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| Title:      | Rodent-specific rearrangement between hyaluronidase and chemokine receptor gene clusters and its implication in cancer resistance, inflammation and aging  |
| Authors:    | Anjoom Thahir A.V. (/jspui/browse?type=author&value=Anjoom+Thahir+A.V.)  |
| Keywords:   | Tumor microenvironment<br>Chemokine signaling<br>Guinea pig vs NMR   |
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| Abstract:   | <p>Cancer is a condition when abnormal cells divide in an uncontrollable manner. Both environment and genetic factors have equal role to play in developing cancer. Cancer is widespread across animal kingdom. But few large animals (elephants, whales etc.) and smaller rodents (naked mole rat, blind mole rat etc.) are extremely resistant to cancer. Elephant genome has lot of copies of tumor suppressor gene, p53, mediate cancer resistance. But in case of naked mole rat, cancer resistance is attributed to the high amounts of High molecular mass hyaluronan in the extra cellular matrix. Recent study reported that oncogene with a neighboring tumor suppressor gene is less prone to amplification. So, it will be interesting to know whether genomic rearrangement near oncogenes or tumor suppressor genes have any role in naked mole rat's cancer resistance. In this study, we have observed a single large rearrangement between hyaluronidase and chemokine receptor gene clusters in rodents. Interestingly it was observed that the organisms, naked mole rat and guinea pig, showing cancer resistant properties have significant long-range rearrangement. The rearrangement is happening exclusively in rodents. It is well reported that chemokine receptors and hyaluronidase are involved in inflammation, cancer progression and aging. Owing to the above knowledge, we suspect that the rearrangement might explain rodents' survival in stressful underground habitat and their ability to develop or resist cancer and their varying lifespan. It will be interesting to know how the chemokine signaling pathway differs regarding chemokine receptor gene expression in different rodents in relation to their physiological and environmental factors. We further hypothesize that 3D chromatin interactions and epigenetic modifications near chemokine receptor gene cluster may vary in time (different stages of aging and cancer) and space (different cells involved in tumor micro environment and inflammatory signaling pathway). Studying involvement of tumor micro environment, in terms of chemokine-chemokine receptor mediated interactions will help in understanding cancer in a broader sense.</p> |
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