Cancer treatments could be guided by evolving ratio of mutated/normal driver genes

Choosing among cancer treatments increasingly involves determining whether tumor cells harbor specific, mutated “oncogenes” that drive abnormal growth and that may also be especially vulnerable or resistant to particular drugs. But according to a new study led by UC San Francisco researchers, in the case of the most commonly mutated cancer-driving oncogene, called *KRAS* (pronounced “kay-rass”), response to treatment can change as tumors evolve, either when a normal copy of the gene from the other member of the matched chromosome pair is lost, or when the cancer cells evolve to produce additional copies of the mutated form of the gene.

The identification of distinctive abnormalities in DNA sequences within the genomes of tumor cells from biopsy specimens is becoming a more common aid to help guide cancer treatment decisions, and the authors of the new study, published in the Feb. 23, 2017, edition of *Cell*, said their discovery of *KRAS* “imbalances” that emerge over time could be added to a growing list of genetic characteristics that may be clinically valuable.

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“In evaluating treatment response in clinical trials going forward, it will be important to understand not only whether a *KRAS* mutation is present, but also how much mutant *KRAS* is present, and whether there is a loss of the normal copy of the *KRAS* gene,” [first author Michael] Burgess said.

[Study can be found here, behind paywall.]

The GLP aggregated and excerpted this blog/article to reflect the diversity of news, opinion, and analysis. Read full, original post: Genetic ‘balance’ may influence response to cancer treat