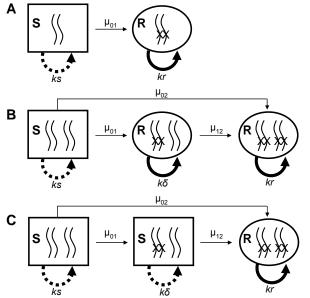
Intellectual Property (Non-confidential)



Predicting Drug Resistance in Cancer



DESCRIPTION

There are several well-documented instances of cancers that can develop acquired resistance to a targeted therapeutic which were initially successful in the treatment of cancer. For example, targeted therapeutics such as the BCR-ABL tyrosine kinase inhibitor imatinib for chronic myeloid leukemia (CML) are widely used, but acquired drug resistance limits their broader success. Cancer cells are prone to mutation, and as these mutations accumulate over time some cells may acquire mutations that protect against therapeutic treatments. Acquired drug resistance is a phenomenon akin to natural selection, in which drug resistant cells give rise to tumors that are no longer drug-responsive. Because these drug-resistant cells begin as a small subpopulation, continued treatment may still appear to be effective; however as the drug-resistant population grows, and mutations continue to accumulate, the cancer becomes exceedingly difficult to treat.

This technology monitors the development of these mutations and allows for clinicians to predict drug resistance and change therapies before drug resistance actually manifests. Additionally, anti-cancer drugs have significant side effects, and if a drug is no longer conferring benefits, continued treatment does more harm than good. By informing clinicians on the optimal time to switch drugs, unnecessary side-effects can be avoided, the risk of developing multi-drug resistance is significantly reduced, and an effective treatment regimen will allow for continuous treatment that does not permit time for the tumor to recover.

KEY ASPECTS

- Identifies when genes targeted by a cancer drug have developed mutations that lead to acquired drug resistance.
- Example genes: PDGFR-, PDGFR-, EGFR, VEGFR, VEGFR1, VEGFR2, VEGFR3m HER-2 (also known as ErbB2), KIT, FLT3, c-MET, FGFR, FGFR1, FGFR3, c-FMS, RET, ABL, BCR-ABL, ALK, ARG, NTRK1, NTRK3, JAK2 and ROS
- Utilizes Fluorescence in situ hybridization (FISH) analysis, PCR, and Sanger sequencing

INTELLECTUAL PROPERTY

Title	US Patent Application	Filed
Acquired drug resistance of cancer vs. de novo genetic mutations	13/491,486	6/7/2012

<u>CONTACT</u>

Matthew Grunseth, M.B.S. Senior Manager, Office of Technology Licensing Telephone: (626) 471-7221 | Email: <u>mgrunseth@coh.org</u>

This material is a summary of public domain and non-confidential City of Hope information. Additional material may be disclosed under a confidentiality agreement.