Mutational load distribution analysis yields metrics reflecting genetic instability during pancreatic carcinogenesis. [1]

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Abstract	Considering carcinogenesis as a microevolutionary process, best described in the context of metapopulation dynamics, provides the basis for theoretical and empirical studies that indicate it is possible to estimate the relative contribution of genetic instability and selection to the process of tumor formation. We show that mutational load distribution analysis (MLDA) of DNA found in pancreatic fluids yields biometrics that reflect the interplay of instability, selection, accident, and gene function that determines the eventual emergence of a tumor. An in silico simulation of carcinogenesis indicates that MLDA may be a suitable tool for early detection of pancreatic cancer. We also present evidence indicating that, when performed serially in individuals harboring a p16 germ-line mutation bestowing a high risk for pancreatic cancer, MLDA may be an effective tool for the longitudinal assessment of risk and early detection of pancreatic cancer.
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