Internal nonlinear dynamics of a short lattice DNA model in terms of propagating kink-antikink solitons

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(Received 27 October 2011; revised manuscript received 9 March 2012; published 13 April 2012)

We study the internal nonlinear dynamics of an inhomogeneous short lattice DNA model by solving numerically the governing discrete perturbed sine-Gordon equations under the limits of a uniform and a nonuniform angular rotation of bases. The internal dynamics is expressed in terms of open-state configurations represented by kink and antikink solitons with fluctuations. The inhomogeneity in the strands and hydrogen bonds as well as nonuniformity in the rotation of bases introduce fluctuations in the profile of the solitons without affecting their robust nature and the propagation. These fluctuations spread into the tail regions of the soliton in the case of periodic inhomogeneity. However, the localized form of inhomogeneity generates amplified fluctuations in the profile of the soliton. The fluctuations are expected to enhance the denaturation process in the DNA molecule.

DOI: 10.1103/PhysRevE.85.041911 PACS number(s): 87.15.−v, 66.90.+r, 05.45.Yv

I. INTRODUCTION

DNA, an important biopolymer, is not only structurally complex, but also performs important biological functions such as transcription, replication, and denaturation. For the occurrence of transcription, the DNA helical strands should unwind and expose the bases for a chemical reaction, in particular to help RNA polymerase, an enzymatic protein, copy genetic information and transport it for protein synthesis [1,2]. However, unwinding of DNA double helical strands is a complex process, which can happen in more than one way. The RNA polymerase may hit a specific site of the DNA molecule and the molecule may undergo fluctuations that will lead to large conformational changes. Also, intrinsic fluctuations of DNA in the form of vibrational energy itself might be trapped in soliton excitations as a result of a balance between dispersion and nonlinearity, which leads to localized base-pair opening. These intrinsic fluctuations act as a source for DNA denaturation. Hence it is important to probe the role of nonlinearity in DNA internal dynamics, in particular in the context of base-pair opening, first by considering continuum models [3]. The presence of solitons in the DNA chain was at first discussed by Englander *et al*. [4]. Yomosa's dynamic plane-base rotator model [5,6], which involves rotational motion of bases, was refined by Takeno and Homma [7–9] and later extended by Zhang [10] by expressing the interstrand interaction through hydrogen bonds in terms of a double-well potential. The well-known Peyrard-Bishop model describes DNA denaturation through the Morse potential for hydrogen bonds [11–13]. In the model of Christiansen *et al*. [14], transverse and longitudinal displacements of the bases were represented in terms of the Toda potential. An extended Peyrard-Bishop model by including the helicoidal structure of DNA was discussed by Barbi *et al*. [15,16]. Alexandrov *et al*. [17,18] studied the impact of sequence-dependent stacking using Monte Carlo simulation [17] and gene transcription via DNA breathing dynamics through Langevin molecular dynamic simulations [18] by treating an extended Peyrard-Bishop-Dauxois model of DNA. The results describe the melting behavior of homogeneous, periodic sequences and the sequence dependence of DNA dynamic features. In the model of Campa [19], the heterogeneous character of DNA was considered. Krumhansl and Alexander [20] considered an asymmetric potential for their model and studied DNA dynamics. Gaeta [21–23] studied soliton dynamics in DNA via torsional motion. Sataric and Tuszynski [24] examined the impact of a protein interaction on the breather dynamics of DNA by extending the model of Peyrard and Bishop, which is more accurate for the formation of localized oscillations in terms of breathers and bubbles. Also, the effect of single nucleotide polymorphism on DNA breathing dynamics was recently studied by Jablensky *et al*. [25] using Langevin molecular dynamic simulations, which stress the importance of the sequence dependence. In particular, this study is related to promoter polymorphism in two overlapping 6p25 genes linked to schizophrenia. The single DNA molecule experiment is another important tool used to unzip the DNA molecule through microscopic modeling [26] and an angular trap [27]. Since the DNA molecular chain is sequence or site dependent, the strands are flexible and the molecule is helical in shape, recently, Daniel and Vasumathi [28–31] and subsequently Daniel and Vanitha [32] made a detailed analytical study of the base-pair opening in an inhomogeneous continuum DNA chain in terms of perturbed kink-antikink and bubble solitons, respectively. In these studies the opening of base pairs is initiated through breaking of hydrogen bonds due to rotational motion of bases in a plane normal to the helical axis of the molecule. Inspite of these developments in the internal dynamics of the DNA molecular chain in terms of moving soliton, slow moving or static soliton itself was found to explain the open states in DNA [5,33–38] in the form of kinks, antikinks, and breathers as solutions of the static sine-Gordon model. The mechanical denaturation of DNA in terms of base-pair openings was understood by studying the stationary solutions of the static sine-Gordon model in both the continuum and discrete limits based on the Peyrard-Bishop model [33]. The equilibrium states of the discrete Peyrard-Bishop Hamiltonian with fixed boundary conditions was studied by Theodorakopoulos *et al*. [34] and the

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thermodynamic instabilities in DNA unzipping or denaturation is understood. The information about the conformation of open states in the form of static kink-antikink solitons can be found also in the earlier work of Yomosa [5]. In a different context, the double helical chain was modeled in terms of a coupled sine-Gordon equation and the open state was expressed in terms of static kink-antikink solitons [35]. Even simple rigid rod models explain the static geometry of DNA segments [36]. Nevertheless, the dynamical aspects of the open-state configuration and its relevance to biological processes are of great interest [37,38].

In spite of the above developments in the nonlinear internal statics and dynamics of DNA, based on various continuum models, an accurate model, however, should take into account the discreteness effect in the base sequence and in this case the dynamics will be governed by nonlinear differential-difference equations. Solving these equations by analytical means is rather difficult; hence the governing dynamical equations should be solved through numerical integration. In addition, natural sequences of DNA are of finite length and its effect cannot be neglected. DNA segments are biologically important because different regions of the DNA molecule are associated with different functions (e.g., promoter, terminator, and coding). These significant regions or segments of the DNA molecule consist of approximately 10–50 base pairs. Therefore, the study of internal dynamics of short and finite-length DNA molecules with a definite number of base pairs is biologically important. Numerical simulation of the internal dynamics of DNA provides an impulse for further investigation and interesting possibilities have been realized in a series of recent works [15,16,19,39,40]. Techera *et al*. [40] discovered two different dynamical regimes in a simplified DNA dynamical model by solving the governing nonlinear Schrödinger equation both analytically and numerically. In the above studies, large-amplitude localized distortions in finite-length DNA molecular structures were considered. Also the asymmetry of the base pairs was neglected. However, Salerno [41,42], introduced a discrete model for DNA promoter dynamics in T7A1 bacteriophage that takes into account information about a specific base sequence along the double helix with reflexive boundary conditions. This study concluded that the sine-Gordon kinks are set in motion at a certain region of the DNA sequence. Later, the unit-mass potential for kinks, which was initially at rest and moving in a slowly varying background, was derived by Salerno and Kivshar [43]. The effect of inhomogeneity on the dynamics of topological solitons in an inhomogeneous DNA molecule was studied numerically in the work by Kovaleva *et al*. [44]. Dominguez-Adame *et al*. [45] investigated the pinning and propagation of kink solitons along periodic and aperiodic inhomogeneous DNA lattice systems. Lennholm and Hornquist [46] performed a genome study of the conjecture made about the promoters as dynamical active regions and found that the part of the promoters where the RNA transcription has started is more active than a random portion of the DNA molecule. Cuenda and Sanchez [47,48] revisited Salerno's sine-Gordon model and claimed that the motion of kinks originates from the bases at the boundary, which are not part of the genome studied. This study also claims that the dynamics of kinks has no special significance with reference to specific regions of the sequence by disproving the recent work of Lennholm and Hornquist [46] and Bashford [49]. Nonbreathing compactonlike modes are considered as a better candidate of nonlinear models for a locally open state and for feasible attachment of enzymes in the recent work of Takeno [50]. A simple two-dimensional discrete model of DNA was proposed by Muto *et al*. [51,52] to explain the longitudinal propagation of energy in a circular DNA model, in which the hydrogen-bond interaction was represented by the Lennard-Jones potential and the sugar-phosphate bridge was represented by an anharmonic potential. This model predicted a significant increase in the lifetime of the open states of the hydrogen bonds due to the role of anharmonicity in DNA denaturation. A real DNA molecule is substantially an inhomogeneous or site or sequence-dependent dynamical system. Recently, Yakushevich *et al*. [53] studied open states in a discrete inhomogeneous DNA double helix by considering the asymmetric nature of the helix and showed that the movement of the soliton representing the open state in the inhomogeneous DNA molecular chain depends on the sequence of base pairs. The inhomogeneity in DNA in that study was considered in terms of a random base sequence, which is not always the case. The breathing behavior of genomic DNA in terms of a base-pair opening was studied using a Langevin molecular dynamic simulation [54]. The higher content of the A-T rich region is also considered as a more suitable site for the formation of a bubble than a molecule with a random sequence [55]. There are only limited studies of soliton propagation in inhomogeneous media, where localized inhomogeneity has been considered in the form of a potential barrier, a delta potential, and discontinuities [28,32,56]. Very recently, Cadoni *et al*. [57,58] studied the propagation of solitons in a fully inhomogeneous medium and proved the possibility of long-lived soliton excitations in the presence of inhomogeneity in the base masses and interpair interactions. In this context, in a very recent paper the present authors studied the impact of localized and periodic inhomogeneities in both stacking and hydrogen bonds on bubble solitonlike excitations but in the continuum limit [32]. It was found that the inhomogeneity does not affect the robust nature of the solitons but introduces small fluctuations in the tail. Motivated by the above, in the present paper we numerically study localized nonlinear molecular excitations through angular rotation of bases in a discrete short DNA double helical molecule with a finite number of site or sequence-dependent base pairs.

The paper is structured as follows. Section II describes the model and dynamical equations for a discrete lattice DNA with a finite number of base pairs that are site dependent. The results of the numerical study of the internal nonlinear dynamics of the short DNA molecule with inhomogeneity under different limits is presented in Sec. III. The results are summarized in Sec. IV.

II. MODEL AND DYNAMICAL EQUATIONS FOR A DISCRETE DNA MOLECULE

We consider a short segment of the B form of a DNA molecule, consisting of two sequence or site-dependent strands, containing 50 base pairs with interstrand and intrastrand interactions through hydrogen bonds and stacking, respectively. In the DNA molecule the strength of the

interaction between complementary bases that occurs through hydrogen bonds is directly proportional to the distance between them. Let (θ_n,ϕ_n) denote the angles of rotation of bases in the xz and xy planes, respectively, and (θ'_n,ϕ'_n) denote the same in the complementary strand. The helical axis of the DNA molecule is directed parallel to the z axis. The distance D^2 between the complementary bases can be written using elementary geometry as [32]

$$D^2 = 2 + 4r^2 + 2\left[S_n^x S_n^{'x} + S_n^y S_n^{'y} - S_n^z S_{'n}^z\right] - 4r\left[S_n^x + S_n^{'x}\right], \tag{1}$$

where $S_n^x = \sin\theta_n\cos\phi_n$, $S_n^y = \sin\theta_n\sin\phi_n$, $S_n^z = \cos\theta_n$, $S_n^{'x} = \sin\theta'_n\cos\phi'_n$, $S_n^{'y} = \sin\theta'_n\sin\phi'_n$, and $S_n^{'z} = \cos\theta'_n$.

Interestingly, in the model proposed here we invoke the analogy of the discrete DNA molecular system with that of an antiferromagnetically coupled anisotropic ferromagnetic spin lattice system (or spin ladder) with a finite number of spin moments. The edges of the coupled spin system are fixed in terms of periodic-type boundaries as in the DNA molecule. The z direction, which is parallel to the helical axis of DNA, is the easy axis of magnetization in the spin system. The spin-spin exchange interaction is restricted to the nearest neighbors, similar to the stacking between adjacent bases in DNA. The above identification leads us to consider the Heisenberg model of the Hamiltonian for an anisotropic site-dependent antiferromagnetically coupled ferromagnetic spin lattice system as the basis for writing the free energy for the DNA molecular system, as given by [32]

$$\begin{aligned} H = \sum_n \{ & -f_n\left[J\left(S_n^x S_{n+1}^x + S_n^y S_{n+1}^y\right) + J_z S_n^z S_{n+1}^z\right. \\ & \left. + J'\left(S_n^{'x} S_{n+1}^{'x} + S_n^{'y} S_{n+1}^{'y}\right) + J'_z S_n^{'z} S_{n+1}^{'z}\right] \\ & + g_n\left[J_c\left(S_n^x S_{n+1}^x + S_n^y S_{n+1}^y\right) + J'_c S_n^z S_n^{'z}\right] \\ & + A\left(S_n^z\right)^2 + A'\left(S_n^{'z}\right)^2\}. \end{aligned} \tag{2}$$

In the Hamiltonian (2) J and J' represent ferromagnetic exchange integrals due to a nearest-neighbor spin-spin interaction in the two lattices, which correspond to the intrastrand interaction constants of the stacking between the nth base and its nearest neighbors in the plane normal to the helical axis. When J_z and J'_z are not equal to J and J', respectively, anisotropy is introduced in the intrastrand interaction in the lattices. Here $f_n(f'_n)$ introduces site-dependent character, which indicates that the intrastrand stacking energy between the bases varies in a specified site-dependent fashion, leading to sequence-dependent character or inhomogeneity in stacking. The term g_n represents a site- or sequence-dependent character in hydrogen bonds. It is assumed that the inhomogeneities in both strands are similar and equal by choosing $f_n = f'_n$. In a DNA molecule, inhomogeneity may arise for several reasons. The presence of different sites along the strands, such as promoter, terminator, and coding, each of which has a very specific sequence of bases in a particular fashion, makes the strands site dependent or inhomogeneous, which makes them soft [59]. In addition, defects caused by the presence of additional molecules in specific sites of the sequence or the presence of abasic sitelike nonpolar imitation of thymine leads to inhomogeneity [60,61]. In contrast, periodic inhomogeneity may arise due to periodic repetition of different sites or simple defects occurring repeatedly along the strands. The terms proportional to J_c and J'_c in the Hamiltonian correspond to the antiferromagnetic spin-spin coupling between the two lattices, which denote the interstrand interaction or hydrogen bonds in the DNA molecule. In the Hamiltonian (2), the terms proportional to A and A' correspond to anisotropy in the lattices that assume only positive values, leading to rotation of the bases restricted to the plane normal to the helical axis of the DNA molecule. We now express the Hamiltonian (2) in terms of the rotational angles ϕ_n and ϕ'_n under the plane-base rotator model ($\theta_n = \theta'_n = \frac{\pi}{2}$) [5,32]. In DNA, the two strands are expected to exhibit similar types of macroscopic behavior [62]; hence we assume $J = J'$ and $J_z = J'_z$. When the anisotropy energies A and A' are much larger than the interstrand and intrastrand interactions, then $\frac{\partial\phi_n}{\partial t} = 2A\cos\theta_n$, $\frac{\partial\phi'_n}{\partial t} = 2A'\cos\theta'_n$, and the Hamiltonian (2) becomes

$$\begin{aligned} H = \sum_n \Bigg\{ & \frac{1}{4A}\left[\left(\frac{\partial\phi_n}{\partial t}\right)^2 + \left(\frac{\partial\phi'_n}{\partial t}\right)^2\right] \\ & - Jf_n[2 - \cos(\phi_{n+1} - \phi_n) - \cos(\phi'_{n+1} - \phi'_n)] \\ & - J_c g_n[1 - \cos(\phi_n - \phi'_n)]\Bigg\}. \end{aligned} \tag{3}$$

In the above Hamiltonian, the moments of inertia $I = \frac{1}{2A}$ and $I' = \frac{1}{2A'}$ and we limit our discussion to $I = I'$ since the physical properties of the two strands are similar. The first two terms in the Hamiltonian (2) represent the kinetic energies of the rotational motion of the nth nucleotide bases accompanied by the potential energy associated with the nth nucleotide, sugar and phosphate and their complementary units.

Having constructed the Hamiltonian for the site-dependent DNA molecule, the corresponding dynamical equation can be obtained by deriving the associated Hamiltonian equations of motion after suitable rescaling of time in the form

$$\begin{aligned} \frac{\partial^2\phi_n}{\partial t^2} = {} & J(1 + \lambda_1 f_n)\sin(\phi_{n+1} - \phi_n) \\ & - J(1 + \lambda_1 f_{n-1})\sin(\phi_n - \phi_{n-1}) \\ & + J_c(1 + \lambda_1 g_n)\sin(\phi_n - \phi'_n), \end{aligned} \tag{4a}$$

$$\begin{aligned} \frac{\partial^2\phi'_n}{\partial t^2} = {} & J(1 + \lambda_1 f_n)\sin(\phi'_{n+1} - \phi'_n) \\ & - J(1 + \lambda_1 f_{n-1})\sin(\phi'_n - \phi'_{n-1}) \\ & + J_c(1 + \lambda_1 g_n)\sin(\phi'_n - \phi_n). \end{aligned} \tag{4b}$$

As the site-dependent character or inhomogeneity along and between the DNA strands, f_n and g_n, respectively, are small, while writing Eqs. (4) we have assumed $f_n = (1 + \lambda_1 f_n)$ and $g_n = (1 + \lambda_1 g_n)$, where λ_1 is a small constant. Equations (4a) and (4b) describe the dynamics of inhomogeneous bases in DNA at the discrete level, when rotational motion of bases in a plane normal to the helical axis is considered. The problem thus reduces to solving the discrete dynamical equations (4a) and (4b) to explain the impact of inhomogeneity on the internal DNA lattice dynamics expressed in terms of soliton modes. A few methods such as the hyperbolic function method [63], the hyperbolic tangent method [64], and the exponential function method [65] are available to solve integrable differential-difference equations, which will give traveling-wave,

solitary-wave, and soliton solutions [66,67]. However, the coupled equations (4a) and (4b) are in general nonintegrable and therefore we attempt to solve them numerically under different limiting cases in the following section.

III. NONLINEAR DYNAMICS OF SHORT LATTICE DNA IN TERMS OF PERTURBED KINK-ANTIKINK SOLITONS

A fourth-order Runge-Kutta method as implemented by MATLAB, with a variable time step, is used to solve the initial value problem found in Eqs. (4a) and (4b) with the periodic boundary conditions $\phi_{n+N} = \phi_n$ and $\phi'_{n+N} = \phi'_n$, where N represents the total number of base pairs chosen for the study here under different limiting cases [68]. This method requires only the initial data and the data at the preceding time step apart from the parameters involved. In this numerical scheme, the spatially discretized ordinary differential equation is passed directly to the MATLAB ordinary differential equation solver ode45. Numerical calculations are performed for a lattice of the DNA molecule composed of 50 base pairs, in which the strands S and S' are composed of even and odd sites, respectively. The parameters are chosen as $J = 0.5$, $J_c = -0.54$, and $\lambda_1 = 0.1$, which stem from the values $J = 0.005$ eV, $J_c = -0.0054$ eV, and 1 time unit equal to 10^{-11} s. We consider the opening of base pairs through breaking of hydrogen bonds due to the rotational motion of the bases. The fluctuation will start from a specific site and the DNA base pairs will open around that point. At the start of the numerical run, the center of the base-pair opening is assigned to some position and then the model is integrated for various time values. During the integration, the fluctuation will start to move from the initial state and emerges as large-amplitude fluctuations as time progresses. In the following we solve Eqs. (4a) and (4b) by numerically integrating the same using the fourth-order Runge-Kutta procedure under various limits that explain different physical situations and obtain the numerical solutions.

A. Homogeneous DNA molecule under uniform, small angular rotation of bases

First we consider a homogeneous DNA molecule by assuming $\lambda_1 = 0$ in Eqs. (4a) and (4b). It is also assumed that, in the B form of the DNA double helix, the difference in the angular rotation of bases with respect to neighboring bases along the strands is small [7,8], so that $\sin(\phi_{n+1} - \phi_n) \approx (\phi_{n+1} - \phi_n)$, $\sin(\phi_n - \phi_{n-1}) \approx (\phi_n - \phi_{n-1})$, and similarly for the complementary strand. The resultant equations read

$$\frac{\partial^2 \phi_n}{\partial t^2} = J(\phi_{n+1} - 2\phi_n + \phi_{n-1}) + J_c \sin(\phi_n - \phi'_n), \tag{5a}$$

$$\frac{\partial^2 \phi'_n}{\partial t^2} = J(\phi'_{n+1} - 2\phi'_n + \phi'_{n-1}) + J_c \sin(\phi'_n - \phi_n). \tag{5b}$$

Equations (5a) and (5b) describe the dynamics of DNA in a plane-base rotator model for the discrete model while considering the dominant angular rotation of bases in a plane normal to the helical axis and ignoring all other small motions of the bases. On adding Eqs. (5a) and (5b), the resultant equation satisfies identically. By subtracting Eq. (5b) from Eq. (5a), we obtain

$$\frac{\partial^2}{\partial t^2}(\phi_n - \phi'_n) = J[(\phi_{n+1} + \phi_{n-1} - 2\phi_n) - (\phi'_{n+1} + \phi'_{n-1} - 2\phi'_n)]. \tag{6}$$

As the two DNA strands are asymmetric in nature, when an open-state configuration is formed in DNA, the two complementary bases may rotate in opposite directions [62] so that $\phi'_n = -\phi_n$. Further, by assuming $\Psi_n = 2\phi_n$, Eq. (6) becomes

$$\frac{\partial^2 \Psi_n}{\partial t^2} = J(\Psi_{n+1} + \Psi_{n-1} - 2\Psi_n) + J_c \sin\Psi_n. \tag{7}$$

Equation (7) is identified as the spatially discretized version of the completely integrable sine-Gordon equation, which admits N-soliton solutions in the form of kinks and antikinks [69]. The term Ψ_n corresponds to the rotational angle of the bases in a plane normal to the helical axis of DNA. The term proportional to J_c represents the hydrogen-bond interaction between bases in DNA. The dynamical study of a similar model was proposed by Salerno [41] to describe the discrete dynamics of a DNA promoter corresponding to the T7A1-promoter base sequence. Equation (7) is numerically integrated for the initial solution

$$\Psi_0 = 4 \arctan \frac{\exp[(n - n_0)a - ct_0]}{\sqrt{(1 - c^2)}}$$

and by choosing the coefficients as $J = 0.5$ and $J_c = -0.54$. The numerical solutions obtained are plotted in Fig. 1. The plots in Figs. 1(a) and 1(b) are similar to the kink and antikink one-soliton solutions of the continuum sine-Gordon equation obtained by solving the equation analytically [69]. The kink and antikink solitons in the figures represent an open-state configuration initiated by the angular rotation of bases and the internal nonlinear dynamics of the DNA molecule. In Fig. 1(c) a sketch of the open-state configuration in terms of the combination of kink and antikink solitons of the sine-Gordon equation (7) is given. The base pairs are found to open locally in the form of a kink-antikink soliton and the soliton propagates along the DNA lattice parallel to the helical axis. In an open-state configuration of similar kind, it was found that at least 10 base pairs will participate [55].

B. Inhomogeneous DNA molecule under uniform, small angular rotation of bases

Next we consider an inhomogeneous DNA molecule ($\lambda_1 \neq 0$) whose dynamics is governed by uniform small angular rotation of bases by assuming $\sin(\phi_{n+1} - \phi_n) \approx (\phi_{n+1} - \phi_n)$, $\sin(\phi'_n - \phi'_{n-1}) \approx (\phi'_n - \phi'_{n-1})$, $\phi'_n = -\phi_n$, and $\Psi_n = 2\phi_n$ in Eqs. (4a) and (4b) and follow the same steps of the procedure as in the previous homogeneous case. The resultant equation reads

$$\frac{\partial^2 \Psi_n}{\partial t^2} - J(\Psi_{n+1} + \Psi_{n-1} - 2\Psi_n) - J_c \sin\Psi_n = \lambda_1 [f_n(\Psi_{n+1} - \Psi_n) + f_{n-1}(\Psi_n - \Psi_{n-1}) + g_n \sin\Psi_n]. \tag{8}$$

Equation (8) is a perturbed discrete sine-Gordon equation, in which terms proportional to λ_1 can be treated as a

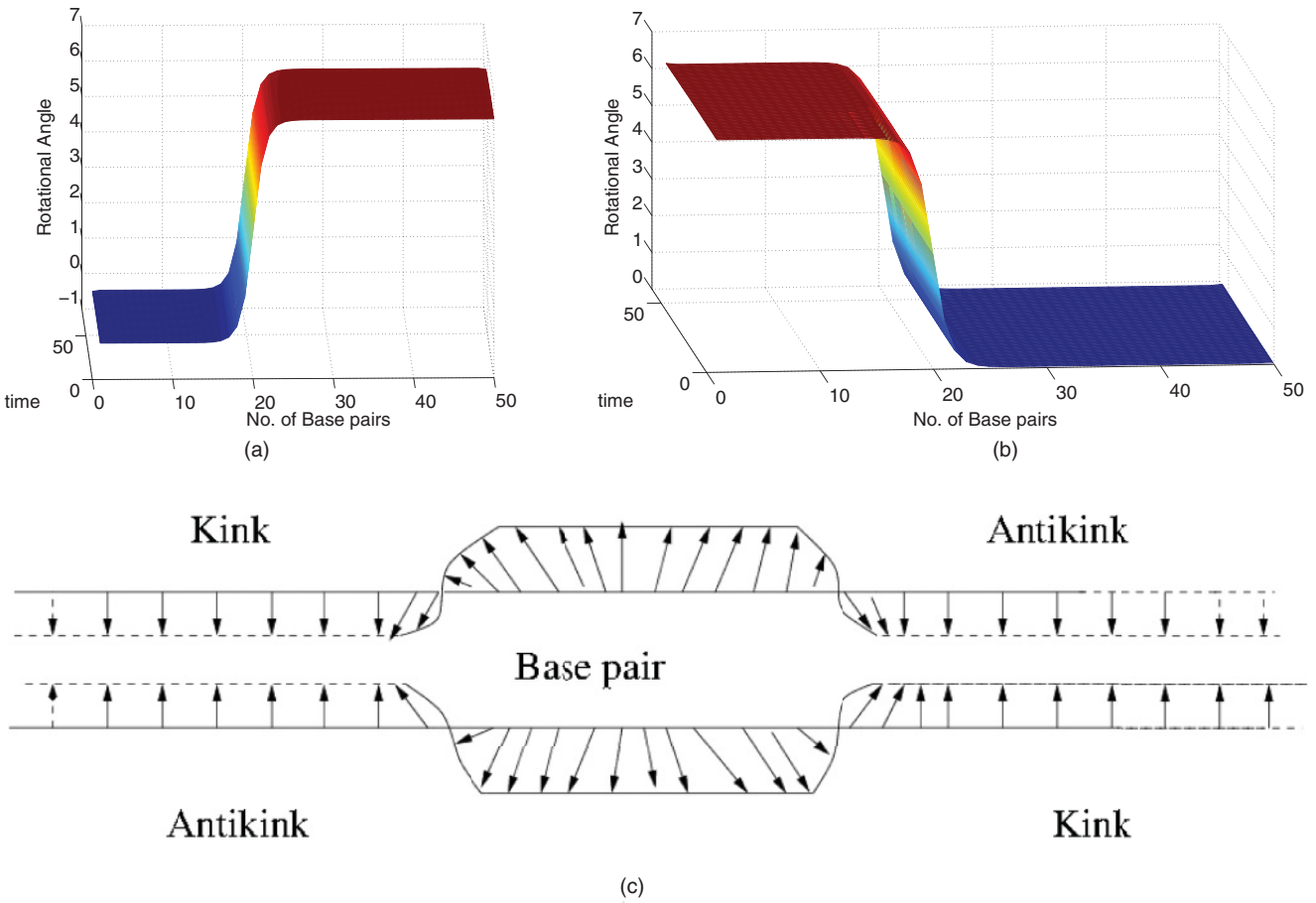

FIG. 1. (Color online) (a) Kink and (b) antikink one-soliton solution obtained by solving Eq. (7) numerically for the parameter values $J = 0.5$ and $J_c = -0.54$. (c) Sketch of the kink-antikink soliton representing the open-state configuration in the homogeneous DNA molecule.

perturbation. Thus, when the DNA strands are sequence dependent, the dynamics is governed by an inhomogeneous discrete perturbed sine-Gordon equation. The effect of inhomogeneity in stacking (f_n and f_{n-1}) as well as in hydrogen bonds (g_n) on soliton excitations is understood by numerically integrating Eq. (8) for the same initial condition

$$\Psi_0 = 4 \arctan \frac{\exp[(n - n_0)a - ct_0]}{\sqrt{(1 - c^2)}}$$

and for the same set of parameter values $J = 0.5$ and $J_c = -0.54$ and by choosing $\lambda_1 = 0.1$. The study of soliton propagation through inhomogeneous media including the kink-impurity interaction and its scattering in the sine-Gordon model has recently received a great deal of interest [44,56–58,70,71]. The trinucleotide repeat sequence amplification causes formation of bubbles that are stable. The results of the above study concluded that the expansion of repeats leads to synchronized DNA breathing behavior, which will trigger simultaneous opening of base pairs. The presence of trinucleotide repeat sequences shifts the base pairs, which will form unstable structures. Here we study the effect of two different forms of inhomogeneities, namely, localized and periodic, by choosing them as hyperbolic secant and cosine functions, respectively.

1. Localized inhomogeneity

To understand the effect of localized inhomogeneity on the soliton excitations in the DNA molecule, we substitute $f_n = A \operatorname{sech} na$ and $g_n = B \operatorname{sech} na$, where A and B are constant amplitudes and $n = 1,2, \ldots,$ in Eq. (8) and numerically integrate the resultant equation for the same initial condition and by choosing the constants as $A = B = 0.9$ and $a = 1.0$. The numerical solutions of Eq. (8) in the form of perturbed kink and antikink solitons are plotted in Figs. 2(a) and 2(b), respectively. From the figures it can be observed that fluctuations appear in both the width and the tails of the kink and antikink solitons. This radiation may be stressed out into the lattice of the DNA strands. It is worth comparing the above results with the recent analytical results on the base-pair opening in an inhomogeneous continuum DNA molecule obtained by solving the governing perturbed continuum sine-Gordon equation analytically using soliton perturbation theory by one of the present authors [29]. Unlike the present case, in the continuum limit, fluctuations were found to occur only in the localized region of the solitons without affecting the tail portion. However, spatial discretization of the model introduces additional small fluctuations in the tail regions of the kink and antikink solitons, which in any case do not affect the robust nature of the kink-antikink soliton and hence the open-state configuration and base-pair opening. In a different context, in

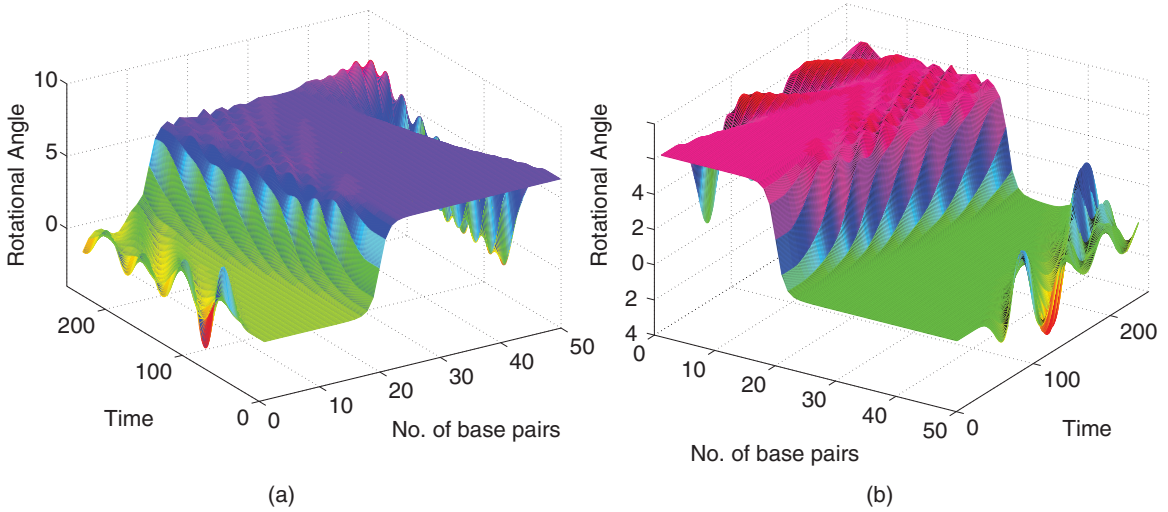

FIG. 2. (Color online) Perturbed (a) kink and (b) antikink one-soliton solution of Eq. (8) in the case of the localized inhomogeneity $f_n = A \operatorname{sech} na$ and $g_n = B \operatorname{sech} na$ with $J = 0.5$, $J_c = -0.54$, and $A = B = 0.9$. The localized inhomogeneity in the stacking and hydrogen bonds introduces small fluctuations in the width and tail regions of the kink and antikink solitons without affecting the robust nature.

the case of an XY spin chain, the model of which identifies with our plane-base rotator model of DNA, the ansatz $\operatorname{sech} na$ energetically favors the deformation of the spin chain [72].

2. *Periodic inhomogeneity*

The periodic inhomogeneity that occurs in the DNA molecule is chosen in the form $f_n = C \cos na$ and $g_n = D \cos na$, $n = 1,2,\ldots,$ where C and D are constants. We substitute the above forms of inhomogeneity in Eq. (8) and numerically integrate the resultant equation for the same set of parameter values and the initial condition as chosen in the case of localized inhomogeneity and by choosing $C = D = 5.0$ and $a = 1.0$. The numerical results of the perturbed kink-antikink solitons are then plotted in Figs. 3(a) and 3(b). Periodic inhomogeneity introduces periodic deformation in the localized region and adds small fluctuations in the tails of the kink and antikink solitons. However, now the periodic fluctuations in the tails get enhanced due to contribution from the spatial discretization of the governing dynamical equation. The periodic inhomogeneity is also responsible for broadening the width of the soliton. In a recent paper Alexandrov *et al.* [54] made a similar study to understand the effect of periodic inhomogeneity through tandem trinucleotide repeat sequences in DNA using a Langevin molecular dynamic simulation and a Markov chain Monte Carlo simulation, which also led to coherent DNA openings and local base-pair breathing dynamics. In addition, this repeated sequence in the DNA has implications for interpretation of genomic data in health and disease.

C. Homogeneous DNA molecule with uniform, large angular rotation of bases

We now consider a homogeneous ($\lambda_1 = 0$) DNA molecule showing uniform ($\phi_n' = -\phi_n$), large angular rotation of bases.

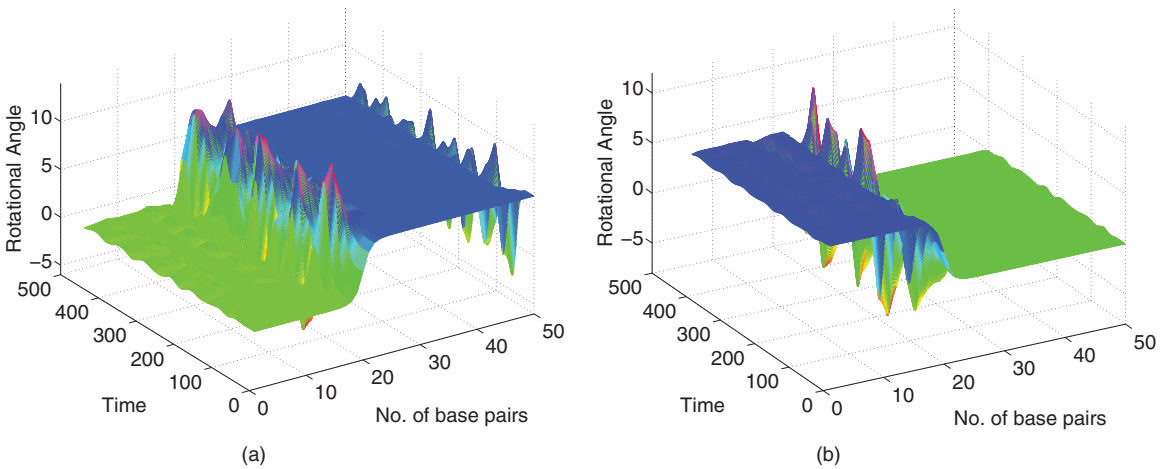

FIG. 3. (Color online) Perturbed (a) kink and (b) antikink one soliton solution of Eq. (8) in the case of periodic inhomogeneity in the form $f_n = C \cos na$ and $g_n = D \cos na$ with $J = 0.5$, $J_c = -0.54$, and $C = D = 5.0$. The periodic inhomogeneity causes periodic deformation in the localized region and adds small fluctuations in the tail regions of the kink and antikink solitons without affecting the robust nature of the soliton. The enhanced periodic fluctuations in the plots also get a contribution from the spatial discretization of the dynamical equation.

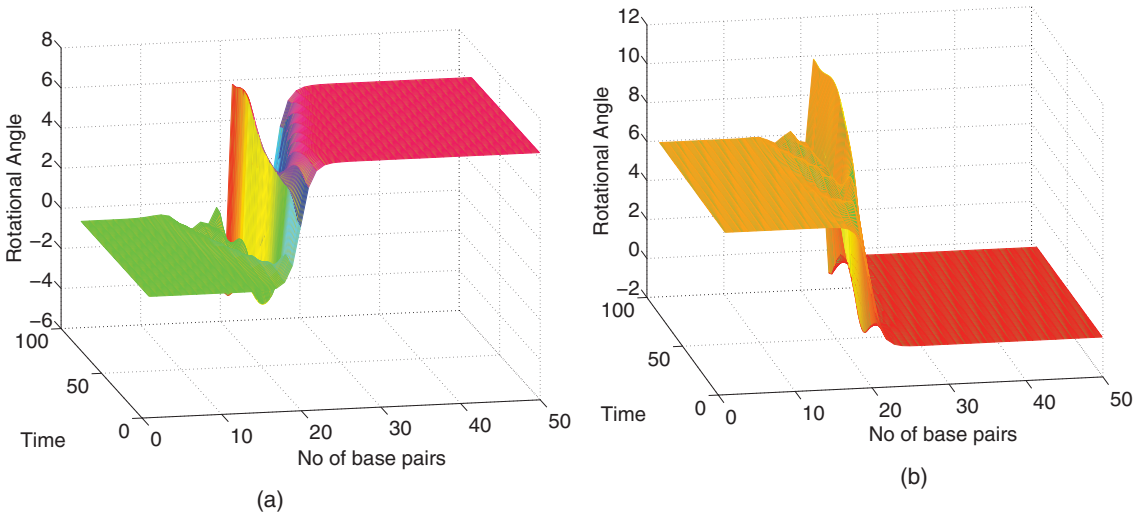

FIG. 4. (Color online) Perturbed (a) kink and (b) antikink soliton solutions of Eq. (9), in a homogeneous DNA molecule with uniform large angular rotation of bases. The parameters are chosen as $J = 0.5$ and $J_c = -0.54$. Motion of the DNA bases interms of large angular rotation of bases in the plane normal to the helical axis introduces large scale fluctuations in the profile of the solitons leaving the tail regions unaffected. The fluctuations resemble the shape of the inhomogeneity.

In this case Eqs. (4a) and (4b) become

$$\frac{\partial^2 \phi_n}{\partial t^2} = J[\sin(\phi_{n+1} - \phi_n) - \sin(\phi_n - \phi_{n-1})] + J_c \sin 2\phi_n. \tag{9}$$

Equation (9) is numerically integrated for the same set of parameters and the initial condition and the results are plotted in Figs. 4(a) and 4(b). In this case large-amplitude sharp localized fluctuations are observed strictly within the profile of the kink and antikink solitons. However, the tail region of the solitons is completely intact and free from any fluctuation. The large-scale fluctuations that appear in the profile of the kink and antikink solitons are due to a large angular rotation of the bases. A comparison of the results of the present case with those of the previous inhomogeneous cases leads to the conclusion that inhomogeneity in the DNA molecule introduces additional fluctuations in the tail regions of the kink and antikink solitons. However, since the large-scale fluctuation occurs within a short period of time, the small fluctuation that may occur in the tail region of the soliton due to spatial discretization of the dynamical equation over an extended period of time could not be observed within this short period.

D. Inhomogeneous DNA molecule with uniform, large angular rotation of bases

In the case of an inhomogeneous DNA molecule ($\lambda_1 \neq 0$) exhibiting uniform ($\phi_n' = -\phi_n$), large angular rotation of bases, the dynamical equations (4a) and (4b) are written as

$$\frac{\partial^2 \phi_n}{\partial t^2} = J[(1 + \lambda_1 f_n)\sin(\phi_{n+1} - \phi_n) + (1 + \lambda_1 f_{n-1})\sin(\phi_n - \phi_{n-1})] + J_c \sin 2\phi_n. \tag{10}$$

Equation (10) is numerically integrated in the case of localized and periodic inhomogeneities separately.

1. Localized inhomogeneity

As before, we substitute the localized inhomogeneities $f_n = A \operatorname{sech} na$ and $g_n = B \operatorname{sech} na$ in Eq. (10) and numerically integrate the resultant equation for the same initial condition and by choosing the parameters as $J = 0.5$ and $J_c = -0.54$ as before and setting $\lambda_1 = 0.1$, $A = B = 0.9$, and $a = 1.0$. The numerical solutions thus obtained are plotted in Figs. 5(a) and 5(b). In this case also, large-scale narrow and sharp pulses occur in the profile of the soliton keeping the tail regions intact. The inhomogeneity favors the growth of large-amplitude excitation and resembles the shape of the inhomogeneity $\operatorname{sech} na$. On comparing the plots in Figs. 4 and 5, it is noted that the sharp amplitude fluctuations generated in the profile region of the solitons due to large angular rotation of bases is significantly enhanced by the localized inhomogeneity. Once again the small fluctuations that will arise due to spatial discretization could not be observed in the short span of time. It is clear from the figures that the formation of large-amplitude excitation due to localized inhomogeneity reduces the width of the soliton slightly.

2. Periodic inhomogeneity

The periodic inhomogeneity that exists in stacking and hydrogen bonds is chosen in the form $f_n = C \cos na$ and $g_n = D \cos na$, where C and D are constant amplitudes. The above forms of inhomogeneities are substituted in Eq. (10) and the resultant equation is numerically integrated. The numerical results are plotted in Figs. 6(a) and 6(b). As found in the figures, periodic inhomogeneities in stacking and hydrogen bonds introduce periodic fluctuations in the profile and in the tail regions of the kink and antikink solitons over a long period of time. The periodic fluctuations that occur in the solitons in this case exhibit a high-density profile over a long period. This is because the source for the generation of periodic fluctuation is due to the periodic inhomogeneity inserted as well as the spatial discretization of the governing dynamical equation. It

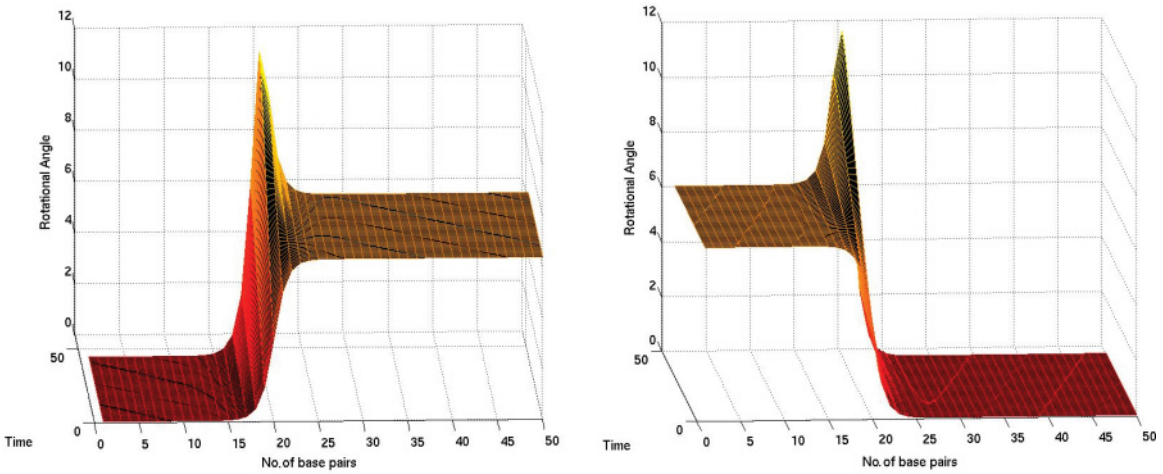

FIG. 5. (Color online) Perturbed (a) kink and (b) antikink soliton solutions of Eq. (10) in the case of an inhomogeneous DNA molecule with localized inhomogeneity in the form $f_n = A\,\mathrm{sech}\,na$ and $g_n = B\,\mathrm{sech}\,na$ with $J = 0.5, J_c = -0.54$ and $A = B = 0.9$. The open state configuration is initiated by uniform ($\phi'_n = -\phi_n$) large angular rotation of bases. The localized inhomogeneity in stacking and hydrogen bonds brings in large scale, narrow and sharp pulses in the profile of the kink and antikink solitons keeping the tail regions intact.

should be noted that the soliton is widened due to periodic inhomogeneity as found earlier.

E. Homogeneous DNA molecule with nonuniform, large angular rotation of bases

Next we consider the internal nonlinear dynamics of a homogeneous ($\lambda_1 = 0$) DNA molecule with nonuniform ($\phi'_n \neq -\phi_n$), large angular rotation of bases. In this case, Eqs. (4a) and (4b) reduce to the set of equations

$$\frac{\partial^2 \phi_n}{\partial t^2} = J\sin(\phi_{n+1} - \phi_n) - J\sin(\phi_n - \phi_{n-1}) + J_c \sin(\phi_n - \phi'_n), \tag{11a}$$

$$\frac{\partial^2 \phi'_n}{\partial t^2} = J\sin(\phi'_{n+1} - \phi'_n) - J\sin(\phi'_n - \phi'_{n-1}) + J_c \sin(\phi'_n - \phi_n). \tag{11b}$$

The numerical solutions of Eqs. (11) for the same initial condition and for the same set of parameter values used in the previous cases are plotted in Figs. 7(a) and 7(b). From the figures it is observed that small-amplitude localized but periodic oscillations occur within the profile region of the kink and antikink solitons leaving the tail regions intact. The occurrence of periodic oscillations in the profile region of the soliton is mainly due to the spatial discretization associated with the nonlinearity of the governing dynamical equation, namely, $\sin(\phi_{n+1} - \phi_n)$ and $\sin(\phi_n - \phi_{n-1})$.

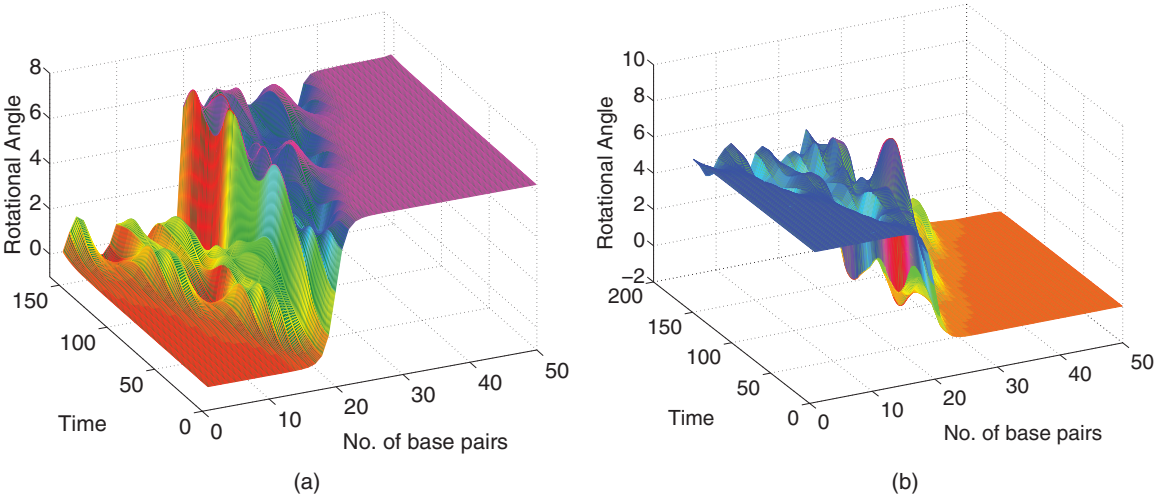

FIG. 6. (Color online) Perturbed (a) kink and (b) antikink soliton solutions of Eq. (10) in a periodic inhomogeneous DNA molecule with the inhomogeneities in the form $f_n = C\cos na$ and $g_n = D\cos na$ and the parameters are chosen as $J = 0.5$, $J_c = -0.54$, and $C = D = 0.9$. The periodic inhomogeneities in stacking and hydrogen bonds add highly dense periodic fluctuations in the entire stretch of the solitons including the width and tail regions.

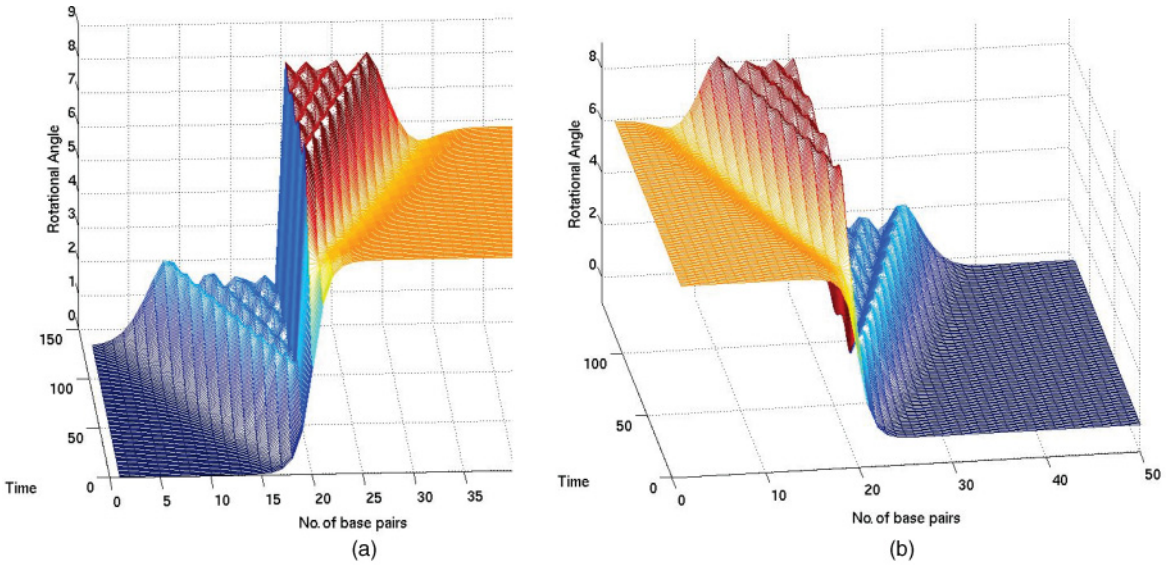

FIG. 7. (Color online) Perturbed (a) kink and (b) antikink soliton solutions of Eqs. (11a) and (11b) in a homogeneous DNA molecule with nonuniform large angular rotation of bases when $J = 0.5$, $J_c = -0.54$. Small amplitude localized but periodic oscillations occur only in the profile region of the solitons with the tail regions intact.

F. Inhomogeneous DNA molecule with nonuniform, large angular rotation of bases

This is the most general case and the internal nonlinear dynamics in this case is governed by the set of discrete coupled equations (4a) and (4b). These equations are numerically integrated for the same initial condition and the same set of parameter values used earlier. To realize the impact of inhomogeneity on the solitons, as in the earlier cases, we consider both localized and periodic forms of inhomogeneities.

1. Localized inhomogeneity

The numerical solutions of Eqs. (4a) and (4b) plotted in Figs. 8(a) and 8(b) correspond to localized inhomogeneities in the form $f_n = A\,\mathrm{sech}\,na$ and $g_n = B\,\mathrm{sech}\,na$. In this case periodic but localized fluctuations appear strictly within the profile region of the soliton in a profound manner. However, this radiation does not spread into the tail region of the solitons. The localization of the periodic fluctuation is because of the localized form of the inhomogeneity.

2. Periodic inhomogeneity

The numerical solution of Eqs. (4a) and (4b) corresponding to the periodic form of inhomogeneities $f_n = C\cos na$ and $g_n = D\cos na$ is plotted in Figs. 9(a) and 9(b). The plots show that periodic inhomogeneity introduces periodic fluctuations in the width and in the tail regions of the kink and antikink solitons. The profound nature of fluctuations in the plots is because of the contributions from the periodic inhomogeneity and the nonlinear spatial discretization. On comparing the above results with that of the case where uniform, large angular

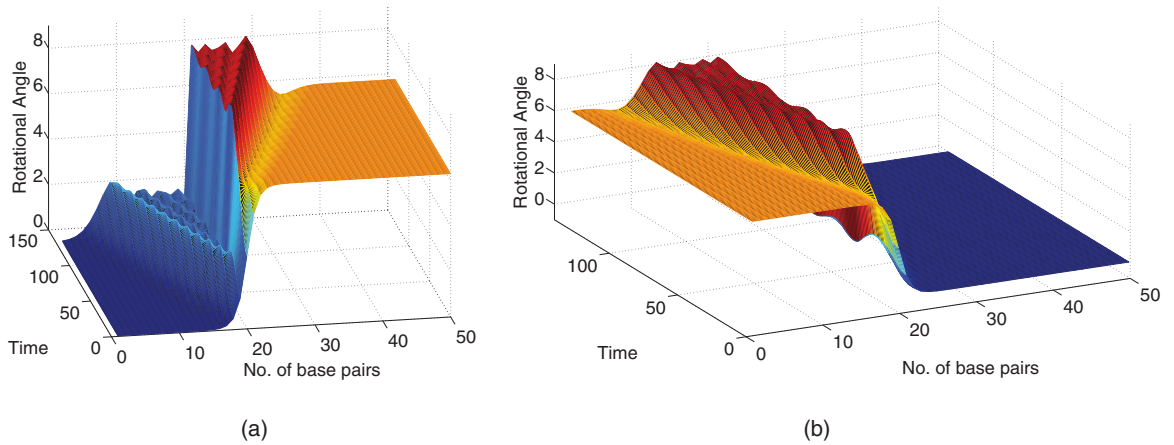

FIG. 8. (Color online) Perturbed (a) kink and (b) antikink soliton solutions of Eqs. (4a) and (4b), in the case of an inhomogeneous DNA molecule whose internal dynamics is governed by nonuniform large angular rotation of bases, with the localized inhomogeneities in the form $f_n = A\,\mathrm{sech}\,na$, $g_n = A\,\mathrm{sech}\,na$ and $J = 0.5$, $J_c = -0.54$, $A = B = 0.9$. Localized and large scale periodic fluctuations are observed within the width of the kink and antikink solitons alone.

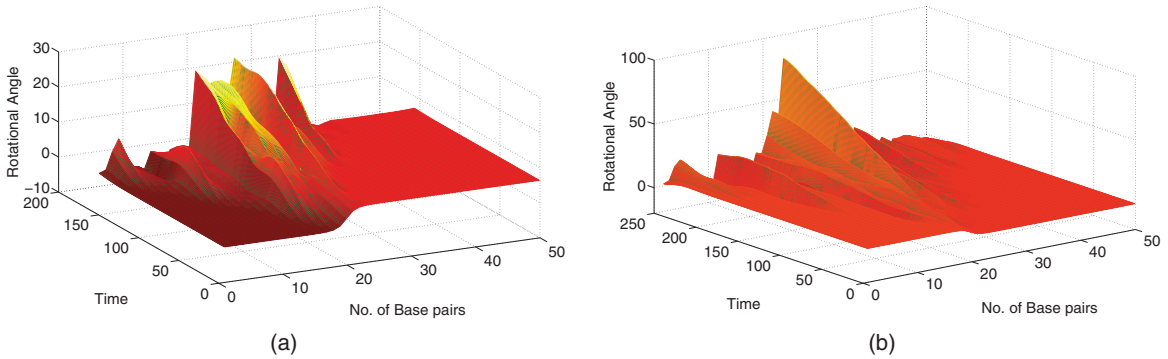

FIG. 9. (Color online) Perturbed (a) kink and (b) antikink soliton solutions of Eqs. (4a) and (4b) in an inhomogeneous DNA molecule with periodic inhomogeneities in the form $f_n = C\cos na$ and $g_n = D\cos na$ for the parametric choices $J = 0.5, J_c = -0.54$, and $C = D = 5.0$ under nonuniform large angular rotation of bases. Periodic fluctuations are observed in the profile and in the tail regions of the kink and antikink solitons.

rotation of bases is considered [see Figs. 6(a) and 6(b)], it is found that nonuniform rotation of bases supresses the periodic character of the fluctuation and hence less pronounced.

IV. CONCLUSION

In this paper the open-state configuration in an inhomogeneous short DNA lattice model is investigated by solving the governing discrete nonlinear dynamical equations numerically. The model considered here to study the internal dynamics is conceived and adapted from two antiferromagnetically coupled short site-dependent ferromagnetic lattices. The Hamilton's equations of motion are constructed for the angular rotation of bases in a plane normal to the helical axis for both the strands. The internal dynamics of homogeneous DNA is then studied by considering uniform and nonuniform small as well as large angular rotation of bases. Two different types of inhomogeneities in terms of localized and periodic functions are considered and their impact on the dynamics, and coherent rotation of bases under different limits has been studied. In the case of homogeneous DNA molecule, under uniform, small angular rotation of bases, the dynamics is governed by the discrete sine-Gordon equation. When the angular rotation of bases is large and nonuniform, the governing dynamical equations become perturbed discrete sine-Gordon equations under different limits. The above equations were integrated numerically, with periodic boundary conditions. The base pair opening or open state configuration under different limits is represented in the form of kink-antikink solitons and their perturbations [55]. Summarized in Table I are the results of the nature of open state configurations in terms of kink-antikink solitons and their perturbations in the form of fluctuations in homogeneous and inhomogeneous (localized and periodic) DNA molecules under uniform and nonuniform as well as small and large angular rotation of bases. From the results it is observed that, in the case of homogeneous DNA molecules, large amplitude angular rotation of bases introduces fluctuations only in the width of the solitons leaving behind the tail regions intact. In inhomogeneous DNA molecules with localized inhomogeneity, when the amplitude of the angular rotation of bases is large, sharp and localized fluctuations are generated within the profile of the kink-antikink solitons which

TABLE I. Nature of open states in terms of kink-antikink solitons in homogeneous and inhomogeneous DNA molecules under uniform/nonuniform and small/large angular rotation of bases.

	Nature of open states in terms of kink-antikink solitons		
Nature of DNA	Uniform small angular rotation of bases	Uniform large angular rotation of bases	Nonuniform, large angular rotation of bases
Homogeneous	Kink-antikink solitons of the discrete sine-Gordon equation. Figs. 1(a) and 1(b)	Large scale fluctuations in the profile of the solitons leaving the tail regions unaffected. Figs. 4(a) and 4(b)	Small amplitude periodic oscillations only in the profile region of the solitons. Figs. 7(a) and 7(b)
Localized inhomogeneous	Small fluctuations in the width and tail regions of the solitons. Figs. 2(a) and 2(b)	Large scale, narrow sharp pulse in the profile of the solitons solitons. Figs. 5(a) and 5(b)	Localized and large scale periodic fluctuations in the width of the kink and antikink solitons. Figs. 8(a) and 8(b)
Periodic inhomogeneous	Periodic deformation in the localized region and small fluctuations in the tails of the solitons. Figs. 3(a) and 3(b)	High density periodic fluctuations in the entire stretch of the solitons including the width and tail regions. Figs. 6(a) and 6(b)	Periodic fluctuations in the profile and in the tail regions of the kink and antikink solitons. Figs. 9(a) and 9(b)

do not spread into the tail regions. However, when the angular rotation of bases is small, the inhomogeneity dominates and the fluctuations spread into the tail regions of the solitons as well. On the otherhand, when inhomogeneity exists in the periodic form, which extends over the entire length of the molecule, fluctuations appear both in the width and in the tail regions of the solitons. It is also noted that, when the amplitude of the angular rotation of bases is large, eventhough uniform, the small fluctuations that develop in the profile of the solitons enlarge and sharpen. Further, nonuniform angular rotation of bases in the DNA molecule generates periodic oscillations in the profile region of the kink and antikink solitons. However, the nonuniformity supresses the periodic fluctuations to some extent. It is concluded from our results that, inhomogeneity as well as nonuniform and large angular rotation of bases, introduce only small fluctuations in the open state configuration represented by kink-antikink solitons without affecting the robust nature and propagation of the solitons. We believe that the fluctuations formed in the regions of the DNA molecule will enhance the denaturation process in the DNA molecule. From our results it is found that the localized form of inhomogeneity gives rise to large, stable and local DNA opening modes. The presence of periodic fluctuations in the width and tail regions of the soliton is due to the spatial discretization and periodic inhomogeneity which can be due to the periodic repetition of base pairs in the DNA molecule. On comparing our results with the recent results of Alexandrov *et al*. [54], related to DNA dynamics with repeat sequence, it may be concluded that the perturbation due to inhomogeneity will not affect the DNA breathing dynamics. However, the periodic perturbation, which is made equivalent to the repeat sequence may be responsible for the interpretation of genomic data in health and disease [54]. Also, it has been found that the large number of repeats may sometimes lead to the formation of non B-DNA structure conformations which will influence several diseases in humans [73]. Further, it was recently shown that the composite models of DNA support solitonic excitations and are able to travel long distances along the DNA chain with real inhomogeneities [58]. In a similar direction, it was earlier found by Dominguez-Adame *et al*. [45] that solitons in periodic lattice DNA can propagate always which is also in confirmation with our results that the robust nature of the soliton is not affected by the inhomogeneities. As the soliton propagates along an inhomogeneous DNA molecule, the emission of phonons may promote penetration of the soliton into the inhomogeneous region [44].

How, the fluctuations developed in the profile region of the solitons spread into the tail regions and stressed out into the lattice generating large amplitude phonons is an important question to be pursued, and the study is underway, the results of which will be published elsewhere.

ACKNOWLEDGMENTS

M.D. acknowledges support from funding for a Department of Science and Technology project. M.V. expresses her sincere thanks to Dr. R. Adhikari for his advice in the numerical work. M.V. also acknowledges the financial support received in the form of visiting student fellowship from the Institute of Mathematical Sciences, Chennai and the Research Fellowship in Science for Meritorious Students from the University Grants Commission.

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