ANDERSON LOCALIZATION IN TWO-CHANNEL WIRES WITH
CORRELATED DISORDER: DNA AS AN APPLICATION

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This research studied the Anderson localization of electrons in two-channel wires with correlated disorder and in DNA molecules. It involved an analytical calculation part where the formula for the inverse localization length for electron states in a two-channel wire is derived. It also involved a computational part where the localization length is calculated for some DNA molecules.

Electron localization in two-channel wires with correlated disorder was studied using a single-electron tight-binding model. Calculations were within second-order Born-approximation to second-order in disorder parameters. An analytical expression for localization length as a functional of correlations in potentials was found.

Anderson localization in DNA molecules were studied in single-channel wire and two-channel models for electron transport in DNA. In both of the models, some DNA sequences exhibited delocalized electron states in their energy spectrum. Studies with two-channel wire model for DNA yielded important link between electron localization properties and genetic information.
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CHAPTER 1

INTRODUCTION

The search for the secret of life is perhaps the most intriguing and rewarding intellectual adventure of all human endeavors. Countless ideas and ideologies have fought over the ultimate answer throughout history. The secret is well hidden, not only because of the infinite delicacy and intrigues of what we may call life, but also the fog of time that divides the questionable origin and its mechanism. At this point in science, we have not yet fully put forward a scientific theory for the origin of life on Earth, but we have accumulated a significant amount of information on how it carries on.

So far all life forms we have found originate from another living organism or organisms, hence they borrow a number of characteristic traits that we name as genetic information from their ancestors. The genetic information organisms pass to their offspring is such a valuable asset that its continuity is a main driving force behind many animal and human behaviors. It is only recent that we have established an understanding on how they pass the information and we are still trying to understand the extent of the effect of that information on an organism’s life span. We have also found out that the genetic information passed on is not always an exact replica or a mix of both ancestors, but that it may show slight variations from its source. Furthermore, we found that such variations may occur in the body of an organism during its lifetime, sometimes leading to fatal diseases.

The genetic information is stored in all living cells of an organism, in form of a very long bio-molecule called the DNA (deoxyribo-nucleic acid). Nucleic acids were first isolated in 1869 by Friedrich Miescher, but it took almost 75 years to demonstrate that DNA was the carrier of genetic information. The structure of DNA was discovered by James Watson and Francis Crick in 1953 [1]. It is a polymer of nucleotides, which consist of phosphorylated sugars and bases. The bases are adenine, thymine, cytosine and guanine. The nucleotides
are arranged in a two-stranded one-dimensional chain. The structure of DNA will be discussed furthermore in the text.

The genetic information within DNA is stored as a sequence of nucleotides. This is a very long sequence and it varies between different species and within the members of the same species. The sequence of nucleotides typically look like ...ATCGATTGCCACTTAGCAGG... (for a single strand). The relationship between the nucleotide sequences of genes and the amino-acid sequences of proteins is determined by the rules of translation, known collectively as the genetic code. The genetic code consists of three-letter 'words' called codons formed from a sequence of three nucleotides (e.g. ACT, CAG, TTT). These codons are signals for the ribosome (protein factory of a cell) and various enzymes to bind a specific amino-acid (building blocks of proteins) with other amino-acids, in relation to the following or preceding codon in the sequence. While there are $64(4^3)$ possible codons, there are only 24 different amino-acids in nature, so some codons point at the same amino-acid.

DNA has regions of different functions. It contains large segments that do not play any role in the transcription (protein coding). These regions are called introns. The regions that are expressed during transcription (sequences that lead to protein coding) are called exons. Along the DNA sequence, there are regions that signal or mark the beginnings of transcription regions. There are sequences that will stop transcription, sequences that enhance the transcription, and sequences that control or regulate the gene expression. There are volumes of studies in almost every property of these various regions of DNA.

The sequence of nucleotides is a random sequence, in the sense that which nucleotide is the next in chain can not be known from previous order. However, the sequences do not appear to be totally uncorrelated. In fact, the DNA sequences display long-range correlations if we focus on parts of DNA. Whether there is a difference between the statistics of intron and exon regions of DNA has also been investigated thoroughly [2, 3, 4, 5, 6]. There is still a wide open and rich field of study for physics to find out the relationships between physical properties and statistical properties of DNA.
The electronic structure of DNA poses a very interesting problem by itself. Surely the mobility of electrons along the sequence effects DNA’s functional behavior (signalling, enzyme-DNA interactions etc.) and its structural-sequential integrity, we just don’t know how. The charge migration is believed to be important for the radiation damage repair [7]. One recent study has shown that single-base mutations on DNA molecules may lead to significant changes in conductance [8]. DNA double helices are also expected to be particularly useful for molecular electronic devices [9, 10, 11]. Numerous studies on conductance of DNA yielded results from insulator, to semi-conductor or metallic [9, 12, 13]. Although it is well-understood now that the current in DNA is due to light particles, i.e. electrons or holes [14], the physical mechanism that provides a wide variety of conducting (or non-conducting) properties for different DNA is still unclear. The electrical conductivity of the DNA molecules exhibits strong environmental dependence that includes variation with temperature, chemical composition of the solution, humidity, quality of the metallic contacts, etc [15]. It is not clear whether these properties play a role in living nature or whether they are just artifacts of the experiments.

Within the statistical nature of DNA, there lies an important clue on the electronic behavior along the molecule, namely quantum localization of electrons (or holes). The quantum mechanical charge carrier has a wavefunction that extends to a certain spatial region. The extent of the wavefunction depends on the type of motion the electron executes, which in return depends on its total energy and the potential function it moves under the influence of. A free electron, for example, extends to infinity. A Gaussian wave packet will spread infinitely in free space. An electron in an ordered system, where potential function exhibits a periodic nature, will extend throughout the whole system (delocalized quantum state) if the electrons energy is within the allowed zone. It is the disordered, random systems that exhibit the crucial property of electron localization, which is the case when the extend of the electrons’ wavefunction, the localization length, is finite and the electron is exponentially localized within a finite region. The localization length determines the conductance properties and electron-ion (molecules or enzymes) interactions for DNA. One dimensional disordered
systems like DNA were considered to have only localized states [16, 17]. However it has been shown that for one-dimensional single-channel wires, a group of systems which DNA is often modeled within, delocalized states may exist under certain conditions [18, 19]. It seemed also probable that delocalized states may exist for one-dimensional two-channel wires, which is a more accurate approximation for DNA. In this thesis, the electron localization lengths for DNA samples in two different tight-binding models (the single channel Fishbone model and the two-channel wire) were calculated. In order to apply the two-channel model, it was necessary to find an analytical expression for the localization length for those systems, taking into account all correlations. That work is also presented in the thesis.

Our knowledge of how DNA exactly functions inside a living organism is limited; indirect observations, snapshots and in vitro observations are the main source of information about the mechanism of the expression of information carried in DNA [20]. Since it is a low-dimensional system with small extend in constrained dimensions, the problem of electronic behavior in DNA is a quantum mechanical one in its nature. Ab-initio studies for single nucleotides or short DNA molecules, or for molecules with periodic repetitions (artifical DNA molecules) can be carried out [21, 22, 23], however it is not possible within today’s technology to study DNA in an exact manner. Instead, we rely on statistical and approximate methods. In this thesis, the DNA molecules are studied using one of such approximate models, the so-called tight-binding model. A detailed discussion on the model follows in the text.

1.1. Structure of DNA

Deoxyribo-nucleic acid, DNA, is a very long chain of nucleotides, phosphorylated sugars attached to bases adenine, thymine, cytosine and guanine. The bases are organic compounds (Fig.1.1).

One nitrogen from each base bonds with a 2'-deoxyribose molecule and they form the nucleotide. Then the 3' hydroxyl group of one nucleotide sugar makes a phosphodiester bond with the 5' phosphate group of the consecutive one as shown in Fig.1.1. The next base in the sequence is then linked to this phosphorylated sugar [24]. The polynucleotide chain has a sense of direction with one end being the 5' phosphate group and the other the 3'
Figure 1.1. Chemical structure of DNA bases and the sugar-phosphate group (yellow and brown) is shown above.

hydroxyl group. For each chain like this, there is a complementary one with the opposite sense of direction. The two chains are linked only through the matching bases; adenine always pairs with thymine (two hydrogen bonds) and guanine with cytosine (three hydrogen bonds) (Fig.1.2). Contrary to the hydrogen bond formation in base pairs of A-T and C-G, all other pairings, i.e. A-G or C-T, lead to a significant repulsive electrostatic potential. The phosphorylated sugars form the backbone of DNA. The electrostatic interactions between the negatively charged backbone and the positively charged bases, and the hydrogen bonds between the hydrophobic bases hold the DNA together.

The length of a DNA molecule can change anywhere between $10^6$ to $10^9$ base pairs (A-T or C-G). The total length of human DNA molecules (for a single cell) is around 3 billion base pairs. These molecules exist in a tightly wrapped structure inside a living cell for most of the
Figure 1.2. Structure of DNA is shown. a) The double-helix structure, the four bases A, T, C and G shown in their complimentary form. b) Graphical depiction of the chemical structure.

time, until they are to be used for replication or protein coding, at which point they unfold. The extended DNA resembles a helical ladder, in which the phosphorylated sugars form the side rails. When they are idle, they are found in forms of chromosomes, long chains of DNA molecules wrapped around histone proteins.

The diameter of the helix is 20 Å and the vertical distance between consecutive bases 3.4 Å. One turn of the helix consists of 10 bases and the twist angle is normally 36°. The values for distances are taken to be the same when the helical structure is unfolded into a ladder structure.

The π-orbitals of adjacent bases, on the same chain or across, are all perpendicular to the length of the DNA strands. These orbitals overlap significantly, creating what is called π-stacking, lowering the total energy of the system and further stabilizing the DNA molecule. The overlap interaction between bases forming base pairs are stronger in comparison to
the overlap interaction between adjacent (consecutive bases along a strand) bases, for two reasons. Firstly, the distance between bases on the same strand is larger than the distance between bases on complimentary strands. Secondly, the \( \pi \)-orbitals of bases on complimentary strand have the same direction, while there is a tilting of orbital directions (a 36° turn at each consecutive base pair) for bases along the same strand in the helical structure [99].
CHAPTER 2

THE CONCEPT OF LOCALIZATION

A crystal is a perfect periodic structure, where every physical property along the directions of translational symmetry is identical. For example, the possibility of finding an electron is the same for each unit cell. A crystal structure is not necessarily three-dimensional. A chain of identical atoms with equal spacings between them is an example of a one-dimensional crystal. The electrons inside the crystal that are not bound to the ions or molecules are called crystal electrons and they occupy quantum states that satisfy the periodicity conditions of the structure. The eigenfunctions of periodic systems are called Bloch wavefunctions. If the potential function of the system has a periodicity

\[
(2.1) \quad V(r) = V(r + \mathbf{T}),
\]

the Bloch wavefunction has the form

\[
(2.2) \quad \psi_k(r) = u_k(r) \exp(i\mathbf{k} \cdot \mathbf{r})
\]

with \( u_k(r) = u_k(r + \mathbf{T}) \). The wavefunction then satisfies

\[
(2.3) \quad \psi_k(r + \mathbf{T}) = \psi_k(r) \exp(i\mathbf{k} \cdot \mathbf{T}), \quad |\psi_k(r + \mathbf{T})|^2 = |\psi_k(r)|^2
\]

and each \( \psi_k \) spreads throughout the whole system. The energies of the wavefunctions are \( E(\mathbf{k}) \), smooth functions of the wavevector \( \mathbf{k} \). At the absolute temperature \( T = 0 \) K, the crystal electrons occupy the eigenstates beginning from the one with the lowest energy until the electrons are depleted. There are no excitations at that temperature, thus eigenstates solely dictate the electronic transport in the crystal. While all crystal wavefunctions are
delocalized, that is they are spread out to the whole crystal, this does not mean that they contribute to conductance. The conductivity of a material is much more complex a property that depends on the whole eigenstate structure. The presence of delocalized states is a necessary but not sufficient condition for conductivity.

The physics of periodic systems is well formulated. However, it is extremely unlikely to find perfect periodicity even for artificial materials. There exist deviations from the perfect periodic structure. These deviations are called defects or impurities. A simple defect may be an atom B at a single site instead of having atom type A at every lattice point (the collection of all points that form the crystal). Similarly, one of the atoms A may shift slightly from its position, or may not be there at all.

- ...A...A...A...A...A...A... Perfect crystal
- ...A...A...A...B...A...A...A...A... Atom A replaced by atom B
- ...A...A...A... A...A...A... A... Missing atom (vacancy)
- ...A...A...A...A...A...A...A... Shift of one of the atoms, topological defect
- ...A...A...A...B...A...B... Multiple defects
- ...A...B...C...A...C...B...B...A... Topological periodicity, quasi-periodic structure
- ...A...B...E...B...C....E...A...A...C... No periodicity or order, complete disorder

Putting aside atomic or molecular scale objects, many material mediums in nature do not have a periodic structure at all. However for most physically, technologically or biologically important materials, it is possible to treat the material medium as quasi-periodic or crystal-like and consider randomness or disorder in the medium as defects. Any system without a perfect periodicity is considered a disordered system. The nature of individual defects and the distribution of these defects determine the strength of disorder. The disorder strength can vary between very weak disorder in crystal-like structures with infrequent defects (e.g. silicon wafers) to complete disorder (e.g. amorphous silicon).

When the disorder is not significant, the physical characteristics of the disordered system can be calculated by perturbation theory taking the perfectly periodic structure as the
zeroth-order basis. Transport properties and conductivity are among the successfully calculable characteristics. The Bloch-Boltzmann quasiclassical theory of electronic transport in lightly disordered conductors has been quite useful in describing the impurity and temperature dependencies of conductance in ordinary relatively pure conductors [25]. Further transport properties such as magnetoresistivity, Hall effect, and thermal conductivity can also be handled with success. However the perturbation theory fails at a point with increasing strength of disorder. The weak-scattering theory can no longer explain phenomena like the increase in resistance with decreasing temperature in disordered dirty systems [26, 27, 28]. The concept of localization comes in to help in understanding strong disorder.

Localization is a wave property. Electromagnetic waves, water waves or particle waves all can display localization. The phenomena of localization exists when waves interact with disordered mediums. Due to disorder, there are many scattering centers inside the medium. Waves undergo a number of multiple scattering processes when they propagate through the material. Let’s consider as an example an electron undergoing a multiple scattering process from an initial state $|k_i\rangle$ with a wavevector $k_i$ into the final state $|k_f\rangle$ with the wavevector $k_f$. The scattering process $P_A \equiv (k_i \rightarrow k_{A,1} \rightarrow k_{A,2} \rightarrow \cdots \rightarrow k_{A,n-2} \rightarrow k_{A,n-1} \rightarrow k_f)$ represents one path involving $n$ scattering events and has a probability amplitude $T_A$. A similar process $P_B \equiv (k_i \rightarrow k_{B,1} \rightarrow k_{B,2} \rightarrow \cdots \rightarrow k_{B,n-2} \rightarrow k_{B,n-1} \rightarrow k_f)$ might exist with probability amplitude $T_B$. In general, the alternative paths accumulate different phases, $\theta_A$ and $\theta_B$. The probability of transition $|k_i\rangle \rightarrow |k_f\rangle$ is then given by

$$|T_A + T_B|^2 = |T_A|^2 + |T_B|^2 + 2|T_A||T_B|\cos(\theta_A - \theta_B).$$

Since in a disordered system, the phases $\theta_A$ and $\theta_B$ are random, the ensemble average $\langle \cos(\theta_A - \theta_B) \rangle = 0$. So the transmission probability is $|T_A|^2 + |T_B|^2$, addition of probabilities of individual processes. This simple addition of probabilities is valid for classical particles, i.e. interference does not contribute. Let’s now consider when $|k_f\rangle = | - k_i\rangle$, which is the case of backscattering. For a system with time-reversal invariance, the probability amplitude $T_A$
of process $P_A$ and probability amplitude $T_B$ of process $P_B$ which correspond to time-reversed process $P_A$ are equal, that is $T_A = T_B$. The phases $\theta_A$ and $\theta_B$ for the time-reversed trajectories are also equal. Then the transition probability for $|k_i \rightarrow | - k_i|$ is $|T_A + T_B|^2 = 4|T_A|^2$ [29]. Thus, we have an enhanced probability for backscattering (scattering in the opposite direction). This phenomena is called coherent-backscattering. The wave interference between paths A and time-reversed A is always constructive independent of disorder, and can completely halt the waves.

This absence of diffusion of waves in random medium is called Anderson-localization. The localization of an electron implies that its wavefunction vanishes exponentially away from the center of localization. The localization length is the measure of the spatial extension of the localized state. Qualitatively,

$$ (2.5) \quad \psi(x) \sim \exp \left( - \frac{|x|}{l(E)} \right) $$

where $l(E)$ is the localization length. The localization length, being a characteristic of a given electronic eigenstate, depends on eigenenergy $E$.

If the localization length considerably exceeds all other relevant lengths in the system (size of the system, mean-free path etc.), then the electron can extend to infinity and contribute to electron transport. The semi-classical Drude conductivity is slightly modified due to coherent-backscattering in this case and the correction to the conductivity is called weak-localization correction. If $l(E)$ is smaller in comparison to the system length, the particles occupying those states do not interact with the boundaries, thus they do not contribute to electron transport.

If all electrons occupy localized states at absolute zero temperature $T = 0\ \text{K}$ the dc-conductivity vanishes and the system is insulating at that temperature. If there are electrons in delocalized states, a non-zero dc-conductance may exist and the system may be conducting at absolute zero temperature. On the other hand, at temperatures above absolute zero some
Figure 2.1. Localization is a wave phenomena. Here localization of water waves in a bath is displayed. a) Crystal like structure. b) The disordered system with localization of waves.

Localized electrons may get excited to delocalized states and the system might conduct again. For the rest of this thesis, all calculations and considerations are carried out at $T = 0$ K.

2.1. Development of Localization Theory, Metal-Insulator Transition and Scaling Theory of Localization

2.1.1. Pre-Scaling Era

Anderson (1958) studied the electronic diffusion in a three-dimensional tight-binding model in which the energies of the different sites (lattice points) are randomly assigned from a given interval according to a constant probability distribution with width $W$. He considered a constant hopping parameter $V$ between sites. He showed that above a critical value of disorder, which he quantifies as $W/V$, all states are spatially localized [16]. The relevance of localization with regard to electronic transport properties of amorphous semiconductors was discussed by Mott [30].
Figure 2.2. Electron states are localized or extended according to their location with respect to the Fermi energy in the energy scale. If the Fermi energy lies in the vicinity of the critical energy, the MIT may occur. The asymptotic behavior of the system in that region forms the basis of one-parameter scaling theory.

The localized states and extended states cannot coexist at the same energy. Quantum mechanical tunneling would admix them and destabilize the localized states [29]. Thus, the localized states must be separated energy wise from the continuum of extended states, without implying any gap in the density of states. The set of localized and delocalized states are separated by a sharply defined energy $E_c$, which is called the critical energy or the mobility-edge. The realization of the random disorder in a medium determines the position of mobility edge. As the Fermi energy is tuned across the critical energy when an external parameter is changed, the system goes through a transition between metallic and insulating phases. This transition is called metal-insulator transition (MIT) or Anderson transition. A quantitative approach to the MIT and calculations for mobility edge may be found in Thouless’s early work [31].

The presence of a mobility edge in disordered systems explains anomalous increase in resistivity for dirty conductors. when $T \to 0$. At high temperatures, motion of electrons in delocalized states are hindered by scattering from other crystal degrees of freedom, like phonons. As the temperature drops, the number of phonons or other scatterers get smaller so the mobility of the electrons increase, which in turn causes decrease in resistance. However as temperature gets closer and closer to absolute zero, the electrons excited from localized states to delocalized states start falling back to localized states. This reduces the number of
free carriers and increases resistance. This increase in resistance is called weak-localization peak.

2.1.2. Single-Parameter Scaling Theory

Scaling theory of localization is a statistical approach to the problem of electron localization in the vicinity of metal-insulator transition region. The so called critical region hosts the states that contribute to the transport mechanisms of the disordered system. The study of localization-delocalization transition as a function of energy across the mobility edge for individual quantum states is too fine-grained. We may however look at a physical quantity that will give information about states at or around mobility edge. That quantity is electrical conductance, which is the inverse of electrical resistance.

Let’s at this point risk a deviation from the line of thought and focus on the conceptual framework that allows us to investigate the conductance as a statistical variable. Conventional statistical physics associates experimentally meaningful quantities with averages over a statistical ensemble of macroscopically different systems. For example, for a given macroscopic concentration of impurities there is a continuum of possible arrangements of their positions in the host crystal. Although the results of measurements of a physical quantity, when performed on specific members of the statistical ensemble, will be different and dependent on the specific configuration of the impurities, or, more generally, on the specific realization of the disorder, the statistical fluctuations of the results will become vanishingly small when compared with, say, the ensemble average, provided the system is sufficiently large, i.e. macroscopic.

Physical quantities are usually assumed to fulfil this criterion, namely that they effectively do not fluctuate within the statistical ensemble of macroscopically equivalent systems in the thermodynamic limit and such quantities are called self-averaging. Formally, the ensemble average of a self-averaging quantity and the most probable value within the ensemble practically coincide when the size of the system is assumed to be infinite. Self-averaging implies that, in practice, measurements done on specific samples-specific realizations of the disorder can be described in terms of ensemble averages.
Let’s take a set $a_L$ of dimensionless physical parameters that vary with the system size $L^d$, where $L$ is to be considered as effective system size such as the inelastic scattering length $L_i$. We define a parameter $\beta$,

$$\frac{d a_L}{d \ln L} = \beta(a_L)$$

The important property of equations like Eq.2.6 is that for increasing $L$, the set $a_L$ goes towards a simple subspace, often a line or even a point. Such points $a^*$ are known as attractors. These points are defined by the condition $\beta(a^*) = 0$. The set of parameters $a_L$ can be reduced to a single parameter, named as the scaling parameter.

Now we shall consider once more the behavior of conductance of disordered systems with its Fermi energy lying in the vicinity of the critical energy. If a scaling parameter related to the quantum transport properties of disordered system is chosen, a thorough investigation of that parameter will shed light on related properties like electron localization. Abrahams, Anderson, Licciardello and Ramakrishnan [40] have come up with $\ln g$ as that scaling parameter, where $g$ is the dimensionless conductance. Their reasoning is simple: at the asymptotic limits of $g \gg 1$ (metallic regime) and $g \ll 1$ (insulator regime), the functional dependence of $\ln g$ to system size $L$ is independent of the size of the system. If we approach the mobility edge either from the metallic regime or from the insulator regime, $\ln g$ can be approximated with the corresponding asymptotic expression. They match each other at the point of transition. The asymptotic expressions for the scaling parameter $\beta$ are

$$\beta \equiv \frac{d \ln g}{d \ln L} = \begin{cases} d - 2, & g \gg 1 \\ \text{const} + \ln g & g \ll 1. \end{cases}$$

The upper equality (2.7) derives from the Drude conductance formula for weakly disorder metals. The lower equality follows from

$$g \sim \exp(-L/I(E)), \quad L \sim -I(E)\ln g$$

15
Figure 2.3. Scaling parameter $\beta$ is a functional of the natural logarithm of the dimensionless conductance. For $\beta > 0$ the behavior is metallic, whereas for $\beta < 0$ it is insulating.

Finally, placing $g$ from Eq.(2.8) into Eq.(2.7), the self averaging quantity-localization length $l(E)$ can be found from

$$
\frac{1}{l(E)} = - \lim_{l \to \infty} \frac{1}{2L} \langle \ln g \rangle
$$

where $\langle \ln g \rangle$ denotes the ensemble averaging, which the scaling theory predicts to reach a limiting value for $L \to \infty$.

A very famous result of the scaling theory of localization is the absence of extended states in 1-D and 2-D systems. Since the upper limit for $\beta(\ln g)$ is given by $d - 2$ in the metallic regime, it is clear that for $d \leq 2$, the value of $\beta < 0$ for all disorder realizations. This means there is no attractor point and the system is always in localized regime. For 3-D systems however, there exists a condition for delocalization, which depends on the strength of disorder or the values of perturbation parameters with which the behavior of dimensionless conductance is approximated around MIT point. Good and detailed explanation of these
points can be found in the review paper by Kramer and McKinnon [38] or in the excellent books of Imry [25] and of Mello and Kumar [29].

The experimental work on the localization of electrons or holes in disordered systems have been a vibrant field of study, and has also seen a revitalization along with the theoretical work recently. The previously mentioned works of Mott [39], Wiesmann [26], Mooij [27] and Gorkov [28] were the pioneering studies into this field.

Application of a magnetic field leads to flux-dependent Aharonov-Bohm phase of a wavefunction, thus producing pronounced changes in the interference of the quantum mechanical transport paths. This was most strikingly demonstrated a decade ago when the experimental discovery of the Aharonov-Bohm-like oscillations of the magnetoresistance of thin normally metallic cylinders provided direct evidence for the existence of quantum interference in the presence of disorder [44].

If quantum interference is the dominant mechanism for the localization of states in a random medium, localization effects should be of importance in other wave phenomena, too [45]. That this is indeed the case has been demonstrated experimentally in recent years by light scattering experiments [43, 46] and microwave localization [87].

2.2. Beyond Scaling Theory and Localization of 1-D Wires

In 1961 Mott and Twose [47] showed for a particular 1-D model, consisting of square barriers of the same height and fluctuation distance between the barriers, that all electronic states are exponentially localized. In 1963, Borland reached the same conclusion for a 1-D chain composed of equal potential barriers of finite width and arbitrary shape separated by zones of zero potential assigned randomly according to a continuous distribution [48]. However Tong later specified that for Borland model extended states could exist for any structural disorder if the individual potential unit exhibits resonances of the transmission [49]. More examples of one-dimensional random potentials is given in the review paper by Ishii [50].

The predictions of scaling theory and naive extension of the properties of the 1-D Anderson model to other potentials have led to the belief that all the states in a random one-dimensional system are exponentially localized. However, further research has shown that delocalized
states may exist. Deterministic quasi-periodic potentials were considered that can generate localized or extended states depending on their parameters [51, 52].

In the models so far considered, the potentials were uncorrelated, i.e. white-noise like. Dunlap [53] and Flores [54] initiated the consideration of short-range correlations in random potentials. The short-range correlations may lead to isolated extended states in the spectrum of the system. The random-dimer model (RDM) of Dunlap and his co-workers was used to explain the high conductivity anomalies observed in certain organic polymers that should behave as insulators [55]. The RDM model has been extensively treated both from a theoretical viewpoint [56, 57, 58, 59, 60] and it was experimentally realized in random semiconductor superlattices [61, 62]. Other models like diluted Anderson-model [63, 64, 65], symmetrical impurities in a pure chain [66], short-range correlations with classical analogues [67, 68] and off-diagonal disorder [69, 70] have also been studied.

The role of long-range correlations has also been analyzed during the last decade. Moura and Lyra considered a 1-D tight-binding disordered model with 1/f-noise type long-range correlations in the sequence of site-energies [80, 81]. They observed numerically the emergence of a continuum of extended states and mobility edges for the carriers, marking the transition between phases of localized states and extended states. Later on, Izrailev and Krokhin [18] established an analytical relation between localization length and potential pair correlator and showed how long-range correlations lead to the appearance of mobility edges. Their result is valid in the second-order approximation over disorder. Though Tessieri [82] later showed that to fourth-order approximation, the extended states are actually still exponentially localized but on much larger scale than the states of the localized phase. Upon passing the 'mobility edges' the inverse of the localization length changes from a quadratic to a quartic dependence on the disorder strength, and for weak disorder it means that the increase of the spatial extension of the electron wavefunctions can be huge. Therefore quantitatively this change in the finite length samples can be considered as a MIT. Similar conclusions have also been reported for other disordered models with long-range correlations [83, 84].
Figure 2.4. The Anderson tight-binding model of a 1-D chain is depicted above. 
\( \varepsilon_i \)'s correspond to on-site energies.

One more important study in the theory of localization for 1-D systems came from Heinrichs [85, 86] who considered two-chain Anderson-model for a one-dimensional wire. In that work, Heinrichs calculated analytically the inverse localization length for a disordered two-chain wire with uncorrelated random potentials. The mentioned work has opened the way to the calculation of localization length for two-channel wires with correlated disorder, which is one of the major findings of this thesis and also the applied model for DNA electron localization problems.

2.3. Analytical Results for Localization Length

Thouless has come up with an analytical solution of problem of Anderson localization in a 1-D chain [17]. The tight-binding Anderson model of a chain (Fig.2.4) is represented by a sequence of potential sites with on-site energies \( \varepsilon_n \) and constant amplitude of hopping between the neighboring sites \( t \). This is an example of the so-called diagonal disorder. The quantum states in this chain are obtained from discrete Schrödinger equation

\[
(2.10) \quad t(\phi_{n+1} + \phi_{n-1}) = (E - \varepsilon_n)\phi_n.
\]

The strength of the fluctuations is measured by the variance \( \varepsilon_0^2 = \langle \varepsilon_n^2 \rangle \). In case of weak disorder, \( \varepsilon_0 \ll t \), Thouless calculated the inverse localization length \( l^{-1}(E) = 1/(E) \) in Born approximation for white-noise potential, where \( \langle \varepsilon_i \varepsilon_k \rangle = \varepsilon_0^2 \delta_{ik} \). The Thouless formula for inverse localization length has the following form
Eq. (2.11) is the first term of the expansion of the Lyapunov exponent $l_0^{-1}(E)$ over variance $\varepsilon_0^2$. The validity of Born approximation suggests scattering is weak, therefor that a localized state covers many sites, i.e. $l_0(E) \gg 1$. It becomes invalid in the vicinity of the band edges, $|E/2t|\to 1$, where $l_0^{-1} \to 0$. More accurate expansion over $\varepsilon_0$ in the vicinity of band edges lead to unusual scaling, $l_0^{-1}(E) \propto \varepsilon_0^{2/3}$ [71, 72]. This "anomalous" behavior of the localization length is later shown to be accompanied by violation of the scaling hypothesis [73, 74, 76].

Izrailev and Krokhn extended result (2.11) into a more general problem of Anderson-chain with correlated disorder [18]. They showed that correlations in the random sequence of the site energies $\varepsilon_n$ strongly affect the interference pattern between the forward and backward scattered wave. In the lowest Born approximation the localization length is determined by a pair correlation function, $\langle \varepsilon_n \varepsilon_{n+k} \rangle = \varepsilon_0^2 \xi(k)$, only. It was found that in the correlated potential the localization length is modified and it is given by the following formula

\[(2.12) \quad l_0^{-1}(E) = \gamma(E) = \varphi(2\mu), \quad \varphi(\mu) = 1 + 2 \sum_{k=1}^{\infty} \xi(k) \cos(\mu k).\]

Here the parameter $\mu$ plays the role of Bloch vector, defining the dispersion relation in a perfectly periodic chain,

\[(2.13) \quad E = 2t \cos \mu.\]

It is clear from (2.12) that if $\varphi(\mu)$ vanishes, the inverse localization length also vanishes and the localization length $l(E) \to \infty$. This happens if the infinite terms in Eq. (2.12) contribute to the sum over $k$. Thus, the long-range correlations may lead to delocalized states forming a band in the energy spectrum. This analytical expression as a novel result also predicted the possibility of sharp mobility-edges.
Figure 2.5. Sharp mobility edge is a sudden change inverse localization length moving along in the energy spectrum. The energy region $l^{-1}(E)=\gamma(E) = 0$ is the delocalized region. $E = 1$ line is the mobility edge.

Krokhin and Izrailev showed in their paper the necessary condition correlation function shall satisfy to give a mobility-edge shown in Fig.2.5. The correlation function shall have the form [18]

$$\xi(k) = \frac{3}{2\pi k} \sin\left(\frac{2\pi k}{3}\right).$$

In 2000, Kuhl and Stöckmann, in collaboration with Izrailev and Krokhin have experimentally demonstrated the existence of a mobility edge experimentally in a microwave transmission experiment [87]. The setup they used (Fig.2.6) is a disordered microwave waveguide. They took a toroidal waveguide, drilled holes on top of it with equal spacings in between. Screws of same thickness were then placed at each hole. Each screw is a scatterer for the microwaves and the depth of the screw determines the reflection coefficient for that screw. It was the random depths of screws that created the disorder in this 1-D like waveguide, and the depths were chosen so that they generated a correlation function similar to Eq.(2.14).

The results of the experiment show (Fig.2.7) the mobility edge for a wave system. This finding has led me into believing that similar delocalized bands (or islands) in energy spectrum might also exist for DNA. My calculations on DNA molecules using Krokhin-Izrailev localization length vs energy relation is given in Chapter 4.

At 2002, Heinrichs published his analytical expression for localization length in two-chain Anderson-model of disordered wires. He generalized the tight-binding model Eq. (2.10) for
two-channel system (Fig. 2.8) by introducing an index $i$ at the wave function $\phi_{n,i}$ and the potential energy $\varepsilon_n$. This index denotes the channel, $i = 1, 2$. A term describing the inter-channel coupling (with constant hopping parameter $h$) is added to the left-hand-side. The second equation for the two-component wave function $\phi_{i,n}$ is obtained by symmetrizing with respect to the index $i$. Finally, Schrödinger equation for the two-channel tight-binding model is written as follows,

\begin{align}
  t(\phi_{1,n+1} + \phi_{1,n-1}) + h\phi_{2,n} &= (E - \varepsilon_{1,n})\phi_{1,n}, \\
  t(\phi_{2,n+1} + \phi_{2,n-1}) + h\phi_{1,n} &= (E - \varepsilon_{2,n})\phi_{2,n}.
\end{align}

(2.15)

In his calculations, Heinrichs considered uncorrelated on-site energies, defining $\langle \varepsilon_n \varepsilon_m \rangle = \varepsilon_0^2 \delta_{ij} \delta_{nm}$. Then for the two-chain Anderson model, the inverse localization length $\gamma(E)$ is given by [85]
Figure 2.7. The transmissivity of the waveguide after a single pass is shown below. The upper graph is transmission vs wavelength (energy is determined by wavelength) for multiple passes. The straight line represents theoretical assumptions, dashed line represents the experimental data.

Figure 2.8. The Anderson tight-binding model of a 2-D wire consists of two connected chains.

(2.16) \[ I^{-1}(E) = \frac{\varepsilon_0^2}{32} \left( \frac{1}{\sin \mu_1} + \frac{1}{\sin \mu_2} \right)^2. \]

where similar to Eq.(2.13), the dispersion in the two-channels is given by,

(2.17) \[ 2 \cos \mu_1 = E - h, \]

\[ 2 \cos \mu_2 = E + h. \]
The dispersion relations for wavenumbers $\mu_1$ and $\mu_2$ reflect the coupling between the two-chains of the wire with coupling constant $h$ creating a shift $\pm h$ in the energy scale. Eq. (2.16) is valid for energy intervals $h - 2 < E < 2 - h$, where both $\mu_1$ and $\mu_2$ are real and waves in both channels are in propagating modes. Later Heinrichs generalized his solution to the intervals $-2 - h < E < h - 2$ and $2 - h < E < 2 + h$, where one of the wave numbers $\mu_i$ is necessarily a pure imaginary number and that wave is in evanescent mode decaying exponentially as a function of the site number. The other mode is still propagating. In that case [86],

$$I^{-1}(E) = \frac{\epsilon_0^2}{16 \sin \mu},$$

where $\mu$ is the real wavenumber for the propagating mode.

In this chapter, I introduced the localization theory, its consequences and applications. The last section summarized the analytical results available to us for calculation of localization lengths in DNA. In the next chapter, the calculation of localization length for a one-dimensional two-chain wire with correlated disorder is presented. The last chapter is about the application of all analytical results for localization length to the specific problem of electron localization in DNA.
CHAPTER 3

CALCULATION OF ELECTRON LOCALIZATION LENGTH FOR TWO-CHANNEL WIRES

We seek to calculate electron localization length for two-channel disordered wires using Eq.(2.9) given in Chapter 2. The inverse localization length $l_{\text{inv}}(E)$ is a functional of the natural logarithm $\ln g$ of the dimensionless conductance $g$. To calculate conductance, we use the Landauer two-probe conductance formula [88, 89]

\begin{equation}
(3.1) \quad g = Tr(\hat{t}\hat{t}^\dagger)
\end{equation}

which describes current transport in a wire. Here $\hat{t}$ is the so-called transmission matrix of the N-channel system

\begin{equation}
(3.2) \quad \hat{t} = \begin{pmatrix}
t_{11} & t_{12} & \cdots & t_{1N} \\
t_{21} & \cdots & \cdots & \cdots \\
\cdots & \cdots & \cdots & \cdots \\
t_{N1} & t_{N2} & \cdots & t_{NN}
\end{pmatrix}
\end{equation}

An incoming wave from channel $j$ of an ideal lead at one end of the disordered wire of length $L$ has a coefficient $t_{ij}$ for transmission into channel $i$ of the lead at the other end. The Landauer conductance does not contain information about the contacts or the lead and it is a sole property of the wire under consideration. We are interested in calculating the electron localization properties in a stand-alone wire independent of the contacts, thus $g$ as defined above is suitable. The correctness of the Landauer formula has been experimentally validated for perfectly transmitting channels [90, 91], the case when Eq.(3.2) reduces to
(3.3) \[ g = N \]

where \( N \) is the total number of channels. Remark that the dimensionless conductance \( g = \frac{G}{2e/h} \), where \( G \) is the conductance and \( 2e/h \) is the quantum of conductance.

We now proceed to the calculation of \( g \). The tight-binding model of a one-dimensional disordered two coupled-chain wire was introduced in Chapter 2. It was generated from a single-electron Hamiltonian. The Hamiltonian is as follows,

\[
H = \sum_{i,n} \epsilon_{i,n} |i,n\rangle \langle i,n| + \sum_n h |1,n\rangle \langle 2,n| + \sum_{i,n} t |i,n\rangle \langle i,n + 1| + h.c.
\]

where site number \( n \) on chain \( i \) is represented by \( |i,n\rangle \). Only the nearest-neighbor sites are coupled with each other; the sites on the same chain are coupled through intra-chain coupling parameter \( t \) and the sites of same number at different chains are coupled through inter-chain coupling parameter \( h \). Electron-electron interaction is neglected in this model. The time-independent Schrödinger equation for the wire was given in Eq.(2.15). We will consider the case \( \langle \epsilon_{i,n} \rangle = 0 \), without loss of generality since it is possible to shift the \( E = 0 \) point on the energy-axis to match the average onsite energy later. The randomness (fluctuations) of the on-site energies is the origin of disorder in our model. The Schrödinger equation can be rewritten after normalizing all parameters with respect to \( t \) \( (h \to h/t,E \to E/t,\epsilon_{i,n} \to \epsilon_{i,n}/t) \). We now write Eq.(2.15) normalized with respect to \( t \), in matrix form,

\[
\begin{pmatrix}
\phi_{1,n+1} + \phi_{1,n-1} \\
\phi_{2,n+1} + \phi_{2,n-1}
\end{pmatrix} =
\begin{pmatrix}
E - \epsilon_{1,n} & -h \\
-h & E - \epsilon_{2,n}
\end{pmatrix}
\begin{pmatrix}
\phi_{1,n} \\
\phi_{2,n}
\end{pmatrix}.
\]

The value of \( \phi_{i,n} \), amplitude of the wavefunction at site \( n \) on chain \( i \), is coupled to the values of \( \phi_{i,n+1} \) and to the value of \( \phi_{j,n} \), the amplitude at the site \( n \) on the other chain. The basis \( \phi_{i,n} \) do not represent uncoupled channels since even at the case all \( \epsilon_{i,n} \to 0 \) the amplitudes of the wavefunction on chains 1 and 2 are still coupled, as shown below
(3.6) \[
\begin{pmatrix}
\phi_{1,n+1} + \phi_{1,n-1} \\
\phi_{2,n+1} + \phi_{2,n-1}
\end{pmatrix} = 
\begin{pmatrix}
E & -h \\
-h & E
\end{pmatrix}
\begin{pmatrix}
\phi_{1,n} \\
\phi_{2,n}
\end{pmatrix}.
\]

We want to use perturbation theory to calculate \( g \). The second-order terms in perturbation diverge if we work with a coupled basis. Thus we seek to diagonalize the interchain coupling term \( h \). The unitary transformation that leads to diagonalization is as follows,

(3.7) \[
\begin{pmatrix}
\psi_{1,n} \\
\psi_{2,n}
\end{pmatrix} = \frac{1}{\sqrt{2}} \begin{pmatrix} 1 & 1 \\ 1 & -1 \end{pmatrix} \begin{pmatrix}
\phi_{1,n} \\
\phi_{2,n}
\end{pmatrix}.
\]

The Schrödinger equation in the \( \psi_{i,n} \) basis has the matrix form

(3.8) \[
\begin{pmatrix}
\psi_{1,n+1} + \psi_{2,n-1} \\
\psi_{2,n+1} + \psi_{2,n-1}
\end{pmatrix} = 
\begin{pmatrix}
E - h - \frac{1}{2}(\epsilon_{1,n} + \epsilon_{2,n}) & \frac{1}{2}(\epsilon_{2,n} - \epsilon_{1,n}) \\
\frac{1}{2}(\epsilon_{2,n} - \epsilon_{1,n}) & E + h - \frac{1}{2}(\epsilon_{1,n} + \epsilon_{2,n})
\end{pmatrix}
\begin{pmatrix}
\psi_{1,n} \\
\psi_{2,n}
\end{pmatrix}.
\]

Now Schrödinger equation is written in the "right" basis. In this basis, the wire is a system of two uncoupled channels for electron wave transmission. In what follows, we develop a perturbation theory for this equation.

3.1. Transfer-Matrix Formalism

The matrix equation (3.8) relates the amplitudes of wavefunction \( \psi_{i,n} \) at site \( n \) to the amplitudes at the nearest neighbor sites. Now we focus the attention at freely propagating waves at the leads. The Landauer conductance formula gives conductance in terms of transmission coefficients \( t_{ij} \) for propagating waves in leads through the wire. The leads have a perfectly periodic structure and the wire have energy fluctuations on periodically arranged sites (there is no topological disorder). The propagating waves on the leads are nothing but Bloch waves (2.3) satisfying
(3.9) \[ \psi_{i,n+1}^\pm = e^{\pm \mu_i} \psi_{i,n}^\pm \]

defining \( \psi_{i,n}^+ (\psi_{i,n}^-) \) as amplitudes of a wave propagating from left-to-right (right-to-left) in channel \( i \).

In the transfer-matrix formalism, the propagation of a wave from left-to-right is represented as a matrix equation,

(3.10) \[
\begin{pmatrix}
\varphi_{j+1} \\
\varphi_j 
\end{pmatrix} = X_j 
\begin{pmatrix}
\varphi_j \\
\varphi_{j-1} 
\end{pmatrix}
\]

where \( X_j \) is the so-called transfer-matrix for site \( j \). Matrix \( X_j \) translates the wave from the left of site \( j \) to the right of site \( j \). For our two-channel wire, Eq.(3.10) is replaced by a 4-dimensional matrix equation,

(3.11) \[
\begin{pmatrix}
\psi_{1,n+1} \\
\psi_{1,n} \\
\psi_{2,n+1} \\
\psi_{2,n} 
\end{pmatrix} = \tilde{X}_n 
\begin{pmatrix}
\psi_{1,n} \\
\psi_{1,n-1} \\
\psi_{2,n} \\
\psi_{2,n-1} 
\end{pmatrix}
\]

The transfer matrix \( \tilde{X}_n \) can be derived from (3.8) according to the definition given above, yielding

(3.12) \[
\begin{pmatrix}
\psi_{1,n+1} \\
\psi_{1,n} \\
\psi_{2,n+1} \\
\psi_{2,n} 
\end{pmatrix} = \tilde{X}_n 
\begin{pmatrix}
E - h - a_n & -1 & b_n & 0 \\
1 & 0 & 0 & 0 \\
b_n & 0 & E + h + a_n & -1 \\
0 & 0 & 1 & 0 
\end{pmatrix} 
\begin{pmatrix}
\psi_{1,n} \\
\psi_{1,n-1} \\
\psi_{2,n} \\
\psi_{2,n-1} 
\end{pmatrix}
\]

where \( a_n = \frac{1}{2}(\varepsilon_{1,n} + \varepsilon_{2,n}) \) and \( b_n = \frac{1}{2}(\varepsilon_{2,n} - \varepsilon_{1,n}) \).
The sites located at the ideal leads have no fluctuations in energy (no disorder) with all on-site energies \( \varepsilon_{i,n} \to 0 \) and \( a_n = b_n = 0 \) as well. As discussed before, the solutions of waves propagating along the leads are Bloch waves in form (3.9). If we insert \( \psi^+_i (\psi^-_i) \) into (3.12), we get for the leads

\[
\begin{pmatrix}
  e^{\pm \mu \psi_{1,n}} \\
  e^{\pm \mu \psi_{2,n-1}} \\
  e^{\pm \mu \psi_{2,n}} \\
  e^{\pm \mu \psi_{2,n-1}}
\end{pmatrix}
= 
\begin{pmatrix}
  E - h & -1 & 0 & 0 \\
  1 & 0 & 0 & 0 \\
  0 & 0 & E + h & -1 \\
  0 & 0 & 1 & 0
\end{pmatrix}
\begin{pmatrix}
  \psi_{1,n} \\
  \psi_{1,n-1} \\
  \psi_{2,n} \\
  \psi_{2,n-1}
\end{pmatrix}.
\]

We can now calculate the wavevector \( \mu \) for the Bloch wave from by simply solving the eigenvalue problem (3.13), \( \tilde{X}_n \psi^+_i = e^{\mu \psi^+_i} (\tilde{X}_n \psi^-_i = e^{-\mu \psi^-_i}) \). After simple algebra, the dispersion relations for the waves propagating in both channels take the form

\[
\begin{align*}
2 \cos \mu_1 &= E - h, \\
2 \cos \mu_2 &= E + h.
\end{align*}
\]

The eigenfunctions for the leads are of the form of plane waves,

\[
\psi_{i,n}^{\pm} = e^{\pm i \mu \psi},
\]

where \( \mu_i \) are taken to be positive, \( 0 \leq \mu_i \leq \pi \) so that these functions correspond to plane waves traveling from left to right and from right to left, respectively.

The transfer matrices for the single slices \( n \) (Fig.3.1) are diagonalized in the basis of the plane waves \( \psi^+_i (\psi^-_i) \) that satisfy the Bloch conditions of the system. The transfer matrix \( \tilde{X}_n \) for slice \( n \) in that basis is,
Figure 3.1. Sites \( n \) on both chains are taken as a slice in the channel basis \( \psi_{i,n} \). The transfer matrix \( \tilde{X}_n \) corresponding to slice \( n \) translates the waves by a single site, adding phases or scattering the waves traveling in both directions.

\[
\begin{align*}
\tilde{X}_n &= X_0 + iX_n = \\
&= \begin{pmatrix}
e^{\mu_1} & 0 & 0 & 0 \\
0 & e^{-\mu_1} & 0 & 0 \\
0 & 0 & e^{\mu_2} & 0 \\
0 & 0 & 0 & e^{-\mu_2}
\end{pmatrix} + i
\begin{pmatrix}
\alpha_{1n}e^{\mu_1} & \alpha_{1n}e^{-\mu_1} & -\beta_n e^{\mu_2} & -\beta_n e^{-\mu_2} \\
-\alpha_{1n}e^{\mu_1} & -\alpha_{1n}e^{-\mu_1} & \beta_n e^{\mu_2} & \beta_n e^{-\mu_2} \\
-\beta_n e^{\mu_1} & -\beta_n e^{-\mu_1} & \alpha_{2n} e^{\mu_2} & \alpha_{2n} e^{-\mu_2} \\
\beta_n e^{\mu_1} & \beta_n e^{-\mu_1} & -\alpha_{2n} e^{\mu_2} & -\alpha_{2n} e^{-\mu_2}
\end{pmatrix},
\end{align*}
\]

where \( X_0 \) is the transfer matrix of the periodic chain and \( X_n^{(1)} \) is the correction due to weak disorder. \( X_n^{(1)} \) is responsible for scattering processes that take place while the waves travel along the channels. The parameters \( \alpha_{i,n} \) and \( \beta_n \) are defined as

\[
\begin{align*}
\alpha_{1n} &= \frac{\epsilon_{1,n} + \epsilon_{2,n}}{4 \sin \mu_1}, \\
\alpha_{2n} &= \frac{\epsilon_{1,n} + \epsilon_{2,n}}{4 \sin \mu_2}, \\
\beta_n &= \frac{\epsilon_{2,n} - \epsilon_{1,n}}{4 \sqrt{\sin \mu_1 \sin \mu_2}}.
\end{align*}
\]

The role of the transfer matrix \( \tilde{X}_n \) is to relate the amplitudes of the plane waves on the left and right side of the slice,

\[
\begin{pmatrix}
\psi_{1,n+1}^+ \\
\psi_{1,n+1}^- \\
\psi_{2,n+1}^+ \\
\psi_{2,n+1}^-
\end{pmatrix} = \tilde{X}_n \begin{pmatrix}
\psi_{1,n}^+ \\
\psi_{1,n}^- \\
\psi_{2,n}^+ \\
\psi_{2,n}^-
\end{pmatrix}.
\]
Now we have the transfer matrix for a single slice and the transfer matrix $X$ corresponding to the whole system is the product

\begin{equation}
X = \prod_{n=1}^{\ell} \hat{X}_n
\end{equation}

The total length of the system is $L a$ where $a$ is the distance between each slice. The transfer matrix of the whole systems relates the amplitude of the waves at both ends of the wire, similar to (3.18),

\begin{equation}
\begin{pmatrix}
\psi^+_{1,L} \\
\psi^-_{1,L} \\
\psi^+_{2,L} \\
\psi^-_{2,L}
\end{pmatrix} = X \begin{pmatrix}
\psi^+_{1,0} \\
\psi^-_{1,0} \\
\psi^+_{2,0} \\
\psi^-_{2,0}
\end{pmatrix}
\end{equation}

3.2. Lyapunov Exponent

The matrices $\hat{X}_n$ are random matrices, because the part $X_n^{(1)}$ contains random on-site energies $\varepsilon_{1,n}$ and $\varepsilon_{2,n}$ of the disordered wire. Since we are considering infinite length wires where $L \to \infty$, the final transfer matrix is a multiplication of infinite number of random matrices.

Lyapunov exponents emerge from the random matrix theory [92], and they are used to characterize the asymptotic behavior of the systems determined by the products of such matrices. The ergodic theorem of Oseledec [93] and Tutubalin [94] states that for a product of infinite number of random matrices belonging to the same statistical ensemble

\begin{equation}
M_L = M_L M_{L-1} \cdots M_1,
\end{equation}

there exists a limiting matrix $\Gamma$,

\begin{equation}
\lim_{L \to \infty} (M_L^T M_L)^{\frac{1}{L}} \equiv \Gamma \geq 0,
\end{equation}
and its eigenvalues can be written as $e^{\lambda_i}$. The maximum of these Lyapunov characteristic exponents $\gamma = \max(\lambda_1, \lambda_2, \ldots, \lambda_d)$ is given by

$$\gamma = \lim_{L \to \infty} \frac{1}{L} \ln(M_L).$$

The Lyapunov exponent (3.23) is directly related to Eq. (2.9) for the inverse localization length. The relation of dimensionless conductance $g$ to the product of random transfer matrices $\hat{X}_n$ is shown in the following section in detail. It is sufficient at the moment to say that in the limit $L \to \infty$, Oseledec’s theorem dictates that we get the maximum of the Lyapunov coefficients. This Lyapunov exponent, in relation to the formula in Eq. (2.9), gives the minimum localization length. The minimum localization length is the one that determines the mobility of the electrons with a given energy inside a disordered medium.

3.3. Scattering Matrix and Conductance

The scattering of plane waves - reflection and transmission - at and between the two ends of the 1-D systems is governed by the scattering matrix $S$,

$$\hat{S} = \begin{pmatrix} \hat{r}^{++} & \hat{t}^{--} \\ \hat{t}^{+-} & \hat{r}^{--} \end{pmatrix},$$

where

$$\hat{t}^{\pm\pm} = \begin{pmatrix} t_{11}^{\pm} & t_{12}^{\pm} \\ t_{21}^{\pm} & t_{22}^{\pm} \end{pmatrix}, \quad \hat{r}^{\pm\pm} = \begin{pmatrix} r_{11}^{\pm} & r_{12}^{\pm} \\ r_{21}^{\pm} & r_{22}^{\pm} \end{pmatrix}.$$

Here $t_{ij}^{++}(t_{ij}^{--})$ and $r_{ij}^{++}(r_{ij}^{--})$ are the transmission and reflection amplitudes in the channel $i$, provided there is a unit flux incident from left(right) in the channel $j$. The transmission matrix $\hat{t}$ in Landauer conductance formula (3.1) is nothing but one of the transfer matrices $t^{--}$ or $t^{++}$, which are equal since the system has time-reversal invariance.

In general, the scattering matrix expresses outgoing wave amplitudes in terms of ingoing ones on either side of the quasi-1D disordered wire via the scattering relations [95, 96],
\[(3.26) \quad \begin{pmatrix} O \\ O' \end{pmatrix} = S \begin{pmatrix} I \\ I' \end{pmatrix}.
\]

Here \( I \) and \( I' \) (\( O \) and \( O' \)) denote ingoing (outgoing) amplitudes at the left and right sides of the disordered region, respectively.

In our case, the matrix \( S \) is defined as follows

\[(3.27) \quad \begin{pmatrix} \psi_{1,0}^- \\ \psi_{2,0}^- \\ \psi_{1,L}^+ \\ \psi_{2,L}^+ \end{pmatrix} = S \begin{pmatrix} \psi_{1,0}^+ \\ \psi_{2,0}^+ \\ \psi_{1,L}^- \\ \psi_{2,L}^- \end{pmatrix}.
\]

Since

\[(3.28) \quad \begin{pmatrix} \psi_{1,L}^+ \\ \psi_{1,L}^- \\ \psi_{2,L}^+ \\ \psi_{2,L}^- \end{pmatrix} = \begin{pmatrix} X_{11} & X_{12} & X_{13} & X_{14} \\ X_{21} & X_{22} & X_{23} & X_{24} \\ X_{31} & X_{32} & X_{33} & X_{34} \\ X_{41} & X_{42} & X_{43} & X_{44} \end{pmatrix} \begin{pmatrix} \psi_{1,0}^+ \\ \psi_{2,0}^+ \\ \psi_{1,0}^- \\ \psi_{2,0}^- \end{pmatrix},
\]

it follows from simple algebra that

\[(3.29) \quad \hat{t} = \hat{t}^{--} = \frac{1}{\delta} \begin{pmatrix} X_{44} & -X_{24} \\ -X_{42} & X_{22} \end{pmatrix},\]

\[(3.30) \quad \delta = X_{22}X_{44} - X_{24}X_{42}.
\]

Finally, the dimensionless conductance \( g \) can be written in terms of elements of transmission matrix \( X \),

\[(3.31) \quad g = \rho \frac{\rho}{|\delta|^2} = \frac{|X_{22}|^2 + |X_{44}|^2 + |X_{42}|^2 + |X_{24}|^2}{|X_{22}X_{44} - X_{24}X_{42}|^2}.
\]
3.4. Calculation of Transfer Matrix in Second-Order Born Approximation

The total scattering matrix $X$ is a product matrices represented with a diagonal part $X_0$ plus non-diagonal part $X^{(1)}_n$ that depends linearly on the on-site energies.

\[
X = \prod_{n=1}^L (X_0 + X^{(1)}_n) = X_0^L + i \sum_n X_0^{L-n} \cdot X^{(1)}_n \cdot X_0^{n-1} - \sum_m X_0^{L-m-n} \cdot X^{(1)}_m \cdot X_0^{m-n} \cdot X^{(1)}_n \cdot X_0^{n-1} \\
+ F(O(\varepsilon^3)) + G(O(\varepsilon^4)) + \cdots
\]

where $F(O(\varepsilon^3)) \ (G(O(\varepsilon^4)))$ is a function of the parameter in third-order (fourth-order) in $\varepsilon$, the disorder in on-site energies. We limit our discussion to the weak disorder case, where

\[
\varepsilon_{i,n} << 1
\]

and continue our calculations within second-order in Born approximation. So, the total transfer matrix $X$ will only include terms that are smaller or equal to second-order in $\varepsilon$. The first three-terms in (3.32) remain in $X = A + iB - C$, where

\[
A = X_0^L = \begin{pmatrix}
 e^{\mu_1 L} & 0 & 0 & 0 \\
 0 & e^{-\mu_1 L} & 0 & 0 \\
 0 & 0 & e^{\mu_2 L} & 0 \\
 0 & 0 & 0 & e^{-\mu_2 L}
\end{pmatrix},
\]

\[
B = \sum_n \begin{pmatrix}
 e^{\mu_1 L} \alpha_{1n} & e^{\mu_1 L} e^{-2\mu_1 n} \alpha_{1n} & -e^{\mu_1 L} e^{(\mu_1 - \mu_2) n} \beta_n & -e^{\mu_1 L} e^{- (\mu_1 + \mu_2) n} \beta_n \\
 -e^{-\mu_1 L} e^{2\mu_1 n} \alpha_{1n} & -e^{-\mu_1 L} \alpha_{1n} & -e^{-\mu_1 L} e^{(\mu_1 + \mu_2) n} \beta_n & -e^{-\mu_1 L} e^{- (\mu_1 - \mu_2) n} \beta_n \\
 -e^{\mu_2 L} e^{(\mu_1 - \mu_2) n} \beta_n & -e^{\mu_2 L} e^{- (\mu_1 + \mu_2) n} \beta_n & e^{\mu_2 L} \alpha_{2n} & e^{\mu_2 L} e^{2\mu_2 n} \alpha_{2n} \\
e^{-\mu_2 L} e^{(\mu_1 + \mu_2) n} \beta_n & e^{-\mu_2 L} e^{- (\mu_1 - \mu_2) n} \beta_n & -e^{-\mu_2 L} e^{2\mu_2 n} \alpha_{2n} & -e^{-\mu_2 L} \alpha_{2n}
\end{pmatrix},
\]

and the terms in $C$ that we need ($C_{22}, C_{24}, C_{42}$ and $C_{44}$).
(3.36) \[ C_{22} = \sum_{m>n} e^{-\mu_1 L} [(1 - e^{2\mu_1 (m-n)}) \alpha_{1m} \alpha_{1n} + (e^{i(\mu_1 - \mu_2)(m-n)} - e^{i(\mu_1 + \mu_2)(m-n)}) \beta_m \beta_n), \]

(3.37) \[ C_{44} = \sum_{m>n} e^{-\mu_2 L} [(1 - e^{2\mu_2 (m-n)}) \alpha_{2m} \alpha_{2n} + (e^{i(\mu_2 - \mu_1)(m-n)} - e^{i(\mu_1 + \mu_2)(m-n)}) \beta_m \beta_n), \]

(3.38) \[ C_{24} = \sum_{m>n} e^{-\mu_1 L} e^{i(\mu_2 - n\mu_2)[(e^{2\mu_2 (m-n)} - e^{-\mu_2 (m-n)}) \beta_m \alpha_{2n} + (e^{\mu_1 (m-n)} - e^{-\mu_1 (m-n)}) \beta_m \alpha_{1n}], \]

(3.39) \[ C_{42} = \sum_{m>n} e^{-\mu_2 L} e^{i(\mu_2 - n\mu_1)[(e^{\mu_2 (m-n)} - e^{-\mu_2 (m-n)}) \beta_m \alpha_{1n} + (e^{\mu_1 (m-n)} - e^{-\mu_1 (m-n)}) \beta_m \alpha_{2n}], \]

Given below are the elements of the total transmission matrix \( X \) that we will use,

\[
X_{22} = e^{-\mu_1 L} \left[ 1 + i \sum_m \alpha_{1m} - \sum_{m>n} (1 - e^{2\mu_1 (m-n)}) \alpha_{1m} \alpha_{1n} - \sum_{m>n} (e^{i(\mu_1 - \mu_2)(m-n)} - e^{i(\mu_1 + \mu_2)(m-n)}) \beta_m \beta_n, \right]
\]

(3.40) \[
X_{44} = e^{-\mu_2 L} \left[ 1 + i \sum_m \alpha_{2m} - \sum_{m>n} (1 - e^{2\mu_2 (m-n)}) \alpha_{2m} \alpha_{2n} - \sum_{m>n} (e^{i(\mu_2 - \mu_1)(m-n)} - e^{i(\mu_1 + \mu_2)(m-n)}) \beta_m \beta_n, \right]
\]

\[
X_{24} = -e^{-\mu_1 L} \left[ i \sum_m e^{i(\mu_2 - m\mu_2)} \beta_m + O(\varepsilon^2) \right],
\]

\[
X_{42} = -e^{-\mu_2 L} \left[ i \sum_m e^{i(\mu_2 - m\mu_1)} \beta_m + O(\varepsilon^2) \right].
\]

The term \( \delta \) in Eq.(3.31) is

\[
\delta = e^{-(\mu_1 + \mu_2) L} \left[ 1 + i \sum_n (\alpha_{1n} + \alpha_{2n}) - \sum_{m>n} (\alpha_{1m} \alpha_{2n} - e^{(\mu_1 - \mu_2)(m-n)} \beta_m \beta_n) - \sum_{m>n} \left[ e^{i(\mu_1 - \mu_2)(m-n)} + e^{-i(\mu_1 - \mu_2)(m-n)} - 2e^{i(\mu_1 + \mu_2)(m-n)} \right] \beta_m \beta_n - \sum_{m>n} [(1 - e^{2\mu_2 (m-n)}) \alpha_{1m} \alpha_{1n} + (1 - e^{2\mu_2 (m-n)}) \alpha_{2m} \alpha_{2n}] \right],
\]

with

35
\[ |\delta|^2 = 1 + \sum_{m,n} (\alpha_{1m} + \alpha_{2m})(\alpha_{1n} + \alpha_{2n}) - 2 \sum_{m,n} (\alpha_{1m}\alpha_{2n}) + 2 \sum_{m,n} \cos(k_1 - k_2)(m-n)\beta_m\beta_n \]
\[ - 4 \sum_{m>n} \cos(\mu_1 - \mu_2)(m-n)\beta_m\beta_n - 4 \sum_{m>n} \cos(\mu_1 + \mu_2)(m-n)\beta_m\beta_n \]
\[ - 2 \sum_{m>n} [(1 - \cos 2\mu_1(m-n))\alpha_{1m}\alpha_{1n} + (1 - \cos 2\mu_2(m-n))\alpha_{2m}\alpha_{2n}] \]

We can simplify the equality a bit more by noting

\[ \sum f(m, n) = \sum f(n, n) + 2 \sum f(m, n) \]

so that

\[ |\delta|^2 = 1 + \sum_{n} \alpha_{1n}^2 + \alpha_{2n}^2 + 2\beta_n^2 \]

\[ - 4 \sum_{m>n} \cos(\mu_1 + \mu_2)(m-n)\beta_m\beta_n \]
\[ + 2 \sum_{m>n} [\cos 2\mu_1(m-n)\alpha_{1m}\alpha_{2n} + \cos 2\mu_2(m-n)\alpha_{2m}\alpha_{1n}] \]

Similar calculations yield \( \rho = 1 + |\delta|^2 \). At the end, we get for inverse localization length \( l^{-1} \)

\[ l^{-1} = - \lim_{L \to \infty} \frac{1}{2L} \ln \left( \frac{1 + |\delta|^2}{|\delta|^2} \right). \]

Let's take a look at \( |\delta|^2 \):

\[ |\delta|^2 = 1 + \beta(\varepsilon^2) \Rightarrow \ln \left( \frac{1 + |\delta|^2}{|\delta|^2} \right) \simeq - \frac{\beta}{2} \]

when we keep only the second-order terms in \( \varepsilon \). This brings us to,
\[ l^{-1} = \lim_{L \to \infty} \frac{1}{4L} \left\langle \sum_m \alpha_{1m}^2 + \alpha_{2m}^2 + 2\beta_m^2 \right\rangle \]

\[ + 4 \left\langle \sum_p \cos((\mu_1 + \mu_2)p) \sum_n \beta_n \beta_{n+p} \right\rangle \]

\[ + 2\left( \sum_p [\cos(2\mu_1 p) \sum_n \alpha_1 \alpha_{1(n+p)} + \cos(2\mu_2 p) \sum_n \alpha_2 \alpha_{2(n+p)}] \right). \]

The result is in terms of \( \alpha \)'s and \( \beta \)'s, previously defined in (3.17). If we trace the conversions back,

\[
\alpha_{1m}^2 = \frac{(\varepsilon_{1,m} + \varepsilon_{2,m})^2}{16 \sin^2 \mu_1} = \frac{\varepsilon_{1,m}^2 + \varepsilon_{2,m}^2 + 2\varepsilon_{1,m} \varepsilon_{2,m}}{16 \sin^2 \mu_1},
\]

\[ \alpha_{2m}^2 = \frac{\varepsilon_{1,m}^2 + \varepsilon_{2,m}^2 + 2\varepsilon_{1,m} \varepsilon_{2,m}}{16 \sin^2 \mu_2}, \]

\[ \beta_m^2 = \frac{\varepsilon_{1,m}^2 + \varepsilon_{2,m}^2 - 2\varepsilon_{1,m} \varepsilon_{2,m}}{16 \sin \mu_1 \sin \mu_2}, \]

and also,

\[ \alpha_{1m} \alpha_{1(m+p)} = \frac{\varepsilon_{1,m} \varepsilon_{1,(m+p)} + \varepsilon_{2,m} \varepsilon_{2,(m+p)} + \varepsilon_{2,m} \varepsilon_{1,(m+p)} + \varepsilon_{1,m} \varepsilon_{2,(m+p)}}{16 \sin^2 \mu_1}, \]

\[ \beta_m \beta_{m+p} = \frac{\varepsilon_{1,m} \varepsilon_{1,(m+p)} + \varepsilon_{2,m} \varepsilon_{2,(m+p)} - \varepsilon_{2,m} \varepsilon_{1,(m+p)} - \varepsilon_{1,m} \varepsilon_{2,(m+p)}}{16 \sin \mu_1 \sin \mu_2}. \]

Defining correlation functions \( \xi_i(p) \) as follows

\[ \varepsilon_{10}^2 \xi_{11}(p) = \frac{\sum_m \varepsilon_{1,m} \varepsilon_{1,(m+p)}}{L}, \]

\[ \varepsilon_{20}^2 \xi_{22}(p) = \frac{\sum_m \varepsilon_{2,m} \varepsilon_{2,(m+p)}}{L}, \]

\[ \varepsilon_{12} \xi_{12}(p) = \frac{\sum_m \varepsilon_{1,m} \varepsilon_{2,(m+p)}}{L}, \]

we obtain the final result for the inverse localization length \( l^{-1}(E) \) as given below:
\[ l^{-1}(E) = \frac{\varepsilon_{10}^2}{64} \left[ \frac{\varphi_{11}(2\mu_1)}{\sin^2 \mu_1} + \frac{\varphi_{11}(2\mu_2)}{\sin^2 \mu_2} + \frac{2\varphi_{11}(\mu_1 + \mu_2)}{\sin \mu_1 \sin \mu_2} \right] + \frac{\varepsilon_{20}^2}{64} \left[ \frac{\varphi_{22}(2\mu_2)}{\sin^2 \mu_2} + \frac{\varphi_{22}(2\mu_1)}{\sin^2 \mu_1} + \frac{2\varphi_{22}(\mu_1 + \mu_2)}{\sin \mu_1 \sin \mu_2} \right] + \frac{\varepsilon_{12}^2}{32} \left[ \frac{\varphi_{12}(2\mu_1)}{\sin^2 \mu_1} + \frac{\varphi_{12}(2\mu_2)}{\sin^2 \mu_2} - \frac{2\varphi_{12}(\mu_1 + \mu_2)}{\sin \mu_1 \sin \mu_2} \right]. \] (3.51)

The structure of this formula for the inverse localization length is similar to the corresponding formula (2.12) for a single-channel system. The localization of an electron occurs due to elastic backscattering processes in both channels with change of the momentum by \(2\mu_1\) and \(2\mu_2\) and due to inter-channel scattering with change of the momentum by \(\mu_1 + \mu_2\). An unperturbed wave, which according to Eq.(3.7) is either symmetric or anti-symmetric is scattered at three random potentials with variances \(\varepsilon_1^2\), \(\varepsilon_2^2\), and \(\varepsilon_{12}\). The correlations enter through the functions \(\varphi_{ij}(\mu)\), which are represented by the Fourier series,

\[ \varphi_{ij}(\mu) = 1 + 2 \sum_{k=1}^{\infty} \xi_{ij}(k) \cos(\mu k). \] (3.52)

The inverse localization length (3.51) is positively defined for all the allowed energies. In the case of uncoupled channels, \(h = 0\), the dispersion relations (3.14) are identical, \(\mu_1 = \mu_2 = \mu\), and the cross-correlation term vanishes. As a result Eq.(3.51) is simplified to the following form

\[ l^{-1}(E) = \frac{1}{\sin^2 \mu} \left( \varepsilon_1^2 \varphi_{11}(2\mu) + \varepsilon_2^2 \varphi_{22}(2\mu) \right). \] (3.53)

For identical channels, \(\varepsilon_{10} = \varepsilon_{20} = \varepsilon_0\) and \(\varphi_{11}(2\mu) = \varphi_{22}(2\mu)\) the single-chain case (2.12) is recovered. Finally, if the potentials in the both channels are uncorrelated (white-noise) then \(\varphi_{ij}(\mu) \equiv 1\) and \(\varepsilon_{12} = 0\). In this case (3.51) reproduces Heinrichs’ result (2.16) [85],

\[ l^{-1}(E) = \frac{\varepsilon_0^2}{32} \left( \frac{1}{\sin \mu_1} + \frac{1}{\sin \mu_2} \right)^2. \] (3.54)

3.5. One Evanescent and One Propagating Mode

In the energy regions \(-2 - h < E < h - 2\) and \(2 - h < E < 2 + h\), one of the wave numbers \(\mu_i\) is necessarily a pure imaginary number. The corresponding wave function decays
exponentially away from the entering point with decrement $|\mu_i|$. Since the transmission matrix is a relation between the propagating wave modes only, the evanescent modes do not contribute to the conductance of a long sample if $L \gg |\mu_i|^{-1}$.

It was shown in Heinrichs' paper [85] that in the case of uncorrelated disorder the evanescent mode term does not contribute to the Lyapunov exponent (2.16) and has to be omitted. Moreover, the coupling between the propagating and evanescent modes is strongly suppressed. This results in an extra factor of 2 in (2.16). In what follows I demonstrate that this scenario of transition from propagating to evanescent regime remains unchanged in the case of correlated potential.

It is worthwhile to note that in weak disorder approximation the formulas for the Lyapunov exponents are invalid in the vicinity of the critical energies $E_c = \pm 2 \pm h$, where the transition from propagating to evanescent regime occurs. At these energies the perturbation $\varepsilon_i/\sin \mu_i \to \infty$ and the Born approximation fails even for weak disorder.

Let us consider the energy domain $2-h < E < 2+h$ where the second mode is evanescent, $\mu_2 = i\kappa$. The transfer matrix of the $n$th site, $\hat{X}_n$, establishes a linear relation between the wave functions on the both side of this site. Since the evanescent mode does not contribute to the conductance, we are interested only on the elements of the transfer matrix which describe scattering from propagating to propagating mode. For the propagating mode with index 1 those elements are at the 2x2 upper left block of the 4x4 transmission matrix $\hat{X}_n$.

\[
\begin{pmatrix}
\psi_{1,n+1}^+ \\
\psi_{1,n+1}^-
\end{pmatrix} = \begin{pmatrix}
\hat{X}_{n,11} & \hat{X}_{n,12} \\
\hat{X}_{n,21} & \hat{X}_{n,22}
\end{pmatrix} \begin{pmatrix}
\psi_{1,n+1}^- \\
\psi_{1,n+1}^+
\end{pmatrix}.
\]

(3.55)

Now we can introduce a transfer matrix for the $n$th site, using the basis of the propagating modes only

\[
\begin{pmatrix}
\psi_{1,n+1}^+ \\
\psi_{1,n+1}^-
\end{pmatrix} = \hat{T}_n \begin{pmatrix}
\psi_{1,n}^+ \\
\psi_{1,n}^-
\end{pmatrix}, \quad \hat{T}_n = \begin{pmatrix}
\hat{X}_{n,11} & \hat{X}_{n,12} \\
\hat{X}_{n,21} & \hat{X}_{n,22}
\end{pmatrix}.
\]

(3.56)
Similar to the transfer matrix $\tilde{X}_n$, the transfer matrix $\tilde{T}_n$ has a zero-order diagonal component and linear over the random potential term,

\begin{equation}
\tilde{T}_n = T_0 + T_n^{(1)} = \begin{pmatrix}
e^{i\mu_i} & 0 \\
0 & e^{-i\mu_i}
\end{pmatrix} + \begin{pmatrix}
ie^{i\mu_i}a_n & ie^{-i\mu_i} \\
-ie^{i\mu_i}a_n & -ie^{-i\mu_i}
\end{pmatrix}.
\end{equation}

The matrix $\tilde{T}_n$, being a unitary matrix, conserves the flux. The total transfer matrix $T$ of a sample is a product of all $\tilde{T}_n$ matrices. Keeping up to quadratic over disorder terms in this product, a formula similar to Eq.(3.32) is obtained,

\begin{equation}
T = \prod_{n=1}^{L} \tilde{T}_n = T_0^L + \sum_{m=1}^{L} T_0^{L-m} \cdot T_m^{(1)} \cdot T_0^{m-1} + \sum_{m>n} T_0^{L-m} \cdot T_m^{(1)} \cdot T_0^{m-n-1} \cdot T_n^{(1)} \cdot T_0^{n-1}.
\end{equation}

In the presence of evanescent mode the dimension of the scattering matrix is also reduced since now it relates the incoming and outgoing components of the propagating wave only,

\begin{equation}
\begin{pmatrix}
\psi_{1,0}^+ \\
\psi_{1,0}^-
\end{pmatrix} = T \begin{pmatrix}
\psi_{1,L}^+ \\
\psi_{1,L}^-
\end{pmatrix}, \quad \begin{pmatrix}
\psi_{1,0}^- \\
\psi_{1,0}^+
\end{pmatrix} = S \begin{pmatrix}
\psi_{1,0}^+ \\
\psi_{1,0}^-
\end{pmatrix}.
\end{equation}

The transmission matrix $\tilde{\ell}$, which determines the conductance becomes a scalar. It is obtained from the scattering matrix,

\begin{equation}
S = \frac{1}{T_{11}} \begin{pmatrix}
T_{21} & 1 \\
1 & -T_{12}
\end{pmatrix}, \quad |\tilde{\ell}| = |T_{11}|^{-2}.
\end{equation}

Thus, in the presence of evanescent mode the conductance (and hence the localization length) is determined by $|T_{11}|^2$. This quantity can be calculated from (3.58) and in the quadratic approximation we get,

\begin{equation}
|T_{11}|^2 = 1 + \sum_{m,n} \alpha_m \alpha_n - 2 \sum_{m>n} \alpha_m \alpha_n + 2 \sum_{m>n} \cos 2\mu_1 (m-n) \alpha_m \alpha_n.
\end{equation}

Now, expanding $\ln |\tilde{\ell}| = -\ln |T_{11}|^2$, the following result for the inverse localization length is obtained,

\begin{equation}
l^{-1}(E) = \frac{1}{32\sin^2 \mu_1} \left[ \epsilon_1^2 \phi_{11}(2\mu_1) + \epsilon_2^2 \phi_{22}(2\mu_1) + 2\epsilon_{12} \phi_{12}(2\mu_1) \right].
\end{equation}
In the counterpart region $-2 - h < E < -2 + h$ the first mode becomes evanescent and the Bloch number $\mu_1$ in (3.62) is replaced by $\mu_2$.

The localization length is a complicated linear functional of the correlation functions $\xi_\mu(k)$. Analysis of the localization length for different classes of short- and long-range correlations requires more work. It is not clear yet what kind of long-range correlations are sufficient for the mobility edge to appear in the spectrum of two-channel system. Here I give numerical results for uncorrelated potential and for exponentially decaying correlation function of the following form

\begin{equation}
(3.63)\quad \xi_{ij}(k) = (-1)^k e^{-\alpha |k|}
\end{equation}

where $\alpha$ is the inverse radius of correlations. Here correlations alternate with anti-correlations. The localization length for this specific correlation function shows oscillatory behavior in the interval of $E$ where both channels are open. Unlike this, in the regions where only one of the channels is propagating the localization length is a monotonic function of energy. There is a discontinuous jump for $l(E)$ at the critical energies $E = \pm(2 - h)$ where a transition from propagating to evanescent mode occurs. These discontinuities are clear evidences of the fact that Born approximation is not valid in the vicinities of the critical points. Extended states do not appear for this class of short-range correlations. They probably may appear if the correlations are of long-range, i.e. the correlations decay as a power law.

3.6. Discussions

In the single-channel case the "inverse" scattering problem - reconstruction of the statistical ensemble of the correlated potentials through the localization length $l(E)$ - has a unique solution. It follows from the general properties of the Fourier integrals that if the Lyapunov exponent vanishes within a finite interval of energies, the correlation function decays as a power law, $\xi(k) \propto 1/k^p$. For a sharp mobility edge the parameter $p = 1$ [75, 18, 76].

In a two-channel system the inverse scattering problem is more complicated since there are three correlators, $\xi_{11}(k)$, $\xi_{22}(k)$, and $\xi_{12}(k)$, which have to be reconstructed from the function $l^{-1}(E)$, given by Eqs.(3.51) and (3.62). However, it is clear from the structure of
Figure 3.2. The localization length vs energy graphs for various correlated/non-correlated disorder is given above. Solid line is for uncorrelated (white-noise) potential. Dashed line is for exponential intra- and inter-channel correlation functions, all three are given by (3.63). Dotted line is for exponential intra-channel correlations and delta-correlated inter-channel scattering, $\langle \varepsilon_i \varepsilon_j \rangle \propto \delta_{ij}$.

The parameters of the model are $h = 0.5$ eV, $t = 1.0$ eV and $\langle \varepsilon_1^2 \rangle = \langle \varepsilon_2^2 \rangle = \langle \varepsilon_{12}^2 \rangle = 0.25^2$.

Eqs. (2.12), (3.51) and (3.62) that the power-decaying correlation functions are necessary in order to have a mobility edge. If the correlations are of a short-range, only a discrete set of resonant extended states may appear in the spectrum.

In particular, an extended state at the band center $E = 0$ was predicted for a two-channel random dimer for some specific parameters of the random potential [77]. The random dimer is an example of a dichotomous sequence where the on-site potential takes on only two values, e.g, $\varepsilon_0$ and $-\varepsilon_0$. For all sites $\varepsilon_{1,n} = \varepsilon_{2,n}$. At a site $n$ the value $\varepsilon_0$ or $-\varepsilon_0$ emerges with probability of $1/2$ and it is repeated at the nearest site $n + 1$. Statistical properties of this model are characterized by the mean value $\langle \varepsilon_n \rangle = 0$, variances $\langle \varepsilon_1^2 \rangle = \langle \varepsilon_2^2 \rangle = \varepsilon_0^2$, $\varepsilon_{12} = \langle \varepsilon_{1,n} \varepsilon_{2,n} \rangle = \varepsilon_0^2$ and the correlators,
\[
(3.64) \quad \xi_{11}(1) = \xi_{22}(1) = \xi_{12}(1) = \langle \varepsilon_n \varepsilon_{n-1} \rangle = 1/2.
\]

and \(\xi_{ij}(k > 1) = 0\). By substituting this correlator into Eq. (3.51), we obtain the inverse localization length for the dimer,

\[
(3.65) \quad \Gamma^{-1}(E) = \frac{\varepsilon_0^2}{8} \left( \cot^2 \mu_1 + \cot^2 \mu_2 \right).
\]

This function does not vanish, if the inter-channel hopping parameter \(h\) is different from zero, i.e. there is no extended state in a two-channel dimer. Unlike this, in a single-channel dimer there are two extended states, which in the case of weak disorder are situated in the vicinity of the band center \(E = 0\) [55]. Indeed, the Lyapunov exponent Eq. (3.65) vanishes quadratically at a single point \(E = 0\) if \(\mu_1 = \mu_2 = \pi/2\). Eq. (3.65) is valid for weakly disordered potential, when \(\varepsilon_0 \ll 1\), therefore it does not show the existence of the extended state at \(E = 0\) for the dimer with \(\varepsilon_0 = 1\) [77].

Finally, I applied the obtained results to the two-stranded DNA molecule to make an analytical investigation. Caetano and Schultz used a DNA-like structure where the four basic nucleotides are evenly represented in a molecule and both strands form uncorrelated sequences of the nucleotides [78]. In terms of my parameters it means that \(\varepsilon_{12} = (\varepsilon_A \varepsilon_T + \varepsilon_C \varepsilon_G)/2\), \(\langle \varepsilon_{1,n}^2 \rangle = \langle \varepsilon_{2,n}^2 \rangle = (\varepsilon_A^2 + \varepsilon_T^2 + \varepsilon_C^2 + \varepsilon_G^2)/4\), and \(\langle \varepsilon_{1,n+k} \varepsilon_{1,n} \rangle = \langle \varepsilon_{2,n+k} \varepsilon_{2,n} \rangle = 0\). Substituting these values into Eq. (3.51) we get,

\[
(3.66) \quad \Gamma^{-1}(E) = \frac{1}{128} \left[ (\varepsilon_A + \varepsilon_T)^2 + (\varepsilon_C + \varepsilon_G)^2 \right] \left( \frac{1}{\sin^2 \mu_1} + \frac{1}{\sin^2 \mu_2} \right) + \frac{1}{64 \sin \mu_1 \sin \mu_2} \left[ (\varepsilon_A - \varepsilon_T)^2 + (\varepsilon_C - \varepsilon_G)^2 \right].
\]

Here the electron energy \(E\), which enters through the dispersion relations Eq. (4.5) and the on-site energies of the nucleotides are counted from the mean site energy \(\langle \varepsilon_{1,n} \rangle\). Since \(\sin \mu_1\) and \(\sin \mu_2\) are positive functions \((0 \leq \mu_1, \mu_2 \leq \pi)\), the Lyapunov exponent Eq. (3.66) is a sum of two positive quantities, independently on the particular values of the on-site energies. This
means that the discussed model of DNA does not allow existence of the extended states. A band of extended states were predicted in Ref.[78], using numerical simulations of the inverse participation ratio. Later this result has been criticized in Ref.[79] on the basis of group theory arguments. Formula (3.66) explicitly shows that in a two-channel system the extended states cannot appear solely due to base pairing A-T, C-G. The base pairing indeed decreases the Lyapunov exponent Eq.(3.51) because of the negative value of the parameter $\varepsilon_{12}$, but this pairing (even being strong) is not enough to give rise to the extended states in the uncorrelated DNA strands. Unlike this, the long-range correlations in a single-stranded model of DNA may lead to a band of extended states [99].
CHAPTER 4

ELECTRON LOCALIZATION IN DNA

Electrons in a DNA molecule can be classified in two groups: The first group consists of the electrons that are strongly bound to the molecular structures of the nucleic bases and sugar phosphate groups. These electrons’ wavefunctions are restricted to the spatial extent of the host molecules. The second group of electrons are those that are loosely bound to the molecules, somewhat free to move (or hop) from one molecule to another, i.e. from base to base or base to sugar phosphate groups. Unlike the first group of electrons, the second group of electrons have wavefunctions that cover several base lengths. They are also the electrons that contribute to electronic transport in DNA. We speak of the localization of that second group of electrons.

The structure of DNA was discussed in Section 1.1. As mentioned there, the most likely mechanism responsible for the electron transport is the hopping between bases due to the overlap of the $\pi$-orbitals ($\pi$ stacking) [97]. Electrons occupying these orbitals have finite quantum mechanical probabilities to jump to the neighboring molecular orbital. The interactions of electrons with the rest of the system is taken into consideration when molecular orbital energies and hopping parameters are calculated. This fact makes it possible to develop a single-electron Hamiltonian when solving for the eigenfunctions and transport properties of the whole DNA molecule.

Two popular models for electronic transport in DNA are considered in this thesis, the Fishbone model and the two-channel wire model. Both are based on tight-binding single-electron Hamiltonians. All calculations are carried out at $T = 0$ K so coupling to phonons etc. is thoroughly ignored. In both models, parameters for electron hopping and on-site energies are borrowed from ab-initio calculations that strongly agree with empirical findings [37, 98].

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4.1. Fishbone Model of DNA

In the Fishbone model of DNA, electron transport in a DNA molecule is considered as discrete jumps between the neighboring base nucleotides. Due to strong A-T and C-G coupling, it is usually considered that there is a single electron channel with a binary sequence of the on-site potentials with energies \( \epsilon_{A-T} = 8.69 \text{ eV} \) and \( \epsilon_{G-C} = 8.31 \text{ eV} \). These on-site energies are taken as the average of the ionization energies of the bases that form the base pair. The corresponding ionization energies are \( \epsilon_A = 8.24 \text{ eV} \), \( \epsilon_T = 9.14 \text{ eV} \), \( \epsilon_C = 8.87 \text{ eV} \) and \( \epsilon_G = 7.75 \text{ eV} \). Each site (base pairs A-T and C-G) in this sequence has a link to the sugar molecules, see Fig.4.1. The sugar molecules attached to the phosphate groups form the backbone structure. From the base site an electron may jump either to the neighboring sites or to one of the backbone sugars. The backbone sugars are not coupled to another backbone sugar. Fig.(4.1) represents the main features of this so-called fishbone model.

The Hamiltonian of the fishbone model is written as follows [98, 99]:

\[
H_F = - \sum_{i=1}^{L} t_i |i\rangle \langle i+1| - \sum_{i=1, q=u,a}^{L} t_i^q |q_i\rangle \langle q_i| + \sum_{i,q} \epsilon_i |i\rangle \langle i| + \epsilon_i^q |q_i\rangle \langle q_i| + h.c.
\]

Here \( |i\rangle \) and \( |q_i\rangle \) are the wave functions at the base and backbone site respectively, \( t_i \) is the hopping parameter between base sites \( i \) and \( i+1 \), \( t_i^q \) is the hopping between the upper, \( q = u \) (lower, \( q = l \)) backbone site and the base site, and \( \epsilon_i \) and \( \epsilon_i^q \) are the on-site energies of the bases and the backbones, respectively. In what follows I will consider the case of the diagonal disorder when the hopping parameters \( t_i = t \) and \( t_i^u = t_i^l = t_b \) are coordinate-independent constants. The role of the off-diagonal disorder was considered in Zhang and Ulloa’s paper [100]. It is convenient to chose \( t \) as a unit of energy, so as all the energies become dimensionless. The randomness in the structure of the DNA molecules is due to the weak fluctuations of the on-site energies, \( \epsilon_i = \epsilon_0 + \delta \epsilon_i \) and \( \epsilon_i^{u,l} = \epsilon_0 + \delta \epsilon_i^{u,l} \), where \( \langle \delta \epsilon_i \rangle^2, \langle \delta \epsilon_i^{u,l} \rangle^2 \ll 1 \). After these simplifications the Schrödinger equation corresponding to Hamiltonian (4.1) can be written in the tight-binding model, similar to Eq.(2.10),

\[
\psi_{i+1} + \psi_{i-1} = (\epsilon_i - E) \psi_i.
\]
Figure 4.1. The depiction Fishbone model for DNA is shown above. The base pairs and backbones have disordered on-site energies while the hopping parameters $t$ and $t_b$ are constant. The base pairs are either A-T or G-C, with energies $\varepsilon_{A-T} = 8.69$ eV and $\varepsilon_{G-C} = 8.31$ eV respectively.

where in place of the on-site energy $\varepsilon_i$, we put $\zeta_i$ [99],

$$\zeta_i = \delta \varepsilon_i + \frac{t_b^2}{(E - \varepsilon_b)^2} \{ \delta \varepsilon_i^u + \delta \varepsilon_i^d \},$$

and the eigenenergy $E$ is replaced by

$$\tilde{E} = E - \varepsilon_0 - \frac{2 t_b^2}{E - \varepsilon_b}.$$  

The effective energy $\tilde{E}$, being the eigenenergy of the tight-binding model Eq.(4.2), lies within the interval $-2 < \tilde{E} < 2$. The dispersion relation for $\tilde{E}$ has a standard form, $\tilde{E} = 2 \cos \mu$.

The dispersion relation for the electron energy $E$ is obtained from Eq.(4.4),

$$E_z(\mu) = \frac{\varepsilon_0 + \varepsilon_b}{2} + \cos \mu \pm \frac{1}{2} \sqrt{(\varepsilon_b + \varepsilon_0 + 2 \cos \mu)^2 - 4 \left[ \varepsilon_b (\varepsilon_0 + 2 \cos \mu) - 2 t_b^2 \right]}.$$  

In the fishbone model the allowed energies form two equivalent conduction bands [98], thus modeling semiconductor behavior of DNA observed in the experiment [13].
Eq.(4.3) is a result of linear expansion over weak fluctuations, therefore both terms are supposed to be small. Because of the singularity in the second term, a narrow interval of energies close to $E = \varepsilon_b$ is excluded from the consideration.

The genetic information is coded in the sequence of potentials $\varepsilon_i$, therefore the fluctuations $\delta \varepsilon_i$ are correlated. Unlike this, the fluctuations of the potentials in the two backbone sequences do not carry genetic information, therefore they are considered to be uncorrelated and statistically independent. Thus, the correlations that exist in the sequence of the random numbers $\zeta_i$ are due to the correlations between the base pairs only,

$$
\langle \zeta_i \zeta_k \rangle = \zeta_0^2 \xi(i-k) + \sigma^2 \frac{t_b^4}{(E - \varepsilon_b)^2} \delta_{ik}.
$$

Here $\zeta_0^2 = \langle \delta \varepsilon_i^2 \rangle$, $\sigma^2 = \langle (\delta \varepsilon_i) \rangle^2 + \langle (\delta \varepsilon_i^d) \rangle^2$, and $\xi(k)$ is the normalized ($\xi(0) = 1$) binary correlation function of the base sequence.

After the calculation of the binary correlation functions, I employed the findings of Izrailev and Krokhin [18] that relates the long-range binary correlation function to the inverse localization length, given in Eq.(2.12). In the next section, I will summarize the numerical calculations on localization lengths of some real human DNA molecules.
Figure 4.3. Binary correlation function of the sequence of nucleotides of Human BRCA gene exon region 11 is given above. The correlation function drops from the value of 1 at $k = 0$ to $\xi \approx 0.1$ at $k \geq 1$, left insert. Correlations extend to distances of a few thousands of base pairs, decaying very slowly. An important feature of this correlation function is close to regular oscillations about zero, right insert.

4.2. Numerical Calculations

The numerical calculations in this thesis are based on the information about DNA sequences available from the GenBank database [101]. I analyzed a number of DNA’s and found a few of them possessing a mobility edge.

To calculate the correlation function $\xi(k)$, a sequence of nucleotides was mapped into a binary one with energies $\pm(\epsilon_{A-T} - \epsilon_{G-C})/2t$. The value of $t = 0.37eV$ [99] and the average backbone energy is taken equal to $\epsilon_0$ since the backbones and the nucleotides in the base sequence are chemically connected. The correlation functions for two different DNA segments are shown in Figs. 4.3 and 4.4. Both correlation functions oscillates with small but slowly decaying amplitude. These oscillations turn out to be the source of the sharp
Figure 4.4. Binary correlation function of the sequence of nucleotides of 51 kbp (kilo base pairs) region of Human Chromosome 22 is graphed above. The region includes both exon and intron regions. The correlation function exhibits behavior similar to that shown in Fig. 4.3 but with oscillations about a small positive value.

metal-insulator transition predicted in Izrailev and Krokhin’s paper [18] for the correlation function Eq.(2.14), which oscillates with power-decaying amplitude.

In the numerical calculations the value of $k$ in Eq.(4.6) does not run to infinity but is cut at the value of $k_{\text{max}} = 400$. This provides good convergence of the Fourier series and an accuracy of $< 1\%$ for the localization length.

The localization length, corresponding to the correlation functions in figures Fig.4.3 and Fig.4.4 is plotted in figures Fig.4.5 and Fig.4.6. Only the region of high-energy band (see Fig.4.2) is shown, since the dependence $l(E)$ in the low-energy band is exactly the same. In the both cases the major part of the allowed energies is occupied by the states localized at the distances of 30-70 base pairs. At these energies DNA macromolecule of the length of $\sim 10^3$ behaves like an insulator. However, there are sharp peaks near $E = 10.2$ eV. At the peak regions the localization length is orders of magnitude larger. My estimates showed that
Figure 4.5. Localization length vs Energy for Human BRCA gene exon region

it can be as long as $10^4 - 10^5$ base pairs. In the experiments usually much shorter segments are examined, which, thus, may exhibit metallic behavior within the peak regions of energies. In Fig.4.5 and Fig.4.6 the peak regions are shown in the inserts. However, the localization length there remains finite, very sharp increase of the localization length should be considered as a mobility edge. Within the peak region the states with localization length $l(E) \sim 10^5$ are practically delocalized. The region of the delocalized states occupies a narrow interval of energies $\sim 20$ meV.

For the both DNA's, the presence of the sharp mobility edges is due to the oscillatory behavior of the correlation function. The long-range correlations by themselves do not necessary lead to the metal-insulator transition. The sharp, step-like transition requires the correlation function given by Eq.(2.14). Any deviation from this specific form leads to the "softening" of the mobility edge and to its disappearance.

4.3. Two-Chain Model of DNA

The Fishbone model of DNA considers only a single-channel for electron propagation. Electron hops from one base pair to another. A better approximation requires taking into account the details of the base pairs. The two bases forming a pair have different ionization
Figure 4.6. Localization length vs Energy for a part of Human Chromosome 22 DNA graphed above was calculated using Fishbone model.

energies and contribute to electron hopping separately. The electron may jump from one base to the other base in the base pair or to a neighboring base on the same strand. The statistics of the chain changes with respect to Fishbone model, because we have four distinct values of on-site energies instead of a binary one.

We model the DNA molecule as a one-dimensional two-chain disordered wire, in exact accord with the wires explained in Chapter 3. The strands of DNA are now called chains and the sites on the chain represent the nucleotides along the strand. The Schrödinger equation for a DNA molecule is written in form

\[
\begin{align*}
t(\phi_{1,n+1} + \phi_{1,n-1}) + h\phi_{2,n} &= (E - \epsilon_{1,n})\phi_{1,n}, \\
t(\phi_{2,n+1} + \phi_{2,n-1}) + h\phi_{1,n} &= (E - \epsilon_{2,n})\phi_{2,n},
\end{align*}
\]

where \( t = 0.37 \) eV and \( h = 1.0 \) eV \([99]\) are the intra-chain and inter-chain hopping parameters, respectively. The on-site energies \( \epsilon_{i,n} \) are one of the four ionization energies of the nucleotides (once again, \( \epsilon_A = 8.24 \) eV, \( \epsilon_T = 9.14 \) eV, \( \epsilon_C = 8.87 \) eV and \( \epsilon_G = 7.75 \) eV). The bases A and T (C and G) are always found opposite of each other, A on one chain and always T on the other one (same for C and G).
We employed the results of Chapter 3 (namely, Eq.(3.51) and Eq.(3.62)) to calculate the inverse localization length numerically. We take $E = 0$ point equal to 8.5 eV, average of the ionization energies of nucleotides. With that, the new on-site energies were $\epsilon_A = -0.26$ eV, $\epsilon_T = 0.64$ eV, $\epsilon_C = 0.37$ eV and $\epsilon_G = -0.75$ eV. Similar to the calculations in Fishbone model, I introduced a cut-off value for $k$ when we sum the $\xi_i(k)$ terms. The summations were run up to $k < 400$.

First, I compared the Fishbone and two-channel model results for BRCA-exon 11 region and a segment of Human Chromosome 22 that includes both intron and exon regions. The values of energy in the two calculations are not comparable, since the average energy $E = 0$ point is not same for the two different models. In addition, the two-channel model results (Fig.4.7 and Fig.4.8) include regions of energy where only one-channel is propagating and the other channel is evanescent. I did not compare the two models as to where the delocalized states occur in the energy spectrum, but whether the delocalized states exist in both models for different DNA regions. I found that exon region 11 of BRCA gene exhibits delocalized electronic states in both of our calculations (Figs.4.5 and 4.7). On the other hand, the studied segment of Chromosome 22 has no delocalized electronic states in the two-channel model (Figs.4.6 and 4.8). Let me point to the difference in the on-site energy distributions (binary disorder for Fishbone model, four valued disorder for two-channel model) as the main reason behind the discrepancies between the two models. However it is not possible to conclusively remark on why Chromosome 22 has only localized states by just investigating the correlators. Following the discussion in Chapter 3, it can be said that oscillating and slow decaying correlators of on-site energies are a necessary but not sufficient condition for delocalized electron states in DNA.

4.4. Gene Expression and Quantum Electron Transport

I aimed to establish a link between the gene expression and the electron localization length for a DNA molecule. The sequence of nucleotides determine the localization lengths of electronic states of a given region. I asked myself whether a link between the functionality of a segment of DNA and its electronic properties exists.
Figure 4.7. Localization length vs energy for BRCA gene exon region 11 is shown.

Figure 4.8. Localization length vs energy for a segment of human Chromosome 22 DNA is shown. The segment includes both exon and intron regions.

In order to get an answer, I investigated the exon and intron regions of various genes for their electron localization properties. The most evident difference in their functionality
Figure 4.9. Localization length vs energy for human SNAP29 gene exon region 5 is shown.

is their role in protein coding. Two among the many DNA molecules we studied, human SNAP29 and SYLB1 genes, are discussed below.

Fig. 4.9 is the graph of localization length vs energy for SNAP29 exon region 5. The peaks ($I(E) > 200$) at various energies represent the states with localization length exceeding the length of the region significantly. Those states are practically delocalized. Just like BRCA-exon 11 and SYLB1-exon 7 regions, SNAP29-exon 5 region has a large number of states that are delocalized. On the other hand, SNAP29-intron 2 and SYLB1- intron 2 regions and the whole genes do not have any delocalized states. This is indeed the general trend for all the DNA molecules I studied.

For the gene sequences, I have found a general rule of localization that apply:

- All exon regions have a number of delocalized states.
- All states in an intron region are localized.
- All states in the whole gene sequence are localized.

The findings differentiate exon and intron regions in existence of delocalized electronic states. The non-existence of delocalized states for the whole (exon + intron) regions is a
Figure 4.10. Localization length vs energy for human SNAP29 gene intron region 2 is shown.

Figure 4.11. Localization length vs energy for human SNAP29 gene whole sequence is shown.

consistency check since if the states in the intron regions are localized, they will localize the states in the whole segment. This is a novel and striking result. It clearly shows that
whether a DNA region has or doesn’t have delocalized electronic states may play a role in its functionality, or vice versa. If we consider all the processes that take place during the protein coding, it makes sense that electronic structure and an important aspect of it, the localization length, has a determining role.

There are no distinct differences between the correlation functions of exon and intron regions, so we do not have a direct means of understanding localization-delocalization from the behavior of the correlation functions alone. Fig.4.15 and Fig.4.16 shows the correlation function (only $\xi_1(k)$ is given in figures) for SNAP29 Exon5 and Intron 2 regions, respectively.
Figure 4.13. Localization length vs energy for human SYLB1 gene intron region 2 is shown.

Figure 4.14. Localization length vs energy for human SYLB1 gene whole sequence is shown.
Figure 4.15. Correlation function $\xi_1(k)$ for SNAP29 gene exon region 5 is shown.

Figure 4.16. Correlation function $\xi_1(k)$ for SNAP29 gene intron region 2 is shown.
CHAPTER 5

CONCLUSIONS

This thesis is a study of Anderson localization of electrons in one-dimensional two-channel wires and in DNA molecules. It involves an analytical calculation part where we derive the formula for the inverse localization length for electron states in a two-channel wire. It also involves a computational part where the localization length is calculated for some DNA molecules.

DNA is a one-dimensional wire. Other examples of a one-dimensional wire are carbon nanotubes and wires etched on semiconducting films deposited on another semiconducting device. Study of the localization of electronic states in multichannel wires is necessary to understand the electron transport properties of 1-D wires.

This study of the localization length vs energy relationships in two-channel wires is the first step in the generalization towards multichannel wires. Indeed, motivated with a similar goal, Heinrichs [85] has found the formula for the inverse localization length vs energy for two-channel and three-channel disordered wires with uncorrelated weak disorder. I generalized the formula for two-channel wires with correlated weak disorder [103].

One technologically interesting application of the results of this thesis has to do with carbon nanotubes. Nanotubes are graphene layers wrapped into a cylinder. The diameter and chirality of nanotubes determine whether they are metallic or semiconducting [105]. In practice most nanotubes are quasi-1D wires with a few number of conducting channels.

The disorder in nanotubes can be of almost any form, depending on the production, deposition/doping or the environment. Most molecules and atoms, including metal atoms/structures attach to nanotubes without altering its electronic state structure significantly [104]. In those cases, nanotubes are weakly disordered systems. I believe this study in localization will be important for various technological applications of nanotubes.
Yet another important field of application is the DNA molecules. My work on electron localization in single-channel Fishbone model for DNA molecules show that the long-range sequential correlations in DNA may in fact lead to electron delocalization [102].

My study in localization of electron states in DNA as a two-channel wire has very striking and far reaching results. First of all, our study shows that DNA molecules can have localized or delocalized electron states depending on their genetic sequence information. Second and more importantly, the calculations give a link between the functionality of DNA regions and their electron localization properties. The general rule of localization itemized in Chapter 4 states that all electron states in intron regions are localized and all exon regions have a number of delocalized states.

At the moment, I do not make any claims as to how or why such a difference in localization of electronic states exists between intron and exon regions. I simply point out at the statistical information available to the scientific community. The vast information on sequences of genes of various organisms can be very quickly analyzed by just looking at the correlations in the sequence disorder. It still remains a laborious task to analyze the sequences in relation to each other, going step by step from chromosome to chromosome, organism to organism, but we propose a possible starting point.

Long sequences of DNA may be mapped according to their electron localization properties using a sliding window technique. The segment of DNA inside the window will be characterized by its electron localization length that can be easily calculated from the correlation function for the sequence of the segment. This will create a sort of chart that can be used as reference or as a fingerprint. The chart will point out to regions of certain electronic properties that may be desirable for various applications.

Another important point of investigation for near future is to see whether small changes in the sequence leads to any change in the localization length. Cancerous DNA segments can be cross-checked with healthy DNA to see if exon regions of cancerous genes show any discrepancy from the general rule of localization.
Finally, I believe that the studies parallel to this work for DNA segments may give some information about how various regions migrate from generation to generation and what role some parts of DNA may have played in the process of evolution.
REFERENCES


