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Editorial

Future of Cancer Treatment and Genomic Toxicity

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INTRODUCTION

Cancer order is sometimes unstable and, therefore, constantly acquires changes at each the ester sequence additionally as chromosomal levels [1-3]. in progress genomic changes, that confer new characteristics to the recipient cells, underlie their progression to advanced sickness states as well as acquisition of drug resistance and treatment failure. Information from our laboratory have shown that accrued number of mutations correlates with poor survival of metastatic tumor patients [2]. One in all the implications of genomic instability and increased modification burden can even be the formation of a lot of neoantigens which facilitate recognition of cancer cells as non-self by immune system. However, continuing acquisition of genomic changes can even give new characteristics to cancer cells which can facilitate them escape immune police work [4]. In step with unstable order, cancer cells show variety of genomic aberrations as well as accrued levels of spontaneous deoxyribonucleic acid breaks. mistreatment muscle system carcinoma and myeloma as model systems, we've got shown that homologous recombination, the foremost precise deoxyribonucleic acid repair mechanism, is dysregulated (or ad lib elevated) in cancer cells and contributes to in progress genomic evolution [3,5], drug resistance [3] and growth of cancer cells in connective tissue growth model [6]. We have recently conjointly shown that apurinic/ apyrimidinic nucleases (APEX1 and APEX2) contribute to accrued deoxyribonucleic acid breaks and homologous recombination activity in metastatic tumor cells [7]. Cancer medication that area unit genotoxic or induce deoxyribonucleic acid harm or breaks, either directly or indirectly, kill cancer cells by increasing the harm to their deoxyribonucleic acid. However, following such treatments the subsets of cancer cells that survive (and not killed by) additionally as traditional cells of the patient currently have accrued levels of deoxyribonucleic acid harm and breaks. This side of chemotherapy poses a risk of development of resistance to treatment in cancer cells and transformation of traditional cells. in step with this view, we've got shown that cancer drug, a therapy agent, induces homologous recombination activity and genomic instability in myeloma cells in vitro [7]. Similarly, sure therapy agents have been coupled to development of secondary cancers [8,9]. There are also reports that counsel that therapy has higher probability of contributing to development of cancer of the blood as compared to radiation. It is, therefore, necessary to develop medication that target mechanisms underlying accrued genomic harm and instability in cancer cells. Such medication has potential to inhibit/delay progression by reducing genomic instability and evolution. There is also evidence that such drugs may have ability to increase cytotoxicity while minimizing/ reducing genomic toxicity caused by chemotherapeutic agents [7].

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