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Cancer genomic epidemiology

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The current paradigm in oncology is that one needs to look only at the tumor to solve cancer. The Vogelstein hypothesis holds that normal tissue acquires mutations in oncogenes, and that cancer-prone patients simply fail to repair their DNA well enough. The tumor is believed to be mutated, alien and unresponsive tissue that must be killed. There are many ways to get cancer, and defective DNA repair is one of the rare ways. Sequencing tumor DNA has shown that each tumor has a surprisingly small number of private mutations not shared by tumors from other patients with the same common cancer diagnosis. Each tumor is a snowflake. Oncology now proposesto treat each tumor based on its gene expression, or DNA mutations, in an exaggerated version of "personalized medicine" that promises to make cancer treatment even more expensive. However, initial results of basing treatment on tumor gene expression have been rather disappointing. Twenty years ago, we found that tissue gene expression cannot identify the earliest steps in a biological process like compensatory renal hypertrophy or kidney failure. Instead, variation in germline DNA (technically, somatic cell DNA) represents the earliest possible cause for any disease. Given the huge amplification characteristic of biological pathways, the only hope for inhibiting a pathway is to block its earliest steps. This is probably why treatment based on tumor gene expression doesn't work. We have found a large number (>2,000) of shared variants (SNPs) in the germline DNA of cancer patients for each of six common cancers in whites: breast, colon, lung, ovary, pancreas, and prostate. These SNPs represent the earliest step in tumorigenesis, and are likely driving the tumor decades later, when it is detected clinically. We estimate that as many as 5 000-10 000 genes, i.e. one third of the genome, may be involved in tumorigenesis. A likely cellular program this large is differentiation. We now believe that the common form of cancer, which occurs after the age of 50, and affects one in three people, involves a common cellular pathway, differentiation. Cancer represents a physiologic response to tissue atrophy ("apoptosis"), gone slightly wrong, simply because oncogenes are over-expressed, and tumor suppresors are under-expressed. While the SNPs we've found make it possible to predict which tumor a patient will get, the many tumorigenesis genes we've found promise more effective, non-toxic "differentiation therapy."

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