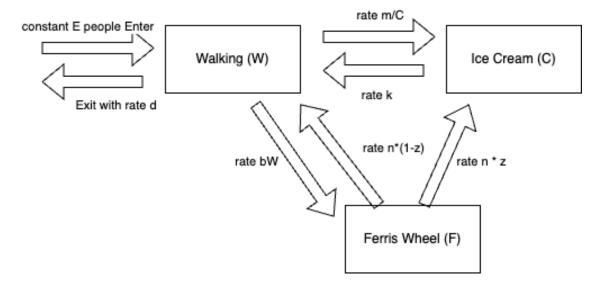
Dynamic Models in Biology HW 7 Jonathan Levine Fall 2023

Problem 1



The diagram above represents the model with 3 state variables. To translate that model into model equations:

$$\dot{W} = E - dW + kC - \frac{m}{C}W - bW^2 + n(1-z)F$$

$$\dot{C} = \frac{m}{C}W - kC + nzF$$

$$\dot{F} = bW^2 - nF$$

The parameters I chose:

E=2;

d = .3;

m = .2;

b = .2;

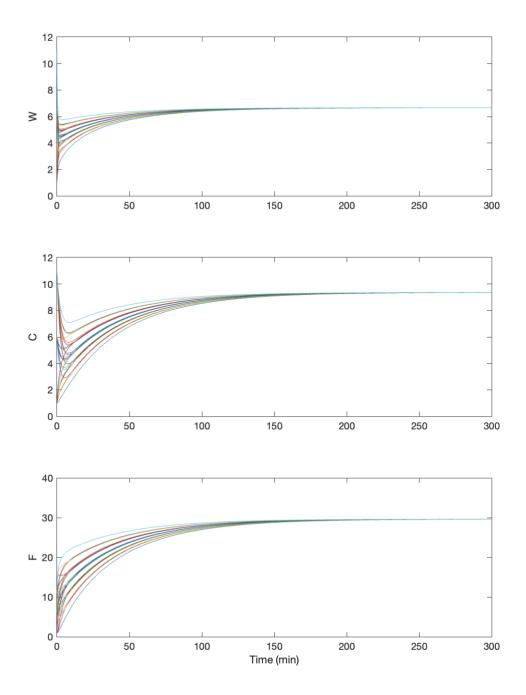
z = .3;

n = .3;

k=.3;

Note that the per-capita rates are less than 1, since it wouldn't make sense for there to be more than 1 person per person electing to do something in this case. With these parameters, the model comes to a stable fixed point independent of the initial conditions.

Here I ran the model for a variety of initial conditions (above and below the fixed point) and you can see that all traces lead to the same fixed point, at around (6.7, 9.3, 29.5). Of course, these dynamics assume that the units of three state variables are continuous, and do not necessarily represent discrete individuals in a 1:1 mapping.



I also solved for the fixed point in WolframAlpha by setting the derivatives to 0 and found the same fixed point:

$$0.3z = 0.2x^2$$

$$0.3y = 0.3 * 0.3z + 0.2 \left(\frac{x}{y}\right)$$
$$2 + 0.3y + 0.3(1 - 0.3)z = 0.3x + 0.2x^2 + +0.2 \left(\frac{x}{y}\right)$$

Solutions

$$x = \frac{20}{3}$$
, $y = \frac{40}{9} - \frac{14\sqrt{10}}{9}$, $z = \frac{800}{27}$

$$x = \frac{20}{3}$$
, $y = \frac{40}{9} + \frac{14\sqrt{10}}{9}$, $z = \frac{800}{27}$

The solution with only positive numbers (no negative people at the fair!) matches the fixed point from the simulation above.

Problem 2

- a. The model seeks to understand population dynamics of tumor cells, pro-tumor immune cells, and anti-tumor immune cells using an ecological framework. By doing this, they can make predictions about the effect of tumor induced immune cell conversion (a parameter in the model) on the viability of a tumor population. They show that this immune cell conversion is essential for tumor growth without this parameter the tumor population is not viable. This highlights a potentially important bottleneck for tumor growth and immune evasion, which can help orient research into the tumor immune microenvironment and guide future immunotherapy.
- b. The state variables are:
 - T (tumor cell population density)
 - P (pro-tumor immune cell population density)
 - A (anti-tumor immune cell population density)

The actual model is a generalized Lotka-Volterra Model:

$$\begin{split} \frac{dT}{dt} &= T \left(r_T - \frac{r_T}{K_T} T + \alpha_{TP} P - \alpha_{TA} A \right) \\ \frac{dP}{dt} &= P \left(-d_P P + \alpha_{PT} T \right) + \omega A T \\ \frac{dA}{dt} &= A \left(r_A - \frac{r_A}{K_A} A + \alpha_{AT} T - \alpha_{AP} P \right) - \omega A T, \end{split}$$

- c. The model is nonlinear.
- d. The model makes a huge simplifying assumption in lumping all immune cell types together. A more complex model would certainly include separate state variables for different immune cell types (macrophages, T-cells, etc.). For these purposes though, the authors' goal is to "explore the potential consequences of immune conversion, not to make quantitative predictions about tumor-immune population dynamics."