The Problem with Randomness

How Quantum Mechanics Impacts the Formation of Cancer

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Abstract

A quantum mechanical view of cancer is one that is rarely discussed. The effects that quantum phenomena have on the formation of cancer are even less studied. This paper brings together a wide variety of research, including the works of some of the greatest scientific minds of our time. The paper seeks to evaluate whether quantum tunneling significantly impacts the formation of cancer-causing mutations to direct future experimental research. In addition, it tries to create an easily digestible resource that covers both the quantum mechanical and the biological foundations of the subject in a way that requires no prior knowledge of these concepts to understand. This is necessary because the data reviewed suggests that there may be a significant likelihood that quantum tunneling is potentially a major cause of cancer, justifying future research.

1. Introduction

Reality is *solid*. This is our most basic way of understanding the world around us. One can place their hand on a desk without it falling through. Most physical phenomena, be it a fly landing on a table or the colliding of planets lightyears away from us, seem to have an underlying truth to them: they are the product of the actions of solid objects.

Even something as pervasive as air is usually seen as something ethereal: science tells us that there should be something there – we breathe it, after all – but, since one cannot see air, we think of it as if it is nonexistent. It is as if for something to seem *real*, it must be solid.

Perhaps this is why it is so jarring to be told otherwise. Advancements in physics have shown that, in fact, not even solids are truly solid. While we have known about the existence of atoms since the early 1800s, quantum mechanics asserts that matter is in a state of constant flux. But what exactly does this mean in terms of cancer? Does it pose a risk to the general population? To find this out, we must first dissect this quantum physical view of matter.

2. Physical

At its very core, quantum mechanics is based on a few key ideas. One of the most important of them is known as Heisenberg's Uncertainty Principle. In our world of solid masses, measuring the speed of an object is relatively easy. A policeman, for example, can measure the speed of a car using machinery that is widely available. However, the more one zooms in on the world around them, the harder it is to accurately measure the speed and position of an object. If we were to isolate a minuscule particle, such as an electron, we would find that it is impossible to accurately calculate both its speed and position. Despite our best efforts, we can only calculate one of these values precisely.

This is where the Uncertainty Principle comes in. It tells us that there is this trade-off between speed and position. (Busch et al., 2007). The more we know about a particle's speed, the less we know about its position and vice versa. In effect, this means that we can never know exactly where a particle is. If we try to measure both the speed and position of a particle, the results become blurred (Schirber, 2009), that is, both do not represent the actual position of the particle, but rather an approximation of the true value. To understand this concept more, let us go back to the electron.

Keeping the Uncertainty Principle in mind, let's construct a model of the behavior of electrons when they are a part of an atom. Thinking about electron motion harkens back to a familiar model of the atom: one where electrons neatly orbit the nucleus in circular paths, not unlike how the planets in our solar system orbit the sun (Schwarz, 2013).

Let us try to apply the Uncertainty Principle to this model of the atom. We know that the electrons in an atom, being subatomic particles, are subject to the Uncertainty Principle. One can measure the position of every electron in an atom, but this poses a problem. Knowing only the positions of the electrons makes it impossible to predict their future motion around the nucleus. Any scientific model worth its salt must be able to make predictions. After all, trying to predict future events is one of the reasons why science exists.

However, we cannot use only the speed of the electron, either. One cannot predict where a car is going to be based on the statement that "It is traveling at 100 miles per hour." Similarly, one cannot predict the future position of an electron given only its speed. This leaves us with one alternative: to use the blurred values given when we try to measure both speed and position.

Naturally, neither of the blurred values will be very precise. But they will tell us the general location of where the electron will be. In order to use this for any predictive models, we must harness the power of an idea that lies at the heart of quantum mechanics: the Probability (Rédei & Summers, 2007).

An electron in a water molecule that is in a drop of rain falling over Spain will almost certainly not appear on the other side of the universe. If we took blurred measurements of the electron's position and speed, we could piece together where the electron is likely to be located. (Dirac, 1926) We can picture this space where the electron is probably located as a spherical shell encasing the atom. The further out we go from this shell, the less likely the electron is to be positioned there. The same applies for the further in we go.

If we combine these different layers of probability surrounding the atom, we get a sphere – a 'cloud' of probability that gradually fades out at the edges. We can use this to predict the most likely position of the electron at any given moment. This is widely accepted to be the true model of the atom – the aptly named Electron Cloud Model (Vlasov, 1993).

However, the outer edge of the electron cloud never really tapers off completely. While the electron will almost certainly not be a few inches away from the atom – an almost astronomical distance for a subatomic particle – the probability of it happening never quite reaches zero. After all, we can never be exactly certain where a particle is until its position is measured.

All this information points toward one thing: a more fluid, gelatinous picture of reality, with the location of particles being hazy rather than clearly defined. Our typical view of the world is significantly altered when this is taken into account.

To understand how this radically changes our view of cancer, we must also understand the chemical basis of genetics.

3. Biological

DNA, or Deoxyribonucleic Acid, holds the key information necessary to construct biological forms. The precise process that genes follow is somewhat more complex than this simple statement would imply, however.

It all starts in the nucleus. The nucleus lies inside our cells and is what sets our cells apart from that of bacteria and other simple organisms (Vellai & Vida, 1999). The heart of the nucleus contains a gigantic mass of genetic information – about *6 feet* of DNA, all intertwined in a tiny ball that can only be described as what looks like a knitting project gone terribly wrong. This genetic tangle works out to be about 3 *billion* base pairs. (Nurk et al., 2022) But what exactly is a base pair?

DNA is a polymer. That is, it is made up of long chains of molecules that are generally referred to as monomers. The monomer that is specific to DNA and other nucleic acids are called

nucleotides. A single nucleotide consists of a chain of atoms referred to as a phosphate group, because of the presence of phosphorous atoms in the structure (Baur, 1974). This phosphate group is bound to a simple sugar, which gives the nucleotide structure. The last part of the nucleotide is the most crucial: the nitrogenous base. There are 4 different nitrogenous bases in DNA, all with slightly different atomic structures (Watson & Crick, 1953). These bases can be thought of as adding different 'flavors' to the nucleotides. The nitrogenous bases directly correspond to the various types of nucleotides, which we will get to a little later.

All in all, there are around 35 atoms in a nucleotide on average. This varies a small amount depending on which nitrogenous base a specific nucleotide has in it. The phosphate group sticks out of each nucleotide and binds itself to another nucleotide, which in turn is bound to another, and so on and so forth. In the end, this forms a long chain of nucleotides (Calladine & Drew, 1992). But this is not enough to form DNA. There needs to be another chain of nucleotides. In order to successfully bond with the initial chain, the new chain cannot be a copy of the initial. In fact, it must be the exact opposite. This leads us back to the nitrogenous bases.

The four nitrogenous bases, A, C, T, and G (which correspond to the chemical names of Adenine, Cytosine, Thymine, and Guanine) have specific preferences when it comes to pairing up. A will only bond with T, and C will only bond with G. So, if we had one strand of DNA that was comprised of the letters ATCGA, it could only bond with the strand that was made of the letters TAGCT. When the two strands bond, they coil around each other and form the familiar double helix of DNA. (Watson & Crick, 1953) A base pair is a combination of two bonded nucleotides, such as an A and a T or a C and a G.

All of the DNA in a human cell was there when that person was born. It originally came from their parents and was copied into every cell formed since. In order to utilize this DNA, every cell comes equipped with the tools to read DNA and turn it into useful materials: proteins. However, DNA is simply too massive to ever leave the nucleus. Therefore, the first step a cell must take to make use of DNA is to first make a short copy. (Bramham & Wells, 2007) If a cell needs to make a certain protein, it will make a copy of the small section of DNA that encodes for that protein.

But it only makes a copy of one of the sections on one of the strands. So, the copy the cell makes is not really DNA anymore. It is Messenger RNA or mRNA. (RNA, or Ribonucleic Acid, is the single-stranded cousin of DNA.) This short segment of mRNA can travel freely outside of the confines of the nucleus, and it eventually navigates to the cell's protein-making factories, the ribosomes. mRNA neatly slides into the ribosome, which itself is made of two strands of RNA (which are called Ribosomal RNA, or rRNA) joined together. (Steitz, 2008) In order to find out what happens next, we must recall what exactly a protein is.

Proteins are like the building blocks of tissues. We usually think of them in terms of muscle (bodybuilders drink *protein* shakes), but proteins are used to build everything from the

skin to the interior walls of the stomach. Like nucleic acids, proteins are also polymers. Instead of nucleotides, proteins are constructed from long chains of amino acids. (Richardson, 1981) Amino acids are delivered into our body when it breaks down anything containing proteins. While one usually thinks of animal products as the type of food that harbors proteins, plant products have them as well, albeit in lower concentrations most of the time.

Now, we can return to the ribosome. Once mRNA slides into the ribosome, a protein can begin to be made. This process begins with Transfer RNA, or tRNA. (Hopper & Phizicky, 2003) Each unit of tRNA has three nucleotides attached to its bottom. This is referred to as an anti-codon. A codon is a set of three nucleotides on mRNA, so an anti-codon must be its opposite. A codon and its matching anti-codon can bond together much like two strands of DNA do. For example, the codon CCG bonds with the anti-codon GGC (Cochella & Green, 2005).

The second major part of tRNA is the top part, which attaches to a specific amino acid. The amino acid carried by tRNA is determined by the anti-codon on the bottom, and, by extension, the specific codon on mRNA that the anti-codon bonds to. tRNA moves to the ribosome and hooks into a specialized ‘docking port’ that leads down through the ribosome directly to the strand of mRNA. The tRNA that has the anti-codon that corresponds to the first codon of mRNA on the strand docks into the ribosome first, bonds with the mRNA, and then deposits its amino acid (Cochella & Green, 2005). It then undocks from the mRNA strand, making room for more tRNA to travel to the ribosome and adds amino acids to a chain in the order that is specified by mRNA. In this way, proteins are formed.

The newly formed amino acid chain folds in on itself in intricate ways. The specific folding patterns of proteins are key to their functions in the body (Orengo et al., 1999). However, this contains a potential for mistakes in the protein manufacturing system: the overreliance on singular codons.

Since proteins fold using the atomic bonds between amino acids, just a single letter switch in a codon can bring the wrong amino acid into the wrong place in a protein. (Maquat, 2001) In this way, a single letter of a codon can cause the protein to misfold and, in the best-case scenario not be able to carry out its function efficiently, or, in the worst case, lethally impact the organism.

This change in DNA structure is known as a mutation. If the proteins regulating the changes cells undergo to divide fail to function, a particularly disastrous outcome can occur: cancer. (Hartwell & Kastan, 1994)

Normally, during the cell cycle, there are specific mechanisms to prevent cells with mutated DNA from spreading in the organism. This regulates the spread of mutation. Specifically, certain proteins go to work ensuring that the cell that is going to clone itself and divide has intact DNA. If not, the cell is sent instructions to self-destruct. (Lawen, 2003)

However, the regulatory protein system that is crucial to preventing the spread of mutations itself can be compromised by a mutation. This can be viewed as a fatal flaw in the cell cycle.

Cells that evade self-destruction, or apoptosis, via a mutation are freed from the constraints of the cell cycle. (Wong, 2011) They divide much faster than the surrounding normal cells, soon forming a colony of mutated cells inside the organism that continues to grow. This is what we refer to as cancer.

There are many causes of mutation, ranging from mistakes in the cell's routine copying of DNA to radiation. (Little, 2000) However, the impact of quantum mechanics on mutation, and, subsequently, cancer, has garnered little attention. The question still stands: How much of a role does quantum mechanics play in the formation of cancer? The answer may lie in a seemingly unexplainable phenomenon known as quantum tunneling.

If one wants to change something, one needs energy. One must heat water in order to bring it to a boil, and an engine needs fuel to work. Despite this seemingly universal property of matter, quantum mechanics provides a workaround, provided one is patient enough. Let us go back to the electron cloud and the Uncertainty Principle.

In classical mechanics, if a particle leaves an atom, there must be a force moving it outwards. However, in quantum mechanics, things are a bit different. This is due to the clouds of probability that surround these subatomic particles. Akin to how electrons have clouds of probability governing where they can be found, other particles such as protons also have such clouds around them as well (*The Proton Radius Problem on JSTOR*, n.d.).

As we found previously, there is a very low, but nonzero, chance that the particle will be found on the outer fringes of its probability cloud. Sometimes, the cloud extends out of the atom itself, and the particle's position can be measured to be *outside* the atom itself. Physicists refer to this phenomenon as Quantum Tunneling. In this way, particles can move without having any force applied to them. (Grifoni & Hänggi, 1998)

While this phenomenon is usually inconsequential to our daily lives, it could potentially mean disaster for our DNA. While there are billions of atoms in a strand of DNA, the number of atoms in a single nucleotide is under 50. So, in a nucleotide, every atom counts. On top of this, DNA uses relatively simple atoms. The heaviest atom in DNA is phosphorus, with 15 protons (Valsami-Jones, 2015). The rest of the atoms in DNA have even lower atomic masses. If a Quantum Tunneling event occurred with a proton escaping an atom in a nucleotide, compromising that atom's role, it very likely would make the nucleotide malfunction. If a nucleotide cannot be read, that codon does not work as intended. And, as expressed before, a single codon can cause an entire protein to malfunction (Studer et al., 2013). Even if a tunneling event happened once per *billion* nucleotides, or 6 times in the DNA of every cell, there are still around 30 *trillion* cells in the average adult human (Sender et al., 2016).

Assuming that the average chance of a proton tunneling event is one in a billion over the lifetime of a cell, that's one hundred and eighty trillion mutations per body. If the mutations are truly random, this means that 30,000 genomes' worth of nucleotides are mutated. While this is just a thought experiment to illustrate how much of an impact quantum tunneling might have on our DNA even at an extremely low probability of it happening (In the range of 0.000000001%), the thought still raises some questions.

Are tunneling events truly random? Can some pattern be discerned? If so, why does tunneling happen more in some areas of the genome and less in others? Are there things we can do to mitigate the risk of tunneling events, possibly making cancer less frequent? The truth is, we don't really know. Many more experiments are needed to answer these questions about phenomena that could be secretly pulling the strings behind one of our society's most feared diseases. The problem with randomness, it seems, is that we don't know enough to protect ourselves from it.

However, one ray of hope for a better understanding of cancer comes from researcher Megan Wolfe of Drexel University, who confirms our suspicions about tunneling. She says that, while the probability of a proton tunneling event is low, it is likely that such events do take place with some frequency, and that they could be a cause of diseases such as cancer.

4. Conclusion

Despite the significant lack of data surrounding spontaneous DNA mutations caused by quantum tunneling, it seems that the phenomenon does not play a negligible role in mutating DNA. It appears that a single codon in one of the many that play a role in forming regulatory proteins being compromised by tunneling is possible, if not probable.

The next step in studying this phenomenon should be the evaluation of exactly how much of a role it plays in the formation of cancer. It very well may be one of the governing forces in its proliferation, affecting countless lives. If this were proven, it would significantly impact the way we understand the disease and ultimately, the way we try to cure it.

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