PAPER

On the possible mechanisms of the selective effect of a non-equilibrium plasma on healthy and cancer cells in a physiological solution

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Abstract

This paper discusses possible mechanisms for the selective effect of weakly ionized nonequilibrium plasma and currents in electrolytes on healthy and cancerous cells in physiological saline in a Petri dish. The interaction with the plasma source leads to a change in osmotic pressure, which affects the electro-mechanical properties of cell membranes in healthy and cancerous cells in different ways. The currents arising in the electrolyte charge the membranes of healthy and cancerous cells to a different potential difference due to the different values of the membranes’ dielectric constants. We hypothesized: 1. The dielectric permeability of cancer cell membranes is lower than that of healthy cells, as is the capacity of a unit of the membrane surface, and therefore, the additional potential difference acquired by the membrane through charging with currents induced in the intercellular electrolyte is greater in cancer cells. This can lead to electroporation of cancer cell membranes, resulting in their apoptosis, but does not affect healthy cells. 2. It is known from the literature that the equilibrium potential differences on the membrane (resting potential) of cancer and healthy cells are noticeably different. Therefore, a change in the potential difference on the membrane due to currents in the extracellular fluid can affect the permeability and transport properties of the membranes. It can also be a reason for the selective effect of the nonequilibrium plasma interaction with healthy and cancerous cells in physiological saline.

Introduction

A number of experimental studies have convincingly shown that the action of weakly ionized nonequilibrium plasma on a physiological solution can selectively affect the cells present in that solution [1–9]. Special attention should be paid to works in which the selective stimulation of apoptosis in cancer cells was observed [4–9].

In [10], we described the importance of accounting for osmotic pressure when analyzing the physiological effects on cellular structures in plasma medicine. The interaction of a weakly ionized plasma jet with a saline solution leads to the diffusive flow of hydrated ions from the interface to the cell structures (figure 1(A)), thus changing the ionic composition of the saline electrolyte. Figure 2 shows a cell in a physiological solution when there is no influence of the plasma jet on the solution (figure 2(A)) and when there is influence of the plasma jet on the solution (figure 2(B)). \( n_{in,\text{w}}, n_{in,\text{ion}} \) are the densities of water molecules and hydrated ions in a cell; \( n_{out,\text{w}}, n_{out,\text{ion}} \) are the densities of water molecules and hydrated ions in the physiological saline near the membrane cells, \( \varepsilon = 81 \) is the dielectric permittivity of electrolyte in and outside the cell; \( n_{in,\text{w}}, \varepsilon_{in} \) are the density of water molecules in the membrane and the dielectric permittivity of the membrane; \( \gamma_{in} \) is the potential difference across the inner and outer surfaces of the membrane. Figure 2(B) shows a cell in a field of currents generated in physiological saline by an external plasma source. Red arrows indicate currents in physiological saline, blue arrows indicate flow of water molecules through a membrane due to an increase in the density of hydrated ions near the cell. Since the cell membrane is permeable to water molecules and poorly permeable to...
hydrated ions (see, for example [11, 12]), an increase in the hydrated ion density near the cell membrane leads to the outflow of water from the cell (the osmotic pressure drop across the cell membranes [6]) and, accordingly, to a decrease in the membrane tension. Estimations of the compression coefficient for the membranes of some types of cells are presented in [13].

It is quite obvious that membrane compression could change the membrane’s porosity and, correspondingly, its dielectric permittivity due to the density change of the water molecule in it and the ion and protein transport through it. However, if the plasma interacting with the electrolyte has a potential different from that of the floating equilibrium in the electrolyte, then ion currents are induced in the electrolyte.

It has to be noted that currents can be excited without plasma when electrodes are immered in an electrolyte to which a potential difference is applied (figure 1(B)) [14–18]. These ion currents can lead to an additional charge on the cell membranes, thus changing the transmembrane potential difference. It is also possible that the combined effect of changes in the ionic composition resulting from the interaction of the plasma with the solution and from the currents induced in the Petri dish (figure 1(C)) is due to the fact that the region of the interaction of the electrolyte with the plasma generally has a potential different from the floating potential in the electrolyte.

Membrane properties in cancer and healthy cells can vary greatly. The module of the all-round compression and shear modulus for hepatocellular carcinoma cells (HCCs) in the measurements [13] are equal to $K_1 = 103.6 \text{ Nm}^{-2}$ and $K_2 = 42.5 \text{ Nm}^{-2}$, respectively, and for hepatocytes, they are $K_1 = 87.5 \text{ Nm}^{-2}$ and $K_2 = 33.3 \text{ Nm}^{-2}$, respectively. The measured viscosity coefficients in the membrane also are different: $\mu = 4.5 \text{ Pa} \cdot \text{s}$ in HCCs and $\mu = 5.9 \text{ Pa} \cdot \text{s}$ in rat hepatoma cells. Resting potentials on the membranes of healthy and cancerous cells also differ markedly. For example, [19] showed that the transmembrane resting potentials in rat hepatocytes and hepatoma cells are $-37.1$ and $-19.8 \text{ mV}$, respectively, and in mouse corneal

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**Figure 1.** Schematics of the typical experimental setups with cell structures in saline in a Petri dish. (A) - plasma jet interacting with air-saline interface. (B) — currents induced by voltage applied to the electrodes. (C) — joint effect of applied voltage and plasma jet.

**Figure 2.** Cell in the physiological saline. (A) — no external source of weakly ionized plasma, (B) — an external source of nonequilibrium plasma interacts with saline. Red arrows indicate currents in physiological saline, and blue arrows indicate flow of water molecules through a membrane due to an increase in the density of hydrated ions near the cell.
fibroblast and fibrosarcoma cells, the corresponding resting potentials are $-42.5$ and $-14.3$ mV. In other words, the resting potential in cancer cells is two times less than in healthy ones, indicating a difference in the transport properties of the membranes of healthy and cancerous cells.

It was shown in [20] that the composition of phospholipids and the carboxyl and phosphate functional groups associated with them in the membranes of healthy and cancer cells differ significantly. This, as well as the difference in the composition of ions in the cell [21, 22], apparently explains the difference in the potential difference on the membrane [13, 19] of healthy and cancer cells, as well as the difference in the total charge of the cell [20–23].

It is possible that a small effect on the mechanical properties of the membranes or on the transmembrane potential difference might have a significant effect on the transport into and out of the healthy and cancer cells.

**Formulation of the problem**

The currents arising in the electrolyte charge the membranes of healthy and cancerous cells to a different potential difference due to the different values of the membranes’ dielectric constant. One of the paper’s hypotheses is that the dielectric permeability of cancer cell membranes is lower than that of healthy cells, as is the capacity of a unit of membrane surface, and, therefore, the additional potential difference acquired by the membrane through charging with currents induced in the intercellular electrolyte is greater in cancer cells. This may lead to electroporation of cancer cell membranes, resulting in their apoptosis, but would not affect healthy cells.

In this brief paper, we would like to bring the scientific community’s attention to the fact that a change in the cell membrane surface charge due to the currents in electrolyte may be responsible for selective action on healthy and cancer cells due to, as mentioned above, the difference in the mechanical-electrical properties of cell membranes and the different responses to the change in the membrane charge. In the electrolyte, currents can be produced by an electrode immersed in a Petri dish or by a plasma jet (figure 1). It should be noted that, in the case of a plasma jet and electrodes (figure 1(C)), the membrane charging will be accompanied by a change in osmotic pressure, which enhances the effect on the cells.

The application of electric fields to biological cells in a conducting medium leads to an accumulation of charge on the outer surface of the membrane and, consequently, to a change in voltage across the membrane. Small changes in the potential on the membrane ($\sim 10–100$ mV) of short duration on the order of milliseconds lead to a disruption of the voltage-dependent channels and, accordingly, to a change in the ion transport through the membrane without destroying the integrity of the membrane [14–16].

With higher electric fields corresponding to higher voltage across the cell membrane, electroporation occurs. Thus, the permeability of the membrane increases to such a level that either the cell needs from seconds to hours to recover (reversible breakdown) or cell death occurs (irreversible breakdown) [14–18]. In [14–16], it was experimentally shown that the critical field strength for the lysing of bacteria (prokaryotic cells) is around 0.1–1 V across the cell membrane, depending on exposure time. In [18], experimental results were given for the influence of electric fields of varying intensity and duration on cancer cells at different values of the medium’s conductivity [18]. It was shown that, as the medium’s salinity (conductivity) increases, the electric field at which cell death occurs begins to fall.

**Mathematical model**

Consider the following model of the cell based on conditions corresponding to figure 1 and figure 2:

1. The cells are thin-walled spheres of radius $a$ that are far enough from each other so that they can be viewed independently of each other in the field of constant currents (figure 3).

2. The cell environment is an electrolyte with conductivity on the order of 2–5 S m$^{-1}$.

3. The internal environment of the cell (sphere) is also an electrolyte with conductivity close to the conductivity of the external medium.

4. The membrane is impermeable to ionic conduction currents, which charge the capacitance, i.e., the current in the electrolyte is closed through the membrane capacitance by the displacement current, $c_m dU_m/dt$, where $c_m$ is the membrane capacitance per unit area and $U_m$ is the voltage on the membrane. The cell (sphere) membrane is charged with currents in the electrolyte until the additional surface charge accumulating on the membrane compensates for the radial field of the currents that charge the membrane.
Consider the case when the time of the field change in the electrolyte is much longer than the charging time of the surface of the sphere (the cell membrane), shown in figure 2. The continuity equation for the currents flowing in the external electrolyte:

\[ \text{div} \vec{j} = \text{div} (\sigma \vec{E}) = \sigma \text{div} (\vec{E}) = -\sigma \Delta \varphi = -\sigma \left( \frac{1}{r^2} \frac{\partial}{\partial r} r^2 \frac{\partial \varphi}{\partial r} + \frac{1}{r \sin \theta} \frac{\partial}{\partial \theta} \sin \theta \frac{\partial \varphi}{\partial \theta} \right) = 0, \]  

(1)

where \( \varphi, \vec{j}, \) and \( \vec{E} \) are distributions of potential, currents, and electric field, and \( \sigma \) is the electrolyte conductivity.

In (1) we used Ohm’s law \( \vec{j} = \sigma \vec{E} \), and the relation \( \vec{E} = -\nabla \varphi \).

Far from the sphere, the current density is constant. Accordingly, the current, potential and the electric field are related by the relation:

\[ \varphi_\infty = -E_0 r \cos \theta = -j_0 r \cos \theta / \sigma, \]  

(2)

Since in our model the membrane is impermeable to ion currents, the membrane is charged until the stationary radial electric field is zero:

\[ E_a = -\frac{\partial \varphi}{\partial r} \bigg|_{r=a} = 0. \]  

(3)

The solutions for (1) with boundary conditions (2), (3) are

\[ \varphi_{r>a} = E_0 r \cos \theta \left( 1 + \frac{a^3}{2r^3} \right), \]  

(4)

and, at \( r = a \),

\[ \varphi_a = \frac{3}{2} E_0 a \cos \theta = \frac{3}{2\sigma} j_0 a \cos \theta. \]  

(5)

From (5), it follows that \( \varphi_a \) in (6) is different at \( \theta = 0 \) and \( \theta = \pi \). Accordingly, the additional potential differences induced by currents on the membrane are as follows:

\[ \varphi_a (0) = \frac{3}{2} E_0 a, \text{ at } \theta = 0, \]  

(6a)

and

\[ \varphi_a (\pi) = -\frac{3}{2} E_0 a, \text{ at } \theta = \pi. \]  

(6b)

When the stationary state is reached, the currents inside the conductive sphere (cell) are absent (the walls of the cells are impermeable to currents), so the potential of the sphere is uniform and constant. We are interested in the additional potential difference on the membrane associated with charging the membrane with external currents, so we set \( \varphi_{\infty} = 0 \).

Since the potential difference across the membrane in cancer cells is almost twice as large as that of healthy cells [19], a potential change of \( \sim 100 \text{ mV} \) can cause irreversible breakdown of the membrane of cancer cells like hepatoma and fibrosarcoma cells but can be safe for healthy cells like hepatocytes and fibroblast cells.

Let us find the value \( j_0 \) at which the additional change of the potential difference on the membrane due to being charged by currents in the electrolyte is \( \sim 100 \text{ mV} \). Therefore, for example, for \( a \approx 11 \mu\text{m} \), corresponding to the size of the hepatocytes of SH-R3s [20] and \( \sigma = 1 \text{ S/m} \) [24], we obtain

**Figure 3.** Schematic of a spherical cell cross-section in a field of currents uniformly distributed over the volume.
\begin{equation}
    j_b \approx \frac{2\sigma}{3\epsilon_0} |\varphi_a| = 6 \cdot 10^3 \text{ A/m}^2.
\end{equation}

Then, let us estimate the charging time of the surface of the sphere to the potential (6). Obviously, this time is determined by the time required to charge the spherical capacitor to the potentials of (6a) and (6b). For simplicity, we shall assume that a thin-walled dielectric sphere does not perturb the electric field near its surface. In this case, the radial current charging the spherical capacitor is
\begin{equation}
    j_r = -j_b \cos (\theta).
\end{equation}

In this case, the estimate of the transition time (characteristic charging time of the capacitor) is
\begin{equation}
    \tau_{ch} \sim \frac{3 \epsilon_m a}{2 \sigma} \approx 0.165 \mu s
\end{equation}

In (9), we substituted \( \epsilon_m \approx 10^{-2} \text{ F/m} \) [24, 25], \( a = 11 \mu m \), and \( \sigma = 1 \text{ S/m} \). If the change of fields in the electrolyte surrounding the cell lasts more than a few tenths of a microsecond, the induced potential on the membrane can be considered quasi-permanent, as defined by (5). If the current ‘at infinity’ changes its direction for a time much shorter than the membrane charging time (9), the change in the membrane potential caused by its additional charging does not affect the operation of the voltage-dependent channels and, accordingly, the cell activity [16]. Note that we have neglected Joule heating. This is a fully justified assumption since an elementary estimate shows that heating of saline with conductivity \( \sigma = 1 \text{ S/m} \) up to \( \Delta T = 1 \text{ K} \) by the current density (7) takes more than 0.1 s. This time is much longer than all the characteristic times of the problem under consideration, even if the heat conduction losses are neglected. We also assume that the transition time (9) is much shorter than the characteristic current termination time that is due to charging the walls of the Petri dish.

Results

It is known that, when a nonequilibrium plasma jet [26] or other type of weakly ionized plasma (for example, a dielectric barrier discharge plasma [1, 27]) interacts with a physiological solution, long-lived solvated (hydrated) ions are formed in the interface. It was shown in [10] that the diffusion of these ions into the depth of the Petri dish, where the studied cell cultures are located, leads to an increase in the density of hydrated ions near the cells and, accordingly, to a change in the osmotic pressure since the membrane is permeable to water but impermeable to hydrated ions. As a result, it is most likely that a change in the concentration of hydrated ions in the intercellular medium makes the solution a hypertonic solution, which leads to the release of excess water from the cell, and hence, cell compression [10].

However, a change in the density of ions in the solution also leads to an increase in the conductivity of the medium and to an increase in the charging current of the membrane. The process of charging the membrane depends on the type of plasma source interacting with the boundary of saline and feeding hydrated ions into the solution. In the case of a Dielectric Barrier Discharge (DBD) plasma source with nanosecond repetitive pulses [27], the amount of time the change of the electric field lasts is much less than the charging time of the membrane \( \tau_{ch} \) (9), and the induced currents obviously cannot noticeably affect the charge on the membrane during a single pulse. However, at a high enough repetition rate of nanosecond pulses or with plasma exposure of a microsecond or longer, charging cell membranes with currents induced in the electrolyte, together with altered osmotic pressure, should certainly affect the mechanical properties of the membranes and the transmembrane transport through ion and other channels.

The dielectric constant of the real phospholipid membrane in the cell, estimated by the experimental values of the capacitance of the membrane from [24, 25], is of the order of \( \epsilon_m \approx 7 - 10 \), while in the ideal phospholipid membrane, the relative dielectric permittivity is \( \epsilon_m \approx 2 - 3 \) [28]. The difference is two- to threefold, apparently due to the presence of water molecules in the pores and defects of the real cell membrane. Since the compression of the membrane can result in its dehydration, the change in osmotic pressure can lead to a change in the electrical properties of the membrane (i.e., an additional potential difference acquired when charging the membrane) up to a loss of dielectric strength (electroporation). Furthermore, if the electrical and mechanical properties of cell membranes in healthy and cancer cells are different, then the induced currents in saline and the osmotic pressure changes cause different physiological consequences, resulting in the selectivity of the plasma effects on cell structures observed in the experiments.

As we saw above with examples of hepatocellular carcinoma (HCC) and hepatocyte cells, the module of the all-round compression of cancer cells significantly exceeds the module of the all-round compression of the healthy cells [13]. Therefore it should be expected that, with the same change in osmotic pressure, the compression of the cancer cells and hence, the displacement of water from it, are less significant than in healthy cells. This means that the change in volume of a cancer cell caused by an additional osmotic pressure differential
is less than a change in the volume of healthy cells. Thus, after the interaction of plasma with saline, the final osmotic pressure drop on the cancer cells membranes is significantly greater than on the membranes of healthy cells. This leads to a greater displacement of water from the membrane of cancer cells than from healthy cells. As a result, the dielectric constant and the specific capacity of cancer cell membranes become significantly smaller than those of healthy cell membranes. Thus, with the same current and exposure time, the membranes of cancer cells are charged to a significantly higher potential difference, leading to a more likely electroporation. This may cause apoptosis in cancer cell, but not in healthy cell membranes. This may explain the selectivity of apoptosis of cell cultures in a Petri dish when interacting with plasma. It should be emphasized that even a small change in the potential difference (5) can affect the transport properties of the membranes of cancer and healthy cells in different ways [11], which can lead to irreversible consequences in cancer cells, resulting in their death.

Conclusions

Nonequilibrium plasma generated by various sources in the near air interface with physiological saline in a Petri dish with cell cultures changes the ionic and molecular composition of the solution. This leads to a change in the osmotic pressure drop in the cells. If the mechanical properties of the membranes in cancerous and healthy cells are different, then a change in the osmotic pressure drop can lead to different physiological consequences for cancerous and healthy cells. In experiment it will manifest as a selective action of nonequilibrium plasma on different cell cultures.

In this paper, we examined additional possible mechanisms of the observed selective effect of plasma on cell structures in a Petri dish, leading to selective apoptosis in cancer cells, but not in healthy cells. Since the modulus of compression of membranes in diseased cells exceeds that of membranes in healthy cells, it should be expected that the decrease in the dielectric constant of membranes in sick cells due to water displacement should be greater than the decrease in the dielectric constant in healthy cells. Therefore, with the same additional charge on the membrane, generated by currents induced in the intercellular solution, the voltage on the membrane and the probability of its breakdown are greater for cancer cells than for healthy ones. Another possible mechanism may be due to the fact that even a small change in the stationary potential difference on the membrane can affect the transport properties of the membranes of cancerous and healthy cells in different ways.

To confirm the mechanisms of selective action proposed in this paper, it is very important to measure the dielectric permittivity of the membranes and its variation due to changes in the osmotic pressure in healthy and cancerous cells.

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