

CANCER AND HUMAN EVOLUTION – A NEW HYPOTHESIS

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Background: Beside the well known particular morphological and physiological traits, certain pathological conditions differentiate Homo sapiens from the most related species, Pan troglodytes. Humans suffer from diseases that are absent or very seldom in chimpanzees: type II diabetes mellitus, psychiatric disorders, Alzheimer's disease, autoimmune disorders, having a greater risk of developing cancer than chimpanzees in the lab (20 vs. 2%). A hypothesis able to explain the mechanisms of human evolution could also explain the onset and mechanism of the specific human diseases. Method: We present here this hypothesis. Results: This hypothesis takes into account the new discoveries in genetics, epigenetics and developmental biology. We identified the insulin/IGF1 biochemical pathway that can play an important role in human evolution. The processes involved in human evolution are also critical for cancer risk, as being involved in cell survival, proliferation and differentiation. Some changes in the regulatory mechanisms of this pathway can be involved in some specific human diseases, especially cancer. Our hypothesis proposes that this condition could be a by-product of the human evolution. IGF1 level correlates better with cancer risk than the presence of an oncogene like BRCA1 or 2. Genetics studies show a strong selection for some proto-oncogenes during human evolution, maybe because they are involved in cell survival and proliferation. Conclusion: This hypothesis represents a new theoretical approach and could provide new therapeutic strategies for cancer.