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THE CHALLENGE

It continues to be a challenge to determine whether or not an individual patient will benefit from a potential treatment option. Scientists and physicians have the "parts list" for diseases like cancer. For many diseases, they also know how the parts fit together. However, it remains a challenge to understand how the system of parts works to the point at which one can reliably predict whether or not a patient will respond to treatment.

THE APPROACH

Edward Stites uses mathematical and computational models to study the behaviors of genetic signaling networks implicated in cancer. Signaling proteins operate within large, complex networks and even when the roles of individual proteins are well understood, the behavior of the network of proteins can be difficult to predict. Stites' methods reveal how these networks promote cancer and respond to treatment. Mathematical models help illuminate the unknowns. They help formulate new hypotheses for experimental testing. The incorporation of data-driven models into cancer research should enable quicker and more efficient progress.

THE INNOVATIONS AND DISCOVERIES

- Stites developed a mathematical model of the RAS gene signaling pathway that revealed multiple unexpected behaviors by the most common cancer-activating mutations in human cancer. This work also demonstrated how mathematical models can be used to study cancer-promoting mutations.
- It has become clear that not all mutations to the same gene behave the same way. Stites' models are capable of predicting the behaviors of different mutations to the same gene, including different responses to treatment. This suggests a role for these computational approaches in personalized cancer medicine.
- Stites' modeling found that certain mutations (such as mutations in the NF1 tumor suppressor gene) amplified the effects of other gene mutations, suggesting that certain combinations of mutations work together to drive cancer. These results were borne out with experiments using cancer cells and were observed in sequenced cancer genomes.

For more information, please visit:
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