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Tunable spin-selective transport through DNA with mismatched base pairs

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Abstract

In our theoretical analysis of spin-selective transport through a homogeneous poly(G)–poly(C) DNA we explore the influence of a mismatched base pair in the DNA chain. The spin polarization of the electrical current through DNA is strongly sensitive to the presence of the mispair in a DNA with less than 20 base pairs. Replacing a canonical G–C base pair by a G–A mispair in homogeneous DNA can strongly decrease, increase up to an order of magnitude, or even change the sign of spin polarization of the electrical current. The mispair induced spin-selective current through DNA also depends on the location of the mispair within the DNA.

Keywords: DNA, spintronics, mispair

(Some figures may appear in colour only in the online journal)

1. Introduction

In the rapidly evolving field of spintronics, molecular spintronics, i.e. spin-based electronics but with organic materials, has made huge strides[3]. In this context, a recent discovery of spin-selective transport [2, 3] through DNA deposited on a gold substrate has opened up an unique possibility to utilize a molecule with widely controllable properties as a spin valve. The spin polarization of the electrical current through DNA is relatively large and for long molecules it can be as large as 60%. DNA consists of four nucleotides, guanine (G), adenine (A), cytosine (C) and thymine (T) that form the canonical Watson–Crick base pairs, G–C and A–T through weak hydrogen bonds and are organized in a helical DNA structure. It is the specific helical structure of DNA that produces the observed strong spin polarization of the electrical current through DNA [4–8]. The strongest spin effect is realized in a homogeneous molecule, e.g. the poly(G)–poly(C) DNA, but incorporation of a defect in such a homogeneous structure can change the spin transport and correspondingly the spin selectivity. Therefore, by introducing defects in a homogeneous DNA structure, spin selective transport through the molecule can be tuned. In [8] it was shown that spin sensitivity of electrical current through DNA strongly depends both on the DNA sequence and on the point mutation of the base pair in the end segment of DNA.

During the past few decades there was a growing interest on the problem of charge transport through DNA. This interest is due to both fundamental importance of this problem and possible technological applications of DNA as a wire in nanoscale devices. The long-range charge transport through DNA has been observed experimentally [9–13] where the charge has been transferred over significant distance $\sim$100 Å. A few mechanisms of charge transfer through the DNA molecule [14, 15] were proposed to explain experimental observations of the charge migration through DNA. The main mechanisms are superexchange and hopping, which include both the polaron hopping and thermally activated hopping. These are the types of incoherent charge transfer, i.e. the transport in this case is determined by incoherent charge dynamics, which includes thermal relaxation and thermal fluctuations. The charge transfer is also strongly affected by dynamical disorder of the DNA molecule, which is introduced through time-dependent fluctuations of the geometrical structure of DNA. The dynamical disorder and the rate of incoherent transport depend on the temperature of DNA and the surrounding medium. At low temperature, $<$100 K (the characteristic energy of the phonon dynamics is $\sim$0.01 eV [16]), these mechanisms of charge transfer are

[3] For reviews, see e.g. [1].

exponentially suppressed. In this case the charge transfer becomes coherent and is described by the coherent dynamics. The corresponding Hamiltonian has a tight-binding form which describe the coherent coupling of the states of the nearest neighbor base pairs. This tight-binding approach to the charge transport through DNA has been widely used in theoretical modeling of the charge dynamics [15, 17–23]. In what follows, first we consider a similar tight-binding model, considering only the coherent charge dynamics and disregarding thermal fluctuations in the DNA molecule and the related polaronic effects, which can be suppressed at low temperatures. Then to study the effect of thermal fluctuations and vibrations of the surrounding media, we introduce in the coherent Hamiltonian the disordered static terms and consider the average characteristics of the electron transport.

Electron transport through DNA is mainly determined by two parameters: the on-site energy, i.e. the electron energy on a given nucleotide and the hopping integrals between the nearest-neighbor nucleotides. Introduction of a defect (in a homogeneous DNA) in the form of a canonical base pair changes both these parameters. At the same time incorporation of a mismatched base pair can change only the hopping integrals, while keeping the same on-site energies for some of the nucleotides. In this case the spin properties of DNA can be fine tuned. The electrical current and the charge transfer through DNA containing different types of mismatched base pairs have been studied experimentally [24–26, 28] and theoretically [29, 30]. It was shown that mispairs (such as T–G, C–A, or G–A) can cause an order of magnitude change in the electrical conductance through a short DNA [24] or even suppress the charge transfer through DNA [26]. In [27] it was shown that the local defects in λ-DNA (nicked DNA with a gap between 3′ and 5′ sites) also strongly modifies the transport through the molecule.

We consider the type of mispairs that causes only local distortion of DNA, i.e. the G·A type mispair in two conformations: G(anti)·A(anti) and G(anti)·A(syn) [31]. These mispairs are known to change the thermodynamical properties of DNA [31–33] and can also influence the magnetic properties [34] and transport through DNA [23]. Here we report on the effects of these mispairs on the spin-selective transport through DNA. The paper is organized as follows. In section 2, we introduce the tight-binding model of the DNA molecule and the main parameters of the model. In section 3, we present the results of the calculations for homogeneous DNA and DNA with thermal disorder. The concluding discussion of the obtained results is presented in section 4.

2. Theoretical framework

We evaluate the conductance through a DNA molecule connected to two leads (electrodes). Our chosen DNA has a finite length and contains one mismatched base pair. Below we consider DNA molecules with the number of base pairs between 5 and 20. Only for such short DNA (with the number of base pairs less than 20), the presence of mispair strongly modifies the spin transport through DNA. Below we also show that for that range of the DNA lengths, the effect of mispairs strongly depends on the DNA length. The Hamiltonian of a DNA molecule connected to two electrodes has the form

$$\mathcal{H} = \mathcal{H}_t + \mathcal{H}_{\text{leads}} + \mathcal{H}_{\text{DNA-leads}}, \tag{1}$$

where $\mathcal{H}_t$ is the tight-binding Hamiltonian which describes the electron transfer within DNA, $\mathcal{H}_{\text{leads}}$ is the Hamiltonian of charge carriers in the right and left leads and $\mathcal{H}_{\text{DNA-leads}}$ describes coupling between DNA and the leads.

We describe the charge transfer within DNA by the double-stranded tight-binding model [15, 20, 35] of DNA. For each strand we consider both the HOMO and LUMO orbitals of the corresponding nucleotides, i.e. for each nucleotide we introduce one HOMO and one LUMO orbitals. With these orbitals we can treat both the electron and hole channels of the charge transport through DNA. The DNA model includes both the interstrand and intrastrand charge hoppings between the nearest nucleotides and the spin–orbit interaction specific for the helix structure of DNA. The corresponding Hamiltonian is

$$\begin{aligned}\mathcal{H}_t = &\sum_{\alpha,K,m,s} \varepsilon_{\alpha,K,m} n_{\alpha,K,m,s} + \sum_{\alpha,K,m,s} t_{\alpha K}^{(m,m+1)} a^{\dagger}_{\alpha,K,m,s} a_{\alpha,K,m+1,s} \\ &+ \sum_{K,m,s} t_{\perp}^{(m,K)} a^{\dagger}_{1,K,m,s} a_{2,K,m,s} \\ &+ \sum_{\alpha,K,m,s,s_1} t_{\text{SO}} a^{\dagger}_{\alpha,K,m,s} \left(\sigma^{(\alpha)}_{m,ss_1} + \sigma^{(\alpha)}_{m+1,ss_1}\right) a_{\alpha,K,m,s_1} \\ &+ \text{h.c.},\end{aligned} \tag{2}$$

where $\alpha = 1, 2$ is the number of the strand, $K = H, L$ labels HOMO and LUMO orbitals, $a_{\alpha,H,m,s}$ and $a_{\alpha,L,m,s}$ are the annihilation operators of an electron with spin $s = \pm 1$ on site (base pair) m at the HUMO and LUMO orbitals in strand α, respectively, $\varepsilon_{\alpha,H,m}$, $\varepsilon_{\alpha,L,m}$ are the corresponding on-site energies, $n_{\alpha,K,m,s} = a^{\dagger}_{\alpha,K,m,s} a_{\alpha,K,m,s}$ and $t_{\alpha K}^{(m,m+1)}$ are the intrastrand hopping integrals between the nearest base pairs m and $m+1$ in strand α, $t_{\perp}^{(m,K)}$ is the interstrand hopping integral within the same base pair m and $K = H, L$ orbitals, t_{SO} is the spin–orbit coupling of the nearest base pairs within the same strand. We assume that the spin–orbit parameter t_{SO} is the same for all orbitals and all nucleotides. The value of the spin–orbit parameter is not well known and is difficult to calculate. It is also very sensitive to the charge distribution in DNA and the properties of the surrounding medium. In [5] the spin–orbit parameter was chosen to be $t_{\text{SO}} = 0.01$ eV, while in [6] this parameter was taken as $t_{\text{SO}} \approx 0.002$ eV. In our present work we have assumed that the spin–orbit parameter takes the value $t_{\text{SO}} = 0.005$ eV, which is in the range of the values considered in [5, 6].

The spin–orbit part of the Hamiltonian is expressed in terms of the matrices [5] $\sigma^{(1)}_{m+1} = \sigma(m\Delta\phi)$ and $\sigma^{(2)}_{m+1} = \sigma(m\Delta\phi + \pi)$, where $\sigma(\phi) = \sigma_x \sin\phi \sin\theta - \sigma_y \cos\phi \sin\theta + \sigma_z \sin\theta$ with $\theta = 0.66$ rad, $\Delta\phi = \pi/5$ and the Pauli matrices σ_x, σ_y, σ_z. The index $m = 1, \ldots, N$ in equation (2) is the number of the base pair and N is the total number of base pairs.

The Hamiltonian of the leads describes the electrons in the right and left contacts and have the form

$$\mathcal{H}_{\text{leads}} = \sum_{Q,k,s} \varepsilon_{Q,k} c^{\dagger}_{Q,k,s} c_{Q,k,s}, \tag{3}$$

where $Q = L, R$ corresponds to left and right contacts, $c_{Q,k,s}$ are the annihilation operators of an electron with spin $s = \pm 1$ and wavevector k in the contact $Q = L, R$. The electron system in each lead is characterized by the corresponding chemical potential, μ_L and μ_R. The bias voltage across DNA is then introduced as $V_b = \mu_L - \mu_R$. Below we calculate the differential conductance, which is defined at $V_b \to 0$ at the variable energy $\varepsilon = \mu_L = \mu_R$, controllable by the gate voltage.

The coupling of the DNA chain to the leads is introduced through hopping between the HOMO and LUMO states of the first base pair of DNA and the left contact and between the HOMO and LUMO states of the N-th base pair and the right contact. The corresponding Hamiltonian is

$$\mathcal{H}_{\mathrm{DNA-leads}} = \sum_{\alpha,K,k,s} V_{1,k} c^{\dagger}_{L,k,s} a_{\alpha,K,1,s} + \mathrm{h.c.} + \sum_{\alpha,K,k,s} V_{N,k} c^{\dagger}_{R,k,\sigma} a_{\alpha,K,N,s} + \mathrm{h.c.}, \quad (4)$$

where $V_{k,1}$ and $V_{k,N}$ are the hopping integrals which characterize the coupling of the DNA chain to the leads. In the above expression we assumed that the coupling strength is the same for both HOMO and LUMO states of DNA. In the following we also assume that the hopping integrals $V_{1,k}$ and $V_{N,k}$ are the same for all states in the contacts, i.e. $V_{1,k} = V_{N,k} = V_0$.

We evaluate the conductance through DNA for two spin components, $s = \pm 1$. The conductance at the electron energy ε is calculated from the Landauer expression

$$G_s = \frac{2e^2}{h} \mathrm{Tr}\left[\mathbf{G}^r(\varepsilon)\mathbf{\Gamma}^L\mathbf{G}^a(\varepsilon)\mathbf{\Gamma}^{R,s}\right], \quad (5)$$

where $s = \pm 1$ is the spin component, $\mathbf{G}^r$ and $\mathbf{G}^a$ are retarded and advanced Green functions of electrons in DNA, $\mathbf{\Gamma}^{R,+1}$, $\mathbf{\Gamma}^{R,-1}$, $\mathbf{\Gamma}^R = \mathbf{\Gamma}^{R,+1} + \mathbf{\Gamma}^{R,-1}$ and $\mathbf{\Gamma}^L$ are the level-width matrices, determining the coupling of the DNA states through the continuous states of the right and left contacts: $\Gamma^L_{p_1;p_2} = 2\pi\rho_L V_0^2 \delta_{i_1,1}\delta_{i_2,1}\delta_{\sigma_1,\sigma_2}$ and $\Gamma^{R,s}_{p_1;p_2} = 2\pi\rho_R V_0^2 \delta_{i_1,N}\delta_{i_2,N}\delta_{s_1,s}\delta_{s_2,s}$. Here $p_i = \alpha_i, K_i, i_i, s_i$ is the combined index and $\rho_{(L,R)}$ is the density of states in the left and right contacts. In our present calculations we assume that $\Gamma^R = \Gamma^L = 0.02$ eV. The coupling of the DNA molecule to the contacts is realized as a tunneling process and is described by the Hamiltonian (4) of the tight-binding type. In this case the only effect of the coupling constants Γ on the results, discussed below, is broadening of the energy levels of the DNA molecule, which results in smoothing of the energy dependence of the current through DNA and corresponding conductance. The values of the parameters Γ were chosen so that in these energy dependencies the levels of the DNA molecule can be resolved. The parameters Γ do not affect the amplitude

The retarded and advanced Green functions can be found from the following expression

$$\mathbf{G}^{r/a}(\varepsilon) = \left[\varepsilon - H_t \pm \frac{1}{2}\left(\mathbf{\Gamma}^L + \mathbf{\Gamma}^R\right)\right]^{-1}. \quad (6)$$

Table 1. The HOMO and LUMO energies (ε_{H} and ε_{L}) of the single nucleobases (in eV).

	Guanine	Adenine	Thymine	Cytosine
HOMO	−9.40	−9.79	−10.46	−10.27
LUMO	−5.37	−5.85	−6.57	−5.90

Table 2. The values of the intrastrand charge transfer integrals (in eV) for different DNA strands, where G(anti) and A(anti) means that guanine and adenine are the part of the mismatched base pair G(anti)·A(anti), while G(syn) and A(syn) means that the guanine and adenine are the parts of G(anti)·A(syn) mispair.

(X_1–X_2)	G–G	C–C	G–G(anti)	G–G(syn)	C–A(anti)	C–A(syn)
H–H	0.056	−0.041	−0.204	0.069	−0.072	0.104
L–L	−0.122	−0.145	−0.204	0.244	−0.103	0.122

Note: Here 'H' and 'L' indicates HOMO and LUMO orbitals, respectively.

Table 3. The values of the interstrand charge transfer integrals (in eV) for different DNA base pairs.

(X_1–X_2)	G–C	G(anti)–A(anti)	G(anti)–A(syn)
H–H	−0.129	−0.125	0.035
L–L	0.065	0.087	−0.071

Note: Here letters H and L means HOMO and LUMO orbitals, respectively.

The conductance calculated from equation (5) have different values for different components of spin, which illustrates the spin-selective transport through DNA. We characterize this spin asymmetry by the following parameter $\Delta G/G = \left(G_{+1} - G_{-1}\right)/\left(G_{+1} + G_{-1}\right)$, which indicates the spin polarization of the electrical conductance. The tight-binding Hamiltonian (2) of the DNA molecule is characterized by the hopping integrals and the on-site energies. These parameters were calculated via the *ab initio* approach [23, 34] and are shown in tables 1–3.

To model the effects of the surrounding medium and the effects of phonons, i.e. vibrations of the DNA molecule on the electron dynamics, we consider a static disordered Hamiltonian with random on-site energies, $\varepsilon_{\alpha,K,m}$ and random interstrand and intrastrand hopping integrals, t. We do not consider the effect of vibrations of the molecule on the strength and profile of the spin–orbit interaction, assuming that it is constant. We assume that the random variables (on-site energies and hopping integrals) at different base pairs are uncorrelated and are described by the Gaussian distribution functions

$$P_\epsilon(\epsilon_{\alpha,K,m}) = \frac{1}{\sigma_E\sqrt{2\pi}} \exp\left[-\frac{(\epsilon_{\alpha,K,m} - \epsilon^{(0)}_{\alpha,K,m})^2}{2\sigma_E^2}\right] \quad (7)$$

and

$$P_t(t) = \frac{1}{\sigma_t\sqrt{2\pi}} \exp\left[-\frac{(t - t^{(0)})^2}{2\sigma_t^2}\right] \quad (8)$$

for on-site energies and hopping integrals, respectively. Here $\varepsilon^{(0)}_{\alpha,K,m}$ and $t^{(0)}$ are the equilibrium values (see tables 1–3)

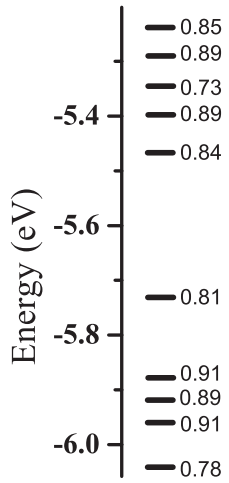

Figure 1. Single-particle energy spectra of LUMO orbitals (electron channel) of DNA with a G(anti) ·A(anti) mispair. The number of base pairs in the molecule is 5. Numbers near the lines are the average values of the z-component of spin.

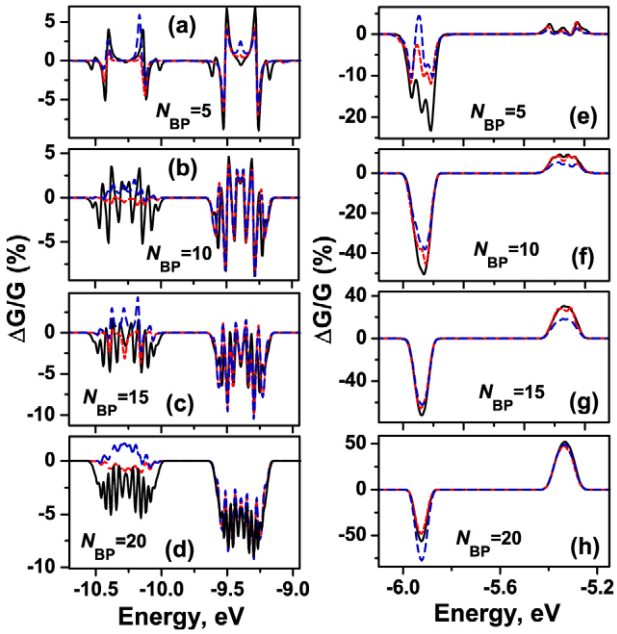

Figure 2. The energy dependence of the spin polarization in (a)–(d) the hole channel (HOMO orbitals) and (e)–(h) the electron channel (LUMO orbitals) for poly(G)–poly(C) DNA (black lines) and for DNA with one mispair: G(anti)·A(anti) (red short-dashed lines) and G(anti)·A(syn) (blue long-dashed lines). The mispair is placed at the midpoint of DNA. The numbers N_{BP} of base pairs in DNA are shown in each panel.

of the corresponding parameters. The distribution functions are characterized by standard deviations σ_E for the on-site energies and σ_t for the hopping integrals. We assume that the standard deviation σ_E is the same for all on-site energies and the standard deviation σ_t is the same for all interstrand and intrastrand hopping integrals.

For a Hamiltonian with disordered on-site energies and the hopping integrals the spin polarization of the electrical conductance is defined as $\Delta\langle G\rangle/\langle G\rangle = \left(\langle G_{+1}\rangle - \langle G_{-1}\rangle\right)/\left(\langle G_{+1}\rangle + \langle G_{-1}\rangle\right)$, where the average conductances $\langle G_\alpha\rangle$ are calculated as the averages over 1000 realizations of the random DNA system.

The non-zero value of spin-polarization (as defined above) of the electrical conductance, which was demonstrated experimentally in [2, 3], is a manifestation of the spin-dependent conductance of DNA. This is an intrinsic property of DNA and is due to the combination of the spin–orbit coupling within DNA and the chiral structure of DNA, which generates the chiral electron states. In figure 1 the energy spectra of the LUMO orbitals (electron channel) is shown for poly(G)–poly(C) DNA with one G(anti)·A(anti) mispair at the midpoint of DNA. Each level is two-fold degenerate. The numbers near the lines are the average values of the z-component of the electron spin. Without spin–orbit interactions these values are 1 for all states. Finite spin–orbit interactions result in correlation of electron spin with direction of propagation along the DNA molecule.

3. Results and discussion

The spin polarization $\Delta G/G$ for short DNA molecules are shown for different energy ranges in figure 2. The left and right columns correspond to HOMO (hole channel) and LUMO (electron channel) orbitals, respectively. The spin polarizations of poly(G)–poly(C) DNA molecules are shown as black lines. The red and blue lines correspond to two types of mismatched base pairs: G(anti)·A(anti) (red lines) and G(anti)·A(syn) (blue lines). The mispairs are placed at the midpoint of DNA. The effect of the mispair on spin polarization is due to two modifications of the DNA Hamiltonian introduced by the mispair. These are the changes of the on-site energies (due to change of the type of nucleotide) and the local intrastrand and interstrand hopping integrals. Since we assume that the on-site energy of guanine in G(anti)·A(anti) and G(anti)·A(syn) mispairs is the same as the energy of guanine in GC base pairs, then, in the presence of the mispair only the on-site energy of cytosine is replaced by the on-site energy of adenine.

For both the hole and electron channels there are two regions with nonzero spin polarizations (figure 2). These regions correspond to two HOMO and two LUMO orbitals of the two strands of DNA. The discrete energy spectrum of the finite DNA molecule results in additional oscillations in the dependence of the spin polarization on the energy (figure 2). Those oscillations are visible in the hole channel, but they cannot be resolved in the electron channel. This is due to smaller intrastrand hopping integrals of the HOMO states. For example, for the C–C strand the intrastrand hopping integrals are -0.041 eV and -0.145 eV in the hole and electron channels, respectively.

For the hole channel (HOMO orbitals) (figures 2(a)–(d)), the effect of mispair on the spin polarization strongly depends on the size of DNA. For a short DNA molecule (figure 2(a)) the spin polarization remains almost the same with the inclusion of the mispairs. With increasing size of DNA from $N_{\mathrm{BP}} = 5$ to $N_{\mathrm{BP}} = 20$, strong changes in the spin polarization due to mispairs are clearly visible. In the low-energy region,

$\Delta G/G$ is decreased in the presence of both types of mispairs (figures 2(*b*)–(*d*)). For $N_{\rm BP} = 20$ (figure 2(*d*)), the G(anti)·A(anti) mispair reduces the spin polarization by almost a factor of 5, while the G(anti)·A(syn) changes the sign of spin polarization. Such a strong modification of the spin polarization is due to changes in the on-site energy of the DNA molecule. The low-energy range corresponds to the C-strand in the poly(G)–poly(C) DNA. In the presence of the mispair the on-site energy within this strand is strongly modified from −10.27 eV (for cytosine) to −9.79 (for adenine), which reduces the spin polatization.

For the higher energy strand (the G-strand in poly(G)–poly(C) DNA) the mispair does not change the on-site energy. In that case the main effect of the mispair is to modify the hopping integrals. The effect of a mispair on the spin polarization is then small (figures 2(*a*)–(*d*)). For the electron channel (LUMO orbitals) (figures 2(*e*)–(*h*)), there are also two main (downward) peaks which now correspond to two LUMO orbitals of the two strands. For the higher energy peak, which corresponds to the C-strand in poly(G)–poly(C) DNA, the mispair changes the on-site energy but this change is small: from −5.90 eV (for cytosine) to −5.85 (for adenine). As a result, in this case there is no strong reduction of the spin polarization, while such a reduction is clearly visible in the hole channel (see lower energy peaks in figures 2(*b*)–(*d*)). For the electron channel the reduction of spin polarization is visible only for the G(anti)·A(syn) mispair and for intermediate DNA lengths, $N_{\rm BP} = 10$ and 15 (figures 2(*f*) and (*g*)). In the low-energy peak where the mispairs changes only the local hopping integrals, the spin polarization is strongly modified by the mispairs only for small DNA molecules (figure 2(*e*)). In this case the spin polarization is strongly reduced and for the G(anti)·A(syn) mispair the spin polarization even changes sign. The general behavior of spin polarization shown in figure 2 illustrates that the spin polarization in the electron channel is almost one order of magnitude larger than the spin polarization in the hole channel, while the effect of mispair is more pronounced in the low-energy hole channel for long DNA and in the low-energy electron channel for short DNA with $N_{\rm BP} = 5$.

For a short DNA the spin polarization becomes sensitive not only to the type of the mispair and the length of DNA but also to the location of the mispair within DNA. In figure 3 the spin polarization $\Delta G/G$ is shown for DNA with $N_{\rm BP} = 15$ base pairs and for different positions of the mispair. The results for the low-energy hole channel are shown in figures 3(*a*) and (*b*). The spin polarization is strongly modified if the mispair is placed near the contacts (at $m = 2$ or $m = 14$ sites). In that case for both types of mispairs the spin polarization is increased by almost an order of magnitude, but the sign of spin polarization is different for different mispairs. For the G(anti)·A(syn) mispair (figure 3(*a*)), the spin polarization becomes positive, while for the G(anti)·A(anti) mispair (figure 3(*b*)) the spin polarization is negative. A strong enhancement of $\Delta G/G$ occurs only when the mispair is near the contacts, whereas when the mispair is away from the contacts, the spin polarization has a weak dependence on its position.

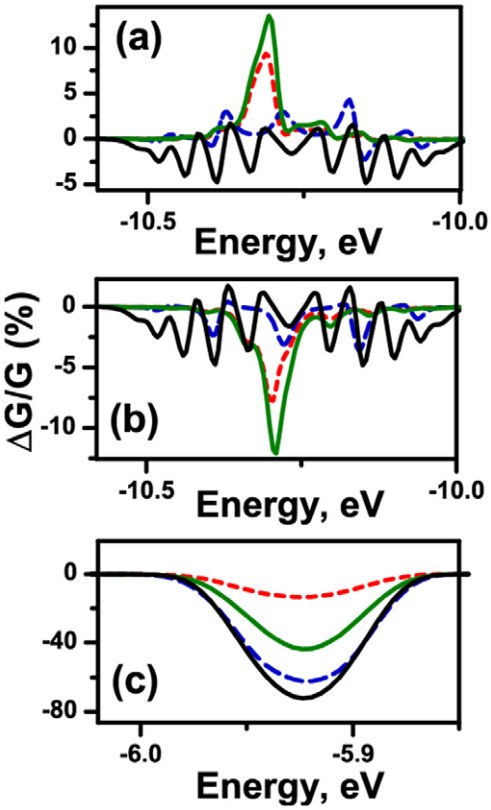

Figure 3. The energy dependence of the spin polarization for DNA consisting of $N_{\rm BP} = 15$ base pairs in the hole channel (*a*), (*b*) and in the electron channel (*c*). The black lines correspond to poly(G)–poly(C) DNA, the blue long-dashed lines—DNA with mispair at $m = 8$ site, the red short-dashed lines—DNA with mispair at $m = 2$ site and the green line—DNA with mispair at $m = 14$ site. The types of mispairs are (*a*) G(anti)·A(syn); (*b*) G(anti)·A(anti); (*c*) G(anti)·A(syn).

For the electron channel, both types of mispairs show a similar behavior with changing locations of the mispair. In figure 3(*c*) we have shown only the results for the G(anti)·A(syn) mispair. Similar to the hole channel, the spin polarization in the electron channel is strongly modified when the mispair is placed near the contact, i.e. at $m = 2$ and 14 sites. However, for the electron channel the spin polarization is strongly decreased by almost an order of magnitude. The reduction is the strongest for the mispair at $m = 2$ base pair (red line in figure 3(*c*)).

The results shown in figures 2 and 3 correspond to an ideal system, which does not take into account the thermal vibrations of DNA and the surrounding medium. Such thermal fluctuations introduce dynamic disorder into the problem that can modify both the on-site energies of HOMO and LUMO orbitals and the interstrand and intrastrand hopping integrals. These modifications should influence the spin polarization of the electrical conductance, $\Delta G/G$. As we mentioned above, to study the effect of the dynamic disorder on spin polarization we consider a random static DNA Hamiltonian with random on-site energies of HOMO and LUMO orbitals and random values of the hopping integrals, the distribution function of which are described by equations (7)–(8). The electrical conductance of such disordered system was calculated as the average over 1000 realizations of random variables.

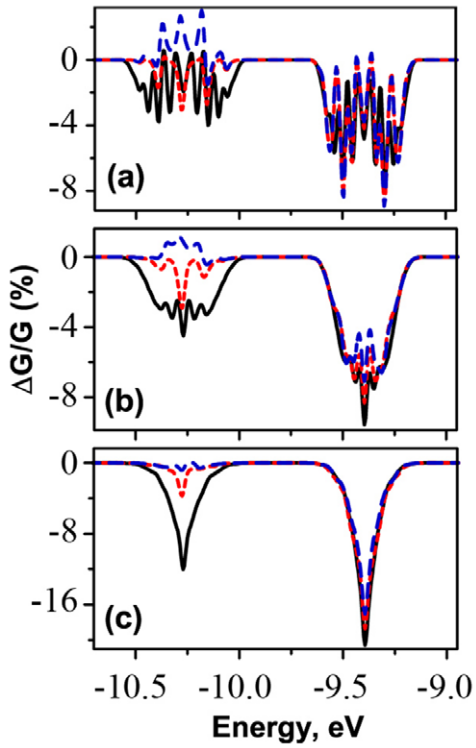

Figure 4. The energy dependence of the spin polarization for poly(G)–poly(C) DNA (black line) and for DNA with one mispair: G(anti)·A(anti) (red short-dashed lines) and G(anti)·A(syn) (blue long-dashed lines). The length of DNA is $N_{\mathrm{BP}} = 15$. The DNA is described by the Hamiltonian with random values of the hopping integrals. The standard deviations for the random variables are (*a*) $\sigma_t = 0.01$ eV, $\sigma_E = 0$ eV; (*b*) $\sigma_t = 0.03$ eV, $\sigma_E = 0$ eV ; (*c*) $\sigma_t = 0.05$ eV, $\sigma_E = 0$ eV.

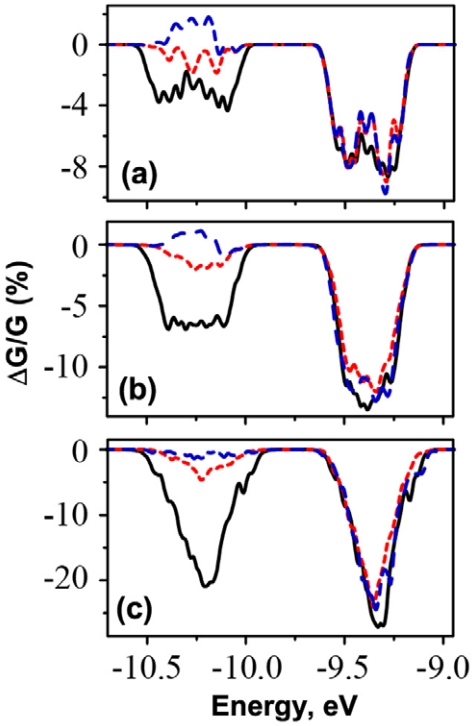

Figure 5. The energy dependence of the spin polarization for poly(G)–poly(C) DNA (black line) and for DNA with one mispair: G(anti)·A(anti) (red short-dashed lines) and G(anti)·A(syn) (blue long-dashed lines). The length of DNA is $N_{\mathrm{BP}} = 15$. The DNA is described by the Hamiltonian with random values of the on-site energies. The standard deviations for the random variables are (*a*) $\sigma_t = 0$ eV, $\sigma_E = 0.01$ eV; (*b*) $\sigma_t = 0$ eV, $\sigma_E = 0.05$ eV; (*c*) $\sigma_t = 0$ eV, $\sigma_E = 0.1$ eV.

In figure 4 the spin polarization of the electrical conductance in the hole channel is shown for DNA with random values of hopping integrals and fixed values of the on-site energies, i.e. $\sigma_E = 0$ and $\sigma_t \neq 0$. For the high-energy peak, with increasing disorder the spin polarization of the electrical conductance becomes less sensitive to the presence of a mispair. However, a different behavior is observed for the low-energy peak. While for a weak disorder, i.e. an ideal poly(G)–poly(C) DNA molecule, a mispair modifies only weakly the spin polarization of conductivity (see figure 4(*a*)), for a strong disorder the spin polarization shows a strong dependence on the presence of a mispair (figure 4(*c*)). For both G(anti)· A(syn) and G(anti)· A(anti) the spin polarization is reduced by almost an order of magnitude when the mispair is introduced into the DNA.

A similar behavior is also observed for DNA with random on-site energies. The corresponding results are shown in figure 5, where the hopping integrals have fixed values but the on-site energies are random. For the high energy peak, the fluctuations of the on-site energies do not increase the sensitivity of $\Delta G/G$ in the presence of a mispair in DNA. For the low-energy peak the disorder strongly enhances the effect of a mispair on $\Delta G/G$. In the presence of strong disorder (figure 5(*c*)), the spin polarization of DNA conductivity containing a mispair is an order of magnitude smaller than that without a mispair.

The results presented in figures 4 and 5 clearly demonstrate that the dynamic disorder does not decrease the effect of mispair on the spin-selective current through DNA, but in some cases it can even enhance this effect. As an example, for the hole current channel, incorporation of a mispair into DNA results in a decrease of spin polarization of DNA conductivity by an order of magnitude.

4. Conclusion

Our studies indicate that by replacing one of the GC base pairs in a poly(G)–poly(C) DNA by a GA mispair, a profound modification of the spin-selective transport through DNA is possible. The current through DNA is determined by two factors: the arrangement of energy levels of the nearest neighbor base pairs and the values of the intrastrand hopping integrals. The maximum current occurs when the interlevel separation is comparable to the corresponding interlevel hopping integral, i.e. intrastrand hopping integrals. The mispair changes locally both the on-site energy levels and the hopping integrals. These changes modify the charge transport through the DNA molecule and correspondingly the spin polarization of electrical current through DNA also gets modified. The mispair is in fact, capable of changing the spin polarization of the charge transport by almost an order of magnitude than in a homogeneous DNA. The effect of

mispair on spin polarization of electrical current depends on the electron energy, i.e. on the channel of electrical current: hole channel (HOMO orbitals) or electron channel (LUMO orbitals).

We considered above only those defects in the DNA molecule that occur in the structure of the base pairs, i.e. mispair defects. With comparable probability, the damage to the molecule can occur also in the DNA backbone [36, 37]. Within the tight-binding model discussed above, those defects in the DNA backbone can modify locally the on-site energies and correspondingly change the spin polarization of the electrical current through DNA.

The changes in the spin polarization of the electrical transport through DNA due to mispair are sensitive to the number of the base pairs within DNA molecule and to the position of the mispair within DNA. The strongest changes in spin polarization of the current in both electron and hole channels are realized when the mispair is placed near the ends of DNA. Thermal vibrations of the DNA molecule and the surrounding medium modify both the on-site energies and the hopping integrals. Although such modifications make the DNA system with mispair more homogeneous, we found that in some cases the finite thermal disorder enhances the effect of mispair on spin polarization of the electrical current. As an example, for the low-energy peak in the hole channel the thermal disorder strongly enhances the changes due to the mispair in spin polarization of the current, while for the higher energy peak in the hole channel the thermal disorder suppresses the effect of the mispair. Therefore, incorporation of a mispair into DNA opens the unique possibility of tuning the spin-selective transport through the homogeneous DNA molecule and can be used to design the DNA-based spin valve with given properties.

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