Lubricating bacteria model for the growth of bacterial colonies exposed to ultraviolet radiation

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In this paper, we study the morphological transition of bacterial colonies exposed to ultraviolet radiation by modifying the bacteria model proposed by Delprato *et al.* Our model considers four factors: the lubricant fluid generated by bacterial colonies, a chemotaxis initiated by the ultraviolet radiation, the intensity of the ultraviolet radiation, and the bacteria's two-stage destruction rate with given radiation intensities. Using this modified model, we simulate the ringlike pattern formation of the bacterial colony exposed to uniform ultraviolet radiation. The following is shown. (1) Without the UV radiation the colony forms a disklike pattern and reaches a constant front velocity. (2) After the radiation is switched on, the bacterial population migrates to the edge of the colony and forms a ringlike pattern. As the intensity of the UV radiation is increased the ring forms faster and the outer velocity of the colony decreases. (3) For higher radiation intensities the total population decreases, while for lower intensities the total population increases initially at a small rate and then decreases. (4) After the UV radiation is switched off, the bacterial population grows both outward as well as into the inner region, and the colony's outer front velocity recovers to a constant value. All these results agree well with the experimental observations [Phys. Rev. Lett. **87**, 158102 (2001)]. Along with the chemotaxis, we find that lubricant fluid and the two-stage destruction rate are critical to the dynamics of the growth of the bacterial colony when exposed to UV radiation, and these were not previously considered.

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I. INTRODUCTION

As an exciting multidisciplinary field, bacterial colonies growing on semisolid agar have served as model systems for studying pattern formation and population dynamics in biological systems. Studies on strains of *Bacillus subtilis* and other bacteria have shown a wide variety of complex patterns depending on nutrient conditions [1–11] and reaction-diffusion equations have been used to model the pattern formation [1,12–19]. In most of the experimental and theoretical studies the environment has been simplified to a uniform one and its changes are due only to the depletion of nutrients or excretion of waste. Actually, the behaviors of bacterial colonies are more complicated.

In nature, to cope with hostile environmental conditions, bacteria have developed sophisticated cooperation and intricate communication capabilities [1,15,20–22]. These include collective production of extracellular "wetting" fluid for movement [1,15,23], direct cell-cell physical interaction via extra-membrane polymers [24,25], long-range chemical signaling [3,10,26–29], and collective activation and deactivation of genes [30,31]. One of the mechanisms for the fundamental movement of bacteria is the secretion of a lubricant layer which makes different media more suitable for swimming (for example, *Bacillus subtilis* secrete "surfactin" [1,32,33]). All these factors lead to complex spatiotemporal patterns in response to adverse growth conditions [1,15].

Recently, experiments on the spatiotemporal response of bacterial colonies of *Bacillus subtilis*, growing in rich nutrient agar, to a sudden change in the environment (by exposing the colony to uniform ultraviolet radiation) have been reported [34]. Among the many interesting phenomena, the most remarkable one is that the bacteria in the central regions of the colonies are observed to migrate towards the colony edge, forming a ring, while they are exposed to UV (ultraviolet) radiation. And after the UV radiation is switched off, the bacterial population is observed to grow both outward as well as inward into the vacated inner region, indicating that the ring pattern is not formed due to depletion of nutrients at the center of the colony. Another remarkable phenomenon is that the colonies' population does not always decrease during UV exposure. For lower intensity UV radiation, the population increases at a small rate initially, this increasing process can last a very long time and then decreases.

A simple reaction-diffusion model was proposed in Ref. [34] to study the mechanism of the observed phenomenology, in which the waste-limited chemotaxis initiated by the UV radiation was considered as leading to what happened in the pattern formation. Using this model, the bacterial population density as a function of distance from the center point of the colony at different exposure times have been simulated under uniform UV radiation strength, and a ringlike pattern finally formed. However, the parameters considered in this model were inadequate to explain other experimental observations. These include the following. (1) The total population grows initially and declines later after the UV radiation turned on, and in Ref. [34] researchers deal with this as a fractional change of the total population of bacteria. (2) For higher UV radiation intensities, after the radiation is switched off, the front velocity on the outside edge of the bacterial colony returns to its preradiation value. (3) For lower UV radiation intensities, a narrow apex also forms at the bacterial colony's front.

Several studies have also been done by Shnerb, Neicu *et al.*, and Lin *et al.* to simulate the response of bacterial colonies exposed to UV radiation [35–37]. Their models incorporate the effect of UV radiation on the growth of a bacterial colony by considering a single convective-diffusion equation. In the single equation they impose the UV radiation regionally, and the bacteria colony migrates mainly with

the movement of the UV source. However, their model is unsuitable for uniform UV radiation which irradiates the entire colony.

In order to more fully model the experimental results we propose a modified lubricating bacteria model. It incorporates chemotaxis which induces bacterial movement, a lubricant layer which restricts the region of movement of the bacteria therefore preventing their unexpected diffusion, and a two-stage destruction process under certain UV radiation. Numerical simulations using the model to simulate morphological transitions of bacterial colonies agree well with experimental observations. Not only are the ringlike patterns formed as in Ref. [34], but other experimentally observed phenomena beyond the scope of that model are also predicted.

This paper is organized as follows. The existing experimental observations of the bacterial colony exposed to the uniform ultraviolet radiation are given briefly in Sec. II. The lubricating bacteria model is proposed in Sec. III. In Sec. IV, we present numerical simulations of the lubricating bacteria model and compare the simulation results with the corresponding experimental observations. Finally, a summary and remarks are given in Sec. V.

II. EXPERIMENTAL OBSERVATIONS

In order to investigate the mechanism of the spatiotemporal response of bacterial populations exposed to UV radiation, experiments of the growth of bacterial colonies exposed to UV radiation were performed in 15 cm diameter Plexiglas petri dishes containing a thin layer of nutrient agar (7 of bacto peptone and 3 agar) [34]. The bacterial colony was initiated with an inoculating needle at a single point source at the center of the petri dish, and the bacteria grew in a thin layer at the surface of the medium. The colony was first allowed to grow in the absence of UV radiation, and then exposed to uniform UV radiation for a fixed period of time. Finally, the UV radiation was turned off and the bacterial colony recovered its growth pattern. The observed phenomena in the experiments [34] can be summarized as follows.

(1) The colony forms a disklike pattern and reaches a constant front velocity when the bacteria grow without UV radiation. (2) As soon as the UV light is turned on, the bacterial population is observed to migrate to the edge of the colony and forms a ringlike pattern finally. The ring forms faster and the front velocity of the colony decreases as the intensity of the UV radiation is increased. (3) For lower UV radiation intensities, the total population increases at a small rate initially and then decreases. For higher intensities, the total population always decreases. (4) After the UV radiation is switched off, the bacterial population is observed to grow both outward as well as inward into the vacated inner region after a recovery time. The front velocity on the outside edge is greater than that on the inside edge.

III. THE MODIFIED MODEL

A. The simple model

The simple reaction-diffusion equations proposed in Ref. [34] consist of three reaction-diffusion equations for bacteria concentration $b(\vec{x},t)$, waste concentration $w(\vec{x},t)$, and chemoattractant concentration $c(\vec{x},t)$:

$$\frac{\partial b}{\partial t} = fb(1-w) - \mu(1-f)b - \nabla \cdot J_c + D_b \nabla^2 b, \tag{1}$$

$$\frac{\partial w}{\partial t} = fb(1-w) + D_w \nabla^2 w, \tag{2}$$

$$\frac{\partial c}{\partial t} = (1-f)b(1-w) + D_c \nabla^2 c. \tag{3}$$

In the above equations, D_b, D_w, and D_c stand for the diffusion coefficients of the bacteria, waste, and chemoattractant, respectively. The first terms on the right-hand side of Eqs. (1) and (2) describe the bacterial growth and the accompanying waste production, respectively. The bacterial growth is limited by the local concentration of waste w and saturates when waste concentration approaches 1. Initially $f=1$ is constant in the absence of the UV radiation and becomes small when UV radiation is switched on. According to Eq. (3), when $f<1$, the bacteria emit a chemoattractant c at a rate which is proportional to the bacterial concentration and is also limited by the waste concentration. The term $-\mu(1-f)b$ in Eq. (1) is responsible for the slow destruction of bacteria by the UV radiation observed experimentally, in which μ is a coefficient representing the rate of the destruction (see next paragraph). The chemotactic flux $J_c=\alpha b(1-b)$ is directed towards the gradient of the concentration of chemoattractant c, and it saturates at large bacterial concentration $b \to 1$ (hard core repulsion), where α is a constant. The last terms in Eqs. (1)–(3) describe linear diffusion of the components. And for simplification, we will call this model I in the following text.

Generally, microorganisms exposed to UV radiation experience an exponential decrease [38]. The single stage exponential decay equation for microbes exposed to UV radiation has the form

$$S(t) = e^{-\mu I t},$$

where $S(t)$ stands for the surviving fraction of the initial microbial population, μ is a standard rate constant, I represents UV intensity, and t is time of exposure. The standard rate constant is independent of intensity.

Thus we can express the slow destruction of bacteria by the UV radiation

$$\frac{\partial b}{\partial t} \sim -\mu I b.$$

We also would like to use this parameter I to represent the reduced UV intensity in the following models. By comparison with the term $-\mu(1-f)b$ in Eq. (1) we see that I equals to $(1-f)$ and so we replace $(1-f)$ and f with I and $(1-I)$ respectively in order to make a clear comparison with actual UV radiation intensities.

Figure 1(a) shows numeric simulations of model I for the bacterial density $b(r)$ as a function of distance r from the center at five unit time intervals after the radiation was turned on, details of the simulation method are described in

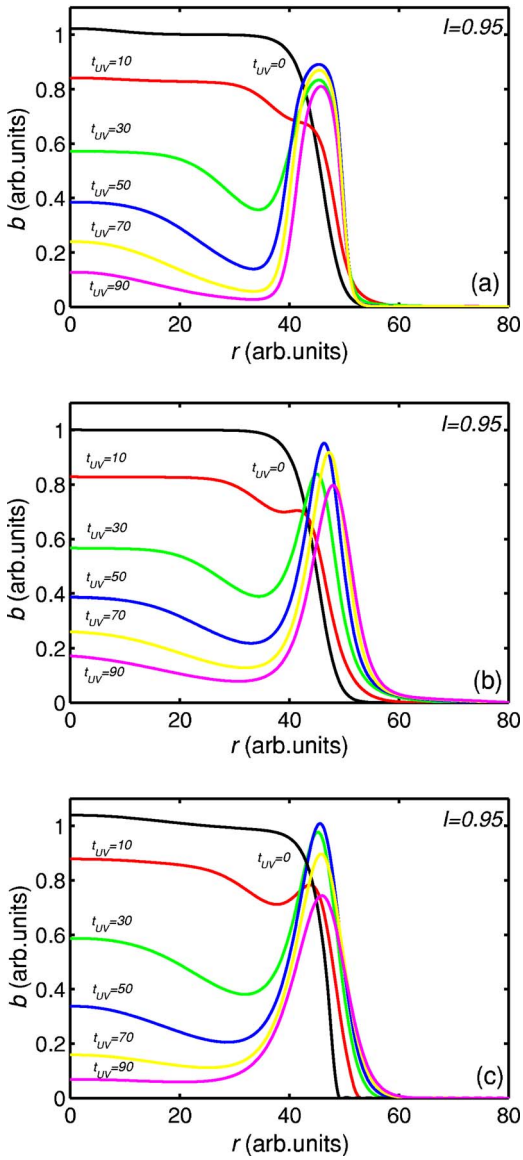

FIG. 1. (Color online) The bacterial population density $b(r)$ as a function of distance r from the center point of the colony at different times for $I=0.95$ [in (a), this is equal to $f=0.05$]. (a) The results of model I. (b) The results of model II, which have modified destruction rate and chemoattrctant flux. The parameters are $D_b=D_c=D_w=1$, $\xi_c=80$, $K_c=100$, $\mu=0.02$, $A=50000$, $B=10$. (c) The results of the model III, which is our final model. The parameters are $D_b=D_l=D_w=D_c=2$, $\gamma=\nu=1$, $\mu=0.1$, $\Gamma=5$, $\xi_c=80$, $K_c=100$, $A=50\,000$, $B=10$. Unless otherwise indicated, the results using models II and III use the same parameter values as those in (b) and (c).

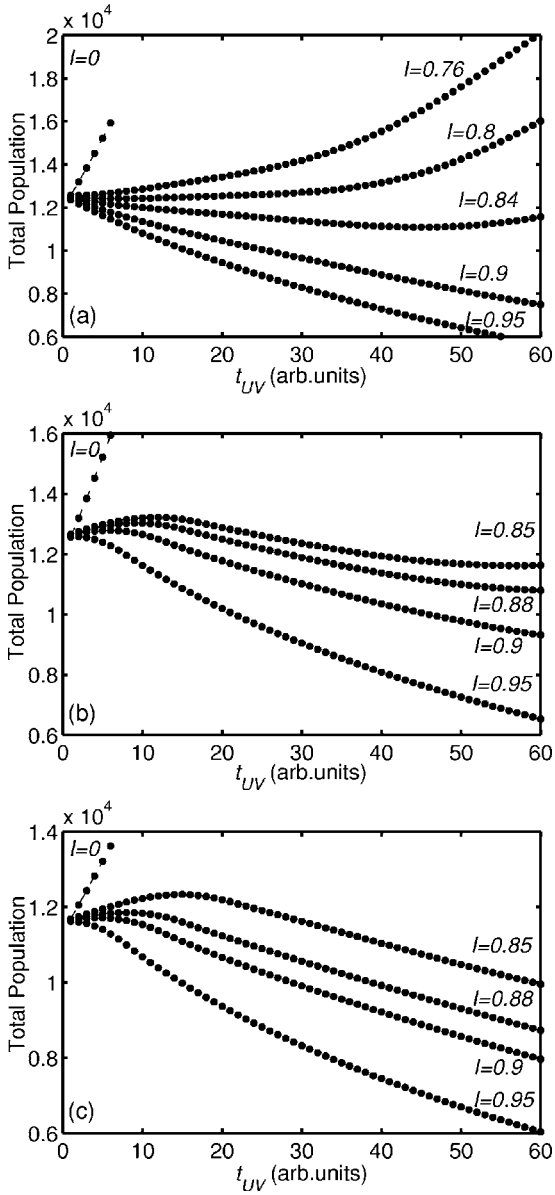

FIG. 2. The total population of the colony vs time for different UV radiation intensities. (a) The results from model I. (b) The results from model II. (c) The results from model III.

Sec. IV. According to the values in Ref. [34], we set $\mu=0.02$, $\alpha=2$, and chose $D_b=D_w=D_c=1$. It can be seen that the model successfully imitated the bacterial density change.

However, for other values of the parameters and, in particular, f, the model failed to correctly predict the phenomenon that after the UV was turned on the total population size slightly grows initially and later declines [see, for example, Fig. 2(a)] which is illustrated in Fig. 3(c) of Ref. [34]. This is due to the behavior of the decreasing term $\mu(1-f)b$ when $f<1$. Even though taking account the increasing term $fb(1-w)$, the increasing time cannot last such a long time before the population begin to decrease.

Model I has further problems due to diffusion of bacteria from the front of the colony for sufficiently small values of I (sufficiently large f). This causes an unexpectedly wide apex [Fig. 3(a)], which leads to a rapid growth in the total bacterial population after a period of population stagnation [Fig. 2(a)]. For larger values of I (smaller values of f), the colony's outer radius undergoes an explosive expansion when the UV radiation is turned of and I is reset to 0 [Fig. 4(a)]. These two predictions of the model do not agree with the experimental results found in Ref. [34].

B. The modified model with exponential decay of bacterial colony's population

As shown above, for lower UV radiation intensities, the expression for the destruction rate of the bacterial population

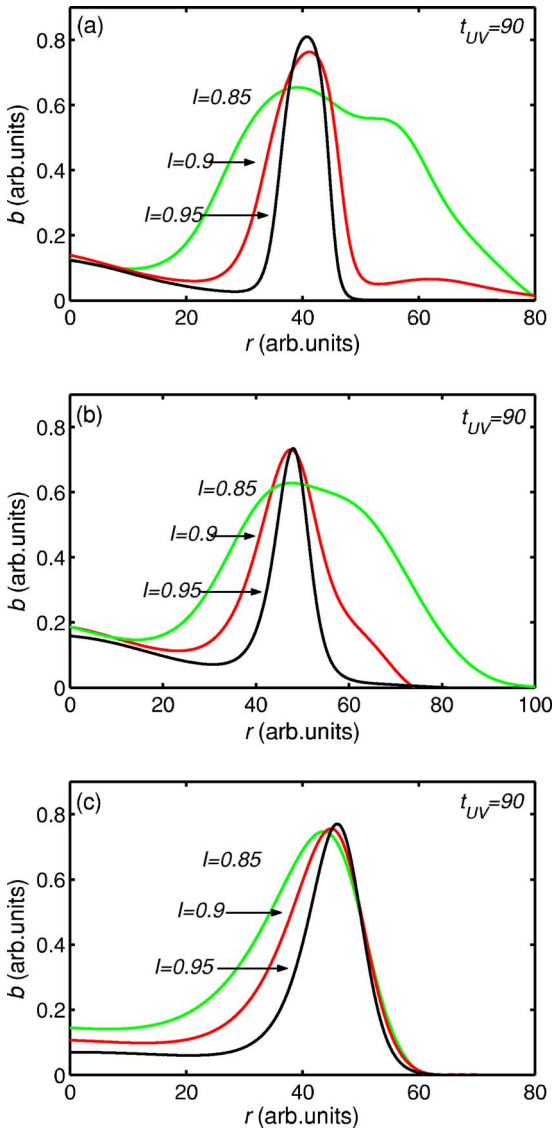

FIG. 3. (Color online) The bacterial population density $b(r)$ as a function of distance r from the center point of the colony at different UV intensities for $t_{\mathrm{UV}}=90$. (a), (b), and (c) are the numeric simulations of the models I, II, and III, respectively. In (a) and (b) there are obvious wide apexes.

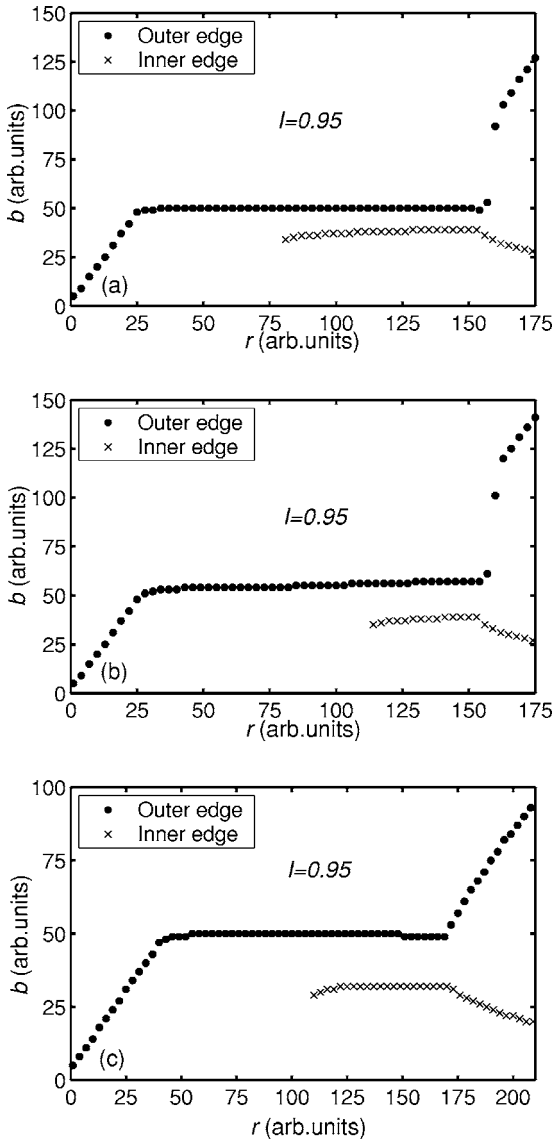

FIG. 4. The radius of the colony as a function of time from the point of inoculation. Three growth periods are considered: the initial growth period, the UV radiation exposure period, and the recovery period. The outer edge of the colony is plotted using (●), and the inner edge using (×). (a) The results of model I, the initial period is for $0<t<25$ and $I=0$; the exposure period is for $25<t<155$ and $I=0.95$; the recovery period is for $155<t<175$ and $I=0$. (b) The results of model II, the three periods are the same as (a). (c) The results of model III, the initial period is for $0<t<40$ and $I=0$; the exposure period is for $40<t<170$ and $I=0.95$; the recovery period is for $170<t<210$ and $I=0$.

is $-\mu Ibt$ in model I. However, it was found that this expression could not satisfactorily model the experimentally observed dynamics of population growth and decay. Even for low values of I it yielded only monotone increases and decreases [see Fig. 2(a)] which, for example, are not in accord with experimental observation No. 3 of Sec. II.

One solution of this problem comes from Kowalski *et al.* [38] who suggest a two-stage survival step function which, in our case, takes the form

$$\frac{\partial b}{\partial t} \sim DS = \begin{cases} -\mu Ibt/2t_c, & t \leqslant 2t_c, \\ -\mu Ib, & t \geqslant 2t_c. \end{cases}$$

They further assume that the threshold t_c is an exponential function of the intensity I:

$$t_c = Ae^{-BI},$$

where A and B are constants. Thus, for a given I, the term $-\mu(1-f)b$ in Eq. (1) which represent the slow destruction of bacteria should change into the above form. We use DS in the model to present this two stage destruction rate expression.

Furthermore, in accordance with the "receptor law" [1,18,39–43], we rewrite the chemoattractant flux as follows:

$$\vec{J}_c = \xi_c D_b b \frac{K_c}{(K_c + c)^2} \nabla c,$$

where ξ_c and K_c are two parameters.

Using these modifications, model I is rewritten as

$$\frac{\partial b}{\partial t} = (1 - I) b(1 - w) + DS - \nabla \cdot \left(\xi_c D_b b \frac{K_c}{(K_c + c)^2} \nabla c \right) + D_b \nabla^2 b, \tag{4}$$

$$\frac{\partial w}{\partial t} = (1 - I) b(1 - w) + D_w \nabla^2 w, \tag{5}$$

$$\frac{\partial c}{\partial t} = Ib(1 - w) + D_c \nabla^2 c. \tag{6}$$

And for simplification, we will call this model model II in the following text.

For the modified bacteria destruction rate and chemoattractant flux, we simulate Eqs. (4)–(6) numerically, the results are shown in Figs. 1(a), 2(b), 3(b), and 4(b). Figure 1(b) shows the bacterial density as a function of distance, and this result agrees well with the experimental result. The fractional changes of the total population as a function of time after the UV radiation is turned on are shown in Fig. 2(b). We see that model II shows the initial growth and later decline in population which model I did not. The initially increasing of total population is due to the different destruction of bacteria.

However from Figs. 3(b) and 4(b) we see that there are still very wide apexes of bacterial density for small UV intensity in model II, which causes the same problem which exists in model I of unexpected rapid growth of total bacterial population after the UV radiation is turned off.

C. The modified model with lubricant layer

For small I, bacteria diffuse from the front of the colony and accumulate in areas away from the colony. Because waste concentrations are very low in these areas, when the value of the reproduction term is bigger than the destruction term μIb, the bacteria will easily reproduce to form a wide apex as shown in Figs. 3(a) and 3(b) and cause a rapid increase in the total population [obvious in Fig. 2(a)]. For large I, because of the strong radiation [μIb is bigger than the reproduction rate $(1-I)b(1-w)$], the bacteria diffuse to a wide area away from the front, and can not reproduce to a considerable population. However, when I becomes 0 again, these bacteria rapidly reproduce and the radius of the colony undergoes an explosive expansion as shown in Figs. 4(a) and 4(b). This is not in agreement with the experimental results of Ref. [34], in which there are no wide apexes of bacteria density for small UV radiation intensities and the bacterial colony's outer front velocity returns to the preradiation velocity after the UV radiation is turned off.

The experimental observations indicate that there must be a mechanism which can hold the bacteria in their colony region to prevent the unexpected diffusion. According to Refs. [1,15,23,32,33], this mechanism is the lubricant layer secreted by the bacterial colony. We consider the model in Ref. [15] which was used to simulate the branching pattern formation of the *Bacillus subtilis* colonies growing on nutrient-poor substrates. After converting to a dimensionless form, the equations of Ref. [15] are

$$\frac{\partial b}{\partial t} = \nabla \cdot (D_b l^\gamma \nabla b) + bn - b, \tag{7}$$

$$\frac{\partial n}{\partial t} = \nabla^2 n - bn, \tag{8}$$

$$\frac{\partial l}{\partial t} = \nabla \cdot (D_l l^\nu \nabla l) + \Gamma bn(1 - l) - \lambda l, \tag{9}$$

$$\frac{\partial s}{\partial t} = b, \tag{10}$$

where $b(\vec{x},t)$ is the density of motile bacteria, $n(\vec{x},t)$ is the concentration of nutrients, $l(\vec{x},t)$ stands for the height of the lubricant layer in which the bacteria swim, and $s(\vec{x},t)$ represents the density of stationary bacteria. In Eq. (7) $\nabla \cdot (D_b l^\gamma \nabla b)$ describes the diffusion of the bacteria, D_b is the diffusion coefficient, and the exponent γ represents the relation of bacterial movement and local lubricant height. The term bn represents the bacteria reproduction rate, and the last term b also stands for the dimensionless sporulation rate. Equation (8) gives the diffusion and consumption rate of the nutrients. In Eq. (9) $\nabla \cdot (D_l l^\nu \nabla l)$ describes the diffusion of the lubricant layer, while the term $\Gamma bn(1-l)$ represents the lubricant production by the bacteria and $-\lambda l$ is absorption into the agar. The parameters Γ and λ are the lubricant production and absorption rates respectively, and the exponent ν plays the same role as γ.

Bacterial movement depends exponentially on the local lubricant height, which means that the lubricant layer restricts the movement region of the bacteria and prevents the unexpected diffusion. We now combine this idea with the previous two models, omitting the terms $s(\vec{x},t)$ and $n(\vec{x},t)$ according to the experimental conditions in Ref. [34], to get

$$\frac{\partial b}{\partial t} = \nabla \cdot (D_b l^\gamma \nabla b) + (1 - I) b(1 - w) + DS - \nabla \cdot \left(\xi_c D_b b \frac{K_c}{(K_c + c)^2} \nabla c \right), \tag{11}$$

$$\frac{\partial l}{\partial t} = \nabla \cdot (D_l l^\nu \nabla l) + \Gamma (1 - I) b(1 - w)(1 - l). \tag{12}$$

With Eqs. (5) and (6), Eqs. (11) and (12) describe the dynamics of the bacterial colonies exposed to UV radiation. We will call this model III in the following text. It will be seen that model III gives a much better description of the morphological transition of bacterial colonies exposed to UV radiation.

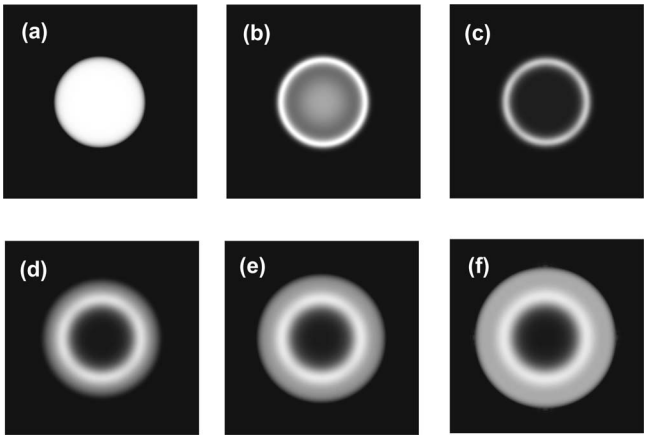

FIG. 5. The spatiotemporal response of the whole bacterial colony under UV radiation. (a)–(c) The images of the bacterial colony as a function of time t_{UV} under UV radiation. (a) $t_{\mathrm{UV}}=0$, (b) $t_{\mathrm{UV}}=30$, and (c) $t_{\mathrm{UV}}=90$. The UV radiation parameter $I=0.95$. (d)–(f) Recovery period of the bacterial population as a function of time t_r after the UV radiation is switched off. The bacterial colony was allowed to recover for (d) $t_r=10$, and (e) $t_r=20$, (f) $t_r=30$. The bacterial population is observed to grow both outward as well as into the vacated inner region.

IV. NUMERIC SIMULATIONS OF THE LUBRICATING BACTERIA MODEL

Equations (5), (6), (11), and (12) are our final modified model for the morphological transition of bacterial colonies exposed to UV radiation. In order to compare it with experimental observations, we carry out numerical simulations in a square lattice with 300×300 lattice cells, space interval $dx=1$, time interval $dt=0.01$. For the initial conditions, we set b to be zero everywhere except in the center, and the other fields to be zero everywhere.

The initial UV radiation intensity is set to 0 ($I=0$) and it remains constant until the colony has expanded to a big enough disklike area. Then, at time $t=t_b$, we fix the value of $I\neq0$, so the growth of the bacteria is restrained and the colony expansion slows down. The bacteria immediately begin to emit chemoattractant c at the edges of the colony, where waste w is below the limiting level of 1. The colony begins to migrate to the edge for chemotaxis. At a later time $t=t_e$, the value of I is again set to 0. To summarize, time between 0 and t_b is the initial growth period, time between t_b and t_e is the UV radiation exposure period and time larger than t_e is the recovery period.

Figures 1(c), 2(c), 3(c), 4(c), and 5–8 show the results of the numeric simulations. Note that the addition of the lubricant layer decreases the expansion velocity of the colony. We compensate for this by setting $D_b=D_w=D_l=D_c=2$ so that the final dimensions of the colony are the same as those in our previous numerical simulations. This does not affect the results and makes comparison with our previous models clearer.

To show the rearrangement of the bacterial colony as a function of time, the bacterial density $b(r)$ as a function of distance r from the center is plotted in Fig. 1(c). We can see that the bacteria move to the edge of the colony due to the attractive chemical c emitted by the fringe bacteria. This result is the same as models I and II, and agrees well with experimental observation.

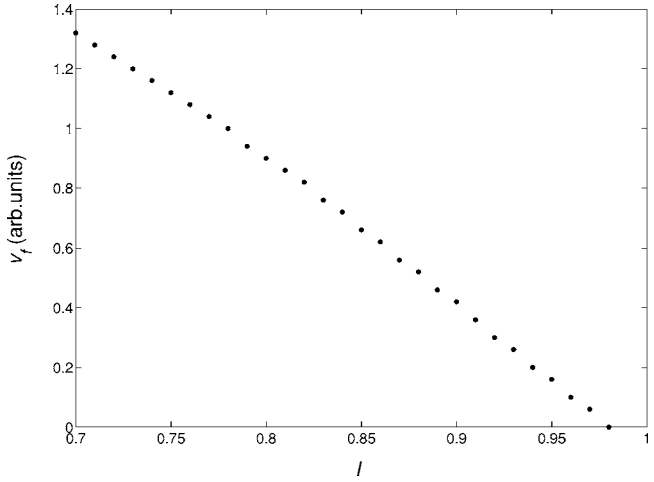

FIG. 6. The bacterial colony's front velocity v_f vs the UV radiation intensity I.

The total population of the bacterial colony as a function of time versus different I is shown in Fig. 2(c). For low values of I the population increases at a small rate initially and then decreases. For high values of I the population always decreases with no obvious initial increase. Comparing Fig. 2(b) and Fig. 2(c), we find that there is an obvious difference between model III and model II for $I=0.85$. In Fig. 2(b), when the UV radiation time $t_{\mathrm{UV}}>40$, the population stops decreasing and begins to increase. This is because the bacteria which diffuse to the outer area begin to reproduce rapidly. Note that Fig. 2(c) did not show this unexpected increase. These results are an improvement on model II and are in good agreement with the observed phenomena [34].

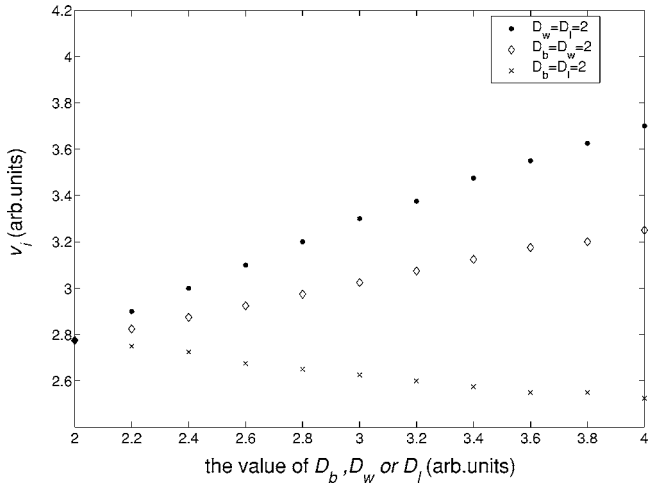

FIG. 7. The bacterial colony's initial front velocity v_i vs the diffusion coefficients D_w, D_b, and D_l, respectively. The solid circles (●) are the v_i vs D_b with the other two parameters $D_w=D_l=2$. The open squares (◇) are the v_i vs D_l with the other two parameters $D_b=D_w=2$. The symbols (×) are the v_i vs D_w with the other two parameters $D_b=D_l=2$.

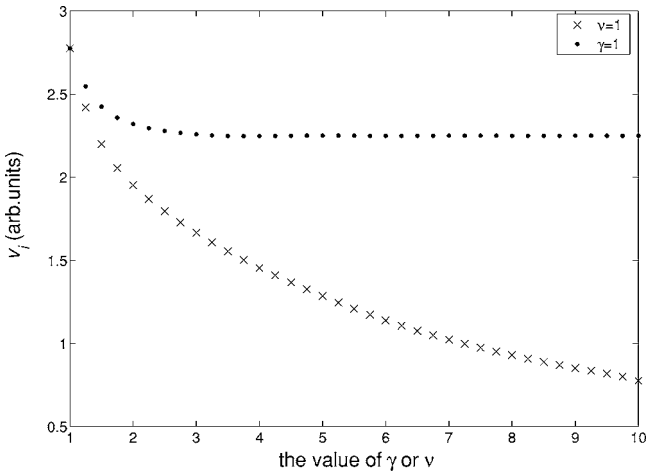

FIG. 8. The bacterial colony's initial front velocity v_i as a function of γ and ν, respectively. Obviously, γ has a lager effect than ν.

The bacterial colony's density distributions are shown in Fig. 3(c) for different UV radiation intensities. Because the final destruction rate is proportional to I, it is obvious that for higher I, the colony has a smaller density in the central area, and the formation of the ring is quicker. Because of the restriction of the lubricant layer, diffusion from the front edge of the colony is greatly impeded. Thus the wide apex does not appear nor does the unexpected population increase. Comparing Fig. 3(c) with Figs. 3(a) and 3(b) we see that this model is in much closer agreement with the experimental observations of Ref. [34].

In Fig. 4(c) we see that the colony grows normally and has a constant front velocity. Note that the initial growth period is longer than that of models I and II because the initial growth velocity in model III is less than that of models I and II and we must therefore use a longer initial growth time in order to achieve a similar colony radius. After the UV radiation is turned on at $t=40$ ($I=0.95$) the front velocity decreases a great deal. Half way through the UV radiation exposure time a ring at the inner edge of the colony appears. Finally, at $t=170$, the UV radiation is turned off ($I=0$) and the bacterial colony once again resumes growth with a constant front velocity. It also grows into the evacuated inner region with a slower growth velocity. Figures 5(a)–5(c), show the spatial-temporal growth of the colony under UV radiation while Figs. 5(d)–5(f) show images of the recovering colony. These results, much better than those of models I and II, agree will with the observed phenomena [34].

In Fig. 6 we show the quantitative relationship between UV radiation intensity I and the front velocity growth of the bacterial colony. We see that the UV radiation intensity is a very significant factor in the expansion of the bacterial colony. In Ref. [34] Delprato *et al.* experimentally measured this relationship and they found for $I=30$, 12, and 7 W/m^2 the front velocities were $v_f=0.004$, 0.006, and 0.010 μm/s, respectively. By comparison we see that our results show the same trends as the experimental results.

The dependences of the initial front velocity v_i vs D_w, D_b, and D_l are shown in Fig. 7. From this figure we can see that D_b plays the main role in altering the velocity v_i. With increase of D_b or D_l the velocity v_i increases, while increasing D_w decreases v_i. A larger D_b means faster diffusion of bacteria in the front of the colony, so the colony will have a larger front velocity. Furthermore, since the lubricant layer is essential for the swimming of the bacteria, a larger D_l also increase the colony's front velocity. On the other hand, for a larger D_w, the waste concentration will have a larger value in the out area near the colony's front. This retards the reproduction rate of the bacteria causing a decrease in the colony's front velocity.

The bacterial and the lubricant fluid diffusion coefficients depend on the height of the fluid and on the positive exponents γ and ν respectively. Diffusion due to the lubricant affects the population at the front of the colony considerably because in that region the height l is less than 1. A higher value of γ or ν lowers the diffusion term $\nabla\cdot(D_b l^\gamma \nabla b)$ or $\nabla\cdot(D_l l^\nu \nabla l)$, and the diffusion of the bacteria or lubricant fluid slows down. γ and ν are very closely related to the nature of the bacteria and the agar. For example, a more viscous fluid results in higher values of both γ and ν. Fig. 8 shows the colony's initial front velocity v_i vs the exponents γ and ν, respectively. We can see that γ has a much larger effect than the ν.

V. CONCLUSIONS AND REMARKS

In this paper a modified lubricating bacteria model was proposed which included the effects of the waste that limits the growth of the bacteria, the lubricant layer and the chemoattractants. Our model also took into account a two-stage population destruction processes due to UV radiation. We found that along with chemoattractant, the two-stage destruction processes and the lubricant layer are essential mechanisms in the movement of *Bacillus subtilis* growing in nutrient rich environments and exposed to uniform UV radiation. Using our model, the followings is shown. (1) Without the UV radiation the colony forms a disklike pattern and reaches a constant front velocity. (2) After the radiation is switched on the bacterial population migrates to the edge of the colony and forms a ringlike pattern. As the intensity of the UV radiation is increased the ring forms faster and the outer velocity of the colony decreases. (3) For higher radiation intensities the total population always decreases, while for lower intensities the total population initially increases at a small rate and then decreases. (4) After the UV radiation is switched off, the bacterial population grows both outward as well as into the inner region, and the colony's outer front velocity returns to a constant value. This all agrees well with experimental observations [34]. Finally, we discuss the dependence of the colony's front velocity on radiation intensities (I), diffusion constants (D_b, D_w, and D_l), and lubricant height related exponents (γ and ν). We show that increases in the UV radiation intensity cause the colony's front velocity to decrease almost linearly. The bacteria diffusion coefficient D_b plays the main role in altering the velocity v_i. However, increases of D_b or the lubricant diffusion coefficient D_l also cause increases in the velocity v_i. On the other hand, increasing the waste diffusion coefficient D_w cause decreases in the velocity v_i. It is also found that γ has a much larger effect on

v_i than the exponent ν. This means that changes in the bacteria and agar's qualities which relate to the bacteria's lubricant-dependent diffusion coefficient can more easily affect the colony's front velocity than the changes which relate to the lubricant's diffusion coefficient.

The spatiotemporal response of bacterial colonies growing on nutrient rich agar exposed to uniform UV radiation is a complex process which involves many mechanisms. The bacterial density distribution is obviously different for different values of I, which is a function of the UV radiation's strength. Under UV radiation, the population migrates to the outer edges of the colony and finally forms a ringlike pattern. Using our model, we explain most of the experimental phenomena, especially the bacterial population dynamics.

In the experiments [34] there are two retarded growing periods. The first is observed after inoculation of the bacteria in the center of the petri dish, and this delay can last tens of hours. It is due to the slowness of the secretion of special enzymes by the bacteria which are needed in the new environment. When the concentration of these enzymes reaches a critical value the bacteria begin to grow fast. The second period of slow growth is observed after the UV radiation is turned off and can last for several hours. During this period the bacteria may be repairing their DNA and this may cause a retardation on growth rate. Once the DNA has been repaired they begin to grow fast again. Our model does not contain terms to describe these two slow growth periods and we are still looking for proper ways to study them.

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