A simple chaotic model for development of HIV virus

Fatemeh Parastesh*, a, Zeinab Aram a, Hamidreza Namazi b, Julien Clinton Sprott c, Sajad Jafari d,a

a Department of Biomedical Engineering, Amirkabir University of Technology, No. 350, Hafez Ave, Valiasr Square, Tehran 159163-4311, Iran

b School of Engineering, Monash University, Selangor, Malaysia

c Department of Physics, University of Wisconsin - Madison, Madison, WI 53706, USA

d Health Technology Research Institute, Amirkabir University of Technology, No. 350, Hafez Ave, Valiasr Square, Tehran 159163-4311, Iran

Email: f.prstsh@gmail.com

Abstract: Studying the growth of HIV virus in the human body as one of the fastest infectious viruses is very important. Using mathematical modeling can make experimental tests easier to process and evaluate. It can also help to predict the disease progress and provide a better insight into the virus development. In this study, a new nonlinear differential equation model is introduced to investigate the interaction of the HIV virus with the body immune system. This is a physiological-based model capable of representing complex behaviors. The bifurcation analysis with a variation of activated healthy T cells is carried out. It is shown that the chaotic development of the virus is available for some ranges of activated healthy T cells. This may explain why the virus develops differently in different individuals or under different circumstances. The chaotic region contains some narrow periodic windows, in which the chaotic mode suddenly ends at some critical points, and the system starts a periodic behavior for a tiny range of active healthy T cells. This may indicate the possibility of controllable development of the HIV virus even when it is in the random-like phase of the disease. For more illustration, the system's state space is represented.

Keywords: HIV virus; mathematical modeling; nonlinear dynamics; chaos; bifurcation; physiological-based model.
1. Introduction

Given the increasing number of HIV-infected patients, it is important to study how this virus develops in the human body. Due to delayed diagnosis and the lack of proper treatment, most of these patients get infected with AIDS (acquired immunodeficiency syndrome) within 5-10 years [1]. At present, researchers have recognized the growth process of AIDS and also found suitable methods for treatment. Most of these researches have focused on this issue from a theoretical perspective, via mathematical modeling and computer simulations [2, 3]. Some of these mathematical models are based on the decay characteristics of the virus in the infected body [4]. In fact, this procedure can help in extending our knowledge about controlling virus development and lead to suggesting some new therapeutic approaches. More importantly, many therapeutic methods can be easily tested on a model, and thus, their effectiveness can be examined without any side effects for the patient. Furthermore, the process of trial and error for therapeutic methods requires time, cost, and a large number of samples, which usually makes them impossible. Since therapeutic methods of coping with the HIV virus interfere with one of the virus proliferation stages, the model can reflect the efficiency of these methods if the virus proliferation stages are put together correctly. For this reason, developing models capable of representing the behaviors of the virus in the body can revolutionize the treatment of the diseases.

One of the procedures for modeling biological systems is dynamical-based modeling [5, 6]. Furthermore, chaotic behavior is known as a specific state of dynamical systems with particular and unique properties [7]. Chaotic behavior is also often a desirable phenomenon in living organisms [8]. Therefore, chaotic-based dynamical models have attracted much attention and have become important in the modeling of biological systems [9]. Generally, chaotic behavior is the justification of many unknown behaviors formerly considered random [10]. Chaos and its applications and also control of chaotic systems have been increasingly studied focusing on either chaotic oscillators individually or in a network. The discovery of chaotic behavior in biological systems by scientists like Walter Freeman [11] created a fundamental change in this context. Chaotic models offer qualitative demonstrations of the desired system rather than quantitative representations.

The HIV virus development is one of the biological mechanisms that seem to exhibit chaotic behavior. The disease caused by HIV has three main stages. In the first stage or acute infection, the patient may have a flu-like illness for a short time. This stage is called the incubation period. As the disease progresses, the more it will interfere with the body immune system and cause infections or even tumors [12]. However, this may not affect individuals whose immune system works well. Finally, the disease will reach the last stage, namely AIDS. In this stage, the number of T lymphocytes is less than 200 cells per microliter. The T lymphocytes are white blood cells mediating the immune system response, which is responsible for the elimination of the foreign invaders [13]. HIV infected individuals are also reported to be at high risk for hepatitis B virus and hepatitis C viral infections [14].

There are many studies on the modeling of the HIV virus and its development in the human body using different theoretical approaches and mathematical models. Some of these studies have used a cellular automata modeling approach [15], while others employed nonlinear differential equations to survey the interactions of the HIV virus [16]. Besides, there are also
some fractional models, due to the strength of the fractal analysis in chaotic systems [17-22]. In the cellular automata method, the virus attack process in the human body is modeled by considering several healthy and infected immune cells arranged next to each other in a two- or three-dimensional space with some neighbor laws for transmission of the disease. Although this method makes it possible to reproduce some spatial localizations and local interactions of the HIV infection, it also has some drawbacks. First, even though the virus substance is different from the immune cells, the virus itself is not included in these models. In these models, only the aggregation of the healthy and nearby infected immune cells, is considered. Second, the motion of virus and immune cells in blood vessels is not considered in such models. For instance, in these models, an infected immune cell can transfer the disease only to its neighbor cells, while the virus transference in the human body does not encounter such limitations. Actually, an infected immune cell can infect many healthy immune cells by the produced viruses moving to different locations.

A variety of dynamical models using nonlinear differential equations have been proposed to study the development of the HIV virus and potential therapeutic methods [22-26]. Bonhoeffer et al. suggested a three-dimensional differential equation model to describe the behavior of the HIV virus in the body at a rate dependent on the virus population and the T cells [27]. Ho et al. showed that the dynamics of the HIV virus model is sensitive to both parameters and initial conditions of the system [28]. In their study, the virus number is affected by the number of both CD4 lymphocytes and CD8 lymphocytes. The CD8 cells involve inhibition of viral transcription and thus are associated with a long term healthy state in the HIV infection [29]. Together, the CD8 lymphocytes play a fighting role, while the CD4 lymphocytes have only a replication role for the virus. Wang studied the interaction of HIV and T cells by differential equations in which the number of viruses and both healthy and infected T cells were taken into account [30]. Moreover, he suggested a prey-predator competitive relationship between healthy T cells and infected T cells. Lund et al. confirmed that chaotic dynamics could be developed in the model of interaction between the HIV virus and the immune system [13]. Hernandez-Vargas et al. presented a discrete-time neural observer, which is tested by application to a model for HIV infection dynamics [31]. Revilla et al. [32] and Yu et al. [33] proposed a method using the recombinant virus, which is a manipulated HIV virus engineered by genetic engineers. This recombinant virus is sent to the HIV-infected body, and then it sticks to the infected T cells and forces them to reproduce. Consequently, the proliferation of the HIV virus is prevented. This recombinant virus does not attack the healthy T cells, and its host cells are only the HIV-infected ones. In this regard, also Ref. [34] discussed the use of engineered therapeutic viruses called 'hunter' viruses to control HIV and other viral infections.

These investigations have revealed that the HIV virus population in the human body varies at random, especially in the phase of AIDs [13]. However, some researchers have confirmed that these behaviors are not random but rather are complex and chaotic [13]. In our study, we seek a proper model which is capable of representing complex behaviors while being simple. We propose three-dimensional differential equations with a special set of parameters where the system can be in the chaotic mode. Furthermore, the saturation phenomenon is included to avoid an endless increase in the population of activated healthy immune cells, infected immune cells, or the virus. We show that the proliferation rate of healthy T cells highly affects the dynamics of virus development. It is found that the healthy immune cells, in turn, determine whether the virus dynamics is periodic or chaotic. In addition, bifurcation behavior is shown
to be available in a short range of activated healthy T cells. Our model produces chaotic behavior for some ranges of activated healthy T cells while it follows a route to periodic behavior for some other ranges. Moreover, the state space for some modes of the system is displayed for more completeness.

This paper is organized into five sections. In the next section, we explain the physiological background of the HIV infection process. After that, we elucidate the mathematical model in the third section. In this part, some further definitions are given to illustrate our modeling approach. The numerical simulation and the calculation results are represented in Section 4. Finally, the discussion and the conclusion are given in Sections 5 and 6.

2. The physiological background

Using a physiologically-based model is critical for obtaining a better understanding of the mechanisms of HIV pathogenesis and for developing treatments. Therefore, in this section, we briefly mention the physiology of the HIV virus infection process. The virus replication cycle begins with the infection of a host cell and ends with the release of mature viral particles. The viruses must come into another cell nucleus since they are not able to be reproduced by themselves. That is because reproduction needs energy and a proteinization mechanism, which exists only in living cells. The host cell for the HIV virus is a vital subtype of immune cells called lymphocytes. That is why the HIV virus is one of the currently best known fatal viruses. On the other hand, the virus entry into the cell is possible only for cells that carry the virus receptor, which is particularly the CD4 molecule for the HIV virus. The lymphocytes that take this type of receptor to absorb the virus are more likely to be infected by the virus, while the other cells resist infection so that any contact with the virus will be in vain. Once the virus enters the cell, its nucleic acid activity begins. The nucleic acids of the virus contain enough genes to inhibit the metabolism of the host cell and conduct the vital chemical reactions of the host cell to proliferate the virus. After that, the produced viruses exit the cell. This can take place in two different ways. In some cases, the virus is released with the decomposition of the host cell, and a large amount of virus is suddenly released. However, for other cases including the HIV virus, the virus exits the host cell without any decomposition of the host cell. Thus, in this case, the host cell is not destroyed and continues to proliferate the HIV virus until it dies.

3. The mathematical model

In this part, we give some definitions to explain the basis of modeling the systems in which two species, materials, cells, etc. interact with one another. In such systems, the increase or decrease of the population of each group influences the population of the other one. To describe the dynamics of two interacting species, a differential equation model known as the prey-predator model has been proposed. Many researchers from different fields have used this model to explain various interactive processes including chemical reactions. The prey-predator model is described as follows [35]:

\[35\]
\[
\frac{dx}{dt} = a_1 x - a_2 xy, \\
\frac{dy}{dt} = a_3 xy - a_4 y,
\]

where \( x(t) \) and \( y(t) \) represent the population of prey and predator, respectively, over the time \( t \). Parameters \( a_1, a_2, a_3, a_4 \) are positive constants. Parameter \( a_1 \) is the natural growth rate of the prey species in the absence of the predator. Parameter \( a_2 \) denotes the effect of the predator on the prey population, which is the only factor reducing the prey population. The parameter \( a_3 \) shows the effect of the predator on the prey and is the only factor increasing the predator population. Increasing the population of a species relies on the amount of available resources. The parameter \( a_4 \) denotes the rate of natural death of the predator species. Note that the equations of Eq. 1 are not completely realistic. In fact, the endless increase of the population of the prey species in the absence of predator (for \( a_2 = 0 \)) is not realistic. Furthermore, the endless decrease of the predator population in the absence of prey (for \( a_4 = 0 \)) is not compatible with the laws of nature. Here in our study, we have tried to compensate for this deficiency in the mathematical model by adding a saturation term to the model equations.

In our proposed model, the HIV virus is the predator species. It attacks the healthy immune cells, which are the prey species so that the healthy immune cells become infected. Thus the healthy immune cell population declines. Simultaneously, the healthy immune cells also attack the viruses and try to destroy them to preserve the body health. On the other hand, as described for the prey-predator equations, decreasing the prey population also decreases the predator population. Additionally, because of the fundamental changes in the immune cells created by the virus, the infected immune cells start to produce virus. The main differences between the interactions of the HIV virus with the immune system and the interactions in the prey-predator model are in two key points:

1. In the prey-predator model, the prey is destroyed while being hunted by the predator. However, the immune cells are not eliminated but rather are infected by the HIV virus. This causes their internal structure to change in a way that helps the virus to proliferate.
2. In the prey-predator model, the predator consumes the prey and grows so that it can reproduce more. However, the host cells are responsible for the proliferation of the virus since the virus is not capable of reproduction by itself.

This relationship between the virus and the immune cell is such a circular causality because the production and survival of the virus rely on the immune cells while the immune cells are also responsible for fighting the virus.

From the above considerations, the revised model is described as follows:

\[
\frac{dx}{dt} = rx - \beta x z - dx^2, \\
\frac{dy}{dt} = \beta x z - ay - cx^2,
\]
\[ \frac{dz}{dt} = \lambda y - bz - \sigma xz - ez^2, \]

where the variable \( x \) is the population of activated healthy immune cells. The variable \( y \) denotes the population of infected immune cells. The activated healthy immune cells become infected after the virus attack. The variable \( z \) is the population of the virus. The parameter \( r \) represents the proliferation of active and healthy T cells. The population of the healthy T cells is decreased when the activated T cells become infected by HIV at a rate \( \beta xz \). The parameter \( d \) is the saturation rate of the healthy T cells. This factor limits the population of healthy T cells even in the absence of the virus. Parameters \( \alpha \) and \( c \) are the morality rate of the infected T cells and the saturation coefficient of their population, respectively. Parameter \( \lambda \) is the proliferation of the virus by the infected T cells. Parameters \( b \) and \( e \) are the constant mortality rate and the saturation rate of the virus, respectively. The virus population also is destroyed while being in a struggle with the healthy T cells at the rate \( \sigma xz \). The parameter setting for Eq. 2 are given in Table 1 where \( r \) is the bifurcation parameter. The parameters of the model have been found by excessive computer search in a way that the model exhibits chaotic behavior.

4. Equilibrium points

To calculate the equilibrium points of the model, the right hand side of Eq.2 is equated to zero. The parameters are set according to Table 1, and \( r \) is considered variable. Solving these equations gives rise to three fixed points as \( E1=(0,0,0), E2=(0,0, -5/13), E3=(25r / 7,0,0) \). Consequently, the eigenvalues of the Jacobian matrix of the model will be \( [0,-0.15,-1.29] \) for the first fixed point, \( [0.35,0.15,1.29] \) for the second fixed point, and \( [-r,-2.21r-\sqrt{4.9r^2 + 0.015r + 0.32 - 0.72},-2.21r+\sqrt{4.9r^2 + 0.015r + 0.32 - 0.72}] \) for the third fixed point. Therefore, the stability of the first fixed point is undefined, and the second fixed point is clearly unstable for any value of \( r \). With considering the variation of \( r \) in the interval \( [0.01,0.04] \), all of the eigenvalues of the third fixed point will be negative and thus the third fixed point is stable.

5. Bifurcation analysis and Lyapunov spectrum

The ability of a biological model to exhibit complex behavior is a great virtue since biological systems exhibit unquestionable complexity. Conversely, the lack of such complexity in a model implies that it is insufficiently realistic. That is to say, the model can be appropriate for modeling of the biological system only if it is capable of demonstrating complex and rich behaviors. In dynamic systems theory, chaotic dynamics is characterized as being the richest and the most elaborate dynamics. In this section, we investigate the qualitative behavior of the model through its bifurcation diagram and state space plot. The initial condition for the variables in the numerical calculations are \( (x,y,z) = (-0.6,-0.66,1.03) \). The model is intended primarily to describe the qualitative characteristics of the real system and changes in its dynamical behavior rather than to make detailed quantitative predictions. However, the model
closely replicates the behavior reported in real systems including nonlinear behavior of the HIV virus.

There have been many proposed differential equation models for HIV virus behavior in the human body. Some of these models cannot exhibit complex behavior due to their lack of richness. One way for a mathematical model to be rich is involving more variables. For example, Ref. [13] discusses the interaction between HIV and the immune system using a five-dimensional differential equation model. With this number of variables, the model can exhibit chaotic behavior. However, too many variables make the model more complicated and involve irrelevant details. On the other hand, there are also some models with fewer variables, but they are not able to represent the desired chaotic behavior. Thus it is important to balance the two factors of simplicity and richness of the model. For this purpose, the model should be capable of demonstrating complex and chaotic behavior, while involving the smallest number of variables. To accomplish this, some more realistic hypotheses should be considered. As mentioned above, our new model is based on the physiological properties of the HIV virus development and employs the concepts of prey-predator equations. Furthermore, a saturation phenomenon is assumed in the form of a logistic-like term to prevent an endless increase of the population of activated healthy immune cells, infected immune cells, or the virus.

To illustrate the qualitative behavior of the model, the bifurcation diagram with a variation of the parameter $r$ is shown in Fig. 1 (a). Bifurcation behavior is observed in the short range of activated healthy T cells for $0.01 < r < 0.04$, as shown in Fig. 1 (a). This confirms that the dynamics of the virus population is sensitive to the rate of activated healthy T cells. The chaotic behavior is observable for about $r = 0.01$ to $r = 0.0141$, which may denote different development of the virus in different individuals or under different circumstances. The system's dynamics follows a route to periodic behavior for $r > 0.0141$. The periodic development of the virus for $0.0141 < r < 0.0175$ is shown in Fig. 1 (a). For $r > 0.0175$, the virus dynamics is well controlled and exhibits a period-1 limit cycle. The corresponding Lyapunov exponents (LEs) of the model and the Kaplan-Yorke dimension ($D_{KY}$) are calculated and shown in Fig. 1 (b) and (c), respectively. Figure 1 (b) shows that in the interval $0.01 < r < 0.0141$, there are one positive, one zero and one negative Lyapunov exponents, thus, the behavior of the model is chaotic. Besides, the Kaplan-Yorke dimension is greater than 2 (Fig. 1 (c)). In this interval, there is a periodic window. In the zoomed window of the Lyapunov spectrum, it can be observed that in this limited range, the largest Lyapunov exponent decays to zero and two other exponents are negative. For $r > 0.0141$, the chaotic behavior changes to the periodic, and therefore, there are two negative and one zero Lyapunov exponents, and consequently the Kaplan-Yorke dimension becomes equal to one.

For an additional illustration, the respective state space plots for different values of $r$ are also displayed. Fig. 2 shows the state space for $r = 0.01$. As expected, a chaotic attractor is evident. The case with $r = 0.0124$ is in one of the periodic windows (the widest one) in the bifurcation diagram. The chaotic behavior ends abruptly and the system dynamics switches to periodic behavior within this tiny window. The chaotic region contains many narrow periodic windows. This may indicate the controlled dynamical behavior of the virus growth that is very sensitive to even small changes in the proliferation of active healthy T cells in different individuals or under different circumstances. The respective state space is shown in Fig. 3 in which the period-
behavior is observable. Figs. 4 and 5 show periodic behavior of the virus development for $r = 0.0155$ and $r = 0.025$, respectively.

6. Discussion

In this study, the interaction between the HIV virus and the immune system of the human body is investigated using a new mathematical model. Unlike some of the previous works, this model can exhibit complex and chaotic behavior while preserving its simplicity by involving only three variables. The main difference between our proposed model and the previous ones is in the two following key points:

1. A saturation phenomenon is assumed in the form of a logistic-like term to prevent an endless increase of the population of activated healthy immune cells, infected immune cells, or the virus.
2. Since the role of the infected immune cells has not been mentioned in the literature, in this model, the infected immune cells do not play any role in absorption or elimination of the virus, and it is just the healthy immune cells that struggle with the virus.

The bifurcation diagram with the variation of activated healthy T cells (parameter $r$) showed the qualitative behavior of the model. Chaotic dynamics is observed for a particular range of the parameter $r$. This may explain why the virus develops differently in different individuals or under different circumstances. The chaotic region also contains some narrow periodic windows, in which the chaotic mode suddenly ends at some critical points, and the system starts a periodic behavior for a tiny range of the parameter $r$. This is a complex dynamical behavior, where a periodic dynamic occurs amidst an otherwise chaotic process. It suggests the possibility of controllable development of the HIV virus even when it is in a random-like phase of the disease. Furthermore, the system dynamics follows a route to the periodic development of the virus for some ranges of the parameter $r$. After that, different dynamics of the population of the virus for some specific values of activated healthy T cells is illustrated in the state space diagram. For $r = 0.01$, the system shows chaotic behavior which might represent the high sensitivity of the virus population to the conditions. However, as the activated healthy T cells increased, for $r = 0.0155$ and $r = 0.025$, for example, the virus population becomes more controllable which is demonstrated in the form of periodic behavior of the activated healthy T cells.

7. Conclusion

In this paper, a new simple mathematical model, yet capable of representing complex behaviors was introduced. It is found that the healthy immune cells, in turn, determine whether the virus dynamics is periodic or chaotic. Also, bifurcation behavior was shown to be available in a short range of activated healthy T cells. Our model produced chaotic behavior for some ranges of activated healthy T cells while it followed a route to periodic behavior for some other ranges. Moreover, the state space for some modes of the system was displayed for more completeness.

The actual dynamics of immune cell regulation or virus proliferation is very complicated. However, understanding these dynamical behaviors can help develop better treatments and even theoretical predictions to optimize the experimental examinations. Certainly, the model
of HIV virus infection represented in this study is not perfectly accurate since many reductions and simplifications are made. However, the strength of this model is its richness and ability to demonstrate complex chaotic behavior while involving only three main variables. The purpose of the work is to develop theoretical studies and more particularly, the improvement of differential equation models that are used to explain some aspects of this disease. In summary to highlight some of the key works done in this research, the following points can be mentioned:

- The model is mathematically simple, involving only three main variables.
- The model is elegant [7].
- The model is rich and able to represent complex chaotic behavior.

**Competing interests**

The authors declare no competing interests.

**Author contribution**

All authors contributed equally to the paper.

**References**

Fatemeh Parastesh was born in Tehran, Iran, in 1992. She received the B.Sc. and M.S. degrees in Biomedical Engineering from the Biomedical Engineering Department, Amirkabir University of Technology, Tehran, Iran, in 2014 and 2017, respectively. She is currently the Ph.D. student in Biomedical Engineering Department of Amirkabir University of Technology. Her research interests include nonlinear dynamics, chaos networks, synchronization and chimera state.

Zainab Aram was born in 1990 in Tehran, Iran. She received her B.Sc degree in the Bioelectrical Engineering from Tehran University, Iran, in 2013. She received her Master of Science in biomedical engineering from AmirKabir University in October of 2016. Her research interests are Neuroscience, Neuroengineering, Dynamical Systems, Chaotic Modeling, Nonlinear Dynamics.

Hamidreza Namazi received his Ph.D. in Mechanical Engineering from Nanyang Technological University (Singapore). Currently, he is working as an adjunct lecturer at Monash University (Malaysia), as a research associate at the University of Calgary (Canada), as a visiting professor at the University of Hradec Kralove (Czech Republic), as an adjunct fellow in Victoria University (Australia), and as a researcher at Masaryk University (Czech Republic). His research interests primarily focus on mathematical and computational analysis and modeling of time series and patterns in mechanical and biomedical engineering. He is the corresponding author of more than 85 peer-reviewed journal papers in this area of research.

Julien Clinton Sprott, born September 16, 1942 in Memphis, Tennessee, received his B.S. in physics from the Massachusetts Institute of Technology in 1964 and his Ph.D. in physics from the University of Wisconsin in 1969. He worked at the Oak Ridge National Laboratory for several years before returning to the University of Wisconsin to join the physics faculty in 1973. In 2008, he became an Emeritus Professor of Physics. His research has been primarily in the area of experimental plasma physics and controlled nuclear fusion. In 1989 his interests turned to nonlinear dynamics, chaos, fractals, and complexity. He has authored or coauthored about 500 scientific papers in these and related fields.

Sajad Jafari was born in Kermanshah, Iran, in 1983. He received his B.Sc., M.S., and Ph.D. degrees in Biomedical Engineering in 2005, 2008, and 2013 from Biomedical Engineering Department, Amirkabir University of technology, Tehran, Iran. He is currently an assistant professor in there (since 2013). His research interests include nonlinear and chaotic signals and systems and complex networks. He serves as editor in International Journal of Bifurcation and Chaos, International Journal of Electronics and Communications, and Radioengineering.
**Table 1.** Parameters of Eq. 2.

**Fig. 1.** (a) The bifurcation diagram of HIV virus development based on Eq. 2 with a variation of active healthy immune cells (parameter $r$) for the parameters in Table 1 and initial conditions $(x, y, z) = (-0.6, -0.66, 1.03)$. (b) The corresponding Lyapunov exponents. (c) The corresponding Kaplan-Yorke dimension.

**Fig. 2.** The state space plot of the system in Eq. 2 for $r = 0.01$ and the parameters in Table 1. The initial conditions are $(x, y, z) = (-0.6, -0.66, 1.03)$.

**Fig. 3.** The state space of the system in Eq. 2 for $r = 0.0124$ and the parameters in Table 1. The initial conditions are $(x, y, z) = (-0.6, -0.66, 1.03)$.

**Fig. 4.** The state space plot of the system in Eq. 2 for $r = 0.0155$ and the parameters in Table 1. The initial conditions are $(x, y, z) = (-0.6, -0.66, 1.03)$.

**Fig. 5.** The state space of the system in Eq. 2 for $r = 0.025$ and the parameters in Table 1. The initial conditions are $(x, y, z) = (-0.6, -0.66, 1.03)$.
### Table 1.

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Fig. 1.
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