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Hans Kollberg, J Transm Dis Immun 2018, Volume 2
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MORE THAN 22 YEARS OF CLINICAL STUDIES ON ANTI-PSEUDOMONAS IGY TO CYSTIC FIBROSIS PATIENTS

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Objectives: Clinical studies have been running in Sweden and in Europe for 22 years with CF-patients on Anti-pseudomonas IgY (Anti-PA IgY) to prevent infections with *Pseudomonas aeruginosa* (*PA*) to find out efficacy and adverse events. The promising results gave Anti-PA IgY an Orphan Drug Designation in 2016.

Studies: Phase II study was conducted during 1995 -2002: Two groups with intermittently PA-infected patients: one group got Anti-PA IgY, the other group was without IgY. Microbiologists did not know from which group the analyses came from. A prolonged study in Sweden continued 2002-2011. Pregnancy: Two CF women, whereof one twice, were on Anti-PA IgY during pregnancies. Transplanted: One boy transplanted 12 years ago due to infections w. *PA* and Atyp.Mycobact. Phase III study 2002 -2017: A multicenter study from nine European countries.

Results: Phase II: Group with Anti-PA IgY: 2.35 positive *PA* cultures/100 months; Untreated group: 7 positive *PA*/100 months. The duration from first to second colonization with PA was significantly prolonged for the treated versus the control group (Kaplan-Meier p=0.015). The time from first *PA* infection until chronic infection occurred was prolonged in the Anti-PA IgY treated group. The time until *PA* was transformed to the severe mucoid form was prolonged. Lung function and BMI were well preserved. Prolonged group: similar effects as those in the first study. Three pregnancies have been carried out well and gave birth to three healthy babies. Transplanted pat.: No new pseudomonas or atypical mycobacterium after transplantation. The few infections in the treated group minimized the need for antibiotics. Phase III: The study was finished in June 2017. Totally 144 countable patients had been included. The results will be ready in spring 2018. All patients have gargled more than 250.000 times and no adverse events have been reported.

Discussion: Anti-PA IgY has shown good results both in efficiency and absence of adverse events. It reduces the use of antibiotics and thus also the risk of resistant bacteria. Gargling is convenient to use. Treatment is cost effective. Cost for Anti-PA IgY is much less than the costs for antibiotics. The costs for days of illness and for hospitalization will be much lower.

Conclusion: Hopefully the now running double-blind, randomized phase III study will give results as expected and Anti-PA IgY might be registered and physicians will be able to give anti-PA IgY to all eligible CF patients.

Biography

Hans Kollberg is Professor emeritus, Pediatrics, Children´s University Hospital, Uppsala. He has a Specialization in Pediatrics from Swedish Medical Board 1966. He holds a Medical Doctors Degree (MD) in Pediatrics from Uppsala University, Sweden 1961. He started his career as Staff physician (1959-1966), Resident Good Samaritan Hospital, Phoenix, Arizona (1966-1967). He extended his service as a Director of the CF Center, University Hospital, Uppsala and Umea in 1968-1982 and 1985-1999. He was professor at the University of Kuwait 1982-1985. He has been a recipient of many awards and grants. He is the Founder of the Swedish Cystic Fibrosis Association, 1969. His research experience includes various programs, contributions and participation in different countries for diverse fields of study. His research interests as a Research Scholar reflect in his wide range of publications in various national and international journals.

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ANOPHELES...THE ANIHALATOR

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Today topic is Malaria, one of the deadliest infections in Sub-Saharan Africa causing more deaths per day than any other outbreak in recent years. Malaria accounts for one of the highest Mortality and Morbidity rates in the world amongst Children esp. in poverty stricken countries with poor socio-economic status with malnutrition and immunosuppression. It also has a very high morbidity rate amongst travelers not taking prophylaxis due to myths regarding the medication, uneducated regarding the signs and symptoms and to seek early treatment.

Due to the above, resistance to medication and options for prophylaxis are limited with poor outcome if not detected early with a very low parasite count and no co-morbidities. Patients are diagnosed with Malaria and are admitted to an ICU setting due to the high infection count and other complications, also increasing the length of stay in hospital, hospital acquired infection rate increasing, complications due to prolonged admission and illness including Thrombi and cardiac complications, malnutrition and Hepato-renal complications. This in itself has a major burden on the economy due to the high cost of ICU management and care.

Biography

Rochelle is a registered medical practitioner, completed her studies in 2010 at the University of Pretoria. She finished her Diploma in Emergency Medicine in 2017 along with her certificate in Travel Medicine in 2016. She accomplished her dispensing license and has been updated with ATLS, ACLS and PALS in South Africa. She works for one of the busiest, private hospital, Emergency departments in South Africa seeing a multitude of trauma and medical emergencies. She is interested in Family and Travel Medicine practice affiliated with the ED with a special interest in Aesthetic Medicine. She is currently serving on the executive board for the Society of Travel Medicine in South Africa working closely with the NICD with all infectious disease monitoring in SA and submitting interesting case studies to Federation of Infectious Diseases in SA (FIDSSA) on behalf of SASTM.

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Yawei Liu et al., J Transm Dis Immun 2018, Volume 2
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NEURONAL IFN-BETA–INDUCED PI3K/AKT-FOXA1 SIGNALING IS ESSENTIAL FOR GENERATION OF FOXA1^{+}TREG CELLS

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Neurons reprogram encephalitogenic T cells ($T_{(enc)}$) to become regulatory T_{reg} cells FoxP3$^{+}T_{regs}$ or FoxA1$^{+}T_{regs}$. We reported previously that neuronal ability to generate FoxA1$^{+}T_{regs}$ was central to preventing neuroinflammation in experimental autoimmune encephalomyelitis (EAE). Mice lacking the cytokine interferon (IFN)β were defective in generating FoxA1$^{+}T_{regs}$ in the brain. Neuron-induced FoxA1$^{+}T_{regs}$ were capable of preventing chronic and demyelinating EAE in mice lacking IFNβ . Here we show that lack of neuronal IFNβ -signaling was associated with lack of neuronal expression of program death-ligand1 (PDL1), which also prevented their ability to reprogram T_{enc} cells to FoxA1$^{+}T_{regs}$. Transfer of IFNβ competent encephalitogenic T cells to mice lacking IFNβ or its receptor; IFN AR in the brain (*Nes^{Cre}:Ifnar$^{fl/fl}$*) led to the absence of FoxA1+T_{regs} generation and aggravated neuroinflammation. We identified that IFNβ activated neuronal PI3K/Akt signaling. Phosphorylated Akt consequently bound to transcription factor FoxA1, which upon translocation to the nucleus induced neuronal PDL1 expression. Conversely, inhibition of PI3K/Akt, or FoxA1 and PDL1 knock-down blocked neuronal ability to generate FoxA1+T_{regs}. Our study identified crucial molecular player's central for neuronal ability to reprogram pathogenic T-cells and to generate FoxA1$^{+}T_{regs}$, which could be a therapeutic target to prevent neuroinflammation.

Biography

Yawei Liu has a medical doctor background and has been doing medical research for more than 10 years. Since her Ph.D., she mainly focused on the role of neurons in the regulation of auto-reactive T cells and central nervous system (CNS) inflammation. We reported a novel function for neurons as being highly immune-competent cells, based on their crucial role in the regulation of T-cell responses and CNS inflammation in models of multiple sclerosis

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Hadaf Dhafir El Yassin et al., J Transm Dis Immun 2018, Volume 2
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ANTI SMOOTH MUSCLE ANTIBODIES (ASMA) AND TUMOR NECROSIS FACTOR (TNF) IN IRAQI PATIENTS INFECTED WITH HEPATITIS C VIRUS

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Background: Hepatitis C virus (HCV) is a serious infectious disease that can cause lifelong infection. Infection with chronic hepatitis C virus (HCV) can lead to autoimmune hepatitis (AIH) in a minority of patients. A genetic predisposition to autoimmune hepatitis from medication may lead to appearance of serum autoimmune antibodies especially anti smooth muscle antibodies (ASMA). Viral infection induces tumor necrosis factor (TNF-alpha) production in hepatocytes. These findings suggest that both parameters may have an important role in the patho-physiology and drug resistance of human liver diseases induced by viruses.

Aim: The aim of the presents study was to evaluate the role of the immunoendocrine system in the pathogenesis of the disease, by measuring serum TNF and antismoothmuscle antibodies (ASMA).

Subject and methods: Sixty- one chronic hepatitis C patients were consequently selected from the Medical city, Gastrointestinal Hospital in Baghdad, Iraq, during the period from July 2014 to September 2014, their median age was 34.8 year, 29 of them were males and 32 were females. All patients were diagnosed having positive for HCV RNA by means of polymerase chain reaction. The study also included twenty apparently healthy adult ages and sex matched considered as controls, which were negatively screened with hepatitis C virus. Peripheral blood sample of 2 ml was aspirated using disposal syringes. Samples were collected between (9.00a.m-12.00p.m.). The blood was allowed to clot in plain tube for 30-45 minutes at room temperature. Sera were obtained by centrifugation of the collected blood and then stored in plain tubes at -20°C. ELISA method was used to measure serum TNF, while ASMA was measured by indirect immunofluoresent assay.

Results: The results of this study showed an increase in mean value of serum TNF in chronic hepatitis C patients accompanied with a 65% increase in ASMS. Significant correlations were found between both parameters studied.

Conclusions: Chronic hepatitis C is associated with an immunological abnormality. Results obtained might shed a light on the type of therapy and drug of choice when managing the disease.

Biography

Prof. Dr. Hadaf Dhafir El Yassin was faculty in University of Baghdad in the Department of Biochemistry, College of Medicine at University of Baghdad. She finished her Post Doctorate in Clinical Biochemistry at Al-Nahrain University. She is currently the head of the Department of Clinical Biochemistry, University of Baghdad, Iraq. She actively participated in 43 local conferences in Iraq and 26 abroad, making a total of 69 conferences attendance and paper presentation. She also published 85 articles.

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THE IMMUNE PROFILE OF PROSTATE EPITHELIAL CELLS BY IL-6 ACTIVATION MEDIATED BY STAT3 AND AKT SIGNALING PATHWAYS

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Introduction: The major signaling transduction of the pro-inflammatory cytokine IL-6 is through the transcription factor STAT3. However, PI3-K/AKT signaling pathway can also activated by IL-6 under prostate pathological conditions.

Objectives: The aim of this study is to evaluate the tissues levels of STAT3/IL-6/ AKT axis signaling in prostate tissues from patients with benign prostatic hyperplasia (BPH) and prostate cancer (PC).

Material & Methods: Immunohistochemical analyses for IL-6, Gp130, phospho-STAT3 (Tyr705) and phospho-AKT (Ser473) were carried out in 25 samples of BPH, 16 samples of PC.

Results: Immunoreactivity to IL-6 was consistently observed in stroma compartment of BPH and cytoplasmic epithelial cell in PC samples. Phospho-AKT was mainly expressed in membrane and the cytoplasm in PC compared to BPH. Immunoreactivity for phospho-STAT3 (Tyr705) was found in the stroma and the nucleus of epithelial and tumoral cells. No significant association was determined (r=0.153, *P*=0.518) when IL-6 and phospho-AKT (S473) were analyzed within BPH patients; whereas a positive correlation emerged between phospho-STAT3 (Tyr705) in the stroma and phospho-AKT (S473). In PC patients, significant relationship was documented between IL-6 and phospho-AKT (Ser473) (r=0.725, *P*=0.02). In addition, the correlation between phospho-AKT (Ser473) and phospho-STAT3 (Tyr705) as well as detected in the nucleus and the stroma were significant.

Conclusion: This suggests that IL-6/AKT axis could be one of mechanism to activate STAT3 by facilitating inflammatory cell migration and chronic inflammation in BPH and promote cancer progression by promoting cell growth in PC.

Biography

Yosra Bouraoui is experienced in immunology, inflammatory and prostate cancer. Yosra worked on prostate tissue by immunohistochemistry to analyze several signaling pathways in response to inflammatory cytokines such as IL-6, IL-1 and TNF alpha. She is expertized in prostate cell culture to study AKT signaling and NF kappa B mediated by IL1. At this moment she is working on petri net model (In silico model) my experiment results on pro inflammatory cytokines and its network in prostate pathologies. Yosra is currently working on the relation between inflammatory cytokine and immune metabolic in prostate tissue.

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PERFLUOROOCTYL BROMIDE VS. PERFLUORODECALIN: WHICH PERFLUOROCARBON IS PREFERABLE FOR ALBUMIN-DERIVED ARTIFICIAL OXYGEN CARRIERS?

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Currently, perfluorodecalin (PFD) and perfluorooctyl bromide (PFOB) are the majorly used perfluorocarbons in the field of artificial oxygen carriers. So far, in our investigations only PFD has been employed as core material of albumin-derived artificial oxygen carriers (A-AOCs). This led to the question of whether PFOB would display an equally safe alternative in A-AOCs. To investigate toxicity, we studied A-AOCs with a PFOB-core in a top load model (TL) and compared the data with results from our previous studies with a PFD-core. TL (+1/6 of blood volume) experiments with 16 healthy Wistar rats were performed with and without (control) A-AOCs (17 vol. %), respectively. After the infusion period (30 min) rats were further observed up to 180 min. During TL systemic parameters, plasma enzyme activities and acid base status were continuously monitored. To confirm hemolysis obtained in the *in vivo* model, supporting *in vitro* studies were performed additionally: whole blood was incubated varying temperature, A-AOCs-concentration and mechanical stress. Blood pressure showed a transient drop during infusion of A-AOCs but was unaffected in the control group. Compared to control animals the PFOB-group displayed increased plasma enzyme activities. All effects after application of only 17 vol. % A-AOCs with PFOB-core were considerably more pronounced compared to 32 vol. % A-AOCs with PFD-core. Furthermore, hemolysis caused by A-AOCs with PFOB-core was significantly more distinct compared to A-AOCs with PFD-core. In conclusion, PFOB should be avoided as core material for A-AOCs because of distinct side-effects already occurring at low dosage.

Biography

Alexandra Scheer completed her Master's degree in Medical Biology in 2015. Since October 2015, she has been working on her Doctoral thesis at Institute of Physiological Chemistry-University Hospital Essen, Germany, in the working group of Dr Katja B Ferenz. Within the scope of this work, she is involved in the development and characterization of artificial oxygen carriers. Since February 2018, she followed Katja B Ferenz in the Institute of Physiology at University Hospital Essen, Germany, to continue her work. Her research interests include "Artificial oxygen carriers, biomaterials, nanoparticles and perfluorocarbons".

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CARDIAC PATCH LOADING MSCS OVEREXPRESSING Tβ4 PROMOTES REPAIR OF THE INFARCTED MYOCARDIUM BY ENDOGENOUS REGENERATIVE MECHANISMS

Yu-zhen Tan, Qiang-li Wang, Hai-jie Wang and **Yong-li Wang**
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Recent studies suggest that the epicardium plays an important role in cardiomyogenesis during development, while it becomes quiescent in adult heart. Thymosin beta 4 (Tβ4) has an effect on activating the epicardium. However, effectiveness of Tβ4 administration is unsatisfactory. Therefore, this study prepared cardiac patch and investigated efficiency of activating the epicardium by Tβ4 released sustainedly from the cardiac patch. Mesenchymal stem cells (MSCs) isolated from bone marrow of rats and mice were transfected with Tβ4. Tβ4 release from the cells was determined with an acquity ultra-performance liquid chromatography system. For preparing of cardiac patch, the cells transfected with Tβ4 and Flag were seeded on PLACL/collagen membrane formulated by electrospinning. The survival and proliferation of the cells on the nanofibers were examined after treatment with hypoxia. In MI models of rats and Wt1*CreERT2/+,* R26*mTmG* mice, the patches were implanted on the epicardium of the infarcted region. In rat models, differentiation of the epicardium-derived cells (EPDCs) and the engrafted MSCs towards cardiomyocytes and vascular cells was examined by Wt1 immunostaining and Flag labeling. In transgenic mouse models, the activated EPDCs expressed GFP. At four week after implantation of the patches, cardiac function was improved significantly, scar area in the infarcted region was reduced obviously. EPDCs increased in subepicardium and myocardium, and some $Wt1^+$ cells and GFP^+ cells expressed CD31, α-SMA or cTnT. Moreover, $c\text{-}kit^+$ cells were observed in subepicardium and myocardium, and a few of them expressed CD31, α-SMA or cTnT. Flag labeling showed that some engrafted MSCs migrated into subepicardium and myocardium. These results suggest that Tβ4 released from the transfected MSCs in PLACL/collagen nanofibrous patches may effectively attenuate left ventricular remodeling and improve cardiac function by activating the epicardial cells and recruiting endogenous stem cells. Our finding provides a novel strategy for myocardial regeneration by enhancing the endogenous regenerative mechanisms.

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Biography

Yu-zhen Tan is a Professor in Department of Anatomy, Histology and Embryology at Shanghai Medical School of Fudan University. She completed her MD at Nanjing Medical University, China and; PhD at Shinshu University School of Medicine, Japan. In 1999, she became a Professor of Shanghai Medical School of Fudan University. She firstly designed and synthesized the self-assembling peptide modified with RGDSP, and investigated effects of the peptide carrying stem cells on repairing the infracted myocardium. She was also first to investigate effects of the PCL/gelatin nanofibrous patch on repairing the infracted myocardium after transplantation on the epicardium. Her research interests include "Stem cell differentiation and cardiovascular regenerative medicine".

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LYMPHATIC ENDOTHELIAL PROGENITOR CELLS AND VEGF-C LOADED WITH SELF-ASSEMBLING PEPTIDE NANOFIBERS PROMOTE LYMPHANGIOGENESIS IN INFARCTED MYOCARDIUM

Hai jie Wang, Hai-feng Zhang, Yu-zhen Tan and **Yong-li Wang**
Shanghai Medical School of Fudan University, China

Lymphatic vessels play a crucial role in draining excess fluid and transport macromolecular substances from extracellular spaces. Disfunction of lymphatic vessels may cause lymph edema and chronic inflammation, leading to fibrosis of the local tissue. This study investigated efficiency of transplantation of lymphatic endothelial progenitor cell (LEPCs) and sustained release of VEGF-C from self-assembling peptide (SAP) on promoting lymphangiogenesis after myocardial infarction (MI). CD34+VEGFR-3+ EPCs were isolated from rat bone marrow. Sustained release of VEGF-C from SAP nanofibers (SAPNs) was detected with ELISA. Compatibility of SAPNs with the cells was accessed with transmission electron microscopy and EB/AO staining. After rat MI models were established with ligation of the anterior descending branch of the left coronary artery, SAP carrying the cells and VEGF-C was injected at the border of the infarcted region. At four week after transplantation, the survival and differentiation of the cells labeled with GFP were examined, and repair of the infarcted myocardium was evaluated. Under induction with VEGF-C, CD34+VEGFR-3+ EPCs could differentiate into lymphatic endothelial cells. The cells spread well along SAPNs. SAPNs protected the cells from apoptosis in the condition of hypoxia, and released VEGF-C sustainedly. After transplantation, cardiac function was improved significantly. The number of the survived cells increased, and some cells differentiated into lymphatic endothelial cells. Density of lymphatic vessels increased, and cardiac edema was reduced. Moreover, angiogenesis and myocardial regeneration were enhanced. These results suggest that SAPNs load LEPCs and release VEGF-C effectively. VEGF-C released from SAPNs induces differentiation of LEPCs towards lymphatic endothelial cells. Loading stem cells and releasing growth factor with SAPNs is a promised strategy for MI therapy.

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3. **Wang G D, Tan Y Z, Wang H J and Zhou P (2017) Autophagy promotes degradation of polyethyleneimine–alginate nanoparticles in endothelial progenitor cells. Int J Nanomed. 12: 6661–75.**

Biography

Hai-jie Wang is a Professor of Department of Anatomy, Histology and Embryology at Shanghai Medical School of Fudan University. He studied Clinical Medicine at Weifang Medical College, China. He completed his MD from Medical School of Shandong University, China in 1987 and PhD from Shinshu University School of Medicine, Japan in 1996. He studied Molecular Medicine at School of Medicine, Yale University from 2005 to 2006 as Visiting Professor. In 1999, he became a Professor of Shanghai Medical School of Fudan University. His research interests include "Differentiation and transplantation of endothelial progenitor cells".

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&
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EFFECT OF LAMININ ON NEUROTROPHIC FACTORS EXPRESSION IN SCHWANN-LIKE CELLS INDUCED FROM HUMAN ADIPOSE-DERIVED STEM CELLS *IN VITRO*

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The Schwann-like cells can be considered as promising in stem cell therapies, at least in experimental models. Human adipose-derived stem cells (ADSCs) are induced into Schwann-like cells (SC-like cells) and are cultured on either a plastic surface or laminin-coated plates. The findings here reveal that laminin is a critical component in extracellular matrix (ECM) of SC-like cells at *in vitro*. The survival rate of SC-like cells on a laminin matrix are measured through MTT assay and it is found that this rate is significantly higher than that of the cells grown on a plastic surface ($P < 0.05$). Schwann cell markers and the myelinogenic ability of SC-like cells at the presence versus absence of laminin are assessed through immunocytochemistry. The analysis of GFAP/S100β and S100β/MBP markers indicate that laminin can increase the differentiated rate and myelinogenic potential of SC-like cells. The expression levels of SCs markers, myelin basic proteins (MBP), and neurotrophic factors in two conditions are analyzed by real-time reverse transcription polymerase chain reaction(RT-PCR).The findings here demonstrated that gene expression of SCs markers, MBP, and brain-derived neurotrophic factors (BDNF) increase significantly on laminin compared to plastic surface ($P < 0.01$). In contrast, the nerve growth factor(NGF) expression is down regulated significantly on laminin-coated plates ($P < 0.05$). The obtained data suggest that production of neurotrophic factors in SC-like cell in presence of laminin can induce appropriate microenvironment for nerve repair in neurodegenerative diseases.

Biography

Dr. Tadjalli is a professor of Histology and Zarinfard is a graduate student at the department of Histology School of Veterinary Medicine, Shiraz University, Shiraz and Dr. Razavi is a professor of Histology Isfahan University of Medical Science, Isfahan, Iran. Her research interest includes Histology, stem cell, regeneration.

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INFLAMMATORY MECHANISM DURING JAPANESE ENCEPHALITIS VIRUS INFECTION

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Japanese encephalitis (JE) is one of the chief causes of acute encephalitis syndrome (AES) in North India with more than 15% confirmed cases. The disease is caused by Japanese encephalitis virus (JEV), a neurovirulent RNA flavivirus transmitted by *Culex* mosquitoes. The virus in natural cycle circulates between pig and mosquitoes or bird and mosquitoes, with pigs being the most important biological amplifiers. Though humans are accidental dead end hosts, JE has generated considerable public anxiety because it mainly remains a disease of children. The disease ranges from non-specific febrile illness to a severe meningoencephalomyelitis illness. The transmission of disease can occur throughout the year in endemic zone, with disease at a peak during monsoon season. Since there is no specific treatment available and vaccination is the best measure to get protection from the disease; it is important to understand the molecular mechanisms in host. The virus has been shown to induce neutrophil infiltration in neural and extra neural tissues. A neutrophil chemotactic protein derived from macrophages had been isolated from JEV induced animal models. It had variety of pathologic effects on host, including vascular permeability and breach in blood brain barrier. The presence of inflammatory chemokine IL-8 was also significantly detected in JE confirmed patients during acute phase of illness. The study had revealed a correlation between IL-8 levels and severity of illness as all severely ill and fatal cases showed higher levels of IL-8 in acute cerebrospinal fluid (CSF) and serum. In cases who recovered completely, the level of IL-8 declined markedly by convalescent phase. The study indicates important interaction between pro inflammatory cytokine, macrophages and neutrophils during JE infection.

Biography

Aditi Singh has over seventeen years of experience in research and academics. After completing her Doctorate in the year 2000 from King Georges' Medical College, she started teaching at undergraduate and post graduate level. During PhD thesis, she tried to understand the pathogenesis of Japanese encephalitis virus in disease confirmed patients; where in pro inflammatory cytokines were studied and evaluated. Her area of research interest is Microbiology, Immunology and Enzymology. Till now, she has authored more than thirty research papers in national and international journals and two book chapters. She has presented more than twenty papers in national and international conferences. Currently, she is working as Associate Professor in Amity University, a leading private University of India.

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&
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INHIBITORY EFFECTS OF DIPEPTIDE ANALOGUE, DAPT, ON γ-SECRETASE CAUSING DECREASE IN AMYLOID-β CONCENTRATION IN NEUROBLASTOMA CELLS

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Alzheimer's disease is a very common type of dementia that destroys memory and other important mental functions. It is a neurodegenerative disorder which affects over 5 million people in the United States alone. In fact, it is the 6th leading cause of death in the United States, and on average, an American is diagnosed with Alzheimer's disease every 66 seconds. This can be a familial or sporadic disease which is primarily caused by the destruction of neurons which starts from the hippocampus and spreads throughout the brain (cerebellum is spared). The apoptosis of the countless neurons seems to be caused by a multitude of factors including amyloid-beta plaques, Tau tangles, and neuronal loss. For the sake of this investigation, there will be a primary focus on the amyloid-beta plaques because the accumulation or buildup of neurotoxic plaques on the neurons seems to be a key factor in Alzheimer's disease. Enzymes called γ-secretase and β-secretase cleave a protein called an amyloid precursor protein (APP) to form these amyloid-beta peptides which can accumulate and form neurotoxic plaques. Previous studies have found that DAPT, a dipeptide analogue, is effective in inhibiting γ secretase thus decreasing amyloid-beta concentration in the brain. This study confirms the efficacy of DAPT in inhibiting γ-secretase, but also investigates the alternative inhibitory effects of other drugs like Activase® rt-PA (alteplase), a tissue plasminogen activator typically used for treatment of stroke, and clonazepam (E64), a pill used to treat panic disorder and anxiety. Although the goal was to see the effects on Aβ40 (40 amino acid amyloid-beta chain) and Aβ42 (42 amino acid amyloid-beta chain) production, only the effects of Aβ40 production were examined due to possible contamination in the Aβ42 tests.

Biography

Arnav Gupta is passionate about exploring causal relationships of drugs commonly used to treat neurodegenerative disorder. Arnav is interested in pursuing a career in neuroscience. His passion in studying the brain and memory has inspired him to do research about Alzheimer's disease. This common disorder presents so many unanswered questions, and Arnav is motivated to do further research in the future to answer some of these questions.

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RECOVERY OF BIOACTIVE PROTEIN FROM INCLUSION BODIES USING MILD SOLUBILISATION AGENT

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Insoluble and inactive protein aggregates known as inclusion bodies are the major bottleneck in the recovery of bioactive recombinant proteins from Escherichia coli. Strong denaturants such as high concentrations of urea and guanidine hydrochloride offer good solubility over a wide range of IBs but result in poor recovery of bioactive protein. Recent studies showing the presence of native-like secondary structure in inclusion bodies have led to the development of mild solubilization agents like organic solvents, alkaline pH and low concentration of denaturants. These mild solubilization agents offer 5-6 times better recovery of bioactive protein from inclusion bodies than aforementioned strong denaturants. In this study, we demonstrated the solubilisation potential of trifluoroethanol (TFE), an organic solvent, in nine inclusion body proteins. Different concentrations of TFE with or without low concentration of denaturant were screened to arrive at an optimal ratio. A mixture of 30% TFE with 3 M urea performed the best at solubilizing maximum amounts of protein. Taking human growth hormone (hGH) as a model protein, mode of action of TFE against strong denaturants was investigated using fluorescent spectroscopy and circular dichroism. The results from these techniques suggested the disruption of tertiary structure and stabilization of secondary structure of protein. Furthermore, the cell number based activity assays indicated the presence of fully functional and bioactive protein recovered from TFE solubilized hGH inclusion bodies. We concluded that TFE could be used as a mild solubilization agent to recover maximum amount of bioactive protein from inclusion body proteins.

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HORMONE IMMUNOTHERAPY IN ENDOCRINE DEPENDENT METASTATIC BREAST CANCER PATIENTS

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Insoluble and inactive protein aggregates knownHormone therapy is advised for ER+ metastatic breast cancer patients due to its efficacy concomitant with low toxicity; however, in most patients the occurrence of resistance is a not well yet understood hurdle to overcome. In these patients, during clinical benefit (CB) from conventional anti-estrogens, the addition of cycles of sequential immunotherapy could prolong the benefit and delay the arising of acquired hormone resistance. In order to validate this hypothesis, in 1992 we started an open exploratory clinical trial. Forty-two of these patients in CB during first line anti-estrogen salvage therapy also received beta-interferon (INF-beta) 3,000,000 IU i.m./day 3 days/week for 1-4 weeks and successively recombinant *interleukin*-2 (IL-2) 3,000,000 IU s.c./day 3 days/week for 5-8 weeks until progression. The immunotherapy cycle lasted 10 weeks and the patient continued anti-estrogen alone during 9-10 weeks, the 11th week being the first week of the successive cycle. At each control visit, routine laboratory examinations and serum measurement of a CEA, TPA, CA15.3 tumor marker (TM) panel were carried out, and an immunological assessment was made (total lymphocytes, CD4+, CD8+, NK cells, T-reg, IL-6, IL-10, IL-12, TNFa, TGFbeta1 and IFN-gamma). The addition of INF-beta-IL-2 sequence significantly prolonged clinical benefit and overall survival from conventional antiestrogens. During CB as opposed to progression, a significant immune stimulation was observed. During CB also a significant CEA, TPA, CA15.3 decrease occurred 24–72 h after interleukin-2 administration. At the progression a significant increase for CEA and for all three markers (standardized values) was found 24–72 h after interleukin-2 administration. In patients who survived less than five years, the Treg cell increase occurred at a significantly shorter time interval than in those who survived longer than five years (20 vs. 45.5 months, respectively; P=0.001). To further confirm these promising results, a multicenter prospective phase II trial is going to be launched by the Cancer Center Institute of Tuscany in Italy.

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REGENERATIVE POTENTIAL OF CD45 NEGATIVE MARROW CELLS RELIES ON TWO SEPARATE SUBPOPULATIONS BEING EITHER ANTI-INFLAMMATORY OR PRO-ANGIOGENIC

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In solid organs, there are the cells with regenerative potential. They can start regeneration of the damaged tissue with a support coming from the bone marrow. In the early 2000s, we started a project in which the mononuclear cells of bone marrow (BMMC) were used to save the legs of patients with critical limb ischemia (CLI). Injection of the cells into the affected limb resulted in several following events which started with a pain release than healing of ulcerations and finally the shortening of a distance of claudication. 40% of 23 patients with CLI receiving BMMC enjoyed remission lasted up to 10 years or more. We learned that several subsets of BMMC may act in concert supporting each other in achieving the goal revascularization of the limb. Revascularization of the infarcted femur was achieved by implantation BMMC via the holes drilled in the head of the femur. It appeared that the cells with the MSC markers played rather a moderate role in preventing the leg amputation as the main actors are endothelial progenitor cells (EPC, CD391+) which are present in the implant. Similarly to leukophoretic product BMMC also the marrow cells centrifuged in own plasma gradient (commercially available kit) is enriched in the cells having MSC and EPC markers. The cells of the latter composition injected intra-articularly into the hip or knee brought a relief in patients with osteoarthritis. We believe that the positive effect of this cellular therapy is due to the presence in a fresh fraction of BMMC a variety of progenitors including MSC and EPC which acting in concert exert the anti-inflammatory, tissue regenerative and angiopoietin effect.

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HOW EFFECTIVE IS MALARIA ERADICATION STRATEGIES IN AFRICA?

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Background: In Africa, malaria has continued to be a big dilemma and a primary cause of mortality and morbidity especially among children under the age of 5, pregnant women and immunocompromised people for example people infected with HIV/AIDS. Despite global efforts in the management and eradication towards malaria, African countries have fallen behind due to many factors. However, the availability of preventative method such as long- lasting insecticide treated bed nets (LLIN), insecticide treated nets (ITN), and indoor residual spraying (IRS) has been instrumental towards eradicating malaria in Africa. While other countries in the world have managed to eradicate malaria, doubts arise in Africa due to the effectiveness of present measures. Consequently, this study evaluates malaria eradication strategies in Africa, the main objective of this study is to detect if eradication strategies such as ITN and IRS methods are reducing the rate of malaria.

Method: A literature search was conducted on scientific databases such as NCBI, Google scholar, PubMed etc. strict inclusion, exclusion criteria were applied in the filtration process of publication and this was done in order to have the best studies to conduct this project. Outcomes of the search were use of ITN/ LLIN vs. non-use.

Results: Seven papers were identified and analyzed. Three groups were identified (Control, LLIN and ITN). The mean value for the control group is 49.69%. The participant in the LLIN group had a mean infection rate of 47.97% and the ITN group had an infection rate of 23.12% during the duration of the study these two groups were using the preventative method. This showed that LLIN and ITN use reduces malaria infection, however according to results obtained ITN reduced malaria infection more than LLIN.

Conclusion: Preventative method to reduce malaria infection is important, the use of LLIN and ITN shows that if used it can prevent people getting infected with malaria.

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PDT - TREATING THE UNTREATABLE

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Background: Treatment of cutaneous lesions by radiotherapy or surgery entails destruction or excision of normal tissue surrounding a lesion. Lesions may be extensive, at sites of poor healing, or adjacent to difficult to reconstruct tissues. This may make treatment by these modalities impossible, or may result in poor cosmetic or functional outcomes. Photodynamic therapy (PDT) offers an important alternative. PDT is a highly selective treatment which can achieve total eradication of malignant and premalignant lesions with minimal damage to normal tissues.

Methods & Results: Patient one had an extensive port wine stain. This had been treated by radioactive Thorium in the past, but this had caused multiple basal cell carcinomas to develop over the whole area. Patient two had a substantial basal cell carcinoma on the back. Photofrin was employed and additional light irradiation using a laser fibre inserted into the base of a thick lesion. Patient three has Gorlin syndrome and had developed a basal cell carcinoma on the lower eye lid. This was eradicated by Photofrin PDT. Patient four presented with an extensive thick plaque of Bowen's disease covering his knee. This resolved after several treatments of Metvix PDT. Patient five was referred with an extensive area of extra-mammary Paget's disease. This was eradicated by Photofrin PDT.

Conclusion: PDT is no longer simply a treatment option for superficial cutaneous non-melanoma carcinoma and pre-malignant conditions. It is the preferred treatment in a range of challenging situations where surgery and radiotherapy are likely to produce suboptimal results.

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3D PRINTING TECHNOLOGY: STUDY BASED ON NANO-SIZED CALCIUM PHOSPHATE AND PCL INK FOR BONE TISSUE REGENERATION

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The objective of this study was to obtain three-dimensionally (3D) printed scaffolds from nano-sized calcium phosphate (amorphous calcium phosphate; ACP) and polycaprolactone (PCL) ink for bone tissue regeneration (BTR). The synthesis of the nano-sized calcium phosphate was performed by wet method. The material was characterized by X-ray diffraction (XRD), infrared spectroscopy (FTIR), scanning electron microscopy (SEM) and determination of the surface area by adsorption of nitrogen. After obtaining the ink, cylindrical scaffolds (15x10 mm) were 3D printed. Mechanical strength, SEM, EDX and porosity were evaluated. Results of XRD analysis showed an intense peak at θ=23.4 corresponding to ACP, as well as other peaks of lower intensities corresponding to HA, ACP and OCP. The FTIR spectrum confirmed that the sample obtained corresponded to the nano-sized calcium phosphate sample. Studies of viscosity showed that the composition of nano-sized calcium phosphate and PCL used was suitable for the preparation of the ink. The SEM analysis shows that the post-printed material maintains the cylindrical structure. It is also observed interconnected pores. On the other hand, the EDX analysis showed the presence of calcium and phosphorus. The analysis of microstructural characterization using the mercury intrusion porosimetry method showed that the porosity was around 70%. The mechanical resistance studies showed values within the range established for this type of material. The nano-sized calcium phosphate obtained through the synthesis process has the necessary quality to be used in the production of inks for 3D printing employed in BTR.

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DIFFERENTIAL PROLIFERATION OF WOLBACHIA INFECTIONS IN AEDES MOSQUITO CELL LINES

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The cell lines of *Aedes albopictus* (C636) obtained at NIMHANS, Karnataka, India are maintained in incubators with M&M insect cell culture medium and BSA to establish *Wolbachia* infections. The cell lines were inoculated with *Wolbachia* (A, B and AB super groups) and the strength of the infection was calculated based on the perpetuation of the cells in the *in vitro* medium. The comparison between control and infected cell lines suggests a two-fold decrease in infected cells reaching confluence. The inoculum strength differs from the source of *Wolbachia* isolated from different insect hosts. *Wolbachia* extracts with double infections (AB) are highly virulent than single infections. Among single infections, *Wolbachia* B super-group is more virulent than A super-group. Further, it was observed that *Wolbachia* derived from *Exorista sorbillans*, *Aedes albopictus*, *Trichogramma japonicum* has greater virulence and cell lines can be infected within few passages. *Wolbachia* isolated from Talicada nyseus did not induce any significant effect on mosquito cell lines. The findings of the current study append the database of potential non-native *Wolbachia* strains that can be introduced in mosquitoes for expression of novel phenotypes. Recent findings reports increased virulence of pathogenic West Nile Virus, when confounded with native *Wolbachia* strains in mosquitoes. Thus screening of alternative *Wolbachia* strains that could be maintained in mosquito cell lines and establishing a *Wolbachia* strain pool for mosquito trans-infection is of significant importance.

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LACK OF MIDDLE EAST RESPIRATORY SYNDROME CORONAVIRUS BUT PREVALENCE OF INFLUENZA VIRUS IN IRANIAN PILGRIMS WITH SEVERE ACUTE RESPIRATORY INFECTIONS- HAJJ 2017

Jila Yavarian, NZ Shafiei Jandaghi, Maryam Naseri, Azadeh Shadab and **Talat Mokhtari Azad**

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More than 2.5 million Muslim pilgrims are gathering in Mecca during the Hajj pilgrimage annually. Hajj is one of the largest mass gatherings of its kind in the world. Transmission of different infectious diseases especially respiratory tract infections during mass gatherings in holly places has a global effect when pilgrims return to their country. The aim of this study was to determine the prevalence of Middle East Respiratory Syndrome coronavirus (MERS-CoV) and influenza virus infections among Iranian pilgrims returning from Hajj in 2017. Throat swabs collected from 132 pilgrims with severe acute respiratory infections (SARI) were examined for presence of MERS-CoV and influenza viruses from 10 September until 4 October 2017 in National Influenza Center, Tehran, Iran. Each sample was tested in a 25 µl reaction for MERS-CoV and influenza A/B by using QuantiFast Probe RT-PCR Kit (Qiagen, Germany). MERS-CoV was tested with targeting the upstream region of the E gene (UpE) for screening and the open reading frame 1b for confirmation. None of the pilgrims tested positive for MERS-CoV, however, 20 (15.2%) were positive for influenza viruses. Influenza A/H3N2, B and A/H1N1 accounted for 60% (12/20), 30% (6/20) and 10% (2/20) of the virus positive samples, respectively. This study showed the prevalence of influenza infections among Iranian pilgrims and suggests continuing surveillance and screening in the pilgrims, appropriate vaccination and other preventive strategies especially nowadays that the risk of influenza pandemic threatens the world. Meanwhile testing for MERS-CoV is necessary for early diagnosis to prevent virus transmission.

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BIOASSAY DIRECTED ISOLATION OF HYPOTENSIVE ALKALOIDS FROM HOLARRHENA PUBESCENS

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Holarrhena pubescens **belongs to the family Apocynacea, commonly known as "kurchi" is highly reputed in traditional medicine as a remedy for amoebic dysentery and other intestinal ailment. Bioassay-directed fractionation by chromatographic methods the ethanolic extract of *Holarrhena pubescens* resulted in the isolation of steroidal alkaloids i.e. Holamide and Pubscinine [1]. Holamide showed a three proton doublet at 1.45 (J=6.56 Hz) and two AB doubles at 3.17 and 3.00 each for on proton (J=12.06 Hz) in the 1H NMR spectrum suggested that it belongs to conanine series of alkaloid (A class of compound with the steroid nucleus and a five members heterocyclic ring with nitrogen). In contrast Pubscinine showed one methyl at 1.28 while the doublet is missing a three proton singlet was observed at 2.28 due to a vinylic methyl indicated a double bond in the 18,20 – epimino ring of the conanine series of alkaloids. In anaesthetized rats, the Holamide and Pubscinine caused a fall in blood pressure in a dose-dependent manner. Pretreatment of animals Atropine completely abolished the hypotensive response of Acetycholine; whereas hypotensive effect of Holamide and Pubscinine were not modified by Atropine [1]. Similarly Acetylcholine produced contractile effect in guinea-pig ileum, which was antagonized by atropine, however both (Holamide and Pubscinine) failed to produced any stimulant response on guinea-pig ileum. These data indicate that the steroidal alkaloids i.e. Holamide and Pubscinine from *Holarrhena pubescens* mediated hypotensive response through a mechanism different to that of Acetycholine.**

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DETERMINATES OF CELL THERAPY EFFICACY FOR TISSUE AND ORGAN REPAIR

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It is not unusual for animal models of disease to inaccurately predict clinical outcome of clinical studies. One such example is stem cell therapy for stress urinary incontinence (SUI) where preclinical studies report almost complete remission of symptoms, whereas clinical studies report only around 50% remission in 50% of patients. The answer is most likely explained because animal models (which create acute SUI in relatively young animals) do not represent the most common clinical scenario where SUI is most common as a chronic disease in peri/post-menopausal women with co-existing risk factors such as obesity and type-2 diabetes. To better predict the effects of cell therapy for UI, we developed a cynomolgus monkey model of urinary incontinence (surgical nerve and muscle damage to the urinary sphincter complex) that reproduces the functional and structural changes in the urinary sphincter complex seen in women with clinical SUI. In these studies, we modeled both acute and chronic SUI in younger and older female NHPs with varying degrees of estrogen deficiencies and impaired glucose/insulin metabolism. With an n=6/experimental group, autologous skeletal muscle precursor cells (skMPCs) were isolated from a muscle biopsy, expanded to 5 million cells and injected directly into the urinary sphincter complex of NHPs with SUI. skMPCs almost completely restored sphincter muscle content and urethral pressures in younger (5-8years) NHPs ($p<0.05$ vs. SUI/no treatment), but not older (15-28 years) NHPs ($p>0.05$ vs. SUI). This same pattern of efficacy was observed in NHPs with acute vs. chronic SUI, in intact vs. ovariectomized NHPs; in normal cycling dominant NHPs vs. dysmenorrheic subordinate NHPs and in normal weight/normal glucose metabolism vs. heavier impaired glucose/insulin ratio NHPs. Thus, there are multiple determinants of cell therapy efficacy that can be modeled in NHPs and are critical to translational applicability of regenerative medicine approaches to tissue repair.

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APOPTOSIS OF IMMUNE CELLS IN TYPE 1 DIABETES MELLITUS

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Type 1 diabetic mellitus (T1DM) is known to be associated with progressive destruction of β-cells of the pancreas. Dysregulated immunity and programmed cell death are an important link in pathogenesis of diabetes. In this study, we examined expression levels of *interleukin-2*, *BCL1*, *ANXA-11* genes in patients with T1DM. The study was done with blood leukocytes of 30 T1DM patients and 70 healthy controls. Reverse-transcription PCR was done with Transcriptor first strand cDNA synthesis kit from Roche Life Science. The study of IL-2 and BCL1 gene expression level in peripheral blood leukocytes indicated that the median gene expression levels of *IL-2* and *BCL1* are increased in patients with T1DM patients compared with control group of healthy persons. The study of apoptosis by annexin test has revealed an increased level of *ANXA-11* expression in T1DM patients. The obtained data can serve as an additional source for understanding the pathogenesis of T1DM mechanisms.

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A KEY TO THE BACKDOOR INTO THE CASTLE: THE CLINICAL RAMIFICATIONS OF IMMUNOEDITING DRIVEN BY ANTIGENIC COMPETITION

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Over the last decade the field of cancer biology has gained considerable data on genomic heterogeneity. This situation creates challenges and possibly opportunities for cancer treatment. The evolution of the tumor at all stages also requires the growing malignancy to confront and avoid the immune system. What we describe here is the interaction of two immune phenomena that work together to change the characteristics of the tumor, i.e., antigenic competition and immunoediting. These two systems are mutually functional and their interaction is capable of altering the characteristics of the tumor for protection and survival in an immune competent host as well as restricting the diversity of the tumor clones. Therefore, the final outcome of these interactions can also become the key to the backdoor into the castle. Through an additional immune manipulation, autologous tumor cell immunization, we can achieve prevention of disease recurrence after surgical resection and by analyzing induced human monoclonal antibodies to the neoantigens, gain in site into the restriction of diversity of the mutant clones. These findings may also open the door for a pathway to immune prevention of cancer.

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