

Gene-specific cancer treatment is common, but there are still some kinks

Elaine Mardis and her colleagues first encountered 39-year-old Lucy (not her real name) in 2010 at the Genome Institute at Washington University in St Louis, Missouri. Lucy had been referred there after a confusing leukaemia diagnosis. Her doctors thought she had a subtype of the disease called acute promyelocytic leukaemia (APL) — one of the most treatable forms.

Mardis, co-director of the Genome Institute, is involved in a university initiative to use whole-genome sequencing and other analyses to launch precision attacks against difficult cancers.

Researchers have learned enough about cancer to know that the way it has been tackled for decades — with cocktails of chemotherapeutic drugs that indiscriminately hit populations of rapidly growing cells — is effective only up to a point. They believe that if they can find the key genetic mutations that drive a particular cancer's growth, they will be able to target the tumour more selectively and with fewer toxic side effects. But they don't yet know enough about which genetic mutations drive a given cancer, let alone how to interrupt the aberrant cellular pathways that result.

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