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Sommario/riassunto	Since the discovery of the Warburg effect in the 1920s cancer has been tightly associated with the genetic and metabolic state of the cell. One of the hallmarks of cancer is the alteration of the cellular metabolism in order to promote proliferation and undermine cellular defense mechanisms such as apoptosis or detection by the immune system. However, the strategies by which this is achieved in different cancers and sometimes even in different patients of the same cancer is very heterogeneous, which hinders the design of general treatment options. Recently, there has been an ongoing effort to study this phenomenon on a genomic scale in order to understand the causality underlying the disease. Hence, current "omics" technologies have contributed to identify and monitor different biological pieces at different biological levels, such as genes, proteins or metabolites. These technological capacities have provided us with vast amounts of clinical data where a single patient may often give rise to various tissue samples, each of them being characterized in detail by genomescale data on the sequence, expression, proteome and metabolome level. Data with such detail poses the imminent problem of extracting meaningful interpretations and translating them into specific treatment options. To this purpose, Systems Biology provides a set of promising computational tools in order to decipher the mechanisms driving a healthy cell's metabolism into a cancerous one. However, this enterprise requires bridging the gap between large data resources,

mathematical analysis and modeling specifically designed to work with the available data. This is by no means trivial and requires high levels of communication and adaptation between the experimental and theoretical side of research.
