Does the introduction of a virus adequately control an exploding population in the case of myxomatosis and the European rabbit in Australia?

by

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Introduction:

Highly adaptable to its environment, the European rabbit now inhabits every continent except Asia and Antarctica. It is widespread in Western Europe and Northwest Africa. It has recently been introduced to South America, North America, and most detrimental, to Australia and New Zealand. In a most devastating blow to the continent, the European rabbit was introduced to Australia in1859 by a rich British landowner named Thomas Austin. Innocently enough, longing for the hunting he missed from back home, he bought 24 rabbits and had them shipped to his estate in Australia. He set them loose on his grounds and proceeded to have some hunting fun.

Unfortunately for the continent, his lack of foresight brought both males and females of the species. Open grasslands, plenty of food sources, rapid breeding ability and few predators at the time brought a wildfire effect in the spreading of this population. In Australia, the rabbit is a pest, and it is from this effect that vernacular English adopted the phrase, "breeding like rabbits."

Because of its extremely high population--which has fluctuated, but has been believed to reach almost a billion at one time--and indiscriminate eating habits, it has continually decimated most of the local sheep and cattle's grass, it has turned once-thriving sheep ranches into wastelands, at one point cutting wool production in half. Many other plant species and competing animals have been driven to the brink of extinction by the rabbits' unstoppable appetite. Selective grazing by the rabbits has changed whole ecosystems and has contributed to soil erosion. Also, the rabbit has been known to drive some smaller mammals (such as native mice) out of their burrows, helping foxes (also recently introduced to Australia) catch these smaller mammals, hurting their populations. Native wildlife has also been hurt by the poison and traps left out to catch the rabbits.

Several methods of containment have been attempted in the past 150 years. Shortly after its introduction in 1859 and subsequent population explosion within 50 years, bounty hunting of the European rabbit was finally implemented.

But in 1950 a highly lethal, rabbit-exclusive virus called myxomatosis was discovered in Brazilian rabbits and was found to be lethal in European rabbits as well. The virus was promptly released into the wild in Australia and effectively spread like wildfire through mosquito and rabbit flea bites, and killed all but 0.2% of the rabbits.

This small percentage that survived built a resistance to the virus, bred like rabbits, and passed on the resistance to the virus onto their offspring. The population exploded again.

However, for a short period of time, a process called co-evolution was in place. The rabbit population built up a genetic resistance to the virus, and the virus evolved into a less deadly form in order to preserve itself. Now, rather than killing 99% of the population, an equilibrium effect was established. Myxomatosis now kills about 50% of susceptible rabbits that are infected.

Eventually, the rabbits built up a much stronger resistance, and the population exploded once more as birth and survival rates increased. However, it is the temporary

phenomenon of equilibrium that occurred that we are interested in studying and portraying. It shows that a population exclusive virus, even one with almost 100% fatality rates can be employed to dramatically reduce an unwanted population and place it in a safe position without long term consequences. Even if this affect is often short lived, as in the case when the control lasted until the mid 80's, it can be employed to restore equilibrium.

The question also rises, how long does such a process last?

The Process of Model building:

The bare structure of this model follows the boundaries of a common epidemic population model with a population that is divided into three parts: those who are susceptible to infection, those who have contracted an infection, and those who have recovered from an infection, assuming that recoveries cannot be re-infected. Once this has been created, then complications concerning resistance, varying death rates and increased birth rates can be created in turn.

Therefore, the model began with a simple population model, which included a birth rate and a death rate to create equilibrium in population. Assume the total population starts at 5,000,000 rabbits, just as a possible starting point. To achieve this equilibrium, the birth and the death rate had to be set at equal values, of 5.5/12.

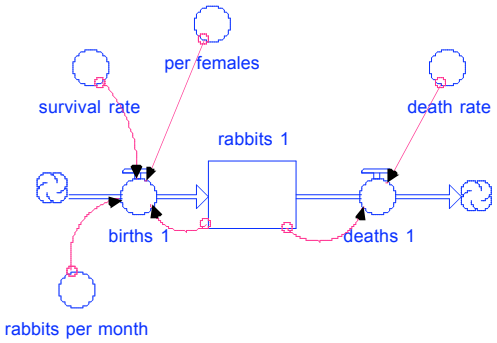

Figure 1: the preliminary model. When death rate and birth rate equaled each other, population reached stability

The second step involved the instigation of a simple population division based on stages of infection: a susceptible population, an infected population, and a recovered population. But because the prospect of infection was complicated, it was only instituted into the model at smaller steps. Therefore infected population and its parameters were added first, and deaths because of infection were excluded to make sure the transfer into infected population went smoothly.

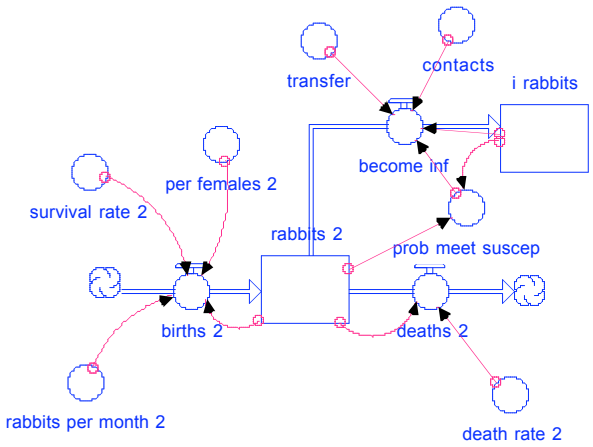

Figure 2: Included infected rabbits. Death rate was reduced, and eventually all rabbits become infected

All previous factors of birth rate and death rate remained constant, however their contents were altered to compensate for the new shifts towards infected. The resulting graph showed susceptible population traveling towards 0 and infected population rising until it encompassed all of the population that did not die of natural causes. This behavior was correct, which therefore allowed the more complicated phase of adding recoveries, infected deaths, and immunities.

The second to last step was the completed epidemic population without levels of immunity factored into it. At this point, number of contacts was reduced from 50 to thirty after more research was accumulated concerning the behavior of mosquitoes, but this was compensated for by an increase in transferability that still made sense within the boundaries of nature. Also, because all healthy rabbits factor into the breeding population, the total healthy rabbits (recovered plus susceptible) tied back into the number of births, rather than exclusively the susceptible. As well, because it is assumed that the rabbits that survive myxomatosis are generally older, the death rate for recovered is set at slightly higher.

The model still achieves a semblance of equilibrium in the short run. Total population does decline slowly, but for now I overlooked this because it was a very small level of decline. The population of susceptible rabbits grows sharply at first, which allows the virus to explode, sharply increasing the number of infected rabbits and sharply decreasing the population of susceptible. Number of recovered rabbits grows slowly, but once it reaches a significantly high level, all populations begin to level out at lower levels.

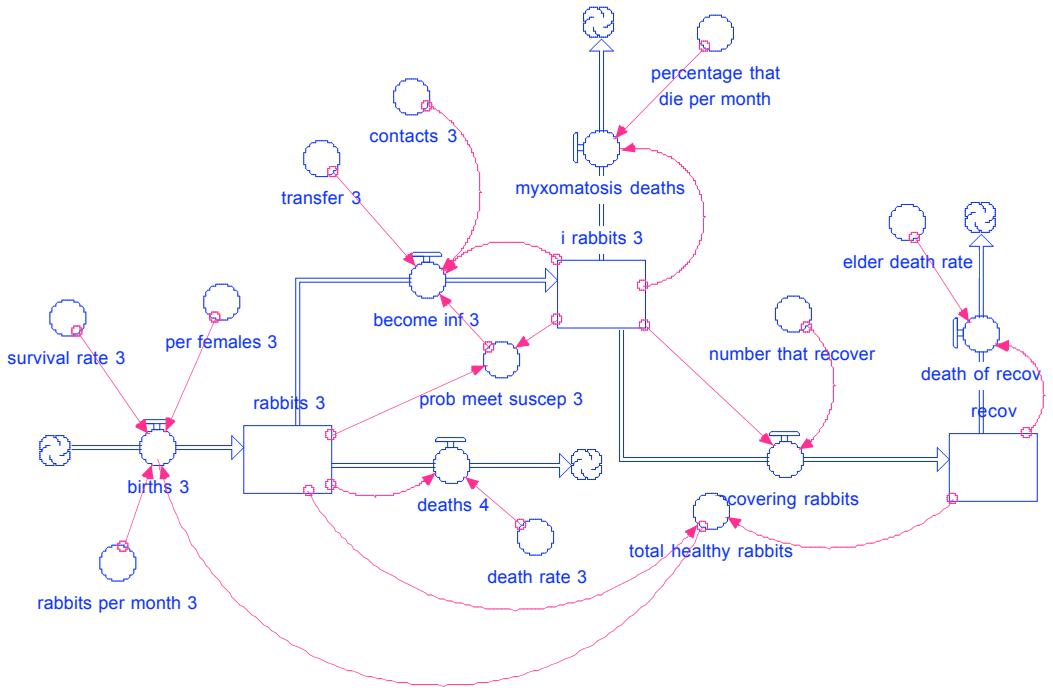

Figure 3: The model is almost complete, but the end result is that the infection slowly decreases the population. This will be changed when genetic resistance is added to the model.

The matter of genetic resistance is complicated to install. Essentially, recovered rabbits have the potential to pass *some* of their ability to recover through their genes, therefore increasing the *chance* that their offspring will also survive. This is significant however. After several decades, 50% of infected rabbits survived rather than the mere 1% at the outbreak.

The knowledge that the virus, too, decreased in its fatality rate is complicated. Therefore rather than include it in the time allotted, it can be included into rabbit resistance.

Analyze the concept of resistance by comparing the number of recovered rabbits in the breeding population to the number of susceptible. Once the number of recovered rabbits reaches and surpasses the number of susceptible—when the ratio is greater than 1:1—a modifier to the myxomatosis death rate comes into play, slowly decreasing it as the ratio grows. By the time recovered population reaches double that of susceptible, the myxomatosis death rate is 90% of what it used to be. When it reaches three times, the death rate has moved to 50% of what it used to be.

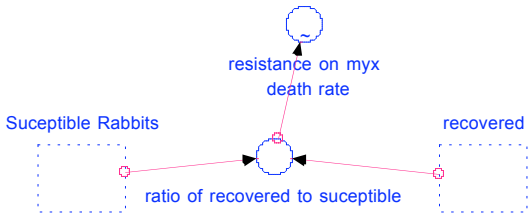

Figure 4: The piece that creates the multiplier "resistance"

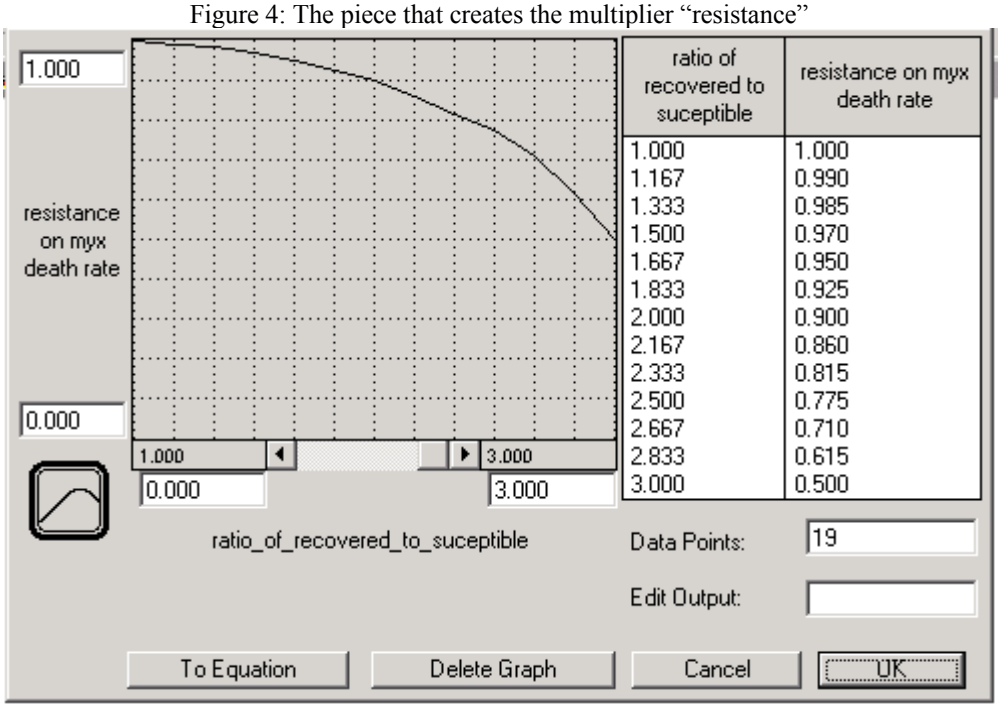

Figure 5: A section of the multiplier graph from 1:1 to 3:1, showing how the effect on Myxomatosis death rate causes it to decrease, eventually ending at .500

The next section will discuss the final product with the addition of the multiplier.

The Finished Model and How it works

The final model works on the premise of three interacting stocks, each representing a piece of the total population, those that are susceptible to infect, those that have become infected, and those that are recovered from infection.

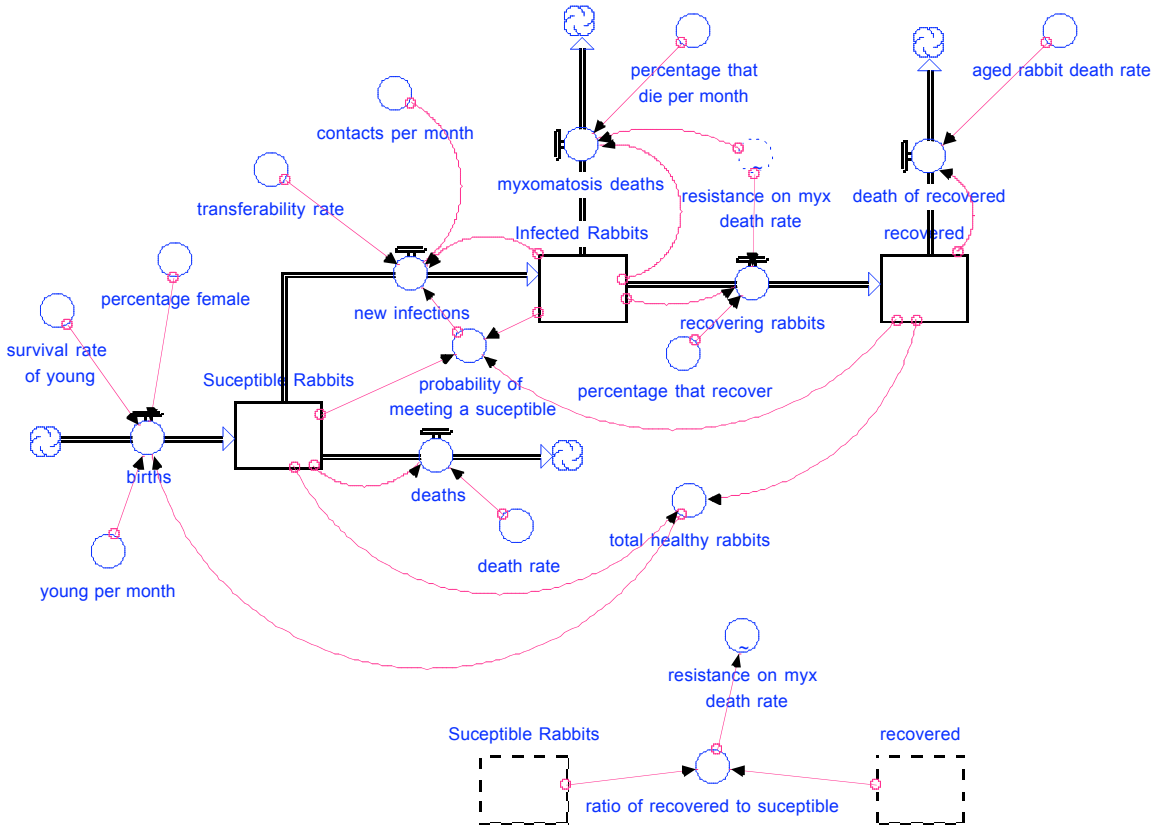

Figure 6: The finished product.

The first section (refer to figure 6) is that of the susceptible. This segment of the population has a single inflow of births, and two outflows. One outflow is for natural deaths (it is assumed that some rabbits live their whole lives without getting infected by myxomatosis, especially in the beginning and ending stages of the virus' introduction). The other outflow includes the rabbits that catch the disease, therefore traveling from the susceptible population to the infected. When rabbits are first born, they are automatically susceptible to the disease, therefore a birth inflow only makes sense attached to susceptible.

Four factors make up the inflow of births to the susceptible population: the number of young a rabbit produces per year, the number of rabbits that reproduce (consisting of susceptible plus recovered population), and the knowledge that not all young born into the wild survive into adulthood. The fourth factor addresses the assumption that half the population is female. Death rate is simply how many rabbits will die per month.

New infections depend on four factors: the rate of transferability (how virulently the disease will pass itself from victim to victim and how able it is to take hold), the number of contacts that the disease makes per year, (the disease is passed on through mosquitoes which have a very short lifespan, therefore each mosquito is considered as a

"contactor" of the disease, and each contactor can only make so many contacts in one year with such a short lifespan), the number of infected rabbits to pass the disease, and the likelihood that the contact will be with a susceptible rabbit.

Those rabbits that have moved into the infected stock now have two possible outlets, they can either die of the disease or they can recover from it. These two things are dependant of one another, obviously. Over time, the percentage that die declines, and the percentage that recover increases to compensate. This is simple to state, but difficult to accomplish.

Initially, the disease killed 99% of all its victims. Survivors of the disease do not necessarily pass any kind of immunity to their offspring, but there is a greater degree of evolutionary resistance. Implementing a resistance factor was accomplished by creating a comparison between recovered rabbits and susceptible. Naturally, as the number of recovered rabbits begins to outnumber the amount of susceptible, the general population will evolve into a more resistant one, as the virus kills off the weaker rabbit.

The normal values of death and recovery were initially set at 98 and 2, respectively. Therefore, .98 multiplied by the amount of infected rabbits die, and .02 multiplied by the infected rabbits recover. A graphical multiplier converter was designed by setting up a ratio of recovered to susceptible. This multiplier was inactive, set to one until the ratio of recovered to susceptible rabbits became greater than one. At that point, the multiplier decreased at an increasing rate, though very slowly at first. This multiplier reduces the myxomatosis death percentage, and factors back into the recovery rate to increase the number of recovered rabbits.

Finally, recovered rabbits are most likely older, and by the time they reach this stage they are more likely to die, therefore a new higher death rate is created and attached to the death outflow.

The Model Feedback & Loop Story

There are a number of feedback loops, both reinforcing and balancing at work in this model, some working to counteract each other. Each stock in the model involves one loop that acts to increase it, and one that acts to counteract it, and thus actx as a controlling agent. The behavior of this model comes down to which loop will dominate the other.

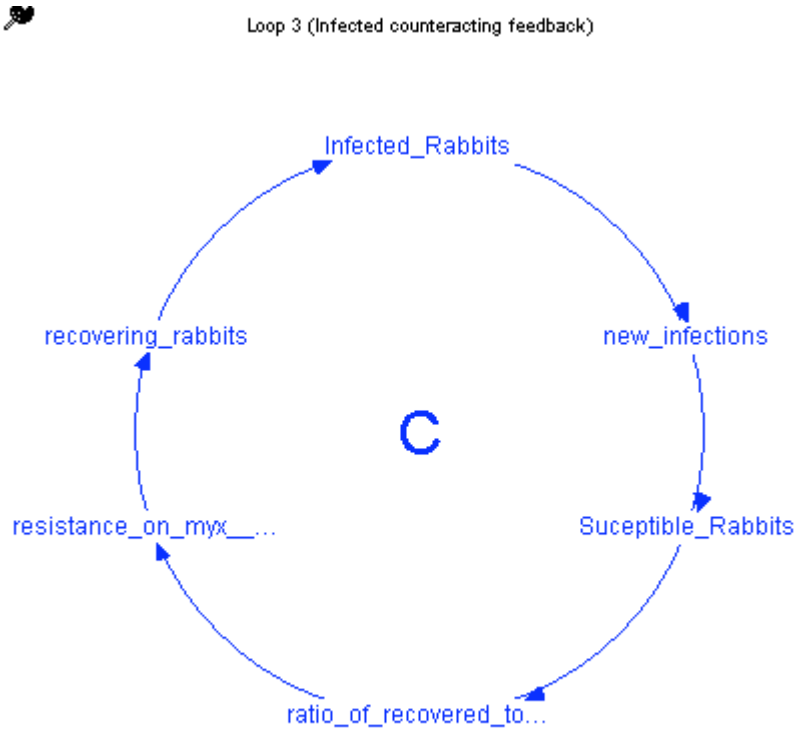

Figure 7: A primary balancing feedback loop that acts to counteract the growth of the infected population

As the number of infected rabbits increase, the number of new infections increase. As new infections increase, susceptible rabbits decrease. This decrease increases the ratio of recovered to susceptible, which increases the resistance to the virus. This resistance increases the number that recover, which acts to decrease the overall number of infected rabbits. In this way, the process counteracts itself.

However, as with all pieces in this model, there is an element that opposes this, one that reinforces the growth of the infected population. Essentially, unchecked, this loop would attempt to cause the exponential increase of infected population.

As the infected rabbits increase, so do the number of new infections simply by process of interaction. More infected being present in the system increases the number of contacts. But as well, the increase in infected rabbits acts to reinforce itself through other parts of the model, as shown below.

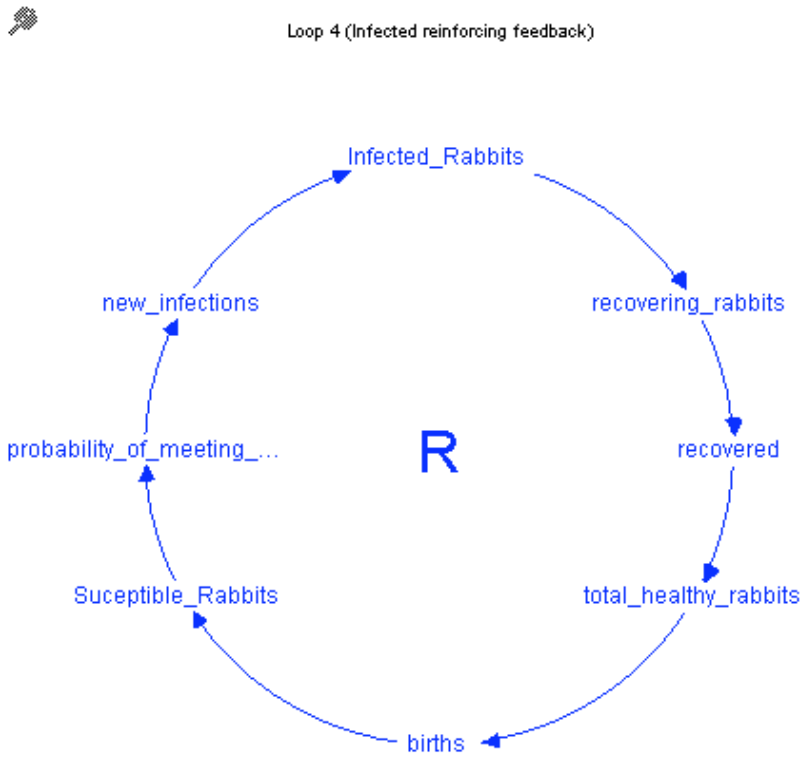

Figure 8: An example of a primary loop that acts to reinforce the infected rabbit population

In this loop, as the infected population increases, the number of rabbits that recover also increases. As recovered rabbits increase the total population of healthy rabbits can produce more offspring, therefore increasing the number of births. New births flow into the susceptible stock, which increases the probability of meeting a susceptible, which then comes back to increase the number of new infections, and following, the number of infected.

For every stock value there are several feedback loops at work, the two described are just some examples of important ones within the system.

These feedback loops are important to the system as a whole because they dictate the nature of growth within the three divisions of population. Over time, different loops have different strengths. Initially, the reinforcing loop for infected population is very strong, which is why there is such an initial climb in infections and reduction of the overall population. But as time proceeds, the balancing feedback loops gain momentum and overcome those that reinforce, allowing for a reasonably stable population.

By examining this feedback we can see how the initial chaos of introducing a virus into the system actually provided stability in the long run. The population climbs drastically, then is decreased drastically, but through the various types of feedback eventually levels at an appropriate amount.

The Model Boundaries

Major Assumptions: In order to make this model sensible, some data had to be manufactured or assumed. For example, it had to be assumed that 50% of rabbits born into the wild die before they are reproducing age. Deviation to this number often resulted in uncontrollable and irregular growth.

Also, the number of contacts made had to be calculated from bits and pieces of information about the nature of disease and the nature of its carrier. Since the virus is passed through an insect host, research was conducted analyzing mosquitoes to determine their behavioral patterns exclusive to certain regions. In some regions mosquitos live longer than others, therefore, assumptions had to be made about their life span, breeding, and feeding habits.

The number of rabbits per reproducing female also had to remain very small, much smaller most likely than is plausible. Other difficult adjustments to death rates and infection rates should be made. Because of time restrictions in the creation process, I was not able to take into account adjustments that would have been made by nature to birth and death rates due to overpopulation, though this was quite an important factor. However, this was not the exact behavior being studied, therefore it was compensated for by simply reducing the birth rate to a suitable level. With more time, perhaps that factor could be taken into account and could aid in population control, because it is quite a realistic one.

Choice of Time: Time specs are adjusted to fit the parameters of the year. The virus itself was introduced in 1950 and in approximately 30 years it had lost its effectiveness. However, for those thirty years, the population was in quite reasonable control. Therefore, time specs were re-adjusted to fit this time span.

Model Testing

For this model to work effectively, it needed to show graphical behavior that fit expectations. Susceptible population should explode initially, and then be reduced drastically in accordance with the sudden explosion of the infected population. Infected however should also quickly be reduced after its initial explosion with the added effect of a very high death rate and increasing number of recoveries. Recovered should grow very late and very slowly, and then reach a level amount around the same time in which the other two sectors of the population level. This was accomplished, and reaffirmed by the graphical results:

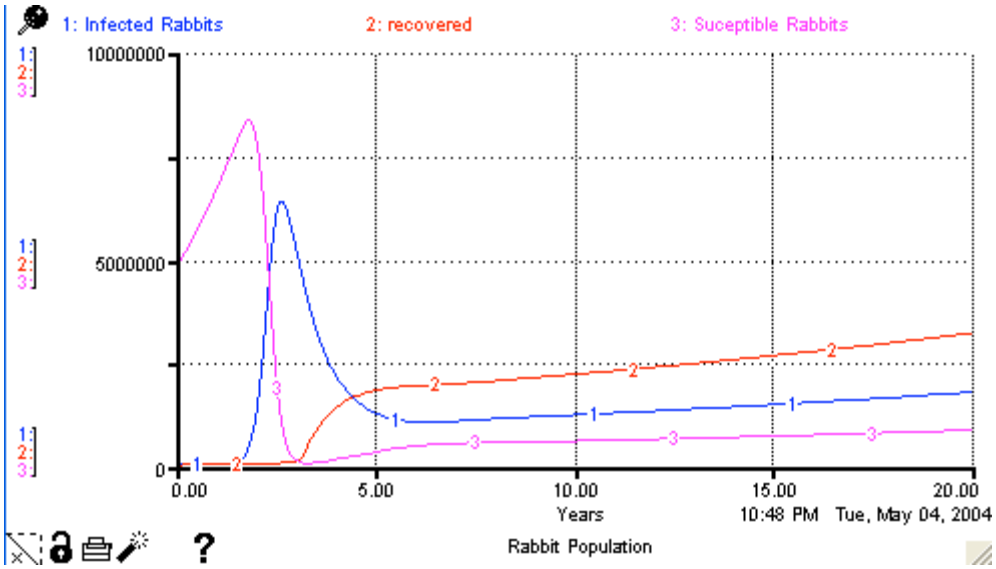

Figure 9: The final graphical analysis showing that the model accomplishing the desired stabilization

The slight increase in population after it has reached stabilization actually makes more sense in this case. The rabbit population did begin to reach a point at which it was out of control around the time period of 30 years after its introduction. As this graph shows, population growth rate begins to climb just as it passes the 20 year mark (This growth will be shown and addressed at a later point.) As well, the more steady pattern of growth is primarily for the recovered population, therefore this new surge in rabbit population presents the problem Australia is now facing: once again, a large population of rabbits, but the disease that would control them is losing its effectiveness.

If the average number of rabbits increased for any reason, would the growth pattern adjust accordingly? For example, if there were any change in the environment--or perhaps as a natural response to the declining population—the growth pattern of total population should grow slightly, following the same pattern.

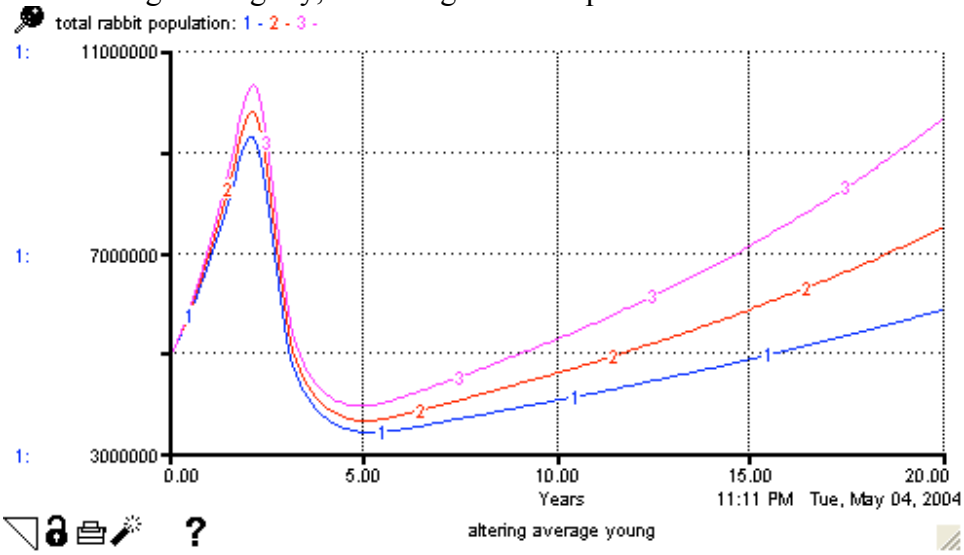

Figure 10: A sensitivity analysis examining average young per female of 2.0, 2.1, and 2.2

The model appears to be behaving in the anticipated manner.

The Results of Modeling and Thinking

The Results: The rabbit population very clearly followed the anticipated and historically proven events almost perfectly. The behavior of population explosion can be controlled in many ways, many of which are instituted by nature, but when nature fails to take control, the artificial introduction of a species-specific virus seems to be rather effective—temporarily. Viruses are conditioned for their own survival, therefore particularly deadly ones, given the time, will evolve into a much less deadly form. Thus was the case of the myxomatosis virus. An initially fatal disease, it was a particularly effective idea, taking into account the time available and the knowledge that rabbits are highly adaptable. The problem exists in the evolutionary behaviors of the virus itself—they were too strong. Self-preservation of the virus population allowed the rabbit population to grow so as to not eliminate the hosts it depended on to survive. This is combination with the high adaptability of rabbits allowed the rabbits to overcome the stabilizing effect of the virus. 30 years later, around the late 1980's the population was once again at a problematic level and growing.

Accurate data shows that this natural system very accurately follows an expected pattern. Therefore, though the introduction of a virus does create an interesting co-evolutionary stabilization, it is only a temporary solution for invading population control.

The final graphs: The final graph is shown below, extended into a 30 year period, showing the stabilization behaviors that have been discussed and explained throughout this report.

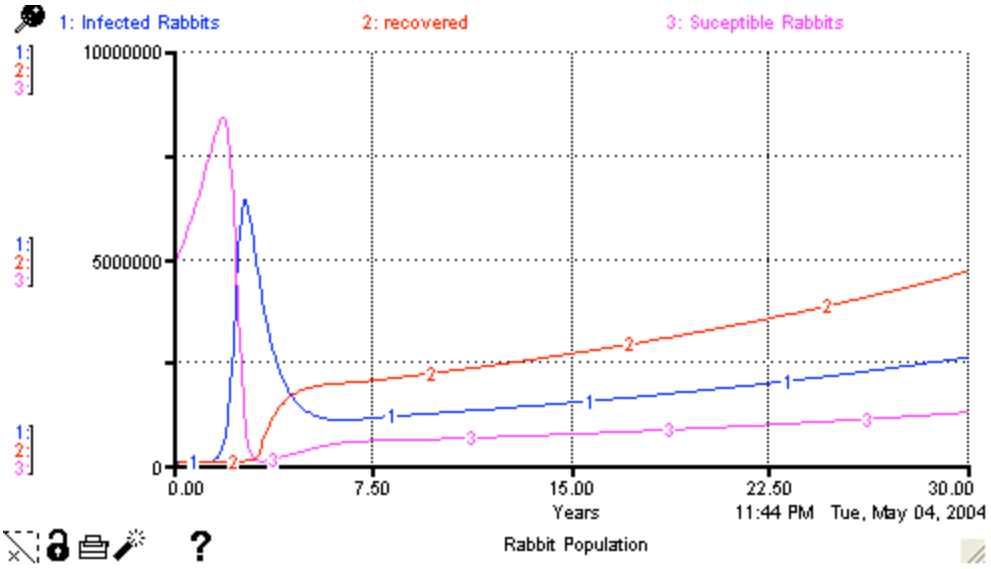

Figure 11: The final graph extended into a thirty year period, notice how the stabilization is reasonably effective for about 20 year period, but it is starting to increase as we pass beyond that point

The Key Learning From the Model Process:

- The introduction of a virus can work effectively, but only from a short period of time
- It is important to build models in pieces so that the pieces can be testing accordingly along the way, rather than having the final model have unknown holes that are difficult to pinpoint.
- The interaction of balancing and reinforcing feedback loops will be what determines the growth of the item being studied
- Several different factors *must* be at work in controlling any population growth, one single method is not effective enough on its own

On its own, the introduction of a virus can be effective in the short term if monitored carefully and not allowed to completely eliminate its host. But in the long term, alternative methods of control and the added affect of nature need to be taken into account. This was accomplished in Australia by introducing birth control into the female population, putting a bounty on rabbit hunting, and building fences. The myxomatosis virus aids in this process, but was surprisingly the most effective and immediate way to get the population into a more manageable level. Therefore, though this equilibrium was not a permanent solution, it was adequate, and allowed researches in Australia time to develop alternative methods of control.

With more time, I would have liked to include an element of natural control that would increase the death rate when population became too crowded. Limited resources would cause the death rate to increase dramatically after competition for survival. I believe that with this piece I could have established a more realistic and long term analysis of this particular population model.

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Appendix – Documented Equations

Omitted