

4th Global Conference on Cancer Science and therapy
 9th World Summit on Virology, Microbiology & Infectious Disease
 6th International Conference on Biomedical Biopharma and Clinical Research
 October 11, 2022 | Webinar

Intelligence strategy: Identifying molecular signatures and network dynamics of pathogenic microbes and complex host interactions

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Microbes play a complex integral role in every major disease, yet are largely unrepresented in traditional models, where the focus relies on narrowly focused symptoms-based markers, rather than the fully articulated microbial pathophysiologies that ultimately give rise to each individual's condition in real life.

We need to go beyond the classical definition of infection, and instead appreciate the complex role that each microbe plays in shaping every human condition, whether disordered or optimal, especially through the full array of proteins that each organism encodes for with the very purpose of interacting with or acting upon its host.

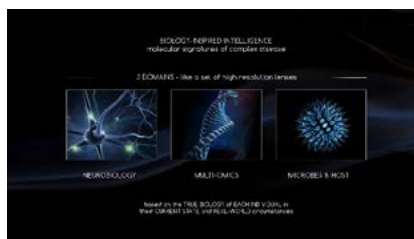
Microbes are neither restricted to designate body parts, or the short list of symptoms we have classically deemed relevant. What's more is that the full array of proteins that microbes encode for have not only the ability to migrate throughout the brain and body, they by their very design remodel their host's biology, beginning with alterations of genetic expression and transcription, with considerable metabolic, cellular, systemic, and cascaded network effects.

This yields intelligence of incredible value, by portraying highly detailed molecular signatures of microbes known to

play causal roles in pathophysiologies of a range of diseases such as Alzheimer's and other forms of dementia, multiple sclerosis and other forms of autoimmunity, psychiatric conditions ranging from PANS/PANDAS to schizophrenia, diabetes, cardiovascular diseases, and host of others.

Finally, a considerable body of high-quality literature exists today, making this intelligence not only relevant, but also actionable.

This presentation is to introduce an intelligence model that can be used to identify microbial signatures pertinent to a range of high impact disease states, with applications for precision medicine, especially drug discovery, clinical intelligence, and advanced diagnostics.



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Role of TBX3 on Epithelial Mesenchymal Transition (EMT) in BRAF mutant negative malignant melanoma

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TBX3 is overexpressed in a broad range of epithelial and mesenchymal-derived cancers. In melanoma, there is a link between oncogenes B-RAF, transcriptional repressor TBX3, and epithelial to mesenchymal transition (EMT). EMT is related to the upregulation of E-cadherin regulators. TBX3 is an additional regulator in BRAF mutation-positive melanoma. TBX3 may act as a significant regulator of oncogenic B-Raf signaling pathways and a promoter of metastasis in B-Raf mutation-positive melanoma. There is an essential correlation between EMT, TBX3, and EMT activation, and BRAF mutations. The mutant B-RAF can up-regulate TBX3 expression in malignant melanoma, which inhibits E-cadherin levels and promotes tumor cell invasion and metastasis. It is more rational to analyze the role of TBX3 in melanoma with low-frequency B-RAF mutation or no BRAF protein and whether it regulates epithelial-mesenchymal transition (EMT). We purposed to examine the effect of TBX3 on EMT in BRAF mutant negative melanoma. The functional loss of E-cadherin repression has been an assay mark of epithelial-to-mesenchymal- transition and several EMT transcription factors have been identified as the cause of this repression. EMT has been allied with up-regulation of the E-cadherin controllers, but it has acknowledged TBX3 as a supplementary

tissue-restricted repressor of E-cadherin in melanoma. TBX3 may act as a significant controller of the oncogenic B-Raf signaling pathway and as a promoter of metastasis in B-RAF-mutant melanomas. In our research, we examined the role of TBX3 in melanoma without or low-frequency BRAF mutation and show up the first time that TBX3 still promotes EMT in BRAF mutant-negative cell lines. We have further discovered, with the support of histological and cytological experiments, the correlation between the expression of TBX3, E-cadherin, N-cadherin, vimentin, and the prognosis of the patients. Therefore, further analysis and research in the future will make it achievable to cure melanoma. The purpose of this study was to examine the role of TBX3 in melanoma without or low-frequency B-RAF mutation and the effect of TBX3 on epithelial-Mesenchymal transition (EMT) in BRAF mutant negative cells. Our results verify, TBX3 is a key molecule regulating epithelial-to-mesenchymal transition (EMT) in malignant melanoma, and TBX3 exhibits a positive regulatory role in promoting EMT regardless of the presence or absence of BRAF mutants. For the first time, we have suggested that TBX3 can still do its function of promoting tumor evolution without BRAF mutations.

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Fluorescence enhancement strategy for evaluation of the minor groove binder DAPI to complementary ssDNA sequence including telomere mimics in (ssDNA@DAPI/LDH)_n ultrathin films

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Nucleic acids, the hereditary material of living beings, perform a vital role in major life activities including growth, heredity, and variation. The genetic information is coded in specific nitrogenous bases, which are the key players in genetic transformation and identification of hereditary diseases. Telomeres are specialized DNA structures present at the end of eukaryotic chromosomes, consisting of repetitive non-transcribed sequences (TTAGGG) and some binding proteins. They protect the chromosomal ends from sticking to each other, and play a key role in the maintenance of genomic integrity. Currently, a number of methods are reported for nucleic acids and telomeres detection but they are not easy going. Therefore, development of sensitive and fast DNA biosensors to measure the nucleic acids and telomeres sequences is of great importance. This paper describes a systematic study on the preparation

of simple ultrathin films (UTFs) composite of fluorescent dye 4',6-diamidino-2-phenylindole (DAPI), having binding properties in the minor groove of AT-rich DNA segments, blending with single-stranded DNA (ssDNA) and incorporation into layered double hydroxides (LDH) nanosheets using layer-by-layer self-assembly method. The resulting UTFs exhibits excellent sensitivity, selectivity and reversibility for long complementary ssDNA sequence on the basis of DNA hybridization and fluorescence enhancement strategy. The dynamic range was 3–20 µg/mL with a detection limit of 20 µg/mL. This new (ssDNA @DAPI/ LDH)_n UTFs, could be an efficient hybrid composite with potential applications in the field of bioluminescent sensing materials.

Keywords: ssDNA, Telomere, DAPI, DNA-biosensor, Layered double hydroxide, Layer by layer co-assembly, Hybridization, Fluorescence enhancement.

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