

Evolution as Learning Yields Hyperbolic Discounting

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Abstract

Learning is modeled as an infection, which jumps from person to person. The rate of infection mimics individual discount rates and induces savings behavior on its own. It is shown that the apparent discount rate, the combination of the agents' true discount rate and the infection rate, decreases over time and approaches the agents' true discount rate. This decrease, known as hyperbolic discounting, is consistent with what is observed in psychology studies, while the limiting case, exponential discounting, is consistent with market level observations. This model closes the gap between individual and market level observations of discounting behavior without explicitly assuming the two kinds of discounting nor relying on commitment mechanisms.

1 Introduction

Beginning with the clinical experiments of Tversky and Kahneman [Tve81] hyperbolic discounting has been observed in numerous experimental settings and has regularly worked better than other discounting models at explaining the observed behavior. It is referred to as, " [the] best documented DU [discounted utility] anomaly " in a recent Journal of Economic Literature survey article[FO02].

Hyperbolic discounting refers to one explanation of the empirical regularity that people act as if they had declining discount rates over time and that they don't seem to follow the "equal difference" rule that exponential, constant rate, discounting implies. If given a choice between a low payoff in the future and another higher one a little later, they will chose the higher, more delayed payoff; but when the choice is brought forward, closer to the present, people change their minds and choose the smaller more immediate payoff.

This behavior may not be a primitive, but rather a derived behavior. Read [Rea01] suggests that hyperbolic discounting may be the result of subadditivity. The argument is that as a stretch of time is subdivided, the amount of discounting increases. This means that the amount an agent will discount a payment in two years depends on if they conceive of it as one two-year period or two one-year periods. Subadditivity can manifest itself as hyperbolic discounting when agents are discounting the future from a fixed now. In the example above, the single two-year period is compared to one of its halves, a one-year period. It then appears that the discount rate falls as the time horizon is increased- an observational equivalent of hyperbolic discounting.

Economics is left with the problem of rationalizing these empirical, experimental observations with the equally good explanatory power of exponential discounting when it is applied to large financial markets. Financial markets have institutionalized exponential discounting by expressing the returns on securities as an annual figure. Economists have also put their blessing on exponential discounting through almost universal use in financial and macroeconomic modeling.

There is a contradiction: individual behavior is better explained by hyperbolic discounting while the behavior of markets is more commonly explained by exponential discounting. It is as if the whole does not equal the sum of the parts. Since our market models are ultimately based on intuition about individual behavior, they must not be contradicted by the facts of individual choice or they must demonstrate a mechanism where individual, observationally, hyperbolic behavior through interactions in the market is observed as exponential. Justifying individual hyperbolic discounting with market level exponential discounting is important to the completeness of the model.

Justifying the contradiction between the individual and market discounting behavior will also provide insight into the functioning and utility of markets in facilitating exchange. If these two discounting methods can be justified in the same model, we can have some assurance that financial markets are not merely a more cost-effective way of trading assets, but that they perform other functions. Financial markets may be the mechanism that allows for time consistent planning that would not be possible without their existence. As a result, financial markets may encourage transmission of ideas, serve as experimental labs for new ideas in valuation, or any one of a dozen different services that go beyond reducing transactions cost.

Models of individual rationality that produce exponential discounting from a hyperbolic discounting typically involve hypothesizing many individuals within each of agent. Each of these individuals is in control for a while, but is aware that, in the future, an alternate self will be in charge of the decision-making[Azf99]. Exponential discounting, and the associated time consistent plans, arise because the individual in charge uses commitment mechanisms, e.g., investments with exit costs, so the future selves will be forced to accept the plans[Lai97].

That methodology takes hyperbolic discounting as a given and then rationalizes other observed exponential behavior with commitment mechanisms. This is one possibility, but there are few exit costs associated with modern financial

markets; stock trades can be made for less than \$8. Commitment mechanisms may be a useful explanation for some investments, notably real estate and tax deferred retirement accounts, but it lacks explanatory power in the broader financial markets. This family of models fails to explain the discounting phenomena with mechanisms that can be realistically observed in the economy. Moreover, they assume the two particular forms of discounting, instead of allowing them to arise as a consequence of more primitive assumptions.

This paper will refrain from assuming hyperbolic discounting and instead explain how it arises naturally in an evolutionary system with agents similar to those that populate a typical dynamic macroeconomic model. An evolutionary system that has two parts, an individual with a classically defined objective function, and a pool of potential policy rules, produces dynamics that are much different than those found with non-evolutionary solution methods. These dynamics mimic the development of hyperbolic discounting and the eventual resolution into exponential discounting.

While this may initially look like just another way of solving a dynamic programming problem, in actuality it is far from it; this method is a dynamic model in its own right. It combines the dynamics of capital accumulation, that the other models explore, with the dynamics of learning about how the markets work. This means that the model will be more applicable in “odd” times, when market rules or the structure of the economy are in flux, when they are expected to be qualitatively different than the present. So, while the typical models work well when explaining market behavior when the uncertainty about the future is only quantitative, i.e., different inflation rates, or productivity growth rates. Evolutionary models will be more effective when uncertainty about the future is qualitative. This has direct applications where the rules of financial markets are changing and evolving, such as the increasing world capital markets integration and the increasing introduction of new financial instrument. It also has applications to explaining the current behavior of US capital markets given the rapidly changing structure of our economy.

The key abstraction is to disentangle observed discounting from the preferences of the individual. Observed discounting is a consequence of the imperfect rules the agents use to make decisions. These rules are a product of the agents’ own preferences and the learning process that produced the rules. Learning lends its own flavor to the observed discounting behavior.

Section 2 will describe the modeling methodology. It will lay out the steps required in the simulations and describe the modeling options of each of the steps.

Section 3 will show how explicitly non-forward looking agents develop, or learn, forward-looking behavior in a viral evolution system. This will be demonstrated by constructing a model where agents only care about consumption today. This system will be expanded in section 3.2 to show the evolving behavior of agents that are explicitly forward-looking with exponential discounting. Discounting will evolve because even if agents don’t care about consumption tomorrow, survival and spread of behaviors will occur tomorrow. The behaviors that involve saving will produce more utility next year, thereby surviving better

and spreading better through the population. Over the years, these behaviors may become widespread. This section may be interpreted as an exploration of the limiting behavior of the evolutionary system.

Section 4 will introduce the concept of hyperbolic and exponential discounting. The definitions in this section will be used in the section that follows to describe the implied discounting behavior of the consumption rules that arise in this evolutionary system.

Section 5 will take a look at the interaction of the classically defined individual with exponential discounting and the pool of potential policy functions. In this section, we will demonstrate that evolutionary systems evolve hyperbolic discounting that eventually resolves into exponential discounting. This may be interpreted as an exploration of the implied discounting dynamics of the evolutionary system.

2 Modeling Methodology

The first step in demonstrating that a viral evolutionary system can induce forward looking behavior in agents that have no preferences over future consumption is to state the objective function of the agents that the viruses will be infecting. This can be a static or a dynamic optimization problem, it does not matter. For this initial example we will use a simple deterministic dynamic programming problem with a few twists.

$$\begin{aligned}
 V_t &= \max_{c_t} \ln(c_t) & (1) \\
 s.t. & \\
 k_{t+1} &= k_t^0 + (1 - \delta)k - c_t \\
 k_0 &= K
 \end{aligned}$$

In the problem shown above as equation 1, the agent chooses consumption in a representative agent problem with production. The capital transition equation is dependent on the current capital stock, k_t , capital's share of income, θ , the depreciation rate, δ , and current consumption, c_t . What makes this problem unusual is that the agent does not care about the future. Only current consumption enters the objective function; future consumption is not considered.¹ For this particular problem, the optimal policy function is obvious; the agent should consume the entire capital stock and production in the first period leaving nothing for the periods that follow.

The traditional approach to this kind of problem is to have the agent "solve" the dynamic programming problem, either by finding the value function or the policy function that maximizes the objective function. In this case the dynamic programming problem will be solved by evolving a good policy function solution to

¹In the actual simulation the utility function is modified so there is a small lower bound on consumption, 0.01, which yields a zero utility result. This modification is useful in later simulations so that younger agents are not immediately better off than older agents and so that the "probability of death" interpretation of the discount rate is accurate.

this dynamic programming problem. The policy function will have a simple fixed functional representation.² The functional form of the policy function is shown as equation 2 below. The two parameters of the policy function are, C , and, α , which can be conceived of as autonomous consumption and a transformation of the marginal propensity to consume.

$$c_t = C + \alpha k_t \tag{2}$$

Each policy function will be represented by its two parameters. As we discovered above, the optimum policy function is $c_t = (1 - \delta)k_t + k_t^\theta$, where the agent consumes all wealth.

These rules will evolve similar to the way viruses evolve. The defining image is of an individual that consults his own consumption rule every period and slavishly follows it. When the agent is doing well with the rule, the agent informs others, teaching others how he is making the consumption and savings decision. Agents that do well evangelize particularly strongly, telling more people about their policy function. Other agents in the economy have a chance of hearing the news about the policy function and they may alter their own policy functions in light of this new policy function. The new policy function will then be used by the agent and propagated in the same way as the first.

This is analogous to the functioning of a physical virus. They exist in our bodies, using our own cells to produce copies of themselves. As individual cells become filled with virus copies they burst distributing the virus and viral cysts to other cells in the body and to other individuals. Viruses that are particularly good at propagating themselves don't kill their host, but keep the host functional and in contact with other potential hosts. That is why the common cold is such a successful virus, it does not kill the host right away, and keeps the host just sick enough to produce a large number of viral cysts, but not sick enough to keep it away from all social contact. This process needs to be operationalized and brought into the context of an evolving consumption policy function.

The steps required to evolve viral policy rules are relatively simple. First, the agents are initialized with random policy functions, and these policy functions are placed in the pool that will later be used to infect the individuals. Thereafter the simulation follows these steps:

1. The agents use their current policy functions to make consumption decisions.
2. Using the utility result of these consumption decisions, the rule for cyst production determines the number of viral cysts, or copies of the agents' policy rule, that the agent will cast off and add to the pool. Higher utility makes for more cysts.
3. These cysts are added to the pool.
4. The rule for catching a new virus is then applied to each of the agents.

²Evolving the functional form needlessly complicates the demonstration

5. If an agent is infected, a virus is drawn from the pool and passed to the agent.
6. The agent then uses the rule for melding the virus with their current policy function to create the new policy function that will be used starting in the next period.
7. Agents that are determined to have died, by running out of capital, are given a new policy function from the pool and another dose of capital.
8. Finally, every virus in the pool is subject to the viral decay rule to determine if they should be removed from the cyst pool.

The process then repeats itself until some convergence criteria on the population of viruses in the pool are reached. This is the pattern of all viral evolution systems. This system is different from the evolution used in classical agent based genetic algorithms. This evolutionary system describes the evolution of a policy rule that cannot propagate itself and is not tied to any one organism. It's the story of a symbiot. The mutation and cross-over functions are still in place but selection is much different, and additional operator, infection, has been added.

There are some specific characteristics that need to be defined in this system. These characteristics revolve around both the agents in the simulation and the pool of viral cysts that infect the agents. The primary characteristics are:

1. The number of agents in the simulation.
2. A rule for cyst production.
3. A rule for catching a new virus.
4. A rule for melding the new virus with the existing policy function in an agent.
5. A method for removing dead agents from the economy.
6. A rule for the survival of an individual virus in the pool of cysts.

Some of these characteristics have little effect on the terminal result; they can be changed freely. Others provide extremely subtle effects that can have a large effect on which policy functions evolve in the system. Each of these characteristics is described below and related to the selection, mutation, crossover, infection paradigm.

2.1 The number of agents in the simulation

The number of agents in the simulation is one of those factors that does not make much of a difference in this context. If the agents actually interacted in the goods market, as they would in a heterogeneous agent problem, rather than just the market for ideas, then the number of agents would be important since

that would determine how closely the simulation approximated the continuum of agents that is usually assumed. For all the simulations in this paper, the number of agents will be fixed at 20. This is large enough to get good “cross fertilization” of ideas and small enough so that the simulations run in a reasonable amount of time. The results of the models are relatively insensitive to the number of agents in the economy. The number of agents in the economy can be thought of as being analogous to the number of significant digits in a calculation- after a certain point, adding extra significant digits doesn’t change the results very much. *The results that will be shown in this paper are not intended to be high precision estimates of policy functions but rather the pattern of how the policy rules evolve over time.*

2.2 A rule for cyst production

The rule for cyst production used in step 2 is a more complicated choice. The usual wisdom is that viral production should be proportional to fitness as measured by the agent’s objective function. While this makes a lot of sense when the fitness has a cardinal interpretation, utility is supposed to be ordinal.³ One policy function producing a larger utility score for an agent than another policy function just means that the result is better, not how much better. It is reasonable that the rule for determining cyst production should be related to the order statistic of the fitness. That means that policy functions that produce a higher utility score should produce more cysts than those that produce a lower utility score simply because it has higher score and not because it is a certain percentage higher.

In these simulations, we will use a rule that increases the number of cysts produced proportionally with the square of the rank. Rank, in this case, is defined in the reverse order of its usual definition, in that the best virus has a high rank, and the worst virus has the rank of one. For the 20 agents in the economy, this particular rule produces a lot of cysts, $\sum_{i=1}^{20} \frac{i^2}{2} = 1,440$.⁴

There are obviously many other choices for this function, and, as in most numerical techniques, there is little basis for one choice over another except that it works well in some contexts. This particular rule was chosen because in experiments that were performed leading up to this demonstration it resulted in reasonably quick evolution in the virus pool without converging too quickly to a homogeneous population.⁵

³This is not strictly true for expected utility models. Expected utility models require that utility is cardinal for intrapersonal comparisons but makes no restrictions on cardinality with respect to interpersonal comparisons.

⁴In this paper the actual rule for the number of cysts that an agent will produce is halved, rounded up for odd numbers.

⁵The limiting factor is not installed computer memory but rather cache size.

2.3 A rule for catching a new virus

Step 3 uses a rule to determine if an agent will catch a new virus from the cyst pool. This is kept as simple as possible and represents a fixed probability of infection for all agents in each period, called the infection rate. This particular parameter, which at first seems innocuous, is actually one of the most important parameters in the simulation. The many effects and interpretations of this particular parameter will be explored in the following sections.

2.4 A rule for melding the new virus with the existing policy function in an agent

Once the agents to be infected are chosen in step 3, the new virus must be combined with the policy function that the agent currently uses to make consumption decisions. This is a combination of the cross-over and mutation operator that is used in standard agent-based evolution. There are many ways to implement cross-over. The simulations in this paper will use simple mean cross-over. In simple mean cross-over, the new policy function parameters, in our case just autonomous consumption, C , and a transformation of the marginal propensity to consume, α , that the agent will use, is just the average of the parameters in the current policy function and the virus that it just caught. Many of the other cross-over operators, like biologically inspired cross-over, which swaps parameters in randomly determined strands, are only becomes useful when there are a large number of parameters. Other types of types of cross-over that involve swapping of parameters, have a nice interpretation only when two policy functions are combined to produce two new policy functions, i.e., sexual reproduction. In the present study two policy functions are combined to produce only one policy function, and so is inappropriate.

Step 3 also includes the mutation operator, which acts on policy rule parameters. The function of this operator is to do the small-scale exploration of the parameter space and allow the slow increases in the fitness of the policy function. Normally this is an extremely important parameter since it prevents genetic drift⁶, and the settling of the policy function on an inefficient local optimum. In the simulations used in this paper, getting the parameters just right is not very important, *those terminal values are not the object of the investigation*. The focus will be on the broad behavior of the evolving system and the path that it takes to its terminal equilibrium. Towards this end, the per parameter mutation rate has been set at a relatively high 0.3. The actual mutation takes the form of adding a Normally distributed random variable with a mean of zero and a standard deviation of 0.1 to the existing parameter.

⁶The tendency for policy functions to clump together away from a local optimum simply because the response surface of the fitness function is relatively flat

2.5 A method for removing dead agents from the economy

Like real viruses, some of the policy functions will cause the death of the agents. Death in this case is defined as running out of capital. When an agent dies, it is immediately removed and a new agent with a new consumption policy function, drawn from the pool of cysts, is created with the same initial capital stock of the agents that were originally created. What is important is that the step of removing dead agents comes after the step where the agents produce cysts that will be added to the cyst pool. This way, if an agent has the “optimum” function of consuming all the capital stock, they can still pass the function on to others.

The very hidden parameter in this model is the capital endowment that is given to each agent. In most dynamics problems with a saddle point characterization, the initial endowment is just represented by a named parameter and then ignored since it does not have an effect on the steady-state of the dynamic system. In this case, the size of the initial endowment is very important. It will determine which of several equilibria the pool will converge to. This topic will be taken up in section 3.

2.6 A rule for the survival of an individual virus in the pool of cysts

The final rule is used in step 8, the shrinking of the cyst pool. Since in each period the agents in the economy will be adding 1,440 viral cysts to the cyst pool, the pool will quickly grow without bound and become unmanageable. To prevent this, each cyst is subject to a fixed probability of being removed from the pool called the decay rate. This means that, while the size of the pool will grow in the early periods, it will eventually top out and remain an approximately fixed quantity. Equilibrium pool size is established when the inflow of cysts, 1,440, is equal to the average number of decaying cysts less the cysts that are removed because they are being used to infect agents. The decay rate insures that there is an equilibrium pool size that is approximately the inflow rate divided by the decay rate.

In section 3 we will actually evolve this particular system and demonstrate the circumstances where the pool converges to policy functions that produce the “optimal” result of consuming all the capital and in which circumstances the agents exhibit forward looking behavior in spite of being explicitly not forward looking.

3 Inducing Forward Looking Behavior

The simulation described above very quickly produces agents that find the “optimal” policy function, consuming all current capital, creating a cyst with this function, and then being replaced by a new individual with a function drawn from the cyst pool. This expected solution always happens when the initial cap-

ital endowment is large, e.g., 40 units. As expected, this evolutionary method is capable of finding the optimal solution to the individuals' consumption problem. This, in itself, is not surprising. What is surprising is that this is not always the case. There are some circumstances where the system will evolve towards a completely different equilibrium, where the agents do not consume all their wealth in the first period.

3.1 Savings without forward looking agents

When the initial capital stock is set to one, the agents start saving, something that is decidedly not optimal from the agent's point of view. Figure 1 below shows the results of a 33 factorial design experiment over the decay rate, infection rate, and depreciation rate.⁷ The simulations were run for 10,000 periods, and the terminal capital for each of the agents was recorded.⁸

Figure 1 shows that when there is a low initial capital stock of one unit, terminal capital stock is nonzero, and is in fact higher than the initial stock, indicating savings behavior. This savings appears to decrease as the infection rate increases. No other solution method would give this result as an optimal solution to the agent's problem since it is not anywhere near the global optimum. The difference between this solution method and the others is the existence of a pool of policy functions. The likely source of this effect is then either the pool itself or the interaction of the pool and the agents. There is an intuitive explanation behind the existence of savings, and the existence of two separate equilibria. The source of impulse to save is the viruses.

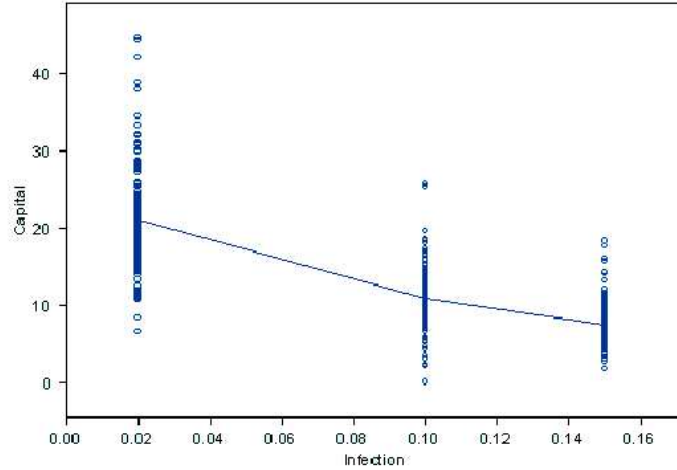
While the agents themselves do not have an interest in future consumption, the long-term cyst production of the virus is very dependent on the average consumption of the agent over time. If the virus directs the agent not to consume everything today, it will produce a smaller number of cysts today, but it can produce more the next period. Eventually, by saving, the average cyst output of the agent will be larger than it would be if the agent consumed the endowment the first period and was replaced by another agent. While it may initially appear that the replacement rule is driving this result, in fact the viral cysts are created and added to the pool before a new virus is drawn for the newly created agent. In this way the analytically optimal rule of consuming everything in the first period can still propagate itself through the population.

The intuition behind the two different possible equilibria is slightly more

⁷A factorial design is an experimental design that matches all level of all factors and is used in situations where there is factor interaction. In this case there was no factor interaction. As a consequence the values of the decay and depreciation rates are unimportant to both the intended analysis and the discussion that follows.

⁸These results were created as part of the testing procedure for the simulation code. This kind of extensive testing is required when working with genetic algorithms since the evolutionary process tends to "cheat", exploiting logical flaws in the simulation design and fitness criteria. A good example of the cheating phenomena can be seen in Graham-Rowe [GR02], where a self-organizing circuit, which was designed to evolve an oscillator, evolved a radio receiver since the fitness criteria was defined as production of an oscillating signal. The circuit used the RF signal from a nearby computer as the basis for its oscillator.

Figure 1: Infection Rate vs. Terminal Capital (Initial Capital = 1)



complicated. A virus in one of the agents has two paths. It can consume the endowment, expel a large number of cysts, and hope that, when the new agent appears, one of their cysts will be chosen to infect the new agent. The other option is to consume only a fraction of the endowment, produce a small number of cysts, and then continue along the same path until they are infected and lose control of the organism, or reach some kind of equilibrium with the capital stock.

The former option is like the person who consumes her entire endowment, feels very happy, and tells society frequently how wonderful it is to save nothing, before keeling over and dying for lack of money. People in the present have a good chance of hearing and accepting this message, as do people in the future, to a lesser extent (copies of the message decay over time). People accepting the message decrease their savings rate, feel happier, and have a good chance of spreading the “Save less” message through their present and future generations. Societal savings approaches zero. The latter option is like the person who consumes enough to feel pretty good, but saves some endowment. Content, he tells society several times (but less often than the enthusiastic spendthrift) about his successful consumption and savings plan. People have only a fair chance of hearing, accepting, and broadcasting the messages he sends out this generation, but he probably survives many more generations to repeat those messages. Over time, the population of converts grows and his savings plan comes to dominate the population.

The best path for an agent is determined by the equilibrium capital stock⁹ for that agent's policy function and the agent's initial endowment. If the equilibrium production for that policy function is less than the initial endowment, the virus that communicates this path will, on average, produce fewer cysts than the virus that communicates consumption of the entire endowment. This occurs because the agent that consumes everything will always be refreshed each period with a new initial endowment, since it "dies". However, if the production at the equilibrium of the capital stock is higher for a virus's policy function than the initial endowment, then saving will produce, on average, a larger number of cysts.

The fitness and survival of the functions that induce savings depends on the trade-offs between the risk of being infected by another virus and the rate that capital increases towards the equilibrium for that function. Slow capital accumulation and its resultant high early consumption keeps a relatively high average cyst output over the short run but increases the probability that the function will be replaced by another before the equilibrium capital stock is reached. Quick accumulation minimizes the short run average cyst production but also minimizes the chance that the function will be replaced before the equilibrium capital stock, and maximum cyst production is reached.

We have established that it is possible to evolve the optimal solution to the posed problem, where the agent consumes the entire endowment every period and we have established that the dynamics of a pool make it possible for other behaviors, that involve saving, to evolve.

The evolutions and gradual improvement can be thought of as learning. True it is not learning in the most efficient, Bayesian, sense, but it demonstrates an improvement in the effectiveness of policy rules over time. Given the interpretation of evolution of policy rules as learning, there is a learning effect on the savings behavior of the individuals, even when the individual explicitly does not care about the future. The dynamics of the learning effects are important to understanding savings behavior.

The next section will expand the exploration of the relationship between forward looking behavior in the agents and the infection rate, which induces forward looking behavior even in agents that should not save. This will provide the basic intuition that will allow a general formulation of discounting in evolving systems.

3.2 Interaction of forward looking agents and viral evolution

In order to explore the interaction of the learning induced forward looking behavior with true forward looking behavior on the part of the individual, it is necessary to alter the objective function of the agents. Only one extra term needs to be added to put the problem from the previous section in the classic

⁹Equilibrium capital stock in this sense is the fixed point in the state transition equation given the consumption rule, i.e., k that satisfies: $k = k^\theta + (1 - \delta)k - C - \alpha k$.

Bellman form. Equation 3 below shows the modified problem with the discounting factor, β .

$$\begin{aligned}
 V_t &= \max_{c_t} \ln(c_t) + \beta V_{t+1} & (3) \\
 s.t. & \\
 k_{t+1} &= k_t^0 + (1 - \delta)k - c_t \\
 k_0 &= K
 \end{aligned}$$

Modifying the simulation is a bit more difficult than modifying the objective function. The difficulty is that the objective function now includes the discounted value of future consumption. The most direct solution is, in each period, use the function that the agent has, run it out several hundred periods and calculate a future consumption series. This series can then be used to calculate the present discounted value of all future consumption and then the appropriate number of viral cysts can be generated.

While this is reasonable, it violates the spirit of the simulations because it puts a lot of computational pressure on the agents. An easier way to put discounting into the model is to directly use the discounting factor as probability of surviving into the next period. This particular interpretation requires modification of steps 2 and 7, the utility and death rules.

Interpreting the discounting factor, β , as the probability of surviving requires that the utility from not surviving is equal to zero and that the utility from surviving is always greater than zero. Viewed in this manner the Bellman equation can be expressed as $V_t = \max_{c_t} \ln(c_t) + \beta V_{t+1} + (1 - \beta)0$.¹⁰ It is clear that the probability of surviving interpretation of the discount parameter is mathematically equivalent to the time preference interpretation.

Discounting can be interpreted axiomatically as a preferring consumption today, or can be a derived property. Viewing discounting as a derived property leads to the interpretation of the discount factor as the probability of surviving into the next period and the restriction that expected utility in the “dead” period is zero. This interpretation can be used in the implementation of the evolutionary method.

Instead of just using the current period consumption for the fitness of the function, the utility from consumption in all periods up to that point is accumulated. There is no need to do any discounting since a modification to step 7, the death step, will effectively create discounting. While it is conceivable that an agent could be alive for the entire 10,000 period simulation it is unlikely since step 7 will also be modified.

¹⁰The probability of death interpretation of the discount rate is only appropriate when the utility function is bounded below at zero. If it were not, there would be circumstances where consumption of some positive quantity would yield a negative utility result and the agent would be better off dead. Recall that in the simulations consumption was modeled as having a lower bound, 0.01, that evaluated to zero utility. The effective utility function is therefore $\ln(c_t) - \ln(0.01)$

In step 7 the “death” of an agent, besides being triggered by a collapse of the capital stock, is also randomly determined. Each agent has only a β probability of surviving into the next period. If they do not survive, the accumulated utility is reset to zero, and they keep their current policy function and capital stock. This particular setup is intended to mimic an infinitely lived agent with discounted utility. The big disadvantage is that discounting tends to be rather sudden: the agent “dies”. This modification allows the system to evolve period-by-period and yields discounted utility results without simulating all future periods for each agent every period. This rule makes the following simulations incomparable to the first simulation. Dead agents are reborn with the same capital stock and policy rule they died with, not a new rule and the initial capital stock.

The discounting simulation was run for 10,000 periods under 81 different scenarios that varied the discount rate and the infection rate. Figure 2 plots the results of that simulation, as level curves of terminal capital stock, given probability of surviving, β , and the infection rate. Initial capital stock was set at one. While the relationship between the terminal capital stock, the infection rate, and the discount rate is not very clear, the plot shows that, generally, increases in the infection rate or decreases in the discount rate decrease the terminal capital stock. The converse can be noted by observing that the largest capital stocks occur when the infection rate is the lowest and the β is the highest (the lower right hand corner).

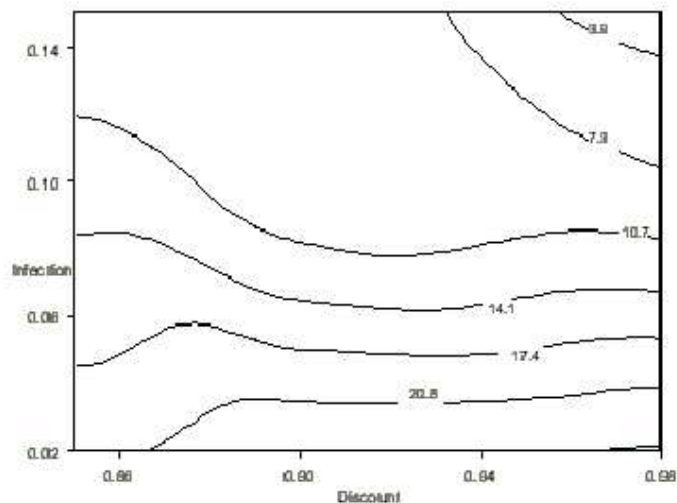
This makes sense because with higher probabilities of survival, there is more success from saving, and because with higher infection rates there is more possibility of learning forward-looking behaviors. What is most interesting about these experimental results is that the effect of the infection rate after 10,000 periods is more pronounced than the effect of the agents’ own probability of surviving. This partially has to do with the slightly different treatment of function replacement and the death of the agent, and the different scales of the two probabilities. It should highlight the fact that learning is important to savings behavior and that the effects of learning can often masquerade as the discount rate.

The intuition for this result is that the virus has two ways of losing the benefits of the accumulated utility and the ability to produce a large number of viral cysts. If the agent dies, the virus loses the benefit of the accumulated utility and the ability to produce more cysts. The virus can also lose the benefit if the agent catches a virus. In that case, the virus is replaced by an inexact copy of itself and again loses the benefit of the accumulated utility since it is no longer able to reproduce through that agent.

The assumption that the agent will not be infected with an identical virus ignores a very important component of the evolutionary system, the cyst pool. Including the pool in the interpretation of the effective discount rate produces many different discounting patterns.

In summary, section 3.1 established that the infection rate is important to determining savings behavior when the agents explicitly did not care about savings. This section expanded that idea and showed that the infection rate

Figure 2: Effect of Probability of Surviving and Infection Rate on Terminal Capital Stock



continues to be important when the agents do care about savings and future consumption. To the extent that infection is thought of as being struck by a new and perhaps better idea, this is a learning process. Most importantly it was demonstrated that the effects of learning could masquerade as a preference for future consumption.

The next section (4) will explain in detail the two major kinds of discounting, hyperbolic and exponential, and how they both can arise in an evolutionary system. This material will be used to explore the theoretical relationship between the agents' probability of death, the infection rate, the probability of self-infection and the "observed" discounting of an agent.

4 Discounting

Discounting is the economists' way of handling future events and making sure that we value future consumption less than present consumption. One of the most common ways of discounting future consumption in macroeconomics is the so-called exponential model, δ^t . This particular model has a very strong practical support since it mimics the effect of compounding interest. It is also the only form of discounting that provides consistent choices in discrete time dynamic programming problems.

The consistency problem is specific to dynamic optimization problems and

the mathematical assumptions that are made in order to make the problems tractable. It is perfectly possible to have an arbitrary discounting function, $\gamma(t)$, in a dynamics problem like the one shown below.

$$\sum_{t=0}^T \gamma(t)u(c_t) \quad (4)$$

With the proper transition equations, provided that the γ function is always positive and finite, a solution can be found at time zero. There are, however, two different kinds of time inconsistency that can arise in dynamic problems, one general and one specific to the Bellman form. The specific problem of consistent discounting arises in dynamic programming problems because of additional assumptions added in Bellman form equations.

Equation 5 below shows a typical kind of dynamic optimization problem, with, c , the control variable, k , the state variable and β the discount of next period's utility. The most interesting thing about this particular form is that it is recursive, i.e., V_t , is defined in terms of V_{t+1} .

$$\begin{aligned} V_t &= \max_{c_t} \ln(c_t) + \beta(t)V_{t+1}(k_{t+1}) & (5) \\ \text{s.t.} & \\ k_{t+1} &= f(k_t) - c_t \end{aligned}$$

This means that it is possible to reinsert the same problem back into the equation, and it should express exactly the same problem.

$$\begin{aligned} V_t &= \max_{c_t} \ln(c_t) + \beta(t) \left[\max_{c_{t+1}} \ln(c_{t+1}) + \beta(t+1)V_{t+2}(k_{t+2}) \right] & (6) \\ \text{s.t.} & \\ k_{t+1} &= f(k_t) - c_t \\ k_{t+2} &= f(k_{t+1}) - c_{t+1} \end{aligned}$$

This is where the “time inconsistency” problem arises. In order for the choices to be time consistent, the agent must make the same decision for k_{t+2} in period t as they would in period $t+1$. A sufficient conditions for this to be true is that the agents face the same problem, and this is only true when $\beta(t) = \beta(t+1)$. In other words, the only way the dynamic problem has a recursive representation is when the discounting is exponential.

The reason that other discounting methods produce time inconsistent choices is that the agent effectively faces a different, recursively defined problem every period, which implies the possibility of different choices.

This particular “time inconsistency” problem arises because of the recursive structure inherent to the Bellman form and is a special case of the general time inconsistency problem, which is that agents change their plans over time even when there has been no change in information.

Hyperbolic discounting is one of the alternative methods that is, as mentioned before, strongly supported by experimental evidence but is “time inconsistent” in the general sense. The discounting interpretation, ignoring the many

figths about the functional form, is that the discounting factor is increasing over time, $\beta(t) < \beta(t + 1)$.

That particular observation becomes more interesting in the “probability of death interpretation”¹¹ of the discounting factor. Exponential discounting means that there is a constant probability of death. Hyperbolic discounting means that there is a decreasing probability of death.

The next section will look at the apparent discounting in evolutionary systems in terms of the discounting functions described above. It will be shown that evolutionary systems have a long-run equilibrium with exponential discounting but pass through an hyperbolic or “time inconsistent” stage on the way to the exponential discounting equilibrium.

5 Discounting Dynamics

Moving back to the problem of viral evolution again, we can rephrase the apparent discounting behavior of the agent-function combination as the probability that the existing policy rule will be able to gather the benefits of the accumulated utility in the next period. This is a compound event that represents the survival of the agent and either no infection by an alternate policy rule or infection by a function that is identical to the one that is currently dictating the agent’s behavior.¹² Equation 7 below expresses the current apparent probability of surviving, $\hat{\beta}(t)$, in terms of the agent’s probability of surviving, β , the probability of infection, d , and the probability that an identical virus will be drawn from the cyst pool, $p(t)$.

$$\hat{\beta}(t) = \beta((1 - d) + dp(t)) \tag{7}$$

It is critical to point out that the apparent probability of surviving is always less than or equal to the agents’ probability of surviving, since the former means appearing to survive unchanged, while the latter means not dying, but possibly having a different policy function. Associated with every apparent probability of surviving is a *tuned policy function* that is, the optimal policy function for the apparent probability of surviving, i.e., one that is tuned to the probability of being replaced by a different policy function. This can be thought of as the analytically derived function that would be found if the actual probability of surviving was equal to the apparent probability of surviving, i.e., the locally optimal policy function. The apparent probability of surviving and the associated tuned policy function will enable us to explore the many different discounting structures that can occur in a particular viral evolutionary system. We will explore this situation with a series of thought experiments that will play out

¹¹This interpretation is strictly only true when the utility from having no consumption in the “dead” state is zero. If the utility functions are logarithmic and the average consumption is less than 1, then it is possible for an agent to be better off dead.

¹²Infection by an identical virus results in no change in the agents state, beyond a possible mutation. All accumulated utility and capital remain intact.

as changing the terms in the apparent probability of surviving function given above.

One of the most obvious features of viral evolution is that if the viral system evolves the analytically optimal policy response, the one you would get with other solution methods, and that policy function fully populates the cyst pool, then the apparent probability of surviving is equal to the agent's probability of surviving and the tuned policy function is equal to the analytically optimal policy function. In this case, it does not matter what the infection rate is since the policy function is always replaced with the same analytically optimal policy response.

$$\hat{\beta}(t) = \beta((1 - d) + d1) = \beta \quad (8)$$

So, if the pool is populated with the analytically optimal policy function, then the apparent discounting of the agents is exponential, $\beta(t) = \beta(t + 1)$. The discount rate and the probability of surviving will be the same in every period, and discounting will be time consistent. Hence, exponential discounting can arise from an evolutionary system.

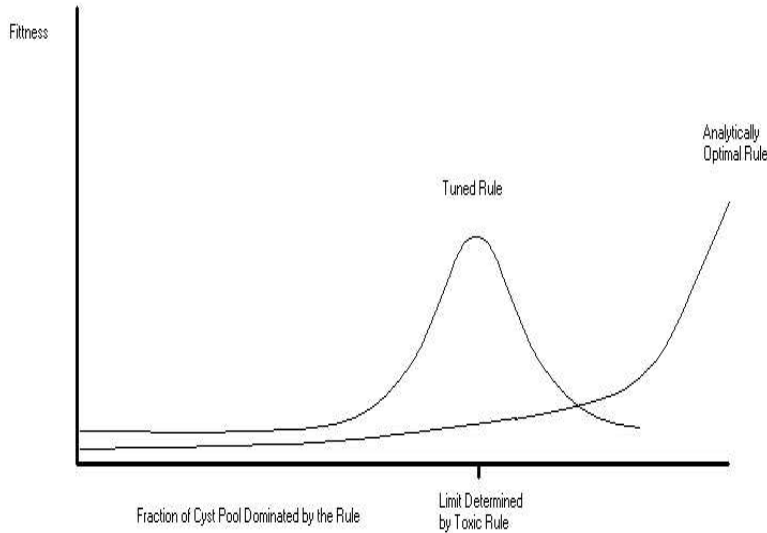
5.1 A Transition Behavior Thought Experiment

The policy function that corresponds to this long-run equilibrium result is unlikely to arise early in the evolution since it will be competing with other policy functions. The analytically optimal policy function is tuned to a probability of surviving of β , but this apparent probability of surviving will not occur until later in the evolutionary process because other policy functions exist in the pool and the probability of being infected by another policy function is positive. Other policy functions that are more closely tuned to the current apparent probability of surviving are likely to dominate and generate more cysts. The following thought experiment should prove this point.

Suppose that the pool is divided into two parts and there is no cross-over or mutation. Infection results in a simple replacement of policy functions. A fraction $p(t)$ of the pool is filled with one of two policy function, and the remaining larger fraction $(1 - p(t))$ is filled with a third, toxic function that causes the death of an individual and no cyst production. The toxic function produces fitness that are so low that any cross-over with a toxic function results in another toxic function.

Now suppose that the fraction of toxic functions in the pool is untouchable, it will always be there and will always be the same fraction. The remainder of the pool will behave as a normal viral cyst pool for the two non-toxic policy functions. Suppose further that we start an economy off with these two nontoxic policy functions, the analytically optimal one, based on the actual probability of surviving, and a tuned policy function, optimal for an apparent probability of surviving. Now assume that the tuned policy function has a higher fitness than the analytically optimal policy function for all fractions of the cyst pool up to $(1 - p)$. The analytically optimal policy function would be more fit if it made up the entire pool, but its fitness falls off as its fraction of the pool falls. It is

Figure 3: Fitness of Two Functions



not the best reaction to the current apparent probability of survival. Figure 3 illustrates this point.

The vertical axis is the fitness measure of the policy function, given the fraction of the pool dominated by the policy function. There are two separate fitness functions, one for the tuned policy function and one for the analytically optimal policy function. When the analytically optimal policy function dominates the pool it has a higher fitness value than the tuned policy function. However, the existence of the fixed fraction of toxic policy functions prevents the analytically optimal policy function from ever controlling a fraction of the pool that would allow it to be more fit than the tuned policy function. As a consequence, no matter what fraction of the pool the analytically optimal policy function dominates, the tuned policy function will always be more fit and will always command a higher fraction of the next period's cyst pool.

This means that the tuned policy function will be contributed to the pool more often than the optimal policy rule. As the tuned policy function increases its share of the pool it will become more and more fit since as its share of the pool increases the apparent probability of surviving approaches its tuned value. Eventually, when the tuned policy function fills the fraction of the pool not filled by the toxic policy function, it will be the best policy function given the composition of the pool. The analytically optimal policy function will not be able to compete.

The most interesting property of viral evolution is that hyperbolic discounting, i.e., declining probability of death discounting, arises naturally from the evolutionary processes. Suppose this time that we have a population of toxic policy functions in the viral pool, but no fixed proportion of toxic rules. Suppose further that we have one agent that actually has a very good, but not the analytically optimal, policy function while the others have the toxic policy function, die, and are replaced by draws from the viral pool. Initially, every infection will be guaranteed to yield the toxic policy function, so there is a very high apparent probability of losing control and a very low apparent probability of surviving. Over time, the good policy function will shed more cysts into the cyst pool. This means that, over time, the probability of the agent with the good policy function being infected by an identical policy function starts to increase, so the probability of losing control to a toxic policy function decreases over time. Because the pool is changing, the tuned policy function, given the current state of the cyst pool, will change over time.

The tuned policy function will tend to dominate the pool until good policy functions push out more of the toxic policy functions, increasing the fraction of the pool devoted to good policy functions. This will in turn change the tuned policy function, reflecting the lower probability of being infected by a toxic function, and the process will continue. Mutation will eventually cause an instance of the correct tuned rule to arise and propagate, since it is the most fit function for the cyst pool composition. Slowly, the fraction of the pool that contains toxic policy functions will shrink and the apparent probability of death will decrease (hyperbolic discounting along the path), until the probability of surviving converges to the agents' actual discount rate (exponential discounting at equilibrium.). Evolutionary pressure forces the probability of surviving to increase over time.

So an evolutionary system like that described can support both exponential discounting that is often observed in capital markets, and hyperbolic discounting that is observed in clinical experiments. Furthermore, hyperbolic discounting arises during the transition period when the economy and policy functions are moving towards a new capital equilibrium and policy function equilibrium where discounting is exponential.

5.2 Transition Behavior Demonstration

These thought experiments can be demonstrated by simulation using the model used to explore the interaction of viral evolution and forward looking agents. The discount rate cannot be observed directly but it can be inferred from the behavior. The key is to assume that the agent's reactions are optimal for some discount rate and then find the discount rate that is consistent with the choice. This discount rate, which I will call the *implied discount rate*, can be calculated using the intertemporal, first-order condition.

Recall that forward-looking agents faced the following problem:

$$V_t = \max_{c_t} \ln(c_t) + \beta V_{t+1} \quad (9)$$

s.t.

$$k_{t+1} = k_t^\theta + (1 - \delta)k - c_t$$

$$k_0 = K$$

The first-order intertemporal condition for this problem is:

$$\frac{1}{c_t} = \beta \left[\frac{\theta k_t^{\theta-1} + (1 - \delta)}{c_{t+1}} \right] \quad (10)$$

Which can be manipulated to give the implied discount rate, $\hat{\beta}$.

$$\frac{c_{t+1}}{c_t(\theta k_t^{\theta-1} + (1 - \delta))} = \hat{\beta} \quad (11)$$

For purposes of simulation, the implied discount rate at time t is time shifted so only contemporaneous and lagged values of consumption and the capital stock are required.

Twenty thousand period simulations were conducted for a variety of factor levels on each of the four main simulation variables: depreciation, viral decay rate, discount rate and the infection rate. The factor levels are shown in table 1 below. In all, 1,440 simulations were made- eight replicates of a full factorial design.

If the thought experiment above is an accurate description then we would expect an initial stage of experimentation in each of the simulations, where a fairly good rule tends to dominate the viral pool and then a slow increase in the implied discount rate. This early chaotic was completed in less then 200 periods for almost all the simulations. What remains is a slowly changing series of per-period average implied discount rates. The average change over time can be shown with a simple regression on time, t .

$$\hat{\beta}_t = \alpha + \lambda t + \epsilon \quad (12)$$

To construct Table 1 below, the first 200 observations were trimmed from each of the 1,440 series of average implied discount rates. The remaining 19,800 observations were regressed on a time trend and the slope parameters collected. The first row of the table shows the mean of all the slope parameters and the confidence bound on that mean. The rows that follow show the average of the slope parameter for the simulations that had zero depreciation, 0.10 depreciation, and so on. In all cases the mean of the time trend is positive, which is consistent with the thought experiment that the probability of survival should increase. Successful rules will slowly adapt to their own dominance of the viral pool increasing the apparent probability of survival over time. This means that discounting follows a hyperbolic pattern of declining probability of death in these simulations. It would be a bit much to claim that learning causes hyperbolic discounting, but it would be reasonable to suppose that learning is tightly bound up with the phenomena.

Table 1: Rate of Change in Implied Discount Rate (1/1,000,000)

Factor	Factor Level	Mean	Confidence Bounds	
			2.5%	97.5%
All		2.70	2.13	3.28
Depreciation	0.00	2.09	1.45	2.73
	0.10	3.33	2.03	4.62
	0.15	2.70	1.74	3.65
Decay Rate	0.10	2.36	1.18	3.54
	0.20	3.22	2.44	4.00
	0.30	2.53	1.53	3.53
Discount Rate	0.00	3.52	1.79	5.25
	0.50	2.99	1.73	4.24
	0.75	2.38	1.34	3.42
	0.90	2.14	1.08	3.20
	0.95	2.49	1.22	3.77
Infection Rate	0.10	2.34	2.08	2.60
	0.15	1.91	1.16	2.67
	0.20	2.57	1.40	3.73
	0.25	3.99	2.17	5.28

6 Summary and Conjectures

This paper has demonstrated a way of using a viral evolutionary system in a macroeconomic framework. The paper has also shown that, while economists can solve many problems with viral evolutionary methods, the pool provides an extra dimension where interesting dynamic behavior can develop. In particular we showed that, even with agents that are explicitly not forward looking, a viral evolutionary system could produce behavior that shows forward-looking interests.

This particular effect can be combined with forward-looking agents to produce hyperbolic discounting that is often assumed in economics rather than derived from some other more elementary process.

There are three potential empirical tests of this model. First, it may be that the empirical regularity of exponential discounting breaks down when there is significant qualitative uncertainty about the economy. In this is the case, financial markets in long-run equilibrium operate with an apparent discount rate equal to the actual discount rate. When the deep parameters of the market change, i.e. there is qualitative uncertainty, the markets must go through a learning process where the discount rate is much higher. If this is the case, then an empirical study of periods with qualitative changes in the economy, e.g., transition from wartime to peace time economies and other rapid structural changes, will cause a change in the risk free rate, either upward or downward

depending on the change, which would then decrease as the learning process proceeded. So if the risk free rate exhibited downward bias, instead of mean reversion this would be empirical evidence of a learning period that is structured like a viral evolutionary system or some other observationally equivalent learning model.

The second potential application has to do with the function of capital markets above and beyond their ability to decrease transactions costs. If we do not observe the learning effects as described in the first hypothesis then it may be that financial markets allow for the development of policy functions that are not so closely tied to the current state of the economy. Put in econometric terms, policy functions are dependent on the “deep” parameters in an economy, e.g., the parameterization of technical growth rather than the actual revelations of technical growth. Unfortunately, a method of testing this hypothesis escapes the author since it may not be falsifiable.

Finally, a more direct test can be conducted under experimental conditions in a lab setting. By observing the changes in implied discounting when agents interact in a market and when they do not may provide compelling evidence of additional market functionality.

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