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Editorial

Open Acess

The Tasmanian Devil's Contagious Cancer

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A Tasmanian devil, the size of a small dog, is a carnivorous marsupial. The endangered animals live only in the state of Tasmania, an island off the southern coast of eastern Australia. Introduction of dingoes to the mainland four centuries ago is thought to have driven the devils to their disappearing island home.

The full scientific name of the Tasmanian devil is Sarcophilus harrisii, perhaps best known as Looney Tunes character Taz.

The cancer "devil facial tumor 1" (DFT1) first appeared in 1996 and has been rocketing through the population ever since.

DFT1 hideously disfigures the animal, with large tumors around the head and in the mouth. Normal biting behavior transmits it. In a new host the tumor explodes, cells rapidly dividing, easily overriding the animal's immune response.

Worse, live cancer cells go from animal to animal, as if the cells are themselves infectious organisms, pathogens. But this isn't the same as passing a sexually transmitted infection from person to person, such as HPV, which is a virus that predisposes to cervical cancer.

DFT1 is deadly, decimating the Tasmanian devil population and driving its extinction. Breeders are trying to establish a colony in New South Wales to save it.

Only three naturally-occurring transmissible cancers are known. Joining DFT1 are canine transmissible venereal tumor and a widespread cancer of soft-shell clams, according to the Transmissible Cancer Group in the Department of Veterinary Medicine at the University of Cambridge. Their study just published in PLOS Biology, with colleagues from Australia and France, reveals the odd genomes of the cancer cells that cause DFT1.

Exploring Strangely Stable Genomic Changes

"In cancer, the genome is shot to hell," a prominent researcher once told me.

Cancer cells, even within an individual, often display an array of the ways that DNA and chromosomes can go off-kilter as they lose control of their division cycle. Healthy human cells growing in culture famously divide 40 to 60 times, the so-called Hayflick limit. Cancer cells, given enough space and nutrients, hormones, growth factors, and transcription factors, divide forever.

A cancer cell that's been in a body longer has more mutations than a more recently generated one. Mutations accrue, their patterns holding the history, the narrative, of that particular cancer in that particular body.

The utter constancy of the genome changes in facial tumor cells in the devils, echoed in laboratory culture, is puzzling. The cancer genomes are much more alike, from devil to devil, than are the cancer cells in a single human.

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