Editorial

Lessons from the wild

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Cancer is not unique to human race. Any multicellular organism including plants is susceptible to developing cancer. The exact incidence of cancer in the wild is at best a speculation. Interest in comparative oncology is recent but has not occupied the place it deserves. There are innumerable species of animal kingdom which do not develop cancer as they do not live long enough to accumulate mutations. There are animals such as "naked mole rats," which are immune to cancer. The rarity of neoplasia in these rats is due to the secretion of high-molecular-mass Hyaluronan (HMH-HA), which, when secreted prevents the cells from overcrowding and tumor formation. It also confers elasticity to the animal to navigate the Burroughs. There are cancers which are communicable as in Tasmanian devils. This is an aggressive nonviral communicable cancer which is lethal. Evolution has ensured suppression of tendencies of developing neoplasms in multicellular organism. Peto's paradox is best explained by the development of guardian genomes such as TP53, development of immune system, bigger cells with reduced metabolism. These mechanisms ensure relative immunity from cancer. For instance, elephants have twenty copies of TP53 in their genome while human beings have only one. This ensures higher apoptosis of mutant and injured cells over repair. The evolution has evolved mechanisms to reduce the possibility of developing cancer in multicellular organism. A comprehensive study and enquiry of this mechanism across the phyla can help understand carcinogenesis with a different perspective.

More than 4 million dogs in the USA suffer from cancer every year. The incidence is more than that seen in humans. Pets share the same habitat as that of their owners but are free from exposure to common carcinogenesis of their human counterparts such as tobacco and alcohol. The cancers seen in these pets are spontaneous, thus amenable to study carcinogenesis.

The limitation of murine models in reflecting the complex characters of oncogenesis is being finally appreciated. This is despite the stellar role of these models in studying initiation of carcinogenesis, promotion, progression, and signaling pathways. Mouse models also do not accurately reflect the process of metastasis and recurrence. Spontaneous cancers in pet dogs are bereft of the disadvantages of the murine models. These cancers occur in immunologically competent animal with an unsullied microenvironment. They mimic the human counterpart more closely. Thus, they serve as better as better models. Hence, there is an increasing interest in adopting pet dogs for early screening of drugs. There is a precedent for this assumption. Liposomal muramyl tripeptide phosphatidylethanolamine was tested in dogs with osteosarcoma. This was a prelude for the phase III studies in children. Dogs have been good models in studying hyperthermia alone and with drugs. Heat dissipation patterns in dogs are akin to humans.

There are genomic similarities between canine cancers and that of human beings. For instance, there is a significant homology between metformin, insulin-like growth factor-1 receptor, mTOR, and high intensity. Thus, drug testing in dogs would be more reliable than murine models. The utility of pets particularly dogs in drug development and testing is being appreciated only recently. The best practice guidelines are yet to evolve. All the stakeholders are still not on the same page. Institutions of excellence in research who are willing to participate in comparative oncology should create large consortiums for research in comparative oncology and clinical trial such an effort will help us understand cancer better, and in drug development. Pharmaceuticals will benefit by weeding out ineffective molecules in the early stage of drug development. Pets and animals in the wild can also E-markers for carcinogenic potential of the million we live in. Yes, there are lessons to learn from the pets and animals in the wild.

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