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Gynecological Cancer's Somatic Mutations: A Short Communication

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INTRODUCTION

Understanding the causes of the mutations that lead to human malignancies is a continuous goal in cancer research, because it is natural to anticipate that if the causes of the diseases are discovered, there will be viable interventions to lessen the risk. As a result, efforts to limit tobacco-related mutagens and UV light exposure, as well as immunizations to prevent oncogenic virus-induced carcinogenesis, have reduced cancer occurrences [1].

What are the origins of the driver mutations that are crucial in the early stages of carcinogenesis besides a small number of environmental agents? Because a vast number of cells in a human tumour have comparable genetic alterations, indicating a shared lineage, cancer is definitely a clonal illness. Secondary, tertiary, and other genetic mutations occur as the tumour expands in size, resulting in a complex mutation mosaic. If a mutation develops in a gene that impacts the fidelity of DNA replication or DNA repair, this genetic diversity is increased even more [2].

ABOUT THE STUDY

The number of normal stem cells in a tissue, the number of times the stem cells divide, and a low incidence of random DNA polymerase errors that occur during each cell division have recently been deemed stochastic and correlated with the number of normal stem cells in a tissue, the number of times the stem cells divide, and a low incidence of random DNA polymerase errors that occur during each cell division. While the quantitative proportion of the background mutation rate owing to random polymerase misincorporation mistakes is still being debated, The number of normal stem cells in a tissue, the number of times the stem cells divide, and a low incidence of random DNA polymerase errors that occur during each cell division have recently been deemed stochastic ("bad luck") and correlated with the number of normal stem cells in a tissue, the number of times the stem cells divide, and a low incidence of random DNA polymerase errors that occur during each cell division. While the quantitative contribution of the background mutation rate due to random polymerase misincorporation errors is debatable, it is logical that somatic mutation rates reflect to some extent stem cell replication rates due to random polymerase misincorporation errors, especially since virtually all non-canonical base pairs require DNA replication to become an inheritable mutation [3].

The nonsense somatic mutation pattern in two tumour suppressor genes is presented here as an exception. We also examine the relative mutagenicity of CpG sequences due to rates of 5-methylcytosine (5-mC) deamination vs. the mistake rate during DNA polymerization. The evidence strongly suggests that selective hydrolytic deamination of 5-mC at CGA Arg codons causes much more non-sense mutations in tumour suppressor genes than random polymerase mistakes. Assuming that mutations caused by polymerase mistakes are random and those nonsense mutations that stop protein synthesis are generally identical; nonsense mutations should be evenly distributed throughout the gene [4].

FUTURE PERSPECTIVE

The ability to create cancer prevention measures depends on whether mutations are mostly random due to stochastic polymerase mistakes and/or exogenous and endogenous DNA damaging agents, or if they are predictable. The argument presented here on the well-known intrinsic risk associated with 5-mC at CpG in Arg CGA codons complements Tomasetti &

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Vogelstein's proposal that random polymerase errors and the number of stem cell divisions play a role in carcinogenesis [5].

CONFLICT OF INTERESTS

No conflict of interest by author.

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Not applicable.

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