

Soliton/exciton transport in proteins

Zachariah Sinkala

Department of Mathematical Sciences, Middle Tennessee State University, Box 34, Murfreesboro, TN 37132, USA

Received 5 October 2005; received in revised form 16 January 2006; accepted 24 January 2006

Available online 6 March 2006

Abstract

The study of electron/proton transport in α -helix sections of proteins have illustrated the existence of soliton-like mechanisms. Recently, Ciblis and Cosic extended investigation to the existence of possible like soliton-type mechanisms in other parts of the protein. They used Quantum Hamiltonian analysis to investigate. In this paper, we investigate the same problem but we use Classical Hamiltonian analysis in our investigation.

© 2006 Elsevier Ltd. All rights reserved.

Keywords: Backbone; Hamiltonian; Protein; Soliton; Transport

1. Introduction

We suggest that folding and conformation changes of proteins may be mediated by interaction with solitons which propagate along the molecular chain. In fact, many biological processes in any living organism are associated with conformational changes, which are a result of space propagation of energy and electrons along protein molecules. For example, the energy released (under normal physiological conditions releases 0.42 eV of energy) under hydrolysis of ATP molecule. The major question is what happens to this energy? How does it perform useful work? Is the energy used through non-equilibrium process or does it thermalize and then work through an equilibrium processes? One hypothesis is in some cases it is transferred along α -helical protein molecules as the vibration oscillation of atoms $C = O$ of peptide groups contained in these molecules. This energy is about half (0.21 eV or 1665 cm^{-1}) of the energy released during ATP hydrolysis. Moreover, the amide-I vibration stays nearly constant from protein to protein, indicating that it is rather weakly coupled to other degrees of freedom. All these factors lead to the assumption that energy released by ATP might stay localized and stored in the amide-I vibration, for example see Davydov (1973). He suggested that the amide-I energy could stay localized through nonlinear interactions of the vibrational

excitation and the deformation in the protein structure caused by the presence of the excitation. The excitation and the deformation balance each other and form a soliton. Therefore, a soliton is a localized packet of energy. Protein molecules also transport electrons from donors to acceptors very effectively. However, Davydov (1973) model is not well suited for vibrational excitons because a vibron transfer is not necessarily analogous to the electronic exciton transfer by exchange interactions, for more details see Takeno (1983).

On one hand, there is much evidence that shows that biological processes can be induced or regulated by the induction of light of particular frequencies (Cosic, 1990, 1994). This is due to light-induced changes in the energetic states of molecules and in particular proteins. The function of some proteins (proton pumps) is directly connected with absorption of visible light of defined wavelengths as in the case of rhodopsins. The strong light absorption is due to the presence of a color prosthetic group bound to the protein, while the frequency selectivity of this absorption is defined by the amino acid sequences of the protein (Cosic, 1990, 1994).

On the other hand, there is evidence that light of defined frequency can induce or enhance some biological processes which are normally controlled only by proteins (Cosic, 1990). All these frequency selective effects of light on biological processes involving protein activation involves energies of the same order and nature as electromagnetic

E-mail address: zsinkala@frank.mtsu.edu.

irradiation of light. These phenomena are discussed in terms of the resonant recognition model (RRM) which proposes that the protein interactions are based on resonant electromagnetic energy transfer within the range of infra-red and visible light (Cosic, 1990).

Solitons have been thought by most biologist as very abstract thing, hardly to imagine. In this paper, we can successfully link solitons with low-frequency phonons (vibration). The latter can be observed and is measurable. Many demonstrations have been made to prove the predicted low-frequency internal motions in biomacromolecules were quite consistent with experimental observations as reported in a series of publications Chou and Chen (1977) and (Chou, 1983a,b, 1984, 1985, 1989a) and (Martel, 1992). Also, their biological functions have been discussed, as presented in Chou (1987, 1988a, 1989b) and Sobell et al. (1983) as well as a comprehensive review Chou's papers (1988b,1989b). For earlier work about solitons which are closely relevant to molecular systems, see the work of Zhou (1989) and Chou (1994).

2. A summarized discussion of RRM model

All protein polymers are made from a linear sequence of 20 different amino acids. The RRM model interprets this linear information using signal analysis methods (Cosic, 1990). Firstly, the amino acid sequences are transformed into numerical series using the electron-ion interaction potential for each amino acid (Veljkovic and Slavic, 1972). Secondly, their values describe the average states of valence electrons in the amino acid. Lastly, numerical series obtained in this way can then be transformed into the frequency domain using fast Fourier transform in order to extract information applicable to the biological function.

To determine the common frequency components for a group of protein sequences, the absolute values of multiples cross-spectral function coefficients are calculated. Peak frequencies in the multiple cross-spectral function denote common frequency components for the analysed sequences. Signal-to-noise ratio for each peak was considered as measure of similarity between analysed sequences. This ratio was calculated as the ratio between signal intensity at the particular peak frequency and the spectrum mean value (ratio of least 20 sequence is considered significant). The presence of a peak with significant ratio in a multiple cross-spectral function of a group of sequences with the same biological function means that all of the analysed sequences within the group have this frequency component in common. This frequency is related to the biological function as it was found in previous investigations (Cosic, 1990) that:

1. such a peak exists only for the group of proteins with the same biological function;
2. no significant peak exists for biologically unrelated proteins; and
3. peak frequencies are different for different biological functions.

Furthermore, it was shown that the proteins and their targets have the same characteristic frequency in common, see Cosic (1990, 1994). Therefore, it can be concluded that these frequencies characterized not only general function but also recognition and interaction between particular protein and its target. This interaction can be considered as resonant energy transfer between interacting molecules. This energy can be transferred through oscillations of a physical field possibly electromagnetic in its nature. As there is evidence that proteins have certain conducting or semiconducting properties (Davydov, 1973), then charge moving through the protein backbone and passing different energy levels caused by different amino acid side groups can produce sufficient conditions for the specific electromagnetic radiation or absorption. The frequency range of this field depends on charge velocity estimated to be $7.87 \times 10^5 \text{ ms}^{-1}$ and the distance between amino acids in protein which is 3.8 \AA , see Cosic (1990, 1994). Having this in mind, the frequency range obtained for protein interactions was 10^{13} – 10^{15} Hz, see Cosic (1990, 1994). All the results obtained in RRM model lead to the conclusion that specificity of protein interactions are based on the resonant electromagnetic energy transfer on a frequency specific for each observed interaction. This has been tested on a number of examples including light absorbing proteins (Cosic, 1990), growth factor activation (Cosic et al., 1989a,b), enzyme activation (Cosic, 1993) and red/far red and blue light receptors in plants (Cosic and Birch, 1994). The physical basis of RRM postulates is the possibility of charge transfer through protein backbone. The possibility and nature of charge or energy transfer along the protein backbone is discussed here.

3. The problem under investigation

We investigate the possible link between a soliton-like mechanism and the resonance recognition process. As the RRM derives a set of frequencies computed from data on the whole length of the protein, it has been assumed that the charge or excitations traveling along the backbone can produce vibrations (oscillations) of particular frequencies. The assumption here is that solitons would travel along the backbone. Therefore, the quotient (soliton velocity/length) of protein backbone could be of the order of RRM frequencies.

4. Preliminary work and known results

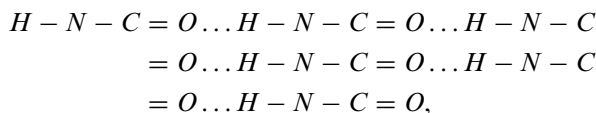
In Davydov (1978) work, it is observed that there are instances of protein to protein reactions where an electron is transferred 30–70 Å from the reactive site unassisted by a chemical carrier. The energy required for an electron to escape its ground state in a protein is approximately 2.3–3.5 eV. The background thermal energy at 300 K is approximately 0.025 eV and optical excitation of the protein is unlikely, see Kharkyanen et al. (1978). This kind of electron transfer is difficult to explain with either

standard chemical theory or by quantum mechanical tunneling, see Davydov (1973).

One possible explanation to the problem of electron transport involves the introduction of solitons. The general properties of solitons are solitary waves (waves localized in space) with the following properties (Ablowitz and Clarkson, 1991):

1. they preserves their shape and velocities,
2. they are extremely stable to perturbations (in particular collisions with small amplitude linear waves),
3. they are even stable with respect to collisions with other solitons. In such collision they pass through each other and recover their speed and shape after interaction. The outcome of the collision of two solitons is a simple phase shift of each excitation.

Davydov (1973) investigated the conditions that would necessitate the formation of excitons and solitons within proteins. His simplest model considers only resonant interactions of vibrational excitations of peptide groups, and this was extended to cover the three spine model for the α -protein. His model assumes that there is a dipole–dipole interaction between the blocks and that there is perturbation of the bond structure within the blocks. This model is equally applicable to electron transport. Careful inspection of the α -helix structure of proteins reveals three channels situated approximately in the longitudinal direction of the sequence



where the dotted lines represent hydrogen bonds. For detailed analysis, it is necessary to consider the interaction of all three channels. Davydov first considered a one-dimensional periodic array of block of atoms.

Here, we will only consider one, since it suffices to convey the basic idea. The Hamiltonian Davydov used to describe the situation is:

$$\begin{aligned} H = \sum_n [E_0 B_n^\dagger B_n - J(B_{n+1}^\dagger B_n + B_n^\dagger B_{n+1})] \\ + \sum_n \left[\frac{v_n}{2m} + \frac{1}{2} w(u_{n+1} - u_n)^2 \right] \\ + \chi \sum_n (u_{n+1} - u_{n-1}) B_n^\dagger B_n \end{aligned} \tag{1}$$

$$= \hat{H}_{CO} + \hat{H}_{ph} + \hat{H}_{int}. \tag{2}$$

Here, B_n^\dagger and B_n are boson creation annihilation operators for quanta of intramolecular vibrations with energy $E_0 = 1665 \text{ cm}^{-1}$ at site n (the CO stretch mode or amide-I mode), u_n and v_n are the molecular displacement and momentum operators for the molecule at site n (the entire peptide group), m and w are the molecular mass and intermolecular force constant, and J is the intersite transfer

energy produced by dipole–dipole interactions. The nonlinear coupling constant χ arises from the modulation of the on-site by the molecular displacements. It is the derivative of the amide-I energy with respect to the length between peptide groups (l) of the adjacent hydrogen bond:

$$\chi \equiv \frac{dE_0}{da}.$$

The vibration part \hat{H}_{CO} , the phonon part \hat{H}_{ph} , and the interaction part \hat{H}_{int} are defined to be individual terms in (2).

For later comparison, we write here the equation of motion for the Heisenberg operator $B_n(t)$,

$$i\hbar \dot{B}_n = E_0 B_n - J(B_{n+1} + B_{n-1}) + \chi B_n(u_{n+1} - u_{n-1}). \tag{3}$$

The form of this equation is such that a phase transformation

$$B_n(t) = \tilde{B}_n(t) \exp\left(\frac{-iE_0 t}{\hbar}\right) \tag{4}$$

removes the energy of the amide-I quantum from the equation, that is, the equation for $\tilde{B}_n(t)$ is (3) but without the term proportional to E_0 .

Davydov minimizes the average value of H with respect to some wave function. This leads to the differential-difference equations. Extensive numerical and theoretical analysis of these differential-difference equations yields the following results: It is reasonable to expect soliton formation at the level of energy released by ATP hydrolysis



and such a soliton travels rather slowly with respect to the speed of longitudinal sound waves. Taking a continuum approximations of the differential-difference equations results in nonlinear Schrödinger equation (NLS), the solution to which is a soliton. Davydov's work was collaborated by a numerical study Hyman et al. (1981). They found out that for soliton to form threshold conditions were necessary:

1. nonlinear cross-coupling between the $C = O$ vibrations and $H..O$ comprehension wave must be sufficiently strong and
2. the $C = O$ vibrations must be energetic enough to provide a self-focusing effect.

Takeno (1983, 1984, 1985, 1986) proposed an alternative model for propagation of biological energy in the α -helix protein. He has argued that the dispersion term in the Davydov model (2), may not be appropriate for the migration of vibrational energy. This particular type of exchange interaction is more relevant for electrons or electronic excitons. His approach is basically classical, he studied vibron solitons in one-dimensional α -helix sections of protein by employing a coupled oscillator-lattice model and therefore does not have the constraint of the number of amide-I quanta. He first introduced his Hamiltonian in

its simplest form, with acoustic phonons, in order to compare with the Davydov model. Takeno has also generalized his theory to deal with more complex systems where the amide-I energy is coupled to both acoustic and optic phonons.

The simplified version of Takeno’s Hamiltonian is

$$\begin{aligned}
 H_T = & \sum_n \left[\frac{p_n^2}{2\mu} + \frac{1}{2} \mu \omega_0^2 \rho_n^2 - 2L \rho_{n+1} \rho_n \right] \\
 & + \sum_n \left[\frac{v_n^2}{2m} + \frac{1}{2} K_a (u_{n+1} - u_n)^2 \right] \\
 & + \sum_n \left[\frac{1}{2} A_a \rho_n^2 (u_{n+1} - u_{n-1}) \right]. \tag{5}
 \end{aligned}$$

Here, ρ_n and p_n are the displacement and momentum coordinates for the high-frequency intramolecular (amide-I) oscillator with mass μ and frequency ω_0 ; L is the coupling strength between neighboring oscillators, which we have restricted to the nearest neighbors. Also, u_n and v_n are the displacement and momentum coordinates for the molecule at site n ; m and K_a are the molecular mass and intramolecular force constant (K_a is the same as w in the Davydov model). The last term couples these two oscillating fields with coupling constant A_a .

In order to make a comparison with the Davydov model, we now for a moment view (5) as a quantum Hamiltonian, with the displacement and momentum coordinates replaced by operators. We introduce creation and annihilation operators for the high-frequency oscillator at site n by the equations

$$\rho_n = \sqrt{\frac{\hbar}{2\mu\omega_0}} (B_n^\dagger + B_n), \quad p_n = i\sqrt{\frac{\hbar\mu\omega_0}{2}} (B_n^\dagger - B_n) \tag{6}$$

then the ρ_n -dependent parts of (5) can be written as

$$\begin{aligned}
 H_v = & \sum_n \hbar\omega_0 \left(B_n^\dagger B_n + \frac{1}{2} \right) - \frac{\hbar L}{\mu\omega_0} \sum_n (B_{n+1}^\dagger B_n^\dagger + B_n^\dagger B_{n+1}^\dagger \\
 & + B_{n+1}^\dagger B_n + B_{n+1} B_n), \tag{7}
 \end{aligned}$$

$$H' = \frac{\hbar A_a}{4\mu\omega_a} \sum_n (B_n^\dagger B_n^\dagger + 2B_n^\dagger B_n + B_n B_n) (u_{n+1} - u_{n-1}). \tag{8}$$

Comparing (8) with the Davydov Hamiltonian it is clear that there are additional $B_n^\dagger B_n^\dagger$ and $B_n B_n$ terms both in the dispersive and interaction parts of the quantum version of the Takeno Hamiltonian. The equation operator B_n obtained from (5) is

$$\begin{aligned}
 i\hbar\dot{B}_n = & \hbar\omega_n B_n - \frac{\hbar L}{\mu\omega_0} (B_{n+1}^\dagger + B_{n+1} + B_{n-1}^\dagger + B_{n-1}) \\
 & + \frac{\hbar A_a}{2\mu\omega_0} (B_n^\dagger + B_n) (u_{n+1} - u_{n-1}). \tag{9}
 \end{aligned}$$

This differs from Eq. (9), the corresponding equations using Hamiltonian (2), by the presence of the creation operators on the right-hand side. The presence of those terms means that a phase transformation of the form (4) cannot remove the energy of the amide-I quantum $\hbar\omega_0 =$

E_0 from the equation. Carrying out that transformation on Eq. (9) produces factors oscillating at $2\omega_0$ in the creation operator terms. In this formulation, the magnitude of E_0 relative to other energies in the problem remains important.

We note that if we drop the creation operators from (8), then we can relate the parameters of the two theories by

$$L = \left(\frac{\mu\omega_0}{\hbar} \right) J, \quad A_a = \left(\frac{2\mu\omega_0}{\hbar} \right) \chi, \quad K_a = w. \tag{10}$$

The equation of motion derived from classical Hamiltonian (5) are

$$\mu\ddot{\rho}_n + \mu\omega_0^2 \rho_n - 2L(\rho_{n+1} + \rho_{n-1}) + A_a \rho_n (u_{n+1} - u_{n-1}) = 0, \tag{11}$$

$$\ddot{u}_n - \frac{a^2 K_a}{m} \frac{(u_{n+1} - 2u_n + u_{n-1}))}{a^2} - \frac{1}{2m} A_a (\rho_{n+1}^2 - \rho_{n-1}^2) = 0. \tag{12}$$

Takeno now proceeds by making a continuum approximations to Eqs. (11) and (12) and obtains this way coupled nonlinear Klein–Gordon equations for the coordinates $\rho(x, t)$ and $u(x, t)$. A rotating-wave approximation then finally leads to an NLS equation.

$$u_{tt} - c_a^2 u_{xx} = \frac{A_a a}{m} \frac{\partial(\rho^2)}{\partial x}, \tag{13}$$

where a is a lattice constant and $c_a = a\sqrt{K_a/m}$ is the sound velocity.

$$i\rho_t = -2La^2 \rho_{xx} + (4L - \mu\omega_0^2 + 2A_a a u_x) \rho. \tag{14}$$

We can look for traveling wave solutions of the form $u(x, t) = u(x - ct)$. Inserting into (13) and integrating once we get

$$u_x(x, t) = -\frac{A_a}{K_a a (1 - s^2)} \rho^2, \tag{15}$$

where $s = c/c_a$. Substituting (15) into (14) we get the nonlinear Schrödinger equation (NLS), but with classical for the amplitude of the amide-I vibration $\rho(x, t)$ compared to Davydov’s NLS equation for the probability amplitude:

$$i\rho_t + 2La^2 \rho_{xx} + (4L - \mu\omega) \rho + \frac{2A_a^2}{K_a (1 - s^2)} \rho^3 = 0. \tag{16}$$

The self-trapped state of amide-I energy is described by the well-known one-soliton solution

$$\rho(x, t) = \alpha \operatorname{sech}[2\alpha a \sqrt{L\kappa}(x - ct)], \tag{17}$$

where c and α are parameters identified as the velocity and amplitude of solitons, respectively, and $\kappa = 2A_a^2/K_a(1 - s^2)$. The situation described by (15) and (16) has a finite interval where the amide-I oscillators are excited by a lattice displacement which pulls the peptide groups closer together in that region. It is easy to show (it can be done similar the way it was shown by Davydov (1982) in the quantum case) that this configuration has a lower energy

Table 1
 α helix parameters, $m_p = 1.672 \times 10^{-27}$ kg

Parameter	Value	Unit	Reference
E_0	1665	cm ⁻¹	(Nevskaya and Chirgadze, 1976)
J	7.8	cm ⁻¹	(Nevskaya and Chirgadze, 1976)
w	13	N m ⁻¹	(Itoh and Shimanouchi, 1972)
m	114	m_p	(Scott, 1982)
χ	0.62	10 ⁻¹⁰ N	(Careri et al., 1984)

than the spatially extended solution to (16) and thus self-trapped (self-focusing).

It can be shown that the soliton moving a velocity c carries an energy

$$E_c = E_0 + \frac{1}{2}m_{sol}c^2, \quad (18)$$

where E_0 is the internal energy of the soliton and m_{sol} is the effective mass of the soliton (Table 1).

5. Applying the classical method

It should be noted that although Davydov, Takeno and others have primary been concerned with the propagation of a soliton in the α -helix, the same equations can be used to model electrons traveling along the protein backbone. Ciblis and Cosic (1997) took three approaches to examine the possibility of solitons being linked to the RRM:

1. direct comparison with known results on soliton work;
2. the derivation of the soliton velocity in terms of fundamental protein parameters based (on Davydov's previous work); and
3. an investigation into the bounds of the soliton velocity within a protein, based on the fact that solitons cannot propagate faster than the speed of compression in a protein.

In this work, we follow the same three above approaches taken by Ciblis and Cosic (1997) but here we apply Takeno's work which is a classical approach compared to Davydov work (which is mainly quantum mechanics approach):

1. Direct comparison with known results on soliton work.
2. The derivation of the soliton velocity in terms of fundamental protein parameters based (on Takeno's previous work).
3. An investigation into the bounds of the soliton velocity within a protein, based on the fact that solitons cannot propagate faster than the speed of compression in a protein.

In Ciblis and Cosic (1997) work, they used a number of equations presented by Davydov (1982), that may be used to define the velocity of a soliton in terms of protein parameters. As mentioned before the equations are based

on a one-dimensional array of cells coupled via dipole-dipole interactions and a local perturbation of that chain. The equations are as follows:

$$E_c = E_0 + \frac{1}{2}m_{dsol}c^2, \quad (19)$$

where E_0 is the internal energy of the soliton, m_{dsol} is the effective mass of the soliton, c is the velocity of the soliton, c_a is the speed of sound through the protein and

$$m_{dsol} = \frac{\hbar^2}{2Ja^2} + \frac{4\chi^4(1 + \frac{3}{2}s^2 - \frac{1}{2}s^4)}{3w^2Jc_a^2(1 - s^2)^3},$$

where \hbar is the Plank's constant divided by 2π , J the dipole-dipole coupling constant between cells of the chain, a the distance between unit cells, w the stretching constant and χ the soliton-phonon coupling constant.

Ciblis and Cosic (1997) substituted m_{dsol} into (19) and solved the resulting equation for the velocity corresponding to a soliton with energy E_c and E_0 . They varied the values of E_0 and E_c calculated values of a soliton's velocity and thus examined if solitons were implicated in RRM interactions.

In this paper, we follow a similar approach used by Ciblis and Cosic, but now we use Takeno's work, and present equations that may define the velocity of a soliton in terms of protein parameters

$$E_c = E_0 + \frac{1}{2}m_{isol}c^2, \quad (20)$$

where E_0 is the internal energy of the soliton, m_{isol} is the velocity of the soliton, c_a is the speed of sound through the protein and

$$m_{isol} = \frac{\hbar^2}{4\hbar La^4} + \frac{4A_a^4\hbar^4(1 + \frac{3}{2}s^2 - \frac{1}{2}s^4)}{3K_a^2\hbar Lc_a^2a^2(1 - s^2)^3}, \quad (21)$$

where

$$s = \frac{c}{c_a}, \quad \hbar L = \mu\omega_0 J, \quad \hbar A_a = 2\mu\omega_0\chi, \quad K_a = w.$$

We substitute m_{isol} into (20) and solve the resulting equation for the velocity corresponding to a soliton with energy E_c and E_0 . This was done using a Computer Algebra System, Mathematica, and the result quantic equation provided four possible values of velocity. By varying the values of E_0 and E_c it is possible to calculate values of a soliton's velocity and thus examined if solitons were implicated in RRM interactions. Numerical results from these investigations are detailed and discussed further in later sections.

5.1. Direct comparison with known results

As it was pointed out in Ciblis and Cosic (1997) that numerical finding (Hyman et al., 1981) of solitons in the α -helix shows that both solitons and excitons could exist in the α -helix of a protein. Their analysis was based on initially approximating a set of difference-differential equations and the initial equations were then decomposed into a coupled system of first-order real equations and then solved. They computed the minimum soliton speed near the

threshold as $1.26 \times 10^3 \text{ m s}^{-1}$, and the maximum theoretical speed for a soliton was found to be $1.1 \times 10^4 \text{ m s}^{-1}$.

The charge velocity approximately computed by RRM estimates is $8 \times 10^5 \text{ m s}^{-1}$ (see Cosic and Birch, 1994). Soliton modeled by Hyman and others is slower by several orders of magnitude. It is also greater than the speed of compression for these peripheral amide-I strands.

Ciblis and Cosic (1997) suggested that assumptions regarding parameters values may need revision. To investigate this further, a model for calculating the stretching constant, K_a , for a section of α -helix was used to see if the values used in Hyman et al. (1981) could be varied. Chou (1983b) in his modeling work used a value for the stretching constant for a hydrogen bond equal to 13 N m^{-1} . The stretching constant used by Hyman and others was 76 N m^{-1} . Chou (1983b) provides a method for calculating the stretching constant for a section of α -helix. Values for the stretching constant using this method for differing sections of α -helix was found to be dependent on the number of constituent of amino acids, see Fig. 1, Ciblis and Cosic (1997). Therefore, K_a the stretching constant varies by an order of magnitude of more. This variation in the H-bond constant was also noted by Scott (1991). Thus, a change in the assumed value of the stretching constant may have on the soliton velocity predicted, and numerous cases with differing values (of an order or more) for K_a need be considered.

5.2. Numerical results

We use formula (20), to predict the velocity of a soliton in the protein backbone. The following assumptions were made to model a soliton in a $N - C - C$ chain with twice the energy of ATP hydrolysis:

$E_c = 1.6 \times 10^{-19} \text{ J}$ approximately twice the energy released by ATP by hydrolysis

$E_0 = 0.328 \times 10^{-19} \text{ J}$ (Hyman et al., 1981)

$J = 1.5 \times 10^{-22} \text{ J}$ (Hyman et al., 1981)

$K_a = 10\text{--}100 \text{ N m}^{-1}$ the range predicted by Chou for section of α -helix

$a = 3.0\text{--}5.0 \text{ \AA}$

$\chi = 1.5 \times 10^{-11} \text{ N}$ (Hyman et al., 1981) threshold for soliton formation

$m = 6.3 \times 10^{-26} \text{ kg}$, the mass of a $N - C - C$ group

$\mu = 7.3 \times 10^{-26} \text{ kg}$, the mass of a amide-I oscillator

$C = O$.

The constants ω_0 , L and A_a can easily be determined since they depend on the above known constants. Using formula (20), two real solutions are obtained. The first solution is a velocity with a very small magnitude of at most order of 10^{-5} m s^{-1} , it is assumed that it has no physical significance. The second solution is of order of $4\text{--}22 \times 10^3 \text{ m s}^{-1}$ and detailed within Table 2. It is assumed that the second solution may have some physical significance. This will be discussed later.

A hypothetical chain of alanine amino acids was examined, using the following constants:

$E_c = 1.6 \times 10^{-19} \text{ J}$ approximately twice the energy released by ATP by hydrolysis

$E_0 = 0.328 \times 10^{-19} \text{ J}$

$J = 1.5 \times 10^{-22} \text{ J}$

$K_a = 100\text{--}1000 \text{ N m}^{-1}$ (to investigate the impact of a stiffer chain)

$a = 10.0\text{--}65.0 \text{ \AA}$

$\chi = 1.5 \times 10^{-11} \text{ N}$

$m = 6.3 \times 10^{-26} \text{ kg}$ the mass of a alanine amino acid

$\mu = 7.3 \times 10^{-26} \text{ kg}$ the mass the vibration (oscillation) of atoms, $C = O$.

Table 2

Soliton velocities in a NCC chain based on the following assumptions: $E_c = 1.6 \times 10^{-19} \text{ J}$, $E_0 = 0.328 \times 10^{-19} \text{ J}$, $K_a = 10\text{--}100 \text{ N m}^{-1}$, $a = 3.0\text{--}5.0 \text{ \AA}$, $\chi = 1.5 \times 10^{-11} \text{ N}$, $m = 6.3 \times 10^{-26} \text{ kg}$, $\mu = 7.3 \times 10^{-26} \text{ kg}$

$R \text{ (\AA)}$	$K_a \text{ (N m}^{-1}\text{)}$		$K_a \text{ (N m}^{-1}\text{)}$		$K_a \text{ (N m}^{-1}\text{)}$		$K_a \text{ (N m}^{-1}\text{)}$	
	$K_a = 10$	$K_a = 20$	$K_a = 30$	$K_a = 40$	$K_a = 50$	$K_a = 60$	$K_a = 70$	$K_a = 80$
3.2	4259.27	5864.12	7116.12	8178.74	9118.38	9969.87	10754.15	11485.01
3.4	4525.47	6230.63	7560.88	8689.91	9688.28	10592.98	11426.29	12202.82
3.6	4791.67	6597.13	8005.64	9201.08	10258.18	11216.10	12098.42	12920.64
3.8	5057.88	6963.64	8450.39	9712.25	10828.08	11839.22	12770.56	13638.45
4	5324.08	7330.15	8895.15	10223.42	11397.98	12462.33	13442.69	14356.26
4.2	5590.29	7696.66	9339.91	10734.59	11967.87	13085.45	14114.83	15074.08
4.4	5856.49	8063.16	9784.67	11245.76	12537.77	13708.57	14786.96	15791.89
4.6	6122.70	8429.67	10229.42	11756.93	13107.67	14331.68	15459.09	16509.70
4.8	6388.90	8796.18	10674.18	12268.11	13677.57	14954.80	16131.23	17227.52
5	6655.10	9162.69	11118.94	12779.28	14247.47	15577.92	16803.36	17945.33
5.2	6921.31	9529.19	11563.70	13290.45	14817.37	16201.03	17475.50	18663.14
5.4	7187.51	9895.70	12008.45	13801.62	15387.27	16824.15	18147.63	19380.96
5.6	7453.72	10262.21	12453.21	14312.79	15957.17	17447.27	18819.77	20098.77
5.8	7719.92	10628.72	12897.97	14823.96	16527.06	18070.38	19491.90	20816.58
6	7986.12	10995.22	13342.73	15335.13	17096.96	18693.50	20164.04	21534.39

Table 3

Soliton velocities in a hypothetical alanine chain based on the following assumptions: $E_c = 1.6 \times 10^{-19}$ J, $E_0 = 0.328 \times 10^{-19}$ J, $K_a = 100\text{--}800$ N m $^{-1}$, $a = 10.0\text{--}65.0$ Å, $\chi = 1.5 \times 10^{-11}$ N, $m = 1.75 \times 10^{-26}$ kg, $\mu = 7.3 \times 10^{-26}$ kg

R (Å)	K_a (N m $^{-1}$)		K_a (N m $^{-1}$)		K_a (N m $^{-1}$)		K_a (N m $^{-1}$)	
	$K_a = 100$	$K_a = 200$	$K_a = 300$	$K_a = 400$	$K_a = 500$	$K_a = 600$	$K_a = 700$	$K_a = 800$
10	24097.51	33942.78	41515.52	47905.81	53538.72	58632.95	63318.64	67680.71
15	36146.26	50914.17	62273.29	71858.71	80308.08	87949.42	94977.97	101521.07
20	48195.01	67885.56	83031.05	95811.61	107077.44	117265.89	126637.29	135361.43
25	60243.77	84856.95	103788.81	119764.52	133846.79	146582.37	158296.61	169201.79
30	72292.52	101828.34	124546.57	143717.42	160616.15	175898.84	189955.93	203042.14
35	84341.27	118799.73	145304.34	167670.32	187385.51	205215.31	221615.26	236882.50
40	96390.02	135771.12	166062.10	191623.23	214154.87	234531.79	253274.58	270722.86
45	108438.78	152742.51	186819.86	215576.13	240924.23	263848.26	284933.90	304563.21
50	120487.53	169713.90	207577.62	239529.04	267693.59	293164.73	316593.22	338403.57
55	132536.28	186685.29	228335.39	263481.94	294462.95	322481.21	348252.54	372243.93
60	144585.04	203656.68	249093.15	287434.84	321232.31	351797.68	379911.87	406084.29
65	156633.79	220628.07	269850.91	311387.75	348001.67	381114.15	411571.19	439924.64

Table 4

Soliton velocity (m s $^{-1}$) limits for a NCC chain

R (Å)	K_a (N m $^{-1}$)		K_a (N m $^{-1}$)		K_a (N m $^{-1}$)	
	$K_a = 20$	$K_a = 40$	$K_a = 60$	$K_a = 80$	$K_a = 100$	$K_a = 200$
2	3662.21	5107.68	6226.24	7172.44	8007.62	11292.15
4	7324.41	10215.36	12452.47	14344.88	16015.24	22584.29
6	10986.63	15323.03	18678.71	21517.33	24022.85	33876.44
8	14648.84	20430.71	24904.94	28689.77	32030.47	45168.59
10	18311.05	25538.39	31131.18	35862.21	40038.09	56460.73
20	36622.09	51076.78	62262.36	71724.42	80076.18	112921.46
40	73244.19	102153.55	124524.71	143448.83	160152.36	225842.93
60	109866.28	153230.33	186787.07	215173.25	240228.53	338764.39
80	146488.37	204307.11	249049.42	286897.67	320304.71	451685.85
100	183110.47	255383.88	311311.78	358622.09	400380.89	564607.32
200	366220.93	510767.76	622623.55	717244.17	800761.78	1129214.63
400	732441.87	1021535.53	1245247.10	1434488.34	1601523.57	2258429.26
600	1098662.80	1532303.29	1867870.65	2151732.51	2402285.35	3387643.89
800	1464883.74	2043071.05	2490494.20	2868976.68	3203047.13	4516858.52

The constants ω_0 , L and A_a can easily be determined since again they depend on the above known constants. The low-speed soliton velocities were calculated to at most order of 10^{-7} m s $^{-1}$. The high-speed soliton solution ranges between 2×10^4 and 5×10^5 m s $^{-1}$. The latter result is of the same order as predicted by RRM. For details of the result, see Table 3. It should be noted, however, that the sound velocity forms an upper limit to the soliton's speed through a protein because as the soliton's velocity approaches the limit the soliton energy tends to infinity. Thus, any high-speed soliton, if in fact existed, would be unstable and the energy might be released as electromagnetic energy. Takeno (1984), provides the following formula for computing the upper bound for the soliton velocity within a one-dimensional protein

$$c_a = a \sqrt{\frac{K_a}{m}}, \quad (22)$$

where c_a is velocity, K_a is the elasticity constant for the chain of units, m is mass of the vibrating units that constitute the protein and a is the non-excited equilibrium distance between the constituent elements of the chain. Since the charge velocity estimated by RRM is 8×10^5 m s $^{-1}$ (Cosic, 1994), then we shall use this as the benchmark to consider the results. There are various ways to group the constituent atoms of the protein backbone into blocks that would act as a single unit within an oscillating chain. It is therefore necessary to investigate the resultant velocities over the likely range of variables. In Table 4, we consider group of atoms whose total mass equals $38u$, $u = 1.6605387 \times 10^{-27}$ kg, and mass of corresponding group of vibration oscillation of atoms is $\frac{117}{57}u$. These masses were chosen because they correspond to a chain consisting of blocks of $N - C - C$ atoms, see Hyman et al. (1981) and Ciblis and Cosic (1997). The range of resultant velocity limits is from 3.6×10^3 to 4×10^6 m s $^{-1}$

Table 5
Soliton velocity limits (m s^{-1}) for protein consisting of $2000u$ sections

R (Å)	K_a (N m^{-1})		K_a (N m^{-1})		K_a (N m^{-1})	
	$K_a = 50$	$K_a = 100$	$K_a = 150$	$K_a = 200$	$K_a = 250$	$K_a = 300$
200	80904.54	112098.44	136336.44	156874.06	175018.38	191450.92
400	161809.09	224196.88	272672.88	313748.12	350036.76	382901.84
600	242713.63	336295.33	409009.31	470622.18	525055.13	574352.75
800	323618.18	448393.77	545345.75	627496.24	700073.51	765803.67
1000	404522.72	560492.21	681682.19	784370.29	875091.89	957254.59

for the range of a and K_a considered. This is within the range of velocities predicted by the RRM but the high values of a and K_a would be accepted as unrealistic scenarios to form the basis for soliton formation.

In Table 5, we consider heavier groups of atoms (corresponding to whole amino acids), where the mass m equals $2000u$ and the corresponding total mass μ of oscillators, $C = O$ equals $\frac{1000}{19}u$. We see that the range of resultant velocity limits is from 8.0×10^4 to $9 \times 10^5 \text{ m s}^{-1}$ for the range of a and K_a considered.

Although overall, the velocities are lower than for *NCC* scenario, the results suggest that perhaps larger sections of the protein may be acting as a coherent unit to form a soliton.

Comparing Tables 4 and 5 suggest that there could be a link between the RRM and a soliton induced electron transport provided the mass of the units cells is of the order of $100u$ with separation of the relevant residues being greater than 20 \AA and the elasticity constant for the chain of units is very high (of the order of 1000 N m^{-1}). Comparison with velocities predicted based on the analysis of the soliton energy equations (21), (20) and the soliton velocity limit values, show that the second solutions, speed solitons, are above the upper bound for a stable soliton. Therefore, link between RRM and solitons could be deduced if:

1. very high stretching constants were shown to exist between amino acids;
2. a physical process which yielded these high-speed solitons could be derived.

However, it is considered that a mechanism that results in electrons traveling at a speed faster than the speed of sound through the backbone, for example an exciton-like mechanism are much more likely to be responsible.

5.3. How does a classical model compare with previous results

Our model is better than Ciblis and Cosic (1997) model for the following reasons:

Although vibron solitons in the present theory in our model and those in Davydov theory used by Ciblis and Cosic (1997) are both described by NLS equation, their

natures are fairly different from each other. This stems from the difference of model Hamiltonian for amide-I vibrons in helical proteins to which the discussion given better is applicable, provided inter-spine interactions are neglected. Namely, vibrons in the present are described by a set of coupled molecular vibration oscillators as given by the first term of the Takeno's Hamiltonian (5), whereas the corresponding ones in the Davydov's theory are regarded as being of quantal nature having the form of excitons with transfer by exchange interactions. The NLS equation here arises from modulations of vibrons by nonlinear coupling with acoustic phonon propagating along the helics of the α -proteins, while that in the Davydov theory directly follows from the quantal Schrodinger equation for exciton probability. The present classical picture of vibron solitons appears to be more appropriate to describe vibrational energy transfer in α -helical proteins as a mobile entity of conformal change. The same explanation holds for application to backbone chain. Additional studies should be taken in the application of Takeno's Hamiltonian to see if they would cause an electron to propagate through a protein backbone.

Acknowledgments

Many thanks to the referee for the valuable suggestions. This work was supported in part by NIA grant from Middle Tennessee State University and NSF HPNC award.

References

- Ablowitz, M., Clarkson, P., 1991. Solitons, Linear Evolutions and Inverse Scattering. Cambridge University Press, Cambridge.
- Careri, G., Buontempo, U., Galluzzi, F., Scott, A.C., Gratton, E., Shymasunder, E., 1984. Spectroscopic evidence for Davydov-like solitons in acetanilide. Phys. Rev. 30B, 4689–4702.
- Chou, K.C., 1983a. Low-frequency vibrations of helical structures in protein molecules. Biochem. J. 209, 573–580.
- Chou, K.C., 1983b. Identification of low-frequency modes in protein molecules. Biochem. J. 215, 465–469.
- Chou, K.C., 1984. Low-frequency vibration of DNA molecules. Biochem. J. 221, 27–31.
- Chou, K.C., 1985. Low-frequency vibration of DNA molecules: beta-sheet and beta-barrel. Biophys. J. 48, 289–297.
- Chou, K.C., 1987. The biological functions of low-frequency phonons: 6. A possible dynamic mechanism of allosteric transition in antibody molecules. Biopolymers 26, 285–295.

- Chou, K.C., 1988a. Collective motion in DNA and its role in drug intercalation. *Biopolymers* 27, 1795–1815.
- Chou, K.C., 1988b. Review: low-frequency collective motion in biomacromolecules and its biological functions. *Biophys. Chem.* 30, 3–48.
- Chou, K.C., 1989a. Quasi-continuum models of twist-like and accordion-like low-frequency motions in DNA. *Biophys. J.* 56, 295–305.
- Chou, K.C., 1989b. Low-frequency resonance and cooperativity of hemoglobin. *Trends Biochem. Sci.* 14, 212.
- Chou, K.C., Chen, N.Y., 1977. The biological functions of low-frequency phonons. *Scientia Sinica* 20, 447–457.
- Chou, K.C., Zhang, C.T., Maggiora, G.M., 1994. Solitary wave dynamics as a mechanism for explaining the internal motion during microtubule growth. *Biopolymers* 34, 143–153.
- Ciblis, P., Cosic, I., 1997. The possibility of soliton/exciton transfer in proteins. *J. Theor. Biol.* 184, 331–338.
- Cosic, I., 1990. Resonant recognition model of protein–protein and protein–DNA interaction. In: Wise, D. (Ed.), *Bioinstrumentation and Biosensors*. Marcel Dekker, New York, pp. 475–510.
- Cosic, I., 1993. Correlation between predicted and measured characteristic frequency of chymotrypsin activation. *Proceedings of the 15th Annual Conference on IEEE Engineering in Medicine and Biological Society*, pp. 1541–1542.
- Cosic, I., 1994. Macromolecular bioactivity: is it resonant interaction between macromolecules?—Theory and applications. *IEEE Trans. Biomed. Eng.* 41, 1101–1114.
- Cosic, I., Birch, S., 1994. Photoreceptors having a similar structure but different absorptions can be distinguished using the RRM. *IEEEEMBS* 16, 265–266.
- Cosic, I., Vojisavljevic, V., Pavlovic, M., 1989a. Prediction of ‘hot spots’ in interleukin-2 based on information spectrum characteristics of growth regulating factors. *Biochimie* 71, 333–342.
- Cosic, I., Vojisavljevic, V., Pavlovic, M., 1989b. The relationship of the resonant recognition model to effects of low intensity light in cell growth. *Int. J. Radiat. Biol.* 56, 179–191.
- Davydov, A.S., 1973. The theory of contraction proteins under their excitation. *J. Theor. Biol.* 38, 559–569.
- Davydov, A.S., 1978. The role of protein in electron transport at large distances. *Phys. Status Solidi (B)* 90, 457–464.
- Davydov, A.S., 1982. *Biology and Quantum Mechanics*. Pergamon Press, New York.
- Hyman, J.M., McLaughlin, D.W., Scott, A.C., 1981. On Davydov’s alpha-helix solitons. *Physica D* 3, 23–44.
- Itoh, K., Shimanouchi, T., 1972. Vibrational spectra of crystalline formamide. *J. Mol. Spectrosc.* 42, 86–99.
- Kharkyanen, V.N., Petrov, E.G., Ukrainskii, I.I., 1978. Donor-acceptor model of electron transfer through protein. *J. Theor. Biol.* 73, 29–50.
- Nevskaia, N.A., Chirgadze, Yu.N., 1976. Infrared spectra resonance interactions of amide-I and amide-II vibrations in the α helix. *Biopolymers* 15, 637–648.
- Martel, P., 1992. Biophysical aspects of neutron scattering from vibration modes of proteins. *Prog. Biophys. Mol. Biol.* 57, 129–179.
- Scott, A.C., 1982. Dynamics of Davydov solitons. *Phys. Rev. A* 26, 578–595.
- Scott, A.C., 1991. Davydov’s soliton revisited. *Physica D* 51, 333–342.
- Sobell, H.M., Banerjee, A., Lozansky, E.D., Zhou, G.P., Chou, K.C., 1983. In: Clementi, E., Sarma, R.H. (Eds.), *Structure and Dynamics: Nucleic Acids and Proteins*. Adenine Press, New York, pp. 181–195.
- Takeo, S., 1983. Exciton soliton in one-dimensional molecular crystals. *Prog. Theor. Phys.* 69, 1798–1801.
- Takeo, S., 1984. Vibron soliton in one-dimensional molecular crystals. *Prog. Theor. Phys.* 71, 395–398.
- Takeo, S., 1985. Vibron soliton and coherent polarization in an exactly tractable oscillator-lattice system. *Prog. Theor. Phys.* 73, 853–873.
- Takeo, S., 1986. Vibron soliton and soliton-induced infrared spectra of crystalline acetanilide. *Prog. Theor. Phys.* 75, 1–14.
- Veljkovic, V., Slavic, I., 1972. General model of pseudopotentials. *Phys. Rev. Lett.* 29, 105–108.
- Zhou, G.P., 1989. Biological functions of soliton and extra electron motion in DNA structure. *Physica Scripta* 40, 698–701.