Cancer as a Consequence of Breaking Through Evolutionary Constraints on Longevity

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Abstract

It is well known that the risk of cancer increases with age, but when viewed through a comparative lens, this is a species-specific trait. An animal is a relatively low background in the animal kingdom. The exceptions, where cancer is a major cause of mortality, are in species with well-developed immune systems, with about 50% of pet dogs that reach the age of 10 years will die from cancer. This is a consequence of having species-specific traits which supports the hypothesis that cancer is a species-specific trait and its underlying cause. Modern pet dogs and cats have achieved a social status equivalent to human family members. This confirms the hypothesis that cancer is a species-specific trait that exists in all species, which has a biological basis. The table shows the relationship of cancer risk in all species, including humans, dogs, cats, and laboratory mice that have undergone their expected evolutionary life span. These species retain cancer protective mechanisms that are acquired over their unique evolutionary histories, replacing natural selection with artificial selection over a very short period of time has not allowed for selection of new adaptive cancer protective mechanisms.

The table shows that the estimated lifetime cancer risk is low in all species except for humans, modern dogs, and laboratory mice that have undergone their expected evolutionary lifespan. While these species retain cancer protective mechanisms that are acquired over their unique evolutionary histories, replacing natural selection with artificial selection over a very short period of time has not allowed for selection of new adaptive cancer protective mechanisms.

Figure 2. Relationship between evolutionary age (red) and estimated lifespan (blue) of long-lived and model vertebrate species. Long-lived species have adapted to their environmental niches over 2 - 60 million years (red bars). Among the adaptive traits in each species, expected lifespan (blue bars) and cancer-protective mechanisms (table) were under strong selection, influenced by reproductive age, lifespan, and body mass. These traits are regulated by natural selection, which can create protective mechanisms in these species. The table shows the estimated lifetime cancer risk is low in all species, except for humans, modern dogs, cats, and laboratory mice that have undergone their expected evolutionary lifespan. While these species retain cancer protective mechanisms that are acquired over their unique evolutionary histories, replacing natural selection with artificial selection over a very short period of time has not allowed for selection of new adaptive cancer protective mechanisms.

COSMIC Signatures in Human and Canine Osteosarcoma

Figure 3. COSMIC signatures from human and canine osteosarcomas. The table and figure each describe the distribution of COSMIC signatures in human and canine osteosarcomas, indicating that distinct mechanisms are responsible for the unique, shared histopathological characteristics of osteosarcoma and its prediction programs and the comparable morphology and organization of these tumors in both species.

When and How Is Dog Cancer a Model?

Adaptive evolution has repeatedly solved the problem of how to diminish cancer risk with increasing life spans.

Advances in biological understanding of cancer risk and life expectancy are improving our ability to design and interpret experiments that will allow us to examine the impact of a different environment on survival. The Cancer Risk and Evolutionary Constraints on Lifespan in Long-Lived Species table provides an overview of cancer risk and lifetime data for different species, highlighting the importance of understanding the evolutionary processes that influence cancer risk and longevity.

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Figure 1. Overlapping Genes Hereditary Mutations or Indels in Histologically Hominoid Human and Canine Tumors Are Infrequent. The top 50 recurrently mutated genes in the COSMIC database, the top 20, 17, 10, and 7 recurrently mutated genes in the Human Cancer Genome Project and the top 4 recurrently mutated genes in the Cancer Genome Atlas were used to determine overlap in name-mapped orthologues among tumors in the same species. For humans and dogs, the top 4 genes were used to determine overlap in name-mapped orthologues among tumors in the same species. For humans and dogs, the top 4 genes were used to determine overlap in name-mapped orthologues among tumors in the same species.