Nonlinear dynamics of DNA bubble induced by site specific DNA-protein interaction

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Abstract. We propose a dynamical model describing interactions between DNA and a specific binding protein involving long-range transmission of biological information. The model couples the hydrogen bonds between the one connecting DNA and protein side chain and the one connecting DNA base pairs since they account for site specificity of the binding. We adopt Morse potentials with coupling terms to construct model of coupled hydrogen bonds. We show that the model gives rise to a breather soliton formation, corresponding to the DNA bubbles, which propagates through DNA chain as the carrier of genetic information. We investigate various kinds of possible coupling dynamics and suggest the model realism in depicting the renaturation or hybridization processes.

1. Introduction

Lossless communication between DNA-binding proteins is the basis of life because it facilitates the gene regulation, transcription and replication of DNA. It has been proposed [1–4] that a protein induces propagating DNA deformations that play role as the carrier for lossless biological communication in DNA chain [5]. Induced conformation in DNA is referred as allosteric effect in DNA. Nonlinearity is necessary since any harmonic wave could not possess a coherent wave in dispersive medium and highly dissipative environment. Thus, the propagating conformation is nicely depicted by a solitonic wave. In this paper, we aim to investigate the dynamics of solitonic wave corresponding to the propagating local conformational distortion in DNA that acts as the information carrier induced by the binding of protein in specific DNA sites. A discussion in biological terms will be conducted to give the mathematical solution a meaning. The relevant preprint is also available in [6].

The notable study of nonlinear localized soliton that propagates through DNA was conducted by Peyrard and Bishop (PB) [7], with their notable breather excitation referred as DNA bubble, which has experimentally been measured in [8]. The dynamics and thermal effects of PB breather are studied in [9,10], while the effect of viscosity and external forces are investigated in [11]. The PB model has been modified to include the protein interaction, such as the statistical model given in [12] and TFAM (Transcription Factor A, Mitochondrial)-DNA interaction model [13] that depicts the allosteric interactions by proteins. However, the effect of the chemical bonds between protein and DNA to an analytical breather is not yet investigated.
Figure 1. Protein-DNA H-bonding. We pick the example case glutamine chain binding to Adenine (A)-Thymine (T) base pair. The H-bonds are in box (x) and in oval (y).

The notable property of DNA-protein interaction is that the protein can bind to a specific region in DNA, depending on its certain DNA sequence or base-pair type. Origin of the specificity in DNA recognition by protein, at chemical signatures, is carried by the base pairs and the DNA shapes [14–16]. The chemical bonds involved are electrostatic, van der Waals’s and hydrogen bond (H-bond); but the latter is the most significant because it possesses the highest transition free energy the most specific binding and the nonspecific case [17]. Thus, the H-bond is responsible for specific DNA-protein interaction.

In this paper, we will derive the solution to our DNA-protein model [18] by using multiple scale perturbation method and later investigate its dynamics. The model Hamiltonian is

\[ H = \sum_n \left\{ \frac{p_{yn}^2}{2m} + D(z)(e^{-\alpha y_n} - 1)^2 + \frac{k}{2}(y_n - y_{n-1})^2 + \frac{p_{zm}^2}{2M} + E(e^{-\beta x_m} - 1)^2 + \frac{\chi^2}{2} a^a b^b f_{mn} \right\}. \]

Here \( p_{yn} \) and \( y_n \) are momentum and position of the stretching mode of a base pair, respectively; while \( D \) is the depth of the Morse potential (second term) of this mode. The DNA-protein stretching \( x_m \) is controlled under a Morse potential by depth \( E \) and is interacting with DNA at site-\( m \). The first three terms are DNA parts consisting of Morse potential for the base pair (the oval dots in figure 1) and the harmonic stacking interaction; the next two terms are protein parts, which also consist of a Morse potential for H-bonds connecting DNA and protein side chain (the rectangle dots in figure 1). The last term is interaction term with coupling constant \( \chi \); it contains Gaussian decay factor \( f_{mn} \) with \( \sigma^2 \) proportional to variance:

\[ f_{mn} = \exp[-\sigma^2(m-n)^2]. \]

The dynamics of the DNA-protein interaction relies heavily on the values of \( a \) and \( b \) in the last term of (1). One can check the implication via perturbation method by implying \( a = b = 1 \). It will be seen that the interaction term pops up in the zeroth-order equations, and it is undesirable for solving perturbations [18]. By considering the biological facts that the protein triggers a local opening in DNA, even when all base pairs are still closed \( (y_n = 0 \text{ initially}) \), it is required that the interaction force in the equation of motion for \( y_n \) not contain the amplitude \( y_n \); otherwise, a closed DNA segment will forever be unopened even though the protein is interacting nearby. Here we take \( a = 2 \) and \( b = 1 \). The structural stability analysis of this Hamiltonian model is investigated in [19].
2. Nonlinear excitations in the DNA-protein interaction

2.1. The multiscale expansion method

To examine how the protein induces a local base-pair conformation (i.e., the opening), we must obtain the analytical form of the nonlinear excitation. A well-known perturbative method has been developed in [20], which is actually based on the multiscale expansion technique. The time $t$ is expanded to $t_0, t_1, \ldots$ where $t_n = \epsilon^n t$ [21] and $\epsilon$ is a small parameter. By using multiple scales method, we treat fast and slowly varying time and spatial scales separately so that we obtain envelope amplitudes in different scales. A naive perturbation cannot, in general, achieve such solutions.

According to original PB approach [22], it is assumed that the oscillations of bases be large enough to be anharmonic but still insufficient to break the H-bond since the Morse plateau is not yet reached. The shifts $Y_n \equiv \alpha y_n$ and $X_m \equiv \beta x_m$ oscillate around the bottom of symmetric potential; hence the transformations $Y_n = \epsilon\phi_n$ and $X_m = \epsilon\psi_m$ can be safely implemented to scale the equations of motion. Here we use the continuum approximation, assuming a long DNA chain with lattice space $a \to 0$, implying $na \to z, m/a \to \rho, ka \to K$, $D(z)/a \to \mathcal{D}$, $f_{ml} \to f(z) = \exp[-\sigma^2(z-z_0)^2]$ and $\chi/a \to \mathcal{X}$. The continuum approximation brings us to the equations of motion for (1),

$$\phi_{tt} - S\phi_{zz} + V(z)\phi = V(z) \left( \frac{3}{2} \epsilon \phi^2 - \frac{7}{6} \epsilon^2 \phi^3 + O(\epsilon^3) \right) - \frac{\mu}{2} \epsilon^2 f(z),$$

$$\psi_{tt} + W\psi = W \left( \frac{3}{2} \epsilon \psi^2 - \frac{7}{6} \epsilon^2 \psi^3 + O(\epsilon^3) \right) - \eta \epsilon \int \phi f(z)dz,$$

where the continuum parameters are

$$V(z) = \frac{2\alpha^2 \mathcal{D}}{\rho}, \quad W = \frac{2\beta^2 E}{M}, \quad S = \frac{K}{\rho}, \quad \mu = \frac{\mathcal{X}{\alpha}}{\rho^2}, \quad \eta = \frac{\mathcal{X}}{\mathcal{M}{\alpha}}.$$

For simplicity hereafter, we write $z \equiv z_0, Z \equiv z_1, t \equiv t_0, T \equiv t_1,$ and $\tau \equiv t_2$. The multiscale expansion yields the time or spatial derivative for each order of $\epsilon^n$ so that we can solve the equations recursively. It should be borne in mind that $\phi = \phi(z, Z, t, T, \tau)$ and $\psi = \psi(t, T, \tau)$ [18]. This method breaks down the nonlinear problem into several nonhomogenous linear ordinary differential equations up to $O(\epsilon^2)$ in $\phi$ and $\psi$; the solutions are [18]

$$\phi^{(0)} = A_1(Z, T, \tau)e^{i\theta} + \text{c.c.},$$

$$\psi^{(0)} = 2(\tau)e^{i\varphi} + \text{c.c.},$$

$$\phi^{(1)} = 3|A_1|^2 - \frac{\mu f(z)}{2\sigma V(z)} |A_2|^2 - \frac{1}{2} A_1^2 e^{2i\theta} + \frac{\mu f(z)}{12\sigma V(z)} A_2^2 e^{2i\varphi} + \text{c.c.},$$

$$\psi^{(1)} = 3|A_2|^2 - \frac{1}{2} A_1^2 e^{2i\varphi} + \frac{\eta}{\sigma} \left[ \frac{A_2^2}{\omega^2} \int A_1 dZ e^{i(\theta-\varphi)} \right] e^{-q^2/4\sigma^2} + \text{c.c.},$$

where $\theta = qz - \omega t, \bar{\theta} = qz_0 - \omega t$ and the phase $\varphi = \sqrt{W}t$. From here we get the dispersion relation,

$$\omega^2 = V(z) + S\epsilon^2.$$

Notice that $\partial A_2/\partial T = 0$, so $A_2$ has no dependence of $T$. Finally, the slow varying envelopes $A_1$ and $A_2$ are determined from the coupled nonlinear Schrödinger-like equations obtained from
zeroing the secular terms (exp(±iθ) and exp(±iφ)) in $O(\epsilon^2)$,

$$
\frac{i}{\partial \tau} A_1 + P_1 \frac{\partial^2 A_1}{\partial \xi^2} + Q_1 |A_1|^2 A_1 = 3\mu f |A_2|^2 A_1, \\
\frac{i}{\partial \tau} A_2 + Q_2 |A_1|^2 A_2 = \eta \gamma \int |A_1|^2 A_2 dZ,
$$

(10)

where

$$
\gamma = \left[ 3 + \frac{2\eta \sqrt{\pi} e^{-q^2/4\sigma^2}}{\omega^2 - 4W} \right] \frac{\sqrt{\pi}}{\sigma}, \quad Q_1 = 4V(z), \\
Q_2 = \left[ 4W + \frac{5\mu \eta \sqrt{\pi}}{12\sigma^2 V(z)} \right], \quad P_1 = \frac{S - V_g^2}{2\omega},
$$

(12)

and $\xi = Z - V_g T$ is a right-moving coordinate having group velocity $V_g = Sq/\omega$. The integral of $|A_1|^2$ with respect to $Z$ over entire space is a finite function of time because we assume a localized solitonic wave.

2.2. Nonlinear excitations

The bright soliton solutions of coupled NLS have been fairly investigated, such as in [23]. We use the Hirota bilinear method [24] to solve (10) and (11) with the transformations

$$
A_1 \equiv \frac{G(\xi, \tau)}{F(\xi, \tau)} \quad \text{and} \quad A_2 \equiv \frac{H(\xi, \tau)}{F(\xi, \tau)} \bigg|_{\xi = \xi_0}
$$

(13)

where $F \in \mathbb{R}$ and $G, H \in \mathbb{C}$. Here we apply a technique that assumes spatial dependency of $A_2$ in the first place, and discard it by inserting a constant $\xi = \xi_0$ after the solution is found. By inserting (13) into (10) and (11), we get the bilinear forms

$$
(iD_\tau + P_1 D_\xi^2) G \cdot F = 0, \\
Q_1 |G|^2 - \mu \gamma f |H|^2 = P_1 D_\xi^2 F \cdot F, \\
iD_\tau H \cdot F = 0, \\
Q_2 |H|^2 = \eta \gamma |G|^2.
$$

(14)

(15)

From (15) we can relate $A_1$ and $A_2$ by $|H|^2 = \eta \gamma |G|^2/Q_2$. The problem of finding one-soliton solution with a common phase is equivalent to solving the NLS in the form of

$$
\frac{i}{\partial \tau} A_1 + P_1 \frac{\partial^2 A_1}{\partial \xi^2} + Q'(z) |A_1|^2 A_1 = 0,
$$

(16)

where

$$
Q'(z) = Q_1 - \frac{3\mu \eta \gamma}{Q_2} f(z)
$$

(17)

is obtained from (15). The solution for bright soliton, $PQ' > 0$, is [20]

$$
A_1(\xi, \tau) = A \text{sech} \left[ A \left( \frac{Q'}{2P_1} \right)^{1/2} \left( \xi - \frac{v_c \tau}{P_1} \right) \right] \\
\times \exp \left[ i \left( \frac{v_c}{2P_1} \right) \left( \xi - \frac{v_c \tau}{P_1} \right) \right],
$$

(18)
where $A$ is the amplitude,

$$A(z) = \left( \frac{v_e^2 - 2v_c v_e}{2 P_1 Q'(z)} \right)^{1/2},$$

(19)

with condition $v_e^2 - 2v_c v_e > 0$. Both $v_c$ and $v_e$ are carrier and envelope wave velocities, respectively, where $v_c = g v_e$ for positive $g$. By taking a common phase, we get that the expression for $A_2$ just differs by the amplitude according to (15),

$$A_2(\tau) = \left( \frac{\eta \gamma}{Q'_2} \right)^{1/2} A_1(\xi, \tau) |_{\xi = \xi_0, z = z_0}.$$  

(20)

To obtain the solution, we first calculate the integral term in (8) by changing the domain $Z$ to $\xi$,

$$\int_{-\infty}^{\infty} A_1 dZ = CA \exp \left[ \frac{iv_e^2}{2 P_1^2} (1 - g) \tau \right],$$

(21)

where

$$C = \frac{2 \pi P_1}{v_c \sqrt{1 - 2g}} \text{sech} \left( \frac{\pi}{2 \sqrt{1 - 2g}} \right).$$

(22)

Inserting (18) and (20) into (6)–(8) and setting $\xi_0$ and $z_0$ to zero, we get

$$Y(z, t) = \epsilon 2 A \text{sech} \Theta \cos(N_+ t + \epsilon^2 A^2 \text{sech}^2 \Theta) \times \{3 - \cos 2(Qz - Mt) - \Lambda(z) \times [1 - \cos 2((Q - q)z - Mt)]\} + \mathcal{O}(\epsilon^3),$$

(23)

$$X(t) = \epsilon 2 A \left( \frac{\eta \gamma}{Q'_2} \right)^{1/2} \text{sech} \theta_0 \cos(N_+ t) + \epsilon^2 A^2 \left( \frac{\eta \gamma}{Q'_2} \right)^{1/2} \text{sech}^2 \theta_0 \times \theta_0 \big[3 - \cos 2(N_+ t)\big] + \epsilon^2 \frac{\eta \sqrt{\pi}}{\sigma} e^{-q^2/4\sigma^2} \times 2CA \left( \frac{\eta \gamma}{Q'_2} \right)^{1/2} \text{sech} \theta_0 \omega_+^{-1} \cos(N_+ + \omega) t + \omega_-^{-1} \cos(N_- + \omega) t] + \mathcal{O}(\epsilon^3),$$

(24)

where

$$\Theta(z, t) = \epsilon v_e \sqrt{1 - 2g} \left( qz - (V_g + \epsilon v_e) \tau \right),$$

(25)

$$\theta_0(t) = \epsilon^2 \frac{v_e \sqrt{1 - 2g}}{P_1^2} t,$$  

(26)

$$Q = q + \epsilon \frac{v_e}{P_1},$$  

(27)

$$M = \omega + \epsilon \frac{v_e}{P_1} \left[ V_g + \epsilon \left( \frac{g v_e}{P_1} \right) \right],$$  

(28)

$$N_\pm = \pm \sqrt{W} + \epsilon^2 g \frac{v_e^2}{P_1^2},$$  

(29)

$$\Lambda(z) = \frac{\mu \eta \gamma}{V(z) Q'_2} e^{-\sigma^2 z^2},$$  

(30)

$$\omega_\pm = \omega^2 \pm 2 \omega \sqrt{W}. $$

(31)
One can see from (23) that if the coupling $\chi = 0$, then $\Lambda(z) = 0$. Hence $y(z,t)$ will be identical to the Peyrard-Bishop breather solution [22], while $x(t) = 0$ as if there is no interacting protein.

We take and adjust the PB parameters from [25],

$$\begin{align*}
\alpha &= 1.2\sqrt{2} \text{Å}^{-1}, \quad D = 0.07 \text{eV}, \quad k = 12 \text{N/m}, \\
q &= 0.18 \text{Å}^{-1}, \quad g = 0.47, \quad v_e = 1888 \text{m/s}.
\end{align*}$$

Here we take constant $D$ for simplicity. The length between two base pairs is $a = 3.4 \text{Å}$ and the nucleotide mass is $m = 5.1 \times 10^{-25} \text{kg}$. Here the value of $q$ corresponds to a wavelength covering 10 base pairs. We take $E = D$ and $\beta = \alpha$ as the connecting hydrogen bonds between adenine and glutamine in the base pairs; they are the same as those connecting A-T. We interpret the decay factor $\sigma/\sqrt{2}$ as the inverse width of the protein. An $\alpha$-helix protein is $12 \text{Å}$ in diameter [26], hence $\sigma = 0.117 \text{Å}^{-1}$. The protein effective mass $M$ is rather free because any proteins could have an arbitrary count of amino acid sequences. Nevertheless, in this case we take it as a glutamic acid weight, $M = 2.47 \times 10^{-24} \text{kg}$. We are left with a free parameter, the coupling constant $\chi$, whose value should have significance in the dynamics of the interaction.

### 3. Discussion and conclusions

To discuss the model relevance with biological reality, we investigate the restrictions of our introduced coupling constant $\chi$ and its ramification with the amplitudes. The value of the coupling is crucial for the complex interaction and should have several interpretations. To see this, let us consider (19) by taking $z = z_0$; we get the restriction

$$Q_1 Q_2 - 3\mu \eta \gamma > 0,$$

or more explicitly, recall that $X = \chi/a$,

$$Q_1 C + (Q_1 D - A)X^2 - BX^3 > 0$$

where

$$\begin{align*}
A &= \frac{9\sqrt{\pi}}{M \rho \beta^2 \sigma}, \quad B = \frac{6\sqrt{\pi}}{M \alpha (\omega^2 - 4W)} e^{-q^2/4\sigma^2}, \\
C &= 4W, \quad D = \frac{15\sqrt{\pi}/2}{12M \rho \beta^2 \sigma^2 V(z)}.
\end{align*}$$
Figure 3. Plot of the solutions with coupling constant $\chi = 0$, $\chi = -0.904$ (local maximum), $\chi = 1$ (approaching singularity) and $\chi = -500$ (highly negative).

If $Q_1 D - A > 0$ then we require

$$\sigma < 0.392\,\text{Å}^{-1},$$

meaning the protein diameter $\sqrt{2}/\sigma > 3.59\,\text{Å}$, which is obviously the case because it is barely a base-pair length. Now we are left with $B$ whose sign depends on $\omega^2 - 4W$. The positive and negative case are

$$M > 8\beta^2E/\omega^2, \quad M < 8\beta^2E/\omega^2,$$

respectively. A positive $B$ will produce a relation between the amplitude $A$ and coupling $\chi$ as in figure 2; the main property is that $A \to 0$ for highly negative $\chi$. In contrast, a negative $B$ will vertically mirror the relation, i.e., $A \to 0$ for highly positive $\chi$. Here our choice falls in the positive case. We will discuss the four interesting values of $\chi$ given in figure 2: zero, local maximum, approaching singularity and highly negative.

For $\chi = 0$ it is shown in (23), figure 3 and figure 4 that the solution is identical to the PB breather when the H-bonds between protein and DNA are not shifted. However, if we take $\chi$ near zero then the DNA is open, while the protein side chain is doing a local oscillation. The interaction induces a local base pairs opening to let the protein recognize a specific sequence of base pairs, while previously the bases are being hindered from outside world by DNA strand.
Figure 4. Plot of the stretch $y$ with respect to DNA chain $z$ and time, (top left) $\chi = 0$, (top right) $\chi = -0.904$, (bottom right) $\chi = 1$ and (bottom left) $\chi = -500$.

Our model only contains one-dimensional bond shifts, and thus it has a radial symmetry. It is possible that the bases twist out while being recognized. The propagating breather soliton is interpreted as the mediator of the allosteric transmissions in DNA [2, 4] that facilitate the long-range information transfer between two vastly separated specific DNA-binding proteins.

Our result for small $\chi > 0$ is in agreement with the statistical model in [12] that implies a breather excitation or amplification of an already existing breather. We should restrict $\chi$ in the value where the amplitudes vary linearly with $\chi$. In addition, from (23) we find that the wavelength near the protein is reduced by $q$ in the $\Lambda(z)$ term. We predict from (24) that the protein will sustain a small-amplitude oscillation that decays over time, by the slowly varying envelope wave occurring over 1,000 ps. This vibration is mechanically due to the recoil from the base-opening process.

The singular point is due to the square root in (19). We cannot interpret this as a totally denatured or separated DNA strands because the phenomena falls outside our small amplitude approximation. On the other hand, we have not found the significance of the local maximum case of $\chi$ other than its relatively high-amplitude property. The local maximum occurs when $Q_1D - A > 0$. For $Q_1D \approx A$ or for very small proteins, the local maximum ceases.

The case of highly negative $\chi$ is particularly interesting because, contrary to the previous cases, it totally reduces the amplitudes. The protein can repress or close the base pairs within its reach, which is restricted by $f(z)$. This can describe the DNA recombination or the reverse mechanism of denaturation. The naturally occurring base pairs closing is the renaturation process catalyzed by proteins such as RAD1O [27], while a similar mechanism can be engineered by DNA hybridization. The base recombination process is crucial for the polymerase chain reaction which is widely used for DNA testing.

At last, the value of effective mass $M$ is still unclear because it depends on the geometrical features of each type of protein and the binding it conducts. Further investigation is needed because the overall interaction dynamics can be extremely different as the coupling behavior is dependent on $M$. It is also interesting to study the base pair zippering by protein because it needs to regularize the thermal fluctuation that forces the bases to open again after being closed.
by the renaturation or hybridization processes.

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