ORIGINAL ARTICLE

# Modeling $Co_2$ and $O_2$ Diffusions across Capillaries, Alveoli and Excess Fats for Primary and Secondary Preventions of Covid-19 in Clinic

**Kang Cheng**[1], **Changhua Zou**[2]

*[1]Basic Medicine, Medical School, Qingdao Binhai University, Qingdao, P.R. China, [2]Biomedical and Biochemical Infophysics, Science Research Institute, Edison NJ 08817, USA*

**ABSTRACT**

**Backgrounds:** The COVID-19 pandemic has killed almost 5 million and infected more than 246 million people in the world as of 10/31/2021. Today the COVID-19 pandemic is still spreading worldwide and is still a major public health crisis. Studies show that the increasing evidence demonstrates a strong correlation between obesity (high body mass index (BMI)) and the COVID-19. Individuals with obesity are linked with large significant increases in morbidity and mortality from COVID-19. How to better prevent COVID-19, cardiac or cerebrovascular strokes and other obesity related diseases are becoming urgent issues in clinical research. **Methods and Results:** In this study, based on the published data and theories of hemodynamics or hematology, we therefore propose modeling mechanisms of clinical hard breathing caused by excess fats, pulmonary hemorrhages, thrombosis, alveolar clots, scabs, edema, as well as air pollution and smoking. Our models qualitatively (formulaically) describe how human lungs (capillaries and alveoli) control diffusions and pressures of $CO_2$ and $O_2$ ; why some people, especially OHS (obesity hypoventilation syndrome) and obesity (overweight) asthma patients, are not mandatory to wear face masks to prevent COVID-19 and other respiratory diseases. **Conclusion:** To clinically prevent primary and secondary COVID-19 and other respiratory diseases without any side effect, we suggest people, especially for the patients with excess fats, to optimize the fatty amounts in the lower respiratory tracts by complete nutrition therapy. We think the body weight index (BWI) is a suitable daily measuring data for the fatty optimization because we have not found any data or method that the fats or lipids amount can be measured directly and daily between capillaries and alveoli in lungs.

**Key words:** capillaries, $CO_2$, $O_2$, diffusion, alveoli, fats, COVID-19, clinic, hemodynamics, hematology, pulmonary hemorrhages, thrombi, clots.

## INTRODUCTION

The COVID-19 pandemic has killed almost 5 million and infected more than 246 million people in the world as of 10/31/2021.[1] Today the COVID-19 pandemic is still spreading worldwide and is still a major public health crisis.

Studies show that the increasing evidence demonstrates a strong correlation between obesity (high body mass index (BMI)) and the COVID-19. Individuals with obesity are linked with large significant increases in morbidity and mortality from COVID-19.[2-4]

Several articles report that obesity hypoventilation syndrome (OHS) is a condition in some obese people in whom poor breathing leads to lower oxygen ($O_2$) and higher carbon dioxide ($CO_2$) levels in the blood. Complications of obesity

**Address for correspondence:** Kang Cheng, Basic Medicine, Medical School, Qingdao Binhai University, Qingdao, P.R. China.

**DOI:** 10.33309/2639-8354.040103

hypoventilation syndrome include pulmonary hypertension; right heart failure, also known as corpulmonale; and secondary erythrocytosis.[5] Overweight and hypertension patients have shortness of breath too,[6] i.e., overweight or obesity patients have a positive correlation with asthma.[7] OHS or overweight people have a high correlation with COVID-19.[2-4]

We proposed biomedical infophysical models of filtering ghost airflows of COVID-19 to defend our upper respiratory tracts and to decrease the related infection and death rates at a human society (country, territory or area) levels in our recently published two articles of preventing or treating (mild) COVID-19 by face mask-wearing.[8-9]

In our other previous studies, we also proposed models of complete nutrition therapy to prevent or cure cardiac or cerebrovascular strokes caused by obesity or overweight.[10-11]

To obtain higher efficiency to prevent COVID-19 and the other respiratory infection diseases, in late 2020, we published another paper for primary and secondary prevention of COVID-19 in clinic at an organ or tissue levels and suggested to often wash or clean our upper respiratory tracts to prevent all respiratory diseases as well as proposed hemodynamics models to maintain optimum transportation of all nutrients in blood flowing by drinking (boiled) warm water and complete nutrition fluids.[12]

How to better prevent COVID-19, cardiac or cerebrovascular strokes and other obesity related diseases are becoming urgent issues in clinical research. Therefore, in this article, we continue our previous studies[8-12] and propose models of $CO_2$ and $O_2$ diffusions across capillaries, alveoli and excess fats (an abnormally high concentration of fats or lipids between capillaries and alveoli in lungs) for primary and secondary preventions of COVID-19 in clinic in a perspective of hemodynamics or hematology.

## METHODS

In this study, we develop our models using published data of hematology and theories of Fick's diffusion law and hemodynamics; we assume a body weight index (BWI) indirectly indicates a fat or lipid amount between capillaries and alveoli in lungs because we have not found any direct method or data for the daily measuring.

## MODELING RESULTS

The published data show that COVID-19 involves at least three human physiological systems: the respiratory, cardiovascular and urinary.

Maintaining the acid/base balance of blood is vital to human survival. The normal blood pH is set at 7.4, which is slightly alkaline or "basic". Blood pH levels below 6.9 or above 7.9 are usually fatal if they last for more than a short time. There are three factors in the managements of pH: the lungs, the kidneys and buffers.[13] Lungs (capillaries and alveoli) control $CO_2$ and kidneys control $HCO_3^{-1}$ (bicarbonate) or $H^+$.[13-16]

### Modeling $Co_2$ and $O_2$ Diffusions across Capillaries, Alveoli and Fats in Lungs

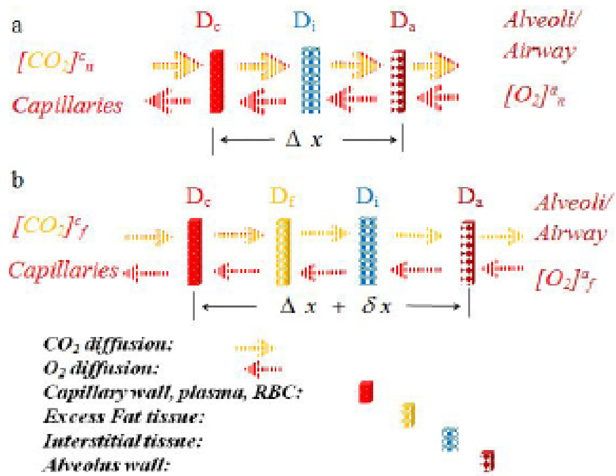
A lung has two circulation (flow) systems: The hemodynamic for the blood flow and the aerodynamic for the air flow.[17-18] Our previous theoretical studies were mostly focused on the upper respiratory tracts and hemodynamic system.[8-9, 12] In this investigation, our major assignments are focused on the mechanisms of the air diffusions across the two circulation (flow) systems of the hemodynamics and aerodynamics in lungs (in the lower respiratory tracts).

A one dimensional (1D) diffusion of particles can be physically modeled using Fick's first law of the pressure-driven flow,[14-15]

$$J=dV/(Adt)=-D(dP/dx)=D[(P_1-P_2)/(x_2-x_1)] \quad (1)$$

Where J represents diffusion volume per unit time and area (flux),[15] d denotes the differentiation, V and A respectively represent diffusion volume and cross area, x and t respectively denote space and time coordinates, P denotes the pressure of diffusions, D denotes the diffusion coefficient or diffusivity, $\nabla$(nabla) and $\Delta$ (delta) respectively denote gradient and variant operators; subscripts 1 and 2 respectively denote the two sides of the diffusion media. We omit Poiseuille's and osmotic pressures because they are not applicable in this investigation.[14-15, 17] We also assume D and A are quasi constants in specific diffusion mediums and ignore the partition coefficient to simplify our modeling procedures.

Figure 1: Equivalent diffusion pathways: a, normal; b, abnormal OHS (excess fats). D represents the diffusion coefficient; [ ] denotes the concentration, $\Delta$x denotes the normal thickness of the combined wall, $\delta$x denotes the increased thickness of the fat, subscripts a, c, f, i and n respectively denote the alveolus wall, capillary wall, excess fat, interstitial tissue and normal; superscripts a and c respectively denote alveoli and capillaries. See the text. The draw is not to the scale.




The meaning of equation 1 in physics is that a pressure gradient in a spatial domain produces (is equivalent to) a volume (quantity) flow (variant) in time domain.

Under normal (optimum ) circumstances the transfers of $CO_2$ and $O_2$ across the capillaries and alveoli in lungs are perfusion (flow) limited, but under certain abnormal (excess fats or over fatty) pulmonary conditions the transfers may become diffusion limited, and when the gases are in chemical combination with hemoglobin, they no longer exert a partial pressure. [14-15]

Neglecting the interaction of different diffusion components, such as $CO_2$ and $O_2$,[19] based on equation 1, we define normal $CO_2$ diffusion from capillaries to alveoli as (Figure 1 a),

$$J_n^{c-a}(CO_2)=dV_n^{c-a}/(Adt)=-D_n^{c-a}(CO_2)[dP_n^{c-a}(CO_2)/dx]=D_n^{c-a}(CO_2)[P_{nc}(CO_2)-P_n^{a}(CO_2)]/|x2-x1| \quad (2)$$

where, D and $\Delta$x (> 0) respectively denote the equivalent diffusivity and thickness of the diffusion, subscript n means the normal, superscripts c, a and - respectively denote capillaries alveoli and to; others have the same meanings as that in equation 1.

For normal exercising people, the faster breathing frequencies increase $CO_2$ volumes and pressures in blood vessels, the increased pressures in the lung capillaries raise the pressure gradients between the capillaries and alveoli in space domain (right sides of the second equal sign in equation 2), the raised gradients enhance $CO_2$ diffusions from the capillaries to the alveoli in time domain (left sides of the second equal sign in equation 2). Note that, $P_n^a(CO_2)$ is roughly a constant, the only way to raise the gradient is to elevate $P_n^c(CO_2)$ usually. Therefore, our modeling equation 2 qualitatively (formulaically) explain how or why $CO_2$ pressure increasing in the blood vessels can elevate exhaling $CO_2$ rates in a perspective hemodynamics.

For the OHS or obesity (overweight) people, there is an abnormal excess fats (over fatty)[27] of alveolar-capillary barrier to limit the diffusion, see Figure 1 b. Based on equations 1 and 2, we define $CO_2$ diffusions across the capillaries, alveoli and excess fats as equation 3,

$$J_f^{c-a}(CO_2)=-D_f^{c-a}(CO_2)[dP_f^{c-a}(CO_2)/dx]=D_f^{c-a}(CO_2)[P_f^c(CO_2)-P_f^a(CO_2)]/|x2' - x1'| \quad (3)$$

Where subscript f means the excess fats, $\delta$x (> 0) denotes the increased thickness of the excess fats (Figure 1b), others are the same as that in equation 2.

Comparing equations 2 and 3 of the normal and abnormal $CO_2$ diffusions, based on hemodynamic or physiological theories or data,[13-16] we assume the both diffusions (fluxes) are approximately equal because human physiological systems will do their best to provide almost constant (average) flow rates (equation 1) to maintain the lives, we also assume the (average) $CO_2$ air pressures do not change in the airways or alveoli, i.e.,

$$J_n^{c-a}(CO_2) = J_f^{c-a}(CO_2) \quad (4)$$

$$P_n^a(CO_2) = P_f^a(CO_2) \quad (5)$$

Therefore, from equations 2 - 5, we obtain,

$$D_n^{c-a}(CO_2)[P_n^c(CO_2)-P_n^a(CO_2)]/|x_2-x_1|=D_f^{c-a}(CO_2)[P_f^c(CO_2)-P_n^a(CO_2)]/|x_2' - x_1'| \quad (6)$$

Because of the excess fats, we assume,

$$D_n^{c-a}(CO_2) > D_f^{c-a}(CO_2) \quad (7)$$

and

$$|x_2' - x_1'| > |x_2 - x_1| \quad (8)$$

Therefore, to make the equation 6 valid, we must have,

$$P_f^c(CO_2) > P_n^c(CO_2) \quad (9)$$

In a perspective hemodynamics, our modeling (un)equations 2 - 9 qualitatively (formulaically) explain how or why $CO_2$ pressure increases in the blood vessels for OHS patients; and the thicker the abnormal fatty tissues or the lower the excess fatty diffusivity, the higher the $CO_2$ pressure in blood vessels and the more dangerous to human lives.

In the same way as that processing $CO_2$ diffusion from the capillaries to alveoli, we can obtain correspondent diffusion (un)equations of $O_2$ from alveoli (with and without excess fats) to the capillaries.

The (un)equations of $O_2$ and $CO_2$ have the same or similar forms. With the (un)equations of $O_2$, we can qualitatively (formulaically) explain how or why $O_2$ pressure decreasing in the blood vessels can enhance inhaling $O_2$ rates for OHS or obesity (overweight) and asthma patients as well as for normal exercising people; the thicker the excess fats or the lower the abnormal diffusivity, the lower the $O_2$ pressure in blood vessels and the more dangerous to human lives.

The mechanisms of that wearing face masks, air pollution and smoking make breathing difficult are the same as or similar to that of difficult breath for OHS or obesity (overweight) asthma patients. For OHS or obesity (overweight) asthma patients, the optimum treatment without any side effect is to reduce weight (excess fats)[7] with the complete nutrition therapy.

We think, our modeling equations qualitatively (formulaically) explain how or why $CO_2$ and $O_2$ pressures respectively increase and decrease in the blood vessels for OHS and asthma patients as well as for normal exercising people; how or why the excess fats, air pollution and smoking make people breath hard; why some people, especially OHS and obesity (overweight) asthma patients, are not mandatory to wear face masks to prevent COVID-19 and other respiratory diseases; and how human lungs (capillaries and alveoli) control diffusions and pressures of $CO_2$ and $O_2$ in a perspective of hemodynamics or hematology.

If we assume the diffusion thickness of the excess fats as a negative value, we obtain modeling equations of lacking fats. Lack of the fats may be also harmful to people health by increasing risks of bleeding, thrombusis and (or) hemorrhage strokes.[20, 11] Therefore, we want the fats in our lungs as well as other organs (tissues) are neither excessive nor little, but optimum.

## The $Co_2$ and $O_2$ Diffusions across Capillaries and Alveoli with Alveolar Clots, Fibrosis, Edema

COVID-19 invasion leads to immune cells, such as macrophages or neutrophils, injuring alveolus walls as well as killing the viruses.[21-22] After containment of an injury, the tissue repair phase starts with removal of toxins and waste products. Clotting (coagulation) reduces blood loss from damaged blood vessels and forms a network of fibrin proteins that trap blood cells and bind the edges of the wound together. A scab forms when the clot dries, reducing the risk of infection. Sometimes a mixture of dead leukocytes and fluid called pus accumulates in the wound.[16]

The wounded alveolus walls may produce pulmonary hemorrhages, edema, clots, fibrosis, scabs and (or) thrombi. The fibrosis, scabs, edema or clots decrease the exchange of $O_2$ and $CO_2$ by decreasing the effective contact area between the alveoli and air, increasing the thickness or decreasing the diffusivity of the diffusion of $O_2$ and $CO_2$,[12] see (un) equations 1 - 9.

Additionally, thrombi, clots can increase the viscosity that can decrease flow rates of $O_2$ and $CO_2$ in blood vessels.[12] Therefore, it is more difficult for the patients to breathe because higher respiratory frequencies are required by the physiological systems to maintain their daily lives. All these dysfunctional factors can be reasons or answers how or why the death rates of COVID-19 patients with pulmonary blood clots or edema are so high.[12,23-25]

## PH, $Co_2$ and $H_2o$ in Human Bicarbonate Buffer System and Kidney Functions

Bicarbonate buffer system maintains our bodies' homeostasis, the related chemical equation is,[13]

$$CO_2+H_2O<=>H_2CO_3<=>H^++HCO_3^{-1} \tag{10}$$

The chemical equation 10 links the three human physiological systems: the respiratory, cardiovascular and urinary that are involved in COVID-19 directly. Lungs (capillaries and alveoli) control $CO_2$ and kidneys control $HCO_3^{-1}$ (bicarbonate) or $H^+$.[16] The chemical equation 10 also indicates a control of pH value in the blood.

• If pH is too high, carbonic acid will donate hydrogen ions ($H^+$) and pH will drop.

• If pH is too low, bicarbonate will bond with hydrogen ions ($H^+$) and pH will rise.

Too much $CO_2$ or too little $HCO_3$ in human blood will cause acidosis.

Too much $HCO_3^{-1}$ or too little $CO_2$ in human blood will cause alkalosis. This condition is less

common than acidosis. $CO_2$ can be lowered by hyperventilation.[13]

So, if you are going into respiratory acidosis chemical equation 10 will move to the right. The body's $H^+$ and $CO_2$ levels will rise and the pH will drop. To counteract this, the body will breathe more and release $H^+$. In contrast, if you are going into respiratory alkalosis the equation will move to the left. The body's $H^+$ and $CO_2$ levels will fall and the pH will rise. So the body will try to breathe less to release $HCO_3^{-1}$.[13]

Therefore, our bodies have the negative feedback systems to manage our multiple physiological circulations.

Based on chemical equation 10, COVID-19, OHS or obesity (overweight) asthma patients are going into respiratory acidosis, i.e., they cannot normally transport $CO_2$ out as well as transport in $O_2$ of the bodies through human bloods. Therefore the concentration of body's $CO_2$ or $H^+$ goes high and the equation will move to the right to maintain their bodies' homeostasis.

Also based on equation 10, $H_2O$ plays a very important role in maintaining human bodies' homeostasis with bicarbonate buffer system, because (many) $H^+$, (little) $HCO_3^{-1}$ and other metabolic by-products or wastes dissolved in $H_2O$ need to be excreted from kidney in the form of urine.[13] The loss of $H_2O$ in the urine or sweat has to be compensated enough, this is one of reasons why or how drinking more warm (boiled) water is very helpful to prevent or cure almost all of the (mild) respiratory diseases,[8-9,12] such as COVID-19 and influenzas.

$H_2O$ plays a very important role not only in the homeostasis maintain but also in the biological energy (ATP) metabolism[13] and (or) OHS (overweight) preventions or treatments. Fats are storages of biological energies, to reduce weights (excess fats) means to reduce the stored energies, the chemical reaction equation is,[26]

$$ATP + H_2O \Rightarrow ADP + P \qquad (11)$$

The equation 11 indicates drinking water is not only inhibiting hungry feelings but also enhancing the chemical reaction in nutrition therapy, i.e. speeding up weight loss by burning the excess fats.

Because SARS-Cov II (COVID-19) is correlated to multiple obesities or over weights related diseases, such as high blood pressure, heart disease, lung disease, diabetes and cancers,[1,28] we strongly suggest to perform comprehensive (holistic) and systematic preventions of all of the diseases by maintaining normal body weight indexes with complete nutrition therapy without any side effect.

## DISCUSSIONS

Increasing levels of $CO_2$ or decreasing levels of $O_2$ in the blood of asthma and OHS patients are similar to do exercise in a perspective view of hematological respiratory mechanism. This is one of reasons why we do not recommend patients with COVID-19 or any respiratory disease to do intensive exercises[9] because of harder breathing.

The diffusion coefficients are proportional to the temperature,[19] therefore, drinking (boiled) warm water, without side effects, can increase the gas diffusions, decrease the viscosity and dissolve the blood clots to increase the blood flow and transportations of all nutrients[17-18,12] to prevent the diseases. Additionally, we felt drinking warm boiled water and (or) and keeping the laryngopharynx and larynx warm (30 C – 37 C) by wearing a scarf are very help to depress the coughing. The modeling results in this investigation are consistent to that in our previous study.[12]

We believe, after OHS or obesity (overweight) asthma patients reduce their weights to normal ranges, they increase their willingness to correctly wear face masks.

## CONCLUSION

To clinically prevent primary and secondary COVID-19 and other respiratory diseases without any side effect, we suggest people, especially for the patients with excess fats, to optimize the fatty amounts in the lower respiratory tracts by complete nutrition therapy. We think the body weight index (BWI) is a suitable daily measuring data for the fatty optimization because we have not found any data or method that the fats or lipids amount can be measured directly and daily between capillaries and alveoli in lungs.

To correctly wear face masks requires optimization of the status of the lower respiratory tracts too. To quit smoking and to reduce air and water pollutions are also key points of the comprehensive preventions of the respiratory diseases. If we systematically follow these guidelines to prevent the multiple diseases, we can get the maximum benefits with the minimum costs.

## ACKNOWLEDGEMENTS

We thank Vivien Cheng (MPH Harvard School of Public Health) for her insights and advisory on paper comments; we thank Clinical Research in Hematology to invite us for publishing this research article. Submitted: 11/9/2021.

## REFERENCES

1. World Health Organization. COVID-19 Weekly Epidemiological Update. Geneva: World Health Organization, Edition 64; published 2 November 2021.
2. Ritter A, Kreis NN, Louwen, Yuan J. Obesity and COVID-19: Molecular mechanisms linking both pandemics, Int. J. Mol Sci 2020;21:5793.
3. Albashir AAD. The potential impacts of obesity on COVID-19, Clinical Medicine 2020;20:(4)e109–13.
4. Popkin BM, Du S, Green WD, et al. Individuals with obesity and COVID-19: A global perspective on the epidemiology and biological relationships. Obesity Reviews. 2020;1–17.
5. NIH. Obesity Hypoventilation Syndrome NHLBI, 10/24/2020. Available from: https://www.nhlbi.nih.gov/health-topics/obesity-hypoventilation-syndrome.

6. Fritz JS. Shortness of breath: what your body might be telling you, Penn Heart and Vascular Blog, – Penn Medicine, 2016;May 20. available from: https://www.pennmedicine.org/updates/blogs/heart-and-vascular-blog/2016/may/shortness-of-breath.
7. Peters U, Dixon AE. The effect of obesity on lung function. Expert Rev Respir Med. 2018; 12(9):755–767.
8. Cheng K, Cheng V, Zou C. Urgent prevention of corona virus disease 2019 (COVID-19): Chinese eating and mask-wearing cultures. Journal of Public Health International. 2020;2(2):8-14.
9. Cheng K, Zou C, Biomedical infophysical models of filtering ghost airflows by wearing masks and maintaining social distancing to prevent COVID-19 and reopen all systems after shutdowns (lockdowns), Journal Of Public Health International. 2020;2:(4)26-39.
10. Cheng K. Health oriented lifelong nutrition controls: preventing cardiovascular diseases caused by obesity. SM J Nutr Metab. 2020;6:5.
11. Cheng K. Prevention of strokes caused by sickle cell anemia, ischemiaor hemorrhage. ARC Journal of Hematology. 2019;4(2):26-30.
12. Cheng K, Cheng V, Zou C. Hemodynamics of maintaining transportation of all nutrients in blood flowing: drinking (boiled) warm water and complete nutrition fluids for primary and secondary prevention of coronavirus disease 2019 in clinic. Clin Res Hematol 2020;3(2):1-5.
13. Young K. Human Physiology, Creative Commons Attribution-Share Alike 3.0 Unported License; 2013. Available from: https://www.wikibooks.org.
14. Jardins D, Terry R. Cardiopulmonary Anatomy & Physiology Essentials for Respiratory Care, 7th Edition, Delmar, a division of Thomson Learning, Inc. Thomson Learning; 2019.
15. Feher J. Quantitative Human Physiology: An Introduction. 2nd ed. New York: Elsevier Inc.; 2017.
16. OpenStax College. Anatomy and Physiology. Houston, Texas: OpenStax College, Rice University; 2013.
17. Fung YC. Biodynamics Circulation. New York: Springer; 1984.
18. Fung YC. Biomechanics: Mechanical Properties of Living Tissues. New York: Springer; 1996.
19. Kensal E. van Holde, W. Curtis Johnson, P. Shing. Principles of Physical Biochemistry. Ho.--2nd ed. Pearson Prentice Hall, Pearson Education, Inc. Upper Saddle River, NJ; 2006.
20. Price AJ, Wright FL, Green J, Balkwill A, Kan SW, et al. Differences in risk factors for 3 types of stroke UK prospective study and meta-analyses. Neurology 2018;90: e298-e306.
21. Borczuk, A.C., Salvatore, S.P., Seshan, S.V. *et al.* COVID-19 pulmonary pathology: a multi-institutional autopsy cohort from Italy and New York City. Mod Pathol. 2020;33:2156–2168.
22. Calabrese F, Pezzuto F, Fortarezza F, Hofman P, Kern I, Panizo A, et al. Pulmonary pathology and COVID-19: lessons from autopsy. The Experience of European Pulmonary Pathologists. Virchows Archiv. 2020;477:359–372.
23. Ferner RE, Levi M, Sofat R, Aronson JK. Thrombosis clinical Thrombosis in COVID-19: clinical outcomes, biochemical and pathological changes, and treatments. The Medicine Centre for Evidence-Based Medicine. 2020;June 4. Available from: https://www.cebm.net/covid-19/thrombosis-in-covid-19-clinical-outcomes-biochemical-and-pathological-changes-and-treatments/.
24. Lax SF, Skok K, Zechner P, Kessler HH, Kaufmann N, Koelblinger C, Vander K, Bargfrieder U, Trauner M. Pulmonary arterial thrombosis in COVID-19 with fatal outcome: results from a prospective, single-center, clinicopathologic case series. Ann Intern Med. 2020;Sep 1. Available from: https://www.acpjournals.org/doi/10.7326/m20-2566.
25. Cerruti L, Boscaro F, Poletto F, Campello E, Spiezia L, Simioni P. Acute pulmonary embolism in COVID-19 related hypercoagulability. Journal of Thrombosis and Thrombolysis. 2020;May 30. Available from: https://doi.org/10.1007/s11239-020-02160-1.
26. Nelson DL, Cox MM. W. H. Lehninger Principles of Biochemistry, fifth edition, Freeman and Company New York, NY, 2008.
27. Elliot JG, Donovan GM, Wang KCW, et al. Fatty airways: implications for obstructive disease. Eur Respir J. 2019;54:1900857.
28. CDC. Underlying medical conditions associated with high risk for severe COVID-19: Information for Healthcare Providers. 2021;Apr. 30. Available from: https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-care/underlying conditions.html.

**How to cite this article:** Cheng K, Zou C. Modeling Co2 and O2 Diffusions across Capillaries, Alveoli and Excess Fats for Primary and Secondary Preventions of Covid-19 in Clinic. Clin Res Hematol 2021;4(1):8-13.
DOI: 10.33309/2639-8354.040103