

Modeling Awareness in a Bacterial Colony

Vladimir Barash
Willard Miranker

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Vladimir Barash, Willard Miranker

Yale University Department of Computer Science

Abstract:

An investigation into the presence of collective awareness in a colony of bacteria foraging in search of nutrient is performed. A model of a bacterial colony is employed, founded upon a qualitative dualist concept of distributed consciousness and a quantitative notion of so-called awareness tokens. Motivated by the Darwinian principle of survival of the fittest, an advanced version of the model incorporates notions of a virtual fitness and a virtual genetic memory. Both models are tested to determine whether the colony has a collective awareness of the location of a nutrient supply. The results indicate that such awareness is present in some cases, and, for the advanced version of the model, it exists in an unstable equilibrium with the colony-wide average of a virtual genetic memory.

1. Introduction

We investigate the presence of collective awareness in a colony of bacteria navigating a space in search of nutrient. The presence of collective awareness at the bacterial colony level has been long speculated: since the 1960's, biologists have known of two species, *V. fischeri* and *V. harveyi*, which seem to exhibit awareness of the presence of conspecifics by emitting light (see Bassler [1] for an overview of the properties of *V. fischeri* and *V. harveyi*). At the same time, the computer science community has analyzed collective awareness in networks of robots (Kornienko et al. [6]) and of abstract agents whose behavior is inspired by reaction-diffusion models (Sato et al. [10]). We integrate these two research trends by devising a model of collective awareness based on both computer scientific and biological methods. Simulations of the model show that collective awareness in a bacterial colony is linked to the reliance of its component organisms on a virtual genetic memory that we shall introduce.

2. Collective Awareness: Background

The notion of collective awareness is strongly related to the dualist theory of mental processes, to the notion of distributed consciousness, and to the Darwinian principle of survival of the fittest. Dualism (Descartes [4], Miranker [8]) provides the philosophical background for collective awareness, while distributed consciousness (Block [3]) suggests possible media where dualist processes may manifest themselves. Survival of the fittest provides a bridge between these philosophical notions and specific population biology mechanisms that may give rise to collective awareness.

2.1 Dualism

In his *Meditations on First Philosophy*, Descartes posited that his mind, an immaterial "thinking thing," (*res cogitans*) existed separately from and interacted with his extended body, a material and non-thinking thing (*res extensa*). Philosophers who agree with Descartes have been occupied with the problem of interactionism – the method whereby an immaterial mind interacts with a material body. The immaterial mind manifests *processes* and *constructs* like awareness and qualia – the subjective aspects of

mental events, e.g., “what a burnt finger feels like” (Nagel [9]). The material body is a collection of physical *tokens*¹ of these constructs and processes. The interactionalism problem, therefore, consists of finding correspondences between mental constructs or processes and physical tokens. To do this, we shall follow the methodology used by Miranker, moreover following the conventions in [8] and refer to such a correspondence as a *mirroring*. Philosophers have attempted to solve this problem by many types of arguments, such as common sense, logical proofs, empirical verification... Common-sense arguments have so far failed to convince either the philosophical or the scientific community, and no one has yet come up with an incontrovertible logical solution to interactionism. Empirical verification has been similarly unsuccessful, but remains one of the best candidates for penetrating and eventually solving the problem due to the recent emergence of empirically based models of mental processes (Kornienko [6]).

2.2 Distributed Consciousness

Even though the problem of interactionism currently has no convincing solution, its very statement suggests something about the nature of processes and their tokens. This problem applies to the interaction between mental and physical phenomena, independently of the forms they take. For instance, if we one day found an intelligence in the Earth's atmosphere, whose thoughts and feelings corresponded exactly to weather patterns, the interactionism problem would be solved – for the relevant mental-physical pair. More generally, interactionism has alerted philosophers and computer scientists to the possible existence of conscious entities whose “minds” and “brains” are very different from human counterparts (Block [3]). The most promising candidates for such entities are digital (e.g. the Internet) and biological networks (e.g. bacterial colonies), wherein consciousness is distributed among a large number of nodes (agents).

Digital networks, from wiring on a chip to pages on the World Wide Web, are composed of connected nodes. The connectedness of these networks allows information –

¹A token of a property or process is a quantitative measure of that property or process. For example, using our terminology, we would say that the pressure p is a token of the specific force, the pressure being a measurable quantity, while the quality of specific force is a dualist construct of Newtonian mechanics

whether in the form of electrical impulses or of user feedback – to flow through them; the more complex the structure of the network, the more complex the flow of information can be. It is possible that in especially complex networks, the nodes might act to support a distributed consciousness, and the measurable information flow between them might serve as a token of the network's mental processes. Biological networks, on the other hand, are generally composed of interacting agents, each of which behaves in a certain deterministic way. The process of interaction continuously creates, alters and destroys short-term ties between these agents. From the dualist perspective, it is possible to conceive of a biological network's consciousness as being distributed among its composite agents, and of the totality of interactions between these agents as the counterpart to the network's awareness. This modeling of biological networks as conscious entities finds support in the recently discovered phenomenon of *quorum sensing* in colonies of certain bacteria species (Bassler [1]).

2.3 Survival of the Fittest

Dualism and distributed consciousness tie into biology, in particular, via the Darwinian principle of survival of the fittest. In populations where virtual genetics plays an important part (such as the virtual bacterial colony memory described in Section 5), this principle is invoked by appending a scalar “fitness” property to each individual in the population, and by establishing a dynamic whereby fitter individuals are more likely to survive over time than less fit ones. If this dynamic is linked to some other property P of the individual (for instance, a measure of distributed consciousness), then, as the simulation proceeds, the fraction of the population that exhibits P will be comprised of mostly fit individuals. Since the fitter will be more likely to survive, their survival acts as a reinforcement mechanism of property P for the population as a whole, and this makes fitness a likely candidate for expressing collective awareness in a biological context.

3. Awareness in Bacteria: Quorum Sensing

Current research into *Vibrio fischeri* and *V. harveyi* shows that these species constantly release autoinducer chemicals, which signal their presence to other bacteria of

the same species. Normally, the bacteria population is diffused in a medium (for instance, sea water), and the autoinducer concentration remains below a critical value. However, if as a group, the bacteria are ever close enough to each other so that the autoinducer concentration exceeds critical, their behavior changes dramatically – *Vibrio fischeri*, for instance, begin to emit chemicals that produce light. This form of communication via chemical exchange, called quorum sensing, allows bacteria to become aware of each other's presence and to use this awareness (mirrored by the luminescence) to increase their chances of survival and reproduction.

Quorum sensing is a very diverse phenomenon. In a 2002 paper, Miller et al. [7] show that quorum sensing regulates virulence factors (toxins, etc.) in *v. cholerae*, the bacterium that causes cholera in humans. The bacteria do not attack the human host until present in sufficiently large numbers; as a result, the attack has a higher chance of penetrating through the immune system's defenses – whereas a premature release of virulence factors would tip off the immune system and foil the bacteria's chances of a successful invasion. In a different vein, Taga and Bassler [11] show that some of the autoinducers responsible for quorum sensing may not be species-specific, allowing the bacteria that use these autoinducers to become aware of different kinds of organisms (i.e. aware of exogenous information). The diversity of quorum sensing strongly suggests this phenomenon as the starting point for a generic model of awareness in bacterial colonies.

4. The Base Model

The base model characterizes a simplified version of quorum sensing. Bacteria in the model do not produce autoinducer chemicals; instead, they are exposed to a nutrient bath, with different concentrations of nutrient at different points in the latter. Each bacterium is able to observe a small portion of the bath, and is predisposed to move to areas of higher nutrient concentration. At the end of every displacement, a bacterium tries to consume nutrient at its present location. If it succeeds, it reproduces by fission; if it fails, it dies. The base model, then, is set up so that bacteria that happen to move towards high-concentrated areas and stay there are the fit bacteria, those that are most likely to leave offspring. Consequently, in the course of simulations of the base model, the bulk of

the bacterial population should shift to nutrient-richer areas of the bath, and this bulk shifting represents a quantitative mirroring of a colonial awareness quality.

4.1 Bacterial Colonies and Nutrient Bath

The base version of the model of collective awareness in bacterial populations (as presented in [8]) consists of a large population of bacteria embedded in a nutrient bath over a certain time period (measured in unit time steps). Each bacterium is idealized as a rod-like object of length d . Its two ends are denoted r and l , respectively. There are no restrictions on the number of bacteria that can occupy the same location² in the nutrient bath at the same time. The bacterial population at x at time t is denoted by $p(x,t)$. The nutrient concentration at x in the bath at time t is denoted by $n(x,t)$. The bath also receives an exogenous influx of nutrient denoted by $n_e(x,t)$.

Each bacterium in the model has a simple life cycle, which consists of three phases: motion, feeding, and death or reproduction (the last depending on the outcome of the two previous phases).

4.2 Bacterial Motion

A single bacterium will change its location as it myopically seeks to improve its nutrient supply. First, the bacterium measures $n(x_r, t)$ and $n(x_l, t)$ and computes the difference and mean of nutrient concentrations at its ends:

$$(4.1) \quad \Delta n(t) = n(x_r, t) - n(x_l, t)$$

$$(4.2) \quad \bar{n}(t) = \frac{n(x_r, t) + n(x_l, t)}{2}$$

Next, the bacterium moves along its length in the direction of the higher nutrient concentration, with its rate of movement proportional to $\Delta n(t)$:

$$(4.3) \quad y(t+1) = x(t) + \alpha z \Delta n(t)$$

where α is a scaling constant, and z is the unit vector:

²For the purposes of the model, a bacterium is taken to be located at its center.

$$(4.4) \quad z = \text{sgn}(\Delta n(t)) \frac{x_r - x_l}{d}$$

After this rectilinear movement, the bacterium makes a random rotation about its center.

Let R be the rotation matrix:

$$(4.5) \quad \begin{bmatrix} \cos 2\pi\theta & -\sin 2\pi\theta \\ \sin 2\pi\theta & \cos 2\pi\theta \end{bmatrix}$$

where θ is chosen at random from the unit interval. Then the final position of a bacterium at the end of its movement phase is given by:

$$(4.6) \quad x(t+1) = R(y(t+1) - \bar{y}(t+1))$$

where $\bar{y}(t+1) = \frac{1}{2}(y_r(t+1) + y_l(t+1))$, and $y_r(t+1), y_l(t+1)$ are determined for the two ends of the bacterium as specified in (4.3).

4.3 Bacterial Feeding

After a bacterium ends its movement phase, it attempts to feed on the nutrient supply at $x(t+1)$. Let β be the amount of nutrient consumed by a bacterium in unit time. Then if:

$$(4.7) \quad n(x, t+1) \geq \beta p(x, t+1)$$

a bacterium at $x(t+1)$ survives, consumes β nutrient, and reproduces in the next phase. Otherwise, a maximally sized set s of bacteria is chosen from $p(x, t+1)$ so:

$$(4.8) \quad n(x, t+1) \geq \beta |s|$$

Here $|s|$ represents the number of bacteria in s . The bacteria chosen survive; the remaining do not consume any nutrient and die in the next phase.

4.4. Bacterial Reproduction and Death

Bacteria that survive reproduce by binary fission. The two daughter bacteria instantly appear in the same location and with the same orientation as their mother. A bacterium that dies is removed from the bath.

5. Advanced Model – Bacterial Genetics

As the basic model represents myopic behavior, it is hard to make or even intuit the argument that the bacterial colony in this model is truly able to “sense” the bath-wide concentration patterns of nutrient. The bacteria that survive the longest in t -time simulations of the base model may have simply gone through a lucky sequence of moves that led them from one nutrient-rich area to another. Accordingly, the final version of the bacterial model includes the virtual notions of *fitness* and *genetic memory*³. Fitness increases the chances of a bacterium's survival at time $t+1$, even in conditions of nutrient shortage, provided the bacterium is at a more nutrient-rich location at $t+1$ than it was at t . Genetic memory modifies a bacterium's rate of movement based on how successful its myopic strategy has been in previous time units, as specified in (5.3) and (5.4).

5.1 Fitness and Modified Bacterial Survival

We now introduce a fitness measure $a(t)$ of a bacterium, calculated just before the feeding phase at time t :

$$(5.1) \quad a(t) = \text{sgn}\left(\frac{\bar{n}(t)}{\bar{n}(t-1)} - 1\right), \quad \text{sgn}(0) = 1$$

A bacterium is said to be fit/not-fit at time $t > 0$ if $a(t) = 1/-1$. All bacteria are considered not fit at the beginning of the model simulation, so $a(0) = -1$.

If equation (4.7) does not hold at $x(t+1)$, the model generates the set s choosing, at first, only among the fit bacteria in $p(x, t+1)$. Then, if the model has already included all the fit bacteria in $p(x, t+1)$ in s , and can further add some number q of non-fit bacteria without violating (4.8), such bacteria are selected at random from the non-fit part of $p(x, t+1)$.

5.2 Genetic Memory

If a bacterium reproduces, it passes on information about its (and its predecessors') fitness values to its two daughters. In the reproductive phase of the

³For convenience, we shall hereafter drop the term virtual

advanced model, these two daughters are given random fitness values $a_{daughter1}, a_{daughter2}$ with the restriction that:

$$(5.2) \quad a_{daughter1} \vee a_{daughter2} = a_{mother}^4$$

Furthermore, each bacterium is assigned a fitness memory value g , initially equal to 0. As the bacterium reproduces, its daughters' g values are updated according to:

$$(5.3) \quad g_{daughter} = g_{mother} + a_{daughter} + \nu \chi$$

where ν is a scaling factor and χ is normally distributed with a 0.5 mean. Finally, the equation of motion (4.3) is modified to read:

$$(5.4) \quad y(t+1) = x(t) + \alpha g z \Delta n(t)$$

The inclusion of genetic memory implies that, as the simulation proceeds in t -time, the movement rates of offspring bacteria will be modified by the successes and failures of its predecessors. The factor ν in (5.3) turns out to be particularly critical for this aspect of the model. On the physical level, this factor represents a bacterium's "trust" in its genetic memory, and it may take on negative values, resulting in bacteria that "mistrust" their genetic memories so much that they actively try to limit their effect on movement. Note that ν is a constant set for the bacterial colony as a whole. The term $\nu \chi$ in (5.3) might also represent a possible mutation effect.

5.3 Awareness in the Advanced Model: a Mirroring Hypothesis

Miranker [8] adopts the interactionist framework to the advanced model of the bacterial colony. A bacterium's a value is termed a token of its awareness; this token corresponds to \hat{a} , the awareness construct, which takes the value "aware" if the token $a = 1$, and "not aware" otherwise. Both the token and the construct have counterparts at the colonial level:

$$(5.5) \quad A(x, t) = \sum_{bacteria\ i\ at\ x} a_i(t)$$

is called the "awareness density token" of the colony, whereas the union of the awareness

⁴Here, \vee is the logical or symbol. (5.2) prevents combinations such as an unfit mother having two fit daughters, and vice versa, as such combinations are genetically unfeasible.

constructs of all the bacteria at x , namely $\hat{A}(x, t)$, is called the “awareness density construct” of the colony.

The set-up of the model suggests that the bacterial population p will mirror the supply of nutrient, so that as the simulation progresses, we will have mirroring effect 1:

$$(5.6) \quad p(x, t) \propto n(x, t)$$

Given that $\hat{A}(x, t)$ is a construct that represents the colony-wide awareness of the nutrient supply, Miranker suggests that $p(x, t)$ and $\hat{A}(x, t)$ are dual, the former being an externally observable quantity, the latter being an externally unobservable quality. With this convention, one can call $p(x, t)$ and $\hat{A}(x, t)$ the sensors/sensations of nutrient, parallel to say olfaction/aroma in human perception.

Miranker also suggests that $A(x, t)$ in the base model mirrors the exogenous supply of nutrient, so that $A(x, t)$ falls to 0 very quickly if said supply is completely cut off. This is mirroring effect 2:

$$(5.7) \quad A(x, t) \propto n(x, t)$$

Mirroring effect 2 is an example of the principle of survival of the fittest acting as a reinforcement mechanism upon the colony, as described in Section 2.3. The fitness property of a bacterium, which often determines its likelihood to survive (as specified in Section 5.1), is linked to its movement towards nutrient-richer areas in the bath. Consequently, in the course of simulations of the advanced model, fit bacteria are much more likely to be in nutrient-rich areas than in nutrient-poor areas (mirroring effect 2).

6. Testing the Advanced Model

We take the nutrient bath to be a rectangular region pixelated into sub-rectangles called cells. The advanced bacterial colony model proposes two mirroring effects described in Section 5.3. In order to more clearly test for the presence of these effects, the notion of a “localized-fill” nutrient bath is introduced, as follows. The “localized-fill” nutrient bath specifies a subset c of cells, such that every cell in c continuously receives a much greater exogenous supply of nutrient than every cell not in c (as specified in Section 6.2). As a result, the average nutrient concentration is much greater in c than

outside c . In the course of simulations of the advanced model, the fraction of the bacterial population located at cells in c is an indicator of the strength of mirroring effect 1; the fraction of the fit bacterial population located at cells in c , an indicator of the strength of mirroring effect 2. These two indicators are determined as follows:

$$(6.1) \quad m_1 = \frac{\sum_{cells \in c} p(cell)}{\sum_{cells \in bath} p(cell)}$$

$$(6.2) \quad m_2 = \frac{fitin}{fitout},$$

where

$$(6.3) \quad fitin = \frac{\sum_{cells \in c} |bacteria\ i\ at\ cell,\ a(i) > -1|}{\sum_{cells \in c} p(cell)}$$

$$(6.4) \quad fitout = \frac{\sum_{cells \notin c} |bacteria\ i\ at\ cell,\ a(i) > -1|}{\sum_{cells \notin c} p(cell)}$$

In (6.3) and (6.4), the absolute value signs denote the number of elements in the collection specified within those signs. m_1 grades for the mirroring of the exogenous nutrient supply by the colony population (5.6). m_2 grades for the mirroring of the exogenous nutrient supply by the colonial fitness (5.7), by determining how many fit bacteria are located at cells in c compared to cells not in c .

6.1 General Specifications

At the beginning of every simulation, a nutrient bath consisting of mxn cells is created and populated randomly with p bacteria, with the restriction $p \ll mxn$. The initial distribution of nutrient in the bath is random. After all the bacteria are created, each undergoes one move phase: this constitutes the initialization of the simulation. Afterwards, each bacterium undergoes the feed, reproduce/die, and move phases, in that

order; the full cycle of phases constitutes one time unit⁵. The simulation is run for a predetermined number T of time units, after which it is stopped and the measures m_1 and m_2 are determined.

6.2 Further Modifications

In addition to the general conditions described in 6.1, the following modifications are performed prior to the beginning of a simulation: first, bacterial reproduction is set to either on or off. The default state is on; if bacterial reproduction is turned off, the reproductive phase produces one daughter bacterium, identical to its mother in all respects except for g . The daughter's g value is specified as follows (compare (5.3)):

$$(6.1) \quad g_{daughter} = g_{mother} + a_{mother} + v\chi$$

Second, the localized-fill pattern of nutrient influx is specified, as follows:

$$(6.2) \quad \begin{aligned} n_e(x, t) &= k\beta, \quad \text{if } x \in c, \quad \forall t \\ n_e(x, t) &= \beta, \quad \text{otherwise, } \forall t \end{aligned}$$

$$(6.3) \quad x \in c, \quad \text{iff } x = (a, b) \text{ such that } \{m/4 < b < 3m/4 \wedge n/4 < a < 3n/4\}, \quad \forall t$$

Where $k > 1.0$ is a scaling factor. The fill dynamics in (6.2 - 6.3) define c (see first paragraph of Section 6) as a rectangle of cells each more than $\sqrt{m+n}/2$ away from the bath edge. Finally, the values of $\alpha \in [0.1, 1.0]$ (see (4.3)) and $v \in [-3.0, 3.0]$ (see (5.3)) are set at the beginning of each simulation.

To account for statistical variance, ten simulations of every identical variant are performed, and means and standard deviations calculated.

6.3 Results: No Reproduction

The results for m_2 values with no reproduction are limited, as the model behaved erratically (producing populations greater than the initial, which is impossible) for those particular runs. No erratic behavior was noted in the course of any other runs.

⁵Immediately after the reproductive or death phases, each cell in the bath receives a specified amount of nutrient influx, specified by $n_e(x, t)$.

Figures 6.1 and 6.2 show the results of testing for mirroring of the exogenous nutrient supply by the bacterial population:

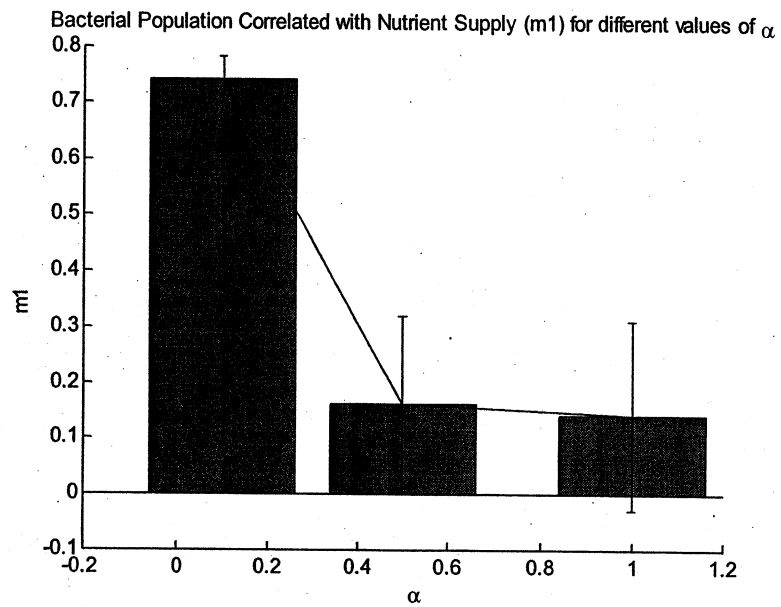


Figure 6.1

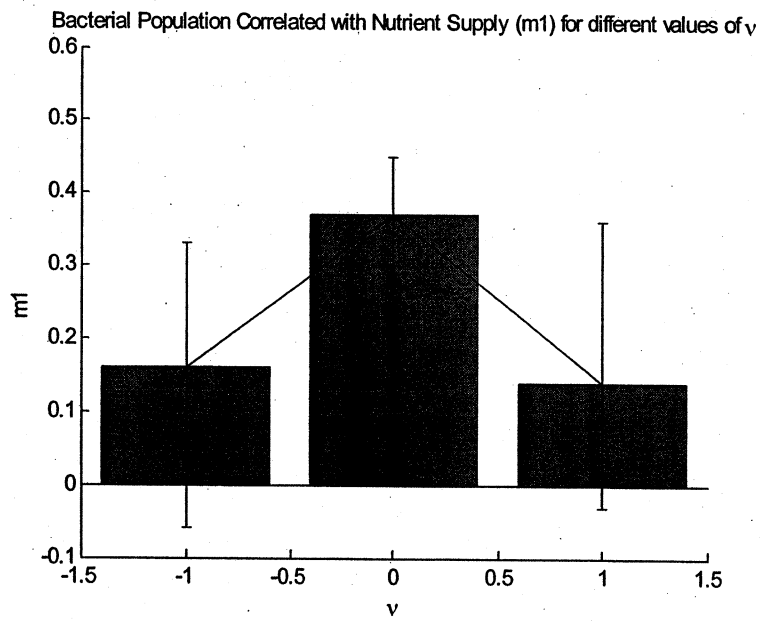


Figure 6.2

Figure 6.3 shows the results of testing for mirroring of the exogenous nutrient supply by the colonial fitness:

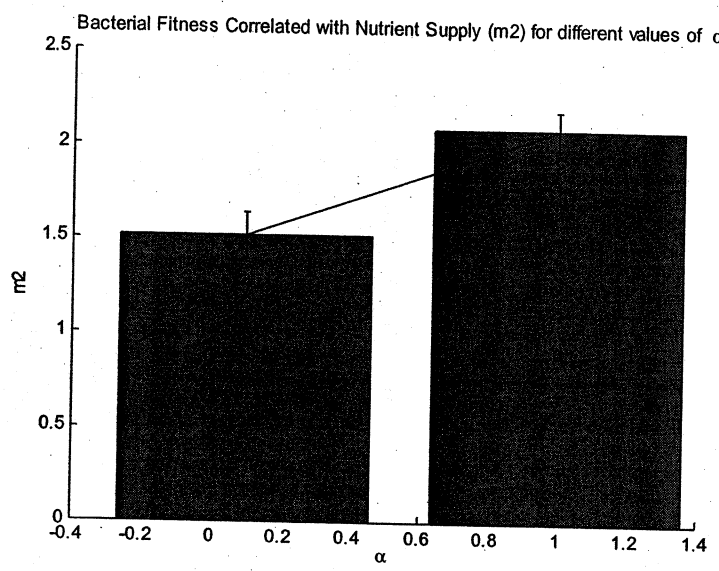


Figure 6.3

6.4 Results: Reproduction

Figures 6.4 and 6.5 show the results of testing for mirroring of the exogenous nutrient supply by the bacterial population:

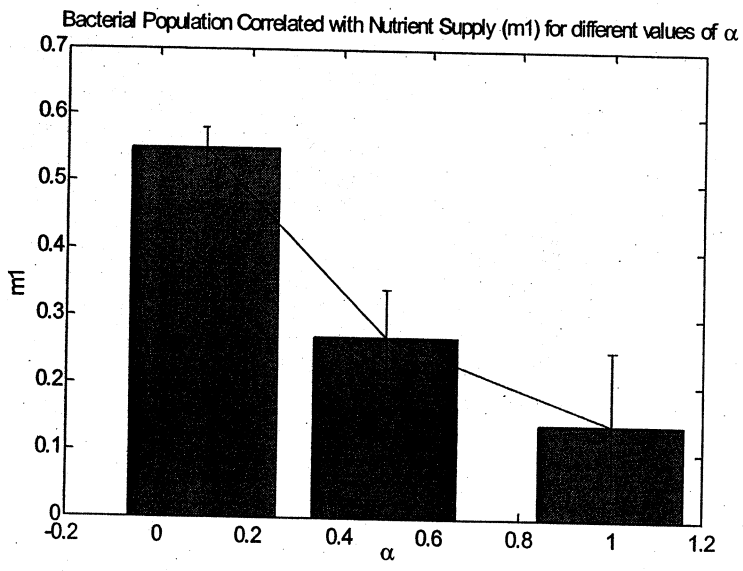


Figure 6.4

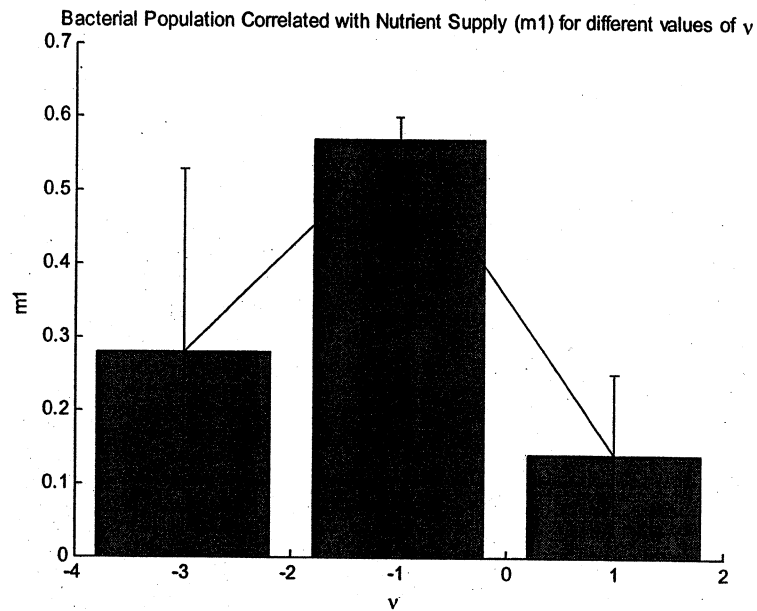


Figure 6.5

Figures 6.6 and 6.7 show the results of testing for mirroring effects of the exogenous nutrient supply by the colonial fitness:

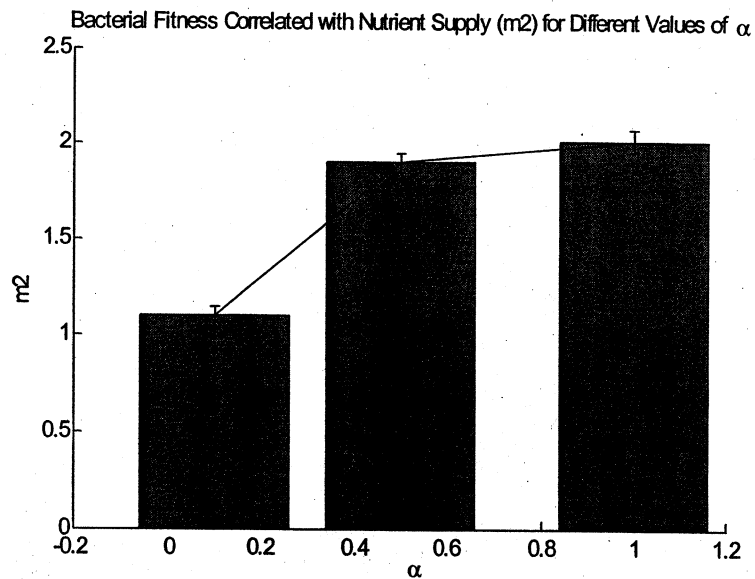


Figure 6.6

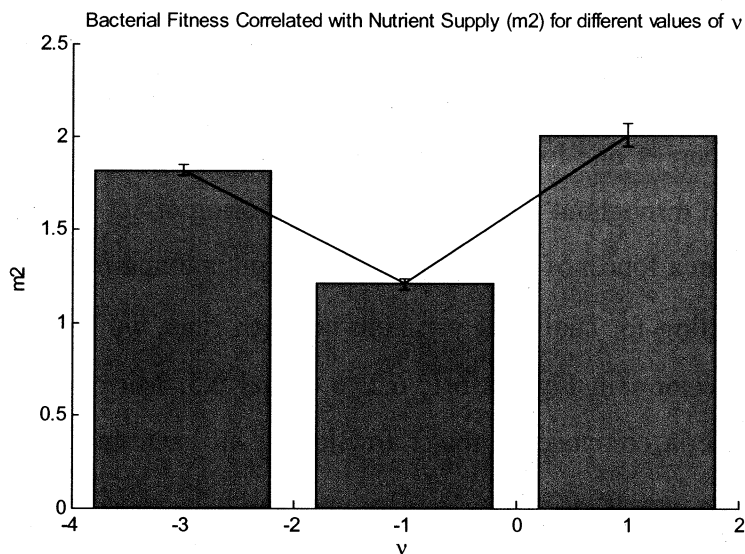


Figure 6.7

Finally, shown in Figure 6.8, are three snapshots taken of the colony at the end of three different simulations, which are particularly relevant for a subsequent discussion of the role of v in the colony's sensitivity to nutrient. The three simulations are performed on an $m \times n = 30 \times 30$ nutrient bath, with $p=100$ bacteria. Reproduction is on and $\alpha = 1$ in all three cases. White, pink and red squares represent concentrations of nutrient, in ascending order. Green squares represent the centers of living bacteria:

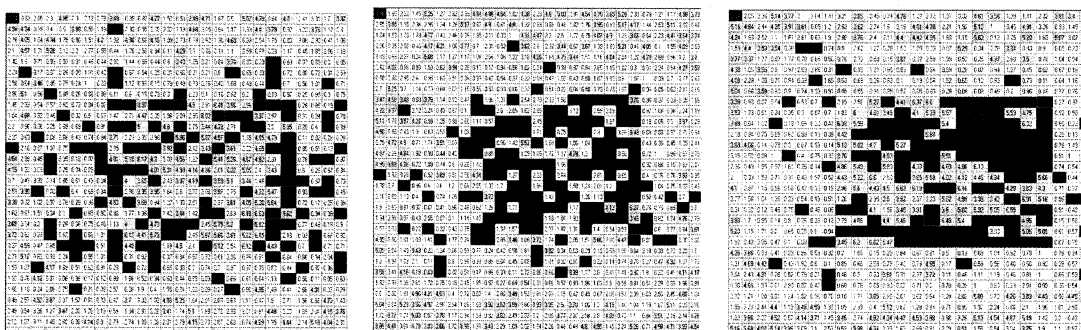


Figure 6.8

Bacterial Sensitivity to Nutrient Location for Different Values of v

From left to right, the snapshots in Figure 6.8 correspond to the values $v=1.0$, -1.0 , 0.0 , respectively (see (5.3)).

7. Interpretation

First and foremost, Figures 6.1-6.7 indicate a statistically significant occurrence of mirroring effects 1 and 2, described in Section 5.3. Since the nutrient-rich area c (see Sections 6, 6.2) occupies one-fourth of the nutrient bath, and the bacteria are initially randomly distributed throughout the nutrient bath, mirroring effect 1 can be said to have occurred if more than a fourth of the bacterial population is found in c (in other words, if $m_1 > .25$). Accounting for statistical variation between runs, we have $m_1 > .25$ for at least one value of α or ν in Figures 6.1, 6.2, 6.4, and 6.5. Mirroring effect 2 can be observed if fit bacteria compose a greater fraction of all bacteria found in c than not found in c (in other words, if $m_2 > 1$). This inequality is valid for all values of all constants in Figures 6.3, 6.6, and 6.7.

Further, it is important to note that a separate series of simulations, not shown here, confirmed that, as nutrient influx was entirely removed, the bacterium population rapidly (within 15 time units in most runs) declined to 0.

Figures 6.1 and 6.4 show a certain amount of correlation between α and m_1 ($\rho = -0.85$ for Figure 6.1, $\rho = -0.96$ for Figure 6.4). The correlation is inverse, so higher α values result in a less pronounced mirroring effect. Since α is a scaling factor that affects the movement of bacteria, we may interpret this by saying that bacteria should not get too sensitive to local changes in nutrient concentration and thus tend to leave nutrient-rich areas almost as soon as they enter them.

The figures corresponding to variation in ν suggest an unstable equilibrium-like relationship between ν and m_1 . Mirroring of nutrient by population is very pronounced for certain values of ν , but barely present for others. The three snapshots in Figure 6.8 serve as visual confirmation of this phenomenon. Since ν affects a bacterium's reliance on its genetic memory, this trend suggests an important property that has to hold for the bacterial population to mirror the bath-wide distribution of nutrient: memory can't be ignored, since ignorance leads to myopic movement. It also can't be relied on too much, or it will produce a deleterious local hypersensitivity effect similar to that caused by high value of α , as discussed in the preceding paragraph.

Finally, returning to the dualist interpretation of the advanced bacterial colony model, if one perceives $p(x,t)$ as a physical counterpart (i.e. a token – see Section 2.1 and footnote 1) of the colony-wide awareness of nutrient supply, then the equilibrium-like relationship between v and m_1 implies that an overly simplistic memory construct does not necessarily induce, and may actually impede, awareness. Only specific levels of reliance on genetic memory allow the bacteria to locate the nutrient on a global scale.

8. Extensions and Future Directions

A first natural extension of the model involves further exploration of the role of α and v for bacterial sensitivity. It is possible to change these parameters into properties of individual bacteria, and allow these bacteria to change α and v from generation to generation, based on previous performance. Combined with survival of the fittest, this modification should allow the colony to reach (evolve) optimal α and v values in the course of the simulation.

A second, more significant extension would be to implement quorum sensing more accurately in the bacterial model. The nutrient bath in the base model is a natural candidate for the virtual counterpart of the autoinducers responsible for the phenomenon of quorum sensing. At the same time, it will be necessary change the source of the exogenous supply to the bacteria themselves, since real bacteria create their own autoinducers. The implementation of the virtual fitness and genetic memory effects in terms of physical intracellular features would be elucidating. Such a physical implementation is made in [8] for model neurons and neural networks in the context of brain modeling.

Further extensions to the model may include quorum sensing between different bacteria species, some of which might be in competition with each other. Such multiple-bacteria models might offer a way to analyze the relationship between quorum sensing and equilibrium in population biology.

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