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Scientists Grow New Potential Medication for Uncommon Leukemia

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Short Communication

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ABSTRACT

Another medication that shows potential in lab studies against an uncommon sort of intense leukemia has been produced by researchers. Furthermore, extra studies propose the same compound could assume a part in prostate growth treatment too, they say. In leukemia, unusual platelets are created in the bone marrow. As a rule, leukemia includes the generation of irregular white platelets - the cells in charge of battling contamination. On the other hand, the strange cells in leukemia don't work in the same route as ordinary white platelets. The leukemia cells keep on growing and partition, in the long run swarming out the typical platelets.

Introduction

Leukemia is a threat (disease) of platelets. In leukemia, unusual platelets are created in the bone marrow. As a rule, leukemia includes the generation of irregular white platelets - the cells in charge of battling contamination. On the other hand, the strange cells in leukemia don't work in the same route as ordinary white platelets. The leukemia cells keep on growing and partition, in the long run swarming out the typical platelets. The deciding result is that it gets to be troublesome for the body to battle contaminations, control dying, and transport oxygen. More than 50,000 instances of leukemia happen yearly in the U.S. There are distinctive sorts of leukemia, based upon how rapidly the ailment creates and the sort of strange cells delivered. Leukemia is called an intense leukemia in the event that it grows quickly [1-9]. Vast quantities of leukemia cells aggregate rapidly in the blood and bone marrow, prompting side effects, for example, tiredness, simple wounding, and defenselessness to diseases. Intense leukemia obliges quick and forceful treatment.

There are around 54,000 new instances of leukemia every year in the U.S. furthermore, around 24,000 passing's because of leukemia. Leukemia makes up around 3% of all new disease cases.

Incessant leukemia's grow gradually over the long run. These leukemia's may not bring about particular side effects toward the start of their course. In the event that left untreated, [10] the cells might in the end develop to high numbers, as in intense leukemia's bringing on comparative manifestations.

Leukemia's are further delegated myeloid or lymphoid, contingent on the sort of white platelet that makes up the leukemia cells. An essential comprehension of the ordinary advancement of platelets is expected to comprehend the diverse sorts of leukemia [11-18]. Ordinary platelets create from undeveloped cells that can possibly get to be numerous cell sorts. Myeloid immature microorganisms develop in the bone marrow and get to be juvenile white cells called myeloid impacts. These myeloid impacts further develop to wind up either red platelets, platelets, or certain sorts of white platelets. Lymphoid undifferentiated organisms develop in the bone marrow to end up lymphoid impacts. The

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lymphoid impacts form further into T or B lymphocytes, unique sorts of white platelets. Myeloid leukemia's are comprised of cells that emerge from myeloid cells, while lymphoid leukemia's emerge from lymphoid cells. Knowing the kind of cell included in leukemia is essential in picking the proper treatment [19-25].

Basic sorts of leukemia

The four most basic sorts of leukemia are intense lymphocytic leukemia, incessant lymphocytic leukemia, intense myeloid leukemia, and interminable myeloid leukemia.

Acute lymphocytic leukemia: (ALL, otherwise called intense lymphoblastic leukemia) is the most widely recognized kind of leukemia in kids, yet it can likewise influence grown-ups. In this sort of leukemia, juvenile lymphoid cells develop quickly in the blood. It influences more than 6,000 individuals for every year in the U.S.

Acute myeloid leukemia: (AML, likewise called intense myelogenous leukemia) includes the quick development of myeloid cells. It happens in both grown-ups and youngsters and influences around 18,000 individuals every year in the U.S.

Chronic lymphocytic leukemia: (CLL) is a moderate developing disease of lymphoid cells that as a rule influences individuals more than 55 years old. It is assessed to influence around 16,000 individuals in the U.S. consistently. It never happens in kids or young people.

Chronic myeloid leukemia: (CML, otherwise called unending myelogenous leukemia) essentially influences grown-ups and happens in around 6,000 individuals consistently in the U.S.

Signs & Symptoms:

The indications and indications of leukemia rely on the kind of leukemia. As expressed before, moderate developing or interminable leukemia may not create any indications at the beginning, while forceful or quickly developing leukemia may prompt serious side effects [26-30]. The indications of leukemia emerge from a loss of capacity of the typical platelets or from gathering of the strange cells in the body.

Signs and manifestations of leukemia ordinarily incorporate the accompanying:

1. Fevers
2. Night sweats
3. Swollen lymph hubs that are generally effortless
4. Sentiments of exhaustion, tiredness
5. Simple draining or wounding, creating pale blue or purplish fixes on the skin or small red spots on the skin, or repeating nosebleeds
6. Incessant diseases
7. Bone or joint agony
8. Weight reduction that is generally unexplained, or loss of hankering
9. Amplification of the spleen or liver, which can prompt stomach agony or swelling
10. Red spots on the skin.

In the event that leukemia cells have invaded the mind, side effects, for example, migraines, seizures, perplexity, loss of muscle control, and retching can happens [30-35]. At present, accessible treatments are just mostly successful in patients with leukemia and the dominant part of patients can't be cured with current treatment methodologies. Subsequently, there is an undeniable need to grow more particular and powerful medications for the treatment of these maladies. In the most recent a quarter century, advance in sub-atomic and cell science has brought about a superior portrayal and comprehension of the sub-atomic irregularities in intense and unending leukemia. These accomplishments have given new chances to the advancement of inventive medications, and leukemia death rates have started to decrease. Sub-atomic focused on medications were initially presented for the treatment of hematological malignancies. Imatinib was the first little molecule targeted medication effectively utilized for the treatment of perpetual myeloid leukemia (CML), and rituximab was the first

tumor-particular immunizer to be presented for the treatment of CD20-positive lymphomas and unending lymphocytic leukemia (CLL). The presentation of imatinib and rituximab has changed the death rates connected with CML and B-cell lymphoid malignancies. In CML the accessibility of tyrosine kinase inhibitors (TKIs) changed the administration and anticipation of this infection [36-40]. Be that as it may, around 20% of CML patients don't accomplish ideal reaction on imatinib treatment and they require option drugs. The accessibility of second era TKIs, for example, dasatinib and nilotinib, has given new remedial would like to patients with imatinib resistance [2]. What's more, new atoms with diverse methods of activity have exhibited action in patients with exceedingly safe mutants, for example, MK0457 and danusertib, which show inhibitory movement against Aurora kinases [3]. Current treatments are often wrong and unsuccessful for intense myeloid leukemia (AML), particularly for more established patients. For these patients, there is an undeniable need to grow better methodologies and new, more particular and dynamic medications. The quest for new medications in AML has prompted the improvement of numerous new antileukemic specialists conceivably dynamic in this type of leukemia [41-45]. Novel operators possibly valuable in the treatment of patients with AML incorporate monoclonal antibodies, atomic target drugs, fresher nucleoside analogs and different medications. These new operators appear to be unrealistic to be therapeudic when directed as monotherapy. They will rather must be utilized as a part of blend with other new operators or with more standard treatment. Nucleoside analogs are clinically imperative antileukemic medications which rival physiologic nucleosides and subsequently, communicate with a substantial number of intracellular targets. As of late, three novel nucleoside analogs, clofarabine, troxacitabine and sapacitabine, have been brought into clinical trials in AML and indicated guarantee [46-50]. Be that as it may, stage II and III randomized trials are important to check whether these medications present preference in the treatment of this malady. Gemtuzumab ozogamicin is a refined IgG4 hostile to CD33 monoclonal immunizer conjugated to calicheamicin, a powerful antitumour anti-microbial that had exhibited movement in intermittent and already untreated AML [51-55]]. This medication was utilized to treat AML from 2000-2010 however was withdrawn from business in June 2010 when a clinical trial demonstrated the medication expanded patient passing and included no advantage over customary leukemia treatments. Another counter acting agent with potential in the treatment of AML is lintuzumab. This is a refined monoclonal immune response coordinated against CD33. However the stage IIb trial of lintuzumab (SGN-33) in patients with AML did not meet the essential endpoint of amplifying general survival and its advancement project will be ceased by the maker (Seattle Genetics, Inc.). These samples demonstrate that the value of initially encouraging antileukemic medications is as often as possible not affirmed in all around composed clinical trials. As of late, FMSlike tyrosine kinase 3 (FLT3) inhibitors, lestaurinib, tandutinib and PKC 412 have been likewise created and tried in AML [56-60]. Preclinical perceptions and clinical studies show that FLT3 inhibitors are promising operators in the treatment of FLT3-transformed AML patients, particularly when utilized as a part of mix with chemotherapy. The most widely recognized grown-up leukemia in the Western world is unending lymphocytic leukemia (CLL). Late treatment of CLL is consolidated cytotoxic chemotherapy with monoclonal antibodies, which has altogether enhanced the nature of reaction, length of time of reaction, and survival [61-65]. Nonetheless, escalated treatment is frequently excessively poisonous, especially in more established patients. Accordingly, the improvement of novel treatment procedures is very attractive, particularly those with focused on activities and lower toxicities. As of late, a few new operators have been investigated and have demonstrated guarantee in CLL treatment including new mAbs and BCL-2 inhibitors, for example, oblimersen, obatoclax, and ABT-263 [66-70]. Moreover, protein kinase inhibitors, for example, flavopiridol, spleen tyrosine kinase inhibitors (Fostamatinib disodium), Bruton's tyrosine kinase (PCI-32765), and phosphatidylinositol 3-kinase inhibitors (CAL- 101) are exceedingly dynamic and very much endured in CLL patients, independent of high-hazard genomic anomalies and propose that these medications may be an imperative new focused on treatment approach for CLL. This is additionally an energizing time in medication advancement for intense lymphoblastic leukemia (ALL). As of late, three novel PNAs, clofarabine, nelarabine and forodesine (immucillin H, BCX-1777), have exhibited promising action in patients with backslid and headstrong ALL [71-75]. Another imperative pattern in ALL medication improvement is the expanding comprehension of the sub-atomic level of the genomic changes that happen in B- and T-cell ALL. Medications focusing on the particles significant to the science of ALL are in ahead of schedule clinical trials: inhibitors of FLT-3, BCR-ABL, mTOR, Bcl-2, ribonucleotide reductase, Aurora A kinase and the proteasome complex. The utilization of hostile to NOTCH1 treatments for T-ALL, incorporating blend treatments with molecularly-

focused on medications are likewise encouraging [76-80]. As of now accessible inhibitors of these objectives can possibly build treatment adequacy, and generally have non-covering toxicities with standard cytotoxic chemotherapy specialists. The improvement of new medications with novel components of activity stays basic to accomplishment in battling leukemias. It is normal that a superior comprehension of the sub-atomic pathogenesis of leukemias will add to the revelation and clinical use of novel medications that will reform restorative procedures and convey reestablished would like to leukemia patients. Novel treatments are being assessed both in preclinical studies and in ahead of schedule clinical trials. This is an energizing time for the advancement of new, compelling medications for the treatment of leukemia, which ought to fundamentally enhance the forecast of this as often as possible lethal sickness soon [81-85].

Complications:

A large portion of the difficulties of leukemia identify with the consumption of ordinary platelets and in addition the symptoms of medicines as portrayed in the past segment, for example, regular diseases, dying, and GVHD in beneficiaries of undeveloped cell transplants. Weight reduction and frailty are further confusions of leukemia and its treatment. Intricacies of any leukemia additionally incorporate a backslide or a movement of the sickness after an abatement has been accomplished with treatment. Different difficulties of leukemia identify with the particular sort of leukemia. For instance, in 3%-5% of instances of CLL, the cells change qualities and change into a forceful lymphoma. This is known as a Richter change. Immune system hemolytic pale, in the body assaults and annihilates red platelets, is another potential confusion of CLL. Individuals with CLL are additionally more inclined to grow second diseases and other blood issue and blood growths [86-90].

Tumor lysis disorder is a condition brought on by the fast demise of malignancy cells when treated. It can happen in any kind of tumor, and it is seen with a few instances of leukemia, especially when huge quantities of leukemia cells are available, for example, with AML or ALL. The quick annihilation of the leukemia cells prompts the arrival of a lot of phosphate, which further causes metabolic variations from the norm and can prompt kidney disappointment.

Youngsters who get treatment for ALL may encounter late unfriendly impacts including focal sensory system (CNS) debilitation, moderating of development, barrenness, waterfalls, and an expanded danger for different malignancies. The occurrence of these late impacts fluctuates relying on the age at treatment and the sort and quality of treatments [91-95].

New potential medication

The compound was produced in the labs who have been laboring for quite a while to recognize a little particle inhibitor that would hinder the association between the protein menin and MLL combination proteins that cause an uncommon sort of intense leukemia. Purported MLL combination leukemia can happen in both grown-ups and youngsters. It speaks to up to 10 percent of intense leukemia in grown-ups, and around 70 percent of intense leukemia in newborn children. Current medications are not extremely powerful, with a little more than 33% of patients surviving five years.

Protein-protein cooperations, for example, the menin-MLL combination protein communications in leukemia are by and large thought to be "undruggable," importance it can be especially difficult to create drugs that objective those connections. Regardless of the trouble, Grembecka says that the MLL-menin collaboration stayed enticing.

"In numerous sorts of tumor, you see different cooperations and transformations that trigger the illness. The MLL-menin affiliation is a better than average solution target in light of the fact that its the essential driver in this kind of leukemia. By obstructing this collaboration, its prone to stop the growth"[96-100].

In a study distributed in Cancer Cell, the analysts tried two mixes they created, MI-463 and MI-503, in cell lines and in mice with MLL leukemia. They discovered the mixes obstructed the MLL-menin connection without hurting typical platelets. The mixes were conveyed into the blood and metabolized at a decent rate, both of which are key issues in growing new medications. The analysts had already tried a before adaptation of the compound, which indicated guarantee. Here, they generously enhanced the drug's intensity and a number of its pharmacologic properties, making it additionally charming for potential use in individuals.

"Despite seemingly unconquerable resistance, we decided to examine making sense of how to piece the MLL-menin cooperation with little molecules. From nothing, we have had the limit to recognize and incredibly enhance a compound and demonstrate that it's got significant potential in blocking MLL combination leukemia in creature models," says Cierpicki, colleague educator of pathology at the U-M Medical School.

Then, prostate tumor scientists at U-M found that menin and MLL assume a part in androgen receptor flagging, which is a key driver of prostate ailment. In a study dispersed in Nature Medicine, the scientists tried the same MLL-menin inhibitors against maiming safe prostate disease cells and mice models [101,102].

Conclusion

"Our study proposes that this MLL-menin inhibitor may likewise have a potential part in a more normal strong tumor, for this situation prostate disease," The mixes must be tried further in the research facility before any clinical trials could be considered. Grembecka and Cierpicki's labs are taking a gander at further refinements and more propelled testing of their inhibitors. Chinnaiyan's group will keep on researching the part of MLL in mutilation safe prostate tumor.

No medications or trials are as of now accessible utilizing a MLL-menin inhibitor.

REFERENCES

1. Gora-Tybor J, Robak T (2008) Targeted drugs in chronic myeloid leukemia. Curr Med Chem 15: 3036-3051.
2. Eiring AM, Khorashad JS, Morley K, Deininger MW (2011) Advances in the treatment of chronic Myeloid Leukemia. BMC Med 9: 99.
3. Okabe S, Tauchi T, Ohyashiki JH, Ohyashiki K (2009) Mechanism of MK-0457 efficacy against BCR-ABL positive leukemia cells. Biochem Biophys Res Commun 380: 775-779.
4. Robak T, Szmigielska-Kaplon A, Pluta A, Grzybowska-Izydorczyk O, Wolska A, et al. (2011) Novel and emerging drugs for acute myeloid leukemia: pharmacology and therapeutic activity. Curr Med Chem 18: 638-666.
5. Estey E (2008) New drugs in acute myeloid leukemia. Semin Oncol 35: 439-448.
6. Lee J, Paek SM, Han SY (2011) FMS-like tyrosine kinase 3 inhibitors: a patent review. Expert Opin Ther Pat 21: 483-503.
7. Hallek M, Fischer K, Fingerle-Rowson G, Fink AM, Busch R, et al. (2010) Addition of rituximab to fludarabine and cyclophosphamide in patients with chronic lymphocytic leukaemia: a randomised, open-label, phase 3 trial. Lancet 376: 1164-1174.
8. Lin TS. (2010) New agents in chronic lymphocytic leukemia. Curr Hematol Malig Rep 5: 29-34.
9. Robak T (2011) New nucleoside analogs for patients with hematological malignancies. Expert Opin Investig Drugs 20: 343-359.
10. Horton TM, Sposto R, Brown P, Reynolds CP, Hunger SP, et al. (2010) Toxicity assessment of molecularly targeted drugs incorporated into multiagent chemotherapy regimens for pediatric acute lymphocytic leukemia (ALL): review from an international consensus conference. Pediatr Blood Cancer 54: 872-878.
11. Madhu Dhar, Lisa Amelse, Nancy Neilsen, Pelagie Favi and Jessica CarterArnold, (2015) Platelet-Rich Plasma Enhances the Cellular Function of Equine Bone Marrow-Derived Mesenchymal Stem Cells. J Stem Cell Res Ther 2015, 5:278
12. Nanditha S, Senthilkumar Muthusamy, Balamanikandasrinivasan Chandrasekaran and Sathya Kannan, (2015) A Judicious Treatment Approach for the Management of Localized Aggressive Periodontitis: A Case Report. J Interdiscipl Med Dent Sci

13. Tasnim Ahsan, Rukhshanda Jabeen, Urooj Lal Rehman, Zeenat Banu and Samar Abbas Jaffri, (2015) Platelet Transfusion; What and When to Transfuse, a Dilemma of Clinical Practice. Intern Med 2015, 5: 181
14. Madhavan Nair, Maria Jose MB, Marisela Agudelo, Adriana Yndart and Mayra E VargasRivera, (2015) Platelets Contribute to BBB Disruption Induced by HIV and Alcohol. J Alcohol Drug Depend 2014, 3:182
15. Claus Vinter B Hviid, Are Hugo Pripp, Ansgar O Aasen and Claus DanckertKrohn, (2014) Postoperative Accumulation of Cyr61/CCN1 in Surgical Wound Fluid Precedes Cytokine Activation and is Disparate from Systemic Alterations. J Infect Dis Ther 2014, 2:181
16. ChidambharamChoccalingam, Premila Samuel, and KidevSwamynathan, (2013) Harris Platelet Syndrome: The Need to Recognise the Entity.. Medical and Health Sciences doi:
17. James P Maloney, Daniel R Ambruso, Norbert F Voelkel and Christopher C Silliman, (2014) Platelet Vascular Endothelial Growth Factor is a Potential Mediator of Transfusion-Related Acute Lung Injury. J Pulm Respir Med 2014, 4: 212
18. Takehisa Iwai, Makoto Umeda, Yoshinori Inoue and Tokyo Medical and Dental University Buerger Disease Research Group, (2014) Pathogenic Mechanism of the Artery and the Vein in Buerger Disease: Our Hypothesis. Angiol 2014, 2:131
19. Vui Heng Chong, Norwani Basir, Aziman Bin Yaakub, (2010) Acute Pancreatitis Complicated by Jejunal Hematoma in a Patient on Anti-Coagulants and Anti-Platelets. JOP. J Pancreas 2010, 11
20. Yancy FerrerAcosta, Marieli Gonzaacutelez, Moacutenica Fernaacutendez and Valance Washington A, (2014) Emerging Roles for Platelets in Inflammation and Disease. J Infect Dis Ther 2014, 2:149
21. Durowoju MO, Babatunde IA, Raheem WA and Ajala MT, (2014) Effect of Antimony on the Tensile Strength and the Morphology of Si Platelets in Recycled Aluminum Piston Alloys. J Material Sci Eng 2013, 3: 137
22. Timori NH and Badlou BA, (2014) The Effect of (Non-) Agitating Condition on Agonist Induced-Aggregation of the 48 hours-Stored Platelet Concentrates. J Blood Disord Transfus 2014, 5: 219
23. Elena Volokhina, Arjen Jakobi, Rolf Urbanus, Eric Huizinga, Henk Sluiter, et. al. (2014) Novel Sequence Variation Affects GPIbα in Post-diarrheal Hemolytic Uremic Syndrome. J Nephrol Ther 2013, S11-007
24. A Simple Method to Quantify Glycogen from Human Platelets. J Cytol Histol 2014, 5: 217
25. Metabolic and Inflammatory Proteins Differentially Expressed in Platelets from Unprovoked Deep Vein Thrombosis Patients. J Proteomics Bioinform Vol.7.1 017-022 (2014)
26. Vivian WY Lee, (2013) Pharmacogenomic Testing of Anti-Platelets Drugs and its Cost-Effectiveness. J Clinic Toxicol 2013 3: e120
27. JihJin Tsai, PoChih Chen, LiTeh Liu, Ko Chang, JuHan Yao, et. al. (2013) Pathogenic Parameters Derived from Activated Platelets in Dengue Patients. Trop Med Surg
28. Felix Kh Kamilov, Galiya A Timirkhanova, Aleksandr V Samorodov, Ferkat A Khaliullin, (2013) Choosing potential dissolution medium to study the influence of water-insoluble substances on aggregation of platelets within preclinical studies under conditions in vitro. Biology and medicine
29. PeiJu Sung, Sayandip Mukherjee, Michael P Blundell and Adrian J Thrasher, (2013) Feeder-Free Derivation of Functional Platelets from Human Induced Pluripotent Stem Cells. J Blood Disord Transfus 2013, 4: 153
30. Dermot Cox, Maria Salvato and Juan Carlos Zapata, (2013) The Role of Platelets in Viral Hemorrhagic Fevers. J Bioterror Biodef 2013, S12:003
31. Susanne Maria Picker, (2012) Pathogen Reduction Technologies: The Best Solution for Safer Blood?. J Blood Disord Transfus 2012, 3: 133

32. Olivier Garraud, Patricia Chavarin, Patrick Fabrigli, Hind HamzehCognasse and Fabrice Cognasse, (2011) Novel Functions of Therapeutic Platelets as “Immune Cells”. J Blood Disord Transfus 2011, S1-005
33. ElSayed Emara and Khaled A. AbdelSater, (2011) Beneficial Effects of Calcium Channel Blocker “Nifedipine” on Abnormalities of Platelets and Lipid Metabolism in Patients with Type II Diabetes Mellitus. J Diabetes Metab 2011, 2:131
34. Jorge Olmos, Rociacuteo Goacutemez and Viviana P. Rubio, (2015) Apoptosis Comparison Effects Between Synthetic and Natural B-Carotene from Dunaliella salina on MDA-MB-231Brest Cancer Cells. J Microb Biochem Technol 7: 051-056
35. Jason B Garrison, Chunmin Ge, Lixiao Che, Derek A Pullum, Guang Peng, et. al. (2015) Knockdown of the Inhibitor of Apoptosis BRUCE Sensitizes Resistant Breast Cancer Cells to Chemotherapeutic Agents. J Cancer Sci Ther/Vol.7.4 121-126
36. Haruhiko Yoshioka, Keita Hoshiai, Toshiya Nakamura, Kayo Horie, Kiyotada Washiya and Jun Watanabe, et. al. (2015) Usefulness of Evaluation of Nuclear Color by Visible-Microscopic Spectroscopy for Objective Differentiation between Non-Cancer and Cancer Cells Prepared Using Liquid-Based Cytology. J Cytol Histol 2015, 6: 308
37. ChingYi Wu, ChiaHua Lin, YuChie Chen, (2015) Using Glucose-bound Fe3O4 Magnetic Nanoparticles as Photothermal Agents for Targeted Hyperthermia of Cancer Cells. J Nanomed Nanotechnol 2015, 5:264
38. Subburayan Karthikeyan, Sugeerappa Laxmanappa Hoti and Nagarajan Rajendra Prasad, (2014) Resveratrol Modulates Expression of ABC Transporters in Non-Small Lung Cancer Cells: Molecular Docking and Gene Expression Studies. J Cancer Sci Ther/Vol.6.12 497-504
39. BL Milner, CB Penny, VE Gibbon, P Kay and P Ruff, (2015) CD133/EpCAM Cancer Stem Cell Markers of Tumour Stage in Colorectal Cancer Cells. J Tissue Sci Eng 2015, 6:143
40. Sumantha Malur Gopalakrishna, Girisha Sirangala Thimappa, Ramesh Puttalingaiah Thylur, Yogisha Shivanna, and Anand Sreenivasan, et. al. (2014) In- vitro Anti-Cancer Screening of Solanumindicum Rhus succedanea, Rheum emodiand Gardenia gummifera Medicinal Plants in Cancer Cells.. PHARMACY AND PHARMACEUTICAL SCIENCES
41. Mehdi Shahbazi, G Hussian Erjaee, and Hoda Erjaee, (2014) Dynamical Analysis of Chemotherapy Optimal Control using Mathematical Model Presented by Fractional Differential Equations, Describing Effector Immune and Cancer Cells Interactions. Journal of Pharmacy and Pharmaceutical Sciences
42. MiHeon Lee, Puja Kachroo, Paul C Pagano, Jane Yanagawa, Gerald Wang, et. al. (2014) Combination Treatment with Apricoxib and IL-27 Enhances Inhibition of Epithelial-Mesenchymal Transition in Human Lung Cancer Cells through a STAT1 Dominant Pathway. J Cancer Sci Ther/Vol.6.11 468-477
43. AnnaLena Gratzke, Kerstin Reimers, Peter M Vogt and Vesna Bucan, (2014) Sensitising Breast Cancer Cells to Chemotherapy by Down Regulation of Lifeguard. J Cancer Sci Ther/Vol.6.10 411-416
44. divWeiguo Hu, Chao Li, Jing Sun, Bo Feng, Daohai Zhang, et. al. (2014) Cancer-Associated-Fibroblast Induces Epithelial-Mesenchymal Transition of Gastric Cancer Cells via Activating Thy-1. J Carcinog Mutagen 2014, 5: 190
45. Takanobu Nakamura, Yasuhiro Suzuki, Yoshifumi Takahashi, Susumu Satomi and Yasufumi Sato, (2014) Paradoxical Augmentation of Tumor Angiogenesis Combined with Down-Regulation of IP-10 after Adenovirus-Mediated Transfer of Vasohibin-1 Gene in Cancer Cells. J Cancer Sci Ther/Vol.6.8 289-297

46. Robert J Walter, Bashar M Attar, Asad Rafiq, Sooraj Tejaswi, Megan Delimata, et. al. (2012) Newcastle Disease Virus LaSota Strain Kills Human Pancreatic Cancer Cells in Vitro with High Selectivity. JOP. J Pancreas 2012, 13
47. Hiroki Takahashi, Hirozumi Sawai, Hitoshi Funahashi, Yoichi Matsuo, Akira Yasuda, et. al. (2007) Antiproteases in Preventing the Invasive Potential of Pancreatic Cancer Cells. JOP. J Pancreas 2007, 8
48. Yasutake Uchima, Tetsuji Sawada, Kosei Hirakawa, (2007) Action of Antiproteases on Pancreatic Cancer Cells. JOP. J Pancreas 2007, 8
49. Xiaomei Ren, Zhang Zhang, Lei Duan, and Ke Ding, (2014) Identification of Niclosamide as a New Lead Compound to Suppress the Metastasis of Prostate Cancer Cells. Med chem 2014, 4: 511
50. ChungPing Hsu, YouLin Wu, WanYun Lee, LiWen Li and JingJenn Lin, (2014) Effects of Targeted Anticancer Medicines on Post-Cell Removal Surface Morphology of Cancer Cells Cultivated on 3-Aminopropyltriethoxysilane Surface. Med chem 2014, S1-007
51. Brian W. Booth, Sonia M Rosenfield and Gilbert H Smith, (2014) Reprogramming Cancer Cell In Vivo. J Stem Cell Res Ther 2014, 4:211
52. nbspDorrah Deeb, Chris Brigolin, Xiaohua Gao, Yongbo Liu, Kirit R. Pindolia and Subhash C. Gautam, et. al. (2014) Induction of Apoptosis in Pancreatic Cancer Cells by CDDO-Me Involves Repression of Telomerase through Epigenetic Pathways. J Carcinog Mutagen 2014, 5: 177
53. Gini C Kuriakose, Satpal Singh, Pradumn K Rajvanshi, William R Surin and Jayabaskaran C, (2014) In Vitro Cytotoxicity and Apoptosis Induction in Human Cancer Cells by Culture Extract of an Endophytic Fusarium solani Strain Isolated from Datura metel L. Pharm Anal Acta 2014, 5: 293
54. Gregory Lee, ChengYuan Huang, Yiting Tang and Hao Zhang, (2014) Potential Roles of Cancerous Immunoglobulins in the Immunology of Cancer Cells. J Clin Cell Immunol 2014, 5: 200
55. Lia Hafiyani, Satoru Yokoyama, Sherif Abdelhamed, Yoshihiro Hayakawa and Ikuo Saiki, (2014) Bufadienolides Overcome TRAIL Resistance in Breast Cancer Cells via JAK-STAT Pathway. Altern Integ Med 2014, 3:154
56. Gregory Lee, ChengYuan Huang, Hao Zhang and Yiting Tang, (2014) The Relationships between Toll-like Receptors and RP215-associated Immunoglobulins Expressed by Cancer Cells. J Cancer Sci Ther/Vol.6.3 077-080
57. Celestial T. Yap, (2014) Evasive Mechanisms in Cancer Cells Pose Challenges to Effective Therapy and Prevention of Cancer Recurrence. Clin Exp Pharmacol 2014, 4: e129
58. Rahamata AliBoina, Marion Cortier, Nathalie Decologne, Cindy RacoeurGodard, Ceacutedric Seignez, et. al. (2014) Activation of Akt by the Mammalian Target of Rapamycin Complex 2 Renders Colon Cancer Cells Sensitive to Apoptosis Induced by Nitric Oxide and Akt Inhibitor. J Carcinog Mutagen 2014, S8-004
59. Wei Li and Bob M Moore II, (2014) The Effect of Arvanil on Prostate Cancer Cells Studied by Whole Cell High Resolution Magic Angle Spinning NMR. Mod Chem Appl 2014, 2: 119
60. Agnieszka J Sok, Agnieszka Gizak, Piotr Mamczur, Aleksandra Piotrowska, Agata Knapik, et. al. (2014) Demethylation with 5-Aza-2′-deoxycytidine Affects Oxidative Metabolism in Human and Mouse Non-small Cell Lung Cancer Cells. J Cancer Sci Ther/Vol.6.2 036-044(2014)
61. Oleksyszyn Jozef, Joanna Wietrzyk and Mateusz Psurski, (2014) Cancer – Could it be Cured? A Spontaneous Regression of Cancer, Cancer Energy Metabolism, Hyperglycemia-Hypoglycemia, Metformin, Warburg and Crabtree Effects and a New Perspective in Cancer Treatment. J Cancer Sci Ther/Vol.6.3 056-061

62. Bipasha Bose and Sudheer Shenoy P, (2014) Stem Cell versus Cancer and Cancer Stem Cell: Intricate Balance Decides Their Respective Usefulness or Harmfulness in the Biological System. J Stem Cell Res Ther 2014, 4:173
63. Carl Parson, Valerie Smith, Christopher Krauss, Hirendra N Banerjee, Christopher Reilly, et. al. (2014) The Effect of Novel Rhenium Compounds on Lymphosarcoma, PC-3 Prostate and Myeloid Leukemia Cancer Cell Lines and an Investigation on the DNA Binding Properties of One of these Compounds through Electronic Spectroscopy. J Bioprocess Biotech 2013, 4:141
64. Daryoush ShahbaziGahrouei, Zeinab Ghasemian, Mohammad Abdolahi, Sohrab Manouchehri, Shaghayegh haghjooy Javanmard and Nasim Dana, et. al. (2013) In vitro Evaluation of Cobalt-Zinc Ferrite Nanoparticles Coated with DMSA on Human Prostate Cancer Cells. J Mol Biomark Diagn 2013, 4: 154
65. Marek Malecki, Jessica Dahlke, Melissa Haig, Lynn Wohlwend and Raf Malecki, (2013) Eradication of Human Ovarian Cancer Cells by Transgenic Expression of Recombinant DNASE1, DNASE1L3, DNASE2, and DFFB Controlled by EGFR Promoter: Novel Strategy for Targeted Therapy of Cancer§. J Genet Syndr Gene Ther 2013, 4: 152
66. Marek Malecki, Jessica Dahlke, Melissa Haig, Lynn Wohlwend and Raf Malecki, (2013) Eradication of Human Ovarian Cancer Cells by Transgenic Expression of Recombinant DNASE1, DNASE1L3, DNASE2, and DFFB Controlled by EGFR Promoter: Novel Strategy for Targeted Therapy of Cancer§. J Genet Syndr Gene Ther 2013, 4: 152
67. Yong Bo Liu, Xiaohua Gao, Dorrah Deeb, Ali S Arbab and Subhash C Gautam, (2013) Pristimerin Induces Apoptosis in Prostate Cancer Cells by Downregulating Bcl-2 through ROS-dependent Ubiquitin-proteasomal Degradation Pathway. J Carcinog Mutagen 2013, S6- 005
68. Karen McGuire, James Jamison, Jacques Gilloteaux and Jack L. Summers, (2013) Vitamin C and K3 Combination Causes Enhanced Anticancer Activity against RT-4 Bladder Cancer Cells. J Cancer Sci Ther/Vol.5.10 325-333
69. Satyajit Patra, Roshan Mascarenhas, Naseer Maliyakkal and Jesi Mathew Aranjani, (2013) Protocatechualdehyde Induces Apoptosis In Human Non-Small-Cell Lung Cancer Cells By Up Regulation Of Growth Arrest And DNA Damage-Inducible (GADD) Genes. Molecular Biology 2013, 2: 113
70. Hui Li, (2013) RNA Trans-Splicing in Normal and Cancer Cells. J Biomol Res Ther 2013, 2: e112 doi: 10.4172/2167-7956.1000e112
71. M Ahmed, K Jamil, (2011) Cytotoxicity of neoplastic drugs Gefitinib, Cisplatin, 5-FU, Gemcitabine, and Vinorelbine on human cervical cancer cells (HeLa). Biology and medicine
72. Mehdi SJ, Ahmad A, Irshad M, Manzoor N, Rizvi MMA, et. al. (2011) Cytotoxic effect of Carvacrol on human cervical cancer cells. Biology and medicine
73. Yiting Tang, Hao Zhang and Gregory Lee, (2013) Similar Gene Regulation Patterns for Growth Inhibition of Cancer Cells by RP215 or Anti-Antigen Receptors. J Cancer Sci Ther/Vol.5.5 200-208
74. Raquel C Montenegro, Rommel R Burbano, Milton N da Silva, Telma G Lemos and Marne C Vasconcellos, (2013) Biflorin, A Naphthoquinone, Inhibitsegfr in Breast Cancer Cells. Med chem 2013, 3: 179
75. Jayaprakash Periasamy, Muthulakshmi Muthuswami, Vignesh Ramesh, Thangaselvam Muthusamy, Amrita Jain, et. al. (2013) Nimesulide and Celecoxib Inhibits Multiple Oncogenic Pathways in Gastric Cancer Cells. J Cancer Sci Ther/Vol.5.4 126-136 (2013)
76. Sachin M Eligar, Radha Pujari, Mohammed Azharuddin Savanur, Nagaraja N Nagre, Srikanth Barkeer, et. al. (2013) Rhizoctonia Bataticola Lectin (RBL) Induces Apoptosis in Human Ovarian Cancer PA-1 Cells and Suppresses Tumor Growth In Vivo. J Glycobiol 2013, S1-001

77. Fatiha El Babili, Jaloul Bouajila, Alex Valentin and Christian Chatelain, (2013) Lawsonia Inermis: Its Anatomy and its Antimalarial, Antioxidant and Human Breast Cancer Cells MCF7 Activities. Pharmaceut Anal Acta 2013; 4: 203
78. Hyunshun Shin, Heather Whitehead, Xian Zhou, Karl L Banta, Juliet V Spencer, et. al. (2013) Synthesis and Evaluation of Ornithine Decarboxylase Inhibitors with Oxime Moiety and MCF-7 Breast Cancer Cells. Biochem & Pharmacol 2013, 2: 111
79. Joseph Molnar, Ilona Mucsi, Helga Engi, Gabriela Spengler, Leonard Amaral, et. al. (2013) The Role of Stroma in Tumour-Host Co-Existence: Some Perspectives in Stroma-Targeted Therapy of Cancer. Biochem & Pharmacol 2013, 2: 107
80. Muthana Ibrahim Maleek, (2013) Omega-3 Fatty Acids Decrease the Proliferation of Rhabdomyosarcoma (RD) and Vero Cell Lines. J Cancer Sci Ther/Vol.5.2 085-088 (2013)
81. QingYi Lu, Lifeng Zhang, Aurelia Lugea, Aune Moro, Mouad Edderkaoui, et. al. (2013) Determination of Rottlerin, a Natural Protein Kinases C Inhibitor, in Pancreatic Cancer Cells and Mouse Xenografts by RP-HPLC Method. J Chromat Separation Techniq 2012, 4: 162
82. Antonia Bellizzi and Stefania Tommasi, (2013) Cancer Cells with Stem Cell-Like Phenotypes and Liver Metastasis from Colon Carcinoma. J Liver 2013, 2: 114
83. R Vidhyalakshmi and C Vallinachiyar, (2013) Apoptosis of Human Breast Cancer Cells (MCF-7) Induced by Polysacccharides Produced by Bacteria. J Cancer Sci Ther/Vol.5.2 031-034
84. Bo Su, Shu Lin Xiang, Jian Su, Hai Ling Tang, Qiang Jin Liao, et. al. (2012) Diallyl Disulfide Increases Histone Acetylation and P21WAF1 Expression in Human Gastric Cancer Cells In vivo and In vitro. Biochem & Pharmacol 2012, 1: 106
85. Thomas Efferth, (2012) Autophagy by Natural Products in Cancer Cells. Biochem Anal Biochem 2012, 1: e128
86. Crispin R. Dass, (2013) Metabolic Shift in Cancer Cells Treated with Chemotherapy Autophagy and Cancer Chemoresistance. Metabolomics 2013, 3:e123
87. Amit K. Maiti, (2012) Elevate the ROS Level to Kill Cancer Cells during Chemotherapy. Chemotherapy 2012, 1: e119
88. Ying Ding and Thu Annelise Nguyen, (2012) Gap Junction Enhancer Potentiates Cytotoxicity of Cisplatin in Breast Cancer Cells. J Cancer Sci Ther/Vol.4.11 371-378
89. Shebl RI, Mohamed AF, Ali AE and Amin MA, (2012) Cerastes cerastes and Vipera lebetina Snake Venoms Apoptotic – Stimulating Activity to Human Breast Cancer Cells and Related Gene Modulation. J Cancer Sci Ther/Vol.4.10 317-323
90. Pranati Sar, Dharmendra K Bhargava, Debomita Sengupta, Bandita Rath, Sanjib Chaudhary and Sandip K Mishra, et. al. (2012) In Human Breast Cancer Cells TRβ Competes with ERα for Altering BCl2/Bax Ratio through SMP30-Mediated p53 Induction. J Cancer Sci Ther/Vol.4.8 227-234
91. Kanagaraj Palaniyandi, Barbara A Pockaj, Sandra J Gendler and XiuBao Chang, (2012) Human Breast Cancer Stem Cells Have Significantly Higher Rate of Clathrin-Independent and Caveolin-Independent Endocytosis than the Differentiated Breast Cancer Cells. J Cancer Sci Ther/Vol.4.7 214-222
92. JuHye Lee, YeonKyong Lee, JaeYong Kim, JaeSeoun Hur, MiKyong Lee and KwonIl Seo, et. al. (2012) Lethariella zahlbruckneri Acetone Extract-Induced Apoptosis of MCF-7 Human Breast Cancer Cells Involves Caspase Cascade and Mitochondria- Mediated Death Signaling. J Nutr Food Sci 2011,S2-004
93. Castro J, Ericsson C, Cashin P, Mahteme H, (2012) Preliminary Finding: Detection of Circulating Cancer Cells in Blood from a Patient with Peritoneal Carcinomatosis Treated with Cytoreductive Surgery and Intraperitoneal Chemotherapy. Surgery Curr Res 2012, 2: 113

94. Zaklina Kovacevic and Des R. Richardson, (2012) Targeting Iron in Cancer Cells: A New Strategy to Inhibit Metastatic Progression. Vitamin Trace Element 2012, 1:e114
95. A. Ivana Scovassi, (2012) Defective Apoptosis and Efficient Autophagy: Two Ways to Protect Cancer Cells from Death. Biochem & Pharmacol 2012, 1: e114
96. Noriaki Sunaga, Katsuya Hiraishi, Tamotsu Ishizuka, Kyoichi Kaira, Yasuki Iwasaki, et. al. (2012) MK615, A Compound Extract from the Japanese Apricot "Prunus mume" Inhibits In vitro Cell Growth and Interleukin-8 Expression in Non-small Cell Lung Cancer Cells. J Cancer Sci Ther 2012, S11-002
97. Zhao Liu, Abhik Bandyopadhyay, Robert W. Nichols, Long Wang, Andrew P. Hinck, et. al. (2012) Blockade of Autocrine TGF-β Signaling Inhibits Stem Cell Phenotype, Survival, and Metastasis of Murine Breast Cancer Cells. J Stem Cell Res Ther 2012, 2:116
98. Roza Zandi, Kai Xu, Hans S. Poulsen, Jack A. Roth and Lin Ji, (2011) Overexpression of the Novel Tumor Suppressor Gene FUS1 Suppresses the Growth of Small Cell Lung Cancer Cells. J Clinic Experiment Pathol 2012, S5: 001
99. Marvin Rubenstein, Courtney MP Hollowell and Patrick Guinan, (2011) Enhanced Delivery of Chemotherapeutic Alkylating Agents into Prostate Cancer Cells Employing the Androgen Receptor as Delivery Vehicle. Metabolomics 2011, 1:103
100. ChangZheng Sun, CuiTao Lu, YingZheng Zhao, Ping Guo, JiLai Tian, et. al. (2011) Characterization of the Doxorubicin-Pluronic F68 Conjugate Micelles and Their Effect on Doxorubicin Resistant Human Erythroleukemic Cancer Cells. J Nanomed Nanotechnol 2011, 2:114
101. Ratana Lim, Martha Lappas, Nuzhat Ahmed, Niels Borregaard, Michael A. Quinn and Gregory E. Rice, et. al. (2011) Effect of Silencing Neutrophil Gelatinase-Associated Lipocalin in Ovarian Cancer Cells on Epithelio-Mesenchymal Transition. J Mol Biomark Diagn 2011, 2: 114
102. GuangRong Yan, NanPeng Chen, YaDong Huang, Wen Ding, GuiWei He, et. al. (2010) Signaling Networks in Gastric Cancer Cells Revealed by Phosphoproteomics. J Proteomics Bioinform Vol.3.4 113-120