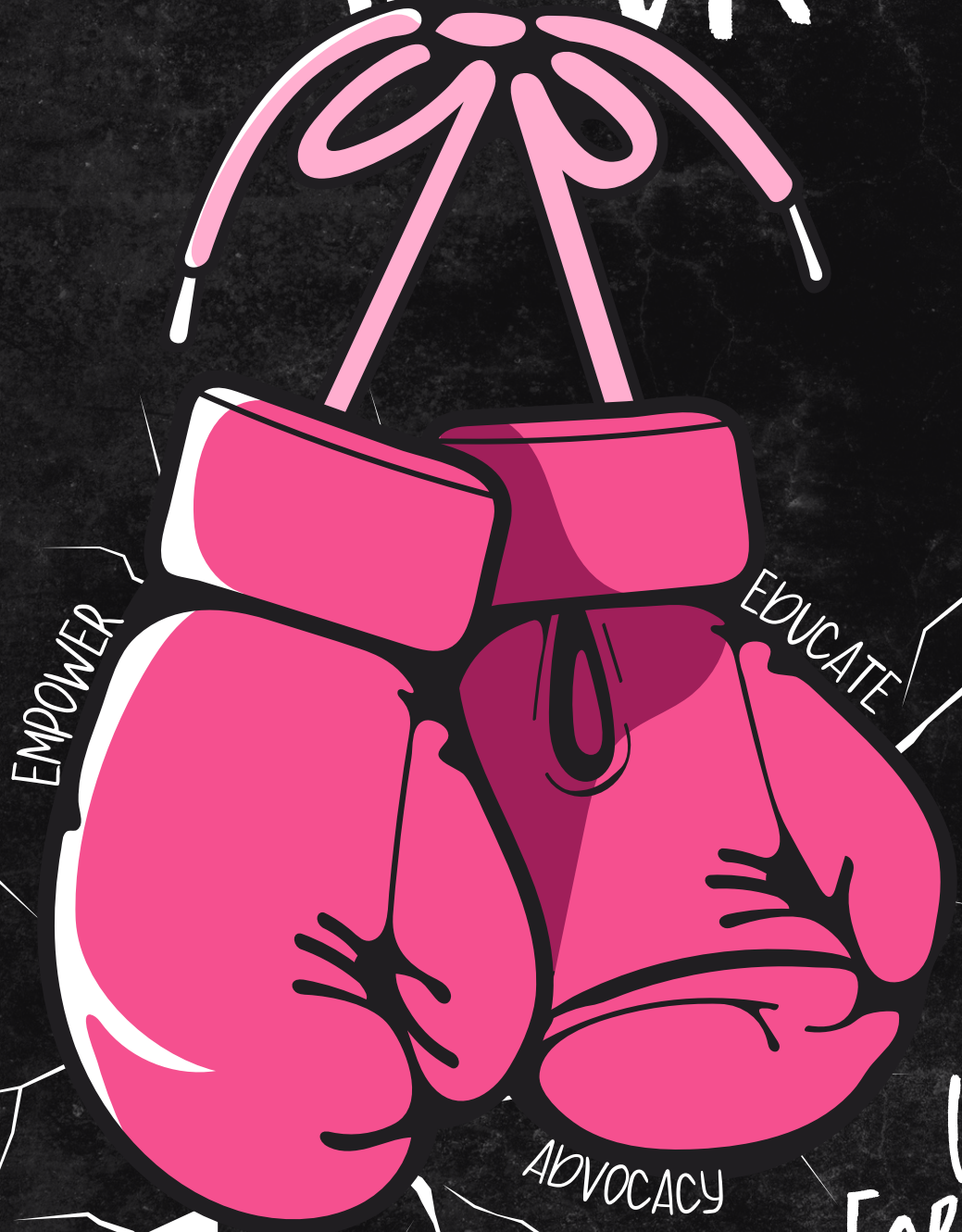
ADVOCACY IN CANCER

HRYA CANCER AWARENESS



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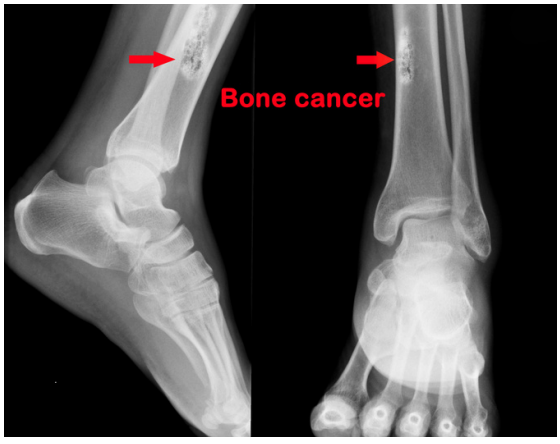
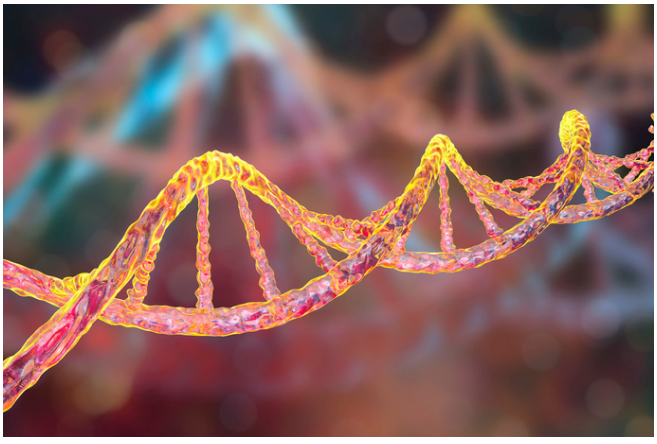
An Overview of Bone Cancer

AUTHOR - YOULAN LI

Abstract

Bone cancer refers to a specific type of cancer that occurs in any bone in the human body, in particular, affects the pelvis or the long bones in the limbs. Various bone cancers affect different groups of people in the population, such as chondrosarcoma which tends to have an impact on adults than children. Treatments for bone cancer depend on the type of bone cancer, some of the common treatments include surgical removal, chemotherapy, and radiation therapy. Even though the exact causes of the majority of bone cancer remain unknown to doctors and researchers, a couple of bone cancers have been proven to be linked to hereditary influences, whereas some of the rest of bone cancers are related to radiation exposure.





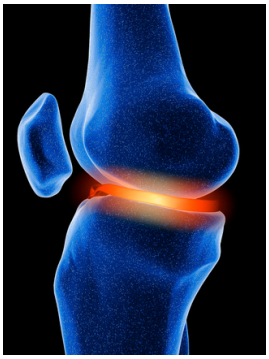
Pathophysiology

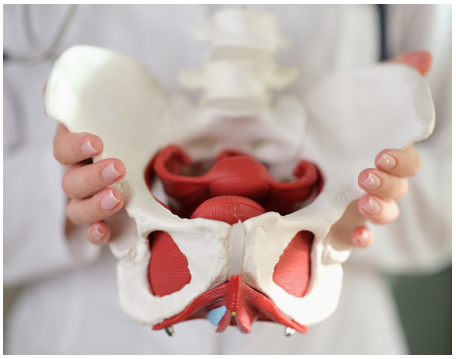
In general, bone cancer is defined by the location where it develops. As a result, the stages of bone cancer development can be classified as primary bone cancers, which are the cancers that initially occur in the bone itself. In contrast, the cancers that are later spread into the bones are named secondary or metastatic bone cancer, such as tumors in the breast, lung, and prostate tumors are some of the most prevalent locations for metastasizing into the bone. According to the statistics estimated by the American Cancer Society and Mayo Clinic, there are about 3,970 new cases diagnosed (2,160 in males; 1,810 in females), with less than 1 percent of all cancers. While some individuals may die of bone cancer, a significant proportion of patients achieve full recovery. The five-year relative survival rate for bone cancer stands at around 66.8%, reflecting the percentage of patients still alive five years after diagnosis.

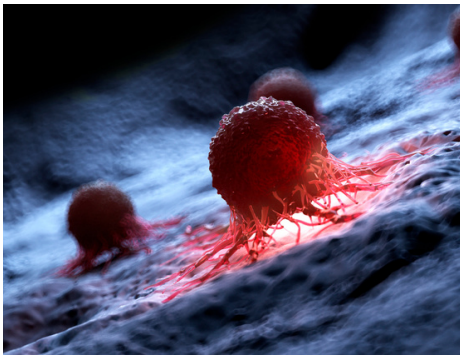
Like many other cancers, bone cancer initiates when healthy bone cells undergo abnormal changes and proliferate uncontrollably, thus forming a mass known as a tumor. Such tumors in the bone have been sorted as either cancerous (malignant) or benign (non-cancerous). A malignant tumor can invade immediately into other parts of the body, causing damage to the bone and may spread to nearby tissues. If tumor cells enter the bloodstream, they can spread and proliferate to distant organs, particularly the lungs, through the process of metastasis. While a benign tumor may also grow, it doesn't spread to other areas of the body. Even though it remains localized in the bone, a benign tumor can gain considerable size, exert pressure on surrounding tissues, weaken the bone, and lead to fractures.

Types of Primary Bone Cancer

The first type of primary bone cancer is osteosarcoma, also known as osteogenic sarcoma. Osteosarcoma is the most prevalent type of bone cancer and typically starts in bone cells in limbs or pelvis. It affects individuals between 30 and 60, exhibiting a higher occurrence in males than females. Osteosarcoma is further classified into three subtypes based on the microscopic appearance of bone cells. Low-grade osteosarcoma demonstrates gradual growth, with the majority of bone cells appearing normal, only a small fraction shows active division. Intermediate-grade osteosarcoma displays slightly accelerated growth compared to the low-grade subtype. Unlike low and intermediate-grade osteosarcoma, high-grade osteosarcoma demonstrates rapid growth. Among children and adolescents, high-grade osteosarcoma is the most frequently occurring. The overall five-year survival rate for all osteosarcoma subtypes averages around 60%, as reported by the American Cancer Society. Secondly, chondrosarcoma originates from cartilage cells and represents the second most common type of this bone cancer. The chances of getting chondrosarcoma increase with age, thus this type rarely emerges in children and adolescents. Even though chondrosarcoma predominantly develops in the pelvis and limbs, with less frequent occurrences in the ribs, skull, chest, shoulder blades, larynx, and trachea, any part of the body containing cartilage is susceptible to the attack of cancer cells. The overall five-year survival rate for chondrosarcoma stands at 75.2%. Except for these 2 common types, there are also other types of cone cancer, such as ewing tumor, fibrosarcoma, malignant fibrous histiocytoma, giant cell tumors of the bone, chordoma, and more.







Diagnosis & Treatment

The diagnosis of bone cancer involves examining the patient's health history and the results from the physical exam. Diverse imaging techniques aid in this diagnosis, with X-ray imaging being the initial tool due to its ability to distinctly reveal most bone tumors. Magnetic resonance imaging (MRI) uses radio waves to produce cross-sectional images of organs, tissues, bones, and blood vessels, and more. These images are then transformed by a computer into a 3D representation. MRI plays a pivotal role in determining whether the cancer has spread to nearby tissues. CT scans, biopsies, and bone scans are some of the other valuable techniques used Tailoring treatment options is contingent upon a range of factors, including the cancer type, size, location, stage, the patient's age, and overall health. Surgical intervention, chemotherapy, radiation therapy, cryosurgery, and targeted therapy are other methods to treat bone cancer other than X-ray and MRI.

Discussion

In essence, bone cancer originates from aberrant cells within the body's bones or cartilage. Different types of bone cancer have different impacts on different age groups, with different five-year survival rates as well. Imaging tools such as X-rays and MRIs are used to diagnose bone cancer and determine specific details (e.g., locations, and sizes). New research from the Journal of Bone Oncology has revealed a new drug called 'CADD522' that blocks a gene associated with spreading the cancer. Ongoing studies are currently discovering the fundamental factors and mechanisms, promoting treatment approaches, and enhancing the overall well-being of individuals living with bone cancer.

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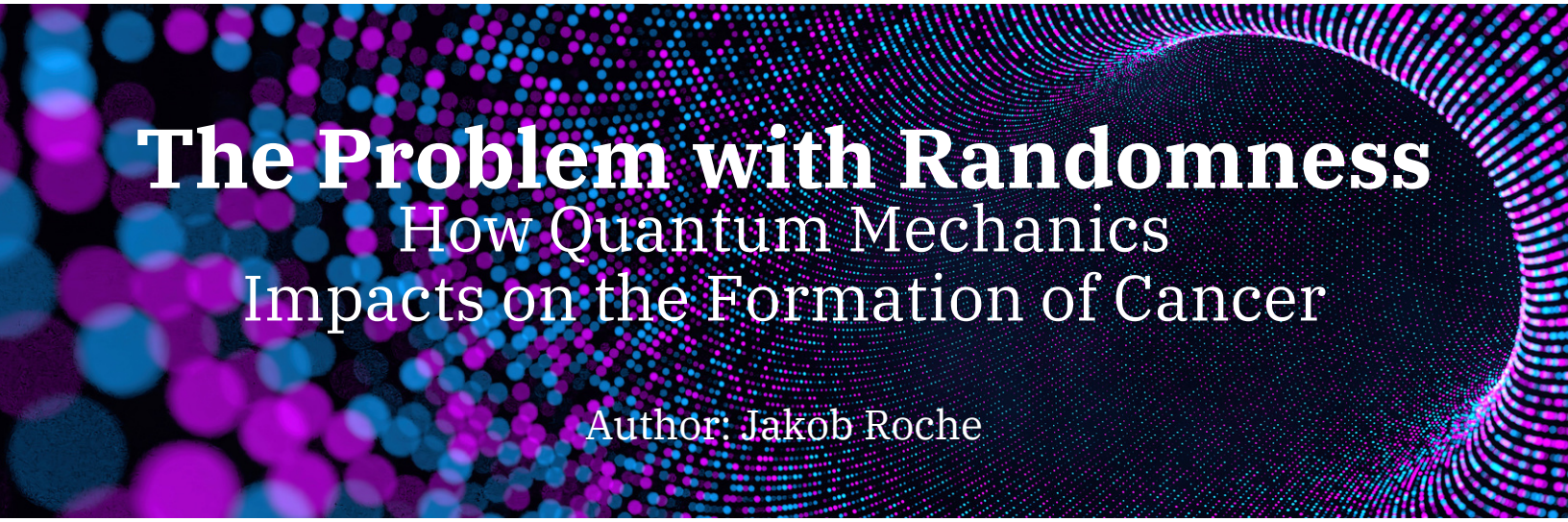


The Problem with Randomness

How Quantum Mechanics Impacts on the Formation of Cancer

Author: Jakob Roche

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 tunnelling 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 tunnelling is potentially a major cause of cancer, justifying future research.

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.

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 behaviour of electrons when they are 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.

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?

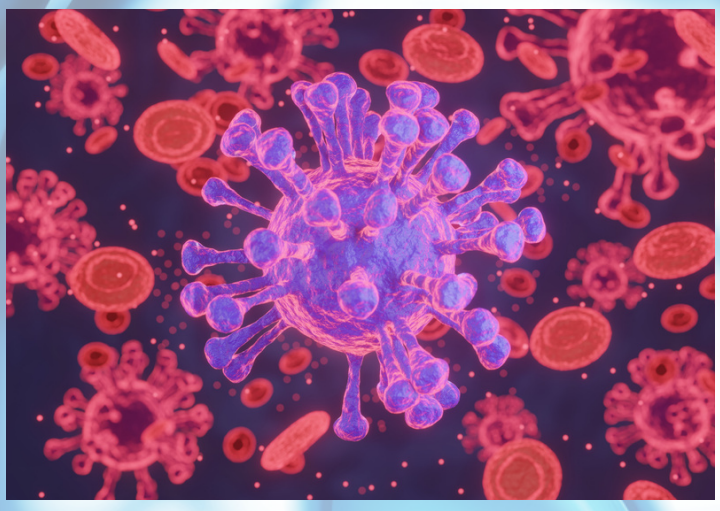
DNA is a polymer. That is, it is made up of long chains of molecules that are generally referred to as monomers. The monomer that is specific to DNA and other nucleic acids are called nucleotides. A single nucleotide consists of a chain of atoms referred to as a phosphate group, because of the presence 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 ‘flavours’ to the ucleotides. 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 e 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 harbours 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 tunnelling. 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 an e-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 tunnelling 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 tunnelling 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 tunnelling events truly random? Can some pattern be discerned? If so, why does tunnelling happen more in some areas of the genome and less in others? Are there things we can do to mitigate the risk of tunnelling 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 tunnelling. She says that, while the probability of a proton tunnelling 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. Conclusion Despite the significant lack of data surrounding spontaneous DNA mutations caused by quantum tunnelling, 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 tunnelling 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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THE GENETICS OF CANCER

Author: Youlan Li

Abstract:

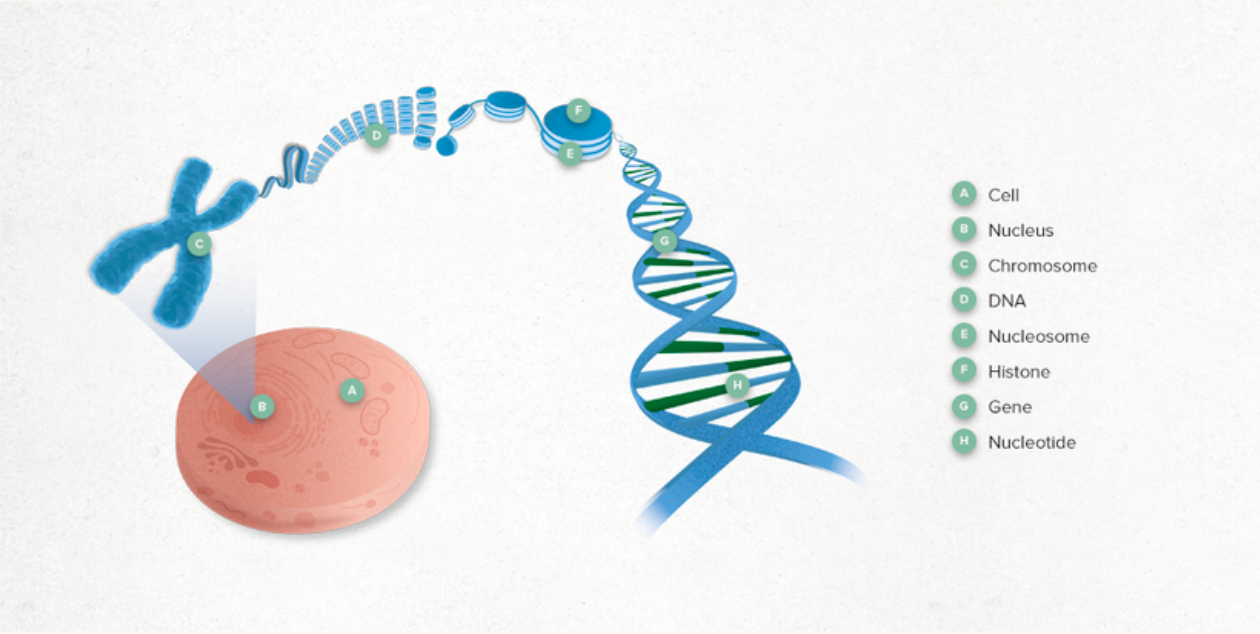
Cancer is a genetic disease, which originates from mutations in the gene that control the growth and division of cells. However, not all mutations lead to cancer, and their impact on the body varies. In most cases, only one mutation won't directly lead to the occurrence of cancer, which is more likely to build up over a certain period, as evidenced by older people having cancer more often than younger populations do. This paper will review the genes linked with cancer, what research has been done, and future research directions discussion.

Introduction:

Cancer results from changes in genes that carry instructions for producing one or multiple proteins. Researchers have identified numerous alterations in DNA sequences that contribute to cancerous cells' onset, growth, and proliferation. The potential reasons behind the changes in DNA include random errors during cell division, carcinogens in the surrounding environment such as chemicals, UV lights, and HPV, and inherited genes from one of the parents. As aforementioned in the abstract section, even though a single mutation in the gene might not significantly impact the appearance of cancer, the accumulation of genetic changes over the years can transform normal cells into cancerous cells. Most cancer cases are believed to occur spontaneously over time due to this cumulative process.

Notably, cancer and genetic alterations cannot be passed down to the offspring, rather, the genetic modification that enhances cancer susceptibility can be inherited if it exists within a parent's egg or sperm cells. For instance, if a parent transmits a mutated BRCA1 or BRCA2 gene to their offspring, their child's likelihood of developing breast cancer and several other types of cancer that link with this gene significantly rises. This is why sometimes cancer seems to cluster and has a higher chance of occurrence in families. It's estimated that as much as 10% of all cancers may stem from genetic changes that are inherited.

There's a rare condition, referred to as family cancer syndromes, in which the gene chance increases the cancer risk that runs in the family. It's crucial to understand that not all cases of cancer clustering within a family can be attributed to a family cancer syndrome. For example, sometimes the prevalence of cancer might be higher in specific families due to shared behaviors or exposures to chemicals or carcinogens, such as smoking, or other factors like obesity. Cancer can also exhibit a familial pattern if family members have a combination of numerous genetic variations, each carrying a risk for cancer.



Specific “cancer genes” linked with cancer:

Some genetic mutations are associated with specific genes. The first to introduce here are tumour suppressor genes, designated as tumour suppressors because they shield against cancer. Essentially, they control the growth of tumour cells by governing the rate of cell division to create new cells, rectifying the errors of DNA, and managing apoptosis (programmed cell death). That being stated, alterations in tumour suppressor genes undermine the regulatory mechanism, leading to uncontrolled cell proliferation, ultimately culminating in the development of a tumour. Among individuals with cancer, the most common mutation in a tumour suppressor gene is observed in p53 or TP53. Due to the vital role that p53 plays in regulating DNA repair and cell division, it has been nicknamed the “guardian of the genome”.

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Current research done:

From Nature, the latest research and reviews on cancer genetics focus on topics such as the identification of a two metastasis-related prognostic signature in the process of predicting the survival of laryngeal squamous cell carcinoma, sub-clonal accumulation of immune escape mechanisms in micro-satellite instability-high colorectal cancers, the clinical impact of the genomic landscape and leukemogenic trajectories in non-intensively treated elderly acute myeloid leukemia patients, and more.

Future research discussion:

One of the ongoing research directions is relating inherited risk factors to cancer genomics. Researchers in NCI's Division of Cancer Epidemiology and Genetics (DCEG) are actively engaged in discovering innovative molecular and genomic patterns within tumours that correlate with inherited genetic variations and environmental influences. This strategy aims to uncover previously unidentified risk factors and provide fresh perspectives into the biological processes underlying cancer development.

Conclusion:

In conclusion, cancer is a genetic disease due to various gene mutations that control the growth and division of cells, including tumour suppressor genes and oncogenes. Current ongoing research by DCEG investigators focuses on innovative molecular and genomic patterns connected with inherited genetic variations and environmental influences.

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THE INTRIGUING LEGACY OF HENRIETTA LACKS

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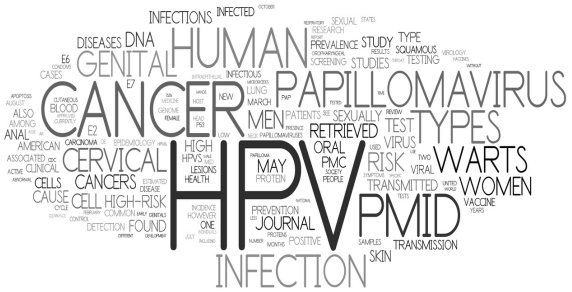
It's 1951, a poor young African-American woman named Henrietta Lacks had just been diagnosed with cervical cancer. Though tragic, this may seem like an everyday occurrence, as what could ever be so significant about a woman more than 70 years ago getting diagnosed with cancer? However, this seemingly mundane diagnosis led to a plethora of research with her cancer cells being revolutionized in an immortal cell line named HeLa, one of the first boundless cell lines that is still being used today to further cancer research. There's no doubt her cells contributed a lot to modern-day cancer research, but one cannot turn a blind eye to this young mother's early death due to the horrible illness along with the complete lack of patient and family consent as her cells were unknowingly taken from her. Her story raises many questions not just about the nature of cancer and why it is so resistant, but also about patient confidentiality, racial and socioeconomic equality, and rights to privacy. [1]

Lacks received radium treatment for her malignant tumour, something that would be more frowned upon today noting how harmful the effects of radium are on the human body. Her cells were taken for a biopsy, a procedure involving analysis of living tissue, and taken to a lab for research. A researcher had been keeping cells from all patients with cervical cancer without asking for their consent, and despite that being incredibly unethical, this led to an epiphany for the research when he observed Lacks' cancer cells – unlike all the other cells that quickly died in his lab, her cells actually started doubling almost every day.

Lacks' cancer cells were definitely one of a kind; so fascinating that even today we don't know the full basis of what made them so impervious to death. It's more or less inferred that the severity of her cancer, numerous duplicates of the human papillomavirus (HPV) genome in those cells, and her having syphilis all contributed to the cancer's extreme resistance. HPV, as a virus, will insert its own DNA into the body of its host, transforming it into a hybrid. In Henrietta Lacks' case, the virus caused her cervical cancer and also contained two major mutations that allowed her cancer cells to thrive that long: the ability to divide uncontrollably and the increased presence of telomerase – enzymes activated during cell division that reconstructs the telomeres, or repeating fragments of DNA at the end of chromosomes, so that they never shorten as an increasing rate of telomere reduction normally causes cells to stop dividing. Lacks also having syphilis would have contributed to her undying cells as her immune system would be weaker than ever before, allowing the cancer to metastasize easily.[2][3]

It's evident her cells contributed a lot to cancer research. The profound discovery that HPV could lead to cervical cancer carved a path to creating the first anti-cancer vaccine that led to a Nobel Prize in 2008 for a doctor involved in that. HeLa cells were also used to determine how the infamous bacterium Salmonella causes infection, create effective treatments against blood disorders, advance the knowledge of HIV infection and its works, deeply analyze how the respiratory illness Tuberculosis works, and so on, with numerous other contributions to oncology. There is even a price tag allotted to HeLa cells, as they can go for over two thousand dollars per millilitre. Profits rising from the cells reached more than 40 billion dollars as of 2021, and the number of studies they were in total to almost 120,000.[4][5]



Alas, the question has to be asked: Was Henrietta Lacks and/or her family ever compensated for her contribution to science? Unfortunately, Lacks died unaware of the legacy her cancer cells left behind, and her family had no inkling of any of this until the early 1970s after some scientists sought them out to research their blood samples. It's safe to say that they were incredibly unhappy with this news, although it was not until 2021 that the Lacks family estate started to demand compensation through a lawsuit for the massive revenue stocked up by some biotechnology companies (i.e. Thermo Fisher Scientific) by using Henrietta Lacks' cells without her consent or knowledge. Although they didn't receive any financial compensation from them, they did get some from John Hopkins University, the one that used her cells in the first place and had managed to reach an agreement with the National Institutes of Health back in 2013 on implementing stricter rules on how and when HeLa cells should be used so that the family can be aware at all times.

Henrietta Lacks remains an important figure in the science community to this day due to her incredible immortal cells, and her immense contribution will continue to be remembered and honoured. She is the reason we have advanced so far in cancer research along with research into other diseases and treatments, and we still have uncharted territory in the medical world that can be guided by her cells. It is amazing to think how we have cultivated so much knowledge from a clump of tiny, stubborn cells swarming in a petri dish, but that's exactly what happened. Through all this, however, we must remember the injustice faced by her and her family as we strive to create an environment in the future with patient confidentiality rules set in stone when we venture out into the unknown.

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