A MATHEMATICAL MODEL OF THE SPREAD OF SARS

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The nature of the rapid spread associated to a fast acting pathogen is generally of great concern. The high death\(^1\) toll of the Severe Acute Respiratory Syndrome (SARS) is alarming. It is the first emerging epidemic of the 21st century and its etiological agent is the coronavirus and in view of this, it is necessary to examine these processes using mathematical tools. The classical diffusion equation describing the properties of an isotropic random motion provides the starting point of the present analysis of air borne diseases. There is currently no evidence of person-to-person transmission of SARS in the world. The oscillatory nature of the solution to the model equation could explain why it is not an easy task to contain pathogens whose behavioural evolution mimics the diffusion equation.

1. Mathematical Epidemics Model and its advantages
This introductory part is channeled towards a brief surve of the advantages of mathematical models. Infectious disease data are highly dependent because infected cases are the cause of further infected cases. To overcome these difficulties, it is necessary to make opportunistic use of mathematical models which have the appropriate chance elements of disease spread built into them. One can thereby, subject to making the correct model assumption, obtain a more efficient analysis as well as directing attention at epidemiologically meaningful parameters.

In general, infection is by direct ‘sufficiently close’ host-to-host contact or by infecting the environment and a susceptible host making ‘sufficiently close’ contact with the environment where in this context, the meaning of environment depends on the particular disease, and might include the linen and cutlery of the household as well as the ambient air. Becker (1989) remarked that infectious diseases were once the main cause of morbidity and mortality in man. This assertion still holds true till date. Nevertheless, nowadays, wars, genocides, religious and ethnic conflicts do contribute a great deal to mortality.

Over the centuries human beings have managed to eradicate the most serious of these diseases from many parts of the world (Bailey, 1975). However, diseases such

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\(1\) In loving memory of Simo Andree Monique.
as malaria and schistosomiasis still affect hundreds of millions of people, while the
number of people affected by leprosy in Africa for example is still many tens of
millions (Becker, 1989). Waterborne diseases and HIV-AIDS pose a serious treat
to humanity and of recent in addition to this list is the Severe Acute Respiratory
Syndrome (SARS), a disease which is fast killing.
Even countries free from more serious infectious diseases still have public health
problems due to emergence of such epidemics, coupled also with the fear of biological
attacks. Becker (1969) posited that with increased air travel the entire world
population is at risk from pandemics of diseases such as influenza, pneumonia, as
well as occasional local outbreaks such as cholera, even in countries essentially free
from these diseases. This has been confirmed with the recent outbreak of SARS in
the Gwandong Province of China. Migration is the prime factor for the appearance
of the disease in other parts of the world. Hence, migration, which is the third
demographic parameter should be given prominence, (Tchuenche (a)) though it
is also the most difficult to model (Martcheva and Milner, 2001). Travelers are
at higher risk of contracting this new form of acute respiratory infection due to
the closeness of individuals for a long time and thanks to WHO and CDC who
have been giving information and guidelines for travelers on their websites. The
passengers aboard a plane in modeling terms form a cohort and the plane have
similar characteristics of a household.
There is always a risk of new infectious disease developing which can pose a serious
treat to mankind. The treat currently posed by AIDS and SARS illustrates this
point fully. More often than not, the epidemic can be controlled if the health and
public authorities cooperate fully and adequate information is passed on to the
population for preventive measures. Although SARS epidemics is probably in its
early stages, some unanswered questions since the first case of SARS was discov-
ered and reported by the late Italian doctor Carlo Urbani, who also succumbed
to the fast and deadly disease still remain: Why are some authorities reluctant to
inform the populace on time? Why are they giving a low figure of cases? This
laxity favours more infections because if a lower number of cases is ascertained,
then the unaccounted cases are still on the streets and continue to infect others.
One thing they have forgotten is that sometimes awareness is the best preventive
measure. We shall not dwell into this any longer.
It is usually impossible to observe the time at which infections occur or to know
which infected person is responsible for transmitting the disease to a particular
susceptible. However, for most infectious diseases we have some understanding of
how a transmission of the disease can occur, and it seems preferable to incorpo-
rate this knowledge into a model aimed at describing the infection process, and of
course, it is important to be sure that the model assumptions are supported both
empirically and by biological as well as sociological considerations, because the
claimed gain in efficiency becomes meaningless if the model is incorrect. Therefore, a desirable by-product of using a model which is specifically formulated to suit the application is that its parameters have well-understood interpretations.

Another important question about a model concerns the degree of complexity to be incorporated into it. An objection often made of mathematically formulated epidemic models is that they involve too many simplifying assumptions. Much is often made of this objection because in fact most method of analysis involve such assumptions, but only mathematically formulated approach explicitly exposes all the assumptions made. Indeed, this property of mathematical formulation coupled with the fact that many concepts, models, methods of analysis and their interpretations are clearly described in a mathematical framework, helps to explain why in most areas of science, mathematics is recognized as a usable and useful means of communication between research workers.

Nevertheless, it is true that many simplifying assumptions are contained in most of the epidemic models to be found in the literature, but it is quite wrong to reject such models merely on the basis of their simplicity, because therein lies a considerable part of their value. A simple model is an idealization and generally cannot be viewed as representing exactly the spread of real epidemics, and there is the obvious advantage that the simple model is more likely to yield to mathematical analysis. It is for the sake tractability and convenience that model building often starts with an oversimplification, which then paves the way to more complex ones. Apart from this advantage, however, simple models can reveal more clearly what the important characteristics are, because in complex ones the important features are often mixed in with less important ones.

Thus, a good epidemic model specifically formulated to describe an infection process will usually explicitly involve parameters with clear epidemiological meanings. In most sciences, the effects of changes are assessed by analyzing the results of repeated experiments. As this is generally not possible with infectious diseases, it is natural to try to overcome this difficulty by constructing a model which adequately describes the basic features of the epidemics in the community and then using the model to predict the consequences of introducing specific changes. Some epidemiologists in the past often regarded the mathematical theory of infectious diseases as a theoretical exercise rather than a body of knowledge having practical relevance. They were somehow short-sighted like some pure mathematicians who refuse to recognize how much modeling goes on within the precincts of pure mathematics. As was pointed out by Rosen (1993), a great deal of what passes for pure mathematics is really applied mathematics. Mathematics is not only a tool, but a key to the understanding of many things. It is an art, a beauty, a wonderful and fascinating subject which is found everywhere (Tchuenche (b)). Some of the mathematical theory can help to give insight to disease spread and it is important
to learn what we can from this theory rather than dismiss all of it as an academic exercise. These are some of the insights to be gained from the study of epidemic models and the uses that can be made thereof. Since infectious diseases continue to pose global public health problems, especially with the incursion of SARS which has no treatment and vaccine, the methods of mathematical analysis described in the next section are aimed at improving our understanding of SARS and its spread through communities, with the hope that such additional knowledge will help in the control of this great masquerader. Due to the complex interplay between mathematics and society, our first perusal at least, the lay-reader should not concern himself unduly with details, but should concentrate on getting a general impression of the process being modeled\footnote{biosocial modeling}.

This work is organized as follows: In the next section we discuss the mode of spread of SARS, while the last section is a brief conclusion.

2. The Model

The model we formulate is over-simplified but realistic enough and its study is a necessary prelude to the construction of more complex ones. As of March 10, 2004, there was no evidence of person-to-person transmission of SARS in the world (WHO), which is possibly an airborne disease and this justifies our choice of the diffusion equation with the assumption that the mode of transmission is random. The key fact here is that even though a process is regarded as random, in many circumstances we can write down deterministic problems in partial differential equations that yields a definite distribution function for the probabilities associated with it. The partial differential equation or the distribution function would be identical with the equation one writes in a deterministic characterization of the phenomenon.

In Brownian motion, small particles move about irregularly in liquid or gas and perhaps the most common manifestation of this phenomenon is the dancing of dust particles in a beam of sunlight. The relatively large dust particles move because of myriads of impacts by the molecules that comprise the surrounding medium (Lin and Segel, 1974). In making a mathematical model of such situation, we should without doubt regard the impacts as occurring randomly. Nevertheless, there is no possibility of computing the trajectory of each molecules, and no interest in doing so, if one wishes to understand the broad outlines of the phenomenon. We assume without loss of generality, isotropic random walk in this study.

Let $C(x, t)$ denote the concentration of matter at point $x$, time $t$ ($t \in \mathbb{R}^+, x \in \mathbb{R}$). If the motion is taken to be completely isotropic (Okubo, 1980), then $C$ satisfies
the following

\[
\frac{\partial C}{\partial t} = D \frac{\partial^2 C}{\partial x^2}
\]

(1)

where \( D \) is the diffusivity. \( C(x, t) \geq 0 \). (No one cares what this equation does when the variable is negative (Rosen, 1993) probably because negative values are biologically meaningless).

Also,

\[
\int_{-\infty}^{\infty} C(x, t) \, dx = 1
\]

(2)

and

\[
\lim_{t \to 0} C(x, t) = M; \quad x = 0
\]

(3)

Condition (2) can be regarded as wind blowing particles across continents and oceans, while equation (3) is required because particles are assumed to start at an origin. Equation (1) is the diffusion equation, which is obtained as the limiting description of random motion (Okubo, 1980). By considering a situation where there is a perfect absorber just to the right of \( x = L \), this means that the particle is removed if it moves to the right from \( x = L \) (a susceptible has possibly inhaled the virus), we obtain the boundary condition

\[
u(L, t) = 0
\]

(4)

The solution \( C_A(x, t) \) that satisfies the differential equation (1), the normalization condition (2) and the initial condition (3) is given by

\[
C_A(x, t) = C(x, t) - C(x - 2L, t)
\]

\[
= \frac{M}{2\sqrt{\pi D t}} e^{-x^2/4Dt} \left( 1 - e^{-\frac{-L(L-x)}{Dt}} \right)
\]

(5)

The subscript \( A \) denotes absorber. One intriguing thing we note here is that in the case of SARS as many other infectious diseases, infectives become immediately a new source of infection. In this case the intermediate sign in equation (5) changes and we have

\[
C_A(x, t) = \frac{M}{2\sqrt{\pi D t}} e^{-x^2/4Dt} \left( 1 + e^{-\frac{-L(L-x)}{Dt}} \right)
\]

(6)

which is similar to the result in the presence of a reflecting barrier with equation (2) taking the form \( \int_{-\infty}^{\infty} C(x, t) \, dx = 1 \). More realistically, we have

\[
\int_{-L}^{L} C(x, t) \, dx = 1 \quad \text{or} \quad \int_{-L}^{L} C(x, t) \, dx = 1
\]
where $E \subset \mathbb{R}^n$ is a neighbourhood of $x$

The distribution function in (6) provides basically the same result as that of a random walk if $D$ is defined appropriately. It is noteworthy to mention here that the presence of a perfectly reflecting barrier means that a particle that reaches $x = L$ at time $t$ and then moves to the right will again be found at $x = L$ at time $t + \Delta t$ ($\Delta t$ is a small increment of time). This is not the case for parasites because a person infected at point $x = L$ time $t$, may not necessary be the one to infect the next individual at that same point at time $t + \Delta t$ due to human migration. This calls for more randomness in the spread of infections.

### 2.1 First passage time

Let $F(L, t)dt$ be the probability that a particle is absorbed at $x = L$ in the time interval $(t, t + \Delta t)$ given that it started at “the origin” when $t = 0$. This also represents the uniform probability that a particle makes its first passage to $L$ in $(t, t + \Delta t)$. Various studies (Lin and Segel, 1974) show that

$$F(L, t) = -D \left( \frac{\partial U_A}{\partial t} \right)_{x=L}$$

From equation (5)

$$F(L, t) = 2LM \left( \frac{D}{\pi t} \right)^\frac{1}{2} e^{-\frac{L^2}{4Dt}}$$

In the case of pathogens, it is not easy to estimate $M$ and only empirical values can be assigned. In such a case, the first passage time for equation (6) given by equation (8) yield an estimate, also because as mentioned earlier, infected individuals are sources of further infections.

When the population sizes get small, the rate equation in (1) progressively lose its meaning (subtle mathematics, but very poor modeling, Rosen (1993)), hence the use of differential equation and all the tools of analysis that come with them are tacitly predicated on a hypothesis, namely that the population sizes are large enough to justify it.

Equation (1) can readily be solved to yield (see Tasaki and Matsumoto 2002, Sneddon 1957).

$$C(x, t) = M \left[ 1 - \text{erf} \left( \frac{x}{2\sqrt{Dt}} \right) \right]$$

where $\text{erf}(z) = \frac{2}{\sqrt{\pi}} \int_0^z e^{-u^2} du; \ z > 0$ represents the error function. From the general solution of the wave equation $y = f(x + \nu t) + g(x - \nu t)$, $\nu$ being the wave velocity, we have that to an observer moving along with the propagating force in the positive direction of $x$ at constant velocity $\nu$, the profile seen in the vicinity of
the boundary is time independent. To analyze the potential profile in the vicinity of the boundary, therefore, it is convenient to adopt a new space variable \( X \) defined by \( X \equiv x - \nu t \). When this variable is adopted, the partial differential equation (1) is reduced to the following ordinary differential equation

\[
D \frac{d^2 C}{dx^2} + \nu \frac{dC}{dX} = 0 \tag{10}
\]

which has a trivial solution given by

\[
C = Me^{\frac{-\nu}{D}(X - X_0)} \tag{11}
\]

where \( X = X_0 \) is the free region.

In the opposite direction of \( x \), equation (10) becomes

\[
D \frac{d^2 C}{dt^2} - \nu \frac{dC}{dY} = 0 \tag{12}
\]

where \( Y \equiv x + \nu t \) and the solution is given by

\[
C = Me^{\frac{\nu}{D}(X - X_0)} \tag{13}
\]

When a space-occupying medium (e.g., air) itself flows, matter are transported accordingly and one method for accounting for this force is by introducing advection (or convection) in the diffusion equation (1) (Okubo, 1980). If for an infinitesimal volume we assume that the diffusion of the instantaneous concentration obeys Fick’s law, then the total instantaneous flux in the \( x \) direction takes the form

\[
J_x = uC - D \frac{\partial C}{\partial x} \text{ and equation (1) becomes}
\]

\[
\frac{\partial C}{\partial t} = -u \frac{\partial C}{\partial x} + D \frac{\partial^2 C}{\partial x^2} \tag{14}
\]

\((u\) is a constant).

In the positive direction, applying the same argument as above we obtain

\[
D \frac{\partial^2 C}{\partial X^2} + \nu \frac{\partial C}{\partial X} - u \frac{\partial C}{\partial X} = 0 \tag{15}
\]

which has similar solution to (13). For a perfectly homogeneous environment, \( u = \nu \) and the ambient air is at a stand still, which is rare indeed! What really determine the spatial distribution of organisms are ecological processes represented by factors such as dispersal and migration, to mention a few. By considering a Malthusian population with diffusion, if

\[
\frac{\partial C}{\partial t} = D \frac{\partial^2 C}{\partial x^2} + \alpha C \tag{16}
\]

\( \Box \)
where $\alpha > 0$ is the renewal rate and $\alpha < 0$ represents the death rate or the intrinsic rate of growth. In the positive direction of $x$, we have

$$D \frac{\partial^2 C}{\partial X^2} + \nu \frac{\partial C}{\partial X} + \alpha C = 0$$

(17)

with solution given by

$$C = Ae^{\xi X}$$

where $\xi$ is the positive solution of the quadratic equation

$$D\xi^2 + \nu \xi + \alpha = 0$$

(18)

The physiologically meaningful solution is

$$\xi = \frac{\nu^2 + \sqrt{\nu^2 - 4\alpha D}}{2D} ; \quad \nu > 2\sqrt{\alpha D}$$

(19)

For $\alpha < 0$, equation (17) reduces to

$$D \frac{\partial^2 C}{\partial X^2} + \nu \frac{\partial C}{\partial X} - \alpha C = 0$$

(20)

while (19) takes the form

$$\eta = \frac{\nu^2 + \sqrt{\nu^2 + 4\alpha D}}{2D}$$

(21)

The general solution of (16) is obtained by a simple “jeu d’esprit” (see Burns et al, (2000), Dunning-Davies (1983))

$$C(x, t) = \frac{M}{2\sqrt{\pi Dt}} e^{(\alpha t - x^2 / 4Dt)}$$

(22)

If $\alpha$ represents the death rate, the concentration subsequently attains a maximum value. Though some of the pathogens are dying, the survivors are virulent enough to cause more infections and one therefore and: how do we control such a population?

Skellam (1951), Kierstead and Slobodkin (1953) obtained the following solution for $0 \leq x \leq L$

$$C(x, t) = A \sin \left( \frac{n\pi x}{L} \right) e^{(\alpha - \frac{n^2 \pi^2}{L^2})t};$$

(23)

($A$ is a constant, $n= 1,2,3,..$) which is oscillatory; for $n = 1$ if $\alpha > \frac{D\pi^2}{L}$, then the exponential term will increase indefinitely with time, and there will be an epidemic. This solution can be inferred directly from the solution of equation

8
by the method of separation of variables (see Weinberger, 1965). The zero solution is unstable and could therefore explain why SARS virus spread so fast and the introduction of a single case in the population could lead to an epidemic (any infectious being a source of proliferation of the virus). Kendall (1948) solved (22) with respect to \( \frac{x}{t} \) and obtained

\[
\nu = \frac{x}{t} = \pm 2\sqrt{\alpha D},
\]

asymptotically.

The density of pathogens is constant if \( \frac{x}{t} = \nu \) (equation (22), whence our earlier assumption \( X = x - \nu t \). But this is not the case because the ambiant air is a turbulent medium and, if the SARS vector flows freely in such a medium for a long time, then from (19),

\[
\frac{x}{t} = \nu > 2\sqrt{\alpha D}.
\]

This could explain why the spread of SARS is quite fast, even in the absence of “sufficiently close” contacts. Detailed data on virus ecology (environmental factors) is required in order to estimate \( \alpha \) and \( D \) independently.

Equation (25) can be interpreted as the population wave front from the initial disturbance propagates with a velocity ultimately greater than \( 2\sqrt{\alpha D} \). If \( 0 \leq x \leq L \), the habitat is confined and no diffusion to the surroundings takes place. Islands provide examples of this case and isolation (quarantine) of cases seems to be one of the best measures to prevent further infections, and the the vicities where a case has been found should be fumigated at regular intervals of time. Sudden appearance of cases far away from the Gwandong province of China may have occur through migration, because the virus cannot travel across oceans in so short a time.

2.2 Superposition method.

Since the blurring of inessential microscope details is one reason why the theory of a number of rather different phenomena is subsumed in a study of a few differential equation, this work is not an exception, for if there were no such natural unifying tendency, the mathematical study of various phenomena would be hopelessly fragmented.

The majority of scientific problems that can be treated by standard mathematical methods are linear. For linear homogeneous problems, the principle of superposition holds; that is, if special solutions to the problem are known, then a linear combination of such solutions yields new solutions.

The elementary or fundamental solution of equation (1) is a unit source solution, which gives the superposition amount of pathogens that diffuse out at time \( t \) to a location between \( x \) and \( x + \Delta x \). Since the diffusion process is the same no matter
where we fix our origin of space measurement and no matter when we start our
clock, $C(x - \sigma, t - \tau)$ represents the effect at point $x$ and time $t$ ($t > \tau$) of a source introduced at point $\sigma$ at time $\tau$. Hence

$$C(x, t) = C(x - \sigma_1, t) + C(x - \sigma_2, t)$$  \hspace{1cm} (26)$$

is a superposition of the effects of two sources one located at $\sigma_1$ and the other at $\sigma_2$. This agrees with our early assumption leading to equation (6). Equation (26) mimics the spread of SARS, since each case is also a source and the total density is the cumulative effects of all the sources. Airborne diseases are usually difficult to control, because each case needs special attention and should be handled with extreme caution. That is why biological weapons are very dangerous, should not be manufactured and if any, should be destroyed immediately with utmost care.

2.3 Migratory Effects

Determination of the basis of animal orientation and navigation during migration has proved to be a vexedly difficult problem (Okubo, 1980). Nevertheless, we look at possible migration of the virus and assume that the latter is chemically stable for the period of travel. In case there is any change (mutation), it is further assumed that the effect of the new pathogen is almost the same as the original one.

If the virus mixes uniformly with a depth of $h$ in the vertical direction, then the diffusion equation is Fickian and reads

$$\beta \frac{\partial C}{\partial x} = D \frac{\partial^2 C}{\partial y^2}$$  \hspace{1cm} (27)$$

where $\beta$ is the velocity of the wind, $(x, y, z) \in \mathbb{R}^3$ represents the cartesian coordinates.

Equation (27) is soluble and yields

$$C(x, y) = \frac{Q}{2h \sqrt{\pi Dx}} e^{-\beta y^2/4Dx}$$  \hspace{1cm} (28)$$

where $Q$ is the rate of emission at the origin $(x = 0 = y)$. When $h$ is small, there is a rapid increase in the proliferation of the virus and this could also explain why mosquitoes bites are elevated at low altitudes.

Since humans are perpetually in movement, we now consider a continuous emission from a moving source. Here, it is assumed that the release of viruses lasts for a sufficiently long time. The following advection-diffusion equation holds

$$\beta \frac{\partial C}{\partial x} = D \left( \frac{\partial^2 C}{\partial y^2} + \frac{\partial^2 C}{\partial z^2} \right)$$  \hspace{1cm} (29)$$

\[10\]
where diffusion in the $x$-direction is ignored (an assumption that is usually accurate). The solution of equation (29), is

$$C(x, y, z) = \frac{Q}{2\pi D_x} e^{-\beta \left(\frac{x^2+y^2}{4D_x}\right)} \quad (30)$$

By ignoring the effect of wind and turbulence, while still assuming isotropic diffusion of Fickian type, equation (29) now takes the form

$$\frac{\partial C}{\partial t} = D \left( \frac{\partial^2 C}{\partial x^2} + \frac{\partial^2 C}{\partial y^2} + \frac{\partial^2 C}{\partial z^2} \right) \quad (31)$$

with solution

$$C(x, y, z, t) = \frac{M}{(4\pi Dt)^{3/2}} e^{-r^2/4Dt}; \quad r^2 = x^2 + y^2 + z^2 \quad (32)$$

The graphical representation of (32) shows isoconcentration contours (spherical concentration) which are formed about the origin, and the concentration actually decreases linearly with distance from the source (Okubo, 1980). Hence, the immediate neighbours of an infected individuals are at high risk of infection. There is an obvious tendency in many communities of susceptibles who are situated a long way from infected persons to have a smaller chance of infection than those who are in closer contact with the disease. If the point source is located on the ground (e.g., mosquitoes breeding sites, SARS patients...), Caslaw and Jager (1959) obtained the solution on the semi-infinite strip $z > 0$ as

$$C(x, y, z, t) = \frac{2M}{(4\pi Dt)^{3/2}} e^{-r^2/4Dt} \quad (33)$$

which is twice the solution in infinite space given by (32). This confirms the fact that proliferation of viruses is high at low altitudes, whence the rapid rate of infection (SARS patients, who represent sources are located on the ground).

If we further assumed that humans migrate but viruses cannot, then the equation of population density for humans takes the form

$$\frac{\partial S}{\partial t} + \frac{\partial S}{\partial a} + \beta \frac{\partial S}{\partial x} = -\lambda S; \quad a \in \mathbb{R}^+, x \in \Gamma \subset \mathbb{R}^3 \quad (34)$$

where $S(a, x, t)$ represent the population density of individuals aged $a$, time $t$, located at point $x$; $\beta$ is the advective velocity. It was shown in Tchuenche (a) that a good estimate of $S$ is

$$\|S(a, x, t)\|_{L^1(\mathbb{R}^+ \times \Gamma)} \leq \frac{M}{|1-\mu|} e^{\frac{1}{1-\mu}-\lambda} t \quad (35)$$
where $\mu$ (the migration constant) is a threshold parameter since for $\mu > 1$, there is departure from endemic regions, say, while for $\mu < 1$, people arrive in region $x$. Immigration is not advisable and the health authorities are right to inform travelers to avoid traveling to any country where a higher scale epidemic is reported. Proper screening of people departing from the area should be done in order to minimize risk of infection in disease free zones.

3. Conclusion

Airborne diseases can be endemic in the community when the total number of susceptible individuals is replenished by births, and immigration. Traveling to such regions is risky. Overcrowding is also an important factor in the transmission dynamics of SARS and public places should be avoided as much as we can whenever a case is reported in a locality. New cases should be quarantined immediately while susceptibles should protect themselves by wearing the mask and remaining indoors if possible. One important question is how long can the virus reside in the ambient air?

We hope the above study has shed more light on the spread of airborne diseases as well as supporting the preventive measures put in place.

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