Pattern formation of a reaction-diffusion system with self-consistent flow in the amoeboid organism *Physarum* plasmodium

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The amoeboid organism, the plasmodium of *Physarum polycephalum*, moves by forming a spatiotemporal pattern of contraction oscillators. This biological system can be regarded as a reaction-diffusion system with spatial interaction via active flow of protoplasmic sol in the cell. We present a reaction-diffusion system with self-consistent flow on the basis of the physiological evidence that the flow is determined by contraction patterns in the plasmodium. Such a coupling of reaction, diffusion, and advection is characteristic of biological systems, and is expected to be related to control mechanisms of amoeboid behavior. Using weakly nonlinear analysis, we show that the envelope dynamics obeys the complex Ginzburg-Landau (CGL) equation when a bifurcation occurs at finite wave number. The flow term affects the nonlinear term of the CGL equation through the critical wave number squared. A physiological role of pattern formation with the flow is discussed. [S1063-651X(99)11501-0]

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I. INTRODUCTION

The plasmodium of *Physarum polycephalum* is a large amoeboid cell, showing contraction-relaxation cycles everywhere within the organism. These local contractions generate intracellular transport of endoplasmic sol [1]. The plasmodium can crawl when the endoplasmic flow is organized throughout the organism. Some types of spatiotemporal pattern of the contraction have been observed after stimulation and discussed in relation to development of amoeboid behavior [2–5]. The contraction apparatus is located at the outer layer of the plasmodium (ectoplasm) [6–8]. Experimental results imply that chemical oscillation is a clock for the rhythmic contraction [9,10]. Possible candidates for the chemicals are, for example, Ca^{2+} and adenosine 5′-triphosphate [3,11,12]. Coupled-oscillator and reaction-diffusion systems have been presented as mathematical models of contraction pattern formation in the plasmodium [13]. They are based on chemical oscillations and diffusion of the chemicals in the ectoplasm. These models have explained experimental results in some simple situations.

Spatial interaction between chemical oscillators occurs by diffusion of chemical substances in cytoplasm, and by protoplasmic streaming in the inner part of the plasmodium (endoplasm) as well. The protoplasmic streaming transports mass and momentum of the endoplasm, and it also transports chemicals in the cytosol. The contraction pattern is modified by inhibition of the streaming [5,14,15]. Miyake *et al.* [15] proposed a model of the information processing system with two levels of subsystems corresponding to the endoplasmic oscillators with long-range interaction and the ectoplasmic ones with short-range interaction. Some more physical models based on hydrodynamics and chemical kinetics have been presented in Refs. [16–18]. These models are constructed on the mechanical interaction of mechanochemical oscillators by viscoelasticity in the plasmodium.

Although the endoplasmic flow evidently exists in the plasmodium, some oscillatory phenomena in the plasmodium can be illustrated with the simple system of diffusively coupled oscillators without any flow effects [13]. Thus it is inevitable to study the role of the endoplasmic flow when we analyze the pattern formation of contraction oscillation in the plasmodium. In the present paper we discuss transportation effect of chemicals by endoplasmic flow on the contraction oscillation. First, we formulate the schematic framework for contraction pattern formation from the view of the oscillatory reaction-diffusion system with self-consistent flow. We do not go into details of reaction kinetics for chemical oscillators and mechanism of the contractile apparatus in the plasmodium. Thereby, we avoid including experimentally unclear and invalid assumptions in our framework. We adopt only known facts and plausible kinetics in the *Physarum* plasmodium. Next, we study weakly nonlinear dynamics of our reaction-diffusion system. Using the method of multiple scales, we obtain the complex Ginzburg-Landau (CGL) equation which describes the envelope dynamics of chemical oscillation near the bifurcation point. We show that the flow term affects the nonlinear term of the CGL equation. A possible role of the plasmodial pattern formation is discussed from a physiological point of view.

II. BASIC EQUATIONS

The plasmodium of *Physarum* has a cytoplasmic cortex (ectoplasmic gel) filled with endoplasmic sol. The ectoplasm makes periodic contraction and relaxation, and it causes intracellular streaming of the endoplasm. A sheet of cytoplasm becomes thick when the endoplasm is flowing into it. Metabolic chemicals regulate contraction cycles [9,10]. These oscillating chemicals are exchanged between endoplasm and ectoplasm, and flow in and out via the streaming. Thus we present the dynamics of rhythmic pattern formation in the *Physarum* plasmodium as the following equations for metabolic elements in the ectoplasmic gel $\mathbf{u}_{\mathrm{gel}}$ and endoplasmic sol $\mathbf{u}_{\mathrm{sol}}$:

$$\frac{\partial h}{\partial t}+\vec{\nabla}\cdot(h\vec{v})=0,$$

$$\frac{\partial \mathbf{u}_{\mathrm{gel}}}{\partial t}=\mathbf{F}_{\mathrm{gel}}(h,\mathbf{u})+\vec{\nabla}\cdot(D_{\mathrm{gel}}\vec{\nabla}\mathbf{u}_{\mathrm{gel}}), \tag{1}$$

$$\frac{\partial \mathbf{u}_{\mathrm{sol}}}{\partial t}+\vec{v}\cdot\vec{\nabla}\mathbf{u}_{\mathrm{sol}}=\mathbf{F}_{\mathrm{sol}}(h,\mathbf{u})+\frac{1}{h}\vec{\nabla}\cdot(D_{\mathrm{sol}}h\vec{\nabla}\mathbf{u}_{\mathrm{sol}}),$$

where h is the thickness of the endoplasmic sol and $\vec{v}$ is the averaged velocity of the endoplasmic flow. The N-component vector $\mathbf{u}$ is a metabolic species in gel and sol, $(\mathbf{u}_{\mathrm{gel}},\mathbf{u}_{\mathrm{sol}})=(u^1,\ldots,u^N)$. Vectors $\mathbf{F}_{\mathrm{gel}}$ and $\mathbf{F}_{\mathrm{sol}}$ represent reaction kinetics among metabolic elements and exchanges of them between the ectoplasm and endoplasm. The quantities D_{gel} and D_{sol} denote diagonal matrices of diffusion constants of metabolic elements. Experiments imply that the chemical oscillator exists in the ectoplasm. Thus we assume the local dynamics of $\mathbf{u}_{\mathrm{gel}}$ has a stable limit cycle which the supercritical Hopf bifurcation gives rise to.

We note that Eq. (1) becomes the closed system if the dynamics of $\vec{v}$, the equation of motion of endoplasmic flow, is given. We consider the facts that metabolic chemicals act on the contractile apparatus in the ectoplasm, and the gradient of contraction force induces the endoplasmic streaming [19]. The contraction pattern determines the streaming, and vice versa. Now we assume that the endoplasmic streaming is the flow with small Reynolds numbers and it has the form of Darchy's law $\vec{v}=-q(h)\vec{\nabla}P(\mathbf{u})$; here P is the intracellular pressure and q is the permeability. This means the stationary flow approximation since deformation of the ectoplasm is very slow compared with variation of the endoplasmic flow. The viscoelasticity of the ectoplasmic cortex is ignored. In Eq. (1) the sol-gel conversion is also ignored, and thus the mass conservation of endoplasmic sol is satisfied. This implies a limitation of the model, such as cell migration, and the formation and reconnection of a network of protoplasmic strands.

In the following, we assume that the thickness h of the endoplasm is almost constant all over the plasmodium, and that the diffusion constants of the metabolic elements are homogeneous in the plasmodium. The intracellular pressure P is expanded around the homogeneous static state $\mathbf{u}=\mathbf{u}_s$ as

$$P(\mathbf{u})=P(\mathbf{u}_s)+\sum_j (u^j-u_s^j)\frac{\partial P}{\partial u^j}(\mathbf{u}_s)+\text{ higher order terms.}$$

Hereafter, we ignore the higher order terms which have no effects on results deduced by the weakly nonlinear analysis. Under these assumptions, we rewrite the metabolic dynamics of Eq. (1) in the form of the reaction-diffusion-advection equations:

$$\frac{\partial \mathbf{u}}{\partial t}+M\vec{\nabla}\mathbf{u}\cdot\vec{\nabla}\mathbf{u}=\mathbf{F}(\mathbf{u};\mu)+D\vec{\nabla}^2\mathbf{u}, \tag{2}$$

where $\mathbf{F}$ is reaction kinetics, M represents a tensor of advection coefficients induced by endoplasmic flow, and D is a diagonal matrix of diffusion constants. The advection quantity M depends on the thickness of the endoplasm and the mechanism inducing the intracellular pressure. We assume that $\mathbf{F}(\mathbf{0};\mu)=\mathbf{0}$ and hence Eq. (2) has a trivial homogeneous steady solution $\mathbf{u}=\mathbf{0}$. We also assume that the trivial solution is stable if $\mu<\mu_c$ (μ_c denotes the bifurcation point) and otherwise it is unstable. The reaction source $\mathbf{F}(\mathbf{u};\mu)$ can be expanded around the trivial solution as

$$\mathbf{F}(\mathbf{u};\mu)=L\mathbf{u}+N_2\mathbf{u}\mathbf{u}+N_3\mathbf{u}\mathbf{u}\mathbf{u}+\cdots,$$

$$L=\left(\frac{\partial F^i}{\partial u^j}\right),\quad N_2=\left(\frac{\partial^2 F^i}{\partial u^j\partial u^k}\right),$$

$$N_3=\left(\frac{\partial^3 F^i}{\partial u^j\partial u^k\partial u^l}\right),\ \ldots\quad \text{at } \mathbf{u}=\mathbf{0}.$$

Linearizing Eq. (2) near the trivial solution, we obtain a solution for some Fourier component,

$$\mathbf{u}=We^{\lambda t}e^{ikx}\mathbf{U}+\text{c.c.},$$

where W is an arbitrary constant and c.c. means complex conjugate. The stability of this component is determined by the eigenvalue problem

$$(L-k^2D)\mathbf{U}=\lambda\mathbf{U} \tag{3}$$

for the eigenvalue λ and eigenvector $\mathbf{U}$. On the bifurcation point, $\mathrm{Re}\,\lambda=0$ and $\partial(\mathrm{Re}\,\lambda)/\partial k=0$ are satisfied at some $k=k_c$ for the maximal eigenvalue(s); here we assume this maximal eigenvalue is simple. In the vicinity of the bifurcation point $(\mu,k)=(\mu_c,k_c)$, the eigenvalue is

$$\lambda=\lambda_c+\left.\frac{\partial\lambda}{\partial\mu}\right|_c(\mu-\mu_c)+\left.\frac{\partial\lambda}{\partial k}\right|_c(k-k_c)+\frac{1}{2!}\left.\frac{\partial^2\lambda}{\partial k^2}\right|_c(k-k_c)^2+\cdots, \tag{4}$$

where the subscript c denotes the bifurcation point, and expansion coefficients are given as

$$\lambda_c=\pm i\omega_c,\quad \left.\frac{\partial\lambda}{\partial k}\right|_c=\pm ic_g,\quad \omega_c,c_g\geqslant 0.$$

Now we consider two oscillatory types of bifurcations: (i) $k_c=0$ and $\omega_c\neq 0$ (Hopf bifurcation); (ii) $k_c\neq 0$ and $\omega_c\neq 0$ (traveling-wave type). In case (i), each component of $\mathbf{u}$ is so smooth in space that the advection term is smaller than the nonlinear and diffusion terms in Eq. (2). Although the weakly nonlinear analysis for this case yields the complex Ginzburg-Landau equation as the envelope dynamics, the advection term has no effect on the CGL equation. In case (ii), the advection term competes with the nonlinear and diffusion terms as shown in the next section.

III. ENVELOPE EQUATION

Let us consider the envelope equation when the bifurcation of traveling-wave type occurs, on a basis of weakly nonlinear analysis [20]. We denote the bifurcation parameter by $(\mu-\mu_c)\sim\epsilon^2$ for $\mu>\mu_c$, and assume that $u^j\sim O(\epsilon)$

$(j=1,\ldots,N)$ in the vicinity of the bifurcation point. In the following analysis, the envelope equation is derived for one spatial dimension system of Eq. (2) with the single traveling wave. In the Appendix we comment on the derivation of envelope equations of Eq. (2) for the counterpropagating waves and for two spatial dimensions [20]. We introduce perturbation expansions and multiple scales,

$$\mathbf{u}\sim\epsilon\mathbf{u}_1+\epsilon^2\mathbf{u}_2+\epsilon^3\mathbf{u}_3+\cdots,\quad L\sim L_0+\epsilon^2L_2+\cdots,$$
$$X=x-c_pt,\quad \xi=\epsilon(x-c_gt),\quad \tau=\epsilon^2t, \tag{5}$$

where $c_p=\omega_c/k_c$ is the phase velocity and c_g is the group velocity. Substitution of Eq. (5) into Eq. (2) yields perturbation equations for each order in ϵ:

$$\left(L_0+D\frac{\partial^2}{\partial X^2}+c_p\frac{\partial}{\partial X}\right)\mathbf{u}_m=\mathbf{b}_m,\quad m=1,2,3,\ldots \tag{6}$$

where $\mathbf{b}_m$ denotes the inhomogeneous term of the mth order equation.

For the first order equation in Eq. (6), the inhomogeneous term is $\mathbf{b}_1=\mathbf{0}$. Then we have a solution

$$\mathbf{u}_1=W(\xi,\tau)e^{ik_cX}\mathbf{V}+\text{c.c.},$$

where $\mathbf{V}$ is an eigenvector of an eigenvalue $\lambda=-i\omega_c$ for the eigenvalue problem (3) on the bifurcation point. For the second order equation, that is $m=2$ in Eq. (6), we expand the solution and inhomogeneous term as a Fourier series in terms of the phase $\phi=k_cx-\omega_ct$,

$$\mathbf{u}_2=\sum_l\mathbf{u}_2^{(l)}e^{il\phi},\quad \mathbf{b}_2=\sum_l\mathbf{b}_2^{(l)}e^{il\phi}.$$

Then the solvability conditions for $\mathbf{u}_2$ are

$$(\mathbf{V}^*,\mathbf{b}_2^{(+1)})=(\overline{\mathbf{V}}^*,\mathbf{b}_2^{(-1)})=0,$$
$$\mathbf{b}_2^{(+1)}=-\frac{\partial W}{\partial\xi}(c_g+2ik_cD)\mathbf{V},\quad \mathbf{b}_2^{(-1)}=\overline{\mathbf{b}}_2^{(+1)}. \tag{7}$$

These conditions are obviously satisfied since

$$c_g=i\left.\frac{\partial\lambda}{\partial k}\right|_c=-2ik_c\frac{(\mathbf{V}^*,D\mathbf{V})}{(\mathbf{V}^*,\mathbf{V})}.$$

Here we use the adjoint eigenvalue problem $(L_0^*-k^2D)\mathbf{U}^*=\overline{\lambda}\mathbf{U}^*$ of Eq. (2). Thus we must advance our calculation to the third order to obtain the envelope equation. For the third order equation in ϵ, we expand $\mathbf{u}_3$ and $\mathbf{b}_3$ as

$$\mathbf{u}_3=\sum_l\mathbf{u}_3^{(l)}e^{il\phi},\quad \mathbf{b}_3=\sum_l\mathbf{b}_3^{(l)}e^{il\phi},$$

then the solvability conditions for $\mathbf{u}_3$ are

$$(\mathbf{V}^*,\mathbf{b}_3^{(+1)})=(\overline{\mathbf{V}}^*,\mathbf{b}_3^{(-1)})=0,$$
$$\begin{aligned}\mathbf{b}_3^{(+1)}=&\frac{\partial W}{\partial\tau}\mathbf{V}-c_g\frac{\partial\mathbf{u}_2^{(1)}}{\partial\xi}+2k_c^2\overline{W}M(\mathbf{u}_2^{(2)}\overline{\mathbf{V}}+\overline{\mathbf{V}}\mathbf{u}_2^{(2)})\\&-WL_2\mathbf{V}-WN_2(\mathbf{u}_2^{(0)}\mathbf{V}+\mathbf{V}\mathbf{u}_2^{(0)})-\overline{W}N_2(\mathbf{u}_2^{(2)}\overline{\mathbf{V}}\\&+\overline{\mathbf{V}}\mathbf{u}_2^{(2)})-|W|^2WN_3(\mathbf{V}\mathbf{V}\overline{\mathbf{V}}+\mathbf{V}\overline{\mathbf{V}}\mathbf{V}+\overline{\mathbf{V}}\mathbf{V}\mathbf{V})\\&-2ik_cD\frac{\partial\mathbf{u}_2^{(1)}}{\partial\xi}-\frac{\partial^2W}{\partial\xi^2}D\mathbf{V},\\ \mathbf{b}_3^{(-1)}=&\overline{\mathbf{b}}_3^{(+1)}.\end{aligned} \tag{8}$$

From these conditions, we obtain the complex Ginzburg-Landau equation:

$$\frac{\partial W}{\partial\tau}=c_1W-c_2|W|^2W+c_3\frac{\partial^2W}{\partial\xi^2},\quad c_j=(\mathbf{V}^*,\mathbf{V}_j)/(\mathbf{V}^*,\mathbf{V}),$$
$$\begin{aligned}\mathbf{V}_1=&L_2\mathbf{V},\\ \mathbf{V}_2=&-2k_c^2M\{\overline{\mathbf{V}},(L_0^{(2,2)})^{-1}(N_2^{(+1)}\mathbf{V}\mathbf{V})\}\\&+N_2\{\mathbf{V},L_0^{-1}(N_2^{(-1)}\{\mathbf{V},\overline{\mathbf{V}}\})\}\\&+N_2\{\overline{\mathbf{V}},(L_0^{(2,2)})^{-1}(N_2^{(+1)}\mathbf{V}\mathbf{V})\}\\&-N_3(\mathbf{V}\mathbf{V}\overline{\mathbf{V}}+\mathbf{V}\overline{\mathbf{V}}\mathbf{V}+\overline{\mathbf{V}}\mathbf{V}\mathbf{V}),\\ \mathbf{V}_3=&D\mathbf{V}-(c_g+2ik_cD)(L_0^{(1,1)})^{-1}(c_g+2ik_cD)\mathbf{V},\end{aligned} \tag{9}$$

where $L_0^{(l,m)}=L_0-(lk_c)^2D+im\omega_c$, $N_2^{(l)}=N_2+lk_c^2M$, and $\{X,Y\}=XY+YX$. According to the dispersion relation (4), $c_1=(\partial\lambda/\partial\mu)_c$ and $c_3=(1/2)(\partial^2\lambda/\partial k^2)_c$ are generically complex constants.

Equation (9) describes the small-amplitude dynamics of the system near the bifurcation point, that is, slow and slight modulation of traveling wave with the wave number k_c and frequency ω_c by the quantity W. We note that the coefficient of the nonlinear term, c_2, depends on the quantity k_c^2M through the vector $\mathbf{V}_2$. For the ordinary (no flow) reaction-diffusion system, this quantity is vanishing in the small-amplitude dynamics of the system. Since the advection term has the form of the gradient of the metabolic species, c_2 depends on the critical wave number k_c.

We consider the effect of the coefficient c_2 on solutions of the CGL equation. The CGL equation has various types of solutions depending on its coefficients [21], but we will not dwell on each of them. We emphasize that c_2 determines the amplitude of nontrivial (finite-amplitude) solutions and induces the instability and bifurcation of the solutions [21]. Nonlinearity is essential to finite-amplitude solutions and bifurcations of the CGL equation. The sign of $\mathrm{Re}(c_2)$ determines the type of bifurcation at $\mathrm{Re}(c_1)=0$: a supercritical bifurcation occurs for $\mathrm{Re}(c_2)>0$, while a subcritical bifurcation occurs for $\mathrm{Re}(c_2)<0$. In the subcritical case, we will need to take into account the higher order terms such as $|W|^4W$ to the CGL equation [21].

We have derived the CGL equation from Eq. (2) in fairly general form. If it is possible to specify the reaction and advection terms based on the precise mechanism in the plasmodium, we can obtain the coefficients of the CGL equation explicitly. In the next section we discuss the advection effect implied by the CGL equation (9) and compare our results with ones derived from reaction-diffusion models without the endoplasmic flow.

IV. DISCUSSION

We have presented the framework of the pattern-formation model for the contraction oscillation in the *Physarum* plasmodium. Considering chemical oscillation to be a clock of the rhythmic contraction, we discussed the transportation of chemicals in the plasmodium. The transportation dynamics is described within the framework that chemical oscillators interact with each other by diffusion and advection couplings. Then we have studied such reaction-diffusion-advection systems to clarify the effect of the endoplasmic flow on contraction pattern formation in the plasmodium. We assumed that the gradient of concentration of endoplasmic chemicals determines the endoplasmic flow. Using the weakly nonlinear analysis, we obtain the following results from Eq. (2): (i) for the Hopf bifurcation, the envelope dynamics is governed by the CGL equation without the flow effect; (ii) for the traveling-wave type bifurcation, the envelope dynamics is governed by the CGL equation and the advection terms affect the nonlinear term of the CGL equation through $k_c^2 M$.

It is well known that the reduction dynamics of the ordinary reaction-diffusion equations is also governed by the CGL equation near the oscillatory bifurcation point [20]. This means that we cannot distinguish the reaction-diffusion system with flow from one without flow by dynamical behavior in the small-amplitude region if system parameters are fixed. Thus, for a weakly nonlinear region, we need precise and careful analysis of the reaction-diffusion-advection systems. The latter result (ii) shows that the nonlinear effect near the bifurcation point stems not only from reaction kinetics but also from the self-consistent flow. This is the point to find out whether the flow affects the weakly nonlinear dynamics of the plasmodial behavior. In the remainder of this section we discuss the effect of the self-consistent flow on physiological behavior.

In general, advection can play an effective role for pattern formation, as the flow of matter often causes instabilities of hydrodynamical systems [22,23]. For example, Rovinsky and Menzinger [24] have shown that a differential flow of chemical species induces instabilities of the homogeneous steady state, and it leads to a traveling-wave pattern without diffusions. In this case, advection terms have a crucial effect on the linear dispersion relation. Contrary to this, the advection studied in the present paper has no effect on the linear stability but modulates the nonlinear dispersion relation. Coefficient c_2 of the nonlinear term in Eq. (9) determines the amplitude of solutions and induces instability and bifurcation of them. As shown in Eq. (9), c_2 depends on the reaction kinetics, diffusion coefficients, and advection coefficients. We remember that the advection quantity M varies according to the thickness of the endoplasm, so it is possible that the *Physarum* plasmodium controls the contraction pattern formation by its thickness.

In the weakly nonlinear region, the self-consistent advection causes the strong dependence of pattern formation on k_c. The critical wave number k_c is regarded as an indicator that an oscillator is in step with its neighboring oscillators, because the phase difference of the metabolic oscillation between neighbors tends to become larger as k_c increases. This point of view implies that phase difference plays an important role for pattern formation such as amoeboid behavior in the *Physarum* plasmodium.

We comment on the reaction-diffusion-advection models presented for the pattern formation of other biological systems. The flow of biological individuals contributes their chemotactic pattern formation in the population systems, such as aggregation of the cellular slime mold *Dictyostelium discoideum* and the motile bacteria *Escherichia coli* [25]. Population density of cells changes as a result of chemotactic motion, and chemotactic substances are produced by each cell. That is, the flux of cellular mass is induced by chemicals. The dynamics of the cell distribution is governed by the diffusion-advection equation. These population models have a framework similar to that of Eq. (2) for contraction pattern formation in the *Physarum* plasmodium. In the chemotaxis models, the gradient of chemicals determines the flux of cellular mass, but the advection term includes a linear part which has the form of cross diffusion. This implies that the weakly nonlinear analysis in the present paper is applicable to these chemotaxis systems, although the flow affects the linear stability of the systems.

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APPENDIX: DERIVATION OF THE ENVELOPE EQUATION IN TWO DIMENSIONS

Before studying the envelope equation in two dimensions, we comment on counterpropagating waves [20]. For the bifurcation of traveling-wave type, counterpropagating waves are possible in one spatial dimension, although we derived the envelope equation for a single traveling wave in Sec. III. The envelope dynamics of the counterpropagating waves is the coupled equations of amplitude W_+ and W_- of a linearized solution

$$W_+ e^{i(k_c x+\omega_c t)}\mathbf{V}_+ + W_- e^{i(k_c x-\omega_c t)}\mathbf{V}_- + \text{c.c.},$$

where $(L_0 - k_c^2 D)\mathbf{V}_\pm = \pm i\omega_c \mathbf{V}_\pm$. The time evolution of $W_\pm$ is governed by the coupled CGL equations with interaction terms $|W_\mp|^2 W_\pm$ [20].

In the case of two spatial dimensions, modes corresponding to an annulus of wave vectors, $|\mathbf{k}| = k_c$, are neutrally stable on the bifurcation point. Thus multimode traveling waves satisfying $|\mathbf{k}_j| = k_c$ of the form

$$\sum_j \{W_{j+}\mathbf{V}_+ \exp[i(\mathbf{k}_j\cdot\mathbf{x}+\omega_c t)] + W_{j-}\mathbf{V}_- \exp[i(\mathbf{k}_j\cdot\mathbf{x}-\omega_c t)]\} + \text{c.c.}$$

are possible [20]. Although we can obtain the multimode envelope equations, it is not clear that their dynamics has structural stability. Such a problem has a relation to pattern selection, and we need more precise analysis of the bifurcation with symmetry. Hereafter we discuss the envelope equation for a single traveling wave of Eq. (2) in two spatial dimensions. We assume that a set of modes near the single plane wave traveling along the x axis, $\exp[i(k_c x-\omega_c t)]$, is dominant across the bifurcation point.

Introducing perturbation expansions and multiple scales,

$$\mathbf{u}\sim\epsilon\mathbf{u}_1+\epsilon^2\mathbf{u}_2+\epsilon^3\mathbf{u}_3+\cdots, \quad L\sim L_0+\epsilon^2 L_2+\cdots, \quad X=x-c_p t, \quad \xi=\epsilon(x-c_g t), \quad \eta=\epsilon y, \quad \tau=\epsilon^2 t, \tag{A1}$$

and substituting Eq. (A1) into Eq. (2), we obtain perturbation equations,

$$\left(L_0+D\frac{\partial^2}{\partial X^2}+c_p\frac{\partial}{\partial X}\right)\mathbf{u}_m=\mathbf{b}_m, \quad m=1,2,3,\ldots. \tag{A2}$$

The inhomogeneous term of the first order equation is vanishing, $\mathbf{b}_1=\mathbf{0}$. For the second and third order equations, the inhomogeneous terms are

$$\mathbf{b}_2=-c_g\frac{\partial\mathbf{u}_1}{\partial\xi}+M\frac{\partial\mathbf{u}_1}{\partial X}\frac{\partial\mathbf{u}_1}{\partial X}-N_2\mathbf{u}_1\mathbf{u}_1-2D\frac{\partial^2\mathbf{u}_1}{\partial X\partial\xi},$$

$$\mathbf{b}_3=\frac{\partial\mathbf{u}_1}{\partial\tau}-c_g\frac{\partial\mathbf{u}_2}{\partial\xi}+M\left(\frac{\partial\mathbf{u}_2}{\partial X}\frac{\partial\mathbf{u}_1}{\partial X}+\frac{\partial\mathbf{u}_1}{\partial X}\frac{\partial\mathbf{u}_2}{\partial X}+\frac{\partial\mathbf{u}_1}{\partial\xi}\frac{\partial\mathbf{u}_1}{\partial X}+\frac{\partial\mathbf{u}_1}{\partial X}\frac{\partial\mathbf{u}_1}{\partial\xi}\right)-L_2\mathbf{u}_1-N_2(\mathbf{u}_2\mathbf{u}_1+\mathbf{u}_1\mathbf{u}_2)-N_3\mathbf{u}_1\mathbf{u}_1\mathbf{u}_1-D\left(2\frac{\partial^2\mathbf{u}_2}{\partial X\partial\xi}+\frac{\partial^2\mathbf{u}_1}{\partial\xi^2}+\frac{\partial^2\mathbf{u}_1}{\partial\eta^2}\right).$$

We write a solution of the first order equations with the slowly varying envelope $W(\xi,\eta,\tau)$,

$$\mathbf{u}_1=W(\xi,\eta,\tau)e^{ik_c X}\mathbf{V}+\text{c.c.}$$

Since the solvability conditions for the second order equation are satisfied by the definition of c_g, we advance our calculation to the third order:

$$(\mathbf{V}^*,\mathbf{b}_3^{(+1)})=(\overline{\mathbf{V}}^*,\mathbf{b}_3^{(-1)})=0,$$

$$\mathbf{b}_3^{(+1)}=\frac{\partial W}{\partial\tau}\mathbf{V}-c_g\frac{\partial\mathbf{u}_2^{(1)}}{\partial\xi}+2k_c^2\overline{W}M(\mathbf{u}_2^{(2)}\overline{\mathbf{V}}+\overline{\mathbf{V}}\mathbf{u}_2^{(2)})-WL_2\mathbf{V} - WN_2(\mathbf{u}_2^{(0)}\mathbf{V}+\mathbf{V}\mathbf{u}_2^{(0)})-\overline{W}N_2(\mathbf{u}_2^{(2)}\overline{\mathbf{V}}+\overline{\mathbf{V}}\mathbf{u}_2^{(2)}) - |W|^2WN_3(\mathbf{V}\mathbf{V}\overline{\mathbf{V}}+\mathbf{V}\overline{\mathbf{V}}\mathbf{V}+\overline{\mathbf{V}}\mathbf{V}\mathbf{V}) - 2ik_cD\frac{\partial\mathbf{u}_2^{(1)}}{\partial\xi}-\left(\frac{\partial^2W}{\partial\xi^2}+\frac{\partial^2W}{\partial\eta^2}\right)D\mathbf{V}, \tag{A3}$$

$$\mathbf{b}_3^{(-1)}=\overline{\mathbf{b}}_3^{(+1)}.$$

From the solvability conditions (A3), we get the envelope dynamics,

$$\frac{\partial W}{\partial\tau}=c_1W-c_2|W|^2W+c_3\frac{\partial^2W}{\partial\xi^2}+c_4\frac{\partial^2W}{\partial\eta^2}, \tag{A4}$$

where coefficients c_1, c_2, c_3 are the same in Eq. (9) and $c_4=-ic_g/2k_c$. The coefficient c_4 implies dispersion waves observed in the nonlinear Schrödinger equation.

[1] N. Kamiya, Protoplasmatol. **8**, 1 (1959).
[2] K. Matsumoto, T. Ueda, and Y. Kobatake, J. Theor. Biol. **122**, 339 (1986).
[3] T. Ueda, in *Oscillations and Morphogenesis*, edited by L. Rensing (Marcel Dekker, New York, 1993), pp. 167–181.
[4] Y. Miyake, S. Tabata, H. Murakami, M. Yano, and H. Shimizu, J. Theor. Biol. **178**, 341 (1996).
[5] T. Nakagaki and T. Ueda, J. Theor. Biol. **179**, 261 (1996).
[6] T. Ueda and K. Gotz von Olenhusen, Exp. Cell Res. **116**, 55 (1978).
[7] W. Naib-Majani, W. Stockem, and K. E. Wohlfarth-Bottermann, Eur. J. Cell Biol. **28**, 103 (1982).
[8] M. Ishigami, Cell Motil. Cytoskeleton **6**, 439 (1986).
[9] A. Grebecki and M. Cieslawska, Protoplasma **97**, 365 (1978).
[10] Z. Baranowski and K-E. W. Bottermann, Eur. J. Cell Biol. **27**, 1 (1982).
[11] Y. Yoshimoto, F. Matsumura, and N. Kamiya, Cell Motil. **1**, 433 (1981); Y. Yoshimoto, T. Sakai, and N. Kamiya, Protoplasma **109**, 159 (1981).
[12] S. Ogihara, Exp. Cell Res. **138**, 377 (1982).
[13] Y. Miyake, Y. Yamaguchi, M. Yano, and H. Shimizu, IEICE Trans. Fundam. Electron. Commun. Comput. Sci. **E76-A**, 780 (1993); K. Takahashi, G. Uchida, Z. Hu, and Y. Tsuchiya, J. Theor. Biol. **184**, 105 (1997); A. Takamatsu, K. Takahashi, M. Nagoa, and Y. Tsuchiya, J. Phys. Soc. Jpn. **66**, 1638 (1997).
[14] Y. Yoshimoto and N. Kamiya, Protoplasma **95**, 111 (1978).
[15] Y. Miyake, M. Yano, and H. Shimizu, Protoplasma **162**, 175 (1991).

[16] G. F. Oster and G. M. Odell, Cell Motil. **4**, 469 (1984).
[17] V. A. Teplov, Yu. M. Romanovsky, and O. A. Latushkin, Bio-Systems **24**, 269 (1991); D. A. Pavlov, Yu. M. Romanovsky, and V. A. Teplov, Biofizika **41,** 146 (1996) [Biophysics **41**, 153 (1996)].
[18] D. A. Smith, Q. J. Mech. Appl. Math. **48**, 39 (1995).
[19] N. Kamiya, R. D. Allen, and R. Zeh, Acta Protozool. **11**, 113 (1972).
[20] Y. Kuramoto, *Chemical Oscillations, Waves, and Turbulence* (Springer-Verlag, Berlin, 1984); A. C. Newell, T. Passot, and J. Lega, Annu. Rev. Fluid Mech. **25**, 399 (1993).
[21] J. T. Stuart and R. C. DiPrima, Proc. R. Soc. London, Ser. A **362**, 27 (1978); H. T. Moon, P. Huerre, and L. G. Redekopp, Phys. Rev. Lett. **49**, 458 (1982); Physica D **7**, 135 (1983); K. Nozaki and N. Bekki, Phys. Rev. Lett. **51**, 2171 (1983); J. Phys. Soc. Jpn. **53**, 1581 (1984); L. R. Keefe, Stud. Appl. Math. **73**, 91 (1983); Phys. Fluids **29**, 3135 (1986); W. van Saarloos and P. C. Hohenberg, Physica D **56**, 303 (1992).
[22] S. Ei and M. Mimura, SIAM (Soc. Ind. Appl. Math.) J. Math. Anal. **21**, 346 (1990).
[23] A. J. Perumpanani, J. A. Sherratt, and P. K. Maini, IMA J. Appl. Math. **55**, 19 (1995).
[24] A. B. Rovinsky and M. Menzinger, Phys. Rev. Lett. **69**, 1193 (1992); **70**, 778 (1993); M. Menzinger and A. B. Rovinsky, Fields Inst. Commun. **5**, 297 (1996).
[25] E. F. Keller and L. A. Segel, J. Theor. Biol. **26**, 399 (1970); **30**, 235 (1971); M. Mimura and T. Tsujikawa, Physica A **230**, 499 (1996); T. Höfer and P. K. Maini, Phys. Rev. E **56**, 2074 (1997).