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Born in 1957 in Rochester, N. Y., USA
Studied Biology, Zoology, and Statistics at the University of Michigan, Ann Arbor and at the University of Florida, Gainesville

FOCUS

PROJECT

How Parasites Escape Host Defense

I will work with the members of our focus group at the Wissenschaftskolleg to evaluate the current status of research on the ecology and evolution of parasites. My own specific interests at present focus on parasites that extend their infections within
hosts by switching the molecules that coat their surface. During an infection, the host learns to recognize and attack the specific surface molecules of the parasite. As the host gains this ability to recognize the particular parasite, the host can kill off the parasite and clear the infection. However, those parasites that can change their coat can escape the host defenses and continue the infection; the host must start again to learn to recognize the new molecular coat. By switching its coat several times, the parasite extends the infection.
In a separate project, I am studying cancer. My work links three different levels: how cancer progresses within a particular individual, how the progressions in different individuals determine the distribution of ages at which individuals get cancer, and how inherited genetic factors influence the ages at which individuals develop cancer.

Recommended Reading

Frank, Steven, A. "Models of parasite virulence." Quarterly Review of Biology 71 (1996): 37-78.
-. Foundations of Social Evolution. Princeton: Princeton University Press, 1998.
-. Immunology and Evolution of Infectious Disease. Princeton: Princeton University Press, 2002.

TUESDAY COLLOQUIUM, 14.11.2006

Cancer: Age of onset reveals biological design

I wish I had the voice of Homer
To sing of rectal carcinoma,
Which kills a lot more chaps, in fact,
Than were bumped off when Troy was sacked.

- J B S Haldane

Cancer occurs mainly in older individuals. But some cancers, such as those of the eye or the bones, occur mostly in children. Why do most cancers happen late in life, but some happen early in life? What does that pattern tell us about the processes that cause cancer?

Here is another puzzle. In those who smoke cigarettes, the probability of getting lung cancer in each year goes up as the individual grows older. But for a smoker who quits at age 60, the probability of getting lung cancer in each year goes up until the age of quitting, but then the probability stays roughly constant in each subsequent year after quitting, neither going up or nor down very much. No one understands why. To study this pattern of constancy after quitting, we would have to understand the main causes of how cancer develops, and how our body is designed to protect against cancer. In my talk, I will discuss how we can think about these issues.

I divide my presentation into four parts: data, causality, design, and reliability.

In the first part on data, I will present observations on the ages of cancer onset in different tissues and under different circumstances. Those patterns of cancer onset establish the puzzles to be explained.

In the second part, I ask: What does it mean to say that a gene causes cancer? What does it mean to say that exposure to a chemical or carcinogen causes cancer? I answer these questions by showing how various causes shift the age of cancer onset.
In third part, I discuss what cancer teaches us about biological design. Cancer is the failure of the body to control the birth and death of cells that make up the body. By studying the failures of controls on cellular birth and death in cancer, we learn of the controls that normally keep the birth and death of our cells in balance.

In the final part, I discuss reliability. Cancer follows from failure of the normal reliability mechanisms that control cells. Thus, we can ask about the design of organisms in relation to measures of reliability and failure, and how aspects of reliability and failure change with age. Interestingly, enhanced systems of reliability may often lead to the design of fallible components. For example, increased reliability in the protection against cancer may lead to reduced efficacy of particular components, such as the repair of DNA damage.

Frank, Steven A. (2017)

Universal expressions of population change by the Price equation : natural selection, information, and maximum entropy production

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=1041215436

Frank, Steven A. (2016)

Common probability patterns arise from simple invariances

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=1041216467

Frank, Steven A. (2015)

D'Alembert's direct and inertial forces acting on populations : the price equation and the fundamental theorem of natural selection

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=1041216041

Frank, Steven A. (2014)

Microbial metabolism : optimal control of uptake versus synthesis

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=788299956

Frank, Steven A. (2013)

A new theory of cooperation

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=794275001

Frank, Steven A. (2013)

Natural selection : VII. History and interpretation of kin selection theory

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=789394502

Frank, Steven A. (2013)

Natural selection : VI. Partitioning the information in fitness and characters by path analysis

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=789394278

Frank, Steven A. (2013)

Microbial evolution : regulatory design prevents cancer-like overgrowths

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=788299816

Frank, Steven A. (2013)

Input-output relations in biological systems : measurement, information and the Hill equation

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=788298585

Frank, Steven A. (2013)

Evolution of robustness and cellular stochasticity of gene expression

https://kxp.k10plus.de/DB=9.663/PPNSET?PPN=786606509