Review of: "Is creeping abandon of human cancer defences evolutionarily favoured?"

Anindita Chakrabarty[1]

1 Shiv Nadar University

Potential competing interests: No potential competing interests to declare.

The manuscript titled “Is creeping abandon of human cancer defences evolutionary favored” was an interesting read. The authors have put forward this hypothesis to justify the extraordinarily high probability of cancer occurrences in human population compared to other animal species with similar or larger body size and longevity (Peto’s paradox). According to the authors such inconsistency may be a result of natural selection favoring loss of tumor suppressive mechanisms acting on humans living as hunter-gatherers until recent past. Such loss might be for saving energy as the food-supply was limited or limiting the dominant males within the tribes reach advanced age. The authors proposed some experiments that involve looking for evidence of preferential loss of tumor suppressor gene activities in present-day aging individuals and fossil records of hunter-gatherers. They also suggested that this can be correlated with the availability of nutritional resources at the prehistoric times. The authors emphasized on the role of epigenetics favoring the gradual loss of tumor suppressive activities.

Present day humans neither have scarcity of food nor do they live as small groups of highly inbred social structures. Hence, factors like limiting food supply would not continue to impact on the loss of tumor suppressive mechanisms in the current day aged populations. I would suggest the authors to look further beyond these theories to explain why gradual loss of cancer would be evolutionary favored and persisting in the current-day population? Could these be associated with chronic DNA damage response triggered by telomere shortening leading to accumulation of damaged senescent cell population and causing low-grade proinflammatory environment? Chronic inflammation indeed is a major culprit for carcinogenesis and can act both genetically and epigenetically.