A practical example of model-based biomarker discovery by quantitative proteomics

The first documented case of cancer dates back to the Egyptians in 1600 BCE. A number of seminal discoveries since the ancient Egyptians have redefined our understanding of cancer. In fact, over the past several years, Cancer researchers have begun to reclassify tumors based on the molecular signalling pathways that cause malignant transformation. As a result, medical oncologists can prescribe drugs that specifically target the aberrant signalling pathway irrespective of the location of the Tumor. Modern cancer therapeutics is becoming targeted rather than systemic. This has brought upon a significant paradigm shift in medical treatment decisions and spurred the precision medicine era. Cancer cells must maintain a malleable phenotype in order to adapt to the selective pressures of the microenvironment and therapeutics. The cells’ ability to modulate its proteome in the context of these selective pressures is an essential component of Tumor progression. Yet, to date, there has not been a systematic evaluation of the proteomic landscape associated with cancer progression, response to therapy and or resistance. We have demonstrated that the proteome of cancer cells which are sensitive to EGFR targeted drugs are quantitatively different from resistant cells. The shift from a sensitive to resistant phenotype was accompanied by a dramatic shift in the proteome. This finding suggests that cancer cells are able to rapidly alter their proteome signature in order to adapt to a selective pressures. Since the signature of resistant cells is different than the parental precursors, the possibility of discovering unique biomarkers of resistance is a reality.

Biography

Kian Kani has completed his Postdoctoral Research at Cedars Sinai Medical Center where he utilized proteomics in order to study therapeutic response to HER axis targeted drugs in cancer and during his Graduate studies at UCLA utilized molecular and bioinformatic tools in order to study control mechanisms regulating aberrant activity of the Human Epidermal Growth Factor Receptor (HER) family in cancer. His focus at USC is to identify and characterize novel cancer biomarkers and his goal is to enhance patient outcome by empowering physicians with the necessary tools for personalized medicine.

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