Nanometer Scale Lindenmayer Systems

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ABSTRACT

Lindenmayer systems or L-systems are a mathematical model of plant growth and cell differentiation proposed by the late biologist Aristid Lindenmayer. L-systems can generate photorealistic computer graphics models of trees, flowers, and other plants as well as other complex structures. An L-system is a set of rewriting rules such as A ➔ Aa and A ➔ a that replace the left hand side of the rule with the right hand side. The rewriting rules are applied repeatedly until only the terminal symbols, such as (a) in the example, that only appear on the right hand sides of the rules remain. A nanometer scale L-system is a set of simple nanomachines that replace target modules with a payload of replacement modules. At nanometer scales, the nanomachines may be able to use thermal diffusion for transport and complementary shapes and chemical affinities, molecular recognition, for targeting. Internal power sources, propulsion systems, guidance, navigation, and control systems may not be needed. Each nanomachine in a nanometer scale L-system is only a few nanometers in cross-section and probably much easier to fabricate than a complex nanorobot. Their small size may enable them to non-destructively enter fragile structure such as cells. Nanometer scale L-systems may be able to assemble complex structures and complex systems of interacting components. The theory of nanometer scale L-systems is presented as well as a discussion of experimental tests and potential practical applications such as the programmed repair of chromosomal and genetic damage in cancer cells.

1. INTRODUCTION

The dream of nanomedicine, nanotechnology applied to medicine, is to cure or control major incurable diseases such as cancer and heart disease[1,2]. These two diseases each cost at least 500,000 lives per year and offer markets of at least $100 billion per year in the United States alone. Unfortunately, a large gap separates present biotechnology and nanotechnology from the sophisticated nanorobots often proposed to address these diseases. It will probably take several decades and tens or hundreds of billions of dollars to fabricate the medical nanorobots[3]. In addition medical nanorobots will probably be bulky, measuring microns in diameter, making safe introduction into the interior and nucleus of cells difficult. Thus a mechanism to perform complex computations, carry out complex modifications of biological structures such as chromosomes, and to assemble large, complex structures at targets inside the cell from nanometer-scale constituents such as proteins or retroviruses that can be more easily introduced into the cell may be needed for effective nanomedicine. Nanometer scale Lindenmayer systems may offer an alternative to integrated nanorobots that can be implemented using present or near-future (next five years) nanotechnologies such as genetic and protein engineering methods, carbon nanotubes, organic dendrimers, nanocrystals, or some combination of these methods.

2. MOLECULAR BUILDING BLOCKS AND LINDENMAYER SYSTEMS

A mechanism to make systems of mechanical parts is the assembly of mechanical systems from standardized simpler matching parts. Extremely complex systems can be constructed from a relatively small number of basic building blocks. The popular Lego toy is an example of this mechanism. Builders and engineers use the same method. Nanoscale building blocks can be small and easily inserted into a cell or the nucleus of a cell. In the absence of nanorobots, some mechanism to assemble the building blocks into systems inside the cell or nucleus is needed. Mobile genetic elements that carry a genetic

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payload and that copy or insert themselves or the payload alone in place of or next to a specific target DNA sequence can assemble or modify complex genetic systems within living organisms. The genetic payload must consist of modular building blocks of genes such as the exons found in eukaryotes, regulatory binding sites, or the DNA sequences targeted by the mobile genetic elements[4].

Targeted mobile genetic elements that carry genetic payloads can implement Lindenmayer-systems or L-systems, a mathematical model of plant growth and cell differentiation proposed by the late biologist Aristid Lindenmayer[5]. The central concept of L-systems is that of rewriting. In general, rewriting is a technique for defining complex objects by successively replacing parts of a simple initial object using a set of rewriting rules or productions[6]. In the context of nanometer scale L-systems, it is more intuitive to refer to the rewriting rules as *replacement rules*. The nomenclature *replacement rule* will be used below in this paper. An L-system is a collection of replacement rules that replace a symbol or group of symbols with a different, usually more complex set of symbols.

A simple example illustrates how a system of targeted mobile genetic elements with genetic payloads can build complex networks of genes. The system consists of a target DNA sequence identified by the symbol α for a network or sub-network of interacting genes, a target DNA sequence identified by the symbol *I* for interface, an exon *P* coding for a folding domain P, a regulatory binding site *Q* recognized by the folding domain P, an exon *R* coding for a folding domain R, and a regulatory binding site *S* recognized by the domain coded by *R*. A semicolon represents a stop codon separating two genes. Each mobile genetic element implements a single replacement rule in the L-system. The domains implement a linear interface system similar to a lock and key. For example, the protein PPR, consisting of the domains P, P, and R in a linear chain, binds only to the matching regulatory binding sites QQS (Figure 1). This linear modular system may be similar to the regulatory proteins made of modular zinc finger motifs arranged in tandem and their associated regulatory binding sites[7].

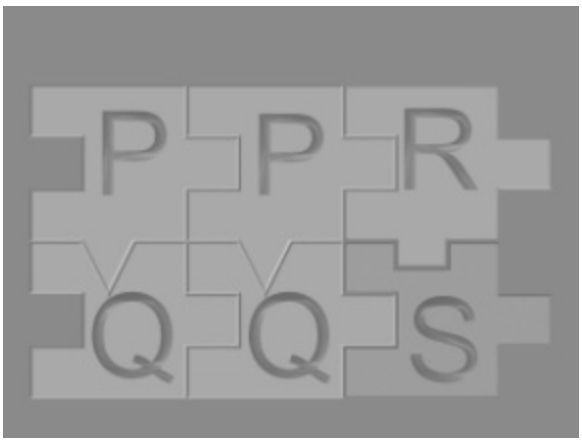


Figure 1 Modular Three Domain Proteins PPR and DNA Regulatory Binding Sites QQS Fit Together

The targeted mobile genetic elements with genetic payloads can be represented symbolically as replacement rules in an L-system. This is a stochastic L-system in which multiple replacement rules have the same left hand side – *I* and α in the example. Which rule is used is random or stochastic. Amongst other advantages, this permits different target sequences *I* to represent distinct and unique interfaces in the network.

$\alpha \rightarrow \alpha I$ $\qquad\qquad$ $I \rightarrow RIS$

$a \rightarrow I$ $I \rightarrow P;Q$

$I \rightarrow PIQ$ $I \rightarrow R;S$

Within the genome, a targeted mobile genetic element with a genetic payload might look something like:

(DNA sequence for a transposase protein that cuts or copies DNA delimited by the transposition start and stop codes and splices at the target DNA sequence such as I) (Transposition start code) (genetic payload of exons, regulatory binding sites and DNA target sequences such as PIQ)(transposition stop code)

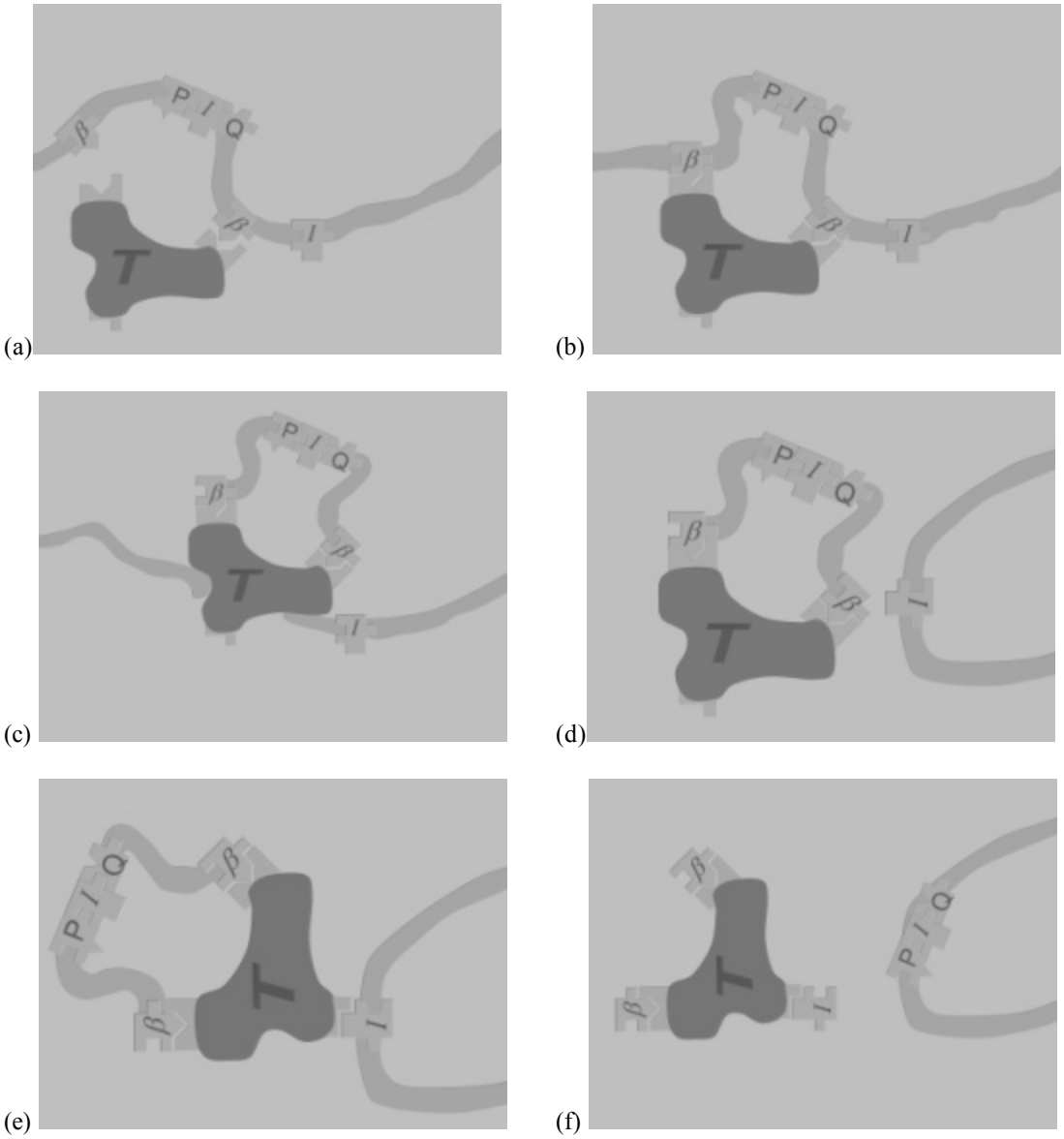
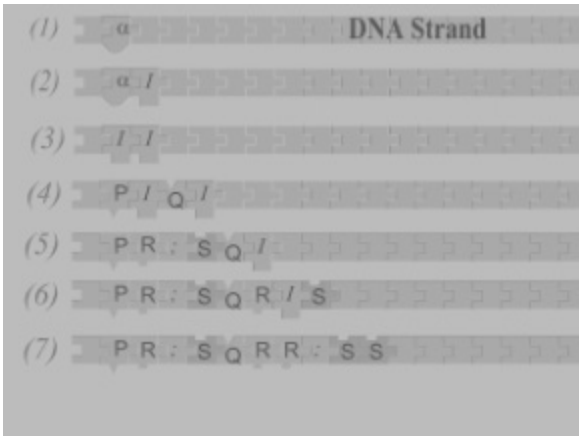


Figure 2 A Transposase T Cuts DNA Segment *PIQ* and Splices at *I* ($I \rightarrow PIQ$)

Figure 2 illustrates a transposase T, a special protein that cuts and splices a DNA segment, cutting the segment *PIQ* and splicing the segment in place of the target DNA sequence *I.* When any of the mobile genetic elements that targets the DNA sequence *I* in the genome encounters the target DNA sequence *I*, it will substitute its genetic payload, represented by the right hand side of the replacement rule, into the genome. The process continues until the last target DNA sequence *I* is consumed. The recursive nature of the replacement rule, the mobile genetic element, where *I* appears on both the left and right hand side of the rule enables the rule to build arbitrarily complex systems.



Step	Rule
(1) → (2)	α → α*I*
(2) → (3)	α → *I*
(3) → (4)	*I* ®*PIQ*
(4) → (5)	*I* ®*R;S*
(5) → (6)	*I* ®*RIS*
(6) → (7)	*I* ®*R;S*

Figure 3 Repeated Substitutions by Targeted Mobile Genetic Elements Build a Network of Genes

Figure 3 illustrates the mobile genetic elements expanding an initial target DNA sequence α into a network of three genes. In step one, the mobile genetic element that implements α → αI replaces α with αI. In step two, the mobile genetic element that implements α → *I* replaces α*I* with *II* in the DNA strand. The process continues until the network of three genes is constructed. This simple example nanometer scale L-system can build larger networks of hundreds or thousands of genes. More sophisticated nanometer scale L-systems can build networks with branches, feedback loops, and other complex structures.

Repeated insertions or substitutions of segments of genes such as the exons and regulatory binding sites by targeted mobile genetic elements with genetic payloads can build complex networks of structural proteins, regulatory proteins, and regulatory binding sites such as the signal transduction networks that include the oncogenes and tumor suppressor genes that are often mutated in cancer[8]. A mutation or activation of these hypothetical naturally occurring mobile genetic elements would restructure the network extensively, changing many genes simultaneously in a coordinated fashion and probably causing substantial chromosomal rearrangements as the mobile genetic elements cut and splice large sections of DNA. This provides a possible mechanism for saltatory evolution[9]. While the view that this actually happens in living organisms is not generally accepted, it is suggested in the work of the late Barbara McClintock, James Shapiro, and some other researchers into mobile genetic elements[10,11,12,13,14,15,16].

In general, a nanometer scale L-system is an L-system implemented at the nanometer scale where the symbols or alphabet of the L-system are nanometer scale supramolecular structures or molecular building blocks such as folding domains in proteins, DNA regulatory binding sites, or segments of genes. The replacement rules of the L-system such as *I* → *PIQ* that replaces the symbol *I* with *PIQ* are implemented with other molecules or nanometer scale supramolecular structures such as the transposases in the mobile genetic element example. The entire L-system is a system of interacting molecules or supramolecular structures. A nanometer scale L-system may be a system of nanocrystals, organic dendrimers, diamondoid structures as suggested by Eric Drexler[17], carbon nanostructures, proteins, or other constituents.

Each replacement rule of a nanometer scale L-system is implemented by a relatively simple molecule or supramolecular structure such as a nanocrystal, organic dendrimer, transposon as in the example, or retrovirus, ranging from 5 to 120 nm in diameter. The small size of the molecule or supramolecular structure that implements each replacement rule makes it relatively easy to introduce the nanometer scale L-system into cells and even into the nucleus of the cells. Nanometer scale L-systems can then assemble complex structures, execute complex programs, or carry out complex modifications of other structures such as chromosomes, all without bulky and difficult to fabricate nanorobots.

2.1 Transport mechanisms for nanometer scale L-systems

Unlike hypothetical nanorobots, nanometer scale L-systems probably cannot use internal propulsion systems such as motors and propellers or sophisticated guidance, navigation, and control systems. Like naturally occurring mobile genetic elements and simple cellular components such as proteins, the nanometer scale L-systems must rely on other mechanisms to be transported throughout a cell or other region of interest.

Thermal diffusion within cells or other regions of interest may offer the simplest transport mechanism. Thermal diffusion avoids the need for sophisticated propulsion systems altogether. Naturally occurring convection currents within cells or other regions of interest may offer another transport mechanism.

2.2 Targeting mechanisms for nanometer scale L-systems

Nanometer scale L-systems depend on the ability to target specific target modules such as regulatory binding sites or other target sequences in the DNA to function. In the simple illustrative examples in this paper, a simple targeting mechanism is presented. A transposase molecule is generally thought to be a dimer comprised of two identical sub-units. These sub-units dimerize to form a roughly triangular transposase with three prongs. Two prongs perform the cutting operation at the transposition start and stop codes. The third prong is the targeting prong. This prong presumably contains a pair of protein α helices that target the insertion or substitution target sequence represented by the symbol α in the example.

Other targeting mechanisms may be possible. For example, the targeting prong of the transposase may be a generic targeting prong that fits into the main groove of the DNA strand without targeting a specific DNA sequence. A regulatory binding protein assists the transposition and insertion process. The regulatory binding protein binds, for example, to a sequence of four regulatory binding sites.

*(target sequence **b**)(target sequence **c**) (target sequence **d**)(target sequence **e**)*

If the target sequences β and χ are carried by the payload DNA and the target sequences δ and ε are found in the target DNA strand, then the regulatory binding protein may be able to direct the insertion of the payload DNA next to or in place of the sequences δ and ε in the target DNA strand.

From the standpoint of living organisms transposition directed by regulatory proteins has many advantages. The genome need only code for a small number of generic transposases. Transposition is under the control of the sophisticated signal transduction networks within the cell. New target sequences can be assembled by transposition and shuffling of the regulatory binding sites.

3. MEDICAL APPLICATIONS OF NANOMETER SCALE L-SYSTEMS

Nanometer scale L-systems have many potential applications in medicine, biology, and other areas of nanotechnology. The possible cure and treatment of cancer using nanometer scale L-systems implemented with naturally occurring or artificial targeted mobile genetic elements with genetic payloads appears to be one of the most important potential applications. Cancer is currently attributed to the accumulation of mutations of proto-oncogenes and tumor suppressor genes within signal transduction networks in a cell. Typically around a dozen mutations of different genes are needed to transform a normal cell into a cancerous cell. Unfortunately, many different sets of mutated oncogenes correspond to the same cancer. Thus, two tumors from two different patients that appear clinically the same may have completely different sets of mutated oncogenes. Cancer is now thought to consist of

over four hundred different types of cancer, each with a different set of mutated genes. This has made any treatment for cancer effective against all or most types of cancer difficult to envision.

A nanometer scale L-system implemented using targeted mobile genetic elements with genetic payloads may offer a solution to this conundrum. The anti-cancer nanometer scale L-system contains a representation of the healthy, undamaged signal transduction network. In healthy, non-cancerous cells it recreates the healthy network. The cells continue to be healthy. In cancerous cells, it overwrites the damaged network, converting the cancer cells to healthy cells. Ideally the cancer cells are converted to stem cells that develop into healthy tissues in the organ where the cells reside. Thus metastatic cells in the lungs or liver are converted to healthy lung or liver tissue. This is important because it is metastatic cells in the lungs and liver that are usually directly responsible for death in cancer patients. The primary tumor is rarely responsible for death and often can be successfully removed by surgery. Failing conversion to stem cells and redifferentiation into organ tissues, the benign former tumors may be surgically removed. If the benign former tumors cannot be surgically removed, some degree of disability will probably result.

3.1 Nanometer scale L-system to Repair a Genetic Network

Figures 4–7 (below) illustrate a simple nanometer scale L-system repairing a small network of three genes. Figure 3 (above) shows the final sequence of substitutions building the completed repaired network of three genes. This nanometer scale L-system is closely related to the first example and consists of the rules:

$a \rightarrow aI$	$I \rightarrow RIS$
$a \rightarrow I$	$I \rightarrow P;Q$
$I \rightarrow PIQ$	$I \rightarrow R;S$

(left side)b(anything)b(right side) ®(left side)(right side)

The additional rule is a transposase that deletes any DNA delimited by the target sequence β. The target sequence β is found in the non-coding DNA flanking the section of the chromosome that needs to be repaired. In the simple example, the section of the chromosome delimited by the target sequence β contains three genes G_1, G_2, and G_3 that may be damaged. For example, G_1, G_2, and G_3 may be constituents of a linear cascade. If G_1, G_2, or G_3 is damaged, the cascade fails and the cell may become cancerous as a result. One would like a way to treat the cancer without a separate drug, system of drugs, or nanomachine for each gene in the cascade.

The target sequences β are flanked by two target sequences α_0 and α_1. The two target sequences α_0 and α_1 concatenated together form the target sequence α recognized by the mobile genetic elements that implement the rules $\alpha \rightarrow \alpha I$ and $\alpha \rightarrow I$ as in the first example. Until the defective section of the chromosome is deleted the other rules will not begin to build the new healthy network of genes. Further, the new network will be built exactly once in the same section of the chromosome.

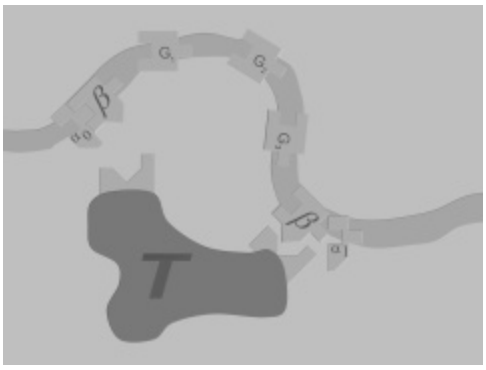


Figure 4 Section of a Chromosome with Three (3) Genes G_1, G_2, and G_3

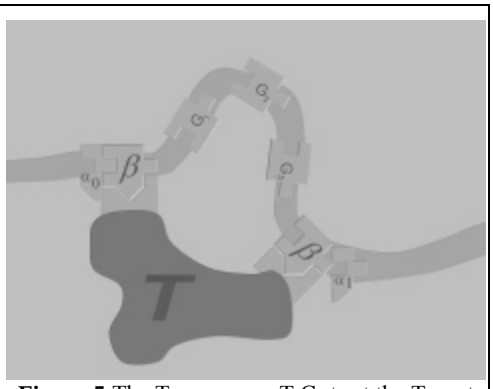


Figure 5 The Transposase T Cuts at the Target Sequences β in Non-Coding DNA Flanking the Three Genes

Figure 6 Transposase T Deletes and Discards the Section Delimited by the Target Sequences β

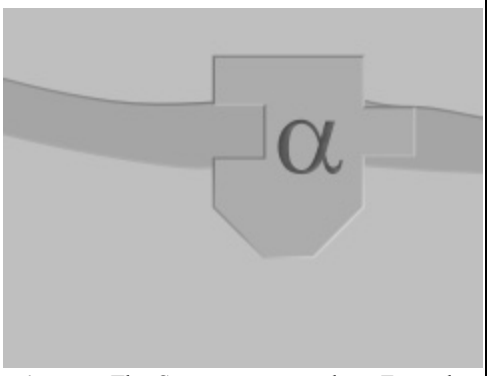


Figure 7 The Sequences α_0 and α_1 Form the Target Sequence α (See Figure 3 for Subsequent Steps)

For illustrative purposes, the example of repairing a section of a chromosome with a nanometer scale L-system uses a stochastic L-system that will not consistently generate the genes G_1, G_2, and G_3. In an actual anti-cancer nanometer scale L-system, a deterministic L-system that consistently generates the genes G_1, G_2, and G_3 would probably be used.

3.2 Nanometer scale L-systems to Generate Repetitive DNA Including Telomeres

The bulk of DNA in the human genome, roughly 97 %, is non-coding DNA. According to generally accepted theory, most of the non-coding DNA is "junk" or selfish DNA that performs no useful purpose[18,19]. In this case the anti-cancer nanometer scale L-system need not rebuild the non-coding DNA within a section of a chromosome that it deletes. For example, if a nanometer scale L-system deletes a one-million (1,000,000) base pair section of a chromosome, it need only contain the information to rebuild about thirty-thousand (30,000) base pairs of coding DNA.

A retrovirus can carry about ten thousand (10,000) base pairs into the nucleus of the cell in a package about 120 nm in diameter. Cellular transport vesicles are similar in size to retroviruses. They may be harnessed or modified to carry a payload of transposons into the nucleus of the cell. If retroviruses can perform targeted insertion into the DNA or can be adapted to perform targeted insertion into the DNA, it requires approximately one-hundred different targeted retroviruses to repair a one-million base pair stretch of a chromosome. In contrast only three targeted retroviruses are needed to replace the thirty thousand (30,000) base pairs of coding DNA.

The author is skeptical of the generally accepted theory that most of the non-coding DNA performs no useful purpose. Further, the highly repetitive DNA sequences that comprise much of the non-coding DNA are known to have an effect on the coding genes. Highly repetitive DNA sequences tend to form chromatin, a complex of DNA and protein, and inhibit the expression of adjacent genes. This causes the so-called "position" effect[20,21,22]. This presents the concern that the nanometer scale L-system needed to repair a cancer cell will be extremely large – for example, hundreds, thousands, or even tens of thousands of distinct targeted retroviruses. The non-coding DNA has many long stretches of repeated DNA sequences. The draft human genome map shows that at least 35 % of the human genome is comprised of non-coding repetitive DNA sequences[23,24]. Briefly, Alu repeats comprise 288 million base pairs (9.9 %). Mammalian interspersed repeats (MIR) comprise 66 million base pairs (2.3 %). Medium reiteration (MER) repeats comprise 50 million base pairs (1.7 %). Long terminal repeats (LTR) comprise 155 million base pairs (5.3%). Long interspersed nucleotide elements (LINE) comprise 466 million base pairs (16.1 %).

Well known examples of repetitive DNA are the tandem repeats comprising the telomeres and the centromeres of chromosomes. The centromeres and telomeres are essential for proper functioning of chromosomes. The telomeres at the ends of the chromosome shorten each time a healthy cell divides. They may provide a clock that controls cellular aging and cause healthy cells to stop dividing after a fixed number of cell divisions. In cancer cells and immortal cell lines the telomeres are improperly regenerated, leading to immortality. It may be necessary to rebuild the telomeres in a controlled way – for example, rebuild the telomere with a specified length corresponding to a certain number of cell divisions before the cell ceases to divide – to repair cancer cells or to reverse the natural aging process.

Highly repetitive sequences can be generated with signal processing techniques such as run-length encoding that can be implemented using appropriately designed L-systems. Thus many parts of the non-coding DNA probably can be represented using relatively simple L-systems. There will be no need to import millions of base pairs of repetitive DNA into the nucleus of the cell in bulk, uncompressed format.

The following nanometer scale L-system implemented with targeted mobile genetic elements with genetic payloads implements run-length encoding and can generate a human telomere of defined length. Run-length encoding represents a repetitive sequence as the number of repeats and the repeated symbol. For example, a sequence of seven A's (AAAAAAA) may be run-length encoded as 7A. The example run-length encoding nanometer scale L-system implements binary numbers (base two) to represent the number of repeats. Note that additional rules can be added to repeat other sequences than (TTAGGG), e.g. (AATTTG), to build other stretches of repetitive DNA such as the centromeres. The rules that implement the binary number system can be reused.

The symbols R_0, R_1, R_2,..., R_N represent sequences in the DNA targeted by the mobile genetic elements. It is likely that these will be 17–20 nucleotide base pair sequences similar to the regulatory binding sites associated with regulatory proteins. R* is a special end-of-run symbol. More efficient run-length encoding schemes may not need the end-of-run symbol. The left parenthesis and right parenthesis () in the end-of-run rule indicate that the left hand side of the rule has been deleted. The characters A, T, and G refer to the standard DNA nucleotide base pairs. (TTAGGG) is the human telomere repeat[25].

$R_1 \rightarrow R_0\ R_0$	$R_2 \rightarrow R_1\ R_1$
$R_3 \rightarrow R_2\ R_2$	$R_4 \rightarrow R_3\ R_3$
....	$R_N \rightarrow R_{N-1}\ R_{N-1}$
(TTAGGG) $R_0 \rightarrow$ (TTAGGG) (TTAGGG)	(TTAGGG)R* $\rightarrow$ ()

Table 1 Nanometer scale L-system to Build Telomere with Run-Length Encoding

As a simple illustrative example, a telomere consisting of thirty-three (33) repeats of the human telomere repeat nucleotide base pair sequence (TTAGGG) can be generated by the initial sequence (TTAGGG) R_5 $R_0 R^*$ as follows.

Sequence in DNA	**Rule applied to make the sequence**
(TTAGGG) R_5 R_0 R^*	(start sequence)
(TTAGGG) R_4 $R_4 R_0$ R^*	$R_5 \rightarrow R_4\ R_4$
(TTAGGG) R_3 R_3 R_3 $R_3 R_0$ R^*	$R_4 \rightarrow R_3\ R_3$
(TTAGGG) R_2 R_2 R_2 R_2 R_2 R_2 R_2 $R_2 R_0$ R^*	$R_3 \rightarrow R_2\ R_2$
(TTAGGG) R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 R_1 $R_1 R_0$ R^*	$R_2 \rightarrow R_1\ R_1$
(TTAGGG) R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 $R_0 R_0$ R^*	$R_1 \rightarrow R_0\ R_0$
(TTAGGG) (TTAGGG) R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 R_0 $R_0 R_0$ R^*	(TTAGGG) $R_0 \rightarrow$ (TTAGGG) (TTAGGG)
…(thirty-one repetitions)	(TTAGGG) $R_0 \rightarrow$ (TTAGGG) (TTAGGG)
(TTAGGG) (TTAGGG)(TTAGGG) R^*	(TTAGGG) $R_0 \rightarrow$ (TTAGGG) (TTAGGG)
(TTAGGG) (TTAGGG)	(TTAGGG)$R^* \rightarrow$ ()

Table 2 Repeated Substitutions Generate a Telomere

A telomere comprised of 1,024 repeats can be represented as (TTAGGG) $R_{10}R^*$ and so forth. This sequence is only about forty-six (46) base pairs. It can be expanded to 6×1024 base pairs by the nanometer scale L-system. Thus, repetitive DNA sequences in the non-coding DNA such as the telomeres and the sequences associated with the centromeres can be generated using simple nanometer scale L-systems. The healthy repetitive DNA can be introduced into the cell in a highly compressed form. Figure 8 illustrates a nanometer scale L-system regenerating the end of a chromosome.

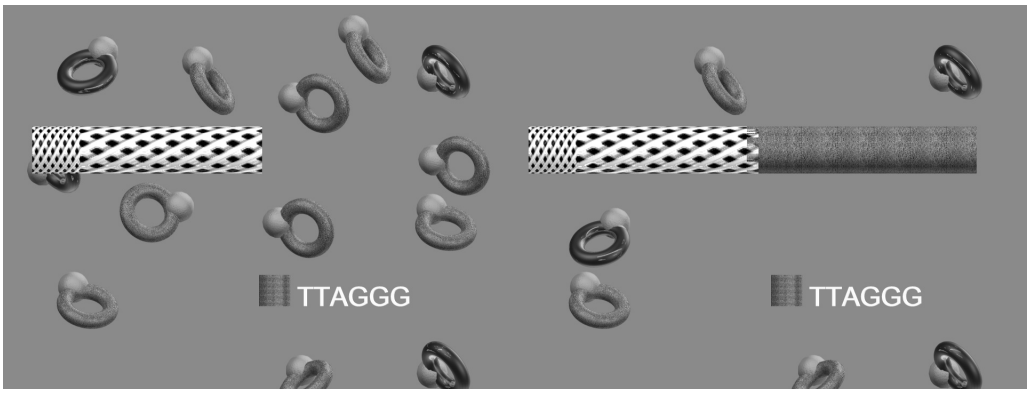


Figure 8 A Nanometer scale L-system Regenerates the End of a Chromosome (Telomere)

4. CONCLUSION

If the anti-cancer nanometer scale L-system is a complete representation of the healthy signal transduction network, it can treat any type of cancer. There is no need for a separate drug, system of drugs, or nanomachine for each type of cancer, each distinct set of mutated oncogenes. The problem then is to infer an L-system for a healthy signal transduction network, something now presumably available or partially available in the recently completed human genome map and in the literature on oncogenes and tumor suppressor genes. This is a tedious task by hand. However, in principle, computational methods for signal, image, and video compression can extract an L-system from gene sequencing data. Run-length encoding can be used for repetitive DNA as illustrated above. Fractal signal encoders can represent complex signals such as still images and video as iterated function systems closely related mathematically to L-systems[26]. The inferred L-system can then be used to direct the automated synthesis of the mobile genetic elements comprising the anti-cancer nanometer scale L-system.

Practical implementation of nanometer scale L-systems is a challenging task. However, nanometer scale L-systems may be implemented by harnessing naturally occurring targeted mobile genetic elements or by adding targeting to naturally occurring untargeted mobile genetic elements. Mobile genetic elements such as insertion sequences, transposons, retrotransposons, and retroviruses are known, naturally occurring biological phenomena that comprise a large fraction of the DNA of human beings and many other life forms. The harnessing or modification of naturally occuring mobile genetic elements to construct nanometer scale L-systems may be much closer to practical realization than complex nanorobots. Further, nanocrystals, organic dendrimers, carbon nanostructures and other objects of current nanotechnology research can probably be adapted to implement nanometer scale L-systems not derived from natural mobile genetic elements. Artifical mobile genetic elements using materials such as nanocrystals, carbon nanostructures, organic dendrimers and so forth may be much closer to realization than complex integrated nanorobots and may circumvent many practical problems with the operation of bulky nanorobots in the body and especially inside cells and the nuclei of cells. The first artificial nanometer scale L-systems will almost certainly be quite simple. The combination of more complex nanometer scale L-systems using mobile genetic elements and the application of signal encoding techniques to the healthy human genome map may offer a powerful way to cure most or even all cancers with a single treatment.

ACKNOWLEDGEMENTS

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