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Application of information-theoretical analysis to develop a molecular beacon-based microfluidic chip technology for cancer diagnostics

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The ability for clinicians to develop personalized therapeutics based on a patient's genetic composition is a challenge, particularly in cancer diagnostics. In many cancers, the genetic composition can vary significantly within the same tumor sample. This not only complicates developing an optimal treatment for a cancer, but also challenges cancer diagnostics as well. Currently, personalized medicine in cancer is based on high-cost genetic testing technology and most of the information acquired is based on correlation studies. Targeted therapy agents are increasingly available for clinical applications, but have produced disappointing results when tested in clinical trials, indicating that there are many challenges that must be addressed to advance this field. In this proposal, we introduce a first-in-the field technique, surprisal analysis (SA), that will revolutionize and reinvigorate the quest for personalized medicine in cancer therapeutics. Furthermore, SA is extended beyond just a theoretical approach by developing targeted microfluidic biotechnology for clinical use.

Biography

Sohila Zadran completed her undergraduate studies in Molecular Cell Biology, with an emphasis Neurobiology at the University of California, Berkeley. She received her doctoral degree in Neuroscience, with an emphasis in Neural Engineering, at the University of Southern California and completed her post-doctoral training at the California Institute of Technology. She is currently a cancer scientist at the University of California, Los Angeles. She is also the founder of Agarionan Corp, a biotech firm affiliated with the California Institute of Quantitative Biology (QB3), a company dedicated to the development of novel cancer biotechnologies.

Treatment of established dermal murine B16F10 melanoma with an attenuated strain of *Toxoplasma gondii* eliminates the treated tumor and stimulates systemic anti-tumor immunity

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While the surgical removal of dermal melanoma cures that lesion, it does nothing to stimulate antitumor immunity that could potentially identify and eliminate occult metastatic disease. An immune based treatment that eliminates the primary dermal melanoma could also potentially generate systemic immunity that will protect against metastatic disease. We have utilized an attenuated strain of *Toxoplasma gondii* (cps) to break tumor-mediated immunosuppression, stimulate an antitumor immune response that eliminates the dermal tumor, and generate systemic antitumor immunity that leads to rejection of subsequent dermal or intravenous challenges with B16F10. *T. gondii* is an obligate intracellular eukaryotic parasite that infects virtually any mammalian species. The cps strain is a uracil auxotroph that can be grown *in vitro* but is unable to replicate *in vivo*. Despite its lack of infectivity, it enters cells and stimulates a strong T-cell mediated immune response characterized by long lasted CD8 effector cells. The presence of cps in the tumor microenvironment modifies the phenotype of tumor infiltrating leukocytes, and along with the expected anti-Toxoplasma immune response there is antigen-spreading so that tumor antigens are responded to and the immune system recognizes subsequent tumor challenges when there is no associated cps. The treatment requires CD8 and NK cells for efficacy but does not require CD4 cells. The treatment also requires that the host express IL-12 and Ifn-g. The treatment requires live cps for efficacy and is effective in mice that are latently infected with another strain of *T. gondii*, so it could function in the high percentage of humans with latent *T. gondii* infection and an established immune response against *T. gondii*.