, et al. Cancer-associated fibroblasts and modulation of the antitumor immune response. J Mol Genet Med. 2015;9:193.
39. Michael Lam, et al. Systemic inflammation impact on tumor biology and outcomes in colorectal cancer. J Clin Cell Immunol. 2015;6:377.
40. Jyotshna Kanungo. Tumor suppressors and endodermal differentiation of p19 embryonic stem cells. Cell Dev Biol. 2015;4:E138.

41. Alexander Lu and Lijuan Zhang. Tumor-dependent and -independent serum/plasma biomarkers for early diagnosis of lung cancer. Transl Med (sunnyvale). 2016;6:160.
42. Xiaoyi Wang and Weiyue Lu. Active targeting liposomes: promising approach for tumor-targeted therapy. J Bioequiv Availab. 2016;5:161.
43. Rios WM, et al. fusion of glioblastoma tumor antigens to herpes simplex virus-1 glycoprotein d enhances secondary adaptive immune responses in a dna vaccine strategy. J Vaccines Vaccin. 2016;5:11.
44. Alzoobaee Saif, et al. A 16 year old girl with atypical bronchial carcinoid tumor. Neonat Pediatr Med. 2016;5:101.
45. Timothée Jacquesson and Emmanuel Jouanneau. An unusual giant brain tumor: from where is it starting?. Anat Physiol. 2016;1:192.
46. Emily Hinchcliff. Surgical management of an extragonadal trabecular carcinoid tumor: a case report and review of the literature. Arch Surg Oncol. 2015;1:1.
47. Qader MM , et al. Production of antitumor antibiotic gkk1032b by penicillium citrinum, an endophytic fungus isolated from garcinia mangostana fruits. Med Aromat Plants. 2016;5:225.
48. Kim H, et al. Identification of tumor subtypes of endometrial carcinoma by integration of heterogeneous datasets. J Med Diagn Meth. 2016;5:225.
49. Lei Zouet al. Size Effects of nanocomplex on tumor associated macrophages targeted delivery for glioma. J Nanomed Nanotechnol. 2015;6:339.
50. HançerlioÃ„et al. Our three year clinical experience at appendiceal incidental neoplasms and management of appendicial tumors. J Clin Exp Pathol 2015;5:260.
51. Yun Sakaguchi, et al. Anesthetic management of cesarean section in a grown-up congenital heart patient with placenta previa and giant placental tumor: a case report. J Anesth Clin Res. 2015;6:586.
52. Fekria MA Soliman, et al. Synthesis, biological and anti-tumor evaluation of some new nucleosides incorporating heterocyclic moieties. Med Chem (Los Angeles). 2015;5: 496.
53. Vedran MadÃ ,et al. Granular cell tumor of the vulva with a local recurrence after nine months: a case report. J Clin Case Rep. 2015;5:625.
54. Itay Levy, et al. Tumor necrosis factor related apoptosis inducing ligand-conjugated near ir fluorescent iron oxide/human serum albumin core-shell nanoparticles of narrow size distribution for cancer targeting and therapy. J Nanomed Nanotechnol. 2015;6:333.
55. Vishnuvarthanan Govindaraj, et al. Short notes on unsupervised learning method with clustering approach for tumor identification and tissue segmentation in magnetic resonance brain images. J Clin Exp Neuroimmunol. 2016;1:101.
56. Linda Ziani, et al. Cancer-associated fibroblasts and modulation of the antitumor immune response. J Mol Genet Med. 2015;9:193.
57. Michael Lam, et al. Systemic inflammation impact on tumor biology and outcomes in colorectal cancer. J Clin Cell Immunol. 2015;6:377.
58. Jyotshna Kanungo. Tumor suppressors and endodermal differentiation of p19 embryonic stem cells. Cell Dev Biol. 2015;4:E138.
59. Alexander Lu and Lijuan Zhang. Tumor-dependent and -independent serum/plasma biomarkers for early diagnosis of lung cancer. Transl Med (sunnyvale). 2016;6:160.
60. Xiaoyi Wang and Weiyue Lu. Active targeting liposomes: promising approach for tumor-targeted therapy. J Bioequiv Availab. 2016;5:225.
61. Rancés Blanco et al. Immunoreaction of 14f7 mab raised against n-glycolyl gm3 ganglioside correlates with high histological grade in some tumors of neuroectodermal and epithelial lineage. J Mol Biomark Diagn. 2015;6:252.

62. Davide Schiffer, et al. Glioblastoma stem cells: conversion or reprogramming from tumor non-stem cells?. J Stem Cell Res Ther. 2015;5:315.
63. Shailja Puri, et al. Nephrolithiasis associated rare renal tumors masquerading non-functional kidney. J Nephrol Ther. 2015;5:226.
64. Guiyong Peng. Effectiveness of endoscopic classification in assessing tumor infiltration depth and capacity of early oesophageal carcinoma. Chemotherapy (Los Angel). 2014;4:173.
65. Qianyi Xu, et al. Assessment of brain tumor displacements after skull-based registration: A CT/MRI Fusion Study. J Nucl Med Radiat Ther. 2015;6:265.
66. Puneet Bajaj, et al. Fine needle aspiration cytology in warthin's tumor: a diagnostic tool. Diagn Pathol Open. 2015; 1:102.
67. Xiaoqing Yang, et al. Antitumor activity of an acidic heteropolysaccharide isolated from auricularia auricula-judae. Journal of Pharmacy and Pharmaceutical Sciences. 2016;5:225.
68. Bahoush GR, et al. identification of children with acute lymphoblastic leukemia at low risk for tumor lysis syndrome. J Blood Disord Transfus. 2015;6:318.
69. Júlio César Nepomuceno. Using the drosophila melanogaster to assessment carcinogenic agents through the test for detection of epithelial tumor clones (warts). Adv Tech Biol Med. 2015;3:149.
70. Beenish Naeem Awan, et al. Bacterial and liposomal vector guided drug delivery system via tumor markers carrier gene to treat neoplasm. J App Pharm. 2016;8:206.
71. Arlen M, et al. The nature and function of immunogenic tumor proteins that characterize pancreatic and colorectal cancer: a review. J Clin Cell Immunol. 2015;6:367.
72. Ayla Gunlemez, et al. Lacrimal sac mucocele in a newborn: a rare mimic of congenital intranasal tumor. J Clin Case Rep. 2015;5:600.
73. Tomoko Nakamura, et al. A placental site trophoblastic tumor complicated with arteriovenous malformation: a case report. J Clin Case Rep. 2015;5:596.
74. Serrano MJ, et al. Circulating tumor cells (CTCs): from detection to dissection. J Clin Diagn Res. 2014;2:120.
75. Rin Iraha, et al. Hepatic Epithelioid Hemangioendothelioma: Vascular penetration in the tumor as a characteristic imaging. finding. J Liver. 2015;4:184.
76. Michael J Strong, et al. brain tumors: epidemiology and current trends in treatment. J Brain Tumors Neurooncol. 2016;1:102.
77. Vladimir A Richter, et al. Antitumor potential of lactaptin. Biol Med (Aligarh). 2014;S2:004.
78. Macias N, et al. Gastric Desmoid Tumor: An infrequent case of intra-abdominal fibromatosis. J Gastrointest Dig Syst. 2016;5:332.
79. Mahsa Khayat-Khoei. Role of stem cells in the anti-tumor effects of the human amniotic membrane. J Cytol Histol. 2015;6:e117.
80. Johannes Routila and Jukka Westermarck. CIP2A as a potential stratification marker and target for tumor responsiveness to dna damaging therapies. J Mol Biomark Diagn. 2015;S2:014.
81. Laura Jimeno, et al. Role of systemic anti-tumor necrosis factor alpha treatment in the reduction of proliferative vitreoretinopathy. J Clin Exp Ophthalmol. 2015; 6:464.
82. Gravey F, et al. Role of stem cells in the anti-tumor effects of the human amniotic membrane. J Cytol Histol. 2011;5:246.
83. Lilia Bardoscia, et al. Pulmonary epithelioid hemangioendothelioma: advances in treatment options despite a rare vascular tumor. Chemotherapy (Los Angel). 2015;4:160.
84. Makiko Yasumizu, et al. Malignant skin tumors in patients with oculocutaneous albinism. Pigmentary Disorders 2015;2:218.
85. Mahendra Kumar Trivedi, et al. The potential impact of biofield treatment on human brain tumor cells: a time-lapse video microscopy. J Integr Oncol. 2015;4:141.

86. Jian-he ZHeng, et al. Defective expression of polarity protein par3 promotes cervical tumorigenesis and metastasis. gynecol obstet (sunnyvale). 2015;5:317.
87. Cristina del Valle Rubido, et al. Ovarian steroid cell tumor associated to endometrial hyperplasia and presenting as postmenopausal vaginal bleeding. Gynecol Obstet (Sunnyvale). 2015;5:316.
88. Shanmukha Ramya, et al. Primary intraosseous squamous cell carcinoma arising from keratocystic odontogenic tumor. J Nucl Med Radiat Ther. 2015;6:253.
89. Oleksandr H. Minchenko, et al. Minchenko. IRE-1alpha signaling as a key target for suppression of tumor growth. Single Cell Biol. 2015;4:118.
90. Aliese Sarkissian and James D Birmingham. Safety and efficacy of high-dose tumor necrosis factor (tnf) inhibitors in the management of pediatric inflammatory diseases. Rheumatology (Sunnyvale) 2015;1000156.
91. Rachel Yanget al. Expression of oncodrivers her-3 and c-met during breast tumorigenesis in brca mutation carriers. J Med Surg Pathol. 2016;1:120.
92. Kristopher JL Irizarry, et al. Leveraging naked mole rat (heterocephalus glaber) comparative genomics to identify canine genes modulating susceptibility to tumorigenesis and cancer phenotypes. J Veterinar Sci Technol. 2015;7:322.
93. Evita B Henderson-Jackson and Farah K Khalil. Pathology of metastatic tumors to bone: effects of decalcification as experienced at a single cancer center. Oncology and cancer case reports. 2016;2:112.
94. Samir Ghani, et al. Protocol for three dimensional histopathological examination of the vermiform appendix in neuroendocrine tumors. J Gastrointest Dig Syst 2016;6:397.
95. Nicoleta C. Arva. Diagnostic challenges in pediatric round blue cell tumors of the bone. Diagn Pathol Open. 2016;1:110.
96. Bacem AE Ottoman. Granular cell tumor of the tongue: a case report with emphasis on the diagnostic and therapeutic proceedings. Oncology and cancer case reports. 2015;1:106.
97. Li Yanan, et al. Improved antitumor effect of survivin responsive conditional replication adenovirus in combination with cisplatin in lung cancer. J Cancer Sci Ther. 2016;8.8:216.
98. Mark N Stein et al. Phase II study evaluating the effect of concomitant ramucirumab on the pharmacokinetics of docetaxel in patients with advanced solid tumors. Clin Pharmacol Biopharm. 2016;5:161.
99. Oral O, et al. Juvenile granulosa cell tumor - a rare neoplasm in newborns. J Carcinog Mutagen. 2016, 5:11
100. Issa C and Daher LA. Severe hyperparathyroidism with secondary osteitis fibrosa cystica and brown tumors mimicking bone metastasis. Endocrinol Metab Syndr. 2016;5:245.
101. Hentati Abdessalem, et al. Chest wall desmoid tumor mimicking a pancoastã¢â€â“tobias tumor. J Pulm Respir Med. 2016;6: 365.
102. Schreiter V, et al. Usefulness of ga-68 hbed-cc psma pet/ct for tumor staging in the initial diagnostic assessment of prostate cancer. J Nucl Med Radiat Ther. 2016;7:29.