EFFECT OF VISCOSITY IN DNA-RNA TRANSCRIPTION

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Abstract: We investigate the nonlinear dynamics of a DNA molecule system using Peyrard-Bishop (PB) model. The effect of viscosity in DNA dynamics is studied both analytically and numerically. The PB model is carried out by a perturbed nonlinear Schrodinger equation and we invoke a multiple-scale perturbation technique to the dynamical equation of PB model and obtain the perturbed soliton. The perturbed NLS equation shows that the velocity and the amplitude of the soliton is varied due to the effect of viscosity which subsequently affects the DNA-RNA transcription and the evolution of soliton parameters has also been analyzed with presence and absence of viscosity of DNA.

Keywords: CNLS, DNA-RNA Transcription, DNA, Perturbed Soliton, Viscosity.

Introduction: One of the first simple mathematical models of DNA was proposed by Englander et al. [1]. Much attention has been focused on the nonlinear effect of DNA molecules in the past years. It seems that there is a strong relationship between the mechanism of the transcription of DNA and the nonlinear effects due to the formation of local denaturation regions [2] and the large fluctuations or breathing of the double helix observed in experiment [3]. Since the idea that nonlinear excitations could play a very important role in the dynamics of DNA was proposed, great progress on this issue has been achieved by means of tools ranging from statistics to dynamics [4-6]. However, the denaturation and the formation of bubbles in DNA have mostly been treated separately [7]. DNA is a biological polymer which plays an important role in the conservation and transportation of genetic information. Many arguments in favor of the nonlinear character of the internal DNA dynamics are k and a number of theoretical models have been proposed to describe the nonlinear properties of DNA. Some of them are successfully used for interpretation of different experimental data, such as the data on hydrogen-tritium exchange, DNA denaturation [8] and others. Biological lattices are very complex structures. They are, as a whole, made of complexes of atoms connected by hydrogen as well as covalent bonds. These theoretical models are based on longitudinal and transverse motions, as well as bending, stretching and rotations [9].

In this paper, we study the influence of viscosity on DNA dynamics. We assume that viscosity, which we deal with, comes from RNAP. At some segments, DNA chain is surrounded by RNAP, which means that our work is relevant for those segments. We show that RNAP causes frictional forces acting on the oscillating nucleotides belonging to DNA. This viscosity brings about demodulation of an incoming solitonic wave, which is crucial for the transcription.

According to the PBD model transversal interactions between nucleotides at the position n, belonging to different strands is modelled by the Morse potential. These forces are weak and consequently. Only the transversal oscillations of the nucleotides are taken into consideration. Of course, the Morse potential is widely used but is not the only choice for intrastrand description. Note that the Morse potential describes both the hydrogen bonds and the repulsive interactions of the phosphate, as well as the surrounding solvent action. The B-form DNA in the Watson-Crick model is a double helix, which consists of two strands s1 and s2 linked by the nearest-neighbour harmonic interactions along the chains. The strands are coupled to each other through hydrogen bonds, which are supposed to be responsible for transversal displacements of nucleotides. This helicoidal structure of the DNA chain implies that nucleotides from different strands become close enough so that they interact through water filaments. This means that a nucleotide at the site n of one strand interacts with both (n+h)th and (n–h)th nucleotides of the other strand. Introducing the transversal displacements u_n, v_n of the nucleotides from their equilibrium positions along the direction of the hydrogen bonds.

The Model Hamiltonian and Dynamical Equation:

The Hamiltonian for the DNA chain is

$$H=\sum\left[\begin{array}{l}\frac{m}{2}(u_n^{\bullet 2})+(v_n^{\bullet 2})+\frac{k}{2}[(u_n-u_{n-1})^2+(v_n-v_{n-1})^2]+\frac{K}{2}[(u_n-v_{n+h})^2+\\(u_n-v_{n-h})^2]+D\exp[-a(u_n-v_n)]\end{array}\right], \quad \rightarrow (1)$$

Where u_n and v_n are the transversal displacements of the nucleotides of mass m at the site n from their equilibrium positions. One can recognize a kinetic energy term. The parameters k and K are coupling constants for the longitudinal and helicoidal interactions respectively. In third term the parameter K, describes the helicoidal interactions along the chain. Finally, the last term is the Morse potential where a and D are the inverse width and the depth of well respectively.

It is obvious that the PBD model assumes two degrees of freedom and both coordinates, u_n and v_n, represent the transversal displacements. It is more convenient to describe the motion of two strands introducing coordinates x_n and y_n, representing the in-phase and the out-of-phase transversal motions are defined as,

$$x_n=\frac{u_n+v_n}{2}, y_n=\frac{u_n-v_n}{\sqrt{2}}, \quad \rightarrow (2)$$

from Eq. (1) and Eq. (2) one can obtain a dynamical equation as

$$m\ddot{y}_n=k(y_{n+1}+y_{n-1}-2y_n)-k(y_{n+h}+y_{n-h}+2y_n)+2\sqrt{2}aD\exp[-a\sqrt{2}y_n-1]\exp[a\sqrt{2}y_n] \quad \rightarrow (3)$$

$$y_n(t)=\varepsilon F_1(\xi)\exp[i\theta_n]+\varepsilon^2[F_0(\xi)+F_2(\xi)\exp[i2\theta_n]]+cc+o(\varepsilon^3) \quad \rightarrow (4)$$

$$\xi=(\varepsilon nl,\varepsilon t),\theta_n=nql-\omega t$$

In order to solve the nonlinear Eq. (3) a semi-discrete approximation can be used [15] and we invoke the derivate expansion and multiple scale method to solve Eq. (3). The essence of this procedure is a series expansion and a limit nl → z where l is a distance between two neighboring nucleotides and z is a continuous variable. Eq.(4) represent a modulated wave and the parameters ω and q are the optical frequency of corresponding linear approximation and the wave number of a carrier wave, the function Fo is real and cc stands for complex-conjugate one can show that the functions Fo and F2 in Eq.(4) can be expressed in terms of F1 as,

$$F_0=\mu|F_1|^2, F_2=\delta_\gamma F_1^2 .$$

While F1 is a solution of the complex nonlinear Schrodinger equation (CNLSE),

$$iF_{1\gamma}=P_\gamma F_{1ss}+Q_\gamma|F_1|^2F_1=0 . \quad \rightarrow (5)$$

Now we introduce the new coordinates as,

$$\tau=\frac{2}{Q_1}\tau_1; S=\sqrt{\frac{2P}{Q_1}S_1}$$

The above equation represents the nonlinear Schrodinger equation with perturbation in cubic term

$$i\frac{\partial F}{\partial\tau_1}+\frac{\partial^2F}{\partial S_1^2}+2|F_1|^2F_1=i\frac{2Q_2}{Q_1}|F|^2F \quad \rightarrow (6)$$

Where,

$\omega_g^2 = \dfrac{4a^2 D}{m}$, $\alpha = \dfrac{-3a}{\sqrt{2}}$ and $\gamma = \dfrac{7a^2}{3}$

In the next section, we adopt a direct perturbation theory to solve Eq. (6)

2. A Direct Perturbation Theory on the PB Model of DNA: The perturbed nonlinear Schrodinger equation is given by, [10, 11]

$$i\frac{\partial F}{\partial \tau_1} + \frac{\partial^2 F}{\partial S_1^2} + 2|F_1|^2 F_1 = i\frac{2Q_2}{Q_1}|F|^2 F \quad , \qquad \rightarrow (7)$$

We consider a nonlinear partial differential equation of the form,

$$K(q, q_t, q_x, \ldots \lambda) = 0 \ , \ 0 < \lambda << 1 , \qquad \rightarrow (8)$$

where K is a nonlinear function of q,qx,.... and λ is an external parameter. Let the unperturbed equation (for λ =0) be given by,

$$K(q_0, q_{0t}, q_{0x}, \ldots \lambda) = 0 \quad , \qquad \rightarrow (9)$$

where q_0 is an exact soliton solution to Eq. (9). We then rewrite qo in terms of these newly defined functions. The unperturbed solution is then rewritten in terms of certain fast and slow variables as,

$$q_0 = \hat{q}_0(\theta_1, \theta_2, ..\theta_m, T; P_1, P_2, ..P_N)$$

Considering Eq. (7) and treating λ as a small parameter, we find that as λ → 0, it admits the following envelope soliton solution of the nonlinear Schrodinger equation

$$\xi_0 = \eta \operatorname{sech} \eta(\theta - \theta_0) \exp[i\xi(\theta - \theta_0) + i(\sigma - \sigma_0)] \qquad \rightarrow (10)$$

Assuming a Poincare-type asymptotic expansion for q and Collecting the coefficients of various powers of λ and we obtain the real and imaginary parts

$$L_2 \hat{\psi}_1 = -\eta^2 \hat{\psi}_1 + \hat{\psi}_{1\theta\theta} + 2\hat{q}_0^2 \hat{\psi}_1 = \operatorname{Im} \hat{F}_1 \qquad \rightarrow (11)$$

$$\operatorname{Re} \hat{F}_1 = \hat{q}_0[\xi T(\theta - \theta_0) - \xi T\theta_0 - \sigma_0 T] , \ \operatorname{Im} \hat{F}_1 = -\hat{q}_0 T + R\hat{q}_0^3 \qquad \rightarrow (12)$$

and L1 and L2 are self-adjoint operators. In writing the above we have assumed only a linear perturbation by retaining terms only up to O(λ).

Velocity and Amplitude of the Soliton: Considering the homogeneous part of Eq. (11), we can check directly that qoθ is a solution to it. Similarly, for Eq. (12), qo is a solution to its homogeneous part. In order that q1/ qo is bounded for all values of θ, we obtain straight forwardly from Eqs. (11-12), the secularity conditions

$$\int_{-\infty}^{\infty} \hat{q}_0(-\hat{q}_0 T + R\hat{q}_0^3) d\theta = 0 \qquad \rightarrow (13)$$

Velocity of the soliton is given by,

$$\int_{-\infty}^{\infty} -\eta^2 \operatorname{sech} \tau \tanh \tau d\tau \left(\eta \operatorname{sech} \tau [\xi_T(\theta - \theta_0) - \xi\theta_{0T} - \sigma_{0T}] \right) \frac{d\tau}{\eta} = 0 \ ; \ \xi_T = 0$$

Amplitude of the soliton is given by,

$$\int_{-\infty}^{\infty} \eta \operatorname{sech} \tau (-\hat{q}_{0T} + R\hat{q}_0^3) \frac{d\tau}{\eta} = 0$$

$$-2\eta_T + \eta_T + \frac{4}{3} R\eta^3 = 0 \ , \ \eta_T = \frac{4}{3} R\eta^3 , \eta = \frac{1}{\sqrt{\frac{8}{3} RT + c}} \qquad \rightarrow (14)$$

Where c is a integration constant. From Eq. (13), it is obvious that soliton moves with constant velocity.

3. Perturbed Solitons: In order to construct the explicit perturbed solution to Eq. (7), we now solve Eqs. (13) and (14). The homogeneous parts of Eqs. (13) and (14) admit, respectively, the following particular solution, $\hat{\varphi}_{11} = \operatorname{sech}\tau \tanh\tau$, $\hat{\varphi}_{12} = -\frac{1}{\eta}[\operatorname{sech}\tau - \frac{3}{2}\tau\operatorname{sech}\tau\tanh\tau - \frac{1}{2}\tanh\tau\sinh\tau]$ and

$\hat{\psi}_{11} = \operatorname{sech}\tau$, $\hat{\psi}_{12} = \frac{1}{2\eta}[\tau\operatorname{sech}\tau - \sinh\tau]$

The explicit form of q_1 is,

$$\hat{q}_1 = i\left(\begin{array}{l} \delta_3\operatorname{sech}\tau + \frac{\delta_4}{2\eta}\tau\operatorname{sech}\tau + \frac{\delta_4}{2\eta}\sinh\tau - \frac{R\eta}{3}\tau\operatorname{sech}\tau\tanh\tau - \frac{R\eta}{12}\operatorname{sech}^3\tau + \\ \frac{R\eta}{6}\cosh\tau\operatorname{sech}\tau + \frac{\eta_\tau}{\eta^2}\tau\operatorname{sech}\tau\tanh\tau + \frac{(\theta-\theta_0)}{4}\tau\operatorname{sech}\tau\tanh\tau - \frac{\eta_\tau}{4\eta^2}\tau^2\operatorname{sech}\tau \\ -\frac{(\theta-\theta_0)}{2}\tau\operatorname{sech}\tau + \frac{R\eta}{6}\tau\operatorname{sech}^2\tau\tanh\tau + \frac{R\eta}{3}\tau\operatorname{sech}\tau\tanh\tau - \frac{(\theta-\theta_0)}{4}\operatorname{sech}^3\tau + \\ \frac{R\eta}{6}\operatorname{sech}^2\tau\tanh\tau\sinh\tau + \frac{R\eta}{3}\tanh\tau\sin\tau - \frac{\eta_\tau}{4\eta^2}\tanh\tau\sin\tau - \frac{(\theta-\theta_0)}{4}\operatorname{sech}^2\tau\sinh\tau \end{array}\right) \rightarrow (15)$$

The above equation represent the perturbed soliton solution.

4. Results and Discussion: In order to completely understand the nonlinear phenomenon involving in the transcription and translational processes we use the method of multiscale perturbation analysis. We constructed the amplitude and velocity component of soliton that assist the motion of soliton. It is found that the soliton propagates with constant velocity and the amplitude varies tremendously. The viscosity of the DNA molecules plays a crucial role in determining the essential, physical properties. In order to study the evolution of the amplitude of soliton when the viscosity of the DNA varies from γ= 8.80x10-4 Pa to γ= 8.90x10-4 Pa. Which is physically admissible. We have plotted the perturbed soliton for the choice of parameters $a = 1.2\times10^{10} m^{-1}$, $D = 0.01\times10^{-18} J$, $h = 5$, $k = 0.08 N/m$, $l = 0.34 nm$, $m = 5.1\times10^{-25} Kg$, $q = \frac{2\pi}{6l}$, c = 0.5. It is highly evident from the Fig. 3(a) that the soliton amplitude is gradually decreasing when the viscosity is increased from γ= 8.80x10-4 Pa to γ= 8.90x10-4 Pa. It is surprising to observe that the amplitude of the soliton is decreasing from 2.9 to 0.2, which is highly significant change. The soliton experiences when it is rolling with the increasing viscosity. A similar appreciable deceleration of the velocity is observed from the Fig. 3(b) and the velocity of the soliton is decreasing from 0.49 to 0.08, when the viscosity is varied from γ= 8.80x10-4 Pa to γ= 8.90x10-4 Pa.

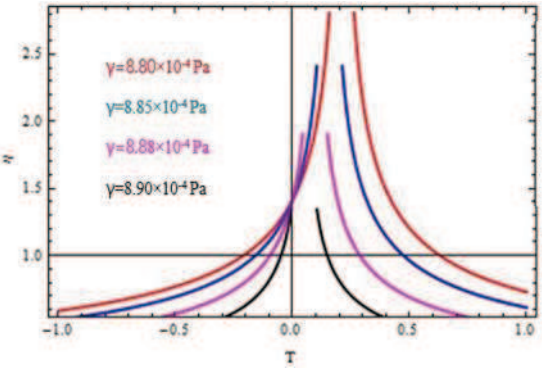

Figure 3(a): Damping of the soliton amplitude by varying the various values of γ.

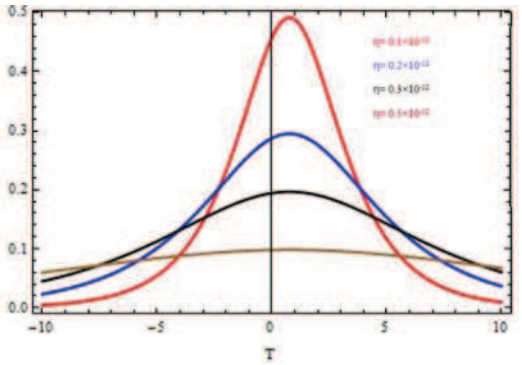

Figure 3(b): Velocity (ξ) changes for varying the values of η.

Conclusions: In order to understand the effect of viscosity on DNA dynamics in terms of soliton like excitations, we derived the dynamical equation of motion for the PB model of DNA, which is governed by a perturbed nonlinear Schrodinger equation. We employ a multiple-scale perturbation technique to the dynamical equation of PB model, and obtain the perturbed soliton. The perturbation analysis confirms that the velocity of the soliton is constant and the amplitude of the soliton is varied due to the effect of viscosity arising from RNAP surrounding the DNA. The viscosity is originated from RNAP, which is highly responsible for DNA-RNA transcription. This declaration of both the amplitude and velocity will play a major role in DNA.

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References:

1. S.W. Englander, N.R. Kallenbanch, A.J. Heeger, J.A. Krumhansl, S. Litwin, "Nature of the Open state in long polynucleotide double helices: possibility of soliton excitations", Proc. Natl. Acad. Sci. USA 77 (1980) 7222.
2. C.R. Calladine and H.R. Drew, "Understanding DNA" (Academic Press, London, 1992).
3. M. Peyrard and A.R. Bishop, "Statistical mechanics of a nonlinear model for DNA denaturation" Phys. Rev. Lett. 62 (1989) 2755.
4. M. Daniel, M. Vasumathi, "Nonlinear molecular excitations in a completely inhomogeneous DNA chain" Phys. Lett. A 372 (2008) 5144;
5. L. V. Yakushevich, "Nonlinear Physics of DNA" (Wiley, Chichester, 1998).
6. T. Dauxois, M. Peyrard, and A. R. Bishop, "Entropy-driven DNA denaturation" Phys. Rev. E 47, R44 (1993).
7. S. Zdravkovi, J. A. Tuszyski and M. V. Satari," Heisenberg Uncertainty Principle and DNA Dynamics", J. Comput. Theor. Nanosci. 2 (2005) 263.
8. S. Zdravkovi and M. V. Satari, "parameter selection in a peyrard-bishop-dauxois model for DNA dynamics", Phys. Rev. E 77 (2008) 031906.
9. M. Daniel, L.Kavitha, "Soliton spin excitations in an anisotropic Heisenberg ferromagnet with octupole-dipole interaction", Phys. Rev. B 59, 13774 (1999).
10. L Kavitha, A Muniyappan," Application of Morse potential in nonlinear dynamics of microtubules" Nonlinear Dyn (2016) 83:1453–1462.

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