A simulation of Virus Diffusion

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Abstract

We introduce the "reaction-diffusion equation" that has potential to express growth of microorganisms, viruses. Instead of theoretical explanation of the equation, we introduce how to solve it in two or more dimensions on personal computers. Since numerical vectors of the equation results are not visual, we draw the time series changes to show where the reaction occurs in time-space.

Keywords: microbe, virus, SARS, COVID, reaction-diffusion

0. Introduction

Virus diffusion in the air is turbulent diffusion, unlike flux (heat, etc.) diffusion. In the body, it is contact and infiltration to body tissues, advection through body fluid tracts, and invasion into landed cells.

The diffusion of fine dust in the air can be described by the advection-diffusion equation. Since the size of virus is same size of fine dust, the behavior of virus population can be described by the convection-diffusion equation in the atmosphere.

In the body, if the virus keeps particle character, it could be described by the convection-diffusion equation.

However, when the virus invades the cell and proliferates, it must add a growth formula term. It is a reaction / convection-diffusion equation.

1. Theory

The phenomenon that RNA (ribonucleic acid) virus propagates and spreads in a human population is discussed based on the partial differential equation. When the genetic information is RNA, the probability of mutation is high [1]. Therefore, the mutation phenomenon must be considered in the proliferation equation.

The virus spreads depends on peoples' flow. Therefore, we make discussions based on a convection-diffusion equation (1). In the virus diffusion phenomenon inside human body, advection terms of the convection-diffusion equation has a large effect.

The properties of various organs inside human body affect viral replication. Originally, these should be taken into consideration. However, that consideration results in huge computational time. The adequacy of organ parameter settings also needs to be discussed.

In this paper, the proliferation coefficient is a spatiotemporal vector in the original expression, but after Eq.(2), it is simplified to a scalar coefficient. It's a simplification that is unavoidable with current PC specs.

$$\partial C(x,t)/\partial t=K(x,t)\partial^2 C(x,t)/\partial x^2+L(x,t)\partial C(x,t)/\partial x, \quad (1)$$

Here, C (x, t) is the amount of virus [numbers / unit volume], {x} is the n-dimensional spatial variable [unit length], {t, 0 <t} is the elapsed time [unit time], K (x, t) is the diffusion coefficient vector [none], and L (x, t) is the advection coefficient vector [none].

Equation (1) is L () = 0, K = const.,

$$\partial C(x,t)/\partial t=K\partial^2 C(x,t)/\partial x^2. \quad (2)$$

That is often solved. As an example: Thermal diffusion phenomenon. The general solution is,

$$C(x,t)=\eta\int_{[-\infty,\infty]}C(y,0)\exp\{(x-y)^2/(4Kt)\}dy,$$

$$\eta=1/\{2(\pi Kt)^{0.5}\}. \quad (3)$$

The solution saves the quantity of the initial value C (x, 0). If C (x, 0) = delta function, then C (x, t) is Gaussian [2].

The virus initially propagates according to the following equation.

$$\partial C(x,t)/\partial t=M\cdot C(x,t),\ M=\text{const.}>0. \quad (4)$$

However, the growth medium is finite. Therefore, in the long run,

$$\partial C(x,t)/\partial t=\{N-C(x,t)\}\cdot M\cdot C(x,t),\ N=\text{Medium volume}. \quad (5)$$

If M = 1 and N = 1, Eq. (5) gives a sigmoid function.

We link Eqs.(1 & 5), and get;

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$\partial C'(x,t)/\partial t = K(x,t)\partial^2 C(x,t)/\partial x^2 + L(x,t)\partial C(x,t)/\partial x$,
$\partial C(x,t)/\partial t = \{N(x,t)-C'(x,t)\} \cdot M \cdot C'(x,t)$, M=scalar constant. (6)

This is a "potential" equation that describes the phenomenon in which a virus propagates, spreads, and advects in a medium. This is one of the reaction-diffusion equations [3]. The theoretical solution of Eq. (6) is unknown. We divide the space-time by meshes, and we approach an approximate solution by simulation.

If the variant virus shares the growth resource (ribosome etc.) written N (), we get the following equation;

$\partial \underline{C'}(x,t)/\partial t = \{N(x,t)-C'(x,t)-C''(x,t)\} \cdot M \cdot C'(x,t)$,
$\partial \underline{C''}(x,t)/\partial t = \{N(x,t)-C'(x,t)-C''(x,t)\} \cdot M'' \cdot C''(x,t)$, $M'' \neq M$, (7)

Eq.(7; Cf. Reference [6] & appendix 1) is an equation that expresses the growth of two kinds of viruses. The advection-diffusion of 2 viruses are 2 equations independently. The equation can also be used to investigate the action of drugs that suppress virus production.

The actions of drugs directly and antibodies accessed to viruses are;
$\partial \underline{C'}(x,t)/\partial t = \{N(x,t)-C'(x,t)\} \cdot M \cdot C'(x,t)$,
$\partial \underline{C'''}(x,t)/\partial t = \rho C'(x,t)C''(x,t)$,
$C(x,t) = \underline{C'''}(x,t)$, ρ=scalar const. $\ll 1$. (8)
The formalism of "$\partial \underline{C'}()/\partial t = \cdots$" is not mathematical; this is algorithmic step-wise expression to calculate plural related equations.

2. Digitizing equations

2.1 Physical approaches

To discretize equation (6), we formulate physical diffusion and advection in 1,2-dimensions.

For diffusion; {C(0,t)=1}→{C(-1,t+1)=p, C(1,t+1)=p, C(0,t+1)=1-2p, 0<p<1}.
{C(0,0,t)=1}→{C(-1,0,t+1)=p, C(1,0,t+1)=p, C(0,-1,t+1)=p, C(0,1,t+1)=p;
C(-1,-1,t+1)=q, C(-1,1,t+1)=q, C(1,-1,t+1)=q, C(1,1,t+1)=q;
C(0,t+1)=1-4p-4q, 0<p<1, 0<q<1}. (9)
The K(x,t) term is expressed by {p,q} real numbers in Eq.(9). The "0" corresponds "x". The "1,-1" is neighbor mesh for "0th" mesh.

For advection; we get,

{C(0,t)=1}→{C(-1,t+1)=p, C(0,t+1)=1-p, 0<p<1} or
{C(0,t)=1}→{C(1,t+1)=p, C(0,t+1)=1-p, 0<p<1}.
[{C(0,0,t)=1}→{C(-1,0,t+1)=p, C(0,0,t+1)=1-p, 0<p<1} or
{C(0,0,t)=1}→{C(1,0,t+1)=p, C(0,0,t+1)=1-p, 0<p<1}] and
[{C(0,0,t)=1}→{C(0,-1,t+1)=q, C(0,0,t+1)=1-q, 0<q<1} or
{C(0,0,t)=1}→{C(0,1,t+1)=q, C(0,0,t+1)=1-q, 0<q<1}]. (10)
Those (p,q) values are expressed L(x,t) in Eq.(6). There is diffusion due to turbulence in the flow. We substitute it with uniform random numbers (expectation 0).
{p, q}→{p+ρ × random#, q+ρ × random#}, $\rho \sim 0.03$. (11)

Now consider the conservation mass. In the field of physics, "div (C) = 0", if there is no "welling point" in space. "div()" is the divergence operator of vector analysis. When simulating a non-reactive tracer such as dust diffusion in the atmosphere with the advection-diffusion equation; we impose the condition of "div()=0", normally.
In the reaction-diffusion equation, there are the "welling points" everywhere in space. Already, addition of the random number terms in equation (11) violates the div = 0 condition. We suppress the violation by expectation zero. The diffusion part has the conservation property.

For virus propagation,
C(x,t+1)=C(x,t)+{1-C(x,t)} · M(x,t) · C(x,t), N=1.
C(x,y,t+1)=C(x,y,t)+{1-C(x,y,t)} · M(x,y,t) · C(x,t), N=1. (12)
The M(x,t) and M(x,y,t) are expressed as M, in Eq.(6). A uniform scalar constant M is sufficient, except for viral species that propagate inside special internal organs.

2.2 Algorithm

When the space is 2 dimensions, we let an integer set (i, j) be the discretized position coordinates of the {x, y} plane.

2.2.1 Definition of the partial differential operator.
We write the adjacent coordinates as; im=i-1,ip=i+1; jm=j-1, jp=j+1. We write the diffusion as followings;
{Mass 1 on C(i,j,t)} → {Mass p on C(i,jm,t+1),
C(im,j,t+1), C(ip,j,t+1), C(i,jp,t+1);
Mass $p/\sqrt{2}$ on C(im,jm,t+1),
C(ip,jm,t+1), C(im,jp,t+1), C(ip,jp,t+1);
Mass $(1-4p-4p/\sqrt{2})$ on C(i,j,t+1)},
$0<p\ll 1$. (13)
If the diffusion rate P (k, L) is 0 at a movement position (k, L), that is, if the place cannot be moved, the movement to

that position is stopped. The total amount of movement is reduced by the contribution. Thus, P () values determination for 8 moving positions are required at all discrete points on the plane.

2.2.2 Definition of the partial differential operator for the time step.

We define the operation as;

$C(i,j,t+1)=C(i,j,t)+\{1-C(i,j,t)\}MC(i,j,t)$, M=scalar constant. (14)

The definition is valid on $P(i,j) \neq 0$. We can introduce an expansion, $M \rightarrow M(i,j,t)$. Such an expanded form may be useful for cancer cell proliferation simulation inside human body.

When two viruses {C (), Cm ()} compete for the same cultured ribosome,

$C(i,j,t+1)=C(i,j,t)+\{1-C(i,j,t)-Cm(i,j,t)\}MC(i,j,t)$, M=scalar constant,

$Cm(i,j,t+1)=Cm(i,j,t)+\{1-C(i,j,t)-Cm(i,j,t)\}Mm \times Cm(i,j,t)$, Mm=scalar constant. (15)

If $\{1-C(i,j,t)-Cm(i,j,t)\}<0$, we force the value to be 0.

When administering the growth-suppressing drug V (), which does not act directly on the virus,

$C(i,j,t+1)=C(i,j,t)+\{1-C(i,j,t)-Cm(i,j,t)-V(i,j,t)\}MC(i,j,t)$, M=scalar constant. (16)

2.2.3 Appearance of mutants.

At the specified time t=T and position (I, J), at the ratio of $0 < \sigma << 1$,

$Cm(I,J,T+1)=\sigma C(I,J,T)$, $C(I,J,T+1)=(1-\sigma)C(I,J,T)$. (17)

Usually, we adopt, M<Mm. (18)

3. Results of numerical simulations

3.1 Definition of the space.

We use 2 dimensional square space that is {x,y}.

Where “x” is [-200,200; integer], whose structure has the square mesh. The “y” direction has same mesh structure.

We set 2 square boxes in the space.

The boxes have centers (-100,0), (100,0). The side length is 98.

The two boxes are connected by one corridor whose width is 10, and the length is 4.

The K(x,t) value inside the boxes is uniform, “0.1”. The K value outside the box is 0 (virus does not spread to the outside).

It is assumed that the virus of "1" is present in one mesh-area at the center of left-box and "t = 0". This is the initial condition. See our paper on atmospheric images for visualization please, of the results [4]. Hereinafter, the brightness of each point of images is set in the range of [10,255]. The physical quantity and brightness of each point have a linear relation.

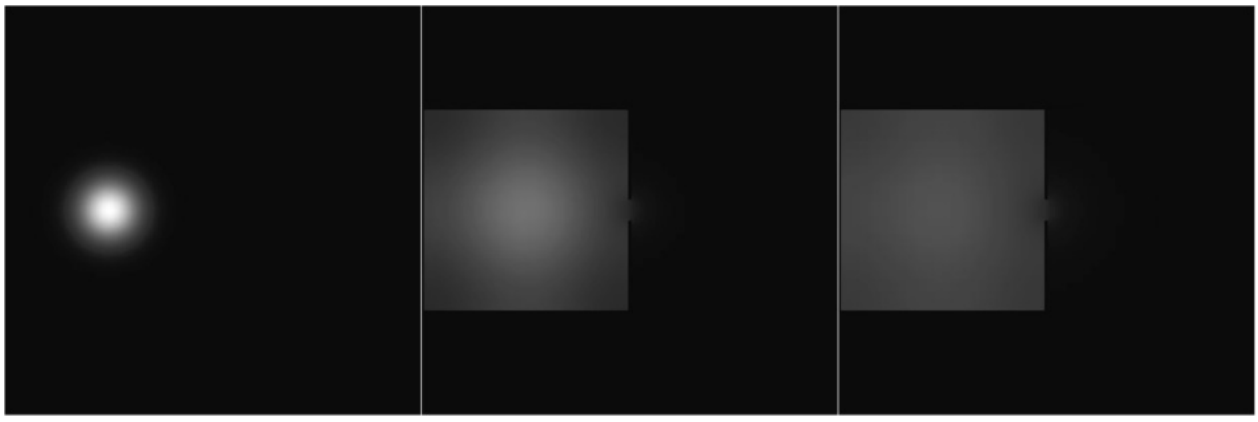

Figure 0: The maximum brightness of 3-images are 8.2, 4.2, 3.1×10^{-5}, respectively. Since there is no proliferation, the virus density is reduced by spreading. At t=8/12k, the virus has passed through a corridor and moved to right-box. It will be dark and unclear in printed matter.

3.2 Test0: the case of diffuse only.

We show the results; They are 3 images, which are virus density at "t = 4,8,12k, k=1000".

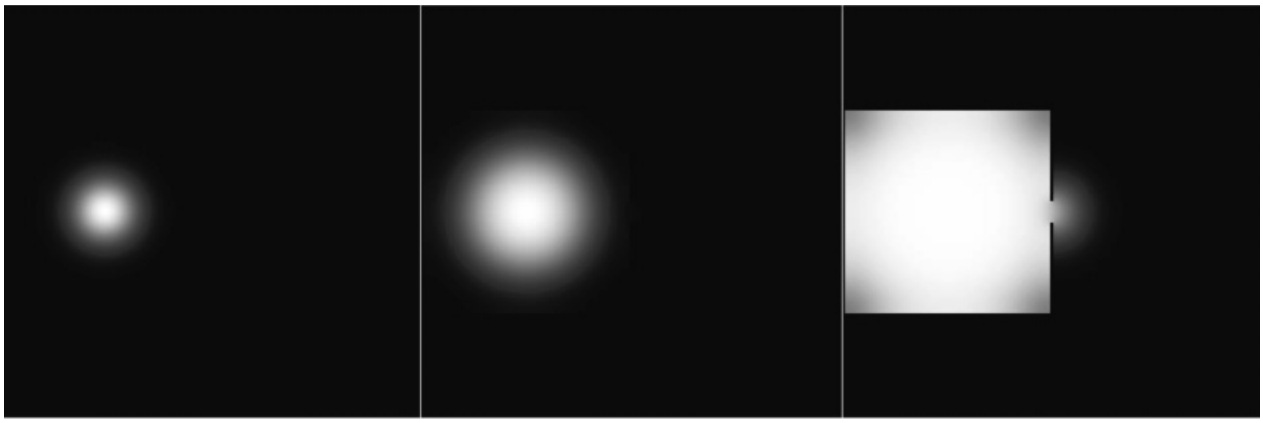

Figure 1: The maximum brightness of 3-images are 0.046, 0.77, 1.0, respectively. Each snap shot is at t=1, 2, 3k.
It is remarkable difference between plain- and reaction-diffusion systems.

3.3 Test1: the case of diffuse and proliferation.

We set M=0.005 of Eq.(6) under the same condition of section 3.2. Proliferation speeds up the development of the system, so we stop the simulation-time at 3k.

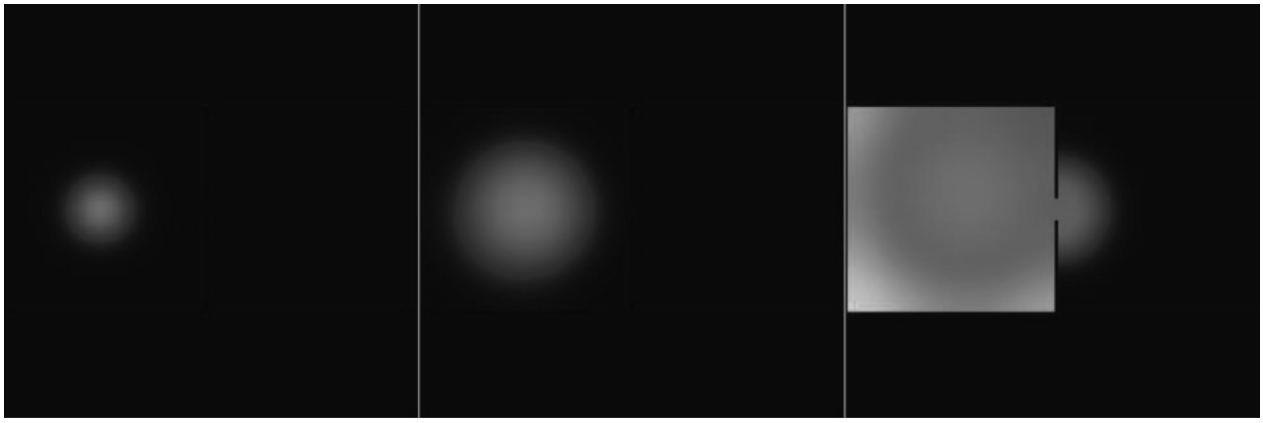

Figure 2: The maximum brightness of 3-images are 0.046, 0.75, 0.93, respectively. Each snap shot is at t=1, 2, 3k.
The mutation location is (-110,10), 10 mesh from the center, southwest direction. The mutation ratio is 0.05.
Old species is displayed by red dots, and the new one is green.
Mutants occur at time t = 200, but at t = 1,2k, their existence is hidden behind the old species and it is not visible.
The existence of a new species can be confirmed at t = 3k. Through a narrow corridor, the new species will grow twice as fast as the old ones, so they will prevail in the 2nd BOX. The simulation shows spread of different types of viruses in different countries. It shows that the number of virus strains that grow faster gradually increases.

3.4 Test3: the case of diffuse, proliferation, and mutation.

We check a case where a virus mutates, and two types of viruses coexist and multiply.

Mutation time is t = 200. Characteristics of new species:

1. Requires the same resources as the old one.

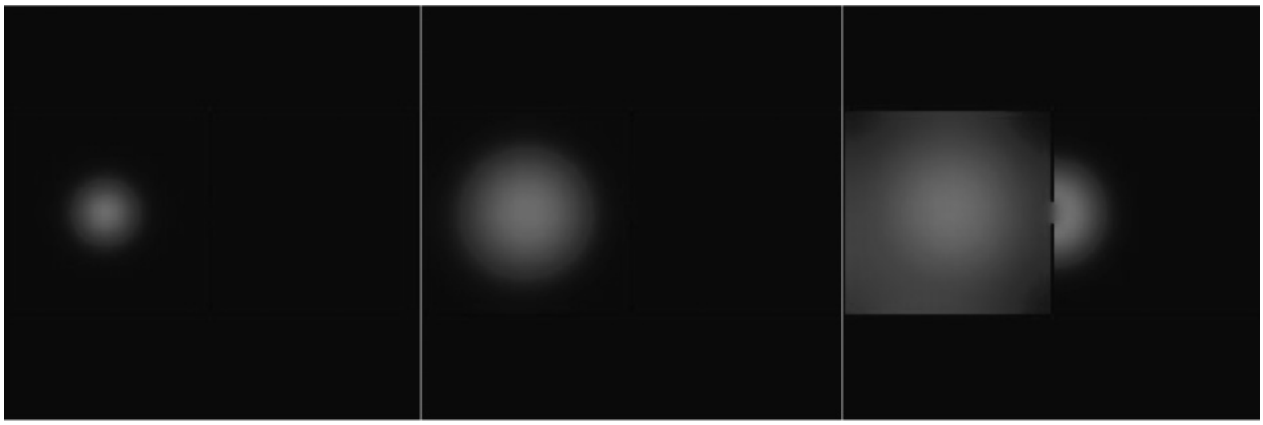

Figure 3: The maximum brightness of 3-images are 0.046, 0.62, 0.58, respectively. Each snap shot is at t=1, 2, 3k.

The drug administration time is t = 1.5k, and the injection-mass per time-steps is linear. The injection is left-BOX only, and the density is homogeneous. Set to consume 50% of the viral replication resource after 0.5k time-steps, and stop administration when 90% of the resource is consumed.

By administration of the drug, virus growth in the left BOX is suppressed. This is reflected in the maximum brightness value (0.93 → 0.58). From the color of distribution, it is effective against the fast-growing mutation virus. Since there is no drug administration in right side BOX, new species (green dots) are growing fast.

This result is acceptable because Favipiravir does not have the ability to break down virus; it cannot eliminate virus grown before administration.

2. The M value of proliferation is doubled (Mm = 2 * M).

The result is in Figure 2.

Figure 4: The maximum brightness of 3-images are 2.2, 2.4, 2.3, respectively. Each snap shot is at t=4, 8, 12k.
The mutation was assumed to occur 5% at t = 200, position (-143, -52). The growth rate of the mutant species is twice that of old species. The flow velocity is 0.05, that is 500 times higher than the diffusion rate ($P=10^{-4}$), so old species in the flow path are not soon replaced by new species.
From the channel, it is visualized that the new virus infiltrates into the tissue due to its rapid proliferation.

3.5 Test4: Presence of drugs that suppress viral growth.
Several mechanisms are known to suppress viral replication. The virus synthesizes its own protein using ribosome, a protein synthesis body of the host cell. Favipiravir is a drug that employs a method which inhibits the mechanism. On our simulation, virus growth is expressed by competing growth-resource with virus and the drug. We get followings.

Figure 5: The maximum brightness of 3-images are 3.8, 5.5, 7.2, respectively. Each snap shot is at t=4, 8, 12k.
The mutation was assumed to occur 5% at t = 200, position (-143, -52). The growth rate of the mutant species is twice that of old species. The flow velocity is 500 times higher than the diffusion rate. And uniform number disturbances (ρ =0.03) are added per t=10 (total 1.2k times).
As in the previous section, under the disturbance flow; old species in the flow path are not soon replaced by new species. From the channel, it is visualized that the new virus infiltrates into the tissue due to its rapid proliferation.
We can know the difference of virus species in tissue and channel.

3.6 Test5: Introduction of advection term.
We set an outer frame that surrounds the space with four sides of the 401×1 [dots] mesh.
The virus does not invade the frame. The inside is a closed space completely, and it has K(x,t)=0.0001, homogeneous virus diffusion area.
Inside that space, we define a rectangular area, outside 301×121, and inside 281×101 meshes.
We define that there is a flow of L (x, t) = 0.05 counterclockwise in this hollow space surrounded by a rectangle with a width

of 10 meshes. There is a closed, 10-mesh channel, where there is a flow and diffusion phenomenon.
There is one unit amount of virus at one point of the flow path, the position (-145, -55). We make it the initial state.

Figure 6: The maximum brightness of 3-images are 3.8, 4.9, 5.4, respectively. Each snap shot is at t=4, 8, 12k.
The mutation was assumed to occur 5% at t = 200, position (-143, -52).
The drug is injected from t=4k, and the position (0,-55), one point in the central in low horizontal channel. The injection mass is 0.1 per every time steps, total mass is 800. The drug-diffusion rate is twice for the virus ($P(i,j)=10^{-3}$, that is 10 times larger than that of Figure 5; To make the drug more effective).
The drug reduces the viral load in the tissue, but does not reduce the virus in the channels. The image of channel is relatively bright compared with the tissue; virus is high density. Especially at the corners of the channel, you can see a particularly dense virus colony. It is known that coronavirus damages to the vascular system. Those indicates that administration of growth inhibitors after inflammation is less effective.

3.7 Test6: Advection and stirring under uniform random numbers.
A uniform random number in [-0.03, 0.03] is added to the two-dimensional flow in the x and y directions. The random number addition repeats per t=10, where the additive effect closes to 0. Thus; the direction of the flow does not change, but the flow is disturbed.

Figure 7: The maximum brightness of 3-images are 3.8, 4.9, 5.4, respectively. Each snap shot is at t=4, 8, 12k.
The mutation was assumed to occur 5% at t = 200, position (-143, -52).
The drug is injected from t=4k, and the position (0,-55), one point in the central in low horizontal channel. The injection mass is 0.1 per every time steps, total mass is 800. The drug-diffusion rate is twice for the virus ($P(i,j)=10^{-3}$, that is 10 times larger than that of Figure 5).
After t = 8k, a dark shadow without virus and inflammation can be seen on the right side of lower flow path. The shadow is the effect of antiviral drugs.

3.8 Test7: Suppressing drug under stirring advection.

Drugs that interfere with virus production are known to be less effective against the coronavirus.

From the simulation of section 3.5 with only the diffusion effect, in the initial stage of high-speed propagation and low viral

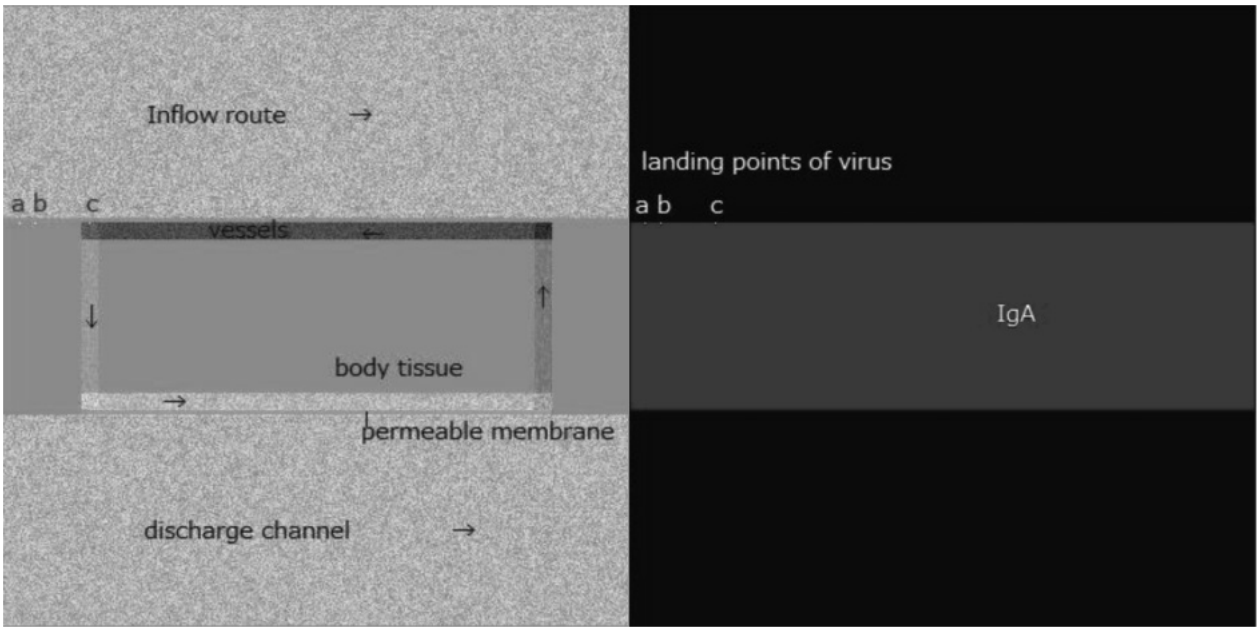

Figure 8: Left: A model of human body; Right: Early virus infection location and distribution of IgA.

The position coordinates in the left and right figures have a one-to-one correspondence.

This model consists of 3 parts (outside-air input, body tissue, and exhaust parts). There is a flow (0.05 [unit speed*]) from left to right in the input/exhaust parts, and there is random number disturbance of expectation 0, section [-0.03,0.03], uniform.

*) definition of the speed: It is expressed as the ratio of mass that moves by one mesh. The "0.05" indicates that 5% moves to the adjacent mesh, where "1" is the mass present in one mesh.

load; If this drug is administered, it will be effective.

That is, it is effective if the drug is spread in the tissue in advance.

On the other hand, as in clinical practice, even if a growth inhibitor is administered after inflammation occurs, new growth can be suppressed, but the virus that has already dispersed and has an adverse effect on tissues cannot be removed. We can't expect the effect.

3.9 Test8: Drug for degrading virus

Currently, such drugs are not found, which directly break down SARS virus. Assuming that the drug has been developed, we simulate it. We adopt a virus decomposition formula, C(x,t+1)=C(x,t)-B, B=0.2*C(x,t)V(x,t). Cf. Appendix 1, Eq.(21 & 22). Where C() is mass of virus, and V() is mass of the drug on each mesh. "0.2" is the ratio of break down.

3.10 Test9: Innate immunity and the virus

Humans have innate immunity (Immunoglobulin A: IgA) originally [5]; under that situation, we simulate the virus propagates. We adopt a following human model.

The body tissue has a thin boundary portion-1 in contact with the input part and a thin boundary portion-2 in contact with the exhaust part. Each portion has 0.001, and 0.01 [unit speed], respectively. Potion-2 has 10 times than that of portion-1. The boundary is assumed to be a permeable membrane with a large diffusion coefficient. There is a flow path of body fluid so as to be in contact with the permeable membrane. The body fluid flow path circulates at a constant rate and there is turbulent diffusion. With this setting, we model the function of discharging foreign substances in body fluids.

The flow of tissue part is 0.05 in both X and Y directions. There is no turbulence.

The flow of input/exhaust parts are 0.05 in X direction with turbulence of 0.03. They have no flow for Y direction, but have Y-

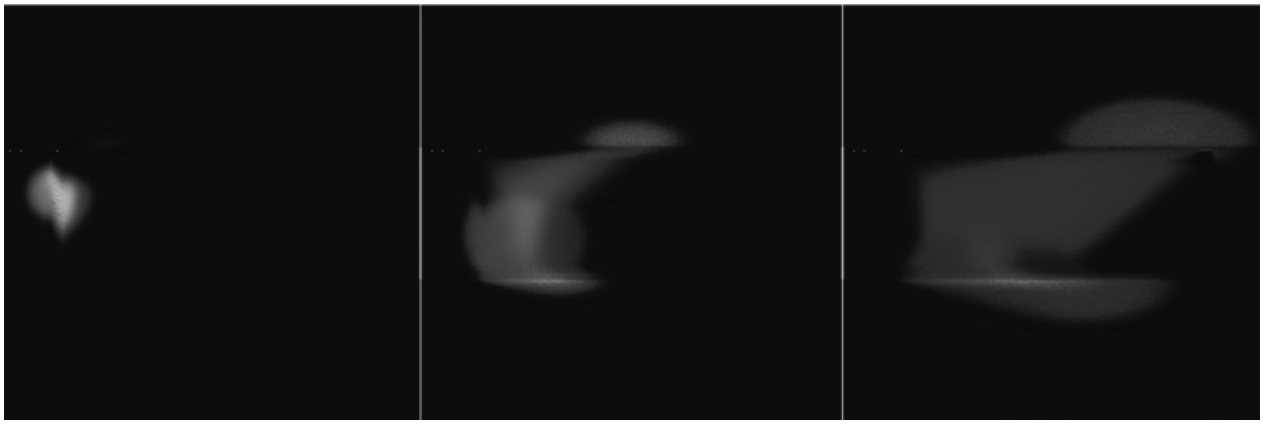

Figure 9: Virus distribution at t=2, 4, 6k.
Initial state of virus distribution:
a: +10 mesh position from the left end, t = 0. The virus distribution that has been advected and spread from a-position is displayed in red.
b: Exists from +20 mesh position from the left end, t = 0.2k. The virus distribution is displayed in green.
c: Exists from +55 mesh position (central part of body fluid tract) from the left end, t = 0.6k. The virus distribution is displayed in blue. The virus species at position a~c are the same.
Relationship between distributed intensity and brightness of each color: The square root of intensity is affine-transformed into the [10,255] interval. Therefore, the low-virus-mass distribution appears to be emphasized.

direction's turbulence of 0.03.

The diffusion coefficient of input- and tissue parts are 0.001; and the exhaust part is 0.003.
The width of fluid transport channel is 10 mesh, and the direction of flow is indicated by arrows in figure-9. The speed of transport route is 0.05 and there is a random number disturbance of 0.03.

The innate immunity (IgA) concentration is 0.00001 [unit concentration] in the blue part of the figure on the right. Initial concentration of the virus is 1; so it is sufficiently dilute of IgA. Moreover, we set that the effect of eliminating virus is reduced to 5% of the multiple value among virus and IgA concentration. The spread of IgA is 0.002 (twice the spread of virus in tissues). This setting makes IgA's virus-suppressing effect is very small. If it is enough, the virus does not multiply and the

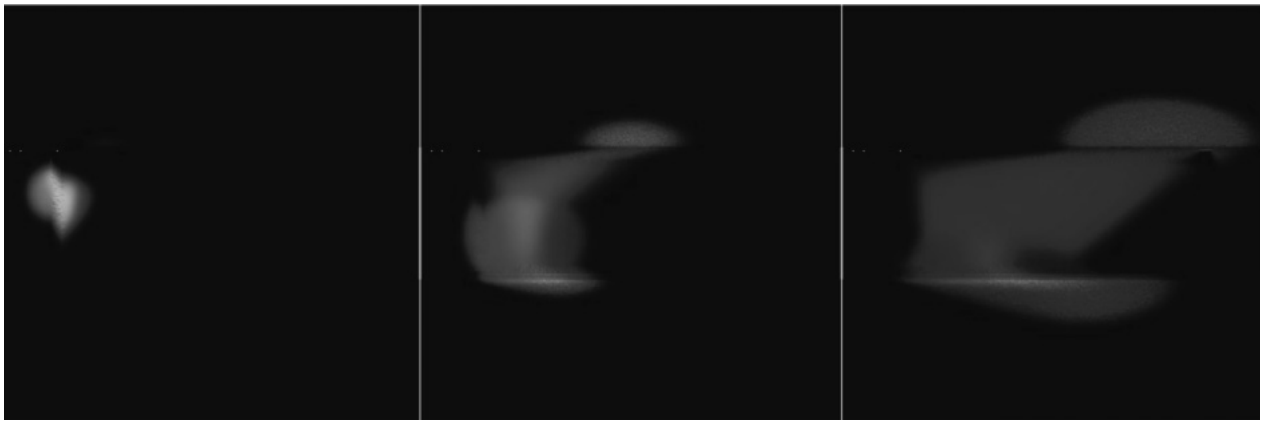

Figure 10: Virus and fragment's distribution at t=2, 4, 6k.
Initial state of virus distribution (see Figure 8):
a: the virus landing point is not used.
b: Exists from +20 mesh position from the left end, t = 0. The virus distribution is displayed in red.
c: Exists from +55 mesh position (central part of body fluid tract) from the left end, t = 0.4k. The virus distribution is displayed in green. The virus species at position b and c are the same.
Concentration of virus fragments: they are displayed by blue. The intensity is accumulated until the simulation end.

image becomes a dark figure.

The virus landing time differs at points (a, b, c), but the spread of body tissues is nearly. Landing on the body fluid transport route reverses the delay of ~600 simulation step time. Viruses are exhausted from both the input and output sides. Coronavirus attacks ACE2 (Angiotensin-Converting Enzyme 2) and invades host cells. Since ACE2 is found in various parts of the body, it is judged that the diffusion rate differs depending on the invading part if it is far from the body fluid transport path. So we did the test.

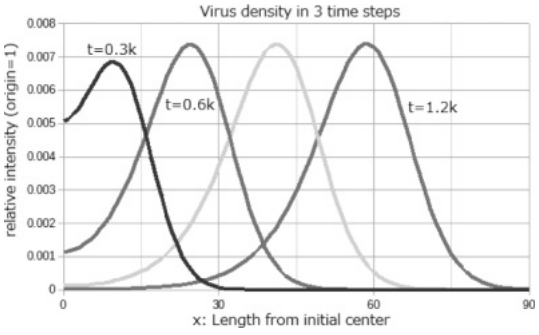

Figure 11: Virus density in 3 time steps per 300 simulation steps.
Virus density is relative intensity for unit=1 at initial origin.

This result indicates that the virus propagates outward from the origin as an increasing Gaussian-like wave.

We calculate the diffusion of the virus into space, by using 1-dimensional reaction-diffusion equation.

3.11 Test10: Traces of virus fragments.
On test 10, under enough concentration of IgA, the trace of virus fragments are visualized.
We set the innate immunity (IgA) concentration is 0.01 [unit concentration], and the effect of eliminating virus is 25% of the multiple value among virus and IgA concentration. Thus, virus decomposed effect is 5000 times larger than that of test 9.
Relationship between distributed intensity and brightness of each color: The square root of intensity is affine-transformed into the [10,255] interval. Therefore, the low-virus-mass distribution appears to be emphasized. The emphasis method is same as figure-9.

In test10, it can be seen how virus debris fragmented by the IgA antibody is transported to output channel.
With sufficient IgA antibody, even if the virus spreads, the concentration is not high; hence the concentration of debris is high.

4. Quantitatively virus diffusion

5. Conclusion

We introduced the "reaction-diffusion equation" that has the potential to express the growth of microorganisms, especially viruses. Instead of a mathematical and theoretical explanation of the equation, we introduced how to actually solve it in two or more dimensions. Since solutions of the equation were not clear on numerical indices, we drew the time series to show where the reaction occurs in space.

What was shown in our simulation,

(1) If the parameters are selected appropriately, the diffusion and suppression of virus can be visualized.

(2) A drug that suppresses virus production is ineffective unless it is present at a high density in the space-time of the production site. If the drug is administered in high concentrations, it can be effective in suppressing the virus. It implies an adverse effect on protein synthesis mechanism of normal human cells.

The parameters have been freely set in order to fit the simulations.

The units is not the units in real world, where units are determined biophysicaly.

Results of the paper show the possibility only, that is, such phenomenon would be happened, within a country, or within a human body.

We do not consider the behavior of persistently infected viruses.

The published PDF will be a monochrome image. The original drawings are color images. I publish them on my blog.

6. References

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[6] Yoshihiro KAWAOKA editted, “Neo-viral science (in Japanese)”, Syueisya co. ltd., 2021.3.22, USBN 978-08-721159-7 C0240;

Shingo IWAMI, “Numerically prove virus characters that cannot be shown in experiments (in Japanese)”, pp.276-287.

Appendix 1:

The following equation is known for the proliferation of hepatitis C virus.

$$dT(t)/dt = -\beta T(t) V(t), \quad (20)$$

$$dI(t)/dt = \beta T(t) V(t) - \delta I(t), \quad (21)$$

$$dV(t)/dt = pI(t) - cV(t). \quad (22)$$

Reference [6] does not explain each item, but it is inferred from the characteristics of the virus.

T (): Amount of resources for viral growth in host cells,

I (): Amount of virus present in cells,

V (): The amount of virus present outside the cell.

{β, δ, p, c} is a parameter.

Characteristics of the virus described by equations (20-22):

(1) There are two types of viruses, {I, V}, which are described by continuous functions. We have introduced analogism into the genetic information, that is, we recognize the contribution of peripheral molecular groups to the functional expression of the genome. It closes to the concept of epigenetics.

(2) The {-δI (t), -cV (t)} term of Eq. (21,22) indicates the spontaneous degradation of the virus. There is no discussion of this mechanism.

(3) The sign of the βT (t) V (t) term is opposite in equation (20,21), and there is no such term in equation (22).

The above speculation was deduced from this feature.

Eq.(7) and Eq.(20) are equivalent. The addition of (21 & 22) can be introduced as an extension of equation (7).

Equation (20-22) is equations that express the phenomenon that 3 quantities {T (), I (), V ()} spontaneously collapse when viewed on a long-term scale. Therefore, there is no conserved quantity.

付録： 概要・結論

微生物、特にウイルスの増殖を表現する可能性のある「反

応拡散方程式」を紹介します。 方程式の数学的および理論的な説明の代わりに、2次元以上で実際に解く方法を紹介します。 方程式の解は数値指数表示では明瞭でないため、時系列を描画して、反応が空間のどこで発生するかを示します。

本シミュレーションで示されたこと、

(1)

パラメータを適切に選択すれば、ウイルスの拡散と薬剤の抑制を可視化できる。

(2)

ウイルス産生を抑制する薬剤は、産生場所の時空間に高密度に存在しないと効果がない。薬剤の高濃度投与は、正常なヒト細胞のタンパク質合成機構に影響を及ぼすことを意味する。

本論文は反応・拡散方程式の生物学への応用可能性のみを示した。シミュレーション・パラメータの正当性については検討していない。