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DEFORMATION FEATURES IN THE CELL WHEN THE CORONAVIRUS ENTERS IT

A mathematical model has been proposed to describe the deformation behavior of a cell, when a coronavirus enters it. The model is continuum-based, and the theory of elasticity is used in calculations. It was found that the deformation process accompanying the penetration of coronavirus consists of two stages. At the first stage, the deformations of cytoplasmic membrane are elastic. At the second stage, the structure of a cytoplasmic membrane is destroyed. The dependence of the energy of the "coronavirus–cell" system on the size of the contact zone that separates the coronavirus and the cell was obtained. The existence of an energy barrier that separates both stages of the deformation process was proved. As a result, the penetration of the coronavirus terminates at the end of the first stage. However, the energy barrier can be overcome due to thermal fluctuations.

K e y w o r d s: coronavirus, cell, deformation, penetration.

1. Introduction

It is known (see, e.g., work [1]) that, in general, the process of coronavirus interaction with a cell is reduced to the transfer of viral RNA into the cell and the subsequent reproduction of the virus in the cell, which results in the cell death. It is generally agreed that RNA is transferred via the penetration of the virus into the cell. This process can be conventionally divided into two stages. At the first stage, the coronavirus is adsorbed on the surface of the cytoplasmic membrane and forms bonds with it (Fig. 1, *b*). At the second stage, the cytoplasmic membrane retracts the coronavirus and wraps it (Fig. 1, *c*). As a result, there arises a membrane bubble inside the cell (Fig. 1, *d*). In essence, this is a coronavirus wrapped in a piece of the cytoplasmic membrane.

To the authors' knowledge, currently there is no mathematical model that would describe the penetration of coronavirus into the cell. In the presented work, we propose one of the possible models for this process.

2. Continuum Model of the "Coronavirus–Cell" System

The deformation behavior of the "coronavirus–cell" system will be studied in the continuum approximation. In this approximation, the coronavirus can be considered as a ball, and the cell as a sphere composed of a shell (the cytoplasmic membrane) filled with fluid (cytoplasm). This approximation is illustrated in Figs. 1 and 2.

The shell material is considered to be an elastic medium.

The virus is known to be a molecular complex of densely packed chain molecules. Therefore, its stiffness substantially exceeds the stiffness of the cell. This circumstance will be taken into account by assuming that the ball–this is the coronavirus in our model–consists of an absolutely hard material. Accordingly, when invading into the cell, the indicated ball is not deformed.

Let R denote the radius of the ball (coronavirus), R_1 the radius of the sphere (cell), and h the thickness of the shell (cytoplasmic membrane). According to work [2], we put $h = (4 \div 6) \times 10^{-9}$ m. The value $R = 6 \times 10^{-8}$ m was borrowed from work [3]. In our calculations, we took the average value $h = 5 \times 10^{-9}$ m. Since $R_1 > 10^{-6}$ m, the following inequalities take place:

$$\frac{R}{R_1} \ll 1, \tag{1}$$

$$\frac{h}{R} \ll 1, \tag{2}$$

$$\frac{h}{R_1} \ll 1. \tag{3}$$

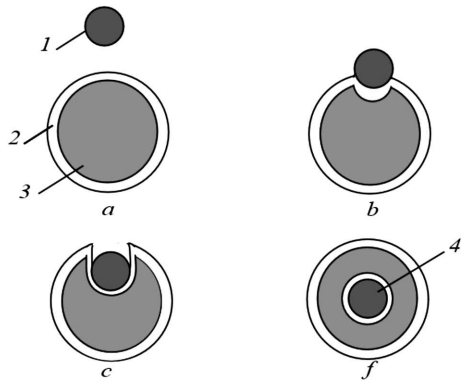

Fig. 1. Hypothetical mechanism of coronavirus penetration into the cell: (*a*) non-interacting coronavirus (*1*) and cell (cytoplasmic membrane (*2*) and cytoplasm (*3*)); (*b*) adsorption of coronavirus on the cytoplasmic membrane surface; (*c*) wrapping of coronavirus with the cytoplasmic membrane material; and (*d*) formation of a membrane bubble (*4*)

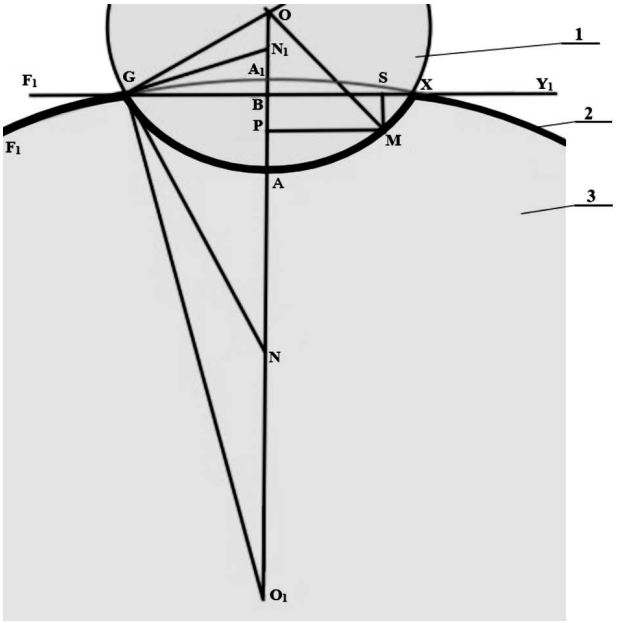

Fig. 2. Schematic diagram of the contact zone between the coronavirus and the cell: coronavirus (*1*), cytoplasmic membrane (*2*), and cytoplasm (*3*)

It is known that the theory of continuous media is based on the concept of mathematically infinitesimally small volume, which must possess, by definition, an infinitesimally small size. This concept is evidently an idealization: in reality, the mathematically infinitesimally small volume is associated with the so-called physically infinitesimally small volume, which has a finite size.

The smallest characteristic size in our case is the quantity h. Therefore, it is reasonable to take it as the size of the physically infinitesimally small volume. This means that the continuum approximation for this system is the zeroth approximation in the small parameters h/R and h/R_1, and h plays the role of a measurement unit. Accordingly, the cytoplasmic membrane is considered in this approximation as a surface.

3. Geometric Parameters of the Continuum-Based Model

Figure 2 reproduces the magnified fragment of Fig. 1, *b*, which illustrates the initial stage of coronavirus penetration into the cell.

The characteristic geometric parameters of this stage are the penetration depth AA_2 denoted as H, and the contact-zone radius $GB = BX$ denoted as a. Introducing the notations $A_1B = b_1$ and $AB = b$, we can write

$$H = b_1 + b. \tag{4}$$

By analyzing the triangles OGB and O_1GB, we obtain

$$a^2 = R^2 - (R - b)^2. \tag{5}$$

The notations for the angles are $\angle GOB = \beta$ and $\angle GO_1B = \beta_1$.

Let us draw two tangents through the point G: GN_1 to the circle centered at O_1 and GN to the circle centered at O. As one can see from Fig. 2, the angle $\angle N_1GN$, which will be denoted as γ, is the sum

$$\gamma = \angle N_1GB + \angle BGN. \tag{6}$$

From the similarity of the triangles N_1GB i N_1GO_1, it follows that $\angle N_1GB = \beta_1$. Moreover, the similarity of the triangles OGN and BGN implies that $\angle BGN = \beta$. Accordingly, expression (6) can be rewritten in the form

$$\gamma = \beta + \beta_1. \tag{7}$$

The angles β and β_1 are related to the radius of the contact zone a via the equalities

$$\sin\beta = \frac{a}{R}, \tag{8}$$

$$\sin\beta_1 = \frac{a}{R_1}. \tag{9}$$

As one can see from Fig. 2, the contact zone GAX is a spherical segment. Accordingly, its area is determined by the expression

$$S = 2\pi Rb. \tag{10}$$

From the smallness of the parameter R/R_1 and formulas (8) and (9), it follows that

$$\beta_1 \ll \beta. \tag{11}$$

Thus, in the zeroth approximation in the parameter R/R_1, we obtain the approximate equality

$$\gamma \approx \beta. \tag{12}$$

It transforms the spherical surface FGA_1XY into the plane F_1GBXY_1. Accordingly, in the zeroth approximation in the parameter R/R_1, we have

$$\beta_1 = 0, \tag{13}$$

and equality (4) looks like

$$H \approx b. \tag{14}$$

4. Mechanism of Cytoplasmic Membrane Destruction at the Coronavirus Penetration Into the Cell

As one can see from Fig. 2, the cell membrane undergoes a bending deformation when contacting with the coronavirus. One can also see that the deformation is maximum at the contact zone boundary. The angle γ can serve as a measure of the bending deformation at this boundary.

At the bending, the upper half of the membrane cross-section is known to be under the action of tensile stresses. The latter are maximum at the boundary. Their magnitude increases with the growth of γ. It is evident that at a certain value γ_1, which, according to formulas (8) and (12), corresponds to the radius of the contact zone $a_1 = R\sin\gamma_1$, the tensile stresses become strong enough to form a crack.

As a rule (see, e.g., work [4]), two stages are distinguished in the development of a crack. At the first stage, the size of the crack is smaller than a certain critical value, and some energy is required for the crack to grow. At the second stage, the size of the crack is larger than the critical one, and the crack grows independently. In our case where the crack is a closed conical surface, the size of the crack is its width, i.e. the distance from the membrane surface to the lower crack edge. Since the tensile stresses are concentrated in the upper half of the membrane cross-section, the critical crack size must be smaller than $h/2$.

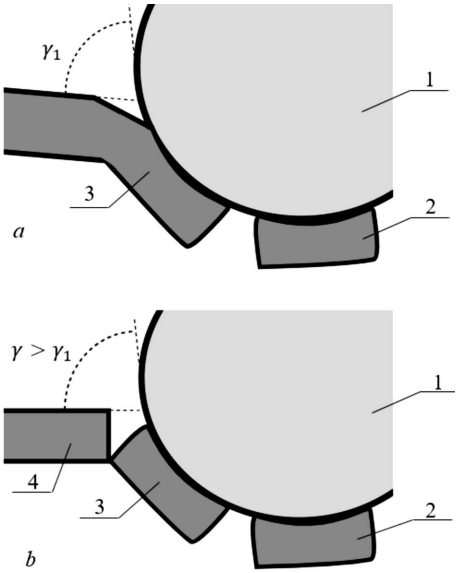

Fig. 3. Schematic diagram of the cytoplasmic membrane destruction: at $\gamma = \gamma_1$ (*a*), at $\gamma > \gamma_1$ (*b*) (coronavirus (*1*) and cytoplasmic membrane sections (*2, 3, 4*))

After the second stage of development, the crack separates sections 2 and 3 (Fig. 3, *a*) of the cytoplasmic membrane. As the coronavirus moves deeper into the cell and, accordingly, the angle γ increases further, there appears a new crack, which separates sections 3 and 4 (Fig. 3, *b*). The latter crack requires much lower energy for its formation than the crack shown in Fig. 3, *a*. Indeed, as one can see from Fig. 3, *b*, on its right edge, section 3 is contiguous with the first crack, which makes weaker the material in its vicinity.

The process of crack formation continues as the coronavirus penetrates into the cell. Therefore, when the coronavirus reaches the middle of the cell, its surface turns out covered with a destroyed cytoplasmic membrane.

According to the described mechanism of destruction, let us divide the process of cell deformation into two stages, by assuming that the first one is realized at $a < a_1$, and the second one at $a > a_1$. We will

also assume that the first and second stages correspond to the situations illustrated in Fig. 1, *b* and *c*, respectively.

5. Energy of the "Coronavirus–Cell" System

Let us express the potential energy U of the "coronavirus–cell" system as the sum

$$U = U' + U'', \tag{15}$$

where U' is the energy of cell deformations induced by the coronavirus, and U'' the energy of the bonds formed by the coronavirus with the cell surface. The energy U will be reckoned from the energy value corresponding to the non-interacting coronavirus and cell.

Let us calculate the energy of the system, if there is no crack in the membrane. The corresponding cell energy will be denoted as U_1, and its components as U_1' and U_1''.

When calculating U', the cylindrical coordinate frame (r, ϕ, z) is used. The line OO_1 is taken as the z-axis, and the point B is the coordinate origin. The z-axis is directed downward.

The approximation given by expression (13) means the replacement of the surface GA_1X by a flat section GBX. Now, GBX is the section ofthe membrane surface that, when being deformed owing to the coronavirus adsorption, transforms into the GAX surface and undergoes bending deformations. This deformation will be characterized by the function $W(r)$ describing the vertical (along the z-axis) displacement at the point of the flat section GBH located at the distance r from point B. For example, the vertical displacement for point S located at the distance $SB = r_S$ from point B is the segment $SM = W(r)$.

Let us determine the profile of the function $W(r)$. First, let us draw the line PM parallel to the line BS. As one can see from Fig. 2, the equalities $SM = BP$ and $BP = OP - OB$ hold true. For the triangle MOP, we have $OP = R\cos\phi$, where ϕ denotes the angle $\angle POM$. According to our notations, $OB = R - b$. Taking the equations given above into account, we obtain

$$W = R\cos\phi - (R - b). \tag{16}$$

The expression $\sin\phi = r/R$ obtained from the triangle OMP allows formula (16) to be rewritten in the form

$$W = \sqrt{R^2 - r^2} - (R - b). \tag{17}$$

Let the displacements W be small and satisfy the inequality

$$W \ll R. \tag{18}$$

Since $W \ll b$, formulas (5) and (10) take the form

$$a^2 = 2Rb, \tag{19}$$

$$S = \pi a^2, \tag{20}$$

and the inequality

$$a^2 \ll 2R^2. \tag{21}$$

is satisfied.

The latter allows the square root in formula (17) to be expanded in a power series in the small parameter r^2/R^2 up to the first-order term. As a result, we obtain

$$W = b - \frac{r^2}{2R}. \tag{22}$$

The displacements W give rise to the appearance of a bending moment acting in the cross-section with the normal r. The flat section GBX, which undergoes bending deformations, is, in essence, a plate. The theory of plate bending (see, e.g., work [7]) gives the following formula for the bending moment intensity M_r:

$$M_r = -D\left(\frac{d^2W}{dr^2} + \nu\frac{1}{r}\frac{dW}{dr}\right), \tag{23}$$

where D is the cylindrical stiffness of the plate, and ν is Poisson's ratio. Substituting expression (22) into formula (23), we obtain

$$M_r = \frac{D(1+\nu)}{R} \quad (r \le a). \tag{24}$$

According to work [5], the transverse force intensity Q_r is determined by the formula

$$Q_r = \frac{dM_r}{dr}. \tag{25}$$

Substituting expression (24) into this formula, we obtain that

$$Q_r = 0 \tag{26}$$

at $r < a$. Therefore, at the point $r = a$, where the bending moment intensity M_r jumps to zero, the transverse force intensity Q must become infinitely large according to Eq. (25).

As was mentioned above, in the continuum theory, the size of a physically infinitesimally small volume is taken as the measurement unit and considered

as an infinitesimally small quantity dr. The infinite value of Q_r at $r = a$ is a consequence of this assumption. Actually, the measurement unit is a finite quantity, which is equal to h in our case. This circumstance enables us to write down the approximate equality

$$Q_r \approx \frac{M_r}{h} \approx \frac{D(1+\nu)}{Rh} \quad (r = a). \tag{27}$$

Accordingly, the vertical force F created by the shell deformation is given by the formula

$$F = Q_r 2\pi a = 2\pi \frac{D(1+\nu)}{h} \frac{a}{R}. \tag{28}$$

Using expression (19), the previous formula can be rewritten in the form

$$F = \frac{2\pi D(1+\nu)}{h} \left(\frac{2}{R}\right)^{1/2} b^{1/2}. \tag{29}$$

Since $F = -\frac{dU'}{db}$, we obtain the following expression for the deformation energy:

$$U_1' = \frac{2}{3} \left(\frac{2}{R}\right)^{1/2} \frac{2\pi D(1+\nu)}{h} b^{3/2}. \tag{30}$$

From the viewpoint of the continuum approach, which is inherent in the accepted model, the formation of bonds between the coronavirus and the cell can be considered as a surface tension variation $\alpha = \alpha_2 - \alpha_1$, where α_2 is the surface tension at the section, where the cell contacts with the coronavirus, and α_1 the surface tension at the same section in the absence of the coronavirus. Accordingly, we have the following formula for the binding energy U_1'':

$$U_1'' = -\alpha S. \tag{31}$$

The sign "$-$" in this formula corresponds to the fact that the formation of bonds leads to the energy reduction.

It is known [6] that the bonds arising between the coronavirus and the membrane surface are the bonds between the spike-like proteins S that form a corona and the proteins in the cytoplasmic membrane. According to the results of works [6, 7], the energy of such bonds is taken to equal 10–12 kcal/mol. Let l be the distance between the membrane proteins that form those bonds. For the quantity α, we have the expression

$$\alpha = \frac{E}{l^2}. \tag{32}$$

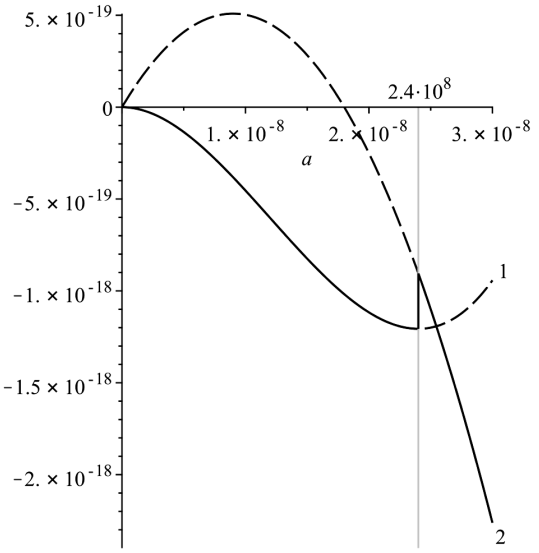

Fig. 4. Dependence of the energy U of the "coronavirus–cell" system on the contact zone radius

According to the results of work [8], $l = 70$ Å. Substituting the indicated values for E and l into formula (32), we obtain $\alpha = 2 \times 10^{-3}$ N/m.

From Eqs. (15), (30), (31), and (20), we have the following formula for the energy U of the system:

$$U_1 = \frac{2}{3} \left(\frac{2}{R}\right)^{1/2} \frac{2\pi D(1+\nu)}{h} b^{3/2} - \alpha\pi a^2. \tag{33}$$

Substituting b by a according to relation (19), we obtain

$$U_1 = \frac{1}{3} \frac{2\pi D(1+\nu)}{R^2 h} a^3 - \alpha\pi a^2. \tag{34}$$

Let $D = 10^{-18}$ nm [9]. For ν, let us take its maximum value, which is equal to 0.5 (see, e.g., work [6]). The corresponding dependence $U_1(a)$ is shown in Fig. 4 (plot *1*).

Now, let us calculate the energy of the system provided that there are cracks. In this case, the energy will be denoted as U_2, and its components as U_2' and U_2''. The energy U_2'' is the energy required to form a crack with a critical size. As was already mentioned, the crack contour is a circle with the radius a. Accordingly, the formula for U_2' looks like

$$U_2' = q 2\pi a, \tag{35}$$

where q is the linear energy density.

The energy associated with the formation of bonds between the coronavirus and the cytoplasmic membrane, U_2'', is identical to the energy U_1''. Therefore,

the energy of the system in this case can be written in the form

$$U_2 = q2\pi a - \alpha\pi a^2. \tag{36}$$

It is known (see, e.g., work [4]) that the basis of the membrane is composed of lipid molecules, which form two adjacent layers. Each of those molecules consists of a hydrophilic "head" and two attached hydrocarbon chains, "tails". The axes of the molecules are directed perpendicularly to the membrane surface. The thickness h_1 of either layer is equal to 2×10^{-9} m.

Let the critical crack size be equal to h_1. The lipid molecules mainly interact with one another via their carbohydrate chains. Let E_1 denote the binding energy between CH_2 groups in two neighbor chains, l_1 the distance between the neighbor bonds that bind those chains, and l_2 the distance between the axes of the interacting chains. The total number of bonds per unit area is $1/l_1l_2$. Accordingly, the energy required for a crack with the width h_1 to appear equals

$$U_1'' = \frac{2\pi a h_1}{l_1 l_2} E_1. \tag{37}$$

By comparing formulas (36) and (38), we obtain the relation

$$q = \frac{h_1}{l_1 l_2} E_1. \tag{38}$$

Since the issue concerns hydrocarbon chains, the known data for paraffins [10] will be used: $l_1 = 2.5 \times 10^{-10}$ m, $l_2 = 5 \times 10^{-10}$ m, and $E_1 = 1.1 \times 10^{-21}$ J. According to formula (38), we obtain $q = 1.8 \times 10^{-11}$ N. The dependence $U_2(a)$ corresponding to this q-value is shown in Fig.4 as plot *2*.

6. Thermodynamic Aspect of Cell Deformation during Coronavirus Penetration

According to the previous consideration, when the coronavirus penetrates into the cell, two deformation processes characterized by the dependences $U_1(a)$ and $U_2(a)$ may take place. According to thermodynamic principles (see, e.g., work [11]), the conditions for those processes to run spontaneously are the inequalities

$$\frac{dU_1}{da} < 0, \tag{39}$$

$$\frac{dU_2}{da} < 0. \tag{40}$$

As one can see from Fig. 4, $\frac{dU_2}{da} > 0$ at $a < 0.8 \times 10^{-8}$ m. This result confirms our previous hypothesis that the deformation should begin in the form of the first process that is not accompanied by the crack formation. This process runs spontaneously until the parameter a becomes equal to 2.4×10^{-8} m.

Despite that the derivative $\frac{dU_2}{da}$ becomes negative at $a > 0.8 \times 10^{-8}$ m, the second process does not begin as far as $a < 2.4 \times 10^{-8}$ m, because of the inequality $U_2 > U_1$. Figure 4 demonstrates that $\frac{dU_1}{da} > 0$ at $a > 2.4 \times 10^{-8}$ m, which means that the first process terminates at $a = 2.4 \times 10^{-8}$ m.

Therefore, we have the value $a_1 = 2.4 \times 10^{-8}$ m for the contact zone radius corresponding to the end of the first stage. At such an a_1-value, the cell shape changes insignificantly. Beyond the contact zone, it remains almost the same as the cell had before its interaction with the coronavirus.

Since $\frac{dU_2}{da} < 0$ at $a > 2.4 \times 10^{-8}$ m, the second deformation process, which is associated with the formation of cracks, can be realized in this interval. However, according to Fig. 4, for this process to start, the system must overcome an energy barrier of about 4×10^{-19} J. This circumstance prohibits the virus from penetrating into the cell, when the radius of the critical zone reaches a value of 2.4×10^{-8} m.

The barrier can be overcome by the system owing to thermal fluctuations. The energy needed to do this is accumulated in the system during a certain time interval, which determines the duration of the lag before the virus starts to destroy the membrane. After the system has overcome the barrier, the second process begins and runs spontaneously.

According to the considered mechanism of destruction, the material of the membrane has to be destroyed, as a result of this process, at all $a > 2.4 \times 10^{-8}$ m. In this case, the material practically loses its elastic properties and, accordingly, its ability to resist a bending deformation. The increase of deformations does not lead anymore to the growth of stresses. The destroyed material behaves itself as a plastic medium (see, e.g., work [12]). In effect, the material loses its resistance to the formation of new bonds between the coronavirus and the cell. The practically unimpeded formation of such bonds is accompanied by substantial membrane deformations and, accordingly, considerable changes in the cell shape. Being wrapped in the membrane material, the coronavirus penetrates into the cell, as is shown in Figs. 1, *c* and *d*. Now, the

structure of the membrane becomes destroyed, which favors the exit of RNA into cytoplasm.

7. Conclusions

Deformations that take place in the cell, when the coronavirus penetrates into it, have the following characteristic features:

1. the process of cell deformation associated with the coronavirus penetration consists of two stages;

2. at the first stage,

• there arise bending, mainly, deformations in the cytoplasmic membrane, which reach the maximum magnitude at the boundary of the contact zone between the cell and the coronavirus;

• those deformations are elastic;

• the cell shape undergoes minor changes;

3. the first stage terminates, when the radius of the contact zone becomes approximately equal to 2.4×10^{-8} m; as a result,

• the material of the cytoplasmic membrane becomes destroyed at the contact zone boundary, which leads to the appearance of an energy barrier of approximately 4×10^{-19} J, which prevents the further penetration of the coronavirus into the cell;

• due to the barrier, the penetration process stops;

• the coronavirus accumulates the energy required to overcome the barrier from the environment due to thermal fluctuations;

• the rate of energy accumulation determines the lag time;

4. the second stage begins after the coronavirus has overcome the barrier; at this stage,

• the membrane destruction continues;

• membrane deformations are plastic;

• the cell shape changes substantially, so the coronavirus, being wrapped in the membrane material, penetrates inside the cell.

1. V.G. Zarnitsyn, M.R. Prauznits, Yu.A. Chizmadzhev. Physical methods for the transfer of nucleic acids into tissues and cells. *Biol. Membr. Zh. Membr. Klet. Biol.* **21**, 355 (2004) (in Russian).
2. A.S. Davydov. *Biology and Quantum Mechanics* (Pergamon Press, 1981).
3. S. Klein, M. Cortese, S.L. Winter, M. Wachsmuth-Melm, Ch.J. Neufeldt, B. Cerikan, M.L. Stanifer, S. Boulant, R. Bartenschlager, P. Chlanda. SARS-CoV-2 structure and replication characterized by in situ cryo-electron tomography. *Nat. Commun.* **11**, 1 (2020).
4. L.D. Landau, E.M. Lifshitz, *Theory of Elasticity* (Pergamon Press, 1959).
5. S.P. Timoshenko. *A Course in the Theory of Elasticity* (Naukova Dumk1, 1972) (in Russian).
6. A.B. Jena, N. Kanungo, V. Nayak, G.B.N. Chainy, J. Dandapat. Catechin and Curcumin interact with corona (2019-nCoV/SARS-CoV2) viral S protein and ACE2 of human cell membrane: insights from Computational study and implication for intervention. *SCI REP-UK* (2021).
7. J.B. Junior. Interaction energies of the human ACE2 molecular recognition by SARS-CoV-2. *Virol. Curr. Res.* **4**, 1 (2020).
8. M.M. Poo, R.A. Cone. Lateral diffusion of rhodopsin in the photoreceptor membrane. *Nature* **247**, 438 (1974).
9. R. Skalak. Rheology of red blood cell membrane. *Microcirculation* **1**, 53 (1976).
10. P.H. Geil. *Polymer Single Crystals* (Interscience Publishers, 1963) [ISBN: 978-0470295106].
11. E.M. Lifshitz, L.P. Pitaevsky, *Statistical Physics: Theory of the Condensed State* (Butterworth-Heinemann, 1980).
12. L.M. Kachanov. *Fundamentals of the Theory of Plasticity* (Dover, 2004).

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ОСОБЛИВОСТІ ДЕФОРМАЦІЙ, ЯКІ ВИНИКАЮТЬ У КЛІТИНІ ПРИ ПРОНИКНЕННІ В НЕЇ КОРОНАВІРУСУ

Пропонується математична модель, яка описує деформаційну поведінку клітини при проникненні в неї коронавірусу. Модель є континуальною, при розрахунках використовуються методи теорії пружності. Встановлено, що процес деформування, який супроводжує проникнення коронавірусу, складається з двох стадій: на першій стадії деформації цитоплазматичної мембрани є пружними, на другій стадії відбувається руйнування її структури. Отримано залежність енергії системи "коронавірус–клітина" від розміру контактної зони, яка розмежовує коронавірус і клітину. Доведено існування енергетичного бар'єра, що розділяє обидві стадії процесу деформування. Ця обставина приводить до зупинки проникнення коронавірусу наприкінці першої стадії. Подолання енергетичного бар'єра, необхідне для подальшого проникнення, відбувається за рахунок теплових флуктуацій.

Ключові слова: коронавірус, клітина, деформація, проникнення.