Perturbed soliton-like molecular excitations in a deformed DNA chain

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Abstract

We study the nonlinear dynamics of a deformed Deoxyribonucleic acid (DNA) molecular chain which is governed by a perturbed sine-Gordon equation coupled with a linear wave equation representing the lattice deformation. The DNA chain considered here is assumed to be deformed periodically which is the energetically favourable configuration, and the periodic deformation is due to the repulsive force between base pairs, stress in the helical backbones and due to the elastic strain force in both the strands. A multiple scale soliton perturbation analysis is carried out to solve the perturbed sine-Gordon equation and the resultant perturbed kink and antikink solitons represent open state configuration with small fluctuation. The perturbation due to periodic deformation of the lattice changes the velocity of the soliton. However, the width of the soliton remains unchanged.

Key words: DNA, Phonon coupling, Soliton Perturbation Theory
1 Introduction

Deoxyribonucleic acid (DNA) plays an important role in the conservation and transformation of genetic information in biological systems [1]. Opening of base pairs in DNA double helix is related to functions like transcription and replication. Base pair opening via nonlinear molecular excitations has been understood by several authors [2,3,4,5,6,7,8,9,10] by proposing different models. Among them the models proposed and used by Yomosa [3,4], as well as by Takeno and Homma [5,6] were based on rotation of bases in a plane normal to the helical axis of DNA, and the nonlinear molecular excitations were governed by kink-antikink solitons. Following Takeno and Homma, recently several authors [11,12,13,14,15,16,17,18] studied soliton-like molecular excitations in DNA by taking into account the rotation of bases. In all the above studies, both the strands of the DNA double helix were considered 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 and also, the main-chain torsion angle indicates that there are elastic strain forces in both the strands [19,20]. The dynamics of this non-rigidity of the strands gives rise to phonons which also play an important role in energy

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transfer in biological systems. In a different context, Davydov [21,22] proposed a model for energy transfer in alpha helix protein molecules and he found that the propagation of molecular vibrations induce longitudinal sound waves (phonons), which in turn provides a potential well that prevents vibrational dispersion, and this coupled excitation propagates as a soliton without loss of energy along hydrogen bonding spines of the alpha helical protein. Thus, the study of nonlinear molecular excitations in DNA double helical chains coupled with phonons in strands or in other words the influence of non-rigidity of the strands in molecular excitations has become an important task which requires a detailed investigation. In this direction, recently, Xiao and his co-workers [23] studied the influence of longitudinal vibration on the soliton excitations in DNA double helix by considering the dynamic plane base rotator model of Takeno-Homma [5,6], and by including the longitudinal vibration and its coupling with hydrogen bonds and stacking. It was shown that the dynamics in this case is governed by a perturbed sine-Gordon equation in the continuum limit, which upon solving using the method of successive approximation by iterations gives soliton under first order approximation, which shows that the effect of longitudinal vibration of the lattice on soliton is small. However, they failed to find the variation of the soliton parameters such as velocity and width explicitly during propagation under iterations. Therefore, in the present paper, we study the nonlinear molecular excitations in DNA double helix with non-rigid elastic strands, by solving the dynamical equation using direct soliton perturbation theory, which provides the variation of velocity and width of
the soliton under perturbation in explicit analytical terms. The paper is organised as follows. In section 2, we consider the model Hamiltonian for the above DNA double helix and derive the dynamical equations. In the continuum limit, the dynamical equations reduce to a perturbed sine-Gordon equation coupled with a linear wave equation representing the longitudinal lattice vibration and this is treated in section 3. In section 4, a multiple scale soliton perturbation theory is developed to investigate the effect of lattice deformation on the open state configuration of DNA represented in terms of kink-antikink solitons of the sine-Gordon equation. The results are concluded in section 5.

2 Model and dynamical equations

We consider the B-form of a DNA double helix with flexible strands, and investigate the nonlinear molecular excitations by considering a plane-base rotator model. In Fig. (1a) we have presented a sketch of the DNA double helix with z-axis parallel to the helical axis. In the figure, $S$ and $S'$ represent the two complementary strands in the DNA double helix, and each arrow in the figure represents the direction of the bases attached to the strand and the dots between arrows represent the net hydrogen bonding effect between the complementary bases. In Fig. 1(b), we present a horizontal projection of the $n^{th}$ base pair in the xy-plane in which $Q_n$ and $Q'_n$ denote the tips of the $n^{th}$ bases, and $P_n$ and $P'_n$ represent the points where the $n^{th}$ bases are attached.
to the strands $S$ and $S'$ respectively.

The conformation and stability of DNA double helical molecular chains are mainly determined by the stacking between the adjacent bases in the strands and the hydrogen bonds between the complementary bases. The Hamiltonian under plane base rotator model involving stacking and hydrogen bonds in terms of the rotational angles $\phi_n$ and $\phi'_n$ of the $n^{th}$ bases (see Fig. 1(b)) in the case of rigid strands as proposed by Yomosa [3,4] and further developed by Takeno and Homma [5,6] is written as

$$H_r = \sum_n \left[ \frac{I}{2}(\dot{\phi}_n^2 + \dot{\phi}'_n^2) + J[2 - \cos(\phi_{n+1} - \phi_n) - \cos(\phi'_{n+1} - \phi'_n)] - \alpha[1 - \cos(\phi_n - \phi'_n)] \right],$$

(1)

where $I$ is the moment of inertia of the nucleotides around the axes at $P_n$ and $P'_n$ in the strands $S$ and $S'$ respectively and thus, the first two terms represent the kinetic energies of the rotational motion of the $n^{th}$ nucleotide bases. In Eq. (1) overdot represents time derivative. Further, the terms proportional to $J$ in Hamiltonian (1) represent the stacking energy between the $n^{th}$ base and its nearest neighbours in the strands $S$ and $S'$ and $\alpha$ represents a measure of the interstrand interaction or hydrogen bonding energy between the complementary bases respectively. Pople’s formula in which the mean energy of the distorted hydrogen bonds is approximately represented in the above form [24]. However, in nature DNA strands are not rigid but flexible and hence,
we assume that the two strands deform elastically and the resultant phonons couple to the stacking and hydrogen bonds. Hence, the part of the Hamiltonian corresponding to the phonon energy, and the energy due to its coupling with the stacking and hydrogen bonds is written as

\[ H_{ph} = \sum_n \left[ \frac{p_n^2}{2M} + \frac{p_n'^2}{2M} + K[(y_{n+1} - y_n)^2 + (y_{n+1}' - y_n')^2] \right], \quad (2a) \]

\[ H_{r-ph} = \sum_n \left[ \beta\{(y_{n+1} - y_n)[1 - \cos(\phi_{n+1} - \phi_n)] + (y_{n+1}' - y_n') \right. \\
\left. \times [1 - \cos(\phi_{n+1}' - \phi_n')]\} - \gamma(y_{n+1} - y_{n-1})[1 - \cos(\phi_n - \phi_n')] \right] \quad (2b) \]

where \( p_n = M\dot{y}_n \) and \( p_n' = M\dot{y}_n' \). In Eqs. (2), \( y_n \) and \( y_n' \) represent the longitudinal displacements of the \( n^{th} \) nucleotides from the equilibrium position in the two strands, and \( M \) is the uniform mass of the nucleotide. \( K \) is the longitudinal elastic constant along the double helical main chain. The stacking energy depends on the distance between the \( n^{th} \) and \( (n+1)^{th} \) base, and the strength of the hydrogen bonds depends symmetrically on the distance between the \( (n-1)^{th} \) and \( (n+1)^{th} \) bases. Thus, \( \beta \) and \( \gamma \) measure the coupling strengths between phonon and stacking as well as hydrogen bonds respectively. The interaction Hamiltonian \( H_{r-ph} \) in Eq.(2b) is chosen to represent the change in stacking energy and hydrogen bonds energy caused by the change in the displacement of the nucleotides along the two strands. As we are going to study the dynamics in the low temperature and long wavelength limit, it is appropriate to consider linear coupling of phonon to the stacking and hydrogen bonds. Now, using the Hamiltonians (1), (2a) and (2b), the total Hamiltonian
H for the system is written as \( H = H_r + H_{ph} + H_{r-ph} \) and the corresponding Hamilton’s equations of motion take the form

\[
\begin{align*}
I \ddot{\phi}_n &= [J + \beta(y_{n+1} - y_n)] \sin(\phi_{n+1} - \phi_n) - [J + \beta(y_n - y_{n-1})] \\
&\quad \times \sin(\phi_n - \phi_{n-1}) + [\alpha + \gamma(y_{n+1} - y_n)] \sin(\phi_n - \phi'_n), \\
I \ddot{\phi}'_n &= [J + \beta(y_{n+1} - y_n)] \sin(\phi'_{n+1} - \phi'_n) - [J + \beta(y_n - y_{n-1})] \\
&\quad \times \sin(\phi'_n - \phi'_{n-1}) + [\alpha + \gamma(y_{n+1} - y_n)] \sin(\phi'_n - \phi_n), \\
M \ddot{y}_n &= 2K(y_{n+1} - 2y_n + y_{n-1}) - \beta[\cos(\phi_{n+1} - \phi_n) - \cos(\phi_n - \phi_{n-1})] \\
&\quad + \gamma[\cos(\phi_{n+1} - \phi'_n) - \cos(\phi'_n - \phi'_{n-1})], \\
M \ddot{y}'_n &= 2K(y'_{n+1} - 2y'_n + y'_{n-1}) - \beta[\cos(\phi'_{n+1} - \phi'_n) - \cos(\phi'_n - \phi'_{n-1})] \\
&\quad + \gamma[\cos(\phi'_{n+1} - \phi''_{n+1}) - \cos(\phi''_{n-1} - \phi'_{n-1})].
\end{align*}
\] (3a)

Eqs. (3a-3d) describe the dynamics of DNA with deformable strands at the discrete level by considering the dominant angular rotation of bases in a plane normal to the helical axis of the DNA, and ignoring all other small motions of the bases combined with longitudinal motion of the nucleotides.

3 Soliton and base pair opening

It is expected that the difference in the angular rotation of bases with respect to neighbouring bases along the two strands in DNA namely \((\phi_{n\pm1} - \phi_n)\) and \((\phi'_{n\pm1} - \phi'_n)\) are small \([5,6]\). Also, as the length of the DNA chain is very large due to the presence of large number of bases compared to the distance between the neighbouring base pairs, we make a continuum approximation by introducing two fields of rotational angles \(\phi_n(t) \rightarrow \phi(z,t), \ \phi'_n(t) \rightarrow \phi'(z,t)\)
and two fields of longitudinal displacement $y_n(t) \to y(z,t)$ and $y'_n(t) \to y'(z,t)$ along the strands where $z = na$ with $l$, the lattice parameter. Also, we make the expansions for $\phi_{n\pm 1} = \phi(z,t) \pm a \frac{\partial \phi}{\partial z} + \frac{a^2}{2l} \frac{\partial^2 \phi}{\partial z^2} \pm \frac{a^3}{3l^2} \frac{\partial^3 \phi}{\partial z^3} + \ldots$, and similar expansions for $\phi'_{n\pm 1}$, $y_{n\pm 1}$ and $y'_{n\pm 1}$. Thus, in the continuum limit under small angular rotation of bases Eqs. (3) upto $O(a^3)$ become

\begin{align}
\phi_{\hat{t}\hat{z}} &= \phi_{zz} - \frac{1}{2} \sin(\phi - \phi') + \epsilon [\beta (y_z \phi_z)_z + \frac{\hat{\gamma}}{2} y_z \sin(\phi - \phi')], \\
\phi'_{\hat{t}\hat{z}} &= \phi'_{zz} - \frac{1}{2} \sin(\phi' - \phi) + \epsilon [\beta (y'_{z} \phi'_{z})_z + \frac{\hat{\gamma}}{2} y'_{z} \sin(\phi' - \phi)], \\
y_{\hat{t}\hat{z}} &= v^2 y_{zz}, \\
y'_{\hat{t}\hat{z}} &= v^2 y'_{zz},
\end{align}

where $\epsilon = \frac{a}{J}$, $v^2 = \frac{2KI}{JM}$ and the suffices $\hat{t}$ and $\hat{z}$ in Eqs.(4) represent partial time and spatial derivatives and the rescaled $a$ is dimensionless. While writing the above equations we have chosen $\alpha = -\frac{1}{2} Ja^2$ and also, the parameter $\gamma$ is rescaled as $\hat{\gamma} = \frac{a^2 \gamma}{4}$. Further, before writing Eqs. (4a-d), we have divided the full equations by $Ja^2$ and rescaled the time variable as $\hat{t} = \sqrt{Ja^2} t$. It is more convenient to describe the transverse motion of the bases in DNA strands in terms of the center of mass co-ordinates. For this, we rewrite Eqs. (4) by subtracting and adding the first two and the last two equations respectively.

Further, to commence the open state configuration of DNA, the two complementary bases are expected to rotate in opposite directions and both the strands are assumed to vibrate in the same direction so that $\phi' = -\phi$ and $y' = y$. Under these conditions, we obtain
\[
\Psi_{tt} - \Psi_{zz} + \sin \Psi = \epsilon [\beta (y_z \Psi_z)_z + \gamma y_z \sin \Psi],
\]

\[
y_{tt} - v^2 y_{zz} = 0,
\]

where \( \Psi = 2\phi \). Eqs. (5) describe the dynamics of bases under a plane-base rotator model of DNA double helical chain with the deformed strands. The terms proportional to \( \beta \) and \( \gamma \) in the right hand side of Eq. (5a) represent the coupling of phonon to the stacking and hydrogen bonds respectively.

When \( \epsilon = 0 \), Eqs. (5a) and (5b) are decoupled, and Eq. (5a) reduces to the completely integrable sine-Gordon equation which admits kink and antikink-type of soliton solutions, and hence we call Eq. (5a) in its present form as a perturbed sine-Gordon equation. The integrable sine-Gordon equation (\( \epsilon = 0 \)) was originally solved for N-soliton solutions using the most celebrated Inverse Scattering Transform (IST) method by Ablowitz and his co-workers [25]. The kink and antikink one soliton solution of the integrable sine-Gordon equation (Eq.(5a) when \( \epsilon = 0 \)) can be written as

\[
\Psi(z, \hat{t}) = 4\arctan \exp[\pm m(z - v\hat{t})], \quad m^{-1} = \sqrt{1 - v^2}.
\]

In Eq.(6), while the upper sign corresponds to kink soliton, the lower sign represents the antikink soliton. Here, \( v \) and \( m^{-1} \) are real parameters that determine the velocity and width of the soliton respectively. The kink and antikink one soliton solutions as given above are depicted in Figs. 2(a) and 2(b). The kink-antikink soliton of the sine-Gordon equation describes an open
state in DNA double helix which is schematically represented in Fig. 2(c). In this figure the base pairs are found to open locally in the form of kink-antikink shape in each strand and the opening is found to propagate along the direction of the helical axis.

Eq. (5b) is the well known one-dimensional linear wave equation which admits wave solution in the form $y = f(z - vt) + g(z + vt)$, where $f$ and $g$ are arbitrary functions. Now, the problem boils down to solving the perturbed sine-Gordon equation (5a) after using the wave solution '$y'$ obtained by solving Eq. (5b).

4 Effect of elastic deformation of strands on base pair opening

4.1 A perturbation approach

When the phonon due to elastic deformation of the strands is coupled to the DNA molecular excitations, it is expected to perturb the kink and antikink solitons in DNA which correspond to the open state configuration. It is further expected that the perturbation due to phonon coupling modifies the shape, width and velocity of the soliton as it propagates along the helical chain. In order to understand this, we solve Eq. (5a) using a suitable perturbation method. One of the most powerful techniques in dealing with perturbed soliton is the soliton perturbation theory which is based on the IST method. However, as the method is very sophisticated it is very difficult to use the same in several
cases. In view of this, many authors used different types of direct methods to study soliton perturbation (see for e.g. refs. [26,27,28,29,30]). In the present paper, we use one such direct perturbation method to solve the perturbed sine-Gordon equation (5a) to understand the effect of phonon interaction on the open state configuration of DNA, which is also dealt in reference [30] in a different context, and also by the present authors recently while studying the nonlinear molecular excitations in an inhomogeneous DNA [17]. The procedure we adapt here is based on the derivative expansion method to linearize the perturbed sine-Gordon equation in the coordinate frame attached to the moving frame. The parameters of the kink-antikink soliton are assumed to depend on a slow time scale in order to eliminate the secular terms. The linearized equations will be solved using the method of separation of variables which will be ultimately related to a generalized eigenvalue problem, the eigenfunctions of which form the bases of the perturbed solution. In the following we use the above approach to find the perturbed soliton solution of Eq. (5a).

4.2 Linearization of the perturbed sine-Gordon equation

In order to study the effect of perturbation due to phonon interaction on the soliton, the time variable \( \hat{t} \) is transformed into several variables as \( t_n = \epsilon^n \hat{t} \), where \( n=0, 1, 2, \ldots \) and \( \epsilon \) is a very small parameter. In view of this, the time derivative and \( \Psi \) in Eq. (5a) are replaced by the expansions

\[
\frac{\partial}{\partial \hat{t}} = \frac{\partial}{\partial t_0} + \epsilon \frac{\partial}{\partial t_1} + \epsilon^2 \frac{\partial}{\partial t_2} + \ldots \]

and

\[
\Psi = \Psi^{(0)} + \epsilon \Psi^{(1)} + \epsilon^2 \Psi^{(2)} + \ldots
\]

and we equate the coefficients of
different powers of $\epsilon$. Thus at $O(\epsilon^0)$ we obtain

$$
\Psi^{(0)}_{zt_0} - \Psi^{(0)}_{zz} + \sin \Psi^{(0)} = 0,
$$

(7)

for which the one soliton solution takes the form $\Psi^{(0)}(z, t_0) = 4\text{arc tan} \exp \zeta$, $\zeta = \pm m_0(z - \xi)$, $\xi_{t_0} = v_0$, where $v_0$ is the velocity of the soliton in the $t_0$ time scale.

Due to perturbation, the soliton parameters namely $m$ and $\xi$ are now treated as functions of the slow time variables $t_0, t_1, t_2, \ldots$. However, $m$ is treated as independent of $t_0$. The equation at $O(\epsilon^1)$ is of the form

$$
\Psi^{(1)}_{\tau \zeta} - \Psi^{(1)}_{\zeta \zeta} + (1 - 2\text{sech}^2 \zeta) \Psi^{(1)} = F^{(1)}(\zeta, \tau),
$$

(8)

where

$$
F^{(1)} = 2\beta [y_\zeta \text{sech} \zeta]_\zeta + 2b\gamma y_\zeta \tanh \zeta \text{sech} \zeta
\vspace{1em}
+ 4v_0 \left[ m_{t_1} + (m^2 \xi_{t_1} - \zeta m_{t_1}) \tanh \zeta \right] \text{sech} \zeta.
$$

(9)

While writing the above equation we have replaced $\sin \Psi^{(0)}$ by $2b \tanh \zeta \text{sech} \zeta$, where $b = \pm 1$, which can be derived using the unperturbed solution given below Eq. (7), and we have also used the transformation $\hat{\zeta} = m(z - vt_0)$ and $\hat{t}_0 = t_0$ to represent everything in a co-ordinate system that is moving with the soliton. Further, we have used another set of transformations given by $\tau = \frac{\hat{t}_0}{2m} - \frac{(1 + v)\hat{\zeta}}{2}$ and $\zeta = \hat{\zeta}$ for our later convenience.

The solution of Eq. (8) is searched by assuming $\Psi^{(1)}(\zeta, \tau) = X(\zeta)T(\tau)$ and
$$F^{(1)}(\zeta, \tau) = X_\zeta(\zeta)H(\tau).$$ Substituting the above in Eq. (8), we obtain

$$\frac{1}{X\zeta}[X_{\zeta\zeta} + (2\text{sech}^2\zeta - 1)X] = \frac{1}{T}[T_\tau - H(\tau)]$$

(10)

In Eq. (10), while the left hand side is independent of \(\tau\), the right hand side is independent of the variable \(\zeta\). Therefore, we can equate the left and right hand sides of Eq. (10) to a constant, say \(\lambda_0\) and write

$$X_{\zeta\zeta} + (2\text{sech}^2\zeta - 1)X = \lambda_0 X_\zeta, \quad T_\tau - \lambda_0 T = H(\tau).$$

(11)

Thus, the problem of constructing the perturbed soliton at this moment turns out to be solving Eq. (11) by constructing the eigenfunctions and finding the eigenvalues. The first part of Eq. (11) is a generalized eigenvalue problem, which is not a self-adjoint eigenvalue problem and differs from the normal eigenvalue problem, with \(X_\zeta\) in the right hand side instead of \(X\). For solving the eigenvalue problem, we consider it in a more general form by writing

$$L_1X = \lambda \tilde{X}, \quad L_1 = \partial_{\zeta\zeta} + 2\text{sech}^2\zeta - 1,$$

(12)

where \(\lambda\) is the eigenvalue. In order to solve Eq.(12) for \(X\), we also consider the following eigenvalue problem.

$$L_2\tilde{X} = \lambda X,$$

(13)
where $L_2$ is to be determined. Now, by combining the above two eigenvalue problems we get $L_2L_1X = \lambda^2X$, $L_1L_2\tilde{X} = \lambda^2\tilde{X}$. From these expressions we conclude that $L_1L_2$ is the adjoint of $L_2L_1$ and also $X$ and $\tilde{X}$ are expected to be adjoint eigenfunctions. Hence, by solving the coupled eigenvalue problem we can find the eigenfunction $X$. Here $L_1$ is known and is given in Eq. (12), but the operator $L_2$ is still unknown. So, by experience we choose $L_2 = \partial_{\zeta\zeta} + 6\text{sech}^2\zeta - 1$.

Now, in order to find the eigenfunctions by solving Eqs. (12) and (13) we choose the eigenfunctions as

$$X(\zeta, k) = p(\zeta, k)e^{ik\zeta}, \quad \tilde{X}(\zeta, k) = q(\zeta, k)e^{ik\zeta},$$

(14)

where $k$ is the propagation constant. On substituting the above in Eqs. (12) and (13) in the asymptotic limit, we obtain the eigenvalue as $\lambda = -(1 + k^2)$.

In order to find the eigenfunctions, we expand $p(\zeta, k)$ and $q(\zeta, k)$ as

$$p(\zeta, k) = p_0 + p_1 \frac{\sinh \zeta}{\cosh \zeta} + p_2 \frac{1}{\cosh^2 \zeta} + p_3 \frac{\sinh \zeta}{\cosh^3 \zeta} + p_4 \frac{1}{\cosh^4 \zeta} + ..., \quad (15a)$$

$$q(\zeta, k) = q_0 + q_1 \frac{\sinh \zeta}{\cosh \zeta} + q_2 \frac{1}{\cosh^2 \zeta} + q_3 \frac{\sinh \zeta}{\cosh^3 \zeta} + q_4 \frac{1}{\cosh^4 \zeta} + ..., \quad (15b)$$

where $p_j$ and $q_j$, $j=0,1,2,...$ are functions of $k$ to be determined. Substituting Eqs. (14), (15a) and (15b) in Eqs. (12) and (13) and collecting the coefficients of $1$, $\frac{\sinh \zeta}{\cosh \zeta}$, $\frac{1}{\cosh^2 \zeta}$, ... we get a set of simultaneous equations. On solving those equations by assuming $p_j = q_j = 0$ for $j \geq 3$, we obtain the eigenfunctions as
\[ X(\zeta, k) = \frac{(1 - k^2 - 2ik \tanh \zeta)}{\sqrt{2\pi(1 + k^2)}} e^{ik\zeta}, \]  
\[ \tilde{X}(\zeta, k) = \frac{(1 - k^2 - 2ik \tanh \zeta - 2\text{sech}^2 \zeta)}{\sqrt{2\pi(1 + k^2)}} e^{ik\zeta}. \]

On comparing Eqs. (16a) and (16b) we can write \( \tilde{X}(\zeta, k) = \frac{X(\zeta, k)}{ik} \). Now, using this in the right hand side of Eq. (12) and comparing the resultant equation with Eq. (11), we obtain \( \lambda_0 = \frac{\text{i}(1+k^2)}{k} \).

The second part of Eq. (11) is a linear inhomogeneous differential equation and it can be solved using known procedures [17]. The solution reads

\[ T(\tau, k) = \frac{1}{i\lambda_0 k(1 + k^2)} \int_{-\infty}^{\infty} d\zeta F^{(1)}(\zeta, \tau) X^*(\zeta, k)(e^{\lambda_0[\tau + \frac{(1+k^2)}{2}\zeta]} - 1), \]  

The first order correction to the soliton can be computed using the following expression.

\[ \Psi^{(1)}(\zeta, \tau) = \int_{-\infty}^{\infty} X(\zeta, k)T(\tau, k)dk + \sum_{j=0,1} X_j(\zeta)T_j(\tau). \]

Here \( X(\zeta, k) \) and \( T(\tau, k) \) are known continuous eigenfunctions which are given in Eqs. (16a) and (17). However, the discrete eigenstates \( X_0, X_1 \) and \( T_0, T_1 \) are unknown. \( X_0 \) and \( X_1 \) are the two discrete eigenstates for the discrete eigenvalue \( \lambda = 0 \) and these states can be found out using the completeness of the continuous eigenfunctions as

\[ X_0(\zeta) = \text{sech} \zeta, \quad X_1(\zeta) = \zeta \text{sech} \zeta. \]
In order to find $T_0$ and $T_1$, we substitute Eq.(18) in Eq.(8) and multiply by $X_0(\zeta)$ and $X_1(\zeta)$ separately, and after using the orthonormal relations, we get

\[
T_1(\tau) = \int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau)X_0(\zeta) d\zeta, \tag{20a}
\]

\[
T_0(\tau) - 2T_1(\tau) = -\int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau)X_1(\zeta) d\zeta. \tag{20b}
\]

As $F^{(1)}(\zeta, \tau)$ given in Eq. (9) does not contain time $\tau$ explicitly, the right hand side of Eqs. (20a) and (20b) are also independent of time, and hence they give rise to secularities and the nonsecular conditions can be written as

\[
\int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau)X_0(\zeta) d\zeta = 0, \tag{21a}
\]

\[
\int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau)X_1(\zeta) d\zeta = 0. \tag{21b}
\]

On substituting the above expressions in Eqs. (20a) and (20b), we choose $T_1(\tau) = 0$ and obtain $T_0(\tau) = C$, where $C$ is a constant, which has to be determined. For this, we integrate Eq. (20b) and obtain

\[
T_0(\tau) = \frac{(1 + v)}{2} \int_{-\infty}^{\infty} \zeta F^{(1)}(\zeta, \tau)X_1(\zeta). \tag{22}
\]

### 4.3 Variation of soliton parameters

In order to find the first order correction, we need to evaluate the eigenstates explicitly for which we need the values of $m_{t_1}$ and $\xi_{t_1}$, which can be found from
the nonsecularity conditions, by substituting the values of $F^{(1)}(\zeta, \tau)$, $X_0(\zeta)$ and $X_1(\zeta)$ respectively from Eqs. (9) and (19). The results give the time evolution of the inverse of the width ($m$) and the velocity ($\xi_1$) of the soliton as

$$m_{t_1} = -\frac{1}{2v_0} \int_{-\infty}^{\infty} \left( \beta [y_\zeta \sech\zeta]_\zeta + b\hat{\gamma} y_\zeta \tanh \zeta \sech\zeta \right) \sech\zeta d\zeta,$$  

(23a)

$$\xi_{t_1} = -\frac{1}{2m^2 v_0} \int_{-\infty}^{\infty} \left( \beta [y_\zeta \sech\zeta]_\zeta + b\hat{\gamma} y_\zeta \tanh \zeta \sech\zeta \right) \zeta \sech\zeta d\zeta.$$  

(23b)

In order to evaluate the integrals found in Eqs. (23a) and (23b) explicitly, we have to substitute the value of '\(y\)' which we have found by solving Eq. (5b). We consider the most general and meaningful wave solution of Eq. (5b) suitable for the problem as the periodic function $y = \sin \zeta$. At this point, it is worth mentioning that Dandoloff and Saxena [31] realized that in the case of an XY-coupled spin chain model which is identifiable with our DNA double helical chain model, the ansatz $cn(\zeta, \kappa)$ with the limit $\kappa \to 0$, energetically favours the periodically deforming spin chain. Hence by substituting $y_\zeta = \cos \zeta$ in Eqs. (23a) and (23b) and on evaluating the integrals we obtain

$$m_{t_1} = 0, \quad \xi_{t_1} = \frac{\pi}{16m^2 v_0} \left[ \pi \beta - b\hat{\gamma}(4 - \pi) \right].$$  

(24)

The parameters $m$ and $\xi$ can be written in terms of the original variable $\hat{t}$ as

$$m = m_0, \quad \xi_\hat{t} \equiv v = v_0 + \frac{\epsilon \pi [\beta \pi - b\hat{\gamma}(4 - \pi)]}{16m^2 v_0},$$  

(25)
where $1/m_0$ is the initial width of the soliton and $v_0$ is the uniform velocity of the soliton in the unperturbed limit. The first of Eq.(25) says that, the width $(m^{-1})$ of the soliton remains constant. However from the second of Eq. (25), we find that the velocity of the soliton gets a correction. It is observed that the correction in velocity depends on the nature of $\beta$ and $\hat{\gamma}$ which can be either positive or negative for $b = \pm 1$. First, we consider the case corresponding to $b = 1$. In this case, when $[\beta\pi - \hat{\gamma}(4 - \pi)] > 0$, the velocity of the soliton gets a positive correction and hence soliton may propagate along the DNA chain without forming a bound state. On the other hand, when $[\beta\pi - \hat{\gamma}(4 - \pi)] \leq 0$, the phonon due to lattice deformation either slows down the soliton or the velocity of the soliton remains unaltered. Finally, if the initial uniform velocity of the soliton before switching on the perturbation due to elastic deformation takes the value $v_0^2 = \frac{\beta\pi - \hat{\gamma}(4 - \pi)}{\beta\pi - \hat{\gamma}(4 - \pi) - 16}$, the soliton is stopped by the deformation.

The stability of the soliton is guaranteed in all the above cases. A similar argument can be made in the case of $b = -1$ with $[\beta\pi - \hat{\gamma}(4 - \pi)]$ replaced by $[\beta\pi + \hat{\gamma}(4 - \pi)]$. Recently Yakushevich et al [14] and Salerno [11] investigated the interaction of soliton with periodic sequence (periodic inhomogeneity), and the results have very close analogy with our results here. It was shown by them that soliton can easily propagate along DNA without forming a bound state. It may also be noted that, Zhang et al [32] obtained similar results in the case of resonant kink impurity interaction and kink scattering in a perturbed sine-Gordon model. In a recent paper, Hwa et al [33], while studying the thermodynamic and dynamic behaviours of twist induced denaturation bubbles in
a long, stretched random sequence of DNA using statistical mechanical models, has shown the localization and delocalization of bubbles along the DNA chain. Finally, Eq. (25) is also similar in form to our recent results of perturbative analysis in the case of an inhomogeneous DNA [17,18]. Thus, we can say that the lattice deformation gives rise to inhomogeneity in the DNA chain.

4.4 First order perturbed soliton

Now, we explicitly construct the first order correction to the one soliton by substituting the values of $X(\zeta, k), X_0(\zeta), X_1(\zeta)$ and $T(\tau, k), T_0(\tau)$ from Eqs. (16a), (19) and (17), (22) and that of $F^{(1)}(\zeta, \tau)$ from Eq. (9) and use the values of $m_{t_1}$ and $\xi_{t_1}$ from Eqs. (24) in Eq. (18) to get

$$
\Psi^{(1)}(\zeta, \hat{t}_0) = \frac{1}{\pi} \left[ \int_{-\infty}^{\infty} \frac{dk}{(1 + k^2)^{3/2}} (1 - k^2 - 2ik \tanh \zeta)e^{ik\zeta} \right.
\times \left. \int_{-\infty}^{\infty} d\zeta' (1 - k^2 + 2ik \tanh \zeta')[\beta \sin \zeta' + \{(\beta - b\hat{\gamma}) \cos \zeta' - \frac{\pi}{8}(\pi \beta - b\hat{\gamma}(4 - \pi))}\tanh \zeta']\sech \zeta' e^{-ik\zeta'} \times \left[ e^{i(1+k^2)}\frac{1}{m}-(1+v)(\zeta-\zeta') - 1 \right] + (1 + v)\sech \zeta \\
\times \int_{-\infty}^{\infty} d\zeta' \zeta'^2 \left[ (\beta \sin \zeta' + \{(\beta - b\hat{\gamma}) \cos \zeta' - \frac{\pi}{8}(\pi \beta - b\hat{\gamma}(4 - \pi))}\tanh \zeta'\right] \sech^2 \zeta'].
$$

We evaluate the integrals in Eq. (26) by finding the values of the residues at poles of different orders using residue theorem (for details see [17,30,34]). After lengthy algebra and some approximations the explicit form of the perturbed
kink (upper sign)-antikink (lower sign) one soliton solution in terms of the original variables is written as

\[
\Psi(z, t_0) \approx 4 \arctan \exp[m_0(z - v_0 t_0)] + \frac{\epsilon \pi [\pi \beta - b \hat{\gamma}(4 - \pi)]}{16m^2v_0} \left[ m(v^2 - 1) + 2vt_0 \right] \sech[m(z - vt_0)].
\] (27)

Having found \(\Psi(z, t_0)\) we find \(\phi(z, t_0)\) using the relation \(\phi = \frac{\Psi}{2}\) and plot the same in Figs. 3(a,b) by choosing \(\beta = \hat{\gamma} = 1, b = -1\) and \(v_0 = 0.4\). From the figures, we observe that the lattice deformation introduces only small fluctuations in the form of periodic oscillations closely resembling the shape of the lattice deformation in the width of the soliton (see Figs. 3(a,b)). We have schematically represented this in Fig. 3(c), where the dotted line along the strands (lattice) represent the periodic deformation of the lattice. It shows that the lattice deformation in DNA does not affect opening of bases in DNA double helix.

5 Conclusion

In this paper, we studied the effect of phonon interaction on base pair opening in DNA by considering the dynamic plane base rotator model. The dynamics of this model in the continuum limit gives rise to a perturbed sine-Gordon equation coupled with a linear wave equation representing longitudinal lattice vibration, which were derived from the Hamiltonian consisting of the stacking
energy, hydrogen bonding energy, energy corresponding to the lattice deformation and its coupling with the stacking and hydrogen bonding energy. In the unperturbed limit, the dynamics is governed by the kink-antikink soliton of the integrable sine-Gordon equation which represents the opening of base pairs in DNA without lattice deformation. In order to understand the effect of lattice deformation on the base pair opening, we carried out a perturbation analysis using multiple-scale soliton perturbation theory. From the results of variation of soliton parameters we observe that when the DNA lattice deforms in a periodic way, the width of the soliton remains constant. However, the velocity of the soliton increases or decreases or remains uniform or even the soliton stops depending on the values of the coupling strengths $\beta$ and $\gamma$. Interestingly, the soliton in all the above cases are found to be stable. From the results of the perturbed soliton we observe that the periodic lattice deformation introduces fluctuation in the width of the soliton. However, there is no change in the topological character of the soliton in the asymptotic region. The above dynamical behaviour may act as energetic activators of the enzyme transport during the process of transcription in DNA.

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References

[1] L. V. Yakushevich, Nanobiology 1 (1992) 343.

[2] S. W. Englander, N. R. Kallenbanch, A. J. Heeger, J. A. Krumhansl and S. Litwin, Proc. Natl. Acad. Sci. USA 77 (1980) 7222.

[3] S. Yomosa, Phys. Rev. A 27 (1983) 2120.

[4] S. Yomosa, Phys. Rev. A 30 (1984) 474.

[5] S. Takeno and S. Homma, Prog. Theor. Phys. 70 (1983) 308.

[6] S. Takeno and S. Homma, Prog. Theor. Phys. 72 (1984) 679.

[7] M. Peyrard and A. R. Bishop, Phys. Rev. Lett. 62 (1989) 2755.

[8] P. L. Christiansen, P. C. Lomdahl and V. Muto, Nonlinearity 4 (1990) 477.

[9] T. Dauxios, Phys. Lett. A 159 (1991) 390.

[10] S. Takeno, Phys. Lett. A 339 (2005) 352.

[11] M. Salerno, Phys. Rev. A 44 (1991) 5292.

[12] G. Gaeta, Phys. Lett. A 143 (1990) 227.

[13] G. Gaeta, Phys. Lett. A 168 (1992) 383.

[14] L. V. Yakushevich, A. V. Savin and L. I. Manevitch, Phys. Rev. E 66 (2002) 016614.

[15] G. Gaeta, Phys. Rev. E 74 (2006) 021921.

[16] M. Cadoni, R. De Leo and G. Gaeta, Phys. Rev. E 75 (2007) 021919.
[17] M. Daniel and V. Vasumathi, Physica D 231 (2007) 10.

[18] M. Daniel and V. Vasumathi, Phys. Lett. A 372 (2008) 5144.

[19] C. R. Calladine, J. Mol. Biol. 161 (1982) 343.

[20] A. C. Scott, Phys. Lett. A 86 (1981) 60.

[21] A. S. Davydov, Solitons in Molecular Systems (Reidel, Dordrecht, 1985) PP. 1-20.

[22] A. S. Davydov, Ukr. Fiz. Zh. 20 (1975) 179.

[23] J. X. Xiao, J. T. Lin and G. X. Zhang, J. Phys. A. Math. Gen. 20 (1987) 2425.

[24] A. Pople, Proc. R. Soc. London Ser. A 205 (1951) 165.

[25] M. J. Ablowitz, D. J. Kaup, A. C. Newell, and H. Segur, Stud. Appl. Math. 53 (1974) 249.

[26] D. J. Kaup, SIAM J. Appl. Math. 31 (1976) 12.

[27] G. L. Lamb, Elements of Soliton Theory (Wiely, New York, 1980) PP. 259-278.

[28] J. Yan and Y. Tang, Phys. Rev. E 54 (1996) 6816.

[29] Y. Tang and W. Wang, Phys. Rev. E 62 (2000) 8842.

[30] J. Yan, Y. Tang and G. Zhou, Phys. Rev. E 58 (1998) 1064.

[31] R. Dandoloff and A. Saxena, J. Phys.: Condens. Matter 9 (1997) L667.

[32] F. Zhang, Y. S. Kivshar and L. Vazquez, Phys. Rev. A 45 (1992) 6019.

[33] T. Hwa, E. Marinari, K. Sneppen and L. Tang, PNAS 100 (2003) 4411.
[34] E. Kreyszig, *Advanced Engineering Mathematics* (John-Wiley, New York, 2002)

PP. 771-794.
Fig. 1. (a) A schematic representation of the structure of B-form DNA double helix. (b) A horizontal projection of the $n^{th}$ base pair in the xy-plane.

Fig. 2. (a) Kink and (b) antikink one soliton solutions of the sine-Gordon equation (Eq. (5a) when $\epsilon = 0$). (c) A sketch of the formation of open state configuration in terms of kink-antikink solitons in a DNA double helical chain.

Fig. 3. The perturbed (a) kink-soliton and (b) antikink-soliton with $\beta = \dot{\gamma} = 1.0, b = -1.0$ and $v_0 = 0.4$. (c) A sketch of the open state configuration in DNA with small fluctuations and periodic deformation in the lattice.
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