

Spatial Models of Chytridiomycosis Dynamics in Frogs

Annette P. TAMINO^{1*} and Rafael P. SALDANA²

¹*Biology Department, Ateneo de Manila University, Loyola Heights, Quezon City, Philippines*

²*Mathematics Department, Ateneo de Manila University, Loyola Heights, Quezon City, Philippines*

**E-mail address: aptamino@admu.edu.ph*

(Received October 27, 2002; Accepted April 15, 2003)

Keywords: Chytridiomycosis, Susceptible, Resistant, Cellular Automata, Epidemiology

Abstract. Simple application of rules governing a one-dimensional Cellular Automata (CA) demonstrated patterns of spread of Chytridiomycosis in three grid-based CA models. Simulation results show that the spread of Chytridiomycosis follow exponential patterns of infection rates for susceptible and resistant frog populations, while there was oscillatory behavior in the simulations for resistant populations and populations subjected to drug treatments. Susceptible populations died out, while resistant and treated populations recovered or stayed healthy. Stable oscillatory patterns in these models provide a quantitative description of the relationship between periods of recovery versus periods of sickness, two variables significantly measured in epidemiology. These CA-based models are useful aids in the analysis of behavior of emerging infectious disease (EID) in wildlife. Applications of theoretical biology in analyzing visualization models of complex systems presents an alternative in understanding population behavior so that possible scenarios that highlight these simulated transmission experiments may strengthen studies in disease control.

1. Introduction

Chytridiomycosis now constitutes a global panzootic disease, infecting and killing amphibian species in six continents. The fungus (*Batrochochytrium dendrobatidis*) causing this has low host-specificity and ability to thrive without an animal vector. Theoretically, an analysis by HERO *et al.* (2002) have attributed this phenomenon to global warming (causing frog species clumping in certain areas) and the introduction of species by international trade (that may have brought the disease to naïve populations). One of the impacts of the drastic decline in amphibian populations and the extinction of significant species is the loss of wealth of animals that play important roles in the ecosystem.

Frogs and other amphibians (toads, salamanders, newts, etc.) act as global sentinels for environmental degradation such as pollution, ozone layer depletion and the introduction of invasive species by their niche specialization and inherent biological peculiarities. In Asian countries, very little study has been done on the dynamics of wildlife diseases as

related to these environmental issues. So the science in uncovering diseases, its origins, spread and control is presently linked with multidisciplinary approaches parallel to technological innovations that have aided humans to understand complex disease dynamics in populations. Many emerging infectious diseases (EID) in wildlife tell us two things. Primarily, disease emergence happens by the change in ecology of host, pathogen or both (SCHRAG and WEINER, 1995). Secondly, spread of disease depends on the critical levels of infection rates and density of susceptible hosts and its vectors . This may contribute to pathogen pollution that impinges itself to unsuspecting host populations.

Wildlife is known as reservoirs of many pathogens and their potential to harm other species of animals and man has to be curbed by standardized surveillance strategies, some of which are impractical to do in actual fieldwork but can be done using computer generated outputs.

This current study is aimed at visualizing the effects of Chytridiomycosis on susceptible and resistant representative species of frogs based on analysis of existing data and by employing a powerful computational tool called Cellular Automata (CA). These are discrete space-time models that have been used to study the dynamics of several biological systems from the levels of cell activity to the levels of clusters of cells and populations of organisms. CA was devised to prove that a logical order exists in the behavior of populations affected by disease spread. By putting a specific frog population as one complex system with many identical discrete components in lattices, having periodic boundary conditions; local interaction between neighboring individuals can be observed as highly influential in the change in cell states. These results account for the dynamics and produced the patterns that were highly observed to belong to all biological systems.

2. Dynamics of Chytridiomycosis

Diseases in amphibians are poorly studied and methods for treatment are not well established. It is only this century that the baffling issues of emerging infectious diseases (EID) from wildlife are being considered significant due to advancing detrimental changes in the environment. Now, the best way an EID is controlled is by isolation of “infectives” (MARANTELLI and BERGER, 2002). It appears that though this disease is widespread in a lot of amphibians, some species were discovered to be resistant to infection and others were more susceptible.

2.1. Possible origins

The origin of Chytridiomycosis has baffled scientists and wildlife biologist because its occurrence cannot be implicated to a specific area pertaining to onset of an EID. This emerging infectious disease that affects a wide-range of amphibian species may have originated as a result of (1) a spill-over from domestic animals to wildlife populations living in proximity, (2) direct human intervention by host or parasite translocation, or (3) there may not be a direct involvement from humans at all, because the pathogen is endemic to the area (DASZAK *et al.*, 2000). The latter condition was thought of when frogs in pristine areas (mostly endemic and or endangered) were turning up dead in Australia (BERGER and SPEARE, 1999; ABC NEWS ONLINE, 2000).

Table 1. CA states.

State	Cell property	Expression
0	Healthy	—
1	Carrier/Quiescent	—
2	Infected	—
3	Terminally ill	—

2.2. Geographical range

Although rare as it may seem, the extent of outbreak in Chytridiomycosis has affected both captive and wild amphibians. It has been identified as the culprit behind mass deaths and extinction of some frog species worldwide in these six continents: Africa, South America, Central America, North America, Europe and Australia and Oceania (ENVIRONMENTAL NEWS NETWORK, 2001) (Table 1). Chytridiomycosis was first noticed by John Nichols in 1990 upon the investigation of the cause of death of California's "arroyo toad" (TENENBAUM, 1991).

2.3. Characteristics of the fungus

Mycologist Joyce Longcore characterized the fungus *Batrachochytrium dendrobatidis* of Phylum Chytridiomycota in 1998 as causing Amphibian Chytridiomycosis 1. Chytrids are true fungi that live in soil, freshwater ponds and streams and feed by degrading chitin. They reproduce asexually and produce a body called a thallus, having rhizoids that take in nutrients on the skin. The thallus is contained in a shell that mature and later on produce zoospores, which are free-swimming forms (affecting metamorphosing tadpoles). The species degrading keratin is *B. dendrobatidis*, where the type specimen was collected in an isolate from the blue poison dart frog, *Dendrobates azureus* (LONGCORE *et al.*, 1999). By comparison of Morphology, histological section ultrastructural and 18s rDNA, the chytrids found in the Old World such as Australia are similar to samples isolated from the New World (North America) (DASZAK *et al.*, 1999). *Batrachochytrium* can also exist as a saprobe without any adult amphibian host. Because of that, it can exist in long-lived tadpoles in the wild and has the capacity to wipe out adult frog populations till extinction (DASZAK, 1998).

2.4. Factors influencing the spread of Chytridiomycosis

The fungus infect amphibians by feeding on keratinized cells specifically the stratum corneum and stratum granulosum without producing much immunological suppression. In Australia, half of 266 collected frogs for three years were infected with the fungus but they have not been infected with any other disease and there was no evidence of depressed immunity, as studied by BERGER and SPEARE (1999). Studied archival collections showed that the disease is present in North America in the 1970's and in Australia during 1978. The onset of international pet trade for outdoor pond stocking, importation for zoo collections and pest control brought introduced frog species in large numbers into different regions (DASZAK, 1998) such as *Litoria* in New Zealand and *Bufo marinus* in the Philippines.

Mass deaths did not occur in all cases of wild amphibians infected by Chytridiomycosis showing that some frogs are resistant and some are susceptible to the disease. The species *Bufo marinus* (sample from Maryland) and *Acris crepitans* (sample from Illinois) have been infected but without observed deaths (MILIUS, 1998). In experimental infections, exposure of susceptible species of frogs to greater than 100 zoospores in water for 12 hours produce 100% infection but more resistant species like *Rana catesbiana* have low incidence for infection even if infected with 5 million spores for five days. Although zoospores are abundant in water, tadpoles are harder to infect. According to Speare (personal communication, 2002), these do not show clinical symptoms or die; infection only happens in the teeth and this is usually shed off until juveniles turn into adults. It is only when the skin of the hind limbs and the tail become keratinized in metamorphosing frogs that infection occurs. Crowding in water sources such as ponds or streams during periods of environmental drought is said to increase transmission rates in Monteverde, Costa Rica (DASZAK, 1998). The same event happened in New Zealand when bell frogs, *Litoria raniformes* (introduced from Australia) were monitored during spring and summer of 1999–2000. Though a few individuals were found infected outside of the pond, majority of the metamorphosing tadpoles uncovered in the pond were dying (WALDMAN *et al.*, 2002). Field surveys in 1998–1999 found out that in Australia most of the infections happen near stream frog communities and the frogs appear healthy upon collection (suggesting that clinical symptoms come in a few days before death) (MAHONY, 2002).

2.5. Symptoms and treatment

The clinical symptoms are usually abnormal posture, lethargy and loss of righting reflex, lesions on the epidermis muscle and the eye leading to ulcerations, hemorrhages and sloughing (DASZAK *et al.*, 1999). Hypothesized mechanisms for death to occur include epidermal hyperplasia impairing cutaneous respiration and osmoregulation and or systemic absorption of fungus toxin. In susceptible species, death occurs between 18–48 days and most clinical signs appear a day or so before death. Since *B. dendrobatidis* do not grow at temperatures higher than 27°C, species mortality with higher temperatures is not 100% complete (Speare, personal communication, 2002). Treatment for juvenile frogs experimentally exposed to *B. dendrobatidis* was done by topical application of Itraconazole (Sporanox) or immersion in the drug with saline solution. Frogs that discontinued the treatment were not free of the infection until death but treated blue and yellow dart-frogs were free of the infection after continuous treatment of 11 days. Control frogs on the other hand died 35 days after exposure to the fungus (NICHOLS and LAMIRANDE, 2002).

3. Cellular Automata in Biology

Cellular automata (CA) provide simple discrete deterministic mathematical models for physical, biological and computational systems (WOLFRAM, 1982). This was conceived by Ulam and von Neumann in the 1940s to organize the investigation of complex extended systems (SIPPER, 1996). Basically, the CA is composed of an array of cells or programmed automata in grids or squares. All cells follow the identical rule or the transition function. This means that each cell evolves in discrete time periods assigned following a periodic boundary condition. The variable that separates each cell or lattice is its state that can either

be a number or property. For two-state CA's the numerical representative is either a 0 or 1 where 1 can be designated as dead and 0 as alive (as an example). Multi-state CA's can also be generated in response to more properties in a system (i.e. healthy, sick, removed). The cells are also allowed to interact with adjacent cells and called neighbors. One cell has left and right neighbors and a periodic boundary condition will be assumed (GREEN, 1993; SIPPER, 1996). Properties of CA such as self-organization allow patterns to emerge as successive states are expressed inside grids. This behavior of CA that is similar to that of biological systems added to its popularity in this field of science. Some other biological mimics include 1) dying out, 2) becoming stable or moving in a cycle with fixed period, 3) growing indefinitely at a fixed speed, and 4) growing and contracting regularly. These factors are considered in the rules for artificial systems.

In studying epidemiology CA concepts were incorporated to indicate the behavior of a disease agent in a host population which is not homogeneously affected by the disease over time. Because of this, patterns emerged showing how actual infections emerge from a particular disease dependent dynamics on the level of the respective local units of infection. This is simply a fixed number of consecutive states (i.e. healthy, infected and removed) that are changed periodically depending on the effects of neighborhood infections. Consequently, a spatially explicit, grid-based simulation model links aspects of local agent transmission between individual animals to aspects of the regional spatiotemporal spread of an epidemic (THULKE *et al.*, 1998).

4. Methodology

There were two kinds of CA applied to 3 kinds of simulations that showed how Chytridiomycosis spreads within the amphibian populations. These were done in conjunction with an actual case study in Chytridiomycosis epidemiology. Deterministic CA is where the outcomes of the cells that underwent transition produced a repeating pattern and was used to simulate behaviour of the disease in 1) susceptible and 2) resistant frog populations. A Stochastic CA on the other hand does not produce a regular pattern when the simulation runs and was used for 3) heterogeneous frog populations that underwent drug treatments.

A cell which is the basic element of a CA was given one state that may evolve in 1 transition period (r) (see Table 1) and was arranged in a spatial web called a lattice; a set of finite grids. For our models, we made use of a one-dimensional lattice. These CA models had multi-states (symbolized by 0, 1, 2, and 3) with periodic boundary conditions and made use of the von Neumann neighborhood. In this neighborhood, an active cell or a cell in question can possibly interact with its four neighbors. Since we used one-dimensional CA with a von Neumann neighborhood, then the transition state of the active cell was influenced by its left and right neighbor. But for a two-dimensional CA with the same neighborhood condition, the active cell interacts with the cell above it, below it and on the left and right side of it. Construction of the CA models was implemented as each of the multi-state dimensions were symbolized by a number which codes for a cell property indicating a real event experienced by affected amphibian populations like varying ranges between sickness (P) and recovery (R) (see Table 1).

Dynamics of the population was observed as each state was allowed to undergo a periodic transition state in response to the disease. One transition period (r) has a time

Table 2. CA rules to give the next state of the cell (the middle number). The asterisk means any state except 0. “Model depend.” means that the state is dependent on respective model, especially on the relationship of P and R involved in the model (see Sec. 4.1).

Neighborhood	State ($r = i$)
000	0
00 or 00	1
0	1
1	2
2	Model depend
3	”
010, 020, 030	”
01*, 02*, 03*	”

equivalent (t), where t is equal to 1 week or 7 days. In one transition (r), a cell proceeds at an “ i ” times step according to a finite set of rules that can be outlined (see Table 2). This set of rules also act as a lookout table that makes the change of state for an active cell automatic. The first column shows the neighborhood of the middle cell and the second column shows its state after i transition period.

Models were done by constructing grids of 25×25 squares in Excel software where each cell has been assigned a random initial state and expressed in the corresponding symbol of Table 1. Graphs of affected populations versus time were done to complement the Excel grid-based models.

The basic assumptions for all models included space limitations that subjects populations to overcrowding thus increasing infection rates. Quiescent stages of the disease in frogs that show no symptoms of infection were incorporated in the model. There was no fixed number of individuals for a population since 1 cell was assumed to have an “ n ” sample of a population that changes its state. Critical periods for increase in recovery or sickness was put between $t = 7-14$ days or $r = 2$ and once this was surpassed by a population, easy recovery and stability of health was achieved at $t = 36$ days.

4.1. Spread in susceptible populations (SP's)

Background case study

In the multi-state CA the addition of death as a property was linked with the species extinction phenomenon if it occurred in large numbers of a population. *Taudactylus acutirostris*, the Australian sharp-snout day frog was currently a missing frog species in the wet tropics. Updated news listed it as extinct in the wild, as the last specimen died of Chytridiomycosis infection in 1995 while in captivity (SPEARE and BERGER, 2000b). The disappearance of the golden toad of Costa Rica and the 2 species of gastric brooding frogs in Australia was also linked with Chytridiomycosis (SPEARE and BERGER, 2000a).

Assumptions

The requirements for this model are species having small clutch size and small geographic range with the tendency to overcrowd in streams and ponds. The equation for this is given as

Table 3. Rules for CA model of SP's.

Neighborhood	State ($r = 1$)	Conditions ($P > R$)
000	0	No variation
0, 00*, *00	1	No variation
1, 01*, *10	2	No variation
02*, *20	3	No variation
2, 03*	3	If 3 repeats 2 transitions, cell dies out

Table 4. Rules for CA model of RP's. After the critical period of $t > 14$ days, cells state oscillate between healthy or infected depending on the state of their neighbors.

Neighborhood	State ($r = i$)	Conditions ($P \leq R$)
000	0	No variation
00*, *00, *0*	1	No variation
1, 01*, *10	2	Revert one step to healthy after critical period of $t > 14$
2	2	"
02*, *20	2	"
020, 010	2	Turn to 0 at $t > 14$, $r = i + 3$

$$P > R, \quad (1)$$

where P and R represent the sick period and recovery period, respectively.

4.2. Modeling spread in resistant populations (RP's)

Background case study

One of the observed resistant species to Chytridiomycosis was the American bullfrog *Rana catesbiana*. Having a cosmopolitan distribution, this amphibian is highly introduced to many areas of the world and was deemed to have been originally from the Americas. Studies showed that these frogs possessed low rates of infection against Chytridiomycosis and no deaths in the population were observed that was linked to the disease (Speare, personal communication, 2002).

Assumptions

The requirements for this model were populations having a broad range of adaptability to a wider range of habitats and microclimates. Many individuals may be carriers of the fungus but do not exhibit symptoms (state of 1 = carrier, quiescent stage). Thus relationship of P and R was shown as $P \leq R$. Sickness occurs in a short period and recovery period is at least equal to the time of sickness.

4.3. A stochastic model of Chytridiomycosis for drug treated captive populations (DTP's)

Background case study

Frogs are quarantined for 2 months before they are added to a collection (MARANTELLI and BERGER, 2002). Studies on topical drugs like Itraconazole showed the variation of some properties in this system.

Table 5. Rules for CA model of DTP's.

Neighborhood	State/Transition	Conditions
Random No. 0	Increase in R	Effective treatment (recovery)
Random No. 1	Increase in P (relapse)	Ineffective treatment (re-infection)
3, 030, *30	3	Dies out, if repeated at $r = i + 3$
000	0	Remains healthy until the end, repeated at $r = i + 2$

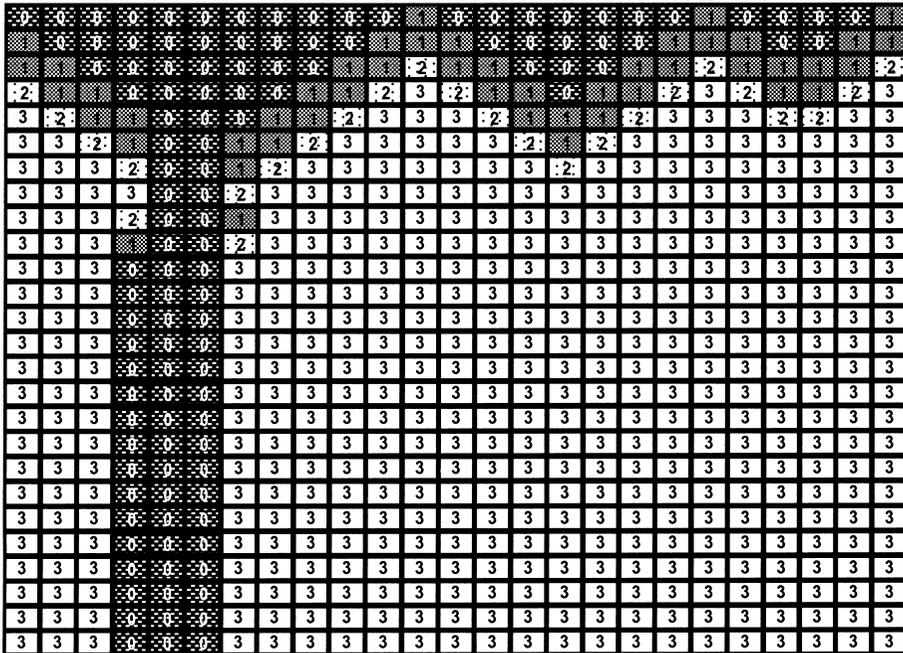


Fig. 1. Deterministic CA model for susceptible frog populations simulating the dynamics of Chytridiomycosis exhibited by cell states: 0 = healthy, 1 = carrier, 2 = infected, and 3 = terminally ill/dead.

Assumptions

Infected frogs can be restored to the healthy state with drug treatments. But medication has varied affects on individuals having different states so that it does not guarantee that 100% of a population under treatment recovery fully although the chance is always 50/50. Because of this we let $P = R$ for this model.

By using a 2-number generator in a computer, either 0 or 1 will be chosen and this will dictate if the frog population will take one step to recovery or sickness again. This is a random event that will simulate if drug administered to a frog sample population was effective leading to restoration of the healthy state or ineffective leading to re-infected state. We assign in our generator 0 if the population assumes one step towards recovery and

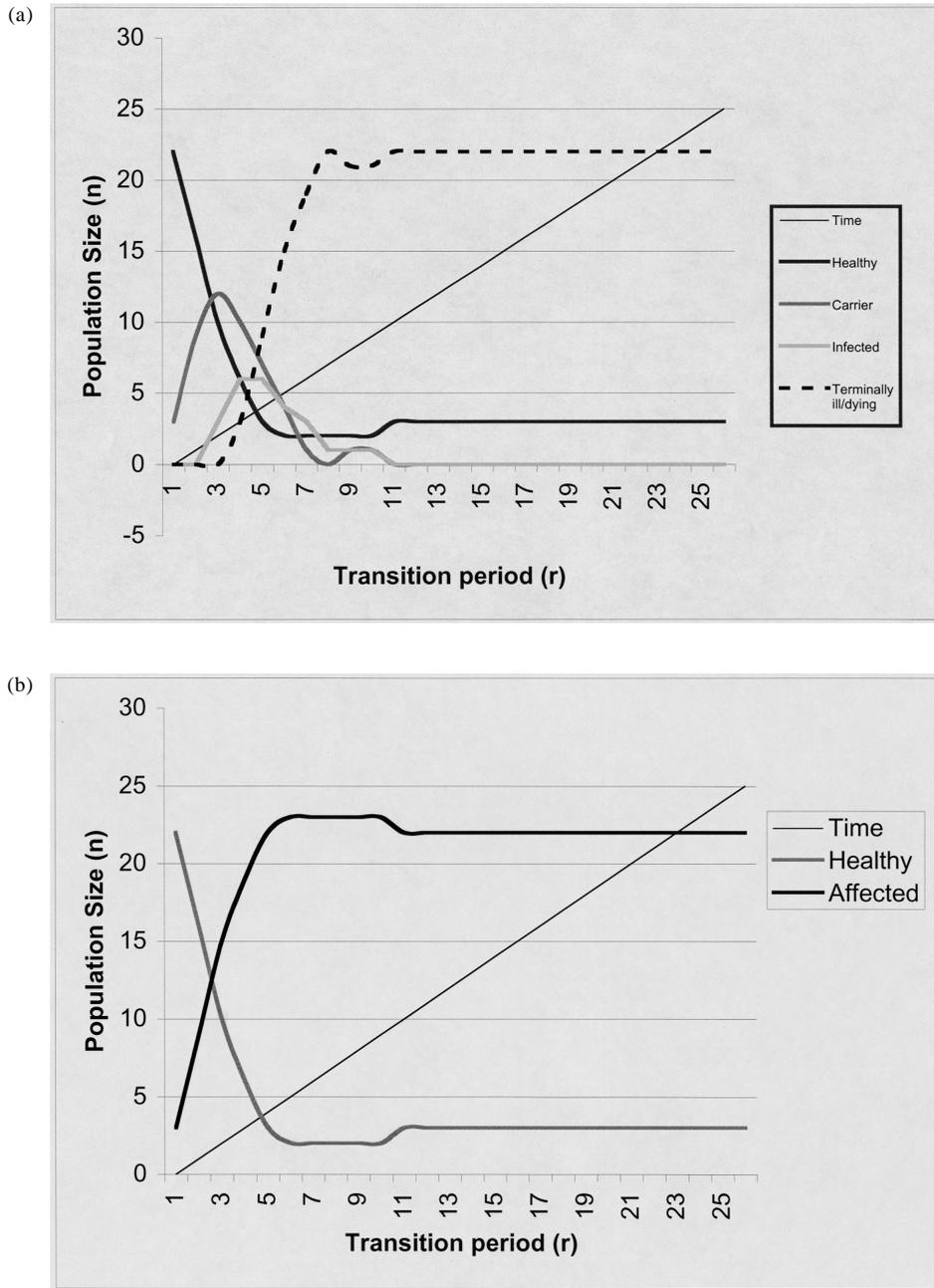


Fig. 2. (a) Graphs showing the change of states in a susceptible frog population (derived from Fig. 1) with the trends in the behaviour of healthy, carrier, infected and terminally ill/dying frog population over increasing time. (b) Dark curve showing all affected populations summed from (a) and lower curve of the healthy populations over increasing time.

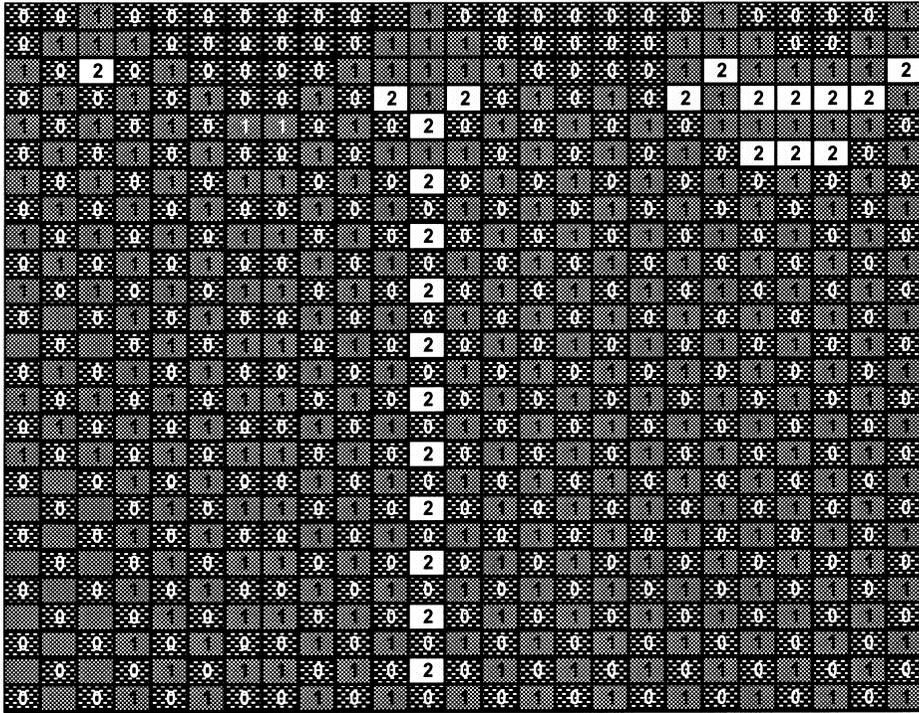


Fig. 3. Deterministic CA model for resistant frog populations simulating the dynamics of Chytridiomycosis.

1 if the population assumes one step towards becoming more ill. If the treatment underway was working, it meant that the cells changes one-step for one transition period to become healthy. If the drug was ineffective, then the cells were changed to the dying state unless the randomizer generates a 0. There is an assumption that there is limited neighborhood interaction during the course of treatment and that the change of states can be reversible. It means that the possibility of reverting back to a sick state was considered in the model. This procedure simulated direct human intervention applied to captive populations. A simple coin toss can also generate 2 random events, heads (for recovery) and tail (for sickness) for simple CA grid models.

5. Results and Discussion

Simulations from Fig. 1 are one-dimensional deterministic CA model for susceptible frog populations where an initial number of individuals were added randomly (i.e. some are healthy, some are sick) at $t = 0$. By following the set of rules and variations for this CA, it was shown that the rates of infection progressed on exponential increase (Fig. 2(b)), and there was a plateau at $t = 7$ till $t = 9$ and a gentle drop after the 9th week. From this CA model,

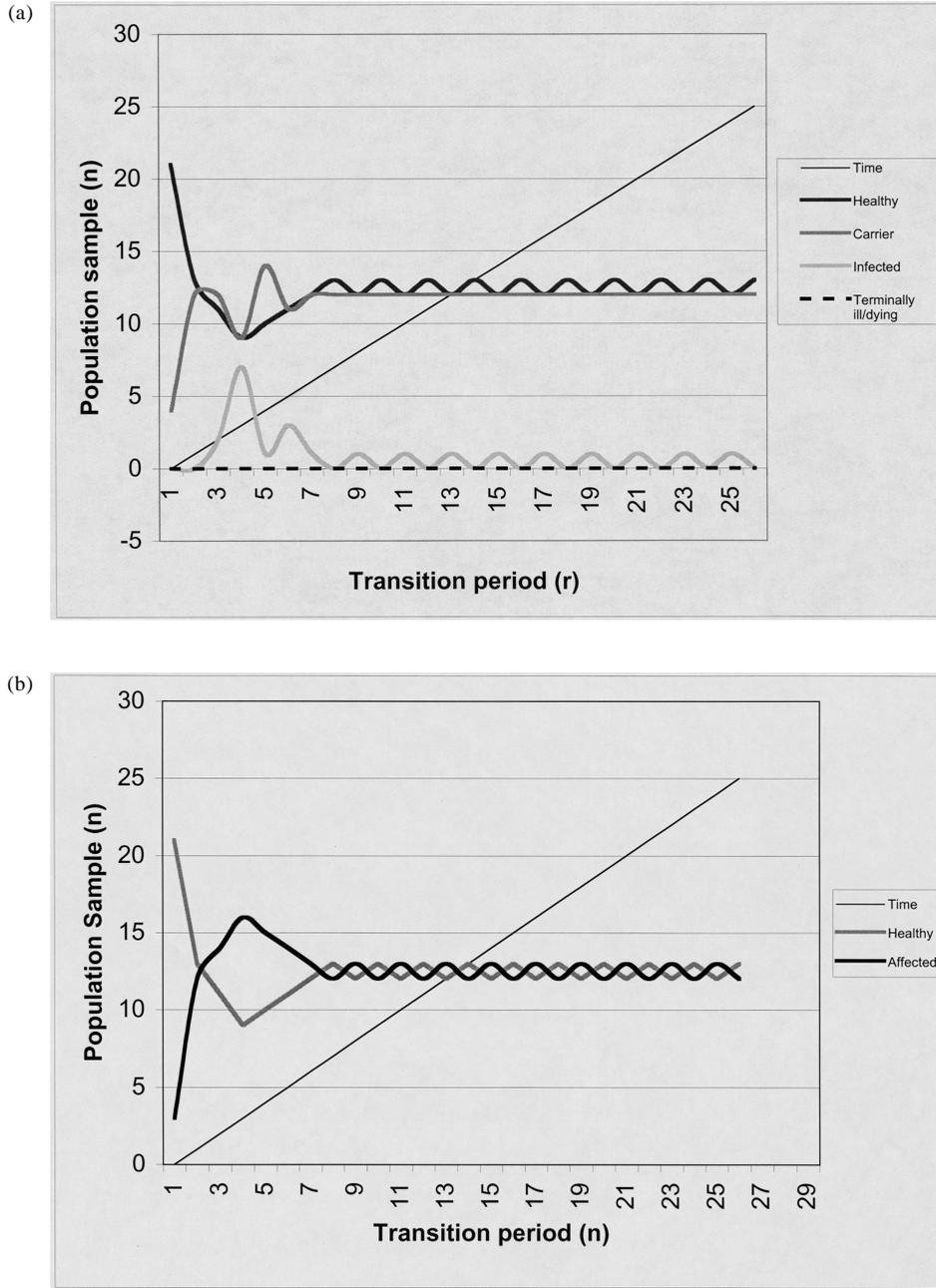


Fig. 4. (a) Graphs showing the changes in states of resistant frog populations (derived from Fig. 3) with the trends of behaviour in healthy, carrier and infected frog population over increasing time. (b) Dark curve showing oscillatory trend of the sum of all disease affected frog populations derived from (a) and lighter curve showing the same oscillatory trend in healthy populations over increasing time.

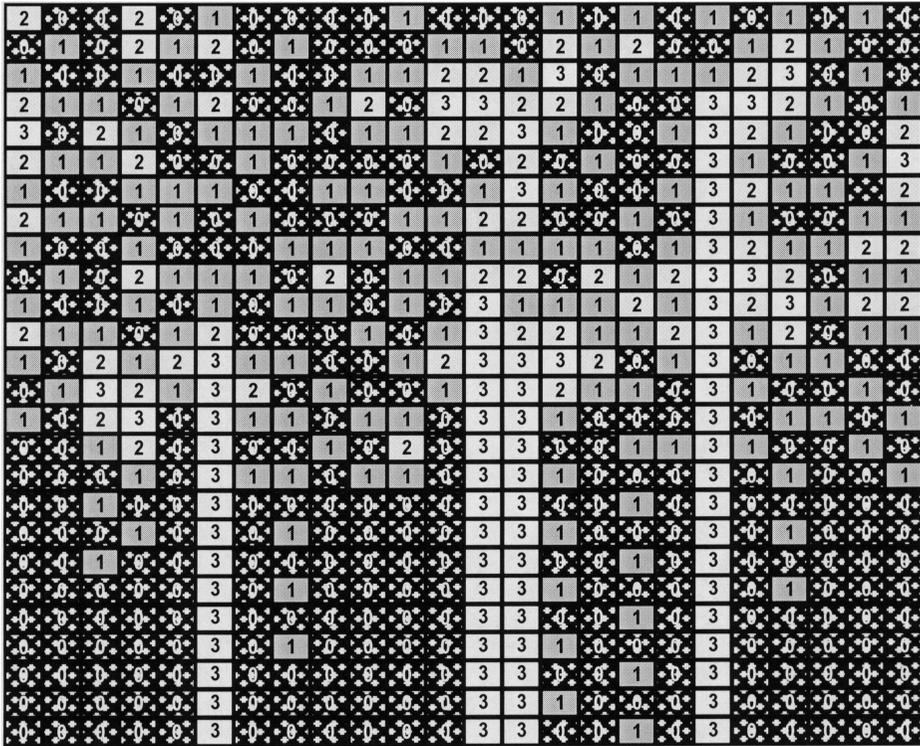


Fig. 5. Stochastic CA model for captive frog populations simulating the dynamics of Chytridiomycosis.

population size (n) of healthy frogs sharply decreased after 5 transition periods or from $t = 0$ to $t = 5$. This was also evident as the rate of dying frogs increased in increments following 2 uneven oscillations until the 9th week when it demonstrated a plateau. Only a few samples of the population remained healthy (there were only 3 cells left). The numbers of carriers and locally infected frogs also declined as the former event was carried out. In general the model followed a Pascal triangle increase, which was shown by the triangular configurations in Fig. 1.

Demonstrations of Fig. 3 are one-dimensional deterministic models that showed the spread of Chytridiomycosis in resistant frog populations, where at $t = 0$ population size (n) for 25 cells of the GBM contained infected samples chosen randomly. The spread of infection initially followed the Pascal triangle increase pattern from $t = 0$ to $t = 4$ that was proven by the exponential curve of the graph for this model (Fig. 4(b)). But as time increased after the fourth transition period, the spread rates followed a periodic uniform pattern. What resulted was a graph that had regular oscillatory motion for both healthy and carrier individuals (Fig. 4(a)). Dying cells in the model were not observed even if few cells reached the infected state. These were able to revert into a healthy state in one transition

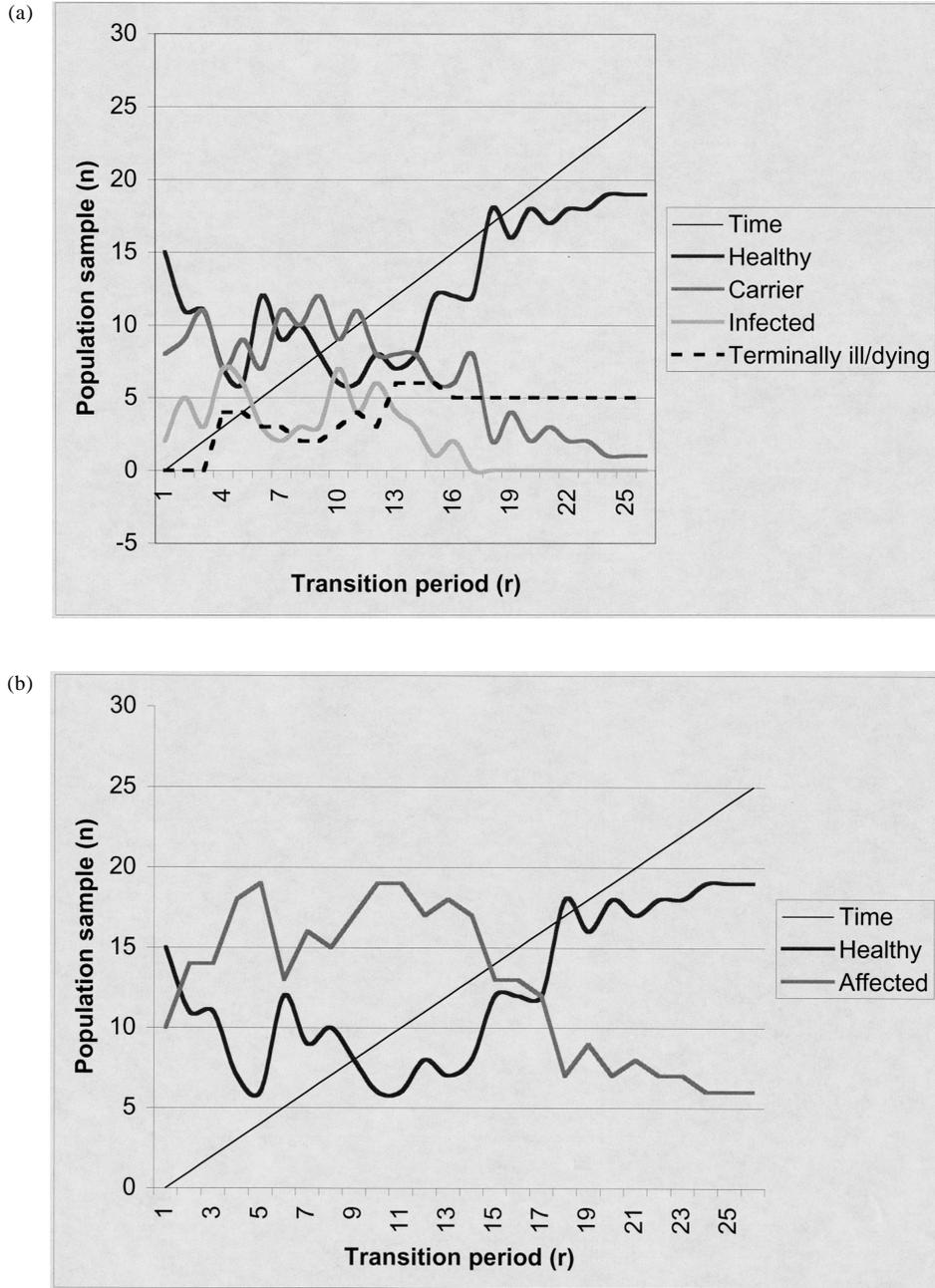


Fig. 6. (a) Changes in states of frog populations subject to random drug treatments (derived from Fig. 5). (b) Rates of Infection in Frog Populations summed from (a).

period (lasting 1 week) as the rules were applied this model.

A stochastic CA was modeled by Fig. 5 where random order was achieved by randomizing (such as following a coin toss system) to predict population sample (n) tendencies for recovery or sickness. The model did not exhibit regular patterns of spread for Chytridiomycosis. At the end time period when the simulation was run ($t = 25$), it was shown that 20% of the infected cells died and 80% were restored to the healthy state. The graph of this model (Fig. 6(b)) showed that infection rates followed a steep increase but declined at the 5th to the 9th week, increasing shortly after that and climbing down till the rates of infection was lowered to allow half of the infected cells to recover. Changes in the population size were noted to increase over time for healthy populations and decrease at one step from both infected and carrier samples. The dying cells/samples graph exhibited a plateau parallel to the time that total infection rates were decreasing at after 17 periods or 17 weeks.

The type of population dynamics manifested in these models all showed exponential growth in infection rates at the start of simulation. But their differences were the emergence of uniform periodic patterns for resistant populations, decline in rates for treated populations and the plateau behavior of the graph for susceptible species. In any biological system, wave fluctuations are a function maintained within a tolerable range of limits. Oscillations then produce wave periods of stability and instability running along those limits. MURRAY (1999) was able to show this by his discussion of local perturbations in populations and said that a steady state population is unstable if the small perturbation found does not reach zero. If we connect this with the model results for resistant species, oscillatory behavior was found occurring at regular intervals such that the amplitude of each wave in the graph cancels out the other. If this would be plotted as a phase plot graph, it would show an elliptic figure equivalent to stable system configurations. This proved that such a system gained a steady state after the 8th week where some population samples were carriers and some reverted back to the healthy state. Chytridiomycosis at this stage had no significant effects on populations of resistant frogs. For susceptible and drug treated populations, exponential rise of infected samples/cells were due to increased transmissions which may have been brought about by an assumption of overcrowding although it does not answer the biology behind species susceptibility. A real example is the case of mass deaths of frogs in Costa Rica during a period of drought where most frogs overcrowded in ponds.

By categorizing the CA type according to Wolfram's categories of one-dimensional CA's, Class 1 governs the CA model for susceptible populations (SP), Class 2 governs the CA model for resistant populations (RP) and Class 3 is fitted for captive populations (DTP) that were given drug treatments for the disease. Class one governs models for SP's since the automata changes into a fixed configuration after a finite number of transition states occurred. This was perceived when the graph for infected population versus time became a flat line. In the model, it was seen when 23 out of 25 cells died out. Class two is found for RP's since the configuration of this automaton followed a periodic cycle or fixed configurations observed in the oscillatory behavior of the model's graph. It is also periodic but end result of the simulation that produced a heterogeneous group of healthy and carrier frogs. For DTP's the configuration is chaotic and aperiodic producing a class three automata that may not be necessarily random at all.

6. Conclusion

Using one-dimensional cellular automata (CA) with periodic boundary conditions and multi-states, patterns of spread of Chytridiomycosis were modeled in three genres of frog populations (susceptible, resistant and drug-treated). It was shown that there are three classes of CA's governing the three kinds of models presented. These models were influenced greatly by the relationship of recovery (R) and sickness (P). Spatiotemporal dynamics was outlined by models of transmission rates in neighborhoods that were affected by proximity (shown by Pascal triangle patterns). In the process of simulation, susceptible populations died out, while resistant and treated populations recovered or stayed healthy. Resulting fluctuations of the graphs these models provide ascertained a quantitative description of the relationship between recovery periods versus periods of sickness, two variables that are significantly measured in epidemiology. Simulation results show that the spread of Chytridiomycosis followed exponential patterns for SP's with incidence of regular oscillatory behavior for RP's. The treated captive population model showed that drug treatments brought significant change in curbing the infected population growth.

These cellular automata-based models can be used to analyze the behavior of emerging infectious disease (EID) in wildlife and its control to develop a testable hypothetical study on the effects of the disease in frogs and amphibians as a whole. It also stresses the significance of biodiversity conservation and environmental education.

Applications of Cellular Automata in the analysis of visualization models for complex systems is an alternative to learn population behavior and create possible scenarios that will highlight the significance of simulated transmission experiments in epidemiology. This is building on the capability to predict disease pathogenicity variations depending on type of species, tempo-spatial relationships and effect of human intervention through controlled experiments and treatments.

7. Recommendations

The produced simulations should be subject for further review by those doing experimental studies on the epidemiology of Chytridiomycosis. Similar simulations may be done using the Ateneo's AGILA high performance computer as well as the use of programming tools (Mathematica). More information is required about the fungus *B. dendrobatidis* and the review of the life cycle features of this fungus to further improve the models shown. It is also important to incorporate the frog's life cycle for future models so that each time period can be described in terms of biological events pertinent to the stages of growth and development of frogs as a biological component of the disease dynamics system.

The present authors would like to thank Dr. Richard Speare of James Cook University, Australia and Dr. Jean-Marc Hero of Griffith University, Australia for their valuable insights and contributions. They also thank Dr. Merab Chan, Dr. Henrietta de la Cruz, Dr. Norman Dennis Marquez of the Ateneo de Manila University Biology and Mathematics Department, CHED-COE,

the Vice President's office of the Loyola Schools and MPB2002 organizers for the acceptance of this paper for conference poster presentation.

REFERENCES

- ABC NEWS ONLINE (2000) Frogs Disappearance Baffles Scientists, <http://www.abc.net.au/news/features/stories/20000120frogs1.htm>
- BERGER, L. and SPEARE, R. (1998) Chytridiomycosis: a new disease of wild and captive amphibians, *ANZCCART Newsletter*, **11**(4), 1–3, <http://www.jcu.edu.au/school/phtm/PHTM/frogs/chynew.htm>
- BERGER, L. and SPEARE, R. (1999) Update on Amphibian Chytrid in Australia, <http://www.jcu.edu.au/school/ohtm/PHTM/frogs/chynew.htm>
- DASZAK, P. (1998) Chytrid fungus and frogs: background on worldwide amphibian disease (unpublished).
- DASZAK, P., BERGER, L. CUNNINGHAM, A., HYATT, A., GREEN, E. and SPEARE, R. (1999) Emerging infectious diseases and amphibian population declines, *Emerging Infectious Diseases*, **5**, 735–748.
- DASZAK, P., CUNNINGHAM, A. and HYATT, A. (2000) Emerging infectious diseases of wildlife-threats to biodiversity and human health, *Science*, **287**, 443–449.
- ENVIRONMENTAL NEWS NETWORK (2001) Fungal Enemy Could Explain Worldwide Amphibian Die-off, January 15, 2002, http://www.enn.com/news/ennstories/2001/08/08232001/fungal_44732.asp
- GREEN, D. (1993) Cellular Automata, Environmental and Information Sciences, Charles Stuart University, <http://life.csu.edu.au/complex/tutorials/tutorial1.html>
- HERO, J. M., WILLIAMS, S. and MAGNUSSON, W. (2002) The ecological characteristics of declining frogs in Australia: Implications for global amphibian declines, Endangered Frog Research Center, School of Environment and Applied Science, Griffith University, Australia.
- LONGCORE, J., PESSIER, A. and NICHOLS, D. (1999) *Batrochochytrium dendrobatidis* gen. et sp. nov., a chytrid pathogenic to amphibians, *Mycologia*, **91**, 219–227.
- MAHONY, M. (2002) Prevalence of chytrid in populations of frogs in eastern New South Wales, *Proceedings from Scientific Conference on Getting the Jump on Amphibian Disease*, <http://www.jcu.edu.au/school/phtm/PHTM/frogs/GJMahon.htm>
- MARANTELLI, G. and BERGER, L. (2002) Quarantine methods for captive collections, *Proceedings from Scientific Conference on Getting the Jump on Amphibian Disease*, <http://www.jcu.edu.au/school/phtm/PHTM/frogs/GJMarBer.htm>
- MILIUS, S. (1998) Fatal skin fungus found in US frogs, *Science News*, **7**.
- MURRAY, J. D. (1999) *Mathematical Biology*, 2nd corrected ed., Springer-Verlag, Berlin Heidelberg, U.S.A.
- NICHOLS, D. and LAMIRANDE, E. (2002) Treatment of cutaneous chytridiomycosis in blue and yellow poison dart frogs (*Dendrobates tinctorius*), *Proceedings from Scientific Conference on Getting the Jump on Amphibian Disease*, <http://www.jcu.edu.au/school/phtm/PHTM/frogs/ginichol.htm>
- SCHRAG, S. and WIENER, P. (1995) Trends Ecology and Evolution.
- SIPPER, M. (1996) A Brief Introduction to Cellular Automata, Computer Based Learning Unit, University of Leeds.
- SPEARE, R. and BERGER, L. (2000a) Global Distribution of Chytridiomycosis in Amphibians, <http://www.jcu.edu.au/school/phtm/PHTM/frogs/chyglob.htm>
- SPEARE, R. and BERGER, L. (2000b) Chytridiomycosis in Amphibians in Australia (unpublished).
- TENENBAUM, D. (1998) *Disappearing Frogs*, <http://whyfiles.org/shorties/frog-fungus.html>
- THULKE, H. *et al.* (1998) Cellular automata in epidemiology—a modeling concept and a wildlife disease, March 20, 2002. [iinwww.ira.uka.de/ca/moca-98/abstracts/thulke.ps](http://www.ira.uka.de/ca/moca-98/abstracts/thulke.ps)
- WALDMAN, B. *et al.* (2002) Discovery of Chytridiomycosis in New Zealand, *Proceedings from Scientific Conference on Getting the Jump on Amphibian Disease*, <http://www.jcu.edu.au/school/phtm/PHTM/frogs/giwaldma.htm>
- WOLFRAM, S. (1982) Cellular Automata as Self-organizing Systems, <http://www.stephenwolfram.com/publications/articles/ca82-cellular/index.html>
- WOLFRAM, S. (1984) Cellular automata as models of complexity, *Nature*, **311**, 419–424.