

A Possible Mechanism for the Rife Effect

Having received numerous responses to my last paper written a few days ago, it's now clear to me that many (most) people who read it didn't fully grasp what I was saying and for the most part completely misinterpreted it. That's not their fault, it's mine, having just carefully re-read the paper I realise that I jumped over a significant part of the detail of the explanation near the end. Since I'm trying to introduce a (revolutionary?) new concept, it's hardly surprising that this didn't help at all!

So I decided to write this "addendum" to the last paper to explain in more detail. And this time hopefully I'll do a better job and convey it all. I won't limit myself to just explaining the technicalities of the electronics, I'll try and relate this directly to the effect in a real biosystem.

A Cell Primer

I would like to start by going back to the question of harmonics and the idea I proposed that harmonics are NOT used in order to randomly hit MOR's in tissue, but that they have a different effect.

What is the "different effect"? I believe it's an effect in which the cell wall or membrane of a target cell (i.e. a bacterium we want to kill) gets charged up or hyperpolarised. I can't think of a simple analogy to make this clear so I'll explain in terms of real cells but starting from the cell itself and work outward toward the electronic implications instead of the other way around. But first we need to understand exactly what cells are.

Real cells (including body cells, bacteria etc) all have an outer "skin" which is called the cell membrane. The cell membrane is typically very thin and usually consists mainly of two layers of a lipoprotein (i.e. literally two lipoprotein molecules stacked end to end). See Fig 1.

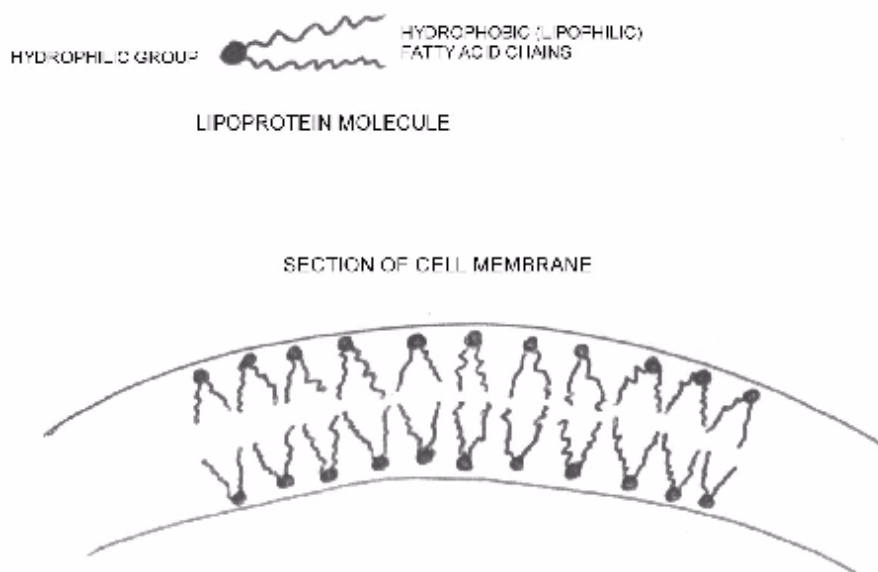


Fig 1. Lipoprotein Molecule and Section of Cell Membrane

As you can see from the diagram each lipoprotein molecule has two different ends - one marked hydrophilic, the other marked hydrophobic (it can also be called lipophilic). What this means is quite simple - the hydrophilic end of the molecule is attracted to (and attracts) water, the other end is repelled by (and repels) water - but it also is attracted to (and attracts) lipids. Lipids are basically “fat” or “oily” molecules.

So the cell membrane automatically organises itself in a simple way. The cell has water both inside and outside, so the hydrophilic ends of the molecules tend to face outwards where they are attracted to the water. The fatty (lipid) ends of the molecules face each other inside the membrane, repelled by the water outside and attracted to each other.

Now bacteria have an additional wall outside the cell membrane see Fig 2. This is called the cell wall. The cell wall is different to the cell membrane. It's much thicker and stronger than the cell membrane. It's composed of varying layers of a different type of molecule called peptidoglycans. Peptidoglycans are simplistically speaking, chains of amino acids with sugar molecules attached. Normal body cells don't have cell walls - so bacteria are actually physically tougher than body cells. The cell wall is constructed from interlocked chains of these peptidoglycans organised into broad layers.

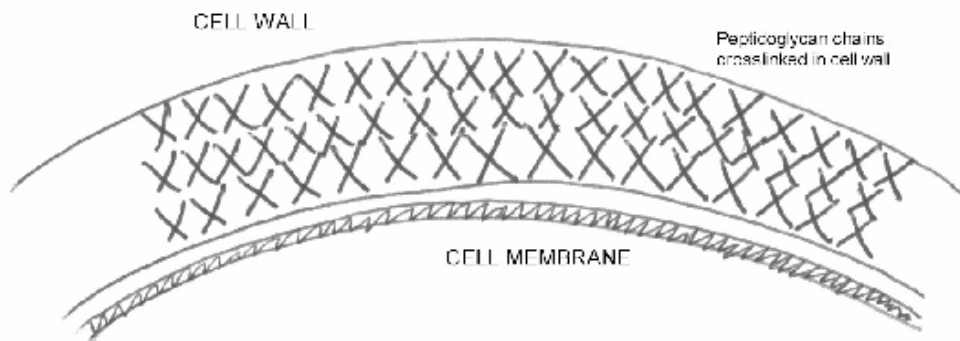


Fig 2. Section of Cell Wall and Cell Membrane

Why do bacteria have these cell walls? Well simply because bacteria are mobile - they can exist outside the body. Body cells are designed to work inside the body and not go wandering off outside the body. So when they're outside the body environment the bacteria need the extra protection of a cell wall. Normal body cells don't need such protection.

What does the cell wall protect against? In a nutshell, pressure. All cells are subject to osmosis, i.e. uptake of water because they have semi-permeable membranes that allow water to cross but not larger molecules. Because the water can cross and the larger molecules, (there is a concentration of large molecules inside the cell already) there is an osmotic

potential and the cells start to absorb water - they blow up like balloons and eventually explode.

In the body, normal tissue cells do **not** explode because the environment they live in is balanced - the fluids outside and between the cells have specific concentrations of salts designed to cancel out the osmotic potential. In this way there is no uptake of water.

But a bacterium which can go outside the body and live in something like fresh water for example needs some protection. It doesn't have this nicely controlled environment and so it needs the cell wall to stop exploding from osmotic pressure. The cell wall lets the bacterium absorb water and pressurise from osmosis, but then holds the pressure in and stops it exploding. So bacteria are pressurised and body cells not - imagine the bacteria to be like balloons, always "inflated" and pressurised with water.

Now for the sake of completeness, I need to mention a third class of cell. There are various primitive animal cells like paramecium, amoebas etc. These are **not** related to bacteria. Bacteria are not considered to be "animals", they form a class of organism called prokaryotes. Simple animals like amoeba are a different class called eukaryotes - animals (us!) also fall into the eukaryotic class.

But an amoeba for example, although similar to one of our body cells, can and does exist outside the body. But eukaryotes don't have cell walls. So how do they stop themselves from exploding from osmotic pressure? It's quite simple, they have built in "pumps" that pump water out when the pressure starts to get too high. But our body cells don't have these pumps. Take a body cell outside the body and drop it in fresh water and it will explode.

So now we know the basics of cell structure we can look at some more detail about the processes and regulation of cell systems and get a better understanding of what's going on.

Transmembrane Potential

All cells which have any difference in the content and concentration of salts inside and outside the cell have something called a transmembrane potential.

Why? Well, salts are chemicals which consist of two different ionic groups that are bound together by electrical attraction between opposite charges. For example lets take normal salt, sodium chloride. Salt consists of one atom of sodium and one atom of chlorine. It has the chemical formula NaCl. These atoms exist as ions in these compounds.

The sodium ion (like most metal ions) is positively charged because it's missing an electron (which is negatively charged). The chlorine ion is negatively charged because it has a spare electron. Now as most people know, when you put two opposite electrical charges together they attract each other (similar charges repel). So in the case of salts, the positive ion attracts the negative ion and vice versa and this is what holds the molecule together. So pure, dry salt is tightly bound together by these electrical charges. There is no net charge on the outside of the salt because all the charges are equal and cancel each other out.

If we now dissolve the salt in water something changes. The ions of the sodium and the chlorine separate. This is due to various reasons. The water can inject or absorb some electrons and form its own ions. Water is H₂O and forms ions of H⁺ and OH⁻. Also the

water molecules can shield the sodium ions from the chlorine ions and so weaken their attraction for each other. The whole process is called ionic solvation.

So in a solution of salt in water we end up with 4 species of different ions, Na^+ , Cl^- , H^+ and OH^- . If we add other salts to the same solution we end up with still more ions. Lets add “potassium salt” KCl . We then add ions of K^+ and more Cl^- .

Now real cells use specific ions for specific chemical purposes. Sodium and potassium are chemically very similar but not identical. So the cell can “fine tune” some biochemical processes by using some sodium for some reactions and potassium for others. Because of this each cell has an optimum amount of sodium and an optimum amount of potassium.

For the purposes of this explanation we don't need to know anything about the actual amounts of sodium and potassium needed. What is important however is the *ratio* of sodium to potassium.

For various biochemical reasons most cells actually need more potassium than sodium to be inside the cell and more sodium than potassium to be outside. I mentioned before that sodium and potassium are very similar but different. This is important.

In practice they are both very small ions and the cell walls and particularly the cell membranes will allow them to pass through.

Now because the sizes of the sodium and potassium ions are different (potassium is bigger than sodium) if you have a limited amount of space (like inside a cell) you can only fit a certain number of them in, and the number of sodium you can fit in will be more than the amount of potassium you can fit in the same space.

Remember that both sodium and potassium ions are positively charged? When you think about it then, it figures that you will have a different number of potassium or sodium ions in the same space depending on the concentration of each, and the fact you have a different number means that there will be a difference in the amount of positive charges (one per ion) inside the cell also depending on the concentration of each type of ion.

This is very important, because if the number of charges inside the cell is not “balanced” with respect to the number of charges outside, then the cell as a whole will take on an electrical charge - in effect you will see an electrical voltage appearing across the membrane that separates the cell contents from the outside.

This voltage appears across all real cells and is called the transmembrane potential.

Changes in the Transmembrane Potential

From the above, although greatly simplified, it's fairly clear that the amount or magnitude (the voltage) of the transmembrane potential will depend entirely on the relative concentration of sodium and potassium inside and outside the cell.

In reality there are more types of ions than just sodium and potassium. For example calcium and magnesium also contribute. But in normal body cells the sodium/potassium ratio is the main factor in determining the transmembrane potential.

Since the cell needs specific amounts of sodium and potassium (as well as other ions) for specific reactions, it has mechanisms actively transport the ions it needs to where they are needed. These are typically “pumps” that actively pump the ions across the membrane. The pump uses ATP (adenosine triphosphate) as its power source.

In a body cell, there is a major pump that deals with sodium and potassium. It pumps potassium into the cell and sodium out of the cell. If the pump didn't operate, the levels of sodium and potassium would stabilise until the ratio would be wrong for optimum biochemical processes.

As the pump operates, it automatically generates the transmembrane potential. Not directly, but indirectly by balancing the ratios of sodium to potassium. In most normal body cells the transmembrane potential stabilises out typically between -50mV and -90mV with an average of -70mV.

If a cell is damaged or diseased the pump can be impaired and so the transmembrane potential will change. However when the cell divides the transmembrane potential also changes - it drops much lower, typically around -15mV until the division is complete.

As an aside, cancerous cells typically have transmembrane potentials around -15mV.

An external voltage applied to the cell can also change the transmembrane potential. The effect is interesting. If the external voltage causes the transmembrane potential to drop below normal, the cell is said to be depolarised. The cell will try to stabilise the problem by increasing it's rate of ionic pumping to bring the transmembrane potential back to normal. If the rate of the pump increases the cell uses more energy and burns more ATP. Eventually it can become exhausted and die. Some cells will respond by dividing in the hope that more than one will increase overall chances of survival - this is probably one of the causes of cancer - uncontrolled division.

If the external voltage however causes the transmembrane potential to increase, the cell is said to be hyperpolarised. In this instance the cell doesn't need to pump, the voltage causes potassium to migrate in and sodium out naturally. In effect it drives the pump in reverse. There is an extremely interesting side effect however - if the pump is driven in reverse it actually *manufactures* ATP! It's truly reversible. Since ATP is the fuel of the cell, hyperpolarisation actually “charges up” cells by refuelling them literally.

Body cells and other cells

Most of the above discussion applies to body cells. It also applies to other cells (bacteria and simple eukaryotes) but these latter types tend to rely on slightly different but very similar mechanisms - they tend to use proton pumps to regulate their processes rather than sodium/potassium pumps. Proton pumps simply pump H^+ ions. Since H^+ ions are usually what determine pH in solution they can also be considered to be pH pumps.

There is one very major difference between sodium/potassium pumps and proton pumps. Proton pumps are much more sensitive and “breakable”. A body cell can withstand depolarisation for several minutes without much ill effect. It can withstand hyperpolarisation

much longer. The effect on the sodium/potassium pump is easily reversible and not very damaging.

A proton pump on the other hand can be badly disrupted almost immediately by either depolarisation or hyperpolarisation. And since the pump regulates pH, any sustained change in the transmembrane potential can cause the cell to die by pH imbalance - it literally dies because of too much acidity or alkalinity.

So the transmembrane potential is critical to the operation of cells.

The Lakhovsky Effect

Georges Lakhovsky, a bioelectrical researcher in the early 20th century developed a theory that the cells of the body could be charged up by applying broadband electrical noise to them. He theorised that if you subjected the body to a broad range of electromagnetic harmonics that some of the harmonics would “charge up” some cells and others would charge up other cells. By charging up the cells in this way he reasoned that he was revitalising the body and could cure disease.

The reason he decided to use broadband harmonics was because he realised that not all cells have an identical electrical environment. Because of this each would have an optimal frequency which would charge it up. But because it was impossible to calculate all the possible variations in real electrical properties of real tissue, he decided that broadband noise would give all the different frequencies needed. He also postulated that the body would only “take” what was needed. That cells which were underpowered would only absorb enough energy to bring them up to optimal potential. Any harmonics which didn’t get absorbed directly by body cells would simply pass through harmlessly.

I don’t know if Lakhovsky knew about transmembrane potentials - but he was amazingly close to the truth. If you take Lakhovsky’s “charging up” of cells to mean hyperpolarising them, then the theory makes a lot of sense. Because, as already explained above, if you hyperpolarise a cell, then you refuel its ATP supply by driving the sodium/potassium pump in reverse.

What about the harmonics? Well I believe Lakhovsky was right about that too. And this is why I suggested that a broad harmonic spread was essential for the Rife effect (I’ll explain the relation between the two in a little while). Because as I tried to explain in the last paper - the electrical characteristics of the body cells (in fact all cells, pathogens as well) vary enormously because of changes in electrical permittivity. And every different type of cell and tissue will absorb EM radiation (or alternating electrical current) at a different, specific frequency, just like a huge number of different radios tuned to different stations. If a cell membrane absorbs radiation (and the cell membrane will be the main structure that experiences external radiation) then the radiation must either depolarise or hyperpolarise the cell membrane.

We already know that cells have active pumps that maintain particular ionic balances and particular transmembrane potentials. Because of this, radiation will be absorbed more easily if it tends to “go with” the pump. If it opposes the pump it will be resisted by the pump. So the cell membrane can act a bit like an electronic diode - it will let current flow more easily in one direction than the other. As a result, such radiation is much more likely to hyperpolarise

cells than depolarise them. And if that is the case, then the cells will be more likely to be charged up and refueled than the opposite. Electronic engineers call this effect “rectification”.

So what I am suggesting is that the Lakhovsky effect is real and that by subjecting cells to broadband electrical noise (harmonics) we can hyperpolarise them. We need the harmonics to cover all the possible “tunings” of different cells in different electrical environments and permittivities.

Rife v Lakhovsky

So is this the whole story? Are the Rife and Lakhovsky effects one and the same thing? The answer is no, they’re not. But I believe they’re intimately related.

The Lakhovsky effect on its own is not supposed to kill bacteria. The Rife effect is. The Lakhovsky effect may kill bacteria by damaging their sensitive proton pumps but in general it just “charges cells up” and makes them more active.

The Lakhovsky effect is a healing modality. Nikola Tesla designed a machine that Lakhovsky used to treat patients with great alleged success. Modern machines such as Dan Dial’s Molecular Enhancer also operate on the Lakhovsky principle and have been shown to have healing effects quite distinct from modern “Rife” machines.

But Rife machines rely on an MOR - a single frequency designed to kill pathogens. So how does the MOR relate to the above? That is what I intend to explain.

Tuning

Unfortunately we need to learn a bit more electronics to carry the explanation properly.

Let’s start with tuning. Above I said that each cell has one frequency that will charge it up, or hyperpolarise it - this is *not* the MOR so don't get this bit confused.

Why one frequency? Well it’s like a radio - each cell has a specific tuning or electrical resonance that will cause it to accept one frequency better than any other. To fully understand this we need to look at what tuning is and how it works in electronics. It’s essential to fully understand this to make sense of the whole theory I’m proposing.

In electronics we make tuning circuits by using two types of electronic components - capacitors and inductors.

A capacitor consists of two electrical conductors (i.e. metal plates, but any conductor will do) with an insulator sandwiched between them. The insulator in a capacitor has a special name, it’s called the dielectric. I want to explain more about capacitors later because I believe this is essential to understanding what I believe the Rife effect to be.

An inductor is typically a coil of wire, but any conductor has some inductance.

Capacitors have an electrical property called capacitance and inductors have a complementary property called inductance. Each property is responsible for a reactance -

remember in the last paper I mentioned that reactances were a kind of resistance specific to waves. Each property is also intimately related to the two special physical properties I mentioned in the last paper - permittivity and permeability.

Now capacitance (the property not the component) is found wherever there is permittivity. And inductance is found wherever there is permeability. As I mentioned in the last paper - every single substance in the universe (even the empty space of the universe itself) has some permittivity and some permeability - so it follows that everything has some capacitance and some inductance.

Now there is special third property we haven't explained yet, and that is resonance.

It just so happens that whenever you find a capacitance combined with an inductance, the combination has the property of resonance - i.e it will "tune in" or resonate when it's subjected to a specific frequency. The frequency of resonance can be calculated if you know the capacitance and inductance. It doesn't matter *how* the capacitance and inductance is combined - there are different electrical effects in different combinations but the fact of resonance per se depends only on there being a capacitance and an inductance, nothing else.

The formula for the frequency of resonance is:

$$\frac{1}{2\pi\sqrt{LC}}$$

Where L is the inductance (in Henries) and C is the capacitance (in Farads)

But hang on a moment - I just said that *everything* - including empty space has permittivity and permeability and that wherever these two are found so is capacitance and inductance. So doesn't that imply then that *everything* in the universe (including the universe itself) has a specific resonant frequency? Simply, *yes*, it does!

I won't go into the wider implications of this but it's an interesting topic in itself!

In electronics we can deliberately exploit the property of resonance. What we do is we connect a capacitor to an inductor. We then adjust the values of the capacitance of the capacitor and the inductance of the inductor according to the equation above until the combination resonates at the frequency we want.

What use is this? Well we can use it as tuning for a radio - in fact that is how radios are tuned. The tuning dial of a radio typically changes either the capacitance or the inductance in the circuit until it resonates at the radio wave frequency we want to pick up.

When waves come in, ones that don't match the resonant frequency of our capacitor/inductor circuit don't have much effect - the ones of the right frequency cause a voltage to appear across our capacitor and inductor combination. In other words, waves of the right frequency get converted into voltages in a tuning circuit.

Tuning a cell

I said before that each cell has a specific frequency which will hyperpolarise it. We can now get some idea how this works by using the explanation above. We know that *everything* in the universe has a resonant frequency - so of course cells do too! So if we feed EM waves of the right frequency into our cell, one particular wave of one particular frequency will be converted into a voltage that will appear across our cell. That voltage as we already know will tend to hyperpolarise the cell membrane.

And each different cell, each different type of tissue, will have a different resonant frequency. Why different? Because each different type of tissue has a different permittivity as I explained in the last paper and for this reason will have a different capacitance. If the capacitance is different the tuning frequency is different as explained by the resonance equation above.

Back to harmonics - in accordance with the above and as described by Lakhovsky - if you want to charge up all cells without needing to know exactly what type of tissue they're in, or how big they are, or what their electrical properties are - you need to simply feed in lots of frequencies at once and hope that some of them will resonate with the cells and cause a change in the transmembrane potential.

This is the reason why I mentioned in the last paper that a broad harmonic signal content was needed - we can't possibly affect even one single cell with just one frequency - because its resonant frequency must change with permittivity (capacitance). And the permittivity changes with frequency itself (strange but true) and with dozens of other factors as well, including even what's adjacent to it!

Harmonics and MOR's

The point most misunderstood in my last paper was that I tried to explain that using harmonics has nothing to do with MOR's. When we first read about Rife technology most of us accept the idea that each pathogen has (at least) one resonant frequency that kills it - the MOR. We then make the assumption that all we have to do is to feed that frequency to the pathogen and it will die. But this assumption is wrong and this is the new concept I mentioned in the last paper. The assumption that there is such a thing as an MOR is *not* wrong. But the assumption that we can produce the MOR by exposing the cell to *just* the one, single MOR frequency on its own *is* wrong.

So up until now everybody has assumed that when someone suggests using lots of harmonics (i.e. lots of frequencies at once) the reason had to be because we were hoping that one of the frequencies just might happen to be the MOR and might kill the pathogen.

What I am suggesting now is completely different. I am now proposing that the reason why we should use harmonics is to cause a Lakhovsky type effect and to charge up the cell by changing its transmembrane potential, *not* in order to just randomly hope we hit the MOR. Hyperpolarising the cell on its own does not necessarily kill the cell - which is a good thing, because if it did, we'd kill ourselves with any exposure to radio frequency noise!

You're probably still confused! :-)) But it's not that difficult and I haven't finished the explanation yet.

What you're probably confused about is why we would want to hyperpolarise the cell membrane if this is *not* the MOR. Because after all we're trying to build a Rife machine, not a Lakhovsky machine aren't we?

You're right. But what I want to go on to explain now is why I think the Lakhovsky effect is an essential part of generating an MOR - it's not the answer in itself, but it's part of a bigger picture which I hope will become clear.

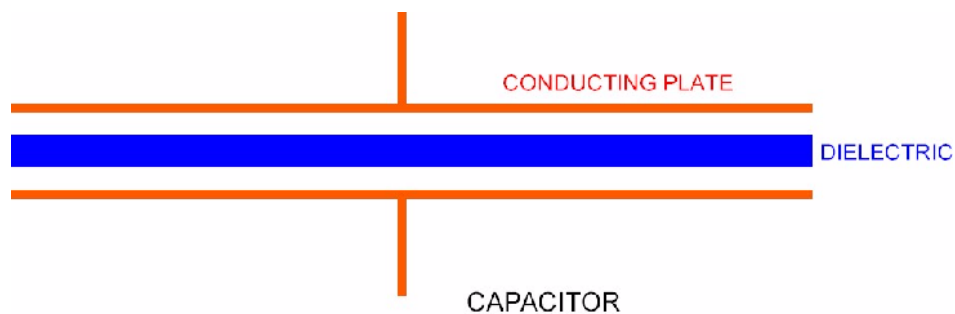
In order to fill in the last part of the puzzle I need to go into some more detail about capacitors. The cell is very much like a capacitor and by understanding a capacitor we can hopefully understand the electrical effects in a cell much better.

Capacitors

I explained before that a capacitor consists of two conducting materials with an insulating material sandwiched between them. The insulator is called the dielectric of the capacitor.

We can easily make a capacitor by taking two metal plates and putting a sheet of insulating material between them - even a sheet of paper will do. The bigger the conducting plates, and the thinner the insulator, the greater the capacitance.

If we want to change the capacitance of the capacitor we can do various things - we can alter the size of the plates (or arrange the plates so that more or less of them overlap). The other thing we can do is to change the material of the dielectric, or change the thickness of the



dielectric. The key property of the dielectric is its permittivity.

Remember if we change the capacitance of a capacitor, we also change the resonant frequency of any circuit its connected to (assuming the inherent inductance stays the same). The second thing to remember is that the capacitor doesn't need to have metal plates - any conducting material will do, it doesn't need to be metal.

Capacitors have several interesting electrical properties. We don't need to go into all of them. The important one for this explanation is that they hold charge; they act like little batteries - you can charge them up and they will keep the charge for a while when the power is switched off. They slowly lose the charge but this is because of other factors in the real world - if we could build a perfect capacitor it would stay charged up forever.

Electronics engineers study capacitor theory and learn how they work. But what is often neglected in conventional electronic teaching is any explanation of what happens in the dielectric (the insulating sheet) when a capacitor is charged. We need to look at this.

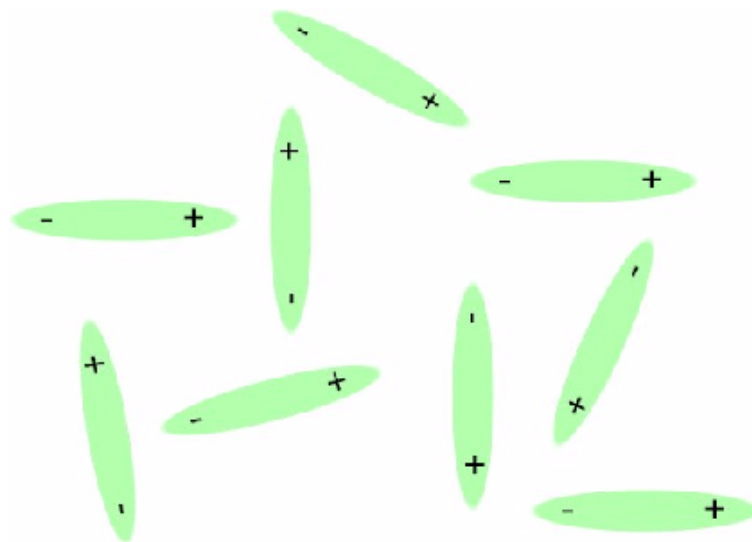
The dielectric is the insulator between the conductors of the capacitor. It's easy to think it's just some inert material that never changes and is never affected by the electrical signals that are fed into the capacitor. This is not true.

The dielectric consists of molecules just like everything else. And molecules react to electrical charges.

We know from basic physics that two different electrical charges attract each other, and that two identical charges repel each other. Well molecules can have charges too. In fact most molecules have some sort of electrical charge distribution inside them - because molecules are made from atoms and atoms have electrons. Some molecules have distinct charges at each end, these are called molecular dipoles and are usually arranged randomly inside the material they are part of. Molecules are held together by different distributions of electrons - this is what chemical bonds are all about.

If we put a molecule in an electric field, the charges of the field will have an effect on the charge distribution in the molecule. And this will affect the chemical bonds of the molecule. the stronger the electric field, the greater the effect.

If we charge up a capacitor we are effectively putting a positive charge on one of its plates,



RANDOM ARRANGEMENT OF MOLECULAR DIPOLES IN NORMAL DIELECTRIC

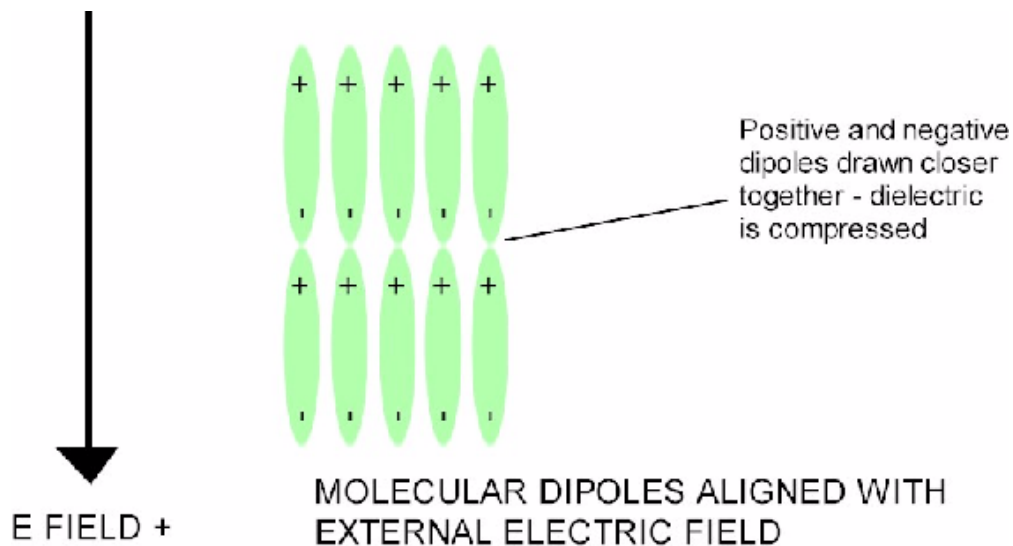
and a negative charge on the other. In between the plates there will be an electric field.

But we already know that between the plates of the capacitor is the dielectric. So this means that when we charge a capacitor we are putting the dielectric in an electric field. And this in turn means that we are putting the molecules that the dielectric is made of in an electric field.

So what will happen? Well we know that opposite charges attract and that like charges repel. If the molecule is capable of significantly rearranging its internal charge distribution (i.e. it's a polar molecule), then what will happen is that the molecules will rearrange their internal charge and will line up so that their internal charges are facing the opposite polarity of the external electric field.

But if the charges in all the molecules suddenly line up with the external electric field, then each molecule in each layer will have its positive end adjacent to the negative end of the molecule in the layer below, and vice versa. And the molecules will not just line up, but they will attract each other as well. Normally the molecules are either arranged randomly, or they are lined up and spaced only in accordance with their own field (as opposed to an external one).

If the dielectric is a soft material (i.e. not very rigid) then this means that the dielectric itself will compress when the capacitor is charged up. When the capacitor is discharged, it will expand. The compression of the dielectric when the capacitor is charged is known as



dielectric stress. This is a good term because the dielectric *is* under stress when the capacitor is charged.

In real electronic capacitors the dielectric is pretty rigid and designed not to suffer from easy molecular charge redistribution.

But if we made a capacitor with a dielectric that was a bit soft, and was made of what are known as polar molecules (molecules which can take on asymmetric charge distributions) then the dielectric in this capacitor would be quite stressed every time it was charged up. The lipoproteins that form the cell membrane are polar molecules like this.

Now let's take this one step further. What would happen if we charged a capacitor like this up and then discharged it, and then kept repeating that? Well, as the capacitor charges the dielectric will be stressed one way (compressed). As the capacitor is discharged the dielectric will be stressed the other way (expanded). If we kept charging and discharging repeatedly then the dielectric would be constantly stressed one way or the other.

I'm sure everyone has at some time taken a piece of metal (such as metal spoon) and bent it back and forward a few times. What happens? The metal breaks. Real materials can usually stand being stressed once - but if you keep stressing them in opposite directions eventually they break.

If you haven't switched off yet, good for you, because now we come to the crux of the matter.

The Cell as a Capacitor.

Remember I said that a cell is a form of capacitor? It also has some inductance - all real materials do. So it has a resonant frequency but the capacitance is the more important property in cells.

A cell consists of a cell membrane (or a membrane and a cell wall) which partially blocks the flow of ions across it. In other words, it's mainly an electrical insulator.

The inside of the cell consists of water containing various ions, maybe a bit more potassium than sodium but it doesn't matter - the inside of the cell is electrically conductive.

Where is the cell? It's suspended in intercellular fluid which is mainly water that also contains various ions. The intercellular fluid is also electrically conductive.

So the cell wall or the cell membrane (or both) are in fact insulators suspended between two conductors - in other words the cell membrane/wall is a dielectric and the cell is a capacitor! Not only that, but the cell membrane consists of polar molecules.

We already know that the cell can be charged up - just like a capacitor - we can hyperpolarise it by applying the right frequency which it will "tune into" and change into a voltage.

And we also know that if we charge up a capacitor and it has a soft, polar dielectric then we will stress the dielectric.

The result is quite simple: if we can find a way of charging and discharging a cell repeatedly, then we will stress its dielectric (the cell wall and/or cell membrane) back and forward just like the piece of metal described above. And what will happen? Eventually it will break!

What happens if the cell wall breaks? The cell will explode under its own osmotic pressure. If the cell membrane breaks the regulatory pumps will break down and the cell will die. Either way the cell will be killed.

It's interesting to note that there are two distinct possible effects. If the cell wall breaks the organism will visibly explode. If the cell membrane breaks but not the wall, the organism may not explode - it will just die. In other words it will be "devitalised".

The Rife Effect

So now we have a possible explanation for the Rife effect. If we can repeatedly hyperpolarise and depolarise a cell then it's likely that we will destroy its outer wall or membrane due to dielectric stress.

But not just any rate of charging and discharging will do. The cell membrane is quite soft, the cell wall is more rigid, but nonetheless "soft" compared to a piece of metal. If you bend something soft back and forth it'll be harder to break than a piece of metal.

But if you can bend a soft material at any rate you like, then you will find there is one point, one frequency at which the energy from the stresses of the bending action will accumulate and if you manage to bend specifically at that rate you will break even a soft object. The rate of discharge in a real cell depends on the electrical conductivity of the cell, if you charge very quickly and do not allow enough time for discharge, then the cell will not be as stressed and the charging/discharging may not have any effect. So there is an optimum rate for maximum stress.

I believe this "optimum rate" of stressing is the Rife MOR.

Now, hopefully all will become clearer. I mentioned before that we can use a spectrum of broad band harmonic waves to charge up or hyperpolarise the cell membrane - although I've stuck to membranes it's not hard to extend the theory to cell walls.

But this is just the charging phase of the capacitor, we haven't accounted for the discharging phase. Remember that I said that real capacitors don't hold charge forever, they discharge themselves? Well so do cells.

So if we feed our spectrum of harmonics to the cell we charge it up. How do we discharge it? Quite simple - we switch the harmonics off so the cell stops charging. The moment it stops charging, it starts discharging automatically because it is "connected" to a conducting medium - the intercellular fluid.

And this is the concept I tried to convey before. The MOR of the cell is the rate of charging and discharging of the cell membrane/wall. It has nothing to do with the harmonics - the only reason we use harmonics is to charge the cell up in the first place. What the MOR is, is the frequency at which we switch on and off the flow of harmonics.

So the *method* of charging the cell (the harmonics) is independent of the *rate* of charging/discharging the cell (the MOR).

So if we design a machine that can produce a spread of electrical harmonics we will charge up cells. If we then simply gate those harmonics on and off we will create the MOR.

The MOR has nothing to do with the pure electrical resonance of the cell. The electrical resonance only determines the tuning of the harmonics needed to charge it up in the first place. The MOR is not exactly a form of direct electrical resonance - if anything it's an indirect, secondary electromechanical resonance related to the structure of the cell's outer covering.

This is the “revolutionary” concept. It’s that an MOR is an indirect, secondary effect that requires a primary effect to enable it. The harmonics are what enable an MOR but are distinct from the MOR itself.

The distinction is vitally important.

One final point needs to be clarified. Does the cell have only one MOR? I think probably not, certain harmonics of the MOR frequency (nothing to do with the charging harmonics above) will also cause stress on the membranes. What we’d expect is that the fundamental MOR will kill the cell most quickly, the harmonics will also do the job but may take longer.

You may ask why it is necessary to go to all this trouble for a new theory. The reason is that basic physics shows that the traditional assumptions of Rife research must be wrong. This is not something I’ve just made up, it’s hard, technical fact.

I realise that some people in the Rife community won’t like this idea - but liking it or otherwise doesn’t change the facts - the electrical environment of real cells and real tissues is so complex that the previously assumed simplistic effects cannot possibly be true under any circumstances.

Conclusion

I concluded from all the technical analysis in this and previous papers that three factors are essential components of the Rife effect.

1. Broad band harmonics or electrical noise is essential to hyperpolarise real cells in widely varying electrical environments inside the body.
2. The rate at which the harmonic spectrum is gated on and off comprises the MOR - the MOR is essentially independent of the wider electrical properties and so the MOR is constant, it doesn’t change with tissue permittivity etc.
3. The delivery device (i.e. plasma tube) needs to have a constantly variable output impedance to match optimally to the diverse range of impedances found in real tissue. I suggest that one way this may be possible is by ramping the voltage and/or current of the tube up and down.

I really hope that most readers will be able to follow through the whole technical argument. I’ve greatly oversimplified a lot of things (it may not seem like it!) but I have. There are numerous other implications as well that I’ve avoided going into because they will only be understood by specialist biologists, chemists and electronics engineers.

For the benefit of specialists I can add a few points:

The dielectric stress is likely to result in separation of the lipoprotein layers of the cell membrane, because there will be strong lateral stress component due to the obvious polar nature of lipoproteins. The isolated lipoproteins are basically endotoxin. The release of endotoxin will trigger Schwartzmann reactions in a live patient which may be mistaken for Herxheimer reactions.

The change in the physical dimensions of the dielectric layer in the cell wall during hyperpolarisation will cause a constant change in both the resonant frequency of the cell (due to the change in the capacitance) and its impedance. In order to sustain oscillation a constant changing harmonic pattern will be necessary to ensure that tuning and hyperpolarisation are maintained. In more simple terms, as you charge up a cell it's tuning will drift and you will lose resonance, so you need to alter the frequency constantly to "follow" the cell's change in resonance point.

A high harmonic content will ensure that some frequencies are "tuned in" by the cell and result in hyperpolarisation. Hyperpolarisation is more likely than depolarisation because the natural ionic potential of the cell will ensure rectification.

There will be constant change of impedance which is why I suggested ramping the plasma tube voltage up and down. Ramping the current up and down (for constant voltage) will do the same thing. The important thing being to ensure that the output impedance of the tube is constantly changing in order to ensure better impedance matching with the target cells. A good impedance match will result in improved energy transfer from tube to cell wall and minimal reflection and wasted power. I believe that a plasma tube is acting as a basic EM antenna but with a variable impedance unlike a conventional antenna which is why it gives a better bioactive effect. But I think it can be improved further by ramping. I also believe that this kind of ramping was a natural consequence of super-regenerative circuitry used by Rife in the original machine.

I have not discussed refraction and reflection effects. Both will occur at interfaces between different tissue types. Enough energy will be needed in a wide dispersal pattern to prevent the possibility of energy being refracted away from the target and/or being reflected off it as opposed to being absorbed.

Pad machines will have different effects from plasma machines but some of the basic principles should still hold regardless. The impedance should be less of a problem, but a wide harmonic spread in current should have the same effect on hyperpolarisation, and is therefore theoretically desirable.

You may be wondering how a small voltage across a cell membrane like 90mV or so can cause dielectric stress because the E field seems very weak. In fact it isn't - remember that a cell membrane is only a few nanometres across - so the E field strength is very high. For example for a 90mV hyperpolarisation and a cell membrane of 10nm, the E field strength is:

$$90\text{mV}/10\text{nm} = 9,000,000 \text{ V/m}$$

In other words, the E field strength can be 9 **MILLION** volts per metre in a real cell membrane! That's hardly a weak field! :-)

The transmembrane potential of a normal cell depends only on the relative ratio of sodium/potassium ions inside and outside the cell. The voltage can be calculated according to the standard electrochemical formula, the Nernst equation. Actual measured potentials in real cells correspond exactly to the predicted Nernst equation values.

What is most interesting is that the potential for any membrane at the point when sodium "saturates" the cell just happens to be -15mV which is the same potential observed in cancer

cells and normal cells that are undergoing division. I'm sure this has important implications for potential cancer treatments.

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