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July 28, 2021 | Webinar

Reconstruction of cell spatial organization based on ligand-receptor mediated self-assembly

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Peking University, Beijing, China

Single-cell RNA sequencing (scRNA-seq) has revolutionized transcriptomic studies by providing unprecedented cellular and molecular throughputs, but spatial information of individual cells is lost during tissue dissociation. While imaging-based technologies such as in situ sequencing show great promise, technical difficulties currently limit their wide usage. Since cellular spatial organization is inherently encoded by cell identity and can be reconstructed, at least in part, by ligand-receptor interactions, here we present CSOmap, a computational strategy to infer cellular interaction from scRNA-seq. We show that CSOmap can successfully recapitulate the spatial organization of tumor microenvironments for multiple cancers, and reveal molecular determinants of cellular interactions. Further, CSOmap readily and enable in silico simulates molecular and cellular perturbation of genes or cell types to gain novel biological insights, especially into how immune cells interact in the tumor microenviroment. CSOmap can be widely applicable to interrogate cellular organizations based on scRNA-seq data for various tissues in diverse systems.

Biography

Xianwen Ren has his expertise in bioinformatics algorithm development in leveraging power inherent in omics data for improving the health and wellbeing. The recent bioinformatics algorithm he developed creates new pathways for investigating the cellular and molecular mechanisms of tumor immune microenvironment in mediating immunotherapies. Based on single-cell RNA-seq data and human ligand-receptor Interactome data, he has developed a bioinformatics algorithm named as CSOmap to successfully reconstruct cellular spatial organizations, which has been validated on a diverse set of organs and tumors. In particular, CSOmap enables in silico gene overexpression/depletion and adoptive cell transfer/depletion to characterize the corresponding spatial changes, which is insightful to identify critical drug targets.

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Randomised controlled trial evidence questions the assumption that pulmonary metastasectomy benefits patients with colorectal cancer

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Statement of the Problem: Over 40 years there has been an increasing number of operations to remove lung metastases from colorectal cancer (CRC) in the belief that otherwise the selected patients would have near zero five-year survival (5YS). Claims for benefit are as high as 60% 5YS. Methodology & Theoretical Orientation: Pulmonary Metastasectomy in Colorectal Cancer (PulMiCC) recruited patients who were potential candidates for metastasectomy and with informed consent collected baseline data on factors known to influence survival. Where there was equipoise patients were offered randomisation into a nested randomised controlled trial.Findings: 512 patients were recruited in 25 centres in Europe and China of whom 263 had elective metastasectomy, 128 did not, 93 were randomised and 28 were excluded. In elective patients 5YS with metastasectomy was 47% versus 22% without. The difference could all be accounted for by differences in the number of metastases, liver involvement andelevation of the tumour marker carcinoembryonic antigen. In the RCT where the arms were very well balanced for all seven known risk factors, there was no statistical difference at any time point and median survival was in fact longer for controls at 3.8 versus 3.5 years.Conclusion & Significance: The zero-survival assumption was refuted. The true survival without metastasectomy in patients who could have been candidates for operation was highly significantly better than the more plausible 5% estimate (P<0.001). The claimed benefit of 60% is also refuted although some very much smaller difference in the long term cannot be excluded. The claimed relief from chemotherapy was not seen. Lung metastases are very rarely isolated disease but a clear signal of systemic blood borne spread. Lung metastases rarely contribute to terminal decline or symptoms. By removing them all that is achieved is loss of the most easily monitored component of the disease.

Biography

Tom Treasure is a cardiothoracic surgeon with a research base as Honorary Professor in the Clinical Operational Research unit at University College London.

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Immunologic reactions to bone and articular implants

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Statement of the Problem: In recent years the number on implantable devices that have been used in orthopedic surgeries has increased exponentially. As the number of people with orthopedic implants has grown, implant failure has become an increasingly important public health issue. While a significant percent of joint implants fails at between 15 and 20 years some authors suggest that one of the main causes is the interaction between the immune system of the host and the material of the implant Methodology & Theoretical Orientation: The search engines used for research comprised of PubMed, Google Scholar and Cochrane Library. Findings: This review aims to summarize relevant and recent data on the immune reactions that are taking place at the juxtaposition between the implant and the patient's tissue, the time frame in which these immune reactions take place and some of the factors that can influence this reaction. The immune reactions can be divided into: hyperacute immune reactions (anaphylactic shock), acute reactions, the transition between the acute phase and the chronic phase and last but not least chronic immune reactions to such implants. Conclusion & Significance: The research being done with regard to implant-related immunology strives to help in solving the problem of long-term implant failure.

Biography

Cristian Paul Dan is a young orthopedic surgeon specialist with an ongoing education, currently specializing in pediatric orthopedic surgery. His continuous interest and work in the field of implantology has crystalized in the recent published articles. Cristian Paul Dan was aided in this endeavor by Adrian Alexandru Dorin Silaşi and Simona Irina Dan. Adrian Alexandru Dorin Silaşi while working in the field of medical oncology has expressed a keen interest in immuno-oncology, interest that has branched out in other fields of immunology as well. Simona Irina Dan while activating in the Department of Physical Medicine and Rehabilitation as medical resident has aided in selecting the articles necessary for the compilation of the research that this team has published. The research was performed under the guidance of the illustrious Prof. Dr. Gheorghe Tomoaia whose extensive research in the field of orthopedic surgery and his vast clinical experience were invaluable for the team.tom.treasure@gmail.com

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Sox2-mediated 5hmc dysregulation in gbm stem cells

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Primary brain tumors are among the most devastating forms of cancer and glioblastoma (GBM) represents the most aggressive and lethal form of the disease. We now know that GBM contain small subsets of cells that display tumor-propagating stem-like phenotypes (i.e. glioma stem cells or GSCs) that act as critical determinants of GBM resistance to current treatments and tumor recurrence for which there is no proven therapy. Altered patterns of DNA methylation are widely reported in human GBM.. Understanding and ultimately targeting the epigenetic mechanisms that induce and maintain these tumor-propagating cell subsets is critical to improving GBM therapy and patient outcomes. DNA methylation is a reversible process and is partially mediated by the ten-eleven translocation (TET) family of enzymes which function as deoxygenases to catalyze the conversion of 5-methylcytosine (5mC) to 5-hydroxymethylcytosine (5hmC). Levels of 5hmC negatively correlates with glioma grade and loss of 5hmC correlates with poor prognosis of GBM patients, strongly suggesting that these enzymes activate tumor suppressing mechanisms. We show that TET2 loss associates with GBM stem cells and correlates with poor survival of GBM patients and identify a SOX2:miR-10b-5p:TET2 axis that represses TET2 expression and induces GBM cell stemness and tumor-propagating potential. In vivo delivery of a miR-10b-5p inhibitor normalizes TET2 expression and function to induce regression of orthotopic GSC-derived xenografts and prolongs animal survival. These findings highlight the importance of TET2 and 5hmC loss in Sox2-driven oncogenesis and their potential for therapeutic targeting.

Biography

I was born in Paraguay, South America and moved to the United States after completing high school to pursue a career in science in the United States of America. Early on in my career I took an interest in personalized medicine and molecular therapies. The long-term goal of my research is to understand the molecular mechanisms involved in regulating stemness and differentiation in neoplastic cells. We want to better understand Glioblastoma (GBM) by studying the molecular mechanism by which these cells acquire a stem-like phenotype and use this knowledge to develop new ways to treat and diagnose the disease

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Interleukin-1α mediates resistance to immunotherapy in melanoma

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Currently, immunotherapies are the most promising treatments for metastatic melanoma. However, most patients with melanoma do not respond or only partially respond to available immunotherapies. Results of preclinical and clinical studies suggest that both innate and acquired resistance play roles in melanoma resistance to immunotherapy. Identifying the factors and mechanisms involved in this resistance could help us design new strategies to avoid resistance and improve therapeutic efficacy.

Inflammation has long been associated with cancer initiation and progression; however, how inflammation causes immune suppression in the tumor microenvironment and resistance to immunotherapy is not well understood. Here, we show that both innate proinflammatory cytokine interleukin-1α (IL-1α) and immunotherapy-induced IL-1α make melanoma resistance to immunotherapy. In a mouse melanoma model, we found that tumor size was inversely correlated with response to immunotherapy. Large tumors had higher levels of IL-1α, Th2 cytokines, polymorphonuclear (PMN)-MDSCs, and regulatory T cells but lower levels of IL-12, Th1 cytokines, and activated CD4+ and CD8+ T cells. We found that therapy with rAd.CD40L (adenovirus-encoded CD40 ligand) increased tumor levels of IL-1α and polymorphonuclear (PMN)-MDSCs. Blocking the IL-1 signaling pathway significantly decreased rAd.CD40L-induced polymorphonuclear (PMN)-MDSCs and their associated PD-L1 expression in the tumor microenvironment and enhanced tumor-specific immunity. Similarly, blocking the IL-1 signaling pathway improved the anti-melanoma activity of anti-PD-L1 antibody therapy. Our study suggests that blocking the IL-1α signaling pathway may increase the efficacy of immunotherapies against melanoma.

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Alpha-fetoprotein complexes with toxins in fixing the cancer brakes

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Research scientist, Canada

The natural cancer brakes that serve the majority of us are apoptosis and the immune system. In cancer, both are broken. Apoptosis can be fixed by special toxins, and immune tolerance can be overcome with immunotherapy. Alpha-fetoprotein (AFP) is the delivery protein produced by the embryo. It crosses three layers of the human hemochorial placenta which separate the two circulations. In the blood, it binds polyunsaturated fatty acids (PUFAs) which the mother has to take by food as she does not produce them herself. AFP has a hydrophobic pocket that binds PUFAs stronger than blood proteins do. AFP delivers PUFAs to embryonic and myeloid-derived suppressor cells (MDSCs) through AFP receptor (AFPR)-mediated endocytosis like a shuttle. Meanwhile, MDSCs suppress embryo rejection by the mother's immune system.

MDSCs forms the protective tumor microenvironment (TME). As well as growing cancer cells, MDSCs can be hit by AFP-toxin preparations. The special toxins activate apoptosis in MDSCs and restore broken ones in cancer cells. MDSCs depletion by AFP-toxin preparations cancels the immune tolerance to tumors. MDSCs-targeted chemotherapy is a perspective cancer immunotherapy. Injectable AFP-toxin non-covalent complexes have demonstrated anticancer activity.

There has been a great desire for enabling the non-invasive delivery of therapeutics across mucosal surfaces. Oral porcine AFP complexes with toxins demonstrated anticancer activity, though they were not traced in the blood of mice. Gastrointestinal (GI) tract lymph nodes are lacking MDSCs and should have other sensitive immune-suppressive cells, which depletion eventually leads to distant metastases reduction. Hence, the cancer cells are not the only target for porcine AFP-toxin complexes, and immune suppressor cells "are more equal than others". The possible role of neonatal Fc receptor (FcRn) in transcytosis of oral porcine AFP complexes with toxins through intestinal enterocytes to GI tract lymph nodes is discussed.

Biography

Dr. Vladimir Nikolayevich Pak graduated from the Novosibirsk State University, received his Ph.D. from the Institute of Bioorganic Chemistry, Moscow, before working at the Novosibirsk Institute of Molecular Biology, Russia, and several biotechnological companies. He has over 35 years of experience in genetic engineering, virology, immunology, and biotechnology. His work focuses on the manufacturing of active pharmaceutical ingredients and the implementation of the drug candidates based on injectable (Reducin) and peroral (Aimpila) forms of AFP+toxin(s) non-covalent complexes. AFP+toxin non-covalent complexes, as well as AFP+toxin chemical conjugates, are likely to have both, direct cytotoxic effect on cancer cells and an immune checkpoint inhibition effect without toxicity. Dr. Vladimir N. Pak is the author of numerous scientific articles, patents, and a book.

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HPV Program in Colombia, A story of light and shadows

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Cervical Cancer (CC) is one of the leading causes of death in Colombian women and the third most frequent type of cancer in the female population with an annual incidence rate of 19.3 per 100,000. The vast majority (80%) of CC cases are attributable to the Human Papilloma Virus (HPV), which can be prevented through vaccination. Colombia went from being one of the leading countries in the world in HPV vaccination rates (with rates higher than 94% for both doses), to a poor immunization coverage levels of about 10.%. The reason for the dramatic decrease in vaccine rates is associated with a media spotlight caused by an episode of unknown etiology in the municipality of Carmen de Bolivar in 2014.,that was amplified by some local politicians and antivax groups Following the media scandal, health authorities opted to stop administering the vaccine in the schools and did not defend the program vigourosly. To change this situation, in 2018 the Colombian League Against Cancer, in association with the American Cancer Society , conducted a pilot project in the department of Arauca, frontier with Venezuela. The intervention consisted of a communication strategy aimed and developed with different population groups that have a role in the vaccination decisión process: girls and adolescents in vaccination age, parents of adolescents girls, educational institutions, health professionals and local media.

With this challenge in mind, the project had three main phases: research/indagation, design and testing, implementation and monitoring. Based on this route, the communications strategy was designed based on the social and cultural characteristics of the audiences involved in the decision to vaccinate against HPV, and specific communication channels and messages were created according to their characteristics. This strategy consisted of activities in the territory such as workshops, focus groups and interviews with key audiences in the territory, an information kit directed to girls and adolescents and also their parents, adittionally a plan for the dissemination of information in social networks and local media.

The results were very encouraging. Vaccination rate of the first dose increased significantly from 4.7% in 2017, to 41% in 2018, to 83% in 2019 and to 62% in 2020. Unfortunately, the covid-19 pandemic appeared and the schools were closed, and obviously the campaign was affected.

The Colombian League Against Cancer, is committed to continue promoting health, especially the HPV vaccination process, therefore, expects the replication of this communications strategy in different territories of the country, in order to increase vaccination against HPV in Colombia in Colombian girls and adolescents.

Biography

Dr Carlos Castro is a medical oncologist from University of British Columbia at Vancouver, Canadá. Has been the Director General of the National Cancer Institute of Colombia, Chairman of the Xaveriana University Cancer Center and the Institute of Oncology of Foundation Santa Fe of Bogota. Was appointed Viceminister of Health from Colombia. Member of the editorial board of “e-cancer”. At the present, is the Medical and Cientific Director of the Colombian League against Cancer.

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Clinical value of egfr copy number gain determined by amplicon-based targeted next generation sequencing in egfr mutated nsclc patients

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Background The clinical relevance of EGFR copy number gain in patients with EGFR mutated advanced non-small cell lung cancer (NSCLC) on first-line tyrosine kinase inhibitor (TKI) treatment has not been fully elucidated.
Objective We aimed to estimate EGFR copy number gain using amplicon-based next generation sequencing (NGS) data and explored its prognostic value.
Patients and Methods NGS data were obtained for 1566 NSCLC patients. EGFR copy number gain was defined based on an increase in EGFR read counts relative to internal reference amplicons and normal controls in combination with a modified z score ≥3.5. Clinical follow-up data were available for 60 patients treated with first-line EGFR-TKI.
Results Specificity and sensitivity of NGS-based EGFR copy number estimations were above 90%. EGFR copy number gain was observed in 27.9% of EGFR mutant cases and in 7.4% of the EGFR wild type cases. EGFR gain was not associated with progression free survival but showed a significant effect on overall survival with a adjusted hazard ratio (HR) of 3.14 (95% CI, 1.46-6.78, P=0.003). Besides EGFR copy number gain, osimertinib treatment in second or subsequent lines and presence of T790M at relapse revealed significant effects in a multivariate analysis with adjusted HR of 0.43 (95% CI, 0.20-0.91, P=0.028) and 0.24 (95% CI, 0.1-0.59, P=0.001), respectively.
Conclusions Pre-treatment EGFR copy number gain determined by amplicon-based NGS data predicts worse OS in EGFR mutated patients treated with first-line EGFR-TKI. T790M at relapse and subsequent treatment with osimertinib predict longer OS.

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The role and mechanism of the degree of immune cell infiltration on the prognosis and immunotherapy of osteosarcoma

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Background: Immunotherapy in osteosarcoma has yielded unsatisfactory results. Hence, identifying a marker for improving the efficacy of immunotherapy and prognosis of osteosarcoma would benefit such population. This study explored the role and mechanism of immune cell infiltration in the tumor microenvironment (TME) on the prognosis and immunotherapy of osteosarcoma.

Methods: This study devised an immune score (IS) based on ssGSEA to evaluate the relative content (RC) of immune cell in osteosarcoma. The Survminer R package was used to analyze the survival and optimal cut-off point for IS and classify the osteosarcoma into high immune cells infiltrating osteosarcoma (HIOS) and low immune cell infiltrating osteosarcoma (LIOS). The relationship between IS and the RC of immune cells was evaluated using Spearman correlation analysis. The Chi-square test compared the recurrence and metastasis rates of osteosarcoma between the two groups. TIDE and GenePattern were used to predict osteosarcoma response to immunotherapy. Using the Kruskal-Wallis test, Spearman correlation analysis, and GSEA, the mechanism of the degree of immune cell infiltration affecting the prognosis and immunotherapy of osteosarcoma was analyzed.

Results: IS reflects the degree of immune cell infiltration in the osteosarcoma TME. HIOS has a lower recurrence rate, metastasis rate, and better immunotherapeutic effect than LIOS. The RC of M1-type macrophages and activity of immune-related pathways positively correlated with IS, while the immune escape and copy number variation (CNV) negatively correlated with IS.

Conclusion: Hyperimmune infiltration promotes osteosarcoma prognosis and immunotherapy associated with a high level of M1-type macrophages, low immune escape, immune-related pathway activity, and low CNV.\

Biography

Qingshan Huang is a PhD student at Peking University, majoring in bone tumors.

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Crispr/cas9 engineered car-t cells therapy: game changer in cancer therapeutics

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CRISPR is the Noble prize winner customized gene editing tool that has taken the research world by storm being the efficient genome editor to fix cancer as well as several hereditary disorders as compared to other gene editing tools like Zinc-finger nucleases (ZFNs), Transcription activator-like effector nucleases (TALENs). CRISPR becomes the game changer in cancer therapeutics after the two recently published path-breaking clinical trials that reported the safety and efficiency of using CRISPR/Cas9 edited, patient-derived T cells (CAR-T cells) to treat refractory cancers.

This article discusses the literature about the mechanism of CRISPR gene editing used in pre-clinical and clinical trials in oncology, focusing mostly on PD-1 knockout CAR-T cell therapy which provides the way for CRISPR to be the most favored technique to help treating cancer and other diseases in future. It also discusses the shortcomings of CRISPR such as unintended on-target and off-target cuts, embryonic germ-line editing and the recent advances that overcome the hurdles and further increase the efficiency of this technique. Till date only somatic cell editing is ethically approved and further research is required to support germ-line editing in humans for the treatment of genetic disorders.

Biography

Afreen Khan obtained her M.SC degree in Medical Biochemistry at Integral Institute of Medical Sciences and Research, Lucknow, India. She is currently in her third-year of her PhD in Medical Biochemistry at Era's Lucknow Medical College, India. Her main research interest centers early diagnosis and treatment of cancers. Her current research is a molecular analysis of various genes that participates in the progression of cancer to have a better understanding of the molecular pathways involved in cancer. Future prospects of her research focusses around development of efficient drug or gene therapy to as a potential cancer therapeutic.

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Health related quality of life and its correlates among people with depression attending outpatient department in ethiopia: a cross sectional study

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Background

Depression is a common mental disorder negatively affects the cognitive, emotion, behavior, functionality and quality of life of people. Poor quality of life results in high rates of relapse, inability to perform occupational and social activities, impaired future outlook, and increases overall health care related costs. However, there is no available evidence regarding the health related quality of people with depression in Ethiopia. Therefore, evaluating the quality of life of people with depression is crucial.

Objective

The aim of this study was to assess the health related quality of life and its correlates among people with depression at Amanuel Mental Specialized Hospital, Addis Ababa, Ethiopia.

Methods

An institutional based cross-sectional study was conducted from May 1st to 30th, 2018. A randomly selected 394 clients with depression were participated in this study. Health related quality of life was measured using world health organization quality of life brief. The collected data were coded and entered to SPSS version 20 for analysis. Step wise multiple linear regression analysis was used to identify the correlates of quality of life and the strength of the correlation was measured by β coefficient with 95% confidence interval.

Results

The mean (±SD) scores of quality of life of people with depression were 41.3±7.5, 42.8±8.2, 38.9±8.9 and 41.8±6.5 for physical, psychological, social and environmental domains, respectively. The Multiple regression analysis showed that age of respondents, age of onset of depression, perceived stigma, living arrangement, social support level and duration of illness were statistically significant predictors of health related quality of life of people with depression in all or at least one domain of quality of life.

Conclusions

This study revealed that nearly half of study participants scored below the mean score in each domain of health related quality of life. This demonstrates a need for improving the quality of life of people with depression through the integration of a positive mental health approach and bio-psychosocial view together with the pharmacological treatments of depression. Moreover, strengthening social support, early identification and treatment of depression and prevention of stigma are also highly recommended to improve the quality of life of people with depression

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