Lecture 11: Why we get sick?

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1. Lessons, Questions and Answers

Lesson 1:

CASP12, a gene found in chromosome 11 of human beings, have been identified in numerous mammals. Its activated product, caspase 12, is a protein closely related to caspase 1 and other members of the caspase family, which process and activate inflammatory cytokines. It's initially believed that *CASP12* in every man should be common predominantly, regardless of some negligible nuance or scarce and extreme cases. However, this is not true. We found that most of human's *CASP12* are inactivated forms, but an extraordinarily higher percentage of activated *CASP12* exists in African and its descent around the world. Figure 1 illustrates the fact mentioned before[1].

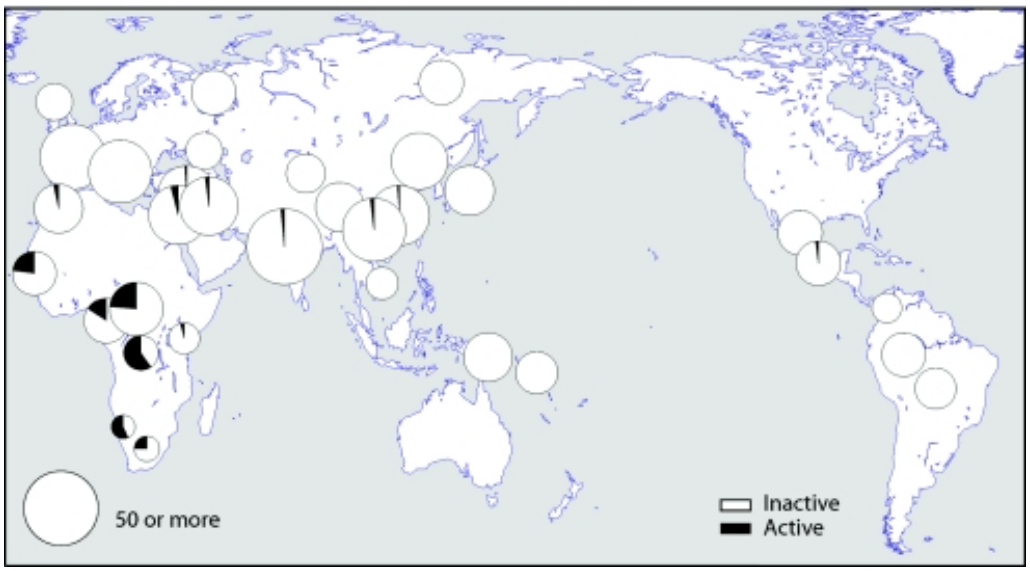

Figure 1. Worldwide distribution of the active and inactive forms of the caspase-12 gene in the HGDP-CEPH diversity panel.

From this figure, we can gain some insight into the migration of our ancestors. It's obvious that the population of our migrating ancestors might have undergone selection and founder effect many times so that the genotype of activated *CASP12*

wasn't brought out of Africa. As what we can see today, activated *CASP12* doesn't spread worldwide, even not appear somewhere.

From another perspective, it can be figure out that caspase 12 isn't pivotal in human body at all. Moreover, there might be some curse and blessing of the inactivation of *CASP12*. The selective advantage of inactivated *CASP12* is thought to be sepsis resistance in populations that experienced more infectious diseases as population sizes and densities increased.

Lesson 2:

Peto's paradox renders an eerie phenomenon that the incidence of cancer doesn't appear to correlate with the number of cells in an organism. It doesn't make sense in the belief that all the organisms have about the same rate of malignant transformation. Every result has its reasons, so does this eerie phenomenon. Exactly, in those long-lived mammals, there are some respective anticancer mechanisms to fight against most of tumors. For instance, elephants have high number of p53 psuedogenes which provides hypersensitivity of stress and conduct apoptosis instead of senescence. This mechanism comes out to be the cancer resistance. As for other examples, some of their mechanisms are indicated in Figure 2[2].

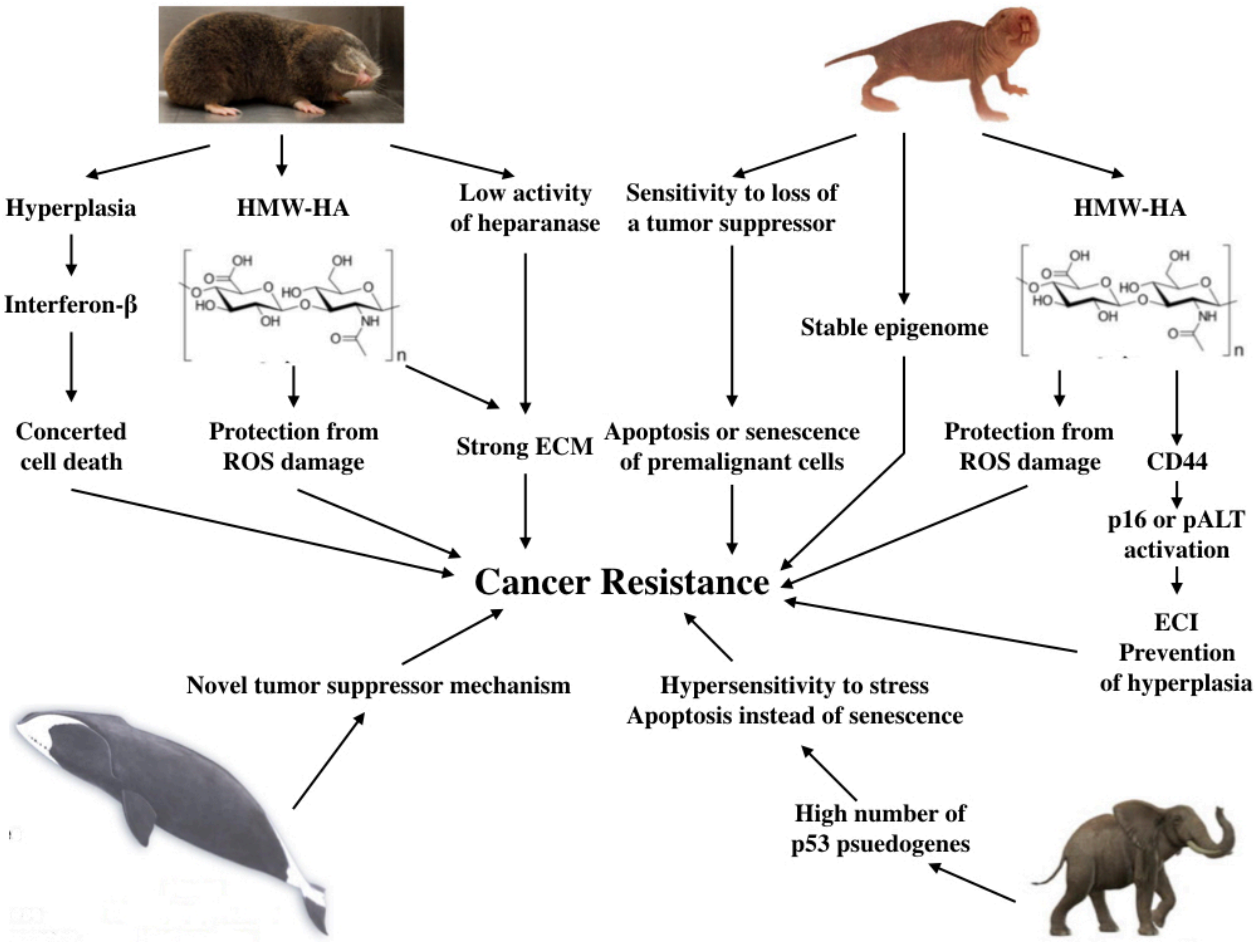

Figure 2. Anticancer mechanisms in naked mole rats, blind mole rats, elephants and whales.

Question 1:

Is rate of evolution related to cancer resistance?

Answer 1:

Reasoning:

Cancer resistance doesn't only represent the less probability for normal cells to transform into tumor cells, but imply the relatively stable genome and epigenome. However, one of the propeller of evolution is variation, which, in essence, depends on the mutation occurred in genome or epigenome. Without variation, all the inheritable information will, from a collective perspective, be passed down identically so that no difference between individuals makes selection hard to conduct its actual functions. Hence, I boldly address a hypothesis, if a strong enough mechanism against tumor

universally appear in a species, the species' rate of evolution will probably be affected, even inhibited by it.

Verification:

Any relevant article is not found.

Lesson 3:

The domestication of livestocks and crops is the foundation stone of development of human culture. Though this explanation is persuasive enough, we still puzzle about why these creatures are domesticated, but not others. It would be extremely irresponsible for us to say human beings had never met other non-domesticated creatures before the civilization established. Actually, those domesticated have some common traits. In terms of livestocks, common traits are smaller brains, sluggish sensors, and multifunctions. These traits helped them much familiar with human beings before domesticated. As for other unfriendly or useless traits, they make great hindrance for those non-domesticated against getting close to human beings. This kind of selective, reasonably, made up the unique and clear phenotypes of domesticated species today.

Lesson 4:

Many sickness today might be on account of the changes of environment, such as dietary habits, resulting in some mechanism in human body not adapting to the new conditions. For instance, type 2 diabetes was attributed to insulin resistance caused by higher blood sugar level and more ingestion.

2. Reference

[1] https://dx.doi.org/10.1086%2F503116

[2] https://dx.doi.org/10.1038%2Fs41568-018-0004-9

3. Postscript—What I've obtained from this course?

This semester, the course, Honor Biology, is the most illuminative one for me without doubt. To state this firmly, please allow me to elaborate what I've obtained from it. In my senior high school days, I never strained to learn Chemistry, even skipped almost every Chemistry class for the sake of escape from the ongoing fear of Chemistry. However, as I learned some lessons in this course, I started to conduct some tentative trial, then made unbelievably great stride on it. With the magnificent breakthrough, I subsequently integrated these new matters with what I'd learned before and coherent and analytic reasoning relevant to biology. I approached a much higher attitude which made me see everything with new perspectives and gave a far clear insight into every implied phenomenon. This is not a learned skill but a transition of mind and sight, a meaningful change in my life.

Another matter I'd obtained is my proficiency in English. Apparently, the way teacher train us DOES definitely work. Though some negligible mistakes still seen in my articles, the fluency and using correct words of mine have improved much better.

For these obvious improvements, I faithfully thank you for teaching this course despite your retirement. I'm anticipating what I will learn from this course next semester.