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Short Communication

Mutasignature Intelligence: Decoding Evolutionary Mutation Patterns in Carcinogenesis

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DESCRIPTION

Mutasignature intelligence refers to the systematic interpretation of mutation patterns as structured biological information that reflects underlying mechanisms of carcinogenic evolution. Instead of viewing mutations as isolated random events, this framework considers them as recognizable signatures generated by specific biological processes, environmental exposures and cellular responses to stress. These mutational signatures encode information about tumor origin, progression pathways and adaptive mechanisms that drive malignancy [1].

Over time, these insults leave characteristic mutational imprints that reflect the nature of the damaging agent and the cellular context in which the damage occurred. Mutasignature intelligence seeks to decode these imprints to reconstruct the evolutionary history of tumor development.

One of the key principles of mutasignature intelligence is that different carcinogenic processes produce distinct mutation patterns. Oxidative stress tends to generate specific base substitutions, while exposure to environmental toxins produces distinct chemical modification patterns [2]. Replication stress introduces structural variations and chromosomal instability, whereas deficiencies in repair mechanisms lead to accumulation of widespread genomic errors.

Cellular repair systems play a central role in shaping mutational signatures. When repair pathways function efficiently, most damage is corrected before it becomes permanent. However, when these systems become overwhelmed or dysfunctional, errors are preserved and propagated through cell division. The resulting mutation patterns reflect both the type of damage and the efficiency of repair mechanisms, creating a combined signature of exposure and vulnerability.

Environmental influences strongly contribute to mutational signature formation. Tobacco exposure, ultraviolet radiation, industrial pollutants, dietary carcinogens and chronic inflammation each produce distinct genomic alterations [3-5]. These alterations accumulate over time, creating a layered record of environmental history within tumor genomes. Mutasignature intelligence uses these patterns to trace the external factors contributing to carcinogenesis and identify high-risk exposure profiles.

Metabolic dysfunction also influences mutation patterns by increasing internal stress within cells. Elevated oxidative metabolism generates reactive intermediates that damage cellular components, including genetic material [6]. Over time, this leads to predictable mutation distributions that correlate with metabolic imbalance. Tumor cells adapt to these conditions by selecting mutations that enhance survival under oxidative stress, further shaping the mutational landscape.

Immune system interactions further shape mutational landscapes. Immune surveillance eliminates highly immunogenic tumor cells, indirectly selecting for clones with less detectable mutation profiles. Over time, this selective pressure alters the overall mutational signature of the tumor, favoring variants that evade immune recognition [7-10]. This process contributes to immune escape and long-term tumor persistence.

Technological advances in high-throughput sequencing have made it possible to analyze mutational signatures at unprecedented resolution. Large-scale genomic datasets reveal recurrent patterns across different cancer types, enabling classification of tumors based on signature composition. These classifications provide insights into tumor origin, progression rate and likely therapeutic response.

Mutasignature intelligence also has important clinical implications. Identifying specific mutational patterns can guide treatment selection by revealing underlying mechanisms of tumor growth. For example, tumors dominated by repair deficiency signatures may respond better to certain targeted therapies. Similarly, tumors influenced by environmental carcinogens may require different therapeutic strategies compared to those driven primarily by metabolic dysfunction.

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In conclusion, mutasignature intelligence provides a structured framework for interpreting mutation patterns as biologically meaningful information rather than random genetic noise. By decoding the signatures left behind by environmental exposure, metabolic stress and repair dysfunction, this approach offers deep insight into the mechanisms driving carcinogenesis. Its integration with sequencing technologies and computational modeling enhances the ability to predict tumor behavior, improve diagnostic accuracy and develop more targeted therapeutic strategies aimed at disrupting the evolutionary logic of cancer development.

REFERENCES

1. Borgonetti V, Benatti C, Governa P, Isoldi G, Pellati F, Alboni S, et al. Non-psychotropic Cannabis sativa L. phytocomplex modulates microglial inflammatory response through CB2 receptors-, endocannabinoids-, and NF-κB-mediated signaling. Phytother Res. 2022;36(5):2246-2263.
2. Shamsudin NF, Ahmed QU, Mahmood S, Shah SA, Sarian MN, Khattak MM, et al. Flavonoids as antidiabetic and anti-inflammatory agents: A review on structural activity relationship-based studies and meta-analysis. Int J Mol Sci. 2022;23(20):12605.
3. Abid R, Ghazanfar S, Farid A, Sulaman SM, Idrees M, Amen RA, et al. Pharmacological properties of 4′, 5, 7-trihydroxyflavone (apigenin) and its impact on cell signaling pathways. Molecules. 2022;27(13):4304.
4. Sharifi-Rad M, Nazaruk J, Polito L, Morais-Braga MF, Rocha JE, Coutinho HD, et al. Matricaria genus as a source of antimicrobial agents: From farm to pharmacy and food applications. Microbiol Res. 2018;215:76-88.
5. Jucá MM, Cysne Filho FM, de Almeida JC, Mesquita DD, Barriga JR, Dias KC, et al. Flavonoids: Biological activities and therapeutic potential. Nat Prod Res. 2020;34(5):692-705.
6. Niedzwiecki A, Roomi MW, Kalinovsky T, Rath M. Anticancer efficacy of polyphenols and their combinations. Nutrients. 2016;8(9):552.
7. Salehi B, Venditti A, Sharifi-Rad M, Kręgiel D, Sharifi-Rad J, Durazzo A, et al. The therapeutic potential of apigenin. Int J Mol Sci. 2019;20(6):1305.
8. Ganapathi R, Kumar RR, Thomas KC, Rafi M, Reddiar KS, George PS, et al. Epstein-Barr virus dynamics and its prognostic impact on nasopharyngeal cancers in a non-endemic region. Ecancermedicalscience. 2022;16:1479.
9. Tsai YC, Hohmann J, El-Shazly M, Chang LK, Dankó B, Kúsz N, et al. Bioactive constituents of *Lindernia crustacea* and its anti-EBV effect via Rta expression inhibition in the viral lytic cycle. Journal of ethnopharmacology. 2020;250:112493.
10. Vágvölgyi M, Girst G, Kúsz N, Ötvös SB, Fülöp F, Hohmann J, et al. Less cytotoxic protoflavones as antiviral agents: protoapigenone 1′-O-isopropyl ether shows improved selectivity against the Epstein-Barr virus lytic cycle. Int J Mol Sci. 2019;20(24):6269.