GENERAL QUESTIONS OF THERMODYNAMICS, STATISTICAL PHYSICS, AND QUANTUM MECHANICS

INTRINSICALLY INDUCED DEFORMATION OF A DNA MACROMOLECULE

P.P. KANEVSKA, S.N. VOLKOV

M.M. Bogolyubov Institute for Theoretical Physics, Nat. Acad. Sci. of Ukraine
(14b, Metrolohichna Str., Kyiv 03143, Ukraine; e-mail: kanevska@bitp.kiev.ua)

UDC 541.183
©2006

The mechanism of the macroscopic bending deformation of a macromolecular chain of the DNA type due to a conformational transformation of the double helix has been substantiated. In the framework of the two-component nonlinear model of conformational transformations of DNA, the shape of a deformed fragment and the energy of its deformation have been found. The estimations of the energy and the amplitude of the bending deformation demonstrate that the deformation of DNA induced by conformational transformations is energy-gained in comparison with that resulting from the elastic mechanism of a bending. Confronting the theoretical results with experimental ones obtained for the anomalous bending of the TATA-box fragment of DNA testifies that the mechanism of the intrinsically induced deformation may be realized for a certain sequence of DNA fragments.

1 Introduction

The study of mechanisms of the bending deformation of DNA is one of the challenging problems of the double helix structural mechanics. The bending of DNA accompanies such key processes of realization of a genetic information as the translation and transcription and also governs the packing of a macromolecule in chromosomes and viruses. The deformation of DNA is usually described in the framework of the elastic rod model, where the macromolecule is considered as a homogeneous chain that obeys the Hooke law. The flexibility of such a chain is characterized by the persistence length, the value of which is determined for macromolecules from experimental data and is well known for DNA [1–3]. In the elastic rod model, the fragments of DNA with their lengths comparable to the persistence one are allowed to bend fluctuationally by small angles (0.2—1° per base pair, depending on their sequence [3]), whereas the fragments shorter than the persistence length are assumed practically rigid.

In the recent years, there have been accumulated the experimental data which testify to the anomalously large — from the viewpoint of the elastic (linear) bending theories — deformations of relatively short fragments of the DNA macromolecule [4–12]. Anomalously large deformations of the DNA double helix occur in the course of the nucleic acid–protein recognition processes [4–9]. Cells demonstrate the formation of loops by a DNA chain, whose length is considerably shorter than the persistence one [10]. It is also known that DNAs in bacteriophages exist in the extremely compact conformation [11]. Responsible for such DNA deformations was considered the interaction between DNA and proteins. Nevertheless, the recent experiment [12] showed that short DNA fragments can form loops spontaneously and without stabilization of the latter by specific proteins.

It is important that, in all indicated experiments, no breaks of hydrogen bonds between base pairs were observed, and the DNA macromolecule remained in the double-stranded helical form. This effect cannot be explained in the framework of the elastic rod model, because the energy of deformation of DNA in such a way, as was observed in [6—9, 12], would lead to the denaturation of pairs of the DNA double chain.

Theoretical and experimental studies of the anomalous deformability of DNA fragments do not

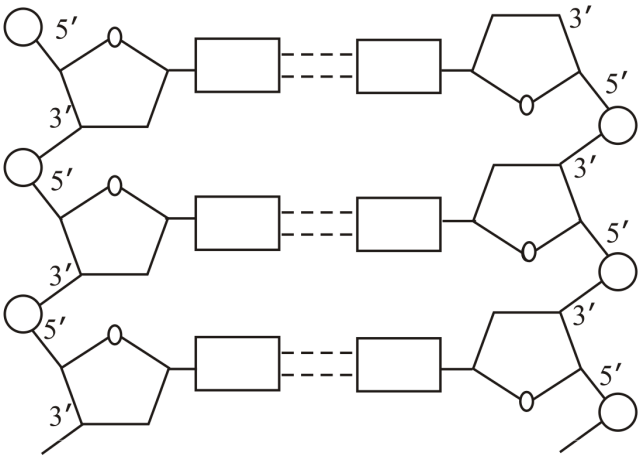

Fig. 1. Schematic illustration of the double helix structure of DNA. Circles stand for phosphate groups, pentagons for sugar rings, and rectangles for bases

explain the mechanism of a deformation of the macromolecule under environmental conditions. For example, the calculations of conformations carried out taking into account all atom-atom interactions allowed one to find possible ways [13] and conditions [14–16] for the structures with a substantially deformed double helix to be formed. But the evaluated energy that is necessary to deform the macromolecule's fragment coincides by value with the estimations of linear models and exceeds the energy of pair melting in a double-stranded helical structure [13].

The analysis of X-ray diffraction [17–20] and electrophoresis [21] data concerning the fragments of the DNA macromolecule proves the anomalous deformation of the double helix and reveals the change of the DNA conformation in its deformed section [17–21]. A possibility for the conformational transformations of the double helix to affect the DNA deformation was considered as long ago as in works [22–24]. However, the mechanism of the influence of internal conformational transformations on the double helix shape could not be clarified because of the complicated structure of the macromolecule.

At the same time, the results of works [17–24] allow one to assert that the anomalous deformation of the macromolecule could be induced by conformational reconstructions of the double helix. That is, the mechanism of the anomalous bending can be a result of the coupling between the conformational mobility and a deformation.

In order to study the influence of the conformational transformations of DNA on the shape of the double helix chain, the relevant nonlinear models were developed in works [25–30] in the framework of a phenomenological approach. In addition to the deformation like that of an elastic rod, those models make allowance for the internal conformational degree of freedom, as well as for the interrelation between the conformational transformations of the macromolecule and its deformation. In works [28–30], it has been shown that the conformational transitions in DNA induce a deformation of the macromolecule chain, which corresponds by its shape to the experimentally observed deformations of the macromolecule's fragments.

In our previous work [29], to understand the mechanism of the deformation induced by internal conformational transformations of the double helix, we built a model of conformational transformation of a DNA double helix fragment that includes a heteronomous conformation typical of the regulatory fragment of DNA (the TATA-box). Literature data on the deformation of the DNA TATA-box are most detailed [6–9, 13–18, 21], which allowed us to confront the theory and the experiment. In work [29], we showed that the deformation of DNA in the heteronomous conformation is described by the bistable macromolecule model. The results of simulations demonstrated that the shape of a deformation of the bistable chain corresponds to the experimentally determined deformation of the TATA-box, so that the developed approach can be used to elucidate the mechanism of deformations.

In this work, we have calculated the energy and the parameters of the deformation of the macromolecule induced by internal conformational excitations. We have substantiated the mechanism of the intrinsically induced deformation of the macromolecule and found the energy-gained ways for its realization in the course of the bending of a TATA-box of DNA.

2 Intrinsically Induced Deformation of a DNA Macromolecule

DNA is a polymorphic macromolecule, whose polymorphism is related to the conformational softness of sugar rings in the double helix chains (Fig. 1) [31]. Under environmental conditions, DNA has double helix conformations that are characterized by the $C2'$-*endo* form of sugar rings and belong to *B*-family. Under definite external conditions, DNA can occur in the *A*-family conformations, with the $C3'$-*endo* form of sugar rings. However, for certain DNA sequences, the conformation that is characterized by the coexistence of various sugar forms ($C2'$-*endo* and $C3'$-*endo*) in the

nucleotide pair of a monomer link of the macromolecule chain is observed. Such a conformation is called heteronomous. It is observed in alternative sequences, in particular, in a TATA-box of DNA.

In the framework of the heteronomous conformation of DNA, two conformational states may be realised. They differ from each other only by the alternative which chain of the double helix includes sugars in the *C*2′-*endo* form and which in the *C*3′-*endo* one (Fig. 2). Therefore, a monomer link of the chain can be in two stable states, which corresponds to the conformational bistability of the macromolecule. It was shown in work [29] that the transition between the heteronomous states of the DNA chain can be described in the framework of the two-component model of a bistable macromolecule, where this transition is accompanied by a bending of the macromolecule.

In order to elucidate the mechanism of the intrinsically induced deformation, consider the bending of a bistable macromolecule, which is a two-component molecular chain. The internal conformational component r describes the rotation of the base pair in the plane that is orthogonal to the main axis of the helix (twist); the rotation occurs owing to the change of the forms of sugars. The external component R describes the displacement of the monomer link resulting from the conformational transition.

In the continual approximation, the conformational mobility energy of a bistable macromolecule looks as

$$E = \frac{1}{2h}\int dz\big[M\dot{R}^2 + m\dot{r}^2 + \\ + gh^2R'^2 + kh^2r'^2 + \Phi(r) + 2\chi R'F(r)\big], \quad (1)$$

where M is the mass of the monomer link, m the reduced mass of nucleosides, h the distance between monomers along the z-axis; χ, g, and k are the parameter of the intercomponent coupling and the force constants of the external and internal subsystems, respectively. The first and second terms in expression (1) compose the kinetic energy of the link. The third and fourth terms describe the interaction along the chain in the nearest neighbor approximation. The fifth term in energy (1) describes the conformational transition energy

$$\Phi(r) = \varepsilon\left(1 - \frac{r^2}{a^2}\right)^2, \quad (2)$$

where a and ε are the distance and the transition barrier between the stable states. The nonlinear potential energy (2) was chosen in the degenerate form, because

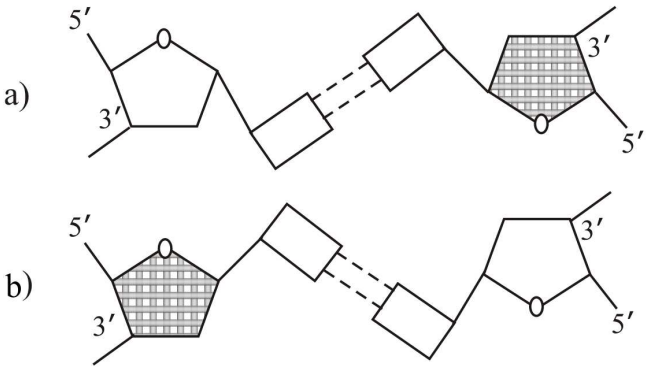

Fig. 2. Schematic illustration of two states of the heteronomous conformation of DNA. (a) The first link includes a sugar ring in the *C*3′-*endo* form, the other link includes a sugar ring in the *C*2′-*endo* form (hatched); (b) vice versa, the first link includes a sugar ring in the *C*2′-*endo* form (hatched), and the other link includes a sugar ring in the *C*3′-*endo* form

the degenerate bistability represents the states of the heteronomous DNA conformation [29], whose deformation is examined in this work. The sixth term describes the coupling between the external and internal components. The potential function $F(r)$ was constructed in such a way that it corresponds to the energy growth on the conformational transition owing to the correlation between the external and internal components:

$$F(r) = \left(1 - \frac{r^2}{a^2}\right). \quad (3)$$

Consider the possible static excitations of system (1). The system of equations for a static excitation looks like

$$gh^2R'' + \chi h\frac{\partial F(r)}{\partial r}r' = 0, \quad (4)$$

$$kh^2r'' - \frac{1}{2}\frac{\partial\Phi(r)}{\partial r} - \chi hR'\frac{\partial F(r)}{\partial r} = 0. \quad (5)$$

We search for those solutions of the system of equations (4) and (5) which have the asymptotic behavior that is characteristic of stable states: at $z \to \pm\infty$, the quantity $r \to \pm a, r' \to 0$, and $R' \to 0$. Then the static excitation looks like a two-component topological soliton:

$$r(z) = \pm a\,\mathrm{th}\left(\sqrt{Q}z\right), \quad (6)$$

$$R(z) = -\frac{\chi}{gh\sqrt{Q}}\,\mathrm{th}\left(\sqrt{Q}z\right), \quad (7)$$

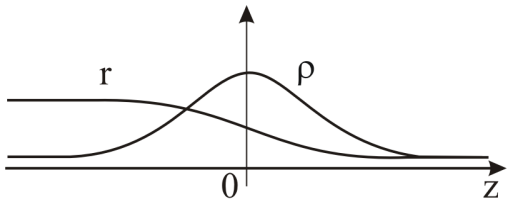

Fig. 3. Schematic illustration of the static conformational excitation of a two-component chain: r is the conformational state of the fragment, and ρ is the chain deformation

where $Q{=}\left(\varepsilon-\chi^2/g\right)/ka^2h^2$.

The shape of a static deformation of the macromolecule can be written down in the form

$$\rho(z)=hR'=-\frac{\chi}{g}\,\mathrm{ch}^{-2}\left(\sqrt{Q}z\right). \tag{8}$$

From this expression, one can see that the deformation of a bistable macromolecule with degenerate potential energy looks like a bell-shaped soliton. In Fig. 3, the profiles of a static deformation and the conformational state of a fragment belonging to the bistable chain are depicted schematically. The component r has the form of a kink and displays the conformational transition between the states of the heteronomous conformation. The deformation of the chain has the form of a bell-shaped soliton.

A deformation of type (8) that is induced by intrinsic conformational changes of the macromolecule will be called hereafter an intrinsically induced deformation. The form of a macromolecule chain deformation is governed by the kind of a non-linearity of the conformational component.

Formulas (6)—(8) demonstrate that the mechanism of the intrinsically induced bending would operate provided that

$$Q>0,\ \ \varepsilon-\frac{\chi^2}{g}>0. \tag{9}$$

Condition (9) determines the ranges of values of the model parameters, at which the intrinsically induced mechanism of deformation is feasible. In the case of a real macromolecule, this condition can be satisfied, first of all, owing to a significant variability of the chain stiffness parameter g and the relatively more stable parameters χ and ε. The stiffness of the double helix chain is governed by such factors as the composition and the sequence of nucleic bases, the conformational reconstruction trajectory, the solvent composition, the concentration of counterions, and interaction with other molecules.

3 Parameters of the Intrinsically Induced Deformation

Let us calculate the DNA bending amplitude according to the mechanism of the intrinsically induced deformation and compare it with experimental data for the bending of a heteronomous DNA in the TATA-box [7–9]. For this purpose, we have to define the parameters of the model for a DNA macromolecule in the heteronomous conformation. We suppose that the distance between monomer links of DNA h = 3 Å [32], and the magnitude of the transition barrier ε = 3 kcal/mol, which corresponds to the value experimentally determined for $B-A$ transitions in DNA [32, 33]. For the distance a between the conformational component wells and the stiffness k of the internal sublattice, we use the relevant values estimated in work [25] for $B-A$ transitions: $a\approx 1$ Å and $k\approx$ 1200 g/s^2. The stiffness g of the external subsystem of the macromolecule considered as a persistence chain corresponds to the experimentally known bending stiffness of DNA, $g=RTP/h$, where P is the persistence length. Hence, in our model, the stiffness of the external subsystem of the double helix considered as a persistence chain $g_p{=}7200$ g/s^2. To calculate g_p, we used the value P = 536 Å, which corresponds to the sequence occurring in the DNA TATA-box [3]. According to condition (9), for the intrinsically induced mechanism of bending to be activated, the inequality $\chi<\sqrt{\varepsilon g}$ must be valid. For further calculations, we put $\chi{=}0.6\sqrt{\varepsilon g_p}=231.6\times10^{-7}$ g × cm/s^2.

Consider the parameters of an intrinsically induced excitation of a bistable macromolecule. The width of conformationally excited solitons is proportional to the quantity $z_0{=}1/\sqrt{Q}$. We shall confine the region of the conformational excitation extension to the section where the structural displacement amplitude exceeds the amplitude of thermal fluctuations $\tilde{r}$. Our calculations show that, for the parameters quoted above, the amplitude of small displacements in one of the wells of the conformational potential energy $\tilde{r}$ = 0.38 Å. The number of pairs in the excited section is determined as $l=2L+1$, where

$$L=\frac{z_0}{h}\mathrm{arcth}\left(1-\tilde{r}/2a\right). \tag{10}$$

To calculate the angle, by which the chain deviates from the initial direction, we note that $\rho/h=dR/dz=$

$\tan\phi$. Then, the general deformation is calculated by the formula

$$\phi = \int\limits_{-Lh}^{Lh} \mathrm{arctg}(R')\frac{dz}{h}. \qquad (11)$$

Let us determine whether the mechanism of the intrinsically induced bending deformation has advantage over the elastic one for the given parameters of the model. We substitute solutions (6) and (7) into energy (1) and integrate over the excitation interval. Then, the energy of the intrinsically induced bending looks like

$$E_{\mathrm{ind}} = \left(\varepsilon - \frac{\chi^2}{g}\right) \int\limits_{-Lh}^{Lh} \frac{dz}{h}\,\mathrm{ch}^{-4}(\sqrt{Q}z). \qquad (12)$$

The elastic portion of the intrinsically induced bending energy has the form

$$E_R = \frac{gh^2}{2}\int_{-Lh}^{Lh}\frac{dz}{h}R'^2 = \frac{1}{2}\frac{\chi^2}{g}\int\limits_{-Lh}^{Lh}\frac{dz}{h}\,\mathrm{ch}^{-4}(\sqrt{Q}z). \qquad (13)$$

After calculating the energy values (12) and (13) for the macromolecule considered as a persistence chain, we obtain that $E_{\mathrm{ind}} > E_R$. Therefore, the intrinsically induced mechanism is not effective for those DNA fragments, the stiffness of which corresponds to the persistence chain. However, it should be taken into account that the bending stiffness is a parameter that depends on external factors. In this case, provided that the stiffness can accept other values in formulas (12) and (13), it turns out that the intrinsically induced mechanism becomes energy-gained if the stiffness $g < 1.5\chi^2/\varepsilon$. On the other hand, from condition (9), we obtain a restriction on g from below: $g > g_0 = \chi^2/\varepsilon$. Thus, the intrinsically induced mechanism turns out to be effective for those DNA fragments, the stiffness of which falls within the interval $(g_0, 1.5g_0)$.

Indeed, according to the experimental data, the bending stiffness for fragments with various sequences can be lower than its persistence value [34–36]. According to the results of studying the intrinsically induced bending of DNA in complexes with proteins by using the cyclization kinetics method, the bending force constant for the TATA-box is half as much as that for the irregular sequence in DNA [34]. The stiffnesses of DNA along the bending directions can also differ by several times, depending on the sequence [35, 36]. In work [36], it was found that, in the case of the TATA-box, the bending force constant along the major groove

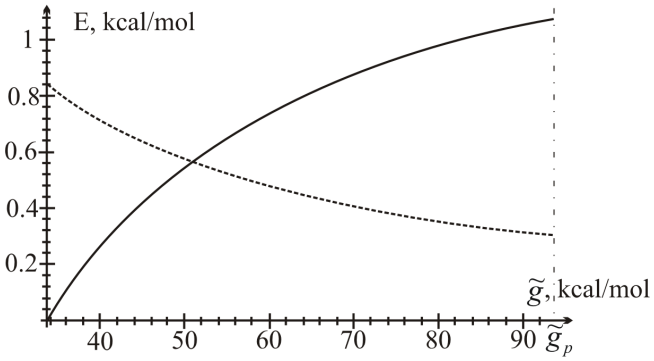

Fig. 4. Dependences of the average energy of the intrinsically induced bending of the DNA macromolecule $E_{\mathrm{ind}}/2L$ (solid curve) and the average energy of the elastic bending $E_R/2L$ (broken curve) on the bending stiffness value, $\tilde{g} = gh^2$

is 4 times lower than that along the minor groove. The neutralization of phosphate groups on the interaction with a protein can also bring about a reduction of the stiffness of the DNA TATA-box [13]. Moreover, the results of a recent experiment [12] show that some specific DNA sequences manifest the anomalously high flexibility.

Let us calculate the dependence of the energy of the intrinsically induced bending of the DNA macromolecule with given parameters on the bending stiffness. In Fig. 4, the solid curve exhibits the results of calculations of the dependence of the average energy of the intrinsically induced bending of the DNA macromolecule $E_{\mathrm{ind}}/2L$ on the bending stiffness within the interval from the value corresponding to the system bistability threshold, $\tilde{g_0}{=}g_0h^2$, to the value corresponding to the bending of the double helix as a persistence chain, $\tilde{g_p} = g_ph^2$. As is seen from the results of calculations, at $\tilde{g} \to \tilde{g_0}$, the energy of the intrinsically induced bending diminishes down to 0. The broken curve in Fig. 4 demonstrates the calculated variation of the elastic deformation energy $E_R/2L$ of the obtained conformational excitation.

In order to explain the DNA bending mechanism by intrinsic conformational changes, let us determine some quantitative characteristics of the static excitation and the bending deformation of a DNA fragment with the heteronomous conformation.

The table presents the results of calculations of the characteristics of the intrinsically-induced excitation carried out for fragments with various lengths: from 4 to 10 base pairs. In the table, l is the number of base pairs in the excited section of the macromolecule, ϕ is the bend angle of the macromolecule chain at the excitation section, and E_{ind} is the excitation energy. For the sake

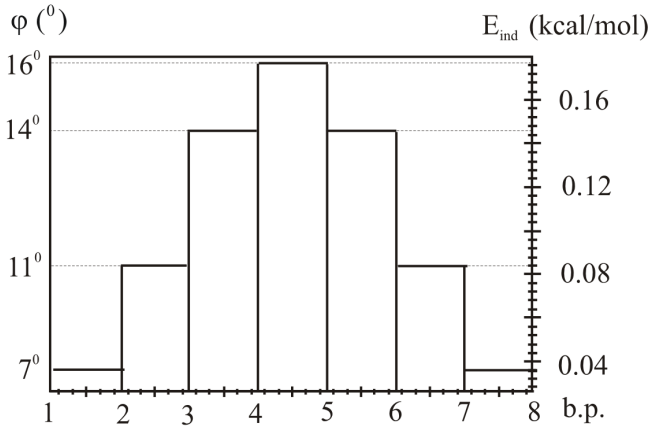

Fig. 5. Bend angle and energy distributions on the intrinsically induced bending of a sequence of 8 base pairs by a total angle of 80°

of comparison with the intrinsically induced mechanism of bending, we also calculated the energy of a chain deformation in the framework of the elastic rod model, $E_{\rm el}$=$gL\,(\phi/2L)^2$, with the corresponding stiffness values. The table also presents the portion E_R of the total bending energy which describes the elastic energy of the excited chain.

According to the table data, the amplitude and the width of the excitation increase as the bending stiffness becomes lower. In so doing, the energy of the intrinsically induced bending decreases, while the energy calculated in the framework of the elastic rod model and the elastic portion of the intrinsically induced deformation increase rather quickly, notwithstanding that the bending stiffness is more than twice lower than the bending stiffness of the double helix considered as a persistence chain.

The data quoted in the table testify that the 8-base-pair fragment of the macromolecule can be deformed to the 80°-bend. The angle deformation that was observed in the experiment for a TATA-box 8 base pairs in length was also of about 80° [7–9].

Also important is to analyze the energy distribution of the static excitation in the deformed fragment. In Fig. 5, we plotted the results of calculations of the bend angle and bend energy distributions along an 8-base-pair excited section of the macromolecule, which is bent according to the intrinsically induced bending mechanism by a total angle of 80°. According to the figure, most deformed is the central part of the fragment. At the ends of the fragment, the deformation brings about displacements, which correspond, by the order of magnitude, to fluctuations $\tilde{r}$ in the internal component. Thus, the energy distribution testifies that the intrinsically induced bending mechanism can be realized in DNA without violating the double-stranded structure of the double helix.

Characteristics of the intrinsically induced excitation

l (b.p)	ϕ (degree)	$E_{\rm ind}$ (kcal/mol)	$E_{\rm el}$ (kcal/mol)	E_R (kcal/mol)	g ($\times g_p$)
4	25	1.64	1.60	1.73	0.530
6	55	0.98	3.46	3.76	0.407
8	80	0.70	5.07	5.54	0.383
10	108	0.54	6.86	7.48	0.373

4 Conclusions

A two-component model for conformational transformations of a bistable macromolecule, which describes the shape and the amplitude of a macroscopic deformation in the DNA double helix chain, has been built.

It has been shown that the proposed mechanism of the intrinsically induced deformation can explain an anomalously large bending, which is observed in the TATA-box of DNA. The mechanism proposed attributes the ability of the fragment to anomalously bend to the properties of the DNA structure with a definite sequence. This conclusion correlates with the results of the recent experiment on studying the properties of the DNA bending deformation [12].

The energy efficiency of the intrinsically induced deformation means that the bend of heteronomous sections in the course of the DNA packing is related to a conformational transition in the chain structure. The mechanism proposed can be applied to describe the DNA compacting in nucleosomas and viruses.

Thus, the proposed mechanism of the intrinsically induced bending explains substantial deformations of bistable macromolecules and can be used for the quantitative estimation of the bending parameters of a polymorphic DNA.

1. *Hagerman P.J.* // Annu. Rev. Biophys. Chem. — 1988. — **17**. — P. 265—286.
2. *Frank-Kamenetskii M.D.* // Itogi Nauki i Tekhn. Ser. Mol. Biol. — 1979. — **15**. — P. 42—73.
3. *Matsumoto A., Olson W.K.* // Biophys. J. — 2002 — **83**, N 1. — P. 22—41.
4. *Schultz S.C., Shields G.C., Steitz T.A.* // Science. — 1991. — **253**, N 5023. — P. 1001—1007.
5. *Ansari A.Z., Bradner J.E., O'Halloran T.V.* // Nature. — 1995. — **374**, N 6520. — P. 371 — 375.
6. *Klug A.* // Ibid. — 1993. — **365**. — P. 486—487.

7. *Kim Y., Geiger J.H., Hahn S., Sigler P.B.* // Ibid. — N 6446. — P. 512—520.
8. *Kim J.L., Burley S.K.* // Nature Struct. Biol. — 1994. — **1**, N 9. — P. 638—653.
9. *Nikolov D.B., Burley S.K.* // Proc. Natl. Acad. Sci. USA. — 1997. — **94**. — P. 15—22.
10. *Schleif R.* // Annu. Rev. Biochem. — 1992. — **61**. — P. 199—223.
11. *Richards K.E., Williams R.C., Calendar R.* // J. Molec. Biol. — 1973. — **78**. — P. 255—259.
12. *Cloutier T.E., Widom J.* // Molec. Cell. — 2004. — **14**. — P. 355—362.
13. *Lebrun A., Shakked Z., Lavery R.* // Proc. Natl. Acad. Sci. USA. — 1997. — **94**. — P. 2993—2998.
14. *Miaskiewicz K., Ornstein R.L.* // J. Biomol. Struct. and Dynam. — 1996. — **13**, N 4. — P. 593—600.
15. *Lavery R., Flatters D.* // Biophys. J. — 1998. — **75**. — P. 372—381.
16. *Flatters D., Young M., Beveridge D.L., Lavery R.* // J. Biomol. Struct. and Dynam. — 1997. — **14**, N 6. — P. 757—765.
17. *Guzikevich-Guerstein G., Shakked Z.* // Nature Struct. Biol. — 1996. — **3**. — P. 32—37.
18. *Juo Z.S., Chiu T.K., Leiberman P.M. et al.* // J. Molec. Biol. — 1996. — **261**. — P. 239—254.
19. *Lu X., Shakked Z., Olson W.* // Ibid. — 2000. — **300**. — P. 819—840.
20. *Olson W.K., Zhurkin V.B.*// Curr. Opinion in Struct. Biol. — 2000. — **10**. — P. 286—297.
21. *Starr D.B., Heopes B.C., Hawley D.K.* // J. Molec. Biol. — 1995. — **250**. — P. 434—446.
22. *Sobell H.M.,Tsai Ch., Gilbert S.G. et al.* // Proc. Natl. Acad. Sci. USA. — 1976. — **73**. — P. 3068—3072.
23. *Manning G.S.*// Biopolymers. — 1981. — **20**. — P. 1261—1270.
24. *Crothers D.M., Haran T.E., Nadeau J.G.* // J. Biol. Chem. — 1990. — **265**, N 13. — P. 7093—7096.
25. *Volkov S.N.* // J. Theor. Biol. — 1990 — **143**. — P. 485 — 496.
26. *Manevich L.I., Savin A.V., Smirnov V.V., Volkov S.N.* // Usp. Fiz. Nauk. — 1994. — **164**, N 9. — P. 937—958.
27. *Volkov S.N.* // Biofiz. Visn. (Kharkiv). — 2000. — **7**, N 2. — P. 7—15.
28. *Volkov S.N.* // Ibid. — 2003. — **1**, N 12. — P. 5—12.
29. *Kanevska P.P, Volkov S.N.* // Ibid. — **2, N** 13. — P. 48—55 (e-print: arXiv/q-bio/BM/0412025).
30. *Volkov S.N.* // J. Biol. Phys. — 2005. — **31**, N 3.
31. *Saenger W.* Principles of Nucleic Acid Structure. — Berlin: Springer, 1984.
32. *Ivanov V.I.* // Molek. Biol. — 1983. — **17**, N 3.— P. 616—621.
33. *Olson W.K., Sussman J.L.* // J. Am. Chem. Soc. — 1982 — **104**, N 1. — P. 270—278.
34. *Zhang Y., Xi Z., Hegde R.S. et al.* // Proc. Natl. Acad. Sci. USA. — 2004. — **101**, N 22. — P. 8337—8341.
35. *Zhurkin V.B.* // Molek. Biol. — 1983. — **17**, N3. — P. 622—638.
36. *Akiyama T., Hogan M.E.* // Proc. Natl. Acad. Sci. USA. — 1996. — **93**. — P. 12122—12127.

Received 14.12.05.
Translated from Ukrainian by O.I. Voitenko

ВНУТРІШНЬО ІНДУКОВАНА ДЕФОРМАЦІЯ МАКРОМОЛЕКУЛИ ДНК

П.П. Канєвська, С.Н. Волков

Р е з ю м е

Обґрунтовано механізм макроскопічної деформації згину ланцюжка макромолекули типу ДНК за рахунок конформаційних перетворень структури подвійної спіралі. В рамках двокомпонентної нелінійної моделі конформаційних перетворень ДНК знайдено форму деформованого фрагмента та енергію утворення деформації. Оцінка енергії та величини деформації згину показує, що індукована конформаційними змінами деформація ДНК є енергетично вигідною в порівнянні з пружним механізмом згинання. Зіставлення результатів теорії з експериментом для аномального згинання фрагмента TATA-бокса ДНК говорить про можливість реалізації механізму внутрішньо індукованої деформації для фрагментів ДНК певної послідовності.