Vol 18 No 12, December 2009
1674-1056/2009/18(12)/5111-06

Chinese Physics B

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A stochastic epidemic model on homogeneous networks*

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(Received 24 March 2009; revised manuscript received 16 May 2009)

In this paper, a stochastic SIS epidemic model on homogeneous networks is considered. The largest Lyapunov exponent is calculated by Oseledec multiplicative ergodic theory, and the stability condition is determined by the largest Lyapunov exponent. The probability density function for the proportion of infected individuals is found explicitly, and the stochastic bifurcation is analysed by a probability density function. In particular, the new basic reproductive number R^*, that governs whether an epidemic with few initial infections can become an endemic or not, is determined by noise intensity. In the homogeneous networks, despite of the basic productive number $R_0 > 1$, the epidemic will die out as long as noise intensity satisfies a certain condition.

Keywords: homogeneous networks, SIS epidemic model, stochastic stability, stochastic bifurcation
PACC: 0250, 0590

1. Introduction

During the past few years, a great deal of epidemiology research works have been done on complex networks.[1−15] These works focus on two different complex networks: the Watts–Strogatz model and the Barabási and Albert model. The first model is a homogeneous network exhibiting small-world properties,[16] and the second one is a typical example of a scale-free network.[17] In these models, individuals are modeled as nodes, and possible contacts between individuals are linked by edges. Each susceptible node is infected with rate ν if it is connected to one or more infected nodes. Infected nodes are cured and become susceptible with rate δ. Thus an effective spreading rate is defined as $\lambda = \nu/\delta$ (without lack of generality, we set $\delta = 1$). The SIS model is generally used to study infections by a stationary average density of the infected individuals, named endemic steady.

However, the above spreading models do not allow for casual contacts. In fact, the spreading rate, which is not only constant unvaryingly, will be disturbed by all kinds of factors. For example, in the Internet and the World Wide Web, the capability of each computer is different in resisting viruses; even more, some viruses will spread only when some files or e-mails are operated, otherwise the viruses will not spread. In the spreading of the disease, all various of random factors including temperature, humidity and some man-made intervention will affect the spreading of the diseases. The temperature difference between day and night, even seasonal change affecting the spreading rate of infectious diseases is a common phenomenon in both temperate and tropical climates.[18] An immediate question that then arises is how to describe these random factors on the complex networks and how the properties of the spreading of the disease change and even the threshold.

In nature and most systems, noise can commonly be regarded as a random and persistent disturbance. The presence of noise in fact can modify the behaviour of the corresponding deterministic evolution of the systems, including asymptotic behaviour, stability, and bifurcation, etc.[19,20] As far as the theory of stochastic bifurcation is concerned, it is still in its infancy since it is much harder to deal with stochastic bifurcation problems than deterministic bifurcation problems. At present, there are mainly two kinds of definitions for stochastic bifurcation available. One is based on the sudden change of shape of the stationary probability density function — the so-called P-bifurcation; and the other is based on the sudden change of sign of the largest Lyapunov exponent — the so-called D-bifurcation.[21,22]

The aim of this paper is to present a comparison of the effect of environmental noise on homogenous networks. In order to estimate the effects of environmen-

*Project supported by the Science Foundation of Shanxi Province of China (Grant No 2009011005-1), the Youth Foundation of Shanxi Province of China (Grant No 2007021006).
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tal noise on homogenous networks, we will investigate the standard SIS model with white noise. By using the Oseledec multiplicative ergodic theory, we determine the stochastic stability of the system; and by utilizing the probability density function of the infected individuals, we analyse the stochastic bifurcation of the system.

The organization of this paper is as follows: Section 2 gives a stochastic SIS epidemic model, and then the condition of the stochastic stability is derived in Section 3. In Section 4 the analysis of the stochastic bifurcation is given. Section 5 ends this paper with a brief conclusion and discussion.

2. The stochastic model on homogeneous networks

In the small-world networks, the Watts–Strogatz model has attracted a great deal of attention. It can be constructed as follows: The starting point is a ring with N nodes, in which each node is symmetrically connected with its $2K$ nearest neighbours. Then, for every node each link connected to a clockwise neighbour is cut as originating from the original node and rewired to a randomly chosen target node with probability p. This procedure generates a random graph with a connectivity distributed exponentially for large k, and an average connectivity $\langle k\rangle = 2K$.[16]

In these networks, one can generally consider that each node has the same number of links, therefore, their connectivity properties can be considered to be homogeneous. At a mean-field level, the equation describing the time evolution of the average density of infected individuals $\rho(t)$ is

$$\frac{\mathrm{d}\rho(t)}{\mathrm{d}t} = -\rho(t) + \lambda\langle k\rangle\rho(t)(1-\rho(t)). \tag{1}$$

In Eq.(1) the first term on the right-hand side represents infected nodes becoming healthy with unit rate. The second term considers the average density of new infected nodes generated by each active node. This is proportional to the infective rate λ, the number of links emanating from each node $\langle k\rangle$, and the probability for any given link points to a healthy node, $1-\rho(t)$.

It is easy to know

$$\lim_{t\to\infty}\rho(t) = \begin{cases} 0, & \text{if } R_0 < 1, \\ \rho^* = 1 - \dfrac{1}{R_0}, & \text{if } R_0 > 1, \end{cases}$$

where $R_0 = \lambda\langle k\rangle$, which is called the basic productive number. It has been shown that there is an epidemic threshold on the homogeneous Watts–Strogatz (WS) small-world network, if $R_0 < 1$ the disease will disappear and all population will become susceptible and the disease-free equilibrium of system (1) is globally asymptotically stable. If $R_0 > 1$ the disease always remains endemic and the endemic equilibrium of system (1) is globally asymptotically stable.

Due to the fact that the stochastic environmental variability λ becomes a random parameter, that is, $\lambda \to \lambda + \sigma\xi(t)$, where $\xi(t)$ is the white noise with mean zero and variance one, and σ is the noise intensity. Thus the equation (1) becomes

$$\begin{aligned}\mathrm{d}\rho(t) = (-\rho(t) + \lambda\langle k\rangle\rho(t)(1-\rho(t)))\mathrm{d}t \\ + \sigma\langle k\rangle\rho(t)(1-\rho(t))\mathrm{d}W(t),\end{aligned} \tag{2}$$

where $W(t)$ is the Wiener process. Let $F(\rho) = -\rho + \lambda\langle k\rangle\rho(1-\rho), G(\rho) = \sigma\langle k\rangle\rho(1-\rho)$, thus equation (2) becomes

$$\mathrm{d}\rho = F(\rho)\mathrm{d}t + G(\rho)\mathrm{d}W. \tag{3}$$

3. Stability of the stochastic model

To determine the stability of the stochastic model, the most common method is to calculate the largest Lyapunov exponent of the system, firstly the linearized equation (2), and then solve the largest Lyapunov exponent of the linearized Itô stochastic differential equations. Based on the Oseledec multiplicative ergodic theorem,[23] the largest Lyapunov exponent is defined as

$$l = \lim_{t\to\infty}\frac{1}{t}\ln \parallel \rho(t,\rho_0) \parallel . \tag{4}$$

The solution of the linearized Itô stochastic differential equations is

$$\begin{aligned}\rho(t) = \rho(0)\exp\left(\int_0^t\left(F^{'}(0) - \frac{G^{'2}(0)}{2}\right)\mathrm{d}s \right. \\ \left. + \int_0^t G(0)\mathrm{d}W(s)\right),\end{aligned} \tag{5}$$

where $F^{'}(0) = \lambda\langle k\rangle - 1$, $G^{'2}(0) = \sigma^2\langle k\rangle^2$.

Introducing the norm $\parallel \rho(t) \parallel = \rho^{1/2}(t,W)$, then the largest Lyapunov exponent of the linearized Itô stochastic differential equations is

$$
\begin{aligned}
l &= \lim_{t\to\infty}\frac{1}{t}\ln\rho^{1/2}(t) = \lim_{t\to\infty}\frac{1}{2t}\ln\rho(t)\\
&= \lim_{t\to\infty}\frac{1}{2t}\left\{\ln\rho(0) + \int_0^t\left(F^{'}(0) - \frac{G^{'2}(0)}{2}\right)\mathrm{d}s + \int_0^t G(0)\mathrm{d}W(s)\right\}\\
&= \frac{2\lambda\langle k\rangle - 2 - \sigma^2\langle k\rangle^2}{4}.
\end{aligned}
\tag{6}
$$

If $\lambda < 1/\langle k\rangle + \sigma^2\langle k\rangle/2$, then $l < 0$, the trivial solution of linear Itô stochastic differential equation is asymptotically stable with probability one, and so does the system (2), the solution of system (2) tends to the disease-free equilibrium with a probability of one (see Fig.1). If $\lambda > 1/\langle k\rangle + \sigma^2\langle k\rangle/2$, then $l > 0$, the trivial solution is unstable with probability one; the solution of system (2) fluctuates around the endemic equilibrium (see Fig.2). If $\lambda = 1/\langle k\rangle + \sigma^2\langle k\rangle/2$, then $l = 0$, the system undergoes a bifurcation, which is called D-bifurcation. The stochastic bifurcations will be discussed in the next section.

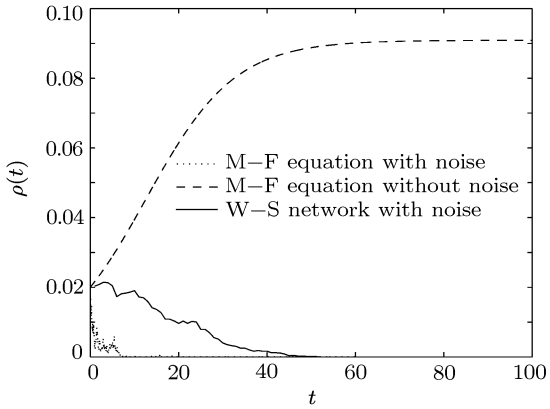


Fig.1. Deterministic and stochastic trajectories of the homogeneous Watts–Strogatz (WS) small-world network model for initial condition with $R_0 = 1.1$, $\sigma = 0.18$, $\langle k\rangle = 6$.

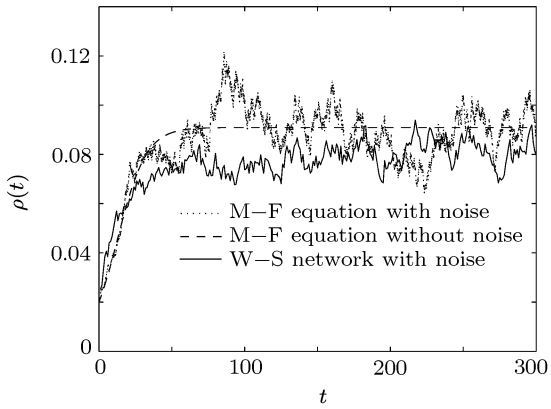


Fig.2. Deterministic and stochastic trajectories of the homogeneous Watts–Strogatz (WS) small-world network model for initial condition with $R_0 = 1.1$, $\sigma = 0.07$, $\langle k\rangle = 6$.

In the numerical implementation of our model, we start with a small-world network with $N = 10000$ and $p = 0.01$. The dynamics proceed by discrete steps. At first the infected node i is chosen at random. If the time elapsed from the moment of entering the infectious cycle up to the current time is larger than the infection time, the node i becomes susceptible. Otherwise, the neighbour j of i is selected with a probability of λ. If j is in the susceptible state, contagion occurs. The node j becomes infected, and its infection time is recorded. If j is already infected, it preserves its state. In order to take into account stochastic noise in the spreading of the disease, we consider the neighbour of i is selected with a random parameter $\lambda + \sigma\xi$. Using Matlab 7.0, we give the numerical simulations.

Numerical simulations show that the introduction of noise in the homogeneous Watts–Strogatz small-world network also will change the dynamic behaviours of diseases. With the different parameters, the proportion of the infected individuals will tend to zero (see Fig.1 and Fig.3) or fluctuate around an stability state (see Fig.2). We can also see from the results of numerical simulations that the difference between the mean-field equation and the WS small-world network model since the mean-field method approximates the real small-world networks.

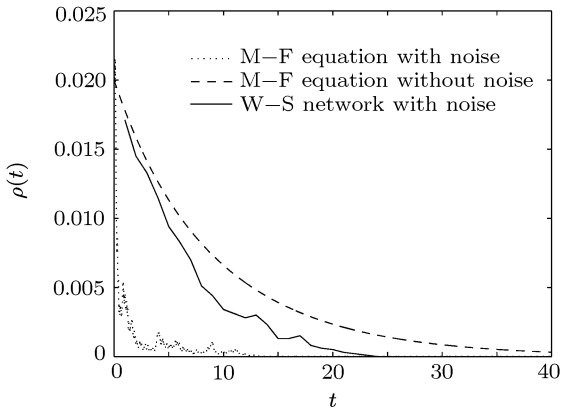


Fig.3. Deterministic and stochastic trajectories of the homogeneous Watts–Strogatz (WS) small-world network model for initial condition with $R_0 = 0.9$, $\sigma = 0.18$, $\langle k\rangle = 6$.

From the biological point of view, the introduction of noise in the deterministic SIS model modifies

the basic productive number $R_0 = \lambda\langle k\rangle$ giving rise to a new basic productive number $R^* = \frac{\lambda\langle k\rangle}{1+\sigma^2\langle k\rangle^2/2}$. If $R_0 < 1$, it is obvious that $R^* < 1$, thus the diseases in the SIS model without and with noise are all extinct (see Fig.3). If $R_0 > 1$, the disease in the SIS model without noise will break out and persist; while in the model with noise, as long as the noise intensity satisfies the following condition $\sigma > \sqrt{2}\sqrt{R_0 - 1}/\langle k\rangle$, the epidemic still will die out with a probability of one (see Fig.1).

We now begin to analyse the effects of the average connectivity and the noise intensity. Fixed the noise intensity, the relation between the basic reproductive number R^* and the average connectivity $\langle k\rangle$ can be expressed by a parabola. It means that there exists a constant $\langle k\rangle^*$, if $\langle k\rangle < \langle k\rangle^*$, the basic reproductive number R_0 increases with $\langle k\rangle$. It is shown that the more people contact with others, the easier the spread of disease. On the contrary, if $\langle k\rangle > \langle k\rangle^*$, the increase of $\langle k\rangle$ will lead to the decrease of R^* (see Fig.4). Figure 4 also gives the curves of R^* with different noise intensities. From the top down, the values of the noise intensity are $0.12, 0.14, 0.16, 0.18$ respectively. Fixed the average connectivity $\langle k\rangle$, R^* is a monotone decreasing function of σ (see Fig.5). Figure 5 also gives the curves of R^* with different average connectivity $\langle k\rangle$. From the top down, the values of the average connectivity $\langle k\rangle$ are 3, 4, 5, 6 respectively.

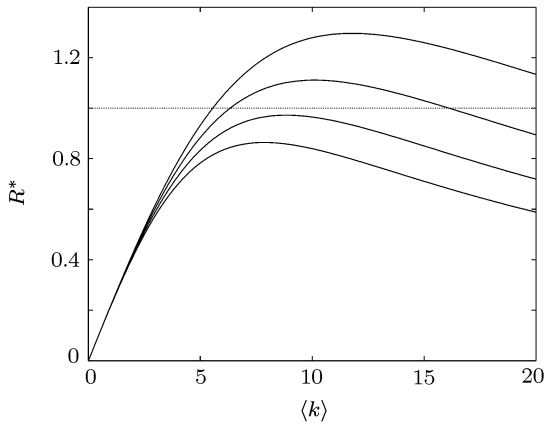


Fig.4. The curves of R^* with different noise intensity.

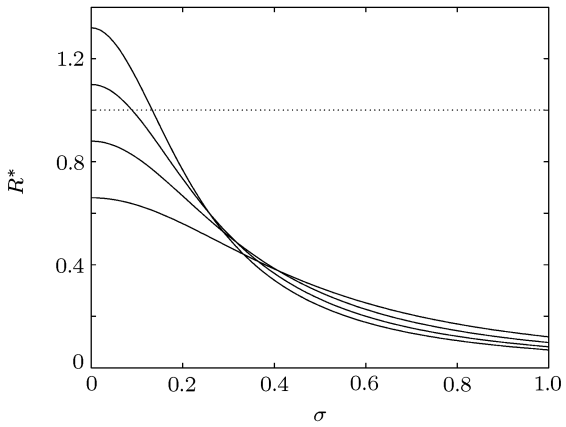


Fig.5. The curves of R^* with different average connectivity.

4. Bifurcations of the stochastic model

Considering the Itô solution of equation (3), the forward equation is

$$\frac{\partial\phi(\rho,t)}{\partial t} = -\frac{\partial}{\partial\rho}(F(\rho)\phi(\rho,t)) + \frac{1}{2}\frac{\partial^2}{\partial\rho^2}((G(\rho))^2\phi(\rho,t)), \tag{7}$$

where $\phi(\rho,t)$ is the probability density function of $\rho(t)$. The steady state density $\phi^*(\rho)$ satisfies the following equation

$$\frac{\mathrm{d}^2}{\mathrm{d}\rho^2}((G(\rho))^2\phi^*(\rho)) - 2\frac{\mathrm{d}}{\mathrm{d}\rho}(F(\rho)\phi^*(\rho)) = 0. \tag{8}$$

When $R_0 > 1$, define $r = \sqrt{R_0 - 1}/\langle k\rangle$, then the solution of Eq.(8) is

$$\phi^*(\rho) = C\rho^{2(r^2/\sigma^2)-2}(1-\rho)^{-2(r^2/\sigma^2)-2} \times \mathrm{e}^{-2/[\sigma^2\langle k\rangle^2(1-\rho)]}. \tag{9}$$

Here C is a normalization constant, which is determined by $\int_0^1 \phi^*(\rho) = 1$. For convenient analysis, we assume $C = 1$.

Suppose that $\sigma < r$. It is easy to know in this case $\lim\limits_{\rho\to 0}\phi^*(\rho) = 0$. By differentiating $\phi^*(\rho)$, we see that the extreme values of $\phi^*(\rho)$ can be determined by the roots of the equation $f(\rho) = 0$, thereinto

$$f(\rho) = 2\sigma^2\rho^2 + (r^2 - 3\sigma^2 + 1/\langle k\rangle^2)\rho + \sigma^2 - r^2. \tag{10}$$

Since $f(0) = \sigma^2 - r^2 < 0$ and $f(1) = 1/\langle k\rangle^2 > 0$, then there must exist some $\rho^* \in (0,1)$, which makes $f(\rho^*) = 0$. So the function $\phi^*(\rho)$ is unimodal (Fig.6).

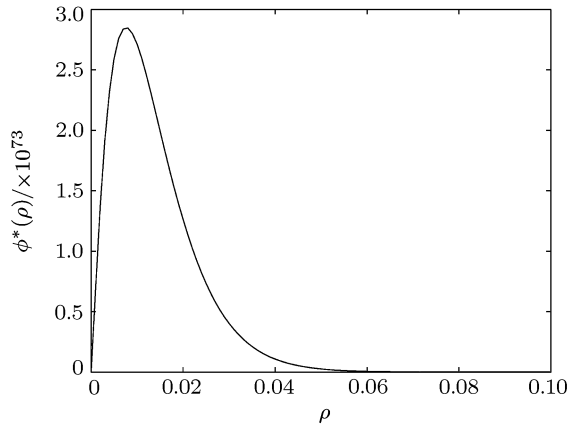


Fig.6. The probability density function $\phi^*(\rho,t)$ with $R_0 = 1.1$, $\sigma = 0.05$, $\langle k\rangle = 6$.

Suppose $r < \sigma < \sqrt{2}r$. Then $\phi^*(\rho)$ is singular and integrable at $\rho = 0$. In Eq.(10), if $\Delta < 0$, that is

$$3\sigma^2 - r^2 + 2\sqrt{2}\sigma\sqrt{\sigma^2 - r^2} > \frac{1}{\langle k\rangle^2}, \qquad (11)$$

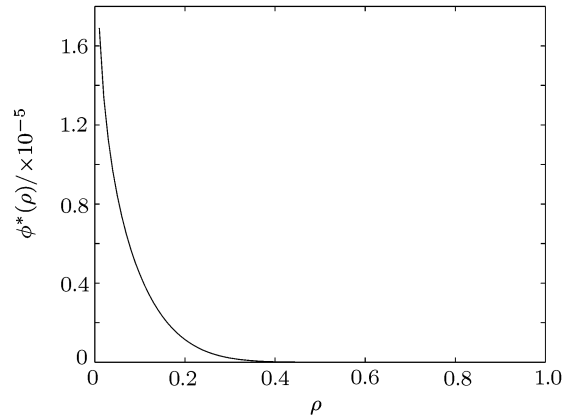


Fig.7. The probability density function $\phi^*(\rho,t)$ with $R_0 = 2.2$, $\sigma = 1.18$, $\langle k\rangle = 6$.

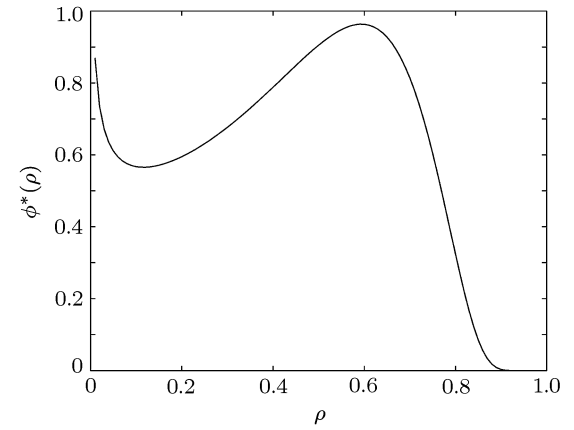


Fig.8. The probability density function $\phi^*(\rho,t)$ with $R_0 = 1.1$, $\sigma = 0.3$, $\langle k\rangle = 6$.

then $f(\rho)$ is strictly positive for $0 < \rho < 1$, hence $\phi^*(\rho)$ is a monotonic decreasing function (Fig.7). If equation (11) is not satisfied, then $f(\rho)$ has two zeros for $0 < \rho < 1$, and the function $\phi^*(\rho)$ is bimodal (Fig.8).

Suppose $\sigma \geq \sqrt{2}r$. Then $\phi^*(\rho)$ is singular and not integrable at $\rho = 0$, thus it can be represented by a δ function (see Fig.9).

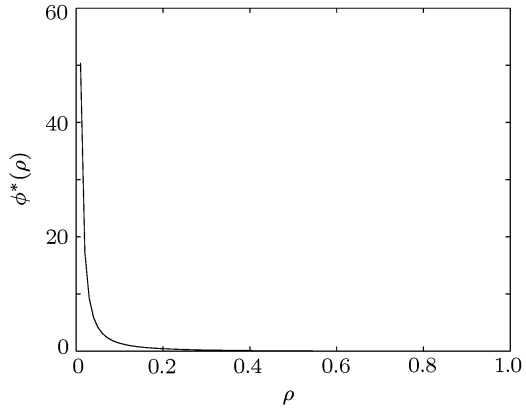


Fig.9. The probability density function $\phi^*(\rho,t)$ with $R_0 = 1.1$, $\sigma = 1.18$, $\langle k\rangle = 6$.

We can see from above, in the limit $t \to \infty$, when $\sigma < \sqrt{2}r$, the probability density function $\phi(\rho,t)$ tends to a distribution defined within a multiplicative constant by $\phi^*(\rho,t)$, and when $\sigma \geq \sqrt{2}r$, $\phi(\rho,t)$ will tend to zero with probability one. The simulations can be seen from Fig.1 and Fig.3 when $\sigma \geq \sqrt{2}r$, and figure 2 gives the deterministic and stochastic trajectories of the homogeneous Watts–Strogatz small-world network model when $\sigma < \sqrt{2}r$.

5. Conclusions

In this paper, a simple stochastic model for the dynamics of an infectious disease on complex networks is considered. The only stochastic component is due to environmental variability, which we incorporate in the model as Gaussian white noise about the disease transmission rate. The largest Lyapunov exponent is calculated by Oseledec multiplicative ergodic theory, and the stability condition is determined. The stochastic bifurcation is analysed by the probability density function. Model analysis shows that there exists a critical value of the density of the noise below which the steady state of the probability distribution of disease prevalence may be found explicitly, but when the density of the noise is greater than the critical value the disease becomes eradicated with a probability of one.

Furthermore, the effects of the average connectivity and the noise intensity are analysed. We can see the positive effect of noise in the analysis of the

stochastic model. The existence of noise can suppress the spreading of the diseases (see Fig.1). The results can explain why the spreading of the diseases is easier in a mild environment than that in an abominable environment. This indicates that noise, if used properly, can be beneficial to some real applications.

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