An Agent-Based Model of the Spread of Devil Facial Tumor Disease in an Isolated Population of Tasmanian Devils

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ABSTRACT

The Tasmanian devil population is being reduced in the wild at an alarming rate due to an epidemic, which is the result of an unusual disease mechanism. Infected animals “inject” cancer cells into other devils, which then clone the cells, developing tumors. These tumors are invariably fatal. Field observers have developed hypotheses that include a life-history change for the species. It is hypothesized that this change has the potential to improve the population’s survivability. An agent-based model of Tasmanian devils is used to evaluate these hypotheses. The model results suggest that the devils’ intra-gender aggression as well as their aggressive mating practices render the life-history change hypotheses’ correctness improbable.

Keywords: Agent-Based Model, Devil Facial Tumor Disease, Ecology, Epidemic, Inference Testing, Tasmanian Devil

INTRODUCTION

An epidemic of devil facial tumor disease (DFTD) is threatening Tasmanian devils (Sarcophilus harrisii) with extinction. Sightings in the Northeast region of Tasmania (where the disease was first discovered in 1996) have declined 95% and have declined 64% over the entire state of Tasmania (DPIW, 2008).

The government of Tasmania, (Byrnes, 2007; DPIW, 2005), has undertaken multifaceted efforts to try to save the devils from extinction. But the outlook, at this time, is bleak for their long-term survival in significant numbers in the wild. At low population levels, their continued survival is significantly threatened by road kill and persecution by farmers (Hawkins, McCallum, Mooney, Jones, & Holdsworth, 2008).

The disease is the result of one infected animal biting another devil and transplanting tumor cells into the victim. An infected animal develops facial tumors which are open sores, with cells that easily separate. These loose cells cause additional tumors in the original animal and also are grafted into other animals by biting (Pearse & Swift, 2006).
Chromosomal studies have shown that all tumor cells are clones of the single ancestor cell, which developed into a tumor in the original victim (McCallum, 2008; Siddle et al., 2007).

Normally, one would expect the victim’s immune system to reject the transplant. As a result of very low genetic diversity in the devils, the tumor cells are not recognized as foreign by most of the devils’ immune systems. Experiments have shown that there exist only a small percentage of devils (possibly < 5%) whose immune systems reject the tumors (Pearse & Swift, 2006; Rex, 2008).

In most populations there are large individual-to-individual variations in two immune system proteins MHC (major histocompatibility complex) class I and II. This variation allows an organism’s immune system to distinguish between its tissue and foreign tissue and reject a transplant. The Tasmanian devils have very little genetic diversity in MHC class I and II proteins and do not reject the tumor cells as foreign tissue (McCallum, 2008; Pickrell, 2007; Sompayrac, 2008).

Jones, Cockburn, Hamede, Hawkins, Hesterman, Lachish, Mann, McCallum, & Pemberton (2008) report a devil life-history change. They cite an apparent unusually rapid evolution of the devils’ life history resulting in an up to 16-fold increase in the proportion of females exhibiting precocious sexual maturity (bearing young when one year old) (Schmid, 2008). They also posit that this change enhances the probability of the Tasmanian devils’ surviving in the wild.

These conclusions appear unlikely to be correct for two primary reasons. First and most serious is the belief that early sexual maturity will increase the survival potential of the species. Kermack & McKendrick (1927) and others’ work, on infectious diseases, suggests that increasing the number of susceptibles may hasten the spread of the disease and demise of the devils in the wild, rather than increasing the potential for species survival (Edelstein-Keshet, 2005).

A second potential problem is their attributing the life history change to phenotypic effects and evolution occurring over a ten-year period (four to ten generations). The change in ratio of early breeders to normal breeders might be a consequence nonparametric statistics and observation errors. If the majority (90% or more in some areas) of normal animals are selectively killed, necessarily the ratio of abnormal to normal will change.

Project Purpose

Hillborn & Stearns (1982) point out in their paper on inference in ecology and evolutionary biology many of difficulties and erroneous conclusions that occur as a result of assumptions about causation.

Are the simplest hypotheses necessarily the best? Is there a single cause? Are the hypotheses being considered mutually exclusive or are they compatible? How do we distinguish between the possibly overlapping predictions of competing complex hypotheses?

These problems are often exacerbated in the field of population biology, whether that of humans or of wildlife, because the researchers’ ability to gather data or test hypotheses is limited by humanitarian, political, social or observational constraints. Models, with appropriate fidelity and resolution, can be valuable tools in overcoming these difficulties (Gilbert, 2008).

The purpose of this project is to evaluate Jones, Cockburn, Hamede, Hawkins, Hesterman, Lachish, Mann, McCallum, & Pemberton (2008) conclusions using (1) agent-based modeling and (2) an equilibrium analysis of the devil population age structure, prior to disease onset.

Approach

A generic, agent-based model of Tasmanian devils was developed to study the spread of DFTD and the impact of early female sexual maturity on population survival. The model environment was closed (except for the introduction of two diseased animals).
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