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RESEARCH ARTICLE

A Causal (Regression) Analysis for Cancer Formation under a Corresponding Virus Attack

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ABSTRACT

Cancer or COVID-19 virus can be serious harmful diseases to our human beings. However, by understanding how these diseases will be formed, we may construct the corresponding statistical causality regression model or even the artificial intelligence one. This may help us develop the corresponding drugs or vaccine. Indeed by applying the techniques as stated in one of my paper, one may even control the mutation or the evolution of the cancer or the COVID-19 virus whenever the Lorenz attract or the Butterfly effects existing during the virus mutation process. This may be a breakthrough in the history of fighting against our human beings' nature enemy — virus.

Key words: Cancer, virus, attack, methylation

BACKGROUND

Cancer is the name given to a group of serious diseases that humans have long tried to cure. Indeed, the latest and most advanced research shows that different viruses play a significant role in the formation of various cancers. In the following, I shall outline a causal (regression) analysis[1] for cancer formation under a corresponding virus attack. According to Zheng *et al*.,[2] alcohol can induce hepatitis C virus (HCV)-hepatocellular carcinoma that causes the DNA methylation of repetitive elements. These elements may include long interspersed nuclear element-1 and All element (Alu). Zheng *et al*.[2] further concludes that HCV infection is highly connected with the loss of DNA methylation in specific REs, implicating molecular mechanisms in liver cancer development.

In vest gating DNA methylation in more depth, Cho *et al*.[3] finds that tobacco smoking may change the transcription and methylation states of extracellular matrix organisation-relatedgenes. It is worth noting here that tuberculosis may increase the risk of lung cancer, which is highly related to smoking. In such a case, DNA methylation patterns will change, altering the tran-scriptionstates of genes. To be more precise, proteins will bind to methylated DNA. This DNA will then form complexes with the proteins that formulated during the process of deacetylated histones Therefore, when DNA has undergone the process of methylation, the nearby histones will also deacetylated, resulting in compounded inhibitory effects on transcription. Similarly, demethylated DNA will not cause the attachment of deacetylated enzymes to the histones, allowing DNA to deacetylated and more mobile, thus promoting transcription.

Next, when transcription indirectly caused by the virus takes place, cellular lipidomics may occur (Chakrabarti *et al*.,).[4] As a result, this could lead to lipid metabolism and cause metabolic health issues. Snaebjornsson *et al*.[5] explains that by altering lipid metabolism, one may even de-velop a possible therapeutic window for cancer treatment. Thus, with reference to my paper in causal (regression) analysis, as shown in,[1] one can deduce the following causality for the possible for motion of cancer:

Virusinfection→DNAmethylation→transcriptionoccurs→lipidomics→metabolicproblem→Cancerformation

Using suitable bio-informatics cancer data, one can follow the procedure as stated in[1] to establish

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a Hayes process model[for just such a virus can conformation mechanism. Specifically, one can perform heterogeneous analysis, together with spatial analysis, to determine the cancer for motion mechanism.[6] Indeed, other virus infection mechanisms, such as COVID-19, can also modeled using the same or similar methods, as long as the details of the infection can be ascertained from cellular experiments.

According to Gourley *et al*.,[7] one can employ the following phenomeno logical model for the hepatitis B virus:

$x'(t)= \lambda - dx - \beta\nu x$ equation (A)

$y'(t)= \beta\nu x - ay$ equation(B)

$\nu'(t)=ky-\mu\nu$ equation(C)

Where x, y, and ν are the numbers of uninfected liver cells, infected cells, and free virions, respectively represent the (constant) rate where the uninfected liver cells are produced;and dx represents the line a term of maintaining tissue homeostas is in the face of hepatocyte turn over.

During infection, healthy liver cells are assumed to become infected at a rate βνx, where β is the mass action rate constant describing the infection process. Infected liver cells are killed by immune cells at rate ay and produce free virions at rate ky, where k is the so-called 'burst' constant. Free virions are cleared by lymphatic can do the mechanism satrate μν, where μ is a constant

MAJOR RESULTS

Mathematically, such a system of differential equations can be made to interact with my HKLam theory.[8] and, in terms of the Lorenz differential system (or other differential equation models such as SERI),can be expressed in the form of a matrix. In addition, it is feasible that these matrices are capable of being written using HKLam's (net-seizing) theorem. The following is an example:

$$\begin{pmatrix} -6 & 6 & 0 \\ -z+\rho & -1 & 0 \\ 0 & x & \beta \end{pmatrix}\begin{pmatrix} L \\ T \\ 1 \end{pmatrix}$$ = Linear regression of causal domino (LRA 1) (equation 1)

$$\begin{pmatrix} -6 & 6 & 0 \\ -z+\rho & -1 & 0 \\ 0 & x & \beta \end{pmatrix}$$ = Linear regression expression (LRE 2) (equation 2)

$$(\text{LRE } 2)\begin{pmatrix} L \\ T \\ 2 \end{pmatrix} = (\text{LRA } 1)$$ (equation 3)

Substitute $\begin{pmatrix} L \\ T \\ 2 \end{pmatrix}$ in equation (3) back into equation (1) and continues this (in fact which is initially an infinite and) recursive process (such that this is a kind of mathematical formalism) until the linear transformation $\begin{pmatrix} L \\ T \\ 2 \end{pmatrix}$ is found in the pre-calculated optimal approximation for a given set of values (σ1, þ1, β1) in the Lorenz attractor. In this case, we have got the wanted real values of the linear transformation $\begin{pmatrix} L \\ T \\ 2 \end{pmatrix}$ at that a particular given set of valued point; say (σ1, þ1, β1). Conversely, if there are sufficiently large amount of $\begin{pmatrix} L \\ T \\ 2 \end{pmatrix}$ s, we can estimate back the corresponding true values of (σ2, þ2, β2) and get the optimal values in the Lorenz attractor. Hence, in terms of weather management, we can determine the consequence risks behind. Thus, we may associate with the most feasible warnings and give in advance by applying some suitable decision theories. In such a case, we are actually using the HKLam Theory to net-seize those changes in our earth-weathering butterfly effect (or the Lorenz attractor). Similar cases happen in other differential equation models such as in the viruses mutation in micro-biology (cancer research) together with the spread of other viruses (influenza and COVID-19 for example) in our public health.

Take for some case studies, lasts consider a set of values for (σ =10, þ, β = 8/3, z = 1, x = 1) and interact with one of the regression model equation in Lam,[1] we may get:

$$\begin{pmatrix} -10 & 10 & 0 \\ -1+\rho & -1 & 0 \\ 0 & 1 & 8/3 \end{pmatrix}\begin{pmatrix} L \\ T \\ 1 \end{pmatrix}$$ = (4.3685.53 +

26.71*wind - 2.185 *wettest -2054.05 * temperature equation (1')

Approximate the matrix by a linear regression, we also have:

$$\begin{pmatrix} -10 & 10 & 0 \\ -1+þ & -1 & 0 \\ 0 & 1 & 8/3 \end{pmatrix} = AX + \varepsilon_0 \quad \text{equation (2')}$$

Thus, by equating the above two equations, we get:

$$(AX + \varepsilon_0)\begin{pmatrix} L \\ T \\ 2 \end{pmatrix} = (4.3685.53 + 26.71*\text{wind} - 2.185$$

*wettest -2054.05 * temperature; or

$$\begin{pmatrix} L \\ T \\ 2 \end{pmatrix} = (AX + \varepsilon_0)^{-1} * [(4.3685.53 + 26.71*\text{wind} -$$

2.185 *wettest -2054.05 * temperature]

equation (3')

Let B = (4.3685.53 + 26.71*wind - 2.185 *wettest -2054.05 * temperature

$$\begin{pmatrix} -10 & 10 & 0 \\ -1+þ & -1 & 0 \\ 0 & 1 & 8/3 \end{pmatrix} [(AX + \varepsilon_0)^{-1} * B] = B$$

$$\begin{pmatrix} -10 & 10 & 0 \\ -1+þ & -1 & 0 \\ 0 & 1 & 8/3 \end{pmatrix} = B * [(AX + \varepsilon_0)^{-1} * B]^{-1}$$

Continue the above process for the second time, we get:

$$B * [(AX + \varepsilon_0)^{-1} * B]^{-1}\begin{pmatrix} L \\ T \\ 3 \end{pmatrix} = B$$

$$B * [(AX + \varepsilon_0)^{-1} * B]^{-1} = A'X + \varepsilon'_0$$

$$(A'X + \varepsilon'_0)\begin{pmatrix} L \\ T \\ 4 \end{pmatrix} = B$$

$$\begin{pmatrix} L \\ T \\ 4 \end{pmatrix} = (A'X + \varepsilon'_0)^{-1} * B$$

$$B * [(AX + \varepsilon_0)^{-1} * B]^{-1} * [(A'X + \varepsilon'_0)^{-1} * B] = B$$

$$B * [(AX + \varepsilon_0)^{-1} * B]^{-1} = B * [(A'X + \varepsilon'_0)^{-1} * B]$$

$$\begin{pmatrix} -10 & 10 & 0 \\ -1+þ & -1 & 0 \\ 0 & 1 & 8/3 \end{pmatrix} = B*[B * [(AX + \varepsilon_0)^{-1} * B]^{-1} * B]$$

$$= B^2 [(AX + \varepsilon_0)^{-1}]^{-1}$$

if we assume $(A*B)^{-1} = B^{-1}* A^{-1}$ and the communicative, distributive properties of matrix multiplication.

If furthermore, the Lorenz matrix can be QR decomposed, then we may have:

$$\begin{pmatrix} -10 & 10 & 0 \\ -1+þ & -1 & 0 \\ 0 & 1 & 8/3 \end{pmatrix} = B^2 [(QR)^{-1}]^{-1}$$

The above sample Lorenz matrix (with assumed parameters) shows that we can always express it in the formative and recursive format using linear regression approximation.[9]

In general, formative recursion computing can be solving using a five-step process:

1. Find out the simplest possible input
2. Visual is and play around with examples
3. Relate hard cases to simpler cases
4. Generalize the pattern
5. Write computer code combining the recursive pattern with the base case.

Next, we apply the mathematical/statistical model to simulate the virus interaction that appears in the biological animal model for the corresponding type of cancer. Doing so allows one to further develop the corresponding hepatitis anti-cancer vaccine through reconstruction of the T-cells using CRISPR technology, and train them to attack those can cells and finally eliminate them. One can evaluate and improve the efficacy of the vaccine using big data analysis.

CONCLUSION

Finally, it is important to note that one can mathematically model the virus infection processes that lead to liver cancer using Wnt signaling (e.g., corresponding to patterns in metabolism in colon cancer).[10] The result being that one might be able to find a suitable virus, such as an oncolytic virus, for the treatment of colon cancer, for example.[11] Moreover, it might be possible to "reconstruct"

the selected virus using CRISPR (according to the mathematical model computed previously) and train it to attack those cancer cells whenever we humans cannot find a suitable virus that is suitable used as a vaccine for our liver cancer like the case in. Hence, we might be able to develop the necessary drugs (that contain the suitable virus) to attack the respective liver cancer virus and balance the micro-organisms that co-existed in the surrounding cancer infected area. Theoretically, the tumor size will gradual bed iminished, and we may even cure the liver cancer caused by the HCV completely. Similarly, we can apply the same mathematical and statistical modeling method for other diseases such as HIV AIDS, and develop corresponding drug using CRISPR re-construction for suitable viruses. This author wants to remark that there is an international study done by the South African professor that when the COVID-19 virus meets the AIDS one in the human body, COVID-19 can be mutated and further evolved in to a harmful one while on the other hand, COVID-19can be mutated an devolved in the Netherland minks to become less harmful just like the case in small pot. This is indeed an interesting result obtained and may be used in the control of virus mutation for future. Actually, HKLam Theory can thus be applied as stated in the above section if the Butterfly effects or the Lorenz attractor exists in such kind of COVID-19 mutation in order to find the saddle or the equilibrium point.[11] Thus, this can then be acted as some form of control to harmful virus evolution. Last but not least, the SARS-CoV-2 virus mutates within HIV patients because of enzymes that copy RNA are prone to make errors.[12] This event implies the needs of the development in corresponding anti-enzymes of SARS-CoV-2 drugs to prevent continuous mutation in that virus among HIV patients. Or else researchers should prevent the errors that occurred during the reverse transcription of RNAs when either of the SAR-CoV-2 or hepatitis virus coexists within the HIV patients. However, the full details of the developing new anti-enzymes drugs or the combinational computation of those reverse transcription of RNAs are out of the scope of our present discussion.

Recently, with the similar concepts of in-depth controlling the reverse transcription of SARS-CoV-2 virus, one of the U.S.A. pharmaceutical company has turned the antiviral drug – Molnupiravir to fight against such virus. The drug is indeed a prodrug of the synthetic nucleoside derivate N4-hydroxycytidine which can cause the copying errors during viral RNA replication together with the harmful mutation effects to the SARS-CoV-2 virus. However, there is a lack of the corresponding virus mutation's big data to determine how much (or quantifying) will the positive evolution (or the deadly effects) of the virus be happened to our normal human COVID-19 patients. Thus, the drug is still in the controversial status. More experiments should be done in order to test the validation of the drug – Molnupiravir's healing efficiency to the SARS-CoV-2 virus.

Last but not least, we researchers or scientists may employ the similar causal regression method (so as to establish the Hayes Process model) as mentioned early in the case of cancer to the newest SARS-CoV-2 virus. As the virus's S protein binds to the receptor ACE2 [13] like the follow-ing listed: Native State —> Receptor Binding —> Intermediate —> Cell Fusion

The result may be a kind of human-made greatly reduced harmful Covid-19 virus just as posted in [14] for the used as a new and improved vaccine with less side effects. COVID-19 can be mutated and devolved in the Netherland minks to become less harmful just like the case in small pot. [15]

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