

**Bubble solitons in an inhomogeneous, helical DNA molecular chain with flexible strands**

M. Daniel\* and M. Vanitha

*Centre for Nonlinear Dynamics, School of Physics, Bharathidasan University, Tiruchirapalli 620 024, India*

(Received 10 May 2011; revised manuscript received 13 July 2011; published 27 September 2011)

Base pair opening in an inhomogeneous, DNA double helical molecular chain with flexible strands is investigated by studying its internal dynamics. For the study, a generalized model which takes into account the energies involved in stacking and hydrogen bonds along with inhomogeneity, helicity, and phonons coupled to the stacking and hydrogen bonds is proposed. The internal dynamics of the proposed DNA model is governed by a perturbed nonlinear Schrödinger equation. The unperturbed, completely integrable nonlinear Schrödinger equation which admits soliton solutions and forming a bubble corresponds to DNA dynamics with homogeneous and rigid strands. The results of the soliton perturbation analysis show that the inhomogeneity in stacking and hydrogen bonds in localized and periodic forms and the helicity do not alter the amplitude under perturbation. However, the flexibility of the strands diminishes the perturbed amplitude. On the other hand, the velocity of the soliton and bubble are unaltered due to all the above effects. However, the position and phase of the soliton and the bubble vary linearly in time. While the position of the soliton depends on the initial velocity, the phase depends on both the initial velocity and the initial amplitude of the soliton. The above effects introduce small fluctuation in the tail of the soliton, without affecting the robust nature of the soliton and the bubble during propagation. The soliton and the bubble obtained as solutions of the internal dynamics of the DNA molecule represent an opening of the base pairs which is essential for the transcription process.

DOI: [10.1103/PhysRevE.84.031928](https://doi.org/10.1103/PhysRevE.84.031928)

PACS number(s): 87.15.-v, 66.90.+r

**I. INTRODUCTION**

DNA is a dynamical complex molecular system with fairly ordered structure and its fluctuation is essential for important functions such as transcription and replication [1], because the genetic information is encoded in the bases which are buried inside the structure, in addition to denaturation. In order to read the genetic code, DNA must be locally opened by structural distortion or by breaking the hydrogen bonds between the bases. It can be caused either by an enzymatic protein molecule which makes large conformational changes when it attacks specific site of DNA or by undergoing large amplitude fluctuations, and this leads to the study of its internal dynamics. This large amplitude motion unwinds the helical strands, allows the base pairs to rotate, and leads to nonlinear molecular excitation, which has become an emerging field of nonlinear DNA dynamics. The nonlinearity in DNA [2] leads to the possibility of coherent, localized excitations in the form of kink-antikink solitons [3,4], breathers [5–7], bubbles [8], charge transport in terms of polarons and bubbles [9], and compactons [10]. Among the different experiments available to explain the internal dynamics of the DNA molecule, Raman scattering [11] interprets the small vibrational modes, hydrogen-deuterium exchange experiments [12] explain the opening of base pairs, microwave absorption experiments [13–15] provide proof for the existence of longitudinal acoustic modes, and fluorescence depolarization experiments [16,17] describe the torsional constants of DNA. A few more attempts were also made to study the internal dynamics of DNA, in terms of solitons through scattering of neutrons and light [18–20]. Single DNA molecule experiments constitute an important tool to unzip the DNA molecule through microscopic modeling [21,22]. The above experimental studies are clear evidence for the

importance of nonlinearity in DNA dynamics and, hence, it needs an understanding on the theoretical front as well. On the theoretical front, the internal dynamics and the nature of nonlinear molecular excitations in the DNA molecule are studied by proposing different models. After the pioneering work of Englander *et al.* [23], who introduced the concept of solitons in DNA dynamics by treating the molecule as chains of pendula, which imitate the local opening of base pairs during motion. Yomosa [24,25] proposed a dynamic plane base-rotator model, by including the rotational motion of bases leading to double well potential. Yomosa's model was further refined by Takeno and Homma [26,27], in which attention was paid to the degrees of freedom. Peyrard and Bishop [28] studied the DNA denaturation process through Morse potential and this model concentrates on the stretching dynamics of DNA with one degree of freedom for each base pair. Using the Toda lattice model, Christiansen and his co-workers [29] explained DNA denaturation by including the transverse and longitudinal motions of the bases in addition to the torsional degree of freedom. The underlying nonlinear excitations in this case appear in the form of breathers. Campa [8] and Barbi [30] also considered the interaction between the complementary bases in the form of Morse potential. The model of Krumhansl and Alexander [31] describes the hydrogen bond interaction by asymmetric double well potential. Gaeta and his co-workers [32–34] made contributions to the study of soliton dynamics in DNA via torsion dynamics. Sataric *et al.* [35,36] examined the impact of protein interaction on the breather dynamics in DNA by extending the model of Peyrard-Bishop, which is more accurate for the formation of localized oscillations in terms of breathers and bubbles. The dynamical equations corresponding to the above models eventually lead to nonlinear evolution equations in the integrable family which were solved analytically [36–38] and numerically [39–42] for kink-antikink, pulse, breather, bubble, and compactonlike modes, which essentially represent the pattern of base pair

\*daniel@cnld.bdu.ac.in

opening in the DNA molecule. In the above models, the DNA double helical molecular chain is considered as consisting of two homogeneous rigid linear chains. However, in nature, the constitution of the DNA molecular chain is sequence-dependent (inhomogeneous), the strands are flexible, and the molecule is helical in shape [43–47]. Recently, Daniel and Vasumathi [43] studied the base pair opening in the DNA molecule by treating it as equivalent to a coupled ferromagnetic lattice or spin ladder. The helicity was taken into account by different authors through torsional coupling [30–32,35], and helical transformation [10] and the dynamics were expressed in the form of kinks and breathers, respectively. Daniel and Vasumathi [45] introduced helicity through twist deformation, in analogy with a cholesteric liquid crystal system and a helimagnet [48–50] and the DNA dynamics is expressed in the form of a kink-antikink soliton. However, helicity was found to increase the width of the soliton [45] and more base pairs participated in the opening process. The inhomogeneous character of the DNA molecular chain introduces small fluctuations in the width of the soliton without affecting the localized pattern of it. Further, flexibility of the DNA strands generate phonons which accelerate the solitons, leaving small fluctuations in the localized region of the solitons without affecting the width and the base pair opening [46].

The number of base pairs that participate in the opening process depends on the nature of the soliton. For instance, due to its broad nature, in a kink-antikink soliton, more base pairs will participate in the opening process. However, in biological processes, it is advantageous to have base pair opening with participation by fewer bases. Therefore, it is preferred to have pulselike nontopological soliton representing bubblelike opening involving few base pairs [39–42]. Recently, there are quite a few studies related to bubble dynamics in DNA in different contexts. Altan-Bonnet *et al.* [51], explained the denaturing process of the helical strands in DNA in terms of bubblelike solitons. From a statistical mechanics point of view, Hanke *et al.* [52,53] studied single DNA denaturation, using the Poland-Scheraga model [54], by putting forwarding the fact that the denatured bubbles open up due to thermal activation. Wu Lian-Ao *et al.* [55] described the bubble dynamics by mapping the Fokker-Planck equation that explains DNA dynamics to a time-dependent Schrödinger equation. The stability of the helix was analyzed through bubble solitons by Villagran *et al.* [56]. Hennig [57] proposed an oscillator network model, which appears like the double helical structure of DNA and obtained a solution in the form of radial breathers and kink shaped patterns resembling an oscillating bubble. When nonelastic effects are included, the amplitude of the soliton grows in time and causes fluctuation. Transfer-matrix studies show the formation of bubbles in flexible DNA loops [58,59]. Vasumathi and Daniel [47], while investigating the dynamics of a protein-DNA complex in a viscous medium, obtained bubbles traveling along the helical chain. Thus, it has become important and necessary to investigate the internal dynamics of DNA in terms of soliton bubbles by proposing a generalized model involving inhomogeneity, flexibility, and helicity of the molecule. Therefore, in the present paper, we propose a generalized model for DNA dynamics by considering the molecule as consisting of two helical, flexible, interacting, site-dependent molecular chains in analogy with the Heisenberg model for a

coupled anisotropic site-dependent spin system and investigate the underlying nonlinear excitations in the form of solitonlike bubbles by solving the governing dynamical equation.

The paper is organized as follows. Section II presents a generalized model for DNA dynamics by including the site-dependent sequence of bases and the helicity and the flexibility of the strands. In Sec. III, the DNA internal dynamics is expressed in terms of a perturbed nonlinear Schrödinger equation. Section IV outlines the soliton perturbation theory to solve the perturbed nonlinear Schrödinger equation. Sections V, VI, and VII are devoted to the results of the perturbation study on the effect of inhomogeneity in base sequences and hydrogen bonds and the helicity and flexibility in DNA strands on soliton excitations, respectively. The results are summarized and concluded in Sec. VIII. The evaluation of several integrals that appear while evaluating the parameters of the soliton and the perturbed part of the soliton solution using residue theorem is given in the Appendix.

## II. A GENERALIZED MODEL FOR DNA INTERNAL DYNAMICS

The models considered earlier by different authors for the study of DNA dynamics treat the molecule as consisting of two homogeneous, rigid, linear chains. Many of the authors, further assumed specific potentials such as Morse, Toda, double well, plane base rotator, etc., to represent the hydrogen bonding between complementary bases. Few authors modeled the system by taking into account the translational, rotational, and torsional motion of bases to represent the energy associated with the hydrogen bonds. Here we consider the DNA molecule as consisting of two sequence- or site-dependent (inhomogeneous), flexible, helical strands or chains with interstrand and intrastrand interactions through hydrogen bonds and stacking, respectively. Interestingly, in the model proposed here, we invoke the analogy of inhomogeneous DNA molecular chain with that of a coupled anisotropic ferromagnetic spin chain and will construct the associated Heisenberg model of the Hamiltonian. The exact form of B-DNA with its helical axis along the  $z$  direction is schematically represented in Fig. 1(a). Each base is depicted by an arrow and the conjugated arrow represents the complementary base. The dots between the arrows in the figure represent the hydrogen bonds between complementary bases. The site-dependent (inhomogeneous) character of hydrogen bonds and base stacking is expressed by the difference in the length of the arrows and the variation in the space between the neighboring arrows. The projection of the  $n$ th base pair in the  $XY$  plane is shown in Fig. 1(b). In the figure,  $B$  and  $B'$  represent the tips of the  $n$ th bases, which belong to the complementary strands  $S$  and  $S'$ , respectively. It is limited in our model here that the two complementary bases rotate in opposite directions. The radius of the circle in the figure is represented as “ $r$ ”. The angles of rotation of bases around the points  $P$  and  $P'$  are represented by  $(\theta_n, \phi_n)$  and  $(\theta'_n, \phi'_n)$  in the  $XZ$  and  $XY$  planes, respectively. The coordinates of  $P$  and  $P'$  are  $(r \cos n\phi_0, r \sin n\phi_0, z_n)$  and  $[r \cos(n\phi_0 + \pi), r \sin(n\phi_0 + \pi), z'_n]$  respectively, where,  $\phi_0 = \frac{2\pi}{p}$  with  $p$ , the number of base pairs per turn in the strands  $S$  and  $S'$ . The rotational motion of bases will cause fluctuation and breaking of hydrogen bonds in the DNA molecule which

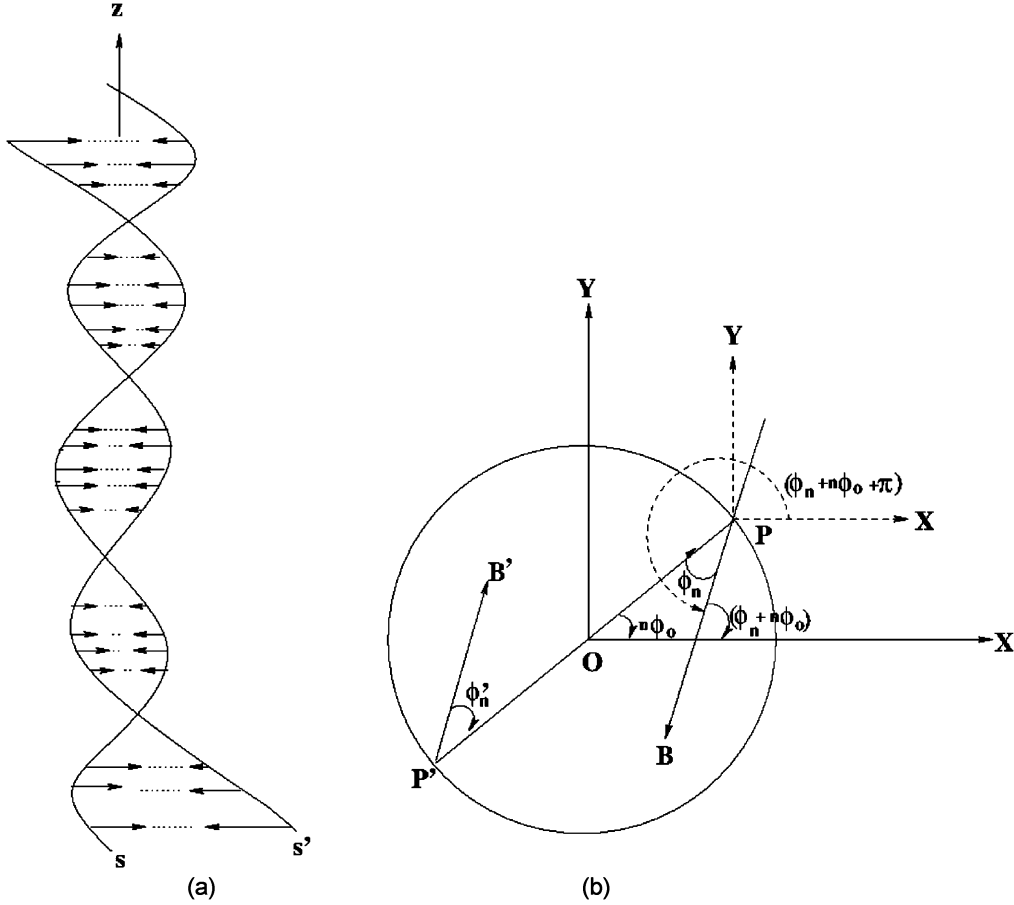


FIG. 1. (a) A schematic representation of the structure of a B-form DNA double helix. (b) A horizontal projection of the  $n$ th base pair in the  $xy$  plane.

leads to opening of base pairs, or in other words, unwinding of the double helical strands. The conformation and stability of the DNA double helix is mainly determined by the stacking energy, in addition to the hydrogen bonds and other forms of energies.

The base-base interaction or the strength of the hydrogen bonds between complementary bases depends on the distance between the complementary bases. In Fig. 1(b), the square of the distance,  $D_n^2$ , between the edges of the arrows  $B$  and  $B'$  can be calculated using the geometry as

$$D_n^2 = 2 + 4r^2 + (z_n - z'_n)^2 + 2(z_n - z'_n)(\cos \theta_n - \cos \theta'_n) + 2[\sin \theta_n \sin \theta'_n \cos(\phi_n - \phi'_n) - \cos \theta_n \cos \theta'_n] - 4r[\sin \theta_n \cos \phi_n + \sin \theta'_n \cos \phi'_n]. \quad (1)$$

The role of hydrogen bonds and stacking in the dynamics can be understood in a better way, when the distance between the bases can be expressed in terms of quasispin operators,

$$\begin{aligned} S_n^x &= \sin \theta_n \cos \phi_n, & S_n^y &= \sin \theta_n \sin \phi_n, & S_n^z &= \cos \theta_n, \\ S_n'^x &= \sin \theta'_n \cos \phi'_n, & S_n'^y &= \sin \theta'_n \sin \phi'_n, & S_n'^z &= \cos \theta'_n, \end{aligned} \quad (2a)$$

$$(2b)$$

for the base at the  $n$ th site in the  $S$  and  $S'$  strands. Further, it is easy to generalize the model in terms of spin operators, when various other interactions are considered in the dynamics.

Using the quasispin operator representation given in Eqs. (2), Eq. (1), after assuming  $z_n = z'_n$ , can be written as

$$D_n^2 = 2 + 4r^2 + 2[S_n^x S_n'^x + S_n^y S_n'^y - S_n^z S_n'^z] - 4r[S_n^x + S_n'^x]. \quad (3)$$

The form of  $D_n^2$  given in Eq. (3) is similar to the Hamiltonian for a Heisenberg spin model. Therefore, the intrastrand base-base interaction or stacking and other useful forces and interactions that stabilize the double helical form of DNA can also be written using the same consideration. In order to use such a quasispin model for the DNA problem, it is reasonable to think that the flexible double strand helix with site-dependent bases can be conceived as an anisotropic twisted and coupled site-dependent spin lattice model or coupled anisotropic helimagnetic spin system. By mapping two such coupled helical spin systems with the two DNA double helices, Daniel and Beula [48] studied the dynamics of DNA by introducing the helical structure through twist deformation in analogy with the structure of a helimagnet [49] (see Fig. 2). and cholesteric liquid crystal system [50]. Under this mapping, the two strands of the DNA molecule correspond to two site-dependent anisotropic, ferromagnetic twisted spin lattices and are antiferromagnetically coupled to each other via the spins or magnetic moments. The direction of the helical axis, that is, the  $z$  direction, is chosen as the easy

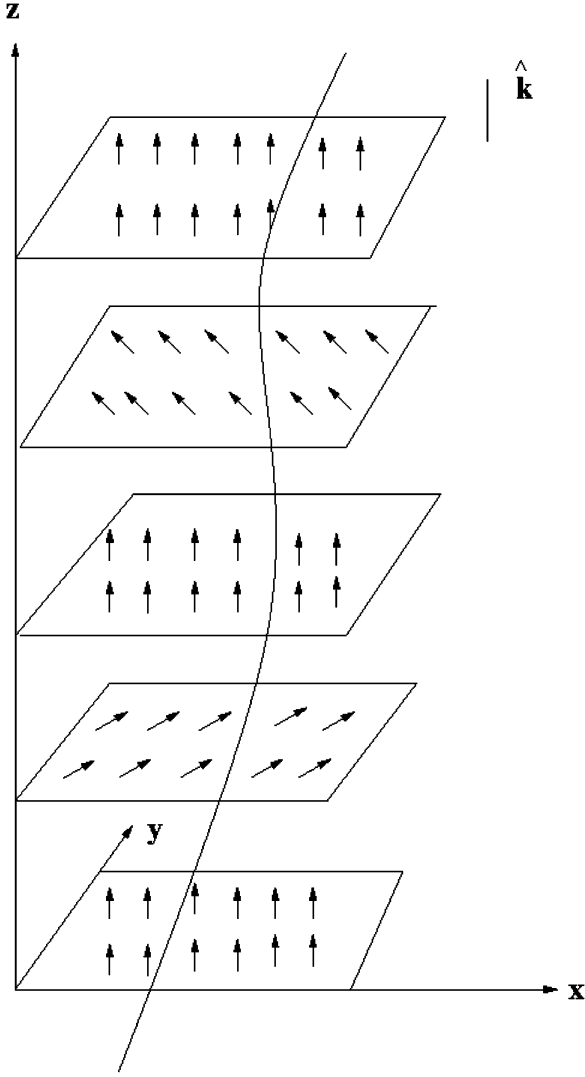


FIG. 2. A schematic representation of a helimagnet.

axis of magnetization of the coupled spin system. Similar to the stacking between adjacent bases in DNA, here the spin-spin exchange interaction is restricted to the nearest neighbors, with the  $n$ th spin interacting with the  $(n + 1)$ th and  $(n - 1)$ th spins. With a view to consider a generalized DNA double stranded helical model, we consider inhomogeneity in stacking and hydrogen bonds and flexibility in the helical strands.

#### A. Hamiltonian of the model

With the above identification, we consider the Heisenberg model of Hamiltonian for an anisotropic, site-dependent, twisted, and antiferromagnetically coupled ferromagnetic spin lattice system or for a suitable site-dependent anisotropic coupled helimagnetic spin system. The total Hamiltonian which consists of the above components is written as [48]

$$H = H_{\text{ex}} + H_{\text{ani}} + H_{\text{hy}} + H_{\text{hel}} + H_{\text{ph}}. \quad (4)$$

The site-dependent exchange Hamiltonian  $H_{\text{ex}}$  (which represents stacking energy in DNA) for the two spin lattices

corresponding to the two strands  $S$  and  $S'$  is written as

$$H_{\text{ex}} = - \sum_n \{ f_n [J_{xy} (S_n^x S_{n+1}^x + S_n^y S_{n+1}^y) + J_z S_n^z S_{n+1}^z] + f'_n [J'_{xy} (S_n^x S_{n+1}^x + S_n^y S_{n+1}^y) + J'_z S_n^z S_{n+1}^z] \}. \quad (5)$$

In the above Hamiltonian,  $J_{xy}$  and  $J'_{xy}$  represent ferromagnetic exchange integrals due to nearest-neighboring spin-spin interaction in the two lattices in the  $xy$  plane, which correspond to the intrastrand interaction constant or the stacking energy between the  $n$ th base and its nearest neighbors in the plane normal to the helical axis in the strands  $S$  and  $S'$ , respectively. When  $J_z$  and  $J'_z$  are not equal to  $J_{xy}$  and  $J'_{xy}$ , respectively, anisotropy is introduced in the intrastrand interaction in the lattices. In Hamiltonian (5),  $f_n$  and  $f'_n$  introduce site-dependent character in the exchange interaction of the spin lattices, which further indicate that the intrastrand stacking energy between bases in the  $S$  and  $S'$  strands varies in a specified site-dependent fashion, leading to sequence-dependent character or inhomogeneity in DNA. In general,  $f_n$  and  $f'_n$  may take different mathematical forms for different types of inhomogeneities. In DNA, inhomogeneity may arise due to more than one reason. The presence of different sites along the strands such as promotor, terminator, coding, etc., each of which has a very specific sequence of bases in a particular function, makes the strands site dependent or inhomogeneous, making them soft [60]. Also, defects caused due to the presence of additional molecules such as drugs in specific sites of the sequence and the presence of abasic sitelike nonpolar mimic of thymine leads to inhomogeneity [61,62]. On the other hand, periodic inhomogeneity may arise due to periodic repetition of different sites or simple defects along the strands.

The Hamiltonian for the magnetocrystalline anisotropy due to crystal field effect in the ferromagnetic spin system is written as

$$H_{\text{ani}} = \sum_n [A(S_n^z)^2 + A'(S_n^z)^2], \quad (6)$$

where  $A$  and  $A'$  are the uniaxial anisotropic coefficients assuming positive values, leading to rotation of bases in a plane normal to the helical axis of the DNA. The Hamiltonian corresponding to the antiferromagnetic spin-spin coupling between the two lattices analogous to interstrand interaction or hydrogen bonds is written as

$$H_{\text{hy}} = \sum_n [J_c g_n (S_n^x S_n^x + S_n^y S_n^y) + J'_c S_n^z S_n^z], \quad (7)$$

where  $J'_c$  and  $J_c$ , which represent the antiferromagnetic exchange interaction between the two spin lattices, correspond to a measure of the interstrand interaction or energy of the hydrogen bonds between the complementary bases of similar sites in both the strands, along the direction of the helical axis ( $z$  direction) and in a plane normal to it, respectively.  $g_n$  represents the inhomogeneity in the interaction between lattices or in the strength of the hydrogen bonds.

In a helimagnet, the helicity is incorporated by using the discrete form of the free energy corresponding to the twist deformation in a cholesteric liquid crystal system. Thus, the Heisenberg model of the Hamiltonian corresponding to the

free energy for the twist, which is responsible for helicity in terms of spin vectors is written as [48]

$$H_{\text{hel}} = \sum_n h \{ [\mathbf{k} \cdot (\mathbf{S}_n \times \mathbf{S}_{n+1})] + q_0 \}^2 + h' \{ [\mathbf{k} \cdot (\mathbf{S}'_n \times \mathbf{S}'_{n+1})] + q_0 \}^2, \quad (8)$$

where,  $\mathbf{S}_n = (S_n^x, S_n^y, S_n^z)$  and  $\mathbf{k} = (0, 0, 1)$ . In Hamiltonian (8),  $q_0 = \frac{2\pi}{q}$  is the pitch wave vector and  $q$  is the pitch of the helix.  $h$  and  $h'$  denote the coefficients associated with the twist deformation in the two lattices which correspond to the two strands of the DNA molecule.

In nature, the spin lattices and also the strands of DNA are not rigid, but flexible, and hence the two strands deform elastically and the resultant phonons couple to the stacking (exchange interaction) and hydrogen bonds (coupling between lattices). Hence, the part of the Hamiltonian corresponding to the phonon energy and the energy due to its coupling is written as

$$H_{\text{ph}} = \frac{p_n^2}{2M} + \frac{p'_n{}^2}{2M} + K(y_{n+1} - y_n)^2 + K'(y'_{n+1} - y'_n)^2 - \hat{\alpha}(y_{n+1} - y_n)(S_n^x S_{n+1}^x + S_n^y S_{n+1}^y)$$

$$- \hat{\alpha}'(y'_{n+1} - y'_n)(S_n^x S_{n+1}^x + S_n^y S_{n+1}^y) + \hat{\beta}(y_{n+1} - y_{n-1})(S_n^x S_n^x + S_n^y S_n^y). \quad (9)$$

In the above Hamiltonian,  $y_n$  and  $y'_n$  represent the longitudinal displacements of the  $n$ th spin/nucleotide from the equilibrium position in the two lattices/strands,  $p_n$  and  $p'_n$  are the associated momenta, and  $M$  is the uniform mass.  $K$  and  $K'$  are the longitudinal elastic constants along the lattices/strands. While  $\hat{\alpha}$  and  $\hat{\alpha}'$  are the coefficients related to the exchange/intrastrand interaction in the two lattices/strands,  $\hat{\beta}$  is a measure of the coupling between the phonons and the hydrogen bonds or interlattice/interstrand interaction. In DNA, the bases are broad molecules lying almost normal to the helical axis. Therefore, for future convenience, while writing Hamiltonian (9), we considered transverse coupling alone, that is, coupling in the  $xy$  plane normal to the helical axis. Now the total Hamiltonian can be obtained by using Hamiltonians (5)–(9) in Eq. (4). The resultant Hamiltonian (4) after using Hamiltonians (5)–(9) in terms of the variables  $(\theta_n, \phi_n)$  and  $(\theta'_n, \phi'_n)$  using Eqs. (2a) and (2b) is written as

$$H = \sum_n \left[ - \{ J_{xy} f_n + \hat{\alpha}(y_{n+1} - y_n) \} \sin \theta_n \sin \theta_{n+1} \cos(\phi_{n+1} - \phi_n) - \{ J'_{xy} f'_n + \hat{\alpha}'(y'_{n+1} - y'_n) \} \sin \theta'_n \sin \theta'_{n+1} \cos(\phi'_{n+1} - \phi'_n) - J_z f_n \cos \theta_n \cos \theta_{n+1} - J'_z f'_n \cos \theta'_n \cos \theta'_{n+1} + \{ J_c g_n + \hat{\beta}(y_{n+1} - y_n) \} \sin \theta_n \sin \theta'_n \cos(\phi_n - \phi'_n) + J'_c g'_n \cos \theta_n \cos \theta'_n + A \cos^2 \theta_n + A' \cos^2 \theta'_n + h \{ \sin^2 \theta_n \sin^2 \theta_{n+1} \sin^2(\phi_{n+1} - \phi_n) + q_0^2 \} + h' \{ \sin^2 \theta'_n \sin^2 \theta'_{n+1} \sin^2(\phi'_{n+1} - \phi'_n) + q_0^2 \} + \frac{p_n^2}{2M} + \frac{p'_n{}^2}{2M} + K(y_{n+1} - y_n)^2 + K'(y'_{n+1} - y'_n)^2 \right]. \quad (10)$$

The quasispin model introduced above implies that the dynamics of bases in DNA can be described by the following equations of motion:

$$\sin \theta_n \frac{\partial \theta_n}{\partial t} = \frac{\partial H}{\partial \phi_n}, \quad \sin \theta_n \frac{\partial \phi_n}{\partial t} = - \frac{\partial H}{\partial \theta_n}, \quad (11a)$$

$$\sin \theta'_n \frac{\partial \theta'_n}{\partial t} = \frac{\partial H}{\partial \phi'_n}, \quad \sin \theta'_n \frac{\partial \phi'_n}{\partial t} = - \frac{\partial H}{\partial \theta'_n}. \quad (11b)$$

When the anisotropy energies  $A$  and  $A'$  are much larger than the other interactions, then upon substituting

Hamiltonian (10), the equations of motion (11a) and (11b) become

$$\frac{\partial \phi_n}{\partial t} = 2A \cos \theta_n, \quad (12a)$$

$$\frac{\partial \phi'_n}{\partial t} = 2A' \cos \theta'_n. \quad (12b)$$

The other two  $\theta_n$  and  $\theta'_n$  equations in Eq. (11) satisfy identically. With the use of Eqs. (12a) and (12b), the Hamiltonian (10) becomes

$$H = \sum_n \left[ \frac{I}{2} \left( \frac{\partial \phi_n}{\partial t} \right)^2 + \frac{I'}{2} \left( \frac{\partial \phi'_n}{\partial t} \right)^2 - \{ J_{xy} f_n + \hat{\alpha}(y_{n+1} - y_n) \} \sin \theta_n \sin \theta_{n+1} \cos(\phi_{n+1} - \phi_n) - \{ J'_{xy} f'_n + \hat{\alpha}'(y'_{n+1} - y'_n) \} \sin \theta'_n \sin \theta'_{n+1} \cos(\phi'_{n+1} - \phi'_n) - J_z f_n \cos \theta_n \cos \theta_{n+1} - J'_z f'_n \cos \theta'_n \cos \theta'_{n+1} + \{ J_c g_n + \hat{\beta}(y_{n+1} - y_n) \} \sin \theta_n \sin \theta'_n \cos(\phi_n - \phi'_n) + J'_c g'_n \cos \theta_n \cos \theta'_n + h \{ \sin^2 \theta_n \sin^2 \theta_{n+1} \sin^2(\phi_{n+1} - \phi_n) + q_0^2 \} + h' \{ \sin^2 \theta'_n \sin^2 \theta'_{n+1} \sin^2(\phi'_{n+1} - \phi'_n) + q_0^2 \} + \frac{p_n^2}{2M} + \frac{p'_n{}^2}{2M} + K(y_{n+1} - y_n)^2 + K'(y'_{n+1} - y'_n)^2 \right], \quad (13)$$

where  $I = \frac{1}{2A}$  and  $I' = \frac{1}{2A'}$  are the moments of inertia of the nucleotides around the axes at  $P_n$  and  $P'_n$ . The first two terms proportional to  $I$  and  $I'$  represent the kinetic energies of the rotational motion of the  $n$ th nucleotide bases accompanied by the potential energy associated with the  $n$ th nucleotide, sugar, and phosphate and their complementary units around the axes at  $P_n$  and  $P'_n$ . In Hamiltonian (13), the nonlinear potential associated with different interactions, bondings, excitations, twist, etc., in terms of the rotational angles is given in Table I.

As we have considered plane ( $xy$ ) base rotator model here, we assume  $\theta_n = \theta'_n = \frac{\pi}{2}$ . Since the physical properties of the two DNA strands are similar, we can also assume that  $J_{xy} = J'_{xy} = J$ ,  $J_z = J'_z$ ,  $J_c = J'_c$ ,  $K = K'$ ,  $\hat{\alpha} = \hat{\alpha}'$ ,  $h = h'$ , and  $f_n = f'_n$ . Further, as the above coefficients associated with the strands  $S$  and  $S'$  are equal [26], we can limit our discussion to the case  $I = I'$ . In view of the above assumptions, the Hamiltonian (13) can be rewritten in the limits of absolute minima of potential as

$$H = \sum_n I \left[ \left( \frac{\partial \phi_n}{\partial t} \right)^2 + \left( \frac{\partial \phi'_n}{\partial t} \right)^2 \right] - [Jf_n + \hat{\alpha}(y_{n+1} - y_n)] \{ [1 - \cos(\phi_{n+1} - \phi_n)] \} - [J'f_n + \hat{\alpha}(y'_{n+1} - y'_n)] \{ [1 - \cos(\phi'_{n+1} - \phi'_n)] \} \\ + [J_c g_n + \hat{\beta}(y_{n+1} - y_n)] [1 - \cos(\phi_n - \phi'_n)] + h \{ 2q_0^2 - [\sin(\phi_{n+1} - \phi_n) - q_0]^2 - [\sin(\phi'_{n+1} - \phi'_n) - q_0]^2 \} \\ + \frac{p_n^2}{2M} + \frac{p'_n{}^2}{2M} + K[(y_{n+1} - y_n)^2 + (y'_{n+1} - y'_n)^2]. \quad (14)$$

### B. Dynamical equation

Having constructed the Hamiltonian for the generalized model, the dynamical equation can be obtained by deriving the associated Hamilton's equations of motion in the form

$$I \frac{\partial^2 \phi_n}{\partial t^2} = \{ [Jf_n + \hat{\alpha}(y_{n+1} - y_n)] \sin(\phi_{n+1} - \phi_n) - [Jf_{n-1} + \hat{\alpha}(y_n - y_{n-1})] \sin(\phi_n - \phi_{n-1}) \\ + [J_c g_n + \hat{\beta}(y_{n+1} - y_n)] \sin(\phi_n - \phi'_n) - 2hq_0 [\cos(\phi_{n+1} - \phi_n) - \cos(\phi_n - \phi_{n-1})] \}, \quad (15a)$$

$$I \frac{\partial^2 \phi'_n}{\partial t^2} = \{ [Jf_n + \hat{\alpha}(y'_{n+1} - y'_n)] \sin(\phi'_{n+1} - \phi'_n) - [Jf_{n-1} + \hat{\alpha}(y'_n - y'_{n-1})] \sin(\phi'_n - \phi'_{n-1}) \\ + [J_c g_n + \hat{\beta}(y_{n+1} - y_n)] \sin(\phi'_n - \phi_n) - 2hq_0 [\cos(\phi'_{n+1} - \phi'_n) - \cos(\phi'_n - \phi'_{n-1})] \}, \quad (15b)$$

$$M \ddot{y}_n = 2K(y_{n+1} - 2y_n + y_{n-1}) - \hat{\alpha} [\cos(\phi_{n+1} - \phi_n) - \cos(\phi_n - \phi_{n-1})] + \hat{\beta} [\cos(\phi_{n+1} - \phi'_{n+1}) - \cos(\phi_{n-1} - \phi'_{n-1})], \quad (15c)$$

$$M \ddot{y}'_n = 2K(y'_{n+1} - 2y'_n + y'_{n-1}) - \hat{\alpha} [\cos(\phi'_{n+1} - \phi'_n) - \cos(\phi'_n - \phi'_{n-1})] + \hat{\beta} [\cos(\phi_{n+1} - \phi'_{n+1}) - \cos(\phi_{n-1} - \phi'_{n-1})]. \quad (15d)$$

Equations (15a)–(15d) describe the dynamics of bases in DNA at the discrete level, when rotational motion of bases in a plane normal to the helical axis is considered. In the  $B$  form of DNA, as the difference in angular rotation of bases with respect to neighboring bases along the two strands is small, we assume that  $\sin(\phi_{n\pm 1} - \phi_n) \approx (\phi_{n\pm 1} - \phi_n)$  and  $\sin(\phi'_{n\pm 1} - \phi'_n) \approx (\phi'_{n\pm 1} - \phi'_n)$ . Also, as the DNA molecular chain is very long, having several thousand base pairs, compared to the distance between the neighboring bases along the strands “ $a$ ”, it is appropriate to make a continuum approximation, which is also valid in the long wavelength and low temperature limit. This is done by introducing two fields of rotational angles,  $\phi_n(t) \rightarrow \phi(z, t)$  and  $\phi'_n(t) \rightarrow \phi'(z, t)$ , where  $z = na$ . The inhomogeneity in both stacking and hydrogen bonds is also expressed by the fields as  $f_n \rightarrow f(z)$  and  $g_n \rightarrow g(z)$ . Also, we introduce the following expansions:

$$\phi_{n\pm 1} = \phi(z, t) \pm a \frac{\partial \phi}{\partial z} + \dots, \quad (16a)$$

$$f_{n\pm 1} = f(z) \pm a \frac{\partial f}{\partial z} + \dots, \quad (16b)$$

$$g_{n\pm 1} = g(z) \pm a \frac{\partial g}{\partial z} + \dots, \quad (16c)$$

$$y_{n\pm 1} = y(z, t) \pm a \frac{\partial y}{\partial z} + \dots. \quad (16d)$$

In a similar way, the expansions for  $\phi'_{n\pm 1}$  and  $y'_{n\pm 1}$  can be written. Since the inhomogeneity is associated with the bases themselves, the same parameter  $a$  is used in all the expansions.

Using the expansions given in Eqs. (16a)–(16d) in Eqs. (15a)–(15d), the equations of motion in the continuum limit reduce to

$$\phi_{tt} = [f(z) + 2h] \phi_{zz} + f_z \phi_z + \hat{\alpha} (y_z \phi_z)_z \\ - \frac{1}{2} g(z) \sin(\phi - \phi') + \hat{\beta} y_z \sin(\phi - \phi'), \quad (17a)$$

$$\phi'_{tt} = [f(z) + 2h] \phi'_{zz} + f_z \phi'_z + \hat{\alpha} (y'_z \phi'_z)_z \\ - \frac{1}{2} g(z) \sin(\phi' - \phi) + \hat{\beta} y_z \sin(\phi' - \phi), \quad (17b)$$

$$y_{tt} = v^2 y_{zz}, \quad (17c)$$

$$y'_{tt} = v^2 y'_{zz}. \quad (17d)$$

In the above equations, the subscripts  $t$  and  $z$  represent partial derivatives with respect to time  $t$  and the spatial variable  $z$ , which have been rescaled, respectively, as  $t \rightarrow \sqrt{\frac{Ja^2}{I}} t$  and  $z \rightarrow \sqrt{\frac{Ja^2}{2h}} z$ . Also, the various parameters are defined as  $J_c \rightarrow \frac{-Ja^2}{2}$ ,  $\hat{\epsilon} = \frac{a}{J}$ ,  $\hat{\beta} = \frac{2\epsilon\hat{\beta}}{a^2}$ ,  $v^2 = \frac{2K}{MI}$ , and  $\hat{\alpha} = \epsilon\hat{\alpha}$ . Upon adding Eqs.

TABLE I. Nonlinear substrate potential for different interactions, excitations, and twist in terms of the rotational angles of the bases in a plane normal to the helical axis.

Type of interaction/bonding/twist	Form of the potential
Homogeneous stacking	$-J[\sin \theta_n \sin \theta_{n+1} \cos(\phi_{n+1} - \phi_n) + \cos \theta_n \cos \theta_{n+1}]$ $-J'[\sin \theta'_n \sin \theta'_{n+1} \cos(\phi'_{n+1} - \phi'_n) + \cos \theta'_n \cos \theta'_{n+1}]$
Inhomogeneous stacking	$-J_{xy} f_n \sin \theta_n \sin \theta_{n+1} \cos(\phi_{n+1} - \phi_n) - J_z f_n \cos \theta_n \cos \theta_{n+1}$ $-J'_{xy} f'_n \sin \theta'_n \sin \theta'_{n+1} \cos(\phi'_{n+1} - \phi'_n) - J'_z f'_n \cos \theta'_n \cos \theta'_{n+1}$
Homogeneous hydrogen bonding	$J \sin \theta_n \sin \theta'_n \cos(\phi_n - \phi'_n) + J' \cos \theta_n \cos \theta'_n$
Inhomogeneous hydrogen bonding	$J_c g_n \sin \theta_n \sin \theta'_n \cos(\phi_n - \phi'_n) + J'_c g'_n \cos \theta_n \cos \theta'_n$
Twist/helicity	$h\{2q_0^2 - [\sin(\phi_{n+1} - \phi_n) - q_0]^2 + [\sin(\phi'_{n+1} - \phi'_n) - q_0]^2\}$
Flexibility/phonon	$\frac{1}{2} \dot{\phi}_n^2 + \frac{1}{2} \dot{\phi}'_n{}^2 + K(y_{n+1} - y_n)^2 + K'(y'_{n+1} - y'_n)^2$
Phonon coupling with stacking	$\frac{1}{2} \dot{\phi}_n^2 + \frac{1}{2} \dot{\phi}'_n{}^2 + K(y_{n+1} - y_n)^2 + K'(y'_{n+1} - y'_n)^2$ $-\hat{\alpha}(y_{n+1} - y_n) \sin \theta_n \sin \theta_{n+1} \cos(\phi_{n+1} - \phi_n)$ $-\hat{\alpha}'(y'_{n+1} - y'_n) \sin \theta'_n \sin \theta'_{n+1} \cos(\phi'_{n+1} - \phi'_n)$
Phonon coupling with hydrogen bonds	$\frac{1}{2} \dot{\phi}_n^2 + \frac{1}{2} \dot{\phi}'_n{}^2 + K(y_{n+1} - y_n)^2 + K'(y'_{n+1} - y'_n)^2$ $+ \hat{\beta}(y_{n+1} - y_n) \sin \theta_n \sin \theta'_n \cos(\phi_n - \phi'_n)$

(17a) and ((17b), the resultant equation satisfies identically and on subtracting Eq. (17b) from (17a), we get

$$(\phi - \phi')_{tt} = [2h + f(z)](\phi - \phi')_{zz} + f_z(\phi - \phi')_z + \hat{\alpha}(y_z \phi_{zz} + y_{zz} \phi_z - y'_z \phi'_{zz} - y'_{zz} \phi'_z) - g(z) \sin(\phi - \phi') + 2\hat{\beta} y_z \sin(\phi - \phi'). \quad (18)$$

As the two DNA strands are asymmetric in nature, they will exhibit similar physical behavior [63]. Also, due to the above, when an open state configuration is formed in DNA, the two complementary bases rotate in opposite directions, so that  $\phi = -\phi'$ . The above limitation further helps to deduce the coupled evolution equations (17a) and (17b) into a generalized sine-Gordon family of equation. Then Eq. (18) becomes

$$\Psi_{tt} = [2h + \hat{\alpha} y_z + f(z)]\Psi_{zz} + f_z \Psi_z + \hat{\alpha} y_{zz} \Psi_z + [\hat{\beta} y_z - g(z)] \sin \Psi, \quad (19)$$

where  $\Psi = 2\phi$ . In analogy with the above choice, it is appropriate to choose  $y' = -y$ , and then Eqs. (17c) and (17d) can be written as

$$y_{tt} = v^2 y_{zz}. \quad (20)$$

Assuming that the inhomogeneity along and between the DNA strands are small, we write  $f(z) = 1 + \lambda_1 f_1(z)$  along the strands and  $g(z) = 1 + \lambda_1 g_1(z)$  between the strands, where  $\lambda_1$  is a small constant. Using the above values of  $f(z)$  and  $g(z)$ , Eqs.(19) and (20) can be written as

$$\Psi_{tt} = \Psi_{zz} - \sin \Psi + \lambda_1 \{ [f_1(z) + h + \hat{\alpha} y_z] \Psi_{zz} + f_{1z} \Psi_z - g_1(z) \sin \Psi + \hat{\alpha} y_{zz} \Psi_z + \hat{\beta} y_z \sin \Psi \}, \quad (21a)$$

$$y_{tt} = v^2 y_{zz}. \quad (21b)$$

While writing Eq. (21a), we have redefined  $h \rightarrow \frac{\lambda_1 h}{2}$ ,  $\hat{\alpha} \rightarrow \lambda_1 \hat{\alpha}$ , and  $\hat{\beta} \rightarrow \lambda_1 \hat{\beta}$ . Equations (21a) and (21b) describe the dynamics of an inhomogeneous DNA double helical molecule with flexible strands in the continuum limit.

### III. DNA DYNAMICS IN TERMS OF PERTURBED NONLINEAR SCHRÖDINGER EQUATION

When the DNA double helical molecule is treated as two homogeneous linear rigid chains, that is, when  $h = \hat{\alpha} = \hat{\beta} = 0$ ,  $f_1(z) = g_1(z) = 0$ , Eq. (21a) reduces to the well-known completely integrable sine-Gordon equation which admits kink-antikink solitons [64]. The kink and antikink soliton solutions of the sine-Gordon equation represent the configuration of base pair opening in the homogeneous DNA molecule with rigid strands. Further, the effect of inhomogeneity in the strands and hydrogen bonds, as well as helicity and flexibility in the strands are studied in detail in a series of papers by one of the present authors [44–46]. However, as the kink-antikink topological soliton of the sine-Gordon equation is considerably broad, a large number of base pairs participate in the opening. However, as mentioned earlier, if the base pair opening can be represented in terms of a pulselike nontopological soliton, which is highly localized with small width, it is expected that fewer base pairs will take part in the opening, which is advantageous. This localized formation of base pair opening can occur and travel in the form of bubbles and breathers along the DNA molecule. Therefore, it is intended to rewrite the dynamical equation (21a) in the form of a generalized nonlinear Schrödinger equation, which will admit localized pulselike soliton with small perturbation.

For this, we use derivative expansion [65,66], which extends the independent variables  $z$  and  $t$  into several variables  $z_0, z_1, \dots$  and  $t_0, t_1, \dots$ , where

$$z_n = \epsilon^n z, \quad t_n = \epsilon^n t. \quad (22)$$

Using the above expansions, we write Eq. (21a) as

$$\Psi_{tt} + 2\epsilon \Psi_{tT} + \epsilon^2 \Psi_{TT} = \lambda_1 \{ ([1 + f_1(z)] + h + \hat{\alpha} y_z) (\Psi_{zz} + 2\epsilon \Psi_{zZ} + \epsilon^2 \Psi_{ZZ}) + f_{1z} \Psi_z + \hat{\alpha} y_{zz} \Psi_z - \{ [1 + g_1(z)] + \hat{\beta} y_z \} \sin \Psi \}. \quad (23)$$

In the above, we have used  $z$  for  $z_0$ ,  $Z$  for  $z_1$ ,  $t$  for  $t_0$ , and  $T$  for  $t_1$ . Now we assume the solution of Eq. (23) in the form of a modulated wave as given below:

$$\Psi = F_1(Z, T)e^{i(kz - \omega t)} + \text{c.c.} + \epsilon[F_0(Z, T) + F_2(Z, T)e^{2i(kz - \omega t)} + \text{c.c.}]. \quad (24)$$

The above solution contains a d.c. term, a first harmonic term and a second harmonic term, and the complex conjugate (c.c.), which explicitly defines  $\Psi$  as a real quantity. Substituting Eq. (24) in Eq. (23) and equating the zero harmonic, first and second harmonic terms, we get the following results.

For the zero harmonic equation,

$$F_{0TT} = F_{0ZZ}. \quad (25a)$$

The zero harmonic equation (25a) is a linear nondispersive wave equation, the solution of which can be written in terms of a linear wave, given by  $F_0 = Ce^{i(Z-T)}$ , where  $C$  is the amplitude of the wave.

For the first harmonic equation,

$$\begin{aligned} -2i\epsilon[\omega F_{1T} + kF_{1Z}] + \epsilon^2[F_{1TT} - F_{1ZZ} - \frac{1}{2}|F_1|^2 F_1] \\ = i\lambda_1[(\hat{\alpha}k^3 + 6\hat{\beta}k)y_Z - i\{k^2 f_1(Z) + g_1(Z) + hk^2\}]F_1. \end{aligned} \quad (25b)$$

While writing the above equation, we have used the dispersion relation  $\omega^2 = 1 + k^2$ .

For the second harmonic equation,

$$F_2 = 0. \quad (25c)$$

Upon using the transformations  $\hat{X} = Z - V_g T$ , where  $V_g$  is the group velocity and  $s = \epsilon T$ , and the redefinitions  $\hat{\beta} = \epsilon\beta$ ,  $\lambda_1 = \epsilon\lambda_1$ , Eq. (25b) transforms into the following equation:

$$\begin{aligned} iF_{1s} + PF_{1\hat{X}\hat{X}} + 2|F_1|^2 F_1 = i\lambda_1[(\hat{\alpha}k^3 + 6\hat{\beta}k)y_{\hat{X}} - i\{k^2 f_1(\hat{X}) \\ + g_1(\hat{X}) + hk^2\}]F_1, \end{aligned} \quad (26)$$

where  $P = (\frac{\omega^2 - k^2}{2\omega^3})$ . On introducing the new spatial variable  $X = (\frac{\omega^2 - k^2}{2\omega^3})^{-\frac{1}{2}} \hat{X}$ , Eq. (26) can be rewritten in the form of the following perturbed nonlinear Schrödinger equation:

$$iF_{1s} + F_{1XX} + 2|F_1|^2 F_1 = i\lambda_1 R[F_1], \quad (27a)$$

where

$$R[F_1] = [(\hat{\alpha}k^3 + 6\hat{\beta}k)y_X - i\{k^2 f_1(X) + g_1(X) + hk^2\}]F_1. \quad (27b)$$

Equation (27a) is called a perturbed nonlinear Schrödinger equation because, when  $\lambda_1 = 0$ , it reduces to the completely integrable nonlinear Schrödinger (NLS) equation:

$$iF_{1s} + F_{1XX} + 2|F_1|^2 F_1 = 0, \quad (28)$$

which admits  $N$ -soliton solutions [67].

#### IV. SOLITON PERTURBATION

The completely integrable NLS equation found in Eq. (28) describes the dynamics of a rigid homogeneous DNA molecular chain when it is treated as two interacting linear chains. The one soliton solution of the NLS equation describes the open state configuration in the DNA molecular chain

which propagates along the direction of the helical axis. Now, the natural question arises as to what the impact of inhomogeneity, helicity, and flexibility of the molecular chain on the propagating solitonic excitations will be. The answer to this question is found by carrying out a multiple scale soliton perturbation analysis on the perturbed NLS equation (27). Different perturbation methods [66,68–77], such as the Lindstedt-Poincaré perturbation method, the method of averaging, and the harmonic balance method, are available. Among the different methods, the multiple-scale perturbation method [66,78], which is widely used, treats the independent variables expanded in different scales and the dependent variables are expanded in asymptotic series. In the following, the details of the multiple scale soliton perturbation theory used to solve the perturbed NLS equation (27) is presented.

##### A. Linearization of the perturbed nonlinear Schrödinger equation

In order to study the effect of perturbation on the soliton, the time variable  $s$  is transformed into several variables as  $s_n = \gamma^n s$ , where  $n = 0, 1, 2, \dots$ , and  $\gamma$  is a very small parameter. Simultaneously,  $F_1$  and  $R[F_1]$  are expanded in asymptotic series, as given below:

$$F_1 = F_1^{(0)} + \gamma F_1^{(1)} + \gamma^2 F_1^{(2)} + \dots, \quad (29)$$

$$R[F_1] = R^{(1)}[F_1^{(0)}] + \gamma R^{(2)}[F_1^{(0)}, F_1^{(1)}] + \dots \quad (30)$$

Substituting the above expansions in Eq. (27), and equating the coefficients of different powers of  $\gamma$ , we obtain the following equations:

$$\gamma^0 : iF_{1s_0}^{(0)} + F_{1XX}^{(0)} + 2|F_1^{(0)}|^2 F_1^{(0)} = 0, \quad (31)$$

$$\begin{aligned} \gamma^1 : iF_{1s_1}^{(1)} + F_{1XX}^{(1)} + 4|F_1^{(0)}|^2 F_1^{(1)} + 2F_1^{(0)} F_1^{(1)} \\ = i[R^{(1)}F_1^{(0)} - F_{1s_1}^{(0)}], \end{aligned} \quad (32a)$$

where

$$R^{(1)} = [(\hat{\alpha}k^3 + 6\hat{\beta}k)y_X - i\{k^2 f_1(X) + g_1(X) + hk^2\}]. \quad (32b)$$

The equation obtained at order of  $\gamma^0$ , that is, Eq. (31), is just the standard NLS equation. It has the single soliton solution which is explicitly written as

$$F_1^{(0)}(X, s_0) = 2\beta \text{sech} \hat{z} e^{-i\theta}, \quad (33)$$

with

$$\hat{z} = 2\beta(X - \xi), \quad \xi = -4\alpha s_0, \quad (34a)$$

$$\theta = \frac{\alpha \hat{z}}{\beta} + \delta = 2\alpha(X - \xi) + \delta, \quad \delta = -4(\alpha^2 + \beta^2)s_0, \quad (34b)$$

where  $\alpha$ ,  $\beta$ ,  $\xi$ , and  $\delta$  are four real parameters which determine the propagating velocity, amplitude, position, and phase of the soliton, respectively. Due to perturbation, the above soliton parameters are assumed to be functions of the slow time variables  $s_0, s_1, s_2, \dots$ . However,  $\alpha, \beta$  are independent of  $s_0$ , and the  $s_0$ -dependent parameters  $\xi$  and  $\delta$  are given by the second equations in Eqs. (34a) and (34b), respectively. The



initial condition for perturbation in the case of the one soliton given in Eq. (33) is written as

$$F_1^{(0)}(X,0) = 2\beta \operatorname{sech}[2\beta(X - X_0)] e^{-i(2\alpha X + \theta_0)}, \quad (35)$$

$$F_1^{(n)}(X,0) = 0, \quad \text{for } n = 1, 2, \dots \quad (36)$$

It follows from Eq. (33) that

$$F_{1s_n}^{(0)} = 2\{[\beta_{s_n} \psi_1(z) + 2\beta^2 \xi_{s_n} \psi_2(z)] + i[\beta(2\alpha \xi_{s_n} - \delta_{s_n}) \phi_1(z) - \alpha_{s_n} \phi_2(z)]\} e^{-i\theta}, \quad (37)$$

where

$$\phi_1(\hat{z}) = \operatorname{sech}\hat{z}, \quad \phi_2(\hat{z}) = \hat{z} \operatorname{sech}\hat{z}, \quad (38)$$

$$\psi_1(\hat{z}) = (1 - \hat{z} \tanh \hat{z}) \operatorname{sech}\hat{z}, \quad \psi_2(\hat{z}) = \tanh \hat{z} \operatorname{sech}\hat{z}. \quad (39)$$

$\phi_1$ ,  $\phi_2$ ,  $\psi_1$ ,  $\psi_2$  are, in fact, discrete eigenfunctions. The linearized NLS equation (32), with the initial conditions given in Eqs. (35) and (36), are reduced to the following form with the use of Eq. (37):

$$\begin{aligned} iF_{1s_0}^{(1)} + 8i\alpha\beta F_{1\hat{z}} + 4\beta^2 F_{1\hat{z}\hat{z}} + 4|F_1^{(0)}|^2 F_1^{(1)} + 2F_1^{(0)} F_1^{(1)} \\ = iR^{(1)} F_1^{(0)} - 2i[\beta_{s_1} \psi_1(z) + 2\beta^2 \xi_{s_1} \psi_2(z) \\ + i\{2\alpha\beta \xi_{s_1} - \beta\delta_{s_1}\} \phi_1(\hat{z}) - \alpha_{s_1} \phi_2(\hat{z})] e^{-i\theta}. \end{aligned} \quad (40)$$

Writing

$$F_1^{(1)} = e^{-i\theta} G_1^{(1)} = [C^{(1)} + iD^{(1)}] e^{-i\theta}, \quad (41)$$

where  $C^{(1)}$  and  $D^{(1)}$  are the real and imaginary parts of  $G_1^{(1)}$ , respectively, the complex equation (41) can be written as the following two real simultaneous equations:

$$\begin{aligned} C_{s_0}^{(1)} + 4\beta^2 \hat{L}_1 D^{(1)} = [g_1(\hat{z}) + k^2 f_1(\hat{z})] F_1^{(0)} e^{i\theta} \\ - 2\beta_{s_1} \psi_1(\hat{z}) - 4\beta^2 \xi_{s_1} \psi_2(\hat{z}), \end{aligned} \quad (42a)$$

$$D_{s_0}^{(1)} - 4\beta^2 \hat{L}_2 C^{(1)} = -(4\alpha\beta \xi_{s_1} - 2\beta\delta_{s_1}) \phi_1(\hat{z}) + 2\alpha_{s_1} \phi_2(\hat{z}), \quad (42b)$$

with

$$C^{(1)}(\hat{z}, 0) = D^{(1)}(\hat{z}, 0), \quad (43)$$

where

$$\hat{L}_1 = \frac{d^2}{d\hat{z}^2} + 2\operatorname{sech}\hat{z}^2 - 1, \quad \hat{L}_2 = \frac{d^2}{d\hat{z}^2} + 6\operatorname{sech}\hat{z}^2 - 1 \quad (44)$$

are two self-adjoint linear differential operators.

### B. Coupled eigenvalue problem

The solutions of Eqs. (42a) and (42b) can be found using the method of separation of variables, with the initial condition given in Eq. (43). The key to the problem is to solve the following types of eigenvalue problems of the operators  $\hat{L}_1$ ,  $\hat{L}_2$ :

$$\hat{L}_1 \Omega = \lambda \zeta, \quad (45a)$$

$$\hat{L}_2 \zeta = \lambda \Omega. \quad (45b)$$

We assume the eigen functions of Eqs. (45a) and (45b) in the form

$$\Omega(\hat{z}, k) = p(\hat{z}, k) e^{ik\hat{z}}, \quad (46a)$$

$$\zeta(\hat{z}, k) = q(\hat{z}, k) e^{ik\hat{z}}, \quad (46b)$$

and expand  $p(\hat{z}, k)$  and  $q(\hat{z}, k)$  as

$$p(\hat{z}, k) = c_0 + c_1 \tanh \hat{z} + \frac{c_2}{\cosh^2 \hat{z}} + c_3 \frac{\sinh \hat{z}}{\cosh^3 \hat{z}} + \dots, \quad (47a)$$

$$q(\hat{z}, k) = d_0 + d_1 \tanh \hat{z} + \frac{d_2}{\cosh^2 \hat{z}} + d_3 \frac{\sinh \hat{z}}{\cosh^3 \hat{z}} + \dots, \quad (47b)$$

where  $c_0$  and  $d_0$  are nonzero coefficients and the coefficients  $c_j$ ,  $d_j$ ,  $j = 1, 2, \dots$ , which are functions of  $\hat{z}$ , are to be determined. Using the completeness or orthonormality relations,

$$\int_{-\infty}^{\infty} \Omega(\hat{z}, k) \bar{\zeta}(\hat{z}, k') dz = \int_{-\infty}^{\infty} \zeta(\hat{z}, k) \bar{\Omega}(\hat{z}, k') dz = \delta(k - k'), \quad (48a)$$

$$\int_{-\infty}^{\infty} \Omega(\hat{z}, k) \zeta_j(\hat{z}) dz = \int_{-\infty}^{\infty} \zeta(\hat{z}, k) \Omega_j(\hat{z}) dz = 0, \quad j = 1, 2, \quad (48b)$$

$$\int_{-\infty}^{\infty} \Omega_j(\hat{z}) \zeta_l(\hat{z}) d\hat{z} = \delta_{jl}, \quad j, l = 1, 2, \quad (48c)$$

$$\int_{-\infty}^{\infty} \Omega(\hat{z}, k) \bar{\zeta}(\hat{z}, k') dk + \sum_{j=1}^2 \Omega_j(\hat{z}) \zeta_j(\hat{z}') = \delta(\hat{z} - \hat{z}'). \quad (48d)$$

Here  $\Omega$  and  $\zeta$  are the eigenfunctions and the bar over these eigenfunctions indicates the complex conjugate, we determine the following eigenfunctions using the procedure used in Ref. [66]:

$$\Omega(\hat{z}, k) = -\frac{1}{\sqrt{2\pi}(k^2 + 1)} [(k + i \tanh \hat{z})^2 - \operatorname{sech}^2 \hat{z}] e^{ik\hat{z}}, \quad (48e)$$

$$\zeta(\hat{z}, k) = -\frac{1}{\sqrt{2\pi}(k^2 + 1)} [(k + i \tanh \hat{z})^2 + \operatorname{sech}^2 \hat{z}] e^{ik\hat{z}}. \quad (48f)$$

### C. Effect of perturbation on the soliton

To find the values of  $C^{(1)}$  and  $D^{(1)}$ , we solve Eqs. (42a) and (42b) with the initial condition  $C_1(\hat{z}, 0) = D_1(\hat{z}, 0) = 0$ , using the variable separation technique by expanding

$$C^{(1)}(\hat{z}, s_0) = \int_{-\infty}^{\infty} c^{(1)}(s_0, k) \zeta(\hat{z}, k) dk + \sum_{j=1}^{\infty} c_j^{(1)}(s_0) \zeta_j(\hat{z}), \quad (49a)$$

$$D^{(1)}(\hat{z}, s_0) = \int_{-\infty}^{\infty} d^{(1)}(s_0, k) \Omega(\hat{z}, k) dk + \sum_{j=1}^{\infty} d_j^{(1)}(s_0) \Omega_j(\hat{z}). \quad (49b)$$

The eigenfunctions have the symmetries mentioned in the following first two equations. Because of these symmetries, the coefficients in Eqs. (49a) and (49b) should satisfy the following relations in order to prove that both  $C^{(1)}$  and  $D^{(1)}$

are real quantities:

$$\bar{\Omega}(\hat{z}, t) = \Omega(\hat{z}, -k), \quad \bar{\zeta}(\hat{z}, k) = \zeta(\hat{z}, k), \quad (50a)$$

$$\bar{\Omega}_j(\hat{z}) = \Omega_j(\hat{z}), \quad \bar{\zeta}_j(\hat{z}) = \zeta_j(\hat{z}), \quad j = 1, 2, \quad (50b)$$

$$\bar{c}^1(s_0, k) = c^{(1)}(s_0, -k), \quad \bar{c}_j^{(1)}(s_0) = c_j^{(1)}(s_0), \quad (50c)$$

$$\bar{d}^1(s_0, k) = d^{(1)}(s_0, -k), \quad \bar{d}_j^{(1)}(s_0) = d_j^{(1)}(s_0). \quad (50d)$$

Substituting Eqs. (49a) and (49b) into Eqs. (42a) and (42b) with the initial conditions  $C^{(1)}(\hat{z}, 0) = D^{(1)}(\hat{z}, 0) = 0$ , and employing Eqs. (48e) and (48f) and the eigenvalue relations  $\hat{L}_1 \Omega_2(\hat{z}) = -2\zeta_2(\hat{z})$ ,  $\hat{L}_2 \zeta_1(\hat{z}) = 2\Omega_1(\hat{z})$ , we get

$$\begin{aligned} & \int_{-\infty}^{\infty} [\dot{c}^{(1)}(s_0, k) + 4\beta^2 \lambda b^{(1)}(s_0, k)] \zeta(\hat{z}, k) dk + \dot{c}_1^{(1)}(s_0) \zeta_1(\hat{z}) \\ & + [\dot{c}_2^{(1)}(s_0) - 8\beta^2 d_2^{(1)}(s_0)] \zeta_2(\hat{z}) = -2\beta_{s_1} \zeta_1(\hat{z}) - 4\beta^2 \xi_{s_1} \zeta_2(\hat{z}), \end{aligned} \quad (51a)$$

$$\begin{aligned} & \int_{-\infty}^{\infty} [\dot{d}^{(1)}(s_0, k) + 4\beta^2 \lambda c^{(1)}(s_0, k)] \Omega(\hat{z}, k) dk \\ & + [\dot{d}_1^{(1)}(s_0) - 8\beta^2 c_1^{(1)}(s_0)] \Omega_1(\hat{z}) + \dot{d}_2^{(1)}(s_0) \Omega_2(\hat{z}) \\ & = \text{Im}[R^{(1)} e^{i\theta}] - (4\alpha\beta\xi_{s_1} - 2\beta\delta_{s_1}) \Omega_1(\hat{z}) + 2\alpha_{s_1} \Omega_2(\hat{z}), \end{aligned} \quad (51b)$$

with  $c^{(1)}(0, k) = c_j^{(1)}(0) = d^{(1)}(0, k) = d_j^{(1)}(0)$ ,  $j = 1, 2$ . The overdot in Eqs. (51a) and (51b) represents the derivative with respect to  $s_0$ . From Eqs. (51a) and (51b) with corresponding initial conditions, we obtain the following ordinary differential equations with suitable zero initial conditions:

$$\dot{c}^{(1)}(s_0, k) + 4\beta^2 \lambda d^{(1)}(s_0, k) = a^{(1)}(k), \quad c^{(1)}(0, k) = 0, \quad (52a)$$

$$\dot{d}^{(1)}(s_0, k) - 4\beta^2 \lambda c^{(1)}(s_0, k) = b^{(1)}(k), \quad d^{(1)}(0, k) = 0, \quad (52b)$$

$$\dot{c}_1^{(1)}(s_0) = a_1^{(1)} - 2\beta_{s_1}, \quad c_1^{(1)}(0) = 0, \quad (53a)$$

$$\dot{d}_2^{(1)}(s_0) = b_2^{(1)} + 2\alpha_{s_1}, \quad d_2^{(1)}(0) = 0, \quad (53b)$$

$$\dot{c}_2^{(1)}(s_0) - 8\beta^2 d_2^{(1)}(s_0) = a_2^{(1)} - 4\beta^2 \xi_{s_1}, \quad c_2^{(1)}(0) = 0, \quad (54a)$$

$$\begin{aligned} \dot{d}_1^{(1)}(s_0) - 8\beta^2 c_1^{(1)}(s_0) &= b_1^{(1)} - (4\alpha\beta\xi_{s_1} - 2\beta\delta_{s_1}), \\ d_1^{(1)}(0) &= 0, \end{aligned} \quad (54b)$$

where

$$a^{(1)}(k) = \int_{-\infty}^{\infty} \text{Re}[R^{(1)} e^{i\theta}] \bar{\Omega}(\hat{z}, k) d\hat{z}, \quad (55a)$$

$$a_j^{(1)}(k) = \int_{-\infty}^{\infty} \text{Re}[R^{(1)} e^{i\theta}] \bar{\Omega}_j(\hat{z}) d\hat{z}, \quad j = 1, 2, \quad (55b)$$

$$b^{(1)}(k) = \int_{-\infty}^{\infty} \text{Im}[R^{(1)} e^{i\theta}] \bar{\zeta}(\hat{z}, k) d\hat{z}, \quad (55c)$$

$$b_j^{(1)}(k) = \int_{-\infty}^{\infty} \text{Im}[R^{(1)} e^{i\theta}] \bar{\zeta}_j(\hat{z}) d\hat{z}, \quad j = 1, 2. \quad (55d)$$

The right-hand sides of Eqs. (54a) and (54b) are independent of  $s_0$  in the moving coordinate system and Eqs. (53a) and (53b) lead to secularity and integrating these equations over  $s_0$  give  $c_1^{(1)}(s_0) = (a_1^{(1)} - 2\beta_{s_1})s_0$  and  $d_2^{(1)}(s_0) = (b_2^{(1)} + 2\alpha_{s_1})s_0$ , which grow infinitely in time. Therefore, we demand that

$$a_1^{(1)} - 2\beta_{s_1} = 0 \rightarrow c_1^{(1)}(s_0) = 0, \quad (56a)$$

$$b_2^{(1)} + 2\alpha_{s_1} = 0 \rightarrow d_2^{(1)}(s_0) = 0. \quad (56b)$$

Equations (54a) and (54b) lead to secularity due to Eqs. (56a) and (56b). Therefore, we get

$$a_2^{(1)} - 4\beta^2 \xi_{s_1} = 0, \quad (57a)$$

$$b_1^{(1)} - (4\alpha\beta\xi_{s_1} - 2\beta\delta_{s_1}) = 0, \quad (57b)$$

with  $c_2^{(1)}(s_0) = 0$  and  $d_1^{(1)}(s_0) = 0$ . By substituting Eqs. (56a) and (57a) and Eqs. (56b) and (57b) into Eqs. (55b) and (55d), respectively, we obtain appropriate formulas for the soliton parameters. The derived formulas for the amplitude  $\beta$ , the velocity  $\alpha$ , the position  $\xi$ , and the phase  $\delta$  are as follows:

$$\beta_{s_1} = \frac{1}{2} \text{Re} \int_{-\infty}^{\infty} R^{(1)} e^{i\theta} \text{sech} \hat{z} d\hat{z}, \quad (58)$$

$$\xi_{s_1} = \frac{1}{4\beta^2} \text{Re} \int_{-\infty}^{\infty} R^{(1)} e^{i\theta} \hat{z} \text{sech} \hat{z} d\hat{z}, \quad (59)$$

$$\alpha_{s_1} = -\frac{1}{2} \text{Im} \int_{-\infty}^{\infty} R^{(1)} e^{i\theta} \tanh \hat{z} \text{sech} \hat{z} d\hat{z}, \quad (60)$$

$$\delta_{s_1} = 2\alpha\xi_{s_1} - \frac{1}{2\beta} \text{Im} \int_{-\infty}^{\infty} R^{(1)} e^{i\theta} (1 - \hat{z} \tanh \hat{z}) \text{sech} \hat{z} d\hat{z}. \quad (61)$$

Equations (58)–(61) give the information about how the amplitude, velocity, position, and phase of the soliton are affected by the perturbation.

In order to understand the effect of perturbation on the soliton solution, we derive the first-order correction to it. As a first step, we get the solutions of Eqs. (52a) and (52b) in a standard way:

$$\begin{aligned} c^{(1)}(s_0, k) &= -\frac{b^{(1)}(k)}{4\beta^2 \lambda} [1 - \cos(4\beta^2 \lambda s_0)] \\ &+ \frac{a^{(1)}(k)}{4\beta^2 \lambda} \sin(4\beta^2 \lambda s_0), \end{aligned} \quad (62a)$$

$$\begin{aligned} d^{(1)}(s_0, k) &= \frac{a^{(1)}(k)}{4\beta^2 \lambda} [1 - \cos(4\beta^2 \lambda s_0)] \\ &+ \frac{b^{(1)}(k)}{4\beta^2 \lambda} \sin(4\beta^2 \lambda s_0). \end{aligned} \quad (62b)$$

After substituting the values of  $c^{(1)}(s_0, k)$  and  $d^{(1)}(s_0, k)$  from Eqs. (62a) and (62b) in Eqs. (49a) and (49b), we get

$$C^{(1)}(\hat{z}, s_0) = \int_{-\infty}^{\infty} c^{(1)}(s_0, k) \zeta(\hat{z}, k) dk, \quad (63a)$$

$$D^{(1)}(\hat{z}, s_0) = \int_{-\infty}^{\infty} d^{(1)}(s_0, k) \Omega(\hat{z}, k) dk. \quad (63b)$$

Substituting Eqs. (63a) and (63b) into Eq. (41) we obtain,

$$\begin{aligned} F_1^{(1)}(\hat{z}, s_0) &= e^{-i\theta} \int_{-\infty}^{\infty} dk [c^{(1)}(s_0, k) \zeta(\hat{z}, k) \\ &+ i d^{(1)}(s_0, k) \Omega(\hat{z}, k)]. \end{aligned} \quad (64)$$

Upon using the values of  $c^{(1)}(s_0, k)$ ,  $d^{(1)}(s_0, k)$ ,  $\Omega(\hat{z}, k)$ , and  $\zeta(\hat{z}, k)$  from Eqs. (62a), (62b), (48e), and (48f), respectively, and after lengthy calculations, we get the following expression

for the first-order correction:

$$F_1^{(1)}(\hat{z}, s_0) = \frac{-ie^{-i\theta}}{8\pi\beta^2} \int_{-\infty}^{\infty} dk \frac{\bar{I}(k)}{(k^2+1)^3} [1 - e^{-4i\beta^2(k^2+1)s_0}] \\ \times (k + i \tanh \hat{z})^2 e^{ik\hat{z}} \frac{ie^{-i\theta}}{8\pi\beta^2} \\ + \int_{-\infty}^{\infty} dk \frac{I(-k)}{(k^2+1)^3} [1 - e^{4i\beta^2(k^2+1)s_0}] \text{sech}^2 \hat{z} e^{ik\hat{z}}, \quad (65)$$

where

$$I(k) = \int_{-\infty}^{\infty} dz [\bar{R}^{(1)} e^{-i\theta} (k + i \tanh \hat{z})^2 - R^{(1)} e^{i\theta} \text{sech}^2 \hat{z}] e^{ik\hat{z}}. \quad (66)$$

Having obtained the general expression and formulas for the variation of the soliton parameters, namely amplitude, velocity, position, and phase and the first-order correction to soliton, we now construct the above quantities explicitly by using specific forms for  $R^{(1)}$  corresponding to different inhomogeneities and also by including the effect of helicity and flexibility of the DNA strands separately in the following sections.

## V. IMPACT OF INHOMOGENEITY ON THE SOLITON EXCITATIONS

The perturbed NLS equation (27) contains information about three different characteristics of the DNA molecule, namely, inhomogeneity, helicity, and flexibility as perturbation. At first, we attempt to understand the effect of inhomogeneity in stacking and hydrogen bonding on the solitonic excitations exhibiting base pair opening in DNA, leaving behind helicity and flexibility by making the coefficients of helicity ( $h$ ) and the phonon coupling with stacking ( $\hat{\alpha}$ ) and hydrogen bonding ( $\hat{\beta}$ ) equal to zero, that is,  $h = \hat{\alpha} = \hat{\beta} = 0$ , respectively, in Eq. (27). In this case, the factor  $R[F_1]$  in the perturbation part of Eq. (27) is proportional to  $k^2 f_1(X) + g_1(X)$ . Inhomogeneity in the DNA molecule may occur due to different reasons. The localized form of inhomogeneity may correspond to the intercalation of a compound between neighboring bases similar to the insertion of a drug molecule and the DNA double helix has to unwind, which leads to distortion of the helix at intercalated sites explaining the function of the molecule. Periodic inhomogeneity may represent periodic repetition of base pairs or defects or molecules along the helical chain. Any form of inhomogeneity in the structure will lead to changes in the functional property of the molecule.

### A. Localized inhomogeneity

#### 1. Variation of soliton parameters

As mentioned, localized inhomogeneity in the DNA molecule may arise because of defects due to the presence of drug molecule in specified sites in the DNA. When the perturbation due to inhomogeneity is switched on, it may have impact on the parameters of the soliton, namely, the amplitude, the velocity, the position, and the phase. This is understood by evaluating the integrals found in the right-hand sides of Eqs. (58)–(61), after substituting the specific form of inhomogeneity in  $R^{(1)}$ . Upon substituting the inhomogeneities

in stacking and hydrogen bonds, namely  $f(\hat{z})$  and  $g(\hat{z})$  in the localized form  $f_1(\hat{z}) = g_1(\hat{z}) = A \text{sech} \hat{z}$ , where  $A$  is the amplitude of the localized inhomogeneity in the right hand sides of Eqs. (58)–(61), and evaluating the resultant integrals, we obtain

$$\beta_{s_1} = 0, \quad \xi_{s_1} = 0, \quad (67a)$$

$$\alpha_{s_1} = 0, \quad \delta_{s_1} = A(1+k^2) \frac{\pi}{3}. \quad (67b)$$

The above equations express the time variation of the amplitude, position, velocity, and phase of the soliton, respectively, due to localized inhomogeneity which can be rewritten after using the transformation  $s_n = \gamma^n s$ , where  $n = 0, 1, 2, \dots$  as

$$\beta_s = \beta_{s_0} + \gamma \beta_{s_1}, \quad \xi_{s_1} = 0, \quad (68a)$$

$$\alpha_s = \alpha_{s_0} + \gamma \alpha_{s_1}, \quad \delta_{s_1} = A(1+k^2) \frac{\pi}{3}. \quad (68b)$$

As  $\beta$  and  $\alpha$  are independent of  $s_0$  ( $\beta_{s_0} = \alpha_{s_0} = 0$ ), we have

$$\beta = \beta_0, \quad \xi = \xi_0 - 4\alpha_0 s, \quad (69a)$$

$$\alpha = \alpha_0, \quad \delta = \delta_0 + \gamma A(1+k^2) \frac{\pi}{3} - 4(\alpha_0^2 + \beta_0^2) s, \quad (69b)$$

where  $\beta_0$ ,  $\alpha_0$ ,  $\xi_0$ , and  $\delta_0$  represent the initial amplitude, velocity, position, and phase of the soliton and  $\gamma$  represents the small perturbation parameter. From Eqs. (69a) and (69b), it is observed that the amplitude ( $\beta$ ) and velocity ( $\alpha$ ) of the soliton remain unchanged even while the soliton excitation crosses the localized inhomogeneous region along the molecule. Also, we understand that the phase ( $\delta$ ) of the soliton depends on the initial amplitude  $\beta_0$  and also the initial velocity  $\alpha_0$  of the soliton and evolves linearly in time. Further, the time variation of the position of the soliton depends on the initial velocity. The above results further show that, except for a displacement and phase shift in the soliton, the localized inhomogeneity in the DNA molecule does not affect the solitonic excitation that represents base pair opening in the molecule. In another context, in a recent paper, Salerno [39,41], while studying the nonlinear wave dynamics of the  $T7A_1$  promotor region with a specific sequence of bases using a perturbed sine-Gordon equation, found that the static solitons acquire energy and they are accelerated, or decelerated, or reflected depending upon the finite velocities present in the dynamically active region. A perturbation study on the effect of localized inhomogeneity on the kink-antikink soliton of the sine-Gordon equation by Daniel *et al.* [44] showed that while the width of the soliton remains constant, the velocity of the soliton gets a small correction.

### 2. Perturbed soliton

Having obtained the variation of the soliton parameters, we now study the effect of the localized inhomogeneity as a perturbation on the NLS-soliton. For this, first we calculate the quantities  $a^{(1)}(k)$  and  $b^{(1)}(k)$  found in Eqs. (55a) and (55c), after using the localized form of the inhomogeneity  $f_1(\hat{z}) = g_1(\hat{z}) = A \text{sech} \hat{z}$  and evaluating the integrals. The results read

$$a^{(1)}(k) = 0, \quad (70a)$$

$$b^{(1)}(k) = \frac{-\sqrt{2\pi} A \beta k (5k^2 - 7)}{3 \sinh\left(\frac{\pi k}{2}\right)}. \quad (70b)$$

Substituting Eqs. (70a) and (70b) in Eqs. (62a) and (62b), we obtain the values of  $c^{(1)}$  and  $d^{(1)}$  as

$$c^{(1)}(k) = \frac{-2A\pi k(5k^2 - 7)}{3\sqrt{2\pi}\beta_0} \left\{ \frac{1 - \cos[4\beta^2(1 + k^2)s_0]}{(1 + k^2) \sinh \frac{\pi k}{2}} \right\}, \quad (71a)$$

$$d^{(1)}(k) = \frac{A\pi k(5k^2 - 7)}{6\sqrt{2\pi}\beta_0} \left\{ \frac{\sin[4\beta^2(1 + k^2)s_0]}{(1 + k^2) \sinh \frac{\pi k}{2}} \right\}. \quad (71b)$$

The first-order perturbation correction  $F_1^{(1)}(\hat{z}, s_0)$  can be obtained by substituting the above values of  $c^{(1)}$  and  $d^{(1)}$  in Eq. (64). The result reads

$$F_1^{(1)}(\hat{z}, s_0) = \frac{Ae^{-i\theta}}{6\pi\beta_0} \int_{-\infty}^{\infty} dk \frac{k(5k^2 - 7)e^{ik\hat{z}}}{(1 + k^2) \sinh \frac{\pi k}{2}} \times \left( \{1 - \cos[4\beta^2(1 + k^2)s_0]\}[(k + i \tanh \hat{z})^2 + \operatorname{sech}^2 \hat{z}] - \frac{i}{2} \sin[4\beta^2(k^2 + 1)s_0][(k + i \tanh \hat{z})^2 - \operatorname{sech}^2 \hat{z}] \right). \quad (72)$$

The integrals in the right-hand side of Eq. (72) can be evaluated with the use of residue theorem [79]. The residue of the above integrand can be found at the poles  $k = i$  and  $k = 2in$ ,  $n = 0, 1, 2, 3, \dots$ . After evaluating the integral using the different integral values found in the Appendix, we get the first-order perturbation correction as

$$F_1^{(1)}(\hat{z}, s_0) = \frac{iAe^{-i\theta}}{3\beta_0} \left[ \frac{115}{9} e^{-2\hat{z}} \{(15 + 4 \tanh \hat{z} + 2 \tanh^2 \hat{z})(1 - \cos 60\beta^2 s_0)\} + 96i\pi\beta^2 s_0 e^{-\hat{z}} (1 + \tanh \hat{z}) \right]. \quad (73)$$

Having obtained the first-order perturbation correction, the perturbed one soliton solution is written by adding the above result Eq. (73) to the unperturbed one soliton solution given in Eq. (33):

$$F_1 = 2\beta \operatorname{sech}[2\beta(Z + 4\alpha T)] e^{-2i\{\alpha Z + 2(\alpha^2 + \beta^2)T\}} + \frac{Ae^{-i\{2\alpha(Z + 4\alpha T) - 4(\alpha^2 + \beta^2)T\}}}{3\beta_0} \left\{ \frac{115}{9} \left[ \left( 15 + 2 \tanh^2 \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) + 4\alpha t \right] \right\} \right) \right] \right. \\ \left. + 4 \tanh 2\beta \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) \right] (1 - \cos 60\beta^2 T) e^{-4\beta \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) + 4\alpha T} \right] \right. \\ \left. + 96i\pi\beta^2 T e^{-2\beta \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) + 4\alpha t} \left\{ 1 + \tanh 2\beta \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) + 4\alpha T \right] \right\} \right\}. \quad (74)$$

Knowing  $F_1$ ,  $\Psi$  can be computed using Eq. (24) and finally the rotational angle  $\phi = \frac{\Psi}{2}$  is obtained as,

$$\phi(z, t) = 2\beta \operatorname{sech}[2\beta(z + 4\alpha t)] \cos\{(2\alpha - k)z + [2(\alpha^2 + \beta^2) + \omega]t\} + \left\{ \frac{A}{3\beta_0} \cos[2\alpha(z + 4\alpha t) - 4(\alpha^2 + \beta^2)t] \right. \\ \left. + \sin[2\alpha(z + 4\alpha t) - 4(\alpha^2 + \beta^2)t] \right\} \left[ \frac{115}{9} \left( 15 + 4 \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t \right] \right\} \right) \right. \\ \left. + 2 \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t \right] \right\} \right] (1 - \cos 60\beta^2 t) e^{-4\beta \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t} \\ \left. + 48\pi\beta^2 T \left( 1 + \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t \right] \right\} \right) e^{-2\beta \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t} \right]. \quad (75)$$

Equation (75) represents the perturbed one soliton representing the rotation of bases in the DNA molecule, under the influence of localized inhomogeneity along the strands and in the hydrogen bonds. In Fig. 3(a), the perturbed envelope one soliton for the base rotation given in Eq. (75) is plotted by choosing the parameters as  $A = 0.0009$ ,  $\alpha = 0.01$ ,  $\beta = 0.9$ ,  $\omega = 1.414$ ,  $k = 1.0$ . In the above, the values of  $\omega$  and  $k$  are chosen such that the dispersion relation is satisfied. To understand the effect of localized inhomogeneity on the

NLS-soliton, we have also plotted the unperturbed envelope one soliton in Fig. 3(b). From Figs. 3(a) and 3(b), one observes that the localized solitonic excitations representing the base pair opening moves along the two DNA strands with localized inhomogeneity. The above inhomogeneity introduces only small localized fluctuation in the tail of the soliton. However, the fluctuation that appears in the tail does not affect the robust nature of the soliton as it propagates along the molecule. This solitonic excitation, in general, may start to travel from

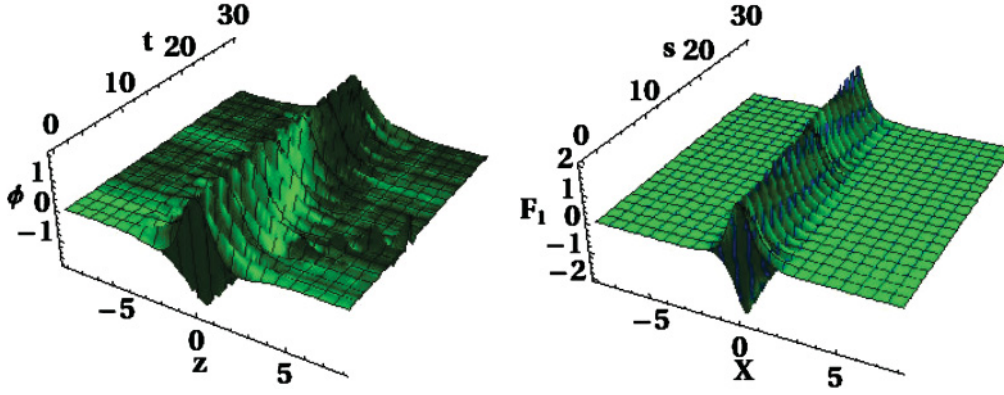


FIG. 3. (Color online) (a) The perturbed one soliton due to localized inhomogeneity in stacking and hydrogen bonds in the form of  $f_1(\hat{z}) = g_1(\hat{z}) = A \operatorname{sech} \hat{z}$ . The values of the parameters used here are  $A = 0.0009$ ,  $\alpha = 0.01$ ,  $\beta = 0.9$ ,  $\omega = 1.414$ ,  $k = 1.0$ . (b) The unperturbed one soliton solution of the NLS equation as given in Eq. (33) for the same parameter values.

the promotor region of DNA which is defined as a point of initiation to the target gene without deceleration with constant velocity since the energetic region gives energy to the transport process [39]. The above result on perturbed soliton corresponds to the rotation of bases in one of the inhomogeneous strands of the DNA molecule. The perturbed solitonic excitations in the complementary strand of the molecule can be obtained from the above result using the relation  $\phi' = -\phi$ . Thus, the combined pattern of the perturbed solitonic excitations that occur in the two strands of the inhomogeneous DNA molecule forms a bubble. In Fig. 4, a sketch of the perturbed bubble soliton propagating along the DNA molecule is given. Here the base pair opening, in the form of a bubble occurs due to the rotational motion of bases. During rotation, the bases of the DNA molecule vibrates and the hydrogen bonds connecting the two strands break, so that the DNA strands separate and this open complex of DNA encloses few broken base pairs leading to local denaturation. The connection between nonlinear energy localization in terms of bubble opening in DNA and its function is explained by Scott [80]. The DNA bubble occurs in different situations such as transcription, replication, nucleotide excision repairs [81], the process of meiotic recombination [82], etc.

### B. Periodic inhomogeneity

When the inhomogeneity appears as a periodic repetition of bases, defects, or additional molecules in the strands of the DNA molecule, it can be expressed by the periodic function  $f_1(\hat{z}) = g_1(\hat{z}) = B \cos \hat{z}$ , where  $B$  is a constant. On substituting the above form of  $f_1(\hat{z})$  and  $g_1(\hat{z})$  in Eqs. (58)–(61), we obtain

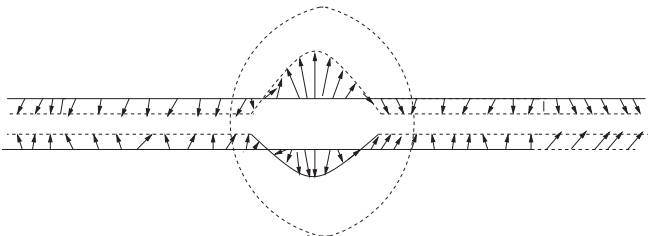


FIG. 4. A sketch of the perturbed bubble soliton propagating along the DNA molecular chain.

the following expressions that represent the time variation of the amplitude, velocity, position, and phase, respectively:

$$\beta_{s_1} = 0, \quad \alpha_{s_1} = 0, \quad (76a)$$

$$\xi_{s_1} = 0, \quad \delta_{s_1} = B \frac{\pi^2}{8} (1 + k^2) \operatorname{csc} h^3 \left( \frac{\pi}{2} \right) \sinh(\pi). \quad (76b)$$

Using the expansion  $s = \gamma^n s$ , where  $n = 0, 1, 2, \dots$  in Eqs. (76a) and (76b) and after integration, we obtain

$$\beta = \beta_0, \quad \alpha = \alpha_0, \quad (77a)$$

$$\xi = \xi_0 - 4\alpha_0 s,$$

$$\delta = \delta_0 + \gamma B \frac{\pi^2}{8} (1 + k^2) \operatorname{csc} h^3 \left( \frac{\pi}{2} \right) \sinh \pi - 4(\alpha_0^2 + \beta_0^2) s, \quad (77b)$$

where  $\beta_0$ ,  $\alpha_0$ ,  $\xi_0$ , and  $\delta_0$  represent the initial amplitude, velocity, position, and phase of the soliton, respectively. Equation (77a) tells that the amplitude and velocity of the solitonic excitation in the DNA molecule are not affected by the perturbation due to the presence of periodic inhomogeneity in stacking and hydrogen bonds. Also, we notice from Eq. (77b) that the position of the soliton depends on the initial velocity of the soliton and varies linearly in time. However, the phase of the soliton depends on both the initial amplitude and the initial velocity of the soliton and varies linearly as time passes. Qualitatively, all the parameters of the soliton undergo similar variation as in the case of localized inhomogeneity.

To obtain the perturbed part of the soliton, we substitute the periodic form of the inhomogeneity  $f_1(\hat{z})$  and  $g_1(\hat{z}) = B \cos z$  in Eqs. (55a) and (55c) and obtain the following expressions for  $a^{(1)}(k)$  and  $b^{(1)}(k)$  after evaluating the integrals:

$$a^{(1)}(k) = 0, \quad (78a)$$

$$b^{(1)}(k) = \frac{-B\beta\sqrt{\pi}}{2\sqrt{2}} \{ [2 - k^2(\pi - 4) + ik(\pi^2 - 4)] \}. \quad (78b)$$

While finding  $b_1(k)$ , we had to evaluate three important integrals based on residue theorem and the values of the integrals are given in the Appendix. Once  $a^{(1)}(k)$  and  $b^{(1)}(k)$  are obtained,  $c^{(1)}(k)$  and  $d^{(1)}(k)$  can be calculated using Eqs. (62a)

and (62b):

$$c^{(1)}(s_0, k) = \frac{B\sqrt{\pi}}{\beta(2\sqrt{2})(1+k^2)} \left[ 2(1+2k^2) - k^2 - \frac{ik}{2}(\pi^2 - 4) \right] [1 - \cos 4\beta^2(1+k^2)s_0], \quad (79a)$$

$$d^{(1)}(s_0, k) = \frac{B\sqrt{\pi}}{\beta(2\sqrt{2})(1+k^2)} \left[ 2(1+2k^2) - k^2 - \frac{ik}{2}(\pi^2 - 4) \right] \sin 4\beta^2(1+k^2)s_0. \quad (79b)$$

The first-order correction  $F_1^{(1)}$  can be found using the above values of  $c^{(1)}$  and  $d^{(1)}$ , in Eq. (64). After substitution, the residue of the integrand can be found by first finding the poles. There are two poles at  $k = \pm i$  of which the pole at  $k = i$  is inside the contour. The first-order correction is obtained as

$$F_1^{(1)}(\hat{z}, s_0) = -16i\pi B\beta(-2 + \pi)\pi s_0 [1 + \tanh(\hat{z})] e^{-\hat{z} - i[2\alpha(\hat{z} + 4\alpha s_0) - 4(\alpha^2 + \beta^2)s_0]}. \quad (80)$$

Making use of the above perturbation correction, the perturbed one soliton solution is written as

$$F_1 = 2\beta \operatorname{sech}[2\beta(Z + 4\alpha T)] e^{-2i\{\alpha Z + 2(\alpha^2 + \beta^2)T\}} - 16i\beta B\pi(-2 + \pi)\pi T \left( 1 + \tanh 2\beta \left\{ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) + 4\alpha t \right\} \right) \\ \times e^{-i[2\alpha(Z + 4\alpha T) - 4(\alpha^2 + \beta^2)T]} e^{-2\beta \left\{ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega Z - kT) + 4\alpha T \right\}}. \quad (81)$$

Using the above value of  $F_1$ , the angle of rotation of bases  $\phi$  can be obtained after finding  $\Psi$  using Eq. (24). The final result reads

$$\phi = 2\beta \operatorname{sech}[2\beta(z + 4\alpha t)] \cos\{(2\alpha - k)z + [2(\alpha^2 + \beta^2) + \omega]t\} \\ - 16\beta B\pi^2 t \left( \sin \left\{ 2\alpha \left[ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t \right] - 4(\alpha^2 + \beta^2)t \right\} \right) \\ \times \left( 1 + \tanh 2\beta \left\{ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t \right\} \right) e^{-2\beta \left\{ \left( \frac{2\omega}{\omega^2 - k^2} \right)^{\frac{1}{2}} (\omega z - kt) + 4\alpha t \right\}}. \quad (82)$$

In Fig. 5, we plot the variation of the rotational angle of bases in the form of perturbed one soliton due to periodic inhomogeneity as found in Eq. (82). The parameters chosen here are  $B = 0.001$ ,  $\beta = 0.9$ , and  $\alpha = 0.01$ , as in the case of localized inhomogeneity. From the figure we observe that the periodic inhomogeneity in stacking and hydrogen bonds which are in the form of  $B \cos z$  introduces periodic fluctuation in the tail of the soliton. The fluctuation that appears in the tail of the soliton grows slowly in time without affecting the robust nature of the soliton. As before, the perturbed solitonic excitation in the complementary strand can be obtained using the relation  $\phi' = -\phi$ , and the pair forms the bubble with a small fluctuation

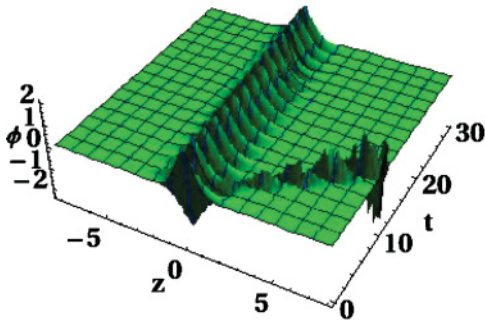


FIG. 5. (Color online) The perturbed one soliton due to periodic inhomogeneity in stacking and hydrogen bonds  $f_1(\hat{z}) = g_1(\hat{z}) = B \cos \hat{z}$  for the parameter value  $B = 0.001$ . All the other parameter values are the same as used in the case of localized inhomogeneity.

in the tail. The bubble in the present case will appear similar to the one found in the case of localized inhomogeneity as shown in Fig. 4. The only difference is that in the present case, the fluctuation that appears in the tail is periodic in nature, unlike the case of localized inhomogeneity, where it was localized.

## VI. EFFECT OF HELICITY ON DNA DYNAMICS

A real DNA molecule has helical shape by its twisting strands and the twisting leads to the study of torsional dynamics. In this section, we attempt to study the effect of helicity on the propagating soliton in a homogeneous rigid DNA helical molecule. The corresponding perturbation part  $R[F_1] = -ikh^2 F_1$  can be obtained by substituting  $f_1 = g_1 = \hat{\alpha} = \hat{\beta} = 0$  in Eq. (27b). Before finding the perturbed soliton due to the above effect, we evaluate the effect due to helicity on the amplitude, velocity, position, and phase of the soliton by substituting the above perturbed part in Eqs. (58)–(61). On evaluating the resultant integrals, we get

$$\beta_{s_1} = 0, \quad \alpha_{s_1} = 0, \quad (83a)$$

$$\xi_{s_1} = 0, \quad \delta_{s_1} = hk^2. \quad (83b)$$

On integrating Eqs. (83a) and (83b) in terms of the variable “ $s$ ” up to first-order approximation ( $\frac{\partial}{\partial s} = \frac{\partial}{\partial s_0} + \gamma \frac{\partial}{\partial s_1}$ ), we obtain

$$\beta = \beta_0, \quad \alpha = \alpha_0, \quad (84a)$$

$$\xi = \xi_0 - 4\alpha_0 s, \quad \delta = \delta_{s_0} - 4(\alpha_0^2 + \beta_0^2)s. \quad (84b)$$

It is evident that the amplitude ( $\beta$ ) and velocity ( $\alpha$ ) of the soliton remain unaltered even when there is helical character

in the DNA molecule. However, the position and phase of the soliton vary linearly as time progresses. Also, the phase of the soliton depends on the initial amplitude and velocity of the soliton, and the position depends on the initial velocity but is independent of the initial amplitude. It may be noted that the above results are qualitatively similar to that of the inhomogeneous case.

In order to derive the first-order perturbation correction on the soliton due to helicity, we substitute the term contributing to perturbation due to helicity, that is, the term proportional to  $h$  in the perturbation in Eqs. (55a) and (55c). After evaluating the integrals, we obtain

$$a^{(1)}(k) = 0, \quad (85a)$$

$$F_1^{(1)}(\hat{z}, s_0) = -\frac{3h}{2\beta\sqrt{2\pi}} e^{-i[2\alpha(\hat{z}+4\alpha s_0)-4(\alpha^2+\beta^2)s]} \left[ \int_{-\infty}^{\infty} dk \left\{ \frac{k^4 e^{ik\hat{z}} [1 - \cos 4\beta^2(1+k^2)s_0] [(k+i \tanh \hat{z})^2 + (\text{sech}^2 \hat{z})]}{(1+k^2)^3 \cosh(\frac{\pi k}{2})} \right. \right. \\ \left. \left. + i \frac{k^4 e^{ik\hat{z}} \sin[4\beta^2(1+k^2)s_0] [(k+i \tanh \hat{z})^2 - (\text{sech}^2 \hat{z})]}{(1+k^2)^3 \cosh(\frac{\pi k}{2})} \right\} \right]. \quad (87)$$

The integrals in the right-hand side of the above equation are evaluated with the aid of residue theorem. The integrands have poles of order 3 at  $k = \pm i$ . In addition, we have a simple pole at  $k = (2n+1)i$  for  $n = 0, 1, 2, 3, \dots$ . The only pole lying within the contour is the simple pole at  $k = +i$  for  $n = 0$ . After evaluating the integral using the different integral values found in the Appendix, we get the first-order perturbation correction as follows:

$$F_1^{(1)}(\hat{z}, s_0) = -\frac{3ih\pi}{64\beta} \left\{ \tanh \hat{z} (1 + \tanh \hat{z}) s_0^2 e^{-\{\hat{z}+i[2\alpha(\hat{z}+4\alpha s_0)-4(\alpha^2+\beta^2)s_0]\}} \right\}. \quad (88)$$

The perturbed one soliton solution is written by adding Eqs. (33) and (88):

$$F_1 = 2\beta \text{sech}[2\beta(Z+4\alpha T)] e^{-2i[\alpha X+2(\alpha^2+\beta^2)T]} - \frac{3ih\pi}{64\beta} e^{-2\beta\{(\frac{2\omega}{\omega^2-k^2})^{\frac{1}{2}}(\omega Z-kT)+4\alpha T\}} \\ \times \left( \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2-k^2} \right)^{\frac{1}{2}} (\omega Z-kT) + 4\alpha T \right] \right\} \left( 1 + \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2-k^2} \right)^{\frac{1}{2}} (\omega Z-kT) + 4\alpha T \right] \right\} \right) \right) T^2. \quad (89)$$

Using the value of  $F_1$ ,  $\phi$  is computed as

$$\phi = 2\beta \text{sech}[2\beta(z+4\alpha t)] \cos\{(2\alpha-k)z + [2(\alpha^2+\beta^2)+\omega]t\} \\ - \frac{3h\pi}{64\beta} \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2-k^2} \right)^{\frac{1}{2}} (\omega z-kt) + 4\alpha t \right] \right\} \left( 1 + \tanh \left\{ 2\beta \left[ \left( \frac{2\omega}{\omega^2-k^2} \right)^{\frac{1}{2}} (\omega z-kt) + 4\alpha t \right] \right\} \right) \\ \times \cos \left\{ 2\alpha \left[ \left( \frac{2\omega}{\omega^2-k^2} \right)^{\frac{1}{2}} (\omega z-kt) + 4\alpha t - 4(\alpha^2+\beta^2)t - (kz-\omega t) \right] \right\} e^{-2\beta\{(\frac{2\omega}{\omega^2-k^2})^{\frac{1}{2}}(\omega z-kt)+4\alpha t\}}. \quad (90)$$

The perturbed one soliton solution for the rotational angle  $\phi$  of the bases is plotted in Fig. 6 by choosing the helicity parameter as  $h = -15.0$ . The above value of  $h$  has also been chosen for molecular dynamic simulation [83]. All the other parameter values are chosen as the same values used in the inhomogeneous cases. From the perturbed one soliton solution given in Eq. (90) and from Fig. 6, we can observe that, initially, the helical character of the molecule introduces fluctuation only in the tail of the soliton without affecting the robust nature of the soliton. We also observe the same effect for different values of helicity. Thus, the helical nature of the DNA chain does not affect qualitatively the propagation of bubblelike base pair opening generated by soliton,

$$b^{(1)}(k) = \frac{6\beta h \sqrt{\pi} k^4 \text{sech}(\frac{\pi k}{2})}{\sqrt{2}(1+k^2)}. \quad (85b)$$

The integrals involved in Eqs. (85a) and (85b) are evaluated using residue theorem [82] and using the values of the integrals found in the Appendix. Substituting Eqs. (85) into Eqs. (62a) and (62b), we obtain

$$c^{(1)}(s_0, k) = \frac{3h}{2\beta\sqrt{2\pi}} \frac{\pi k^4 [1 - \cos 4\beta^2(1+k^2)s_0]}{(1+k^2)^2 \cosh(\frac{\pi k}{2})}, \quad (86a)$$

$$d^{(1)}(s_0, k) = \frac{3h}{2\beta\sqrt{2\pi}} \frac{\pi k^4 \sin 4\beta^2(1+k^2)s_0}{(1+k^2)^2 \cosh(\frac{\pi k}{2})}. \quad (86b)$$

Substituting Eqs. (86a) and (86b) into Eq. (64), we obtain the first-order perturbation correction as

even while the DNA molecule is treated as coupled linear chains.

## VII. EFFECT OF FLEXIBILITY IN DNA STRANDS (PHONON) ON DNA DYNAMICS

The base pair opening via nonlinear molecular excitations in an inhomogeneous DNA chain and a helicoidal DNA is so far understood by considering the DNA strands as rigid lattices. However, in nature, the force between purine bases in consecutive base pairs is repulsive and this force is resisted by stress in the helical backbones of DNA. Also, the presence of elastic strain forces in both the strands of DNA [84,85],

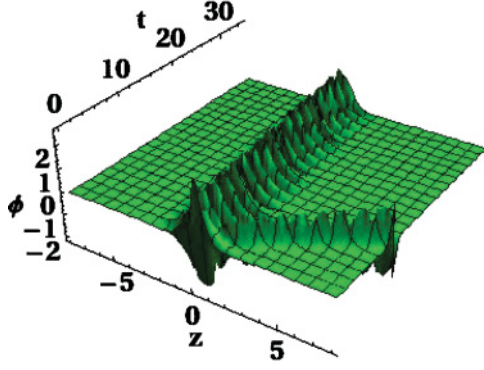


FIG. 6. (Color online) The perturbed one soliton representing base pair opening with helicity  $h = -15.0$ . The other parameter values are the same as those used in the case of inhomogeneity.

indicates the nonrigidity of the strands leading to the formation of phonons, which plays an important role in energy transfer in biological systems. It should be pointed out that the DNA molecule has elastic properties, which is unusual due to its helicoidal symmetry and the sequence of base pairs. Xiao and his co-workers [86] studied the effect of longitudinal vibrations and its coupling with hydrogen bonding and stacking by considering the dynamic plane-base rotator model of Takeno and Homma [26,27]. In the continuum limit, the dynamics is governed by a perturbed sine-Gordon equation and it was solved using the method of successive approximation. The results showed that the effect of longitudinal vibration of the lattice on the soliton is small. Very recently, Daniel and his co-worker [46], while studying the effect of longitudinal vibration of bases in the strands on the DNA dynamics, found that the dynamics is governed by a perturbed sine-Gordon equation coupled with a linear wave equation representing the lattice deformation which modulates the velocity of the kink-antikink soliton that represents base pair opening. In this section, we study the effect of flexibility of homogeneous strands on DNA dynamics by solving the associated perturbed NLS equation (27) by setting  $f_1 = g_1 = h = 0$  along with the wave equation found in Eq. (21b) for the lattice displacement. In particular, we investigate the effect of periodic lattice deformation on the solitonlike base pair opening in the DNA double helix. On assuming  $f_1 = g_1 = h = 0$ , the perturbed NLS equation (27) coupled with the linear wave equation (21b) in the transformed variable become

$$iF_{1s} + F_{1XX} + 2|F_1|^2F_1 = i\lambda_1[k(\hat{\alpha}k^2 + 6\hat{\beta})]y_XF_1, \quad (91a)$$

$$y_{ss} - v^2y_{XX} = 0. \quad (91b)$$

Here  $\hat{\alpha}$  and  $\hat{\beta}$  measure the coupling strengths of the phonon with the stacking and the hydrogen bonds, respectively. Equation (91b) is the well known one-dimensional linear wave equation which admits wave solution in the form  $y = f(X - vs) + g(X + vs)$ . The perturbed NLS equation (91a) is solved after substituting the above wave solution in it. The formation of phonons due to elastic deformation of the DNA strands coupled with DNA molecular excitations will perturb the solitonlike base pair opening in DNA. As before, the perturbation is expected to modulate the parameters of the soliton, namely, amplitude, velocity, position, and phase. In order to understand this, we solve Eqs. (58)–(61) using the

same procedure which we used in the case of inhomogeneity and helicity and evaluate the soliton parameters which then will be used to derive the first-order perturbation correction to the one soliton. From Eqs. (58)–(61), after using  $R^{(1)} = k(\hat{\alpha}k^2 + 6\hat{\beta})\cos(\hat{z} - vs)F_1$  and evaluating the integrals, we obtain

$$\beta_{s_1} = -\beta k(\hat{\alpha}k^2 + 6\hat{\beta})\pi \operatorname{csch}\left(\frac{\pi}{2}\right), \quad \alpha_{s_1} = 0, \quad (92a)$$

$$\xi_{s_1} = 0, \quad \delta_{s_1} = 0. \quad (92b)$$

Rewriting the above, in terms of the original variable “ $s$ ” up to the first-order approximation ( $\beta_s = \beta_{s_0} + \epsilon\beta_{s_1}$ ) and after integration, we get

$$\beta = \beta_0 e^{-\epsilon k(k^2\hat{\alpha} + 6\hat{\beta})\pi \operatorname{csch}(\frac{\pi}{2})s}, \quad \alpha = \alpha_0, \quad (93a)$$

$$\xi = \xi_0 - 4\alpha_0s, \quad \delta = \delta_{s_0} - 4(\alpha_0^2 + \beta_0^2)s. \quad (93b)$$

The first of Eq. (93a) says that coupling of phonon to stacking and hydrogen bonds makes the amplitude of the soliton to vary exponentially as time progresses. The quantity  $\epsilon[k(k^2\hat{\alpha} + 6\hat{\beta})\pi \operatorname{csch}(\frac{\pi}{2})]$  that appears in the exponential of  $\beta$  is a small positive quantity, and hence the amplitude decays exponentially as time progresses. However, the velocity of the soliton remains unaltered. On the other hand, the position and phase of the soliton vary linearly as time progresses due to the coupling of lattice distortion in terms of phonon with stacking and hydrogen bonds. Also, the position of the soliton depends on the initial velocity of the soliton and the phase depends on the initial amplitude and velocity of the soliton as in the previous cases.

Having evaluated the soliton parameters, the first-order perturbation correction to the one soliton solution will be derived now. For this, first, we evaluate  $a^{(1)}(k)$  and  $b^{(1)}(k)$  from Eqs. (55a) and (55c) and get

$$a^{(1)}(k) = \frac{\hat{\beta}k\sqrt{\pi}(\hat{\alpha}k^2 + 6\hat{\beta})}{2\sqrt{2}(k^2 + 1)}[k^2(\pi^2 - 1) - 2], \quad (94a)$$

$$b^{(1)}(k) = 0. \quad (94b)$$

Upon substituting Eqs. (94a) and (94b) in Eqs. (62a) and (62b), we obtain,

$$\begin{aligned} c^{(1)}(s_0, k) &= \frac{k(\hat{\alpha}k^2 + 6\hat{\beta})[\pi k^2(\pi^2 - 1) - 2\pi] \sin 4\beta^2(1 + k^2)s_0}{(8\sqrt{2\pi})\beta(1 + k^2)^2}, \end{aligned} \quad (95a)$$

$$\begin{aligned} d^{(1)}(s_0, k) &= -\frac{k(\hat{\alpha}k^2 + 6\hat{\beta})[\pi k^2(\pi^2 - 1) - 2\pi][1 - \cos 4\beta^2(1 + k^2)s_0]}{(8\sqrt{2\pi})\beta(1 + k^2)^2}. \end{aligned} \quad (95b)$$

Substitution of Eqs. (95a) and (95b) in Eq. (64) yields the following integral for the first-order perturbation



correction:

$$F_1^{(1)}(\hat{z}, s_0) = -\frac{e^{-i\theta}}{16\pi\beta} \int_{-\infty}^{\infty} dk \left[ \frac{[\pi k^3(\pi^2 - 1) - 2\pi](\hat{\alpha}k^2 + 6\hat{\beta})e^{ik\hat{z}}}{(1+k^2)^3} \{\sin[4\hat{\beta}^2(1+k^2)s_0] \right. \\ \left. \times [(k+i \tanh \hat{z})^2 + \text{sech}^2 \hat{z}] + i\{[1 - \cos 4\hat{\beta}^2(1+k^2)s_0][(k+i \tanh \hat{z})^2 - \text{sech}^2 \hat{z}]\} \right]. \quad (96)$$

The integrals involved in the above expression are evaluated using the residue theorem by first finding the poles. Here the integrand has a pole of order 3 at  $k = i$ . Even though the number of terms involved in the integration is very large, yet, it is evaluated using the technique of integration by parts with much attention. Finally, we get the first-order perturbation correction as

$$F_1^{(1)}(\hat{z}, s_0) = \frac{-2i\pi(6\hat{\beta} - \hat{\alpha})}{1149} (1 + \tanh \hat{z}) [128\hat{\beta}^2 \tanh \hat{z} \{5(\pi^2 - 1) - 4\hat{\alpha}(\pi^2 - 512\pi - 249) \\ - 4i\alpha(7\pi^2 - 256\pi - 503)\} e^{-\hat{z}} + 5\pi\hat{\beta}^2(2 + i(1 - \pi^2))] s_0 e^{-i[2\hat{\beta}(\hat{z} + 4\alpha s_0) - 4(\alpha^2 + \hat{\beta}^2)s_0]}. \quad (97)$$

Using the above first-order perturbation correction, the corresponding perturbed one soliton solution is obtained by adding Eq. (97) to the unperturbed one soliton solution given in Eq. (33):

$$F_1 = 2\beta \text{sech}[2\beta(Z + 4\alpha T)] e^{-i[2\alpha Z + 4(\alpha^2 - \beta^2)T]} - \frac{2i\pi(6\hat{\beta} - \hat{\alpha})}{1149} (1 + \tanh \hat{z}) [128\hat{\beta}^2 \tanh \hat{z} \{5(\pi^2 - 1) \\ - 4\hat{\alpha}(\pi^2 - 512\pi - 249) - 4i\alpha(7\pi^2 - 256\pi - 503)\} e^{-\hat{z}} + 5\pi\hat{\beta}^2[2 + i(1 - \pi^2)]] s_0 e^{-i[2\hat{\beta}(\hat{z} + 4\alpha s_0) - 4(\alpha^2 + \hat{\beta}^2)s_0]}, \quad (98)$$

with  $\hat{z} = 2\beta[(\frac{2\omega^3}{\omega^2 - k^2})^{1/2}(Z - \frac{k}{\omega}T)]$ . Using the above perturbed one soliton solution for  $F_1$ ,  $\Psi$  is calculated using Eq. (24) and consequently, the perturbed one soliton for the angular rotation of bases is obtained as

$$\phi = 4\beta \text{sech}[2\beta(z + 4\alpha t)] \cos[2(\alpha z + 2(\alpha^2 - \beta^2)t) - (kz - \omega t)] \\ + \frac{8\pi(6\hat{\beta} - \hat{\alpha})}{1149} \sin \left\{ (kz - \omega t) + 2\beta \left[ \left( \frac{2\omega^3}{\omega^2 - k^2} \right)^{1/2} \left( z - \frac{k}{\omega} \right) t + 4\alpha t \right] - 4(\alpha^2 + \beta^2)t \right\} \\ \times \left\{ -2 \tanh \left[ \left( \frac{2\omega^3}{\omega^2 - k^2} \right)^{1/2} \left( z - \frac{k}{\omega} \right) t \right] \left\{ 1 + \tanh \left[ \left( \frac{2\omega^3}{\omega^2 - k^2} \right)^{1/2} \left( z - \frac{k}{\omega} \right) t \right] \right\} \right. \\ \left. \times e^{-2\hat{\beta}[(\frac{2\omega^3}{\omega^2 - k^2})^{1/2}(z - \frac{k}{\omega})t - 4\alpha t]} [\pi\hat{\beta}^2 t \{-\pi^2[\hat{\alpha} + 340] + (\hat{\alpha} - 325)\}] \right\}. \quad (99)$$

The perturbed one soliton for the rotational angle  $\phi$  of the bases is plotted in Fig. 7 by choosing the coupling strengths of the phonon with the stacking and the hydrogen bonds, respectively, as  $\hat{\alpha} = 0.01$  and  $\hat{\beta} = 0.1$ . All the other parameters are chosen as in the inhomogeneous case.

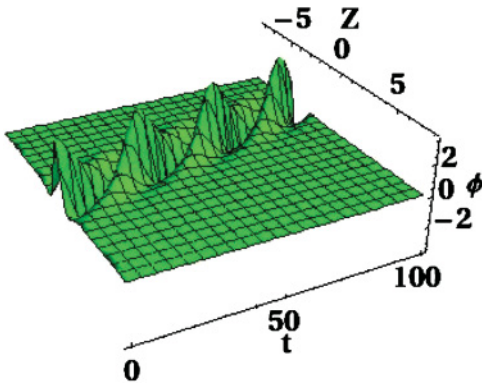


FIG. 7. (Color online) The perturbed one soliton due to flexibility when  $\hat{\alpha} = 0.01$  and  $\hat{\beta} = 0.1$ . All the other parameters are chosen as in the inhomogeneous case.

are chosen by the same values as in the inhomogeneous case. From the perturbed one soliton solution given in Eq. (99) and from Fig. 7, we observe that the elastic motion due to flexibility of the strands introduces small quakelike motion in the localized region of the soliton, which damps out slowly as time progresses. This kind of quakelike motion may produce a hole which will be the entrance for intercalators into the DNA [87]. The relation  $\phi' = -\phi$  introduces similar pattern of soliton in the complementary strand, thus forming a bubble containing fewer base pairs. Thus, the coupling of phonon to the stacking and hydrogen bonds modulate the bubble. However, the modulation dies out slowly and the robust nature of the bubble is maintained asymptotically.

## VIII. CONCLUSIONS

In this paper, base pair opening in an inhomogeneous (in stacking and hydrogen bonds) DNA double helical molecular chain with flexible strands is investigated through an understanding of the underlying internal dynamics of the molecule. To study the internal dynamics of the system, a generalized model with the Hamiltonian containing energies

TABLE II. Variation of soliton parameters and nature of perturbed soliton due to inhomogeneity, helicity, and flexibility.

Nature of perturbation	Soliton parameters				Perturbed soliton solution
	Amplitude	Velocity	Position	Phase	
Localized inhomogeneity	Unaltered	Unaltered	Varies linearly with time	Varies linearly with time	Localized aperiodic fluctuation appears in the tail of the soliton
Periodic inhomogeneity	Unaltered	Unaltered	Varies linearly with time	Varies linearly with time	Periodic fluctuation in the tail of the soliton
Helicity	Unaltered	Unaltered	Varies linearly with time	Varies linearly with time	Fluctuation appears in the tail of the soliton
Flexibility	Diminishes	Unaltered	Varies linearly with time	Varies linearly with time	Quakelike motion develops in the localized region and diminishes

corresponding to the stacking, hydrogen bonds, phonons and their coupling, along with inhomogeneity in stacking and hydrogen bonds and helicity is proposed. In fact, the model is based on an analogy with the Heisenberg model for two antiferromagnetically coupled twisted site-dependent ferromagnetic spin chains or, equivalently, two antiferromagnetically coupled inhomogeneous helical spin systems under the plane base rotator model. As the physical properties of both the strands of DNA are equal, all the energy coefficients and the moments of inertia of the complementary strands have been assumed to be equal. The Hamilton's equations of motion are then constructed for the angular rotation of bases in a plane normal to the helical axis, for both the strands, and for the lattice (strand) distortion. The hydrogen bonds connecting the two strands of the DNA molecule are broken easily, when the complementary bases open through rotational motion. Since the two DNA strands are asymmetric in nature, the open state configuration is easily formed when the two complementary bases rotate in opposite directions ( $\phi = -\phi'$ ). When it is assumed that the bases open for a small angle, in the continuum limit, the dynamical equations reduce to a generalized sine-Gordon equation coupled with a linear wave equation for lattice distortion. Under strong approximation of homogeneous, rigid, and nonhelical molecular chains, the dynamical equation will reduce to the completely integrable sine-Gordon equation, which admits kink-antikink-type of solitons representing base pair opening in the DNA molecule. However, since the above topological soliton will accommodate a large number of base pairs during opening, it is attempted here to reduce the dynamical equation to a perturbed NLS equation which will possess perturbed pulselike soliton solution so that fewer base pairs take part in the opening. This has been achieved by using a derivative expansion for the independent variables combined with a modulated wave solution for the dependent variable of the dynamical equation. In the resultant perturbed NLS equation, the perturbation corresponds to the effects due to inhomogeneity in stacking and hydrogen bonds, helicity, and flexibility of the strands. The problem then boils down to solving the perturbed NLS equation using the multiple scale soliton perturbation theory to understand the effect of (i) the inhomogeneity in stacking and hydrogen bonds, (ii) the helical nature of the strands, and (iii) the flexibility of the strands on the NLS-soliton that represents base pair

opening. The analysis brings out the variation of the soliton parameters such as amplitude, velocity, position, and phase. In the perturbation analysis, the nonlinear evolution equation is written as equivalent to a set of coupled eigenvalue problems and the perturbed soliton solution is constructed by using the eigenfunctions as the basis solutions. The perturbation analysis is carried out for all the above three cases—namely, inhomogeneity, helicity, and flexibility—separately by finding the variation of soliton parameters and the perturbed soliton solution. The inhomogeneity in the DNA molecule may arise due to different reasons. A localized inhomogeneity in DNA may arise due to defects caused by the presence of additional molecules such as drugs, carcinogens, mutants, and dyes in specific sites of the DNA sequence. Therefore, the localized inhomogeneity is assumed in the form of “Asechz” which is also amenable to analytical analysis. A periodic inhomogeneity may arise due to periodic repetition of different sites or simple defects along the strands. The helicoidal nature of the DNA molecule was incorporated into the model by including a twisting motion of the strands in analogy with twist deformation in cholesteric liquid crystals. Since the strands of DNA are flexible in nature, they deform elastically and the resultant phonons due to longitudinal stretching couple to the stacking and hydrogen bonds.

The variation of the amplitude, velocity, position, and phase of the soliton and bubble due to inhomogeneity in stacking and hydrogen bonds, helicity, and flexibility as perturbation have been found. A survey of the key results of the perturbation analysis is given in Table II. In all the cases with rigid strands, the amplitude and velocity of the soliton and bubble are unaffected due to perturbational effects. However, flexibility of the strands and the elastic motion diminish the perturbed amplitude. However, the position and phase of the soliton vary linearly with time. Also, while the position of the soliton depends on the initial velocity of the soliton, the phase depends on the initial velocity as well as the initial amplitude of the soliton. The results show that, when the DNA molecule is considered as two coupled rigid homogeneous linear continuum chains, in the limit of small angular rotation of bases, the internal dynamics is governed by the completely integrable NLS equation which admits  $N$ -soliton solutions. The pair of solitons formed in the complementary strands form a bubble and propagates along the molecule.

When there is a localized inhomogeneity in the base stacking and hydrogen bonds, it introduces small localized fluctuations in the tail of the solitons and hence in the bubble [see Fig. 3(a)]. However, the robust nature of the propagating soliton and bubble are not affected. The breaking of hydrogen bonds and opening of bases through rotation occurs nonuniformly, because of the fluctuation due to inhomogeneity in stacking and hydrogen bonds. On the other hand, if the inhomogeneity in stacking and hydrogen bonds is in the periodic form, the fluctuation in the tail of the soliton and hence the bubble appear in a periodic pattern (see Fig. 5). Bubbles start the transcription process by opening up a local region of about 20 base pairs. While moving along the helical strand, if a damaged site is recognized, these bubbles form around it and the damaged site will be removed (see Ref. [80]). In the same way, the helical nature of the DNA molecule introduces fluctuation in the tail of the soliton and bubble (see Fig. 6). When the strands of the DNA molecule are flexible, phonon excitations are generated, which in turn introduces fluctuation in the localized region of the soliton. The vibrations along the strands which is responsible for the formation of phonons increase the stretch of the hydrogen bonds and lead to unzipping of the DNA strands. The base pair opening in the form of solitons and bubble will lead to transcription process in the DNA molecule in addition to local denaturation of the molecule. Also, these phonon modes possess a number of applications to biological problems such as drug intercalation when there is inhomogeneity in the DNA molecule, dynamic mechanism of allosteric transition in antibody molecules, DNA radiosensitivity, DNA breathing, and protein-DNA interaction. As localized and periodic inhomogeneities in stacking and hydrogen bonds, the helical nature and flexibility of strands do not affect the robust nature of the soliton and bubble, the biological process in general will not be affected due to these effects. This confirms that the generalized model proposed here to understand the base pair opening in DNA molecule through the internal dynamics is a right model for the study. Having understood the base pair opening in an infinite DNA molecular chain, the natural question arises as to how the internal dynamics of a short DNA molecule with suitable boundaries which may correspond to a specific biological function contributes to base pair opening. Investigations based on numerical simulation along this line are in progress and the results will be presented elsewhere.

#### ACKNOWLEDGMENTS

The work of M.D. forms part of a major research project sponsored by DST. M.V acknowledges Bharathidasan University and University Grants Commission for financial support.

#### APPENDIX

In this appendix, we evaluate several integrals which appear in the perturbation analysis, while evaluating the soliton parameters and the perturbed soliton using the residue theorem. Before evaluation of the actual integrals, let us

calculate the following integral, which will be the basis for evaluation of all the other integrals:

$$I_1(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \tanh \hat{z} d\hat{z}. \quad (\text{A1})$$

Consider a closed path “ $c$ ” in the plane  $\varrho = \tilde{z} + i\Upsilon$  noted as follows:

$$\oint_c f_1(\varrho) d\varrho = \oint_c e^{-ik\varrho} \tanh \varrho d\varrho. \quad (\text{A2})$$

Here  $\tanh \varrho$  is a periodic function with an imaginary period  $i\pi$  and the closed integral  $c$  has the boundary  $-\infty < \tilde{z} < \infty$  and  $0 \leq \Upsilon \leq \pi$  and the integrand is analytic except for a simple pole at  $\varrho = \frac{i\pi}{2}$ . The integrand (A1) is divergent, but in physical problems, the integral limits are, of course, convergent. To keep the integral convergent, we assume  $e^{-ik\hat{z}}$  replaced by  $e^{-(ik+\tilde{h})\hat{z}}$  for ( $\hat{z} < 0$ ) and by  $e^{(-ik-\tilde{h})\hat{z}}$  for ( $\hat{z} > 0$ ), where “ $\tilde{h}$ ” is a very small positive number. Since  $\tilde{h}$  is so small that the only effect is the integral along the two straight line segments being zero, we obtain

$$\oint_c f_1(\varrho) d\varrho = [1 - e^{-k\pi}] I_1(k). \quad (\text{A3})$$

From the definition of residue theorem, we have

$$\oint_c f_1(\varrho) d\varrho = 2\pi i \text{Res} f_1(\varrho_0). \quad (\text{A4})$$

Using the standard procedure, we obtain the value of the residue corresponding to the pole  $\varrho = \frac{i\pi}{2}$  as

$$\text{Res} f_1(\varrho) = \lim(\varrho - \varrho_0) = e^{\frac{k\pi}{2}}. \quad (\text{A5})$$

Substituting the residue found in Eq. (A5) into (A4) and then comparing with (A3), we get

$$I_1(k) = \oint_c e^{-ik\varrho} \tanh \varrho d\varrho = \frac{-i\pi}{\sinh(\frac{k\pi}{2})}. \quad (\text{A6})$$

Starting from Eq. (A6), using integration by parts, we obtain the following formulas successively:

$$I_2(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \text{sech}^2 \hat{z} d\hat{z} = \frac{k\pi}{\sinh(\frac{k\pi}{2})}, \quad (\text{A7})$$

$$I_3(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \tanh \hat{z} \text{sech}^2 \hat{z} d\hat{z} = \frac{-i\pi k^2}{2 \sinh(\frac{k\pi}{2})}, \quad (\text{A8})$$

$$I_4(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \tanh^2 \hat{z} \text{sech}^2 \hat{z} d\hat{z} = \frac{-\pi k(2 - k^2)}{6 \sinh(\frac{k\pi}{2})}, \quad (\text{A9})$$

$$I_5(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \text{sech}^4 \hat{z} d\hat{z} = \frac{\pi k(k^2 + 4)}{6 \sinh(\frac{k\pi}{2})}, \quad (\text{A10})$$

$$I_6(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \cos \hat{z} \text{sech}^2 \hat{z} d\hat{z} = \frac{2\pi \cosh(\frac{k\pi}{2}) \cosh(\frac{\pi}{2})}{\cosh(k\pi) + \cosh \pi}, \quad (\text{A11})$$

$$I_7(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \cos \hat{z} \text{sech}^3 \hat{z} d\hat{z} = \frac{\pi [(2 + k) \cosh(\frac{k\pi}{2}) \cosh(\frac{\pi}{2}) - 2k \sinh(\frac{k\pi}{2}) \sinh(\frac{\pi}{2})]}{\cosh(k\pi) + \cosh \pi}, \quad (\text{A12})$$

$$\begin{aligned}
 I_8(k) &= \int_{-\infty}^{\infty} e^{-ik\hat{z}} \cos \hat{z} \tanh \hat{z} \operatorname{sech} \hat{z} d\hat{z} \\
 &= \frac{2\pi[-ik \cosh(\frac{k\pi}{2}) \cosh(\frac{\pi}{2}) + i \sinh(\frac{k\pi}{2}) \sinh(\frac{\pi}{2})]}{\cosh(k\pi) + \cosh \pi},
 \end{aligned}
 \tag{A13}$$

$$I_9(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \operatorname{sech} \hat{z} d\hat{z} = \frac{-\pi}{\cosh(\frac{\pi k}{2})}, \tag{A14}$$

$$I_{10}(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \tanh \hat{z} \operatorname{sech} \hat{z} d\hat{z} = \frac{-ik\pi}{\cosh(\frac{\pi k}{2})}, \tag{A15}$$

$$I_{11}(k) = \int_{-\infty}^{\infty} e^{-ik\hat{z}} \tanh^2 \hat{z} \operatorname{sech} \hat{z} d\hat{z} = \frac{\pi}{2 \cosh(\frac{\pi k}{2})}. \tag{A16}$$

- 
- [1] L. Stryer, *Biochemistry*, 4th ed. (W.H. Freeman, New York, 1995).
- [2] L. V. Yakushevich, *Nonlinear Physics of DNA* (Wiley-VCH, Berlin, 2004).
- [3] R. V. Polozov and L. V. Yakushevich, *J. Theor. Biol.* **130**, 423 (1988).
- [4] J. A. Gonzalez and M. M. Landrove, *Phys. Letts. A* **292**, 256 (2002).
- [5] K. Forinash, M. Peyrard, and B. Malomed, *Phys. Rev. E* **49**, 3400 (1994).
- [6] J. A. D. Wattis, S. A. Harris, C. R. Grindon, and C. A. Laughton, *Phys. Rev. E* **63**, 061903 (2001).
- [7] J. Cuevas, F. Palmero, J. F. R. Archilla, and F. R. Romero, *Phys. Letts. A* **299**, 221 (2002).
- [8] A. Campa, *Phys. Rev. E* **63**, 021901 (2001).
- [9] G. Kalosakas, K. Q. Rasmussen, and A. R. Bishop, *Synth. Met.* **141**, 93 (2004).
- [10] S. Takeno, *Phys. Lett. A* **339**, 352 (2005).
- [11] H. Urabe, Y. Tominaga, and K. Kubota, *J. Chem. Phys.* **78**, 5937 (1983).
- [12] C. Mandal, N. R. Kallenbach, and S. W. Englander, *J. Mol. Biol.* **135**, 391 (1979).
- [13] G. S. Edwards, C. C. Davis, J. D. Saffer, and M. L. Swicord, *Phys. Rev. Lett.* **53**, 1284 (1984).
- [14] M. E. Davis and L. L. Van Zandt, *Phys. Rev. A* **37**, 888 (1988).
- [15] Ch. T. Zhang, *Phys. Rev. A* **40**, 2148 (1989).
- [16] P. R. Selvin, D. N. Cook, N. G. Pon, W. R. Bauer, M. P. Klein, and J. E. Hearst, *Science* **255**, 82 (1992).
- [17] M. D. Barkley and B. H. Zimm, *J. Chem. Phys.* **70**, 2991 (1979).
- [18] V. K. Fedyanin and L. V. Yakushevich, *Stud. Biophys.* **103**, 171 (1984).
- [19] W. K. Schroll, V. V. Prabhu, E. W. Prohofsky, and L. L. Van Zandt, *Biopolymers* **28**, 1189 (1989).
- [20] K. F. Baverstock and R. B. Cundall, *Nature (London)* **332**, 312 (1988).
- [21] S. Cocco, R. Monasson, and J. F. Marko, *Phys. Rev. E* **65**, 041907 (2002).
- [22] S. Cocco, R. Monasson, and J. F. Marko, *Phys. Rev. E* **66**, 051914 (2002).
- [23] S. W. Englander, N. R. Kallenbach, A. J. Heeger, J. A. Krumhansl, and A. Litwin, *Proc. Natl. Acad. Sci. USA* **77**, 7222 (1980).
- [24] S. Yomosa, *Phys. Rev. A* **27**, 2120 (1983).
- [25] S. Yomosa, *Phys. Rev. A* **30**, 474 (1984).
- [26] S. Takeno and S. Homma, *Prog. Theor. Phys.* **70**, 308 (1983).
- [27] S. Takeno and S. Homma, *Prog. Theor. Phys.* **72**, 679 (1984).
- [28] M. Peyrard and A. R. Bishop, *Phys. Rev. Lett.* **62**, 2755 (1989).
- [29] P. L. Christiansen, P. C. Lomdahl, and V. Muto, *Nonlinearity* **4**, 477 (1991).
- [30] M. Barbi, S. Cocco, and M. Peyrard, *Phys. Lett. A* **253**, 358 (1999).
- [31] J. A. Krumhansl and D. M. Alexander, in *Structure and Dynamics: Nucleic Acids and Proteins*, edited by E. Clementi and R. H. Sarma (Adenine, New York, 1983).
- [32] G. Gaeta, *Phys. Lett. A* **143**, 227 (1990).
- [33] G. Gaeta, *Phys. Lett. A* **168**, 383 (1992).
- [34] G. Gaeta, *Phys. Rev. E* **74**, 021921 (2006).
- [35] M. V. Sataric and J. A. Tuszynski, *Phys. Rev. E* **65**, 051901 (2002).
- [36] M. V. Sataric, L. Matsson, and J. A. Tuszynski, *Phys. Rev. E* **74**, 051902 (2006).
- [37] T. Dauxios, *Phys. Lett. A* **159**, 390 (1991).
- [38] M. Cadoni, R. De Leo, and G. Gaeta, *Phys. Rev. E* **75**, 021919 (2007).
- [39] M. Salerno, *Phys. Rev. A* **44**, 5292 (1991).
- [40] M. Peyrard, S. Cuesta Lopez, and G. James, *J. Biol. Phys.* **35**, 73 (2009).
- [41] M. Salerno, *Phys. Letts. A* **167**, 49 (1992).
- [42] M. Gueron, M. Kochoyan, and J. J. Leroy, *Nature (London)* **328**, 89 (1987).
- [43] M. Daniel and V. Vasumathi, *Physica D* **237**, 10 (2007).
- [44] M. Daniel and V. Vasumathi, *Phys. Lett. A* **372**, 5144 (2008).
- [45] M. Daniel and V. Vasumathi, *Phys. Rev. E* **79**, 012901 (2009).
- [46] V. Vasumathi and M. Daniel, *Phys. Lett. A* **373**, 76 (2008).
- [47] V. Vasumathi and M. Daniel, *Phys. Rev. E* **80**, 061904 (2009).
- [48] M. Daniel and J. Beula, *Phys. Rev. B* **77**, 144416 (2008).
- [49] R. M. White, *Quantum Theory of Magnetism* (Springer, New York, 1982).
- [50] P. G. de Gennes, *Physics of Liquid Crystals* (Clarendon, Oxford, 1974).
- [51] G. Altan Bonnet, A. Libchaber, and O. Krichevsky, *Phys. Rev. Lett.* **90**, 138101 (2003).
- [52] A. Hanke and R. Metzlar, *J. Phys. A* **36**, L473 (2003).
- [53] R. Metzlar, T. Ambjornsson, A. Hanke, and H. C. Fogedby, *J. Phys. Condens. Matter* **21**, 034111 (2009).
- [54] D. Poland and H. A. Scheraga, *J. Chem. Phys.* **45**, 1456 (1966).
- [55] Wu. Lian-Ao, S. S. Wu, and D. Segal, *Phys. Rev. E* **79**, 061901 (2009).
- [56] E. Villagran, J. Bernal, and M. Aguero, *Revista Mexicana de Fisica* **51**, 580 (2005).
- [57] D. Hennig, *Eur. Phys. J. B* **37**, 391 (2004).
- [58] J. Yan and J. F. Marko, *Phys. Rev. Lett.* **93**, 108108 (2004).

- [59] P. Ranjith, P. B. Sunil Kumar, and G. I. Menon, *Phys. Rev. Lett.* **94**, 138102 (2005).
- [60] M. Hisakado and M. Wadati, *J. Phys. Soc. Jpn.* **64**, 1098 (1995).
- [61] J. Ladik and J. Cizek, *Int. J. Quantum Chem.* **26**, 955 (1984).
- [62] E. Cubero, E. C. Sherer, F. J. Luque, M. Orozco, and C. A. Laughton, *J. Am. Chem. Soc.* **121**, 8653 (1999).
- [63] G.-f. Zhou and C.-T. Zhang, *Phys. Scr.* **43**, 347 (1991).
- [64] M. J. Ablowitz, D. J. Kaup, A. C. Newell, and H. Segur, *Stud. Appl. Math.* **53**, 249 (1974).
- [65] M. Remoissenet, *Phys. Rev. B* **33**, 2386 (1986).
- [66] J. Yan, Y. Tang, G. Zhou, and Z. Chen, *Phys. Rev. E* **58**, 1064 (1998).
- [67] V. E. Zakharov and A. B. Shabat, *Zh. Eksp. Teor. Fiz.* **61**, 118 (1971) [*Sov. Phys. JETP* **34**, 62 (1972)].
- [68] G. L. Lamb, *Elements of Soliton Theory* (Wiley, New York, 1980).
- [69] P. G. Drazin and R. S. Johnson, *Solitons: An Introduction* (Cambridge University Press, Cambridge, 1989).
- [70] D. J. Kaup, *Phys. Rev. B* **27**, 6787 (1983).
- [71] D. J. Kaup, *Phys. Rev. B* **29**, 1072 (1984).
- [72] J. P. Keener and D. W. Mclaughlin, *J. Math. Phys.* **18**, 2008 (1977).
- [73] J. P. Keener and D. W. Mclaughlin, *Phys. Rev. A* **16**, 777 (1977).
- [74] R. L. Herman, *J. Phys. A* **23**, 1063 (1990).
- [75] R. L. Herman, *J. Phys. A* **23**, 2327 (1990).
- [76] A. H. Nayfeh and D. T. Mook, *Nonlinear Oscillations* (Wiley, New York, 1979).
- [77] P. Hagedorn, *Nonlinear Oscillations* (Oxford Science, Oxford, 1988).
- [78] Y. Tang and W. Wang, *Phys. Rev. E* **62**, 8842 (2000).
- [79] E. Kreyzig, *Advanced Engineering Mathematics* (Wiley, New York, 2002).
- [80] A. C. Scott, *Phys. Rev. A* **31**, 3518 (1985).
- [81] E. Evans, J. Fellows, A. Coffey, and R. D. Wood, *EMBO J.* **16**, 625 (1997).
- [82] P. R. Bianco and S. C. Kowalczykowski, *Nature (Nature)* **405**, 308 (2000).
- [83] J. A. H. Cognet, Y. Boulard, and G. V. Fazakerley, *J. Mol. Biol.* **246**, 209 (1995).
- [84] C. R. Calladine, *J. Mol. Biol.* **161**, 343 (1982).
- [85] A. C. Scott, *Phys. Lett. A* **86**, 60 (1981).
- [86] J. X. Xiao, J. T. Lin, and G. X. Zhang, *J. Phys. A* **20**, 2425 (1990).
- [87] K. C. Chou and B. Mao, *Biopolymers* **27**, 1795 (1988).