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Abstract

Immunotherapeutic agents have been researched for many decades as an alternative treatment for cancer. Current research demonstrates that immunotherapy is safer than radiotherapy or chemotherapy. This is attributed to immunotherapy's mechanism of utilizing the body's own defense system, as opposed to absorbing harmful chemicals. Two forms of immunotherapy that have been effective in curing cancer without the added danger of chemical toxicity are PNC-27 and PNC-28. These peptides were created by a supercomputer at SUNY Downstate Medical Center in New York in 2000. PNC-27 and PNC-28 work with the MDM2 -P53 tumor suppressor complex. It acts as a competitive inhibitor for binding, increasing the half-life of P53 in the cell and assisting with the elimination of cancer cells (Sarafraz-Yazdi E, Bowne WB, et al 2010). These immunotherapy agents also have the ability to bind to the cell membrane and lyse the cell. The clinical trials for PNC-27 and 28 were successful, and the drug is currently in use outside of the United States. Although this form of immunotherapy does come with some side effects, research illustrates that this form of immunotherapy can be a successful strategy in eliminating the cancer and ensuring that a relapse does not occur.

Abbreviations

MDM2- Mouse Double Minute 2 Homolog P53- Tumor protein, Tp53

HDM2- Human Double Minute Homolog 2

Introduction

Cancer is one of the three leading causes of death across the globe. Although researchers have been discovering cures for many diseases, the cure for cancer still eludes them. There are over 240 different forms of tumors that currently exist, and each type comes with a unique mechanism that can help it evade treatment. Tumors are caused by the ongoing replication of a cell that does not adhere to cell regulations for replication (Vassilev A, DePamphilis, 2017). Due to the fact that tumors arise from our own cells, in order to cure cancer, researchers have to come up with a treatment that exclusively destroys malignant cells, keeping the healthy cells intact. While there are many cures being evaluated in current research that target all cells, PNC-27 and PNC-28 have the ability to target cancer cells only.

There are millions of cells in the human body. In order for cells to thrive, the cells need to be replicated to ensure continuity. New cells are constantly being created as old cells are dying. Cells are converted to tumor cells when the checkpoints that are responsible for monitoring the division do not proceed as expected. (Chao et al., 2017) When the cell metastasizes and travels to another region within the body, the tumor is transformed into a potentially lethal cancer.

When a cell goes through the division process, it replicates all of its chromosomes and sends an identical copy to the new version of the cell that was formed. The process by which this is done is called mitosis. The cell has three phases that ensure that cell replication is proceeding correctly. These three checkpoints that ensure the legitimacy of DNA are known as the M Phase (Mitosis), G1 Phase (Gap 1), and the G2 Phase (Gap 2). At the G1 phase, the accuracy of the DNA is evaluated before proceeding. At G2, the chromosome duplication is assessed. (Veron, 2017) After these checkpoints, the cell goes on to produce identical daughter cells. In a normal, healthy cell, if a cell does not pass these checkpoints successfully, the cell is destroyed. These checkpoints are the main regulators of replication and ensure a low rate of errors in the DNA.

The process of mitosis guarantees perpetuation of life, but it can prove lethal if it proceeds unchecked. Genetic mutation (regardless of whether it was caused by carcinogens or through random errors during DNA replication) takes place in cells throughout the lifetime of an organism (Ahluwalia, 2009). When one of these mutations results in the breakdown of cell cycle regulation, it gives rise to an "unregulated proliferating descendant clone of that particular cell" (Cooper, 2000). This clone of cells can form a tumor. Tumors arise with significant frequency, but most tumors pose slight risk to their host cell due to tumor cell localization. These tumors are known to be benign tumors. It is generally evident when a tumor is benign since the cells bear a close resemblance and role comparable to normal cells. (Mims, et al. 2015).

One of the most significant stages in the diagnosis of cancer patients is the series of events leading to the development of tumor cell invasion and metastasis. Cancer metastasis is the dispersion of cancer cells to tissues and organs outside the original site of the tumor. Once a tumor has left its original source, it travels and forms additional tumors in other organs. This process occurs in three main developments: invasion, intravasation, and extravasation.

Metastasis occurs due to a loss of cell to cell adhesion. Initially, the malignant tumor cells isolate from the main mass at the original source. Subsequently, the cells proceed to invade the surrounding stroma. This development causes the secretion of substances to degrade the basement membrane and extracellular matrix of the organ it has invaded (Shields JA, Shields CL, 2016). The secretion of substances helps express the proteins that mediate the control of the tumor's motility. In addition to the tumor's ability to migrate to other organs, there is a process called angiogenesis. The process of angiogenesis is necessary for the tumor to exist and keep itself satiated. Angiogenesis is the development of new blood vessels that aid with local diffusion. This helps the cell sustain

itself with nutrients, oxygen and transporting metabolic wastes (Nowak-Sliwinska P, 2018).

After invading other organs, the tumor then goes through the introversion stage. Introversion allows for the tumor to make pathways throughout the blood so it can travel through the body and settle in another location. The tumor networks with the endothelial cells through biochemical interactions. This process is regulated by a carbohydrate-carbohydrate reaction.

Adhesion to the endothelial cells creates more solid bonds to penetrate the endothelium and the basement membrane (More SK, Vomhof-Dekrey EE, Basson MD., et al 2019). Through this process, the new tumor reproduces.

As mentioned previously, the common driving force behind cancer is a genetic mutation, but there are many distinctions in how cancer can evolve. There are different mutations that can alter the genes standard structure and function. It is generally not possible to determine what the underlying cause of the mutations. However, research has linked different risk factors to an increased probability of cancer. Carcinogens and viral or genetic factors have all been linked to cancer. For example, exposure to the environment plays a role in how genes will be transcribed. The atmosphere and our food contain many toxins that can cause mutations. UV radiation that is induced by exposure to the sun without ensuring proper protection causes thymine dimers, which researchers believe causes skin cancer. Smoking is also a significant cause of cancer.

Chemicals in tobacco smoke harm the cleansing system that the body employs to remove toxins. Thus, smokers are not as capable of handling toxic chemicals as those with healthy lungs and blood (O'Keeffe LM 2017). Moreover, smoking damages DNA, and generates potent carcinogens which not only affect the trachea, bronchial system and lungs, but also permeate the entire system.

Methods

Data was collected using ProQuest and PubMed databases, and the National Library of Medicine. The images, graphs, and diagrams are in the research articles referenced. Additional materials were found in the SUNY (State University of New York) Downstate research library.

Radiation Therapy Mechanism:

Treatment of cancer originally involved surgical removal techniques alone. Over the previous centuries, radiation therapy has been discovered as an alternate treatment. Radiation therapy treats cancer by focusing beams of intense energy on a specific part of the body to eliminate cancer cells. Some of these forms of energy typically includes X-rays, but can also use high-energy particles or waves, such as gamma rays, electron beams, or protons. The larger the amount of energy involved in the therapy, the more penetration is accomplished by the ionizing ray into the cancer tissue (Orth M, Lauber 2014). The ionizing ray kills the cells that are actively dividing. Although radiation is effective for preventing division of rapidly dividing cells, it can cause only minimal breaks in the DNA. Nevertheless, these breaks stop cancer cells from replicating and producing, activating their elimination. It can be used to shrink early stage cancer, or treat advanced symptoms of cancer.

Radiation therapy attempts to strike a balance by eliminating the harmful dividin cells and minimizing damage to the normal cells around the tumor site. Normal cells can often repair much of the damage caused by radiation. The majority of people with cancer receive radiation therapy as part of their treatment plan, along with a combination with other treatments.

Physicians prescribe radiation therapy to treat all forms of cancer, including benign tumors. Although radiation therapy is effective, research shows that radiation therapy has a large number of side effects. These include tooth decay, third degree skin burns, and difficulty breathing (Murphy, et al. 2019). Radiation therapy is rationed, and there is a limit to the amount of radiation people can safely receive. Depending on how much radiation an organ has received during the first round of treatments, the body may not have the ability to receive radiation a second time.

The major lethal side effect of radiation therapy is the relapse of the cancer. Because radiation therapy damages healthy cells, these damaged cells can metastasize to other locations and cause a recurrence of the cancer. Most types of leukemia cancer, including acute myelogenous leukemia (AML), chronic myelogenous leukemia (CML), and acute lymphoblastic leukemia (ALL) are all forms of cancer that originate from radiation exposure. Myelodysplastic syndrome (MDS), a bone marrow cancer that may evolve into leukemia, has also been associated with previous radiation exposure" (Cidon,EU 2016). Not all cancers may return immediately after radiation therapy. Research shows that solid tumors take much longer to develop, and don't come back quickly. Most of these cancers are not spotted for years after the radiation therapy has caused the damage, and some are diagnosed more than a decade later. We may be inevitably perpetuating the disease itself by using radiation therapy as an option.

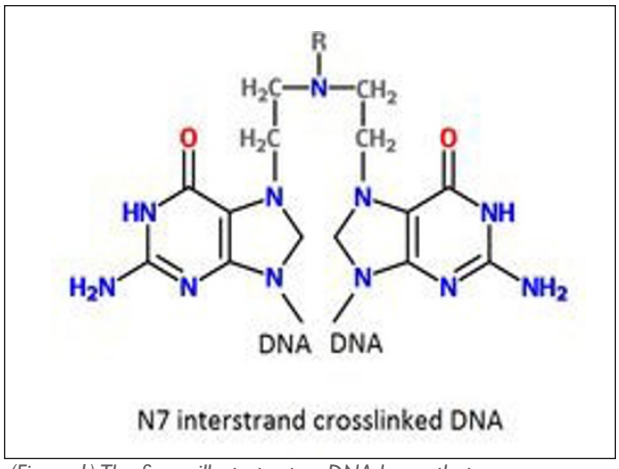
C Hemotherapy Mechanism:

A common form of treatment that is currently in use is chemotherapy. Chemotherapy is a systemic treatment,

and travels through the bloodstream. The treatment is often given to the patient for a specific time, ranging from 6 months to a year. In cases where there is no possible cure, chemotherapy may be given to extend the patient's life, or relieve painful symptoms. The drugs are usually given by IV or orally. Chemotherapy has a lower probability of damaging cells that are at rest, such as most normal cells. Physicians can prescribe the treatment after surgery was performed to remove the original tumor, in order to eliminate the individual cancer cells that may have metastasized before they are able to form a tumor.

The mechanism of chemotherapy is to eliminate cancer cells by targeting different cells at different phases of the cell cycle in order to halt the replication. The chemotherapy damages the genes inside the nucleus of cells, and attack cells that are at the point of splitting. Some damage the cells while they are replicating their gene before they split (Johnstone RW, 2016). Some chemotherapeutic agents attack the DNA of the cell by adding a methyl group, in order to prevent them from producing additional clones of the cancer cell. These are known as alkylating agents, and they attach to DNA, RNA, and proteins through covalent bonding.

Alkylating agents will attack at any point in the cell cycle. There are many types of alkylating groups, such as nitrogen mustards, nitrosoureas, and aziridines. The molecules bind to the DNA and change the DNAs conformation. DNA is composed of a double strand, and the

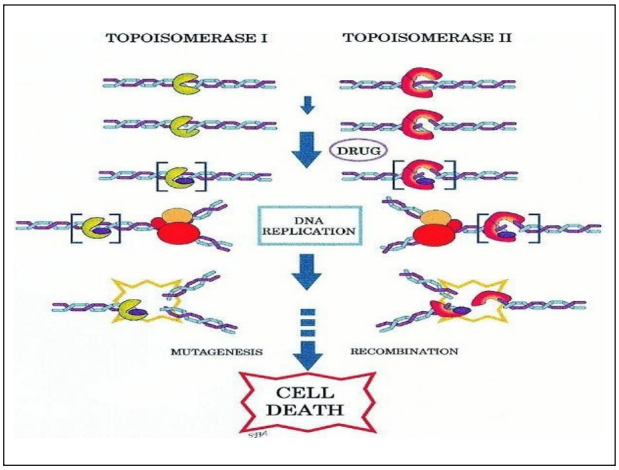


(Figure 1) The figure illustrates two DNA bases that are cross-linked by a nitrogen mustard (Siddik ZH 2005

molecules may either bind two times to one strand of DNA (known as a "intrastrand crosslink") or may bind once to both strands (called a "interstrand crosslink")" (Figure 1). If the cell attempts to replicate or fix cross-linked DNA during cell division, the DNA strands break (Cancer Chemotherapy and Biotherapy, 2005). This leads to a form of programmed cell death called apoptosis.

Another chemotherapy drug that interferes with transcription and translation is a topoisomerase inhibitor. When the DNA double-strand helix is unwound during DNA replication or transcription, the adjoining DNA (that has not been opened yet) is wound up extremely tightly. The topoisomerase inhibitor produces single- or double-strand breaks into DNA, reducing the tension in the DNA strand ahead of the replication point (Figure 2) This allows the normal unwinding of DNA to happen during replication or transcription. Inhibition of topoisomerase does not allow either of these processes to continue (Nitiss JL, 2007). Other chemotherapy drugs interfere with cell metabolism. This blocks off nutrients and oxygen supply, which causes growth arrest.

Research has shown that chemotherapy has many side effects. Some of these side effects include fatigue, hair loss, infection, fertility problems, weight loss, damage to



(Figure 2) Topoisomerase I and II Inhibitors (Huda W. 2016)

lung tissue, heart problems, kidney problems, and nerve damage (Uzun, 2019).) Studies show that one of the primary drawbacks is that chemotherapy does not achieve remission. One reason is that the original treatment is not successful in eliminating all the cancer cells. The remaining cancer cells that are left in the organ grew into a new tumor. Another reason that chemotherapy is not successful in achieving remission is that some cancer cells have metastasized to other parts of the body and started forming a new tumor in the new location. Cancer cells may also develop a mechanism that makes it become resistant to treatment when it relapses, thereby blocking a possible cure (Stadlr, WM, 2014).

Immunotherapy

Researchers have discovered that one of the best methods

for treating cancer is immunotherapy. Immunotherapy is a biological therapy that utilizes substances made from living organisms to treat cancer. Some immunotherapy treatments use genetic engineering to improve immune cells' cancer-fighting capabilities and are commonly referred to as gene therapies. Many immunotherapy treatments for preventing, managing, or treating different cancers can also be used in combination with surgery, chemotherapy, radiation, or targeted therapies to aid their overall effectiveness (Yang, Wang, Wang, 2019).

Immunotherapy boosts the immune system's ability to identify, target, and remove cancer cells. While many cells replicate naturally, this behavior is tightly regulated by an assortment of factors, including the genes within cells. When growth is not required at certain points, cells are instructed to halt growth. Cancer cells acquire defects that cause them to ignore these inhibitive signals, and their replication proceeds in an unregulated and rapid manner. Because cancer cells grow and behave through abnormal methods, this can alert the immune system of a potential threat, which can recognize and eliminate cancer cells through a process called immunosurveillance.

Immunosurveillance is an extremely effective method for eliminating pathogens from the body, but the process isn't always successful when it pertains to cancer. Even though the immune system can prevent or slow cancer growth, cancer cells have ways to evade destruction by the immune system. For instance, cancer cells may have genetic changes that make them appear less discernible to the immune system, have proteins on their surface that turn off immune cells, or change the normal cells around the tumor so they interfere with how the immune system responds to the cancer cells (Pardoll D. 2015).

Cancer cells develop ways to evade and escape the immune system, which allows them to continue to thrive and metastasize to other organs (Prendergast GC, Jaffee EM, 2007).

Therefore, immunotherapies are designed to enhance the cancer-fighting capabilities of immune cells and utilize the body's own protective measures to fight the tumors.

NC-27 and PNC-28 Peptides

PNC-27 and PNC-28 are synthetic peptides designed to specifically target and destroy cancer cells. Therapeutic peptides have the ability to treat an extensive range of diseases and cancers and contain a large number of advantages over proteins or antibodies. They are easily synthesized in a lab and are not toxic to the human body. They are manufactured to have a very specific affinity for the targeted cell. PNC-27 and PNC-28 were created using a super computer by Dr. Matthew Pincus and Dr. Joseph Michl of SUNY Downstate Medical Center. The peptides were created with a specific fold that only interacts with a particular structure present on cancer cells. It can be administered by nebulizer, vaginal or rectal suppository or intravenously at the tumor site. Many patients travel to foreign countries to receive this treatment. PNC-27 and PNC-28 have been used with exceptional results, and over 500 patients have had a high success rate with the drug since 2007. PNC-27 and PNC-28 peptides have been shown to be highly effective in specifically targeting a large variety of different cancers. These peptides have had high success rates with breast cancer, leukemia, melanoma, and pancreatic cancer lines. It has proven to be the most effective when taken simultaneously with immune system boosters, proper hydration, and a diet that avoids excess sugars and red meat.

P 53 Mechanism for PNC-27 AND PNC 28

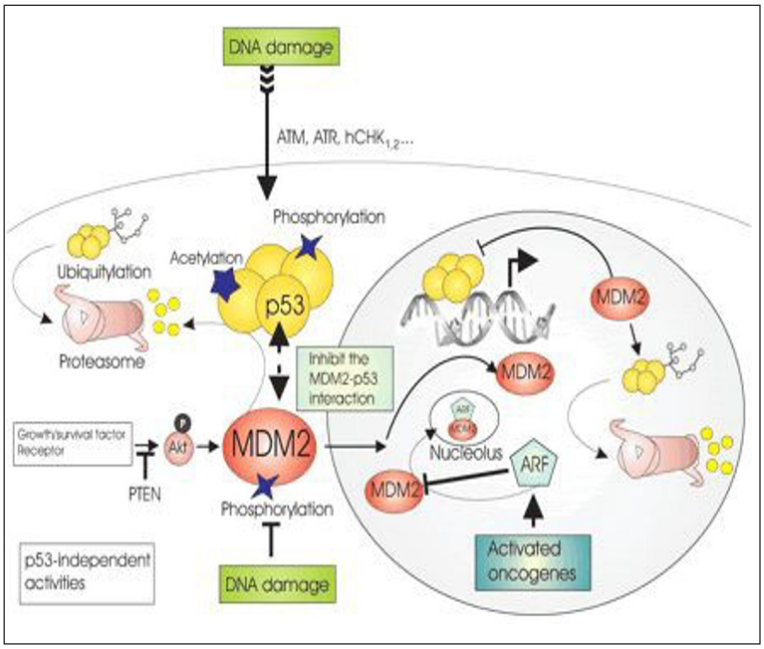
TP53 or "tumor protein 53" is a gene that is located on the seventeenth chromosome of humans. It codes for a protein that regulates the cell cycle and hence functions as a tumor suppressor. P53 has been described as "the guardian of the genome", referring to its role in keeping the cell stable by preventing genome mutation (Uehara 2018). TP53 has 4 domains that are responsible for activating transcription factors, tetramerization of proteins, recognizing DNA sequences, and checking damaged DNA (Sabapathy et al 2019).

The p53 tumor suppressor has a very significant role in growth arrest, DNA repair, and apoptosis, and it causes a response to different attacks on the cell. Rapid induction of high p53 protein levels by various stress types prevents inappropriate propagation of cells carrying potentially mutagenic, damaged DNA (Zhang et al., 2014). In normal cells, p53 is an extremely unstable protein that has a half-life ranging from 5-30 minutes. The p53 protein is present at very low levels in the cell, due to continuous degradation. This continuous degradation is primarily regulated by MDM2. Research over the past decade has assigned MDM2 as the central controller of p53 by regulating the p53 tumor suppressor function (Carotenuto, 2019).

A mutation of the p53-MDM2 complex during the induction of p53 can lead to the accumulation of active p53 in the cell (Figure 3). Consequently, p53 half-life extends from minutes to hours. This can aid p53 in stopping cancer. Both PNC-27 and PNC-28 were tested as agents that would competitively block p53 from interacting with HDM-2. By acting as a competitive inhibitor, the peptides would prolong the half-life of p53 in cancer cells. This eventually leads to more efficiency in inducing apoptosis of cancer cells.

Mechanism through Membrane and Affinity to HDM2

The PNC-27 and PNC-28 cancer peptides are substances that causes the death of cancer cells only. PNC-27 and PNC-28 accomplish this due to their affinity for binding to a protein called HDM-2. Cancer cells have high levels of HDM-2 present in their cell membranes. (Ehsan Sarafraz-Yazdi, Wilbur B. Bowne, 2010). Dr. Ehsan Sarafraz-Yazdi (Sarafraz-Yazdi E et al 2015) explains in detail how the PNC-27 and PNC- 28 cancer peptide manifests themselves, and what their mechanisms could mean for progress in the field of cancer research. The study points out that the peptide's mechanism hinges on the fact that there are formations of oligomeric pores in the plasma membrane of tumor cells. (American Association for Cancer, 2010) The oligomeric pores are present exclusively in cancer cells. Additionally, the observations illustrated that PNC-27 and PNC-28 were capable of distinguishing between cancerous and non-cancerous cells.



(Figure 3) A chart of the disruption of mdm2 and the p53 complex (Hashimoto N, Nagano H, 2010)

In a 2009 study, researchers discovered that the " three-dimensional structure of PNC-27's and PNC-28's p53 residues of its amino acids may be superimposable onto the structure for the same residues bound to HDM-2." This discovery alerted researchers to the fact that that PNC-27 and PNC-28's could target HDM-2 in cancer cells' membranes (Pincus, et al (2011)).

Upon further research, it was discovered that by "inserting untransformed cells that are not prone to PNC-27 and PNC-28 with HDM-2 containing a localized membrane signal, these cells then became inclined towards PNC-27 and PNC-28". This research established that the PNC-27 peptide was able to exclusively mark HDM-2 in the membranes of cancer cells and destroy them through membranolysis, while at the same time leaving the healthy cells alive (Sookraj, et al (2010). Additional research established that PNC-27 and PNC-28 uses the entire peptide when destroying cancer cells, as opposed to fragments. (Cancer chemotherapy and pharmacology, 2010).

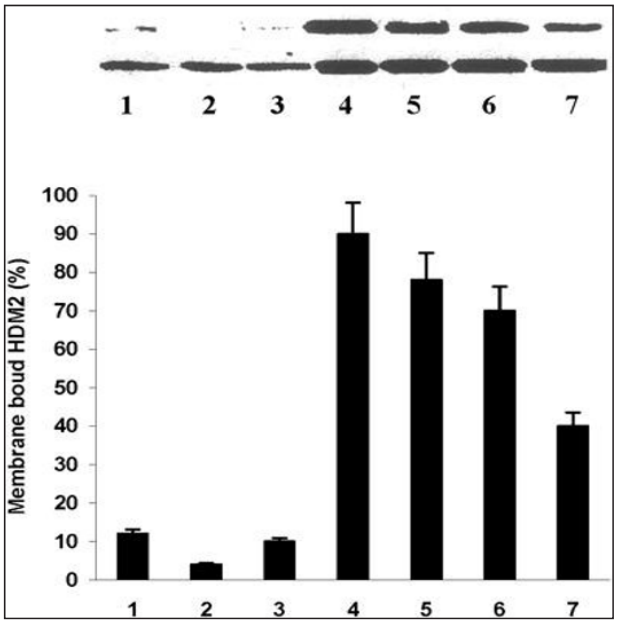
"The PNC-27 peptide has an HDM-2 binding domain that has the same residues 12-26 of p53 and a transmembrane-penetrating domain (Sarafraz-Yazdi, et al, 2010). PNC-28 is a p53 peptide from its HDM2-2-binding domain (residues 17-26), which contains the penetratin sequence enabling cell penetration on its carboxyl terminal end. This domain is connected to a transmembrane-penetrating sequence, also referred to as the membrane residency peptide (MRP). When p53 binds to the HDM-2 protein through the 12–26 amino acid sequence, HDM-2 causes ubiquitination of p53 that targets it for proteolysis in the proteasome. Both PNC-27 and PNC-28 peptides , which contains a sequence that is identical to that of PNC-27 but lacks the first six amino acid residues of PNC-27 (i.e., p53 residues 17-26), are highly toxic to a wide variety of cancer cells with IC50 values that range from around 75 ug/ml (18.6 uM) to 200 ug/ml (50 uM)." "While PNC-27 and PNC-28 are toxic to cancerous cell, neither peptide affects normal cells that are in culture, even at the highest doses (around 500 ug/ml) tested". This discovery illustrates that both peptides would not be toxic to the human body, in contrast to the effects of many chemotherapeutic agents (Davitt K, Babcock BD, 2014).

In a newer study that was analyzed in 2014 and published in Annals of Clinical & Laboratory Science, researchers concluded that "the anti-cancer peptide, PNC-27 and PNC-28, induces tumor cell necrosis of a poorly differentiated non-solid tissue mammalian leukemia cell line that depends on expression of HDM-2 in the plasma membrane of these cells." (Davitt, et al (2014). Studies based on information discovered in earlier experiments illustrated that the PNC-27 and PNC-28 peptides were able to destroy solid tissue tumor cells through the mechanism of binding to HDM-2 proteins in their cell membranes, an effect which was an effect that was different from the p53 activity in those cells.

The scientists wanted to determine whether PNC-27

peptide would also be effective against non-solid tissue tumor cells (Davitt et al., 2014). Their study determined whether the non-solid tissue tumor cells expressed HDM-2 in their membranes in the first place (solid tissue cells were found to do so), and find if PNC-27 could cause these cell's death by the HDM-2 binding. The researchers observed that these non-solid tumor cells do express HDM-2 in cell membranes.

They also noted that the PNC-27 peptide could cause cell destruction through membranolysis through the same HDM-2 binding mechanism as it did in the solid tissue tumor cells, independent of the p53 pathway. The research established that the peptides are successful for both solid and non-solid tumors, making it a valuable drug that could treat a variety of cancers.



(Figure 4) "Blots of whole cell lysates (Lower) and membrane fractions (Upper) for H (M) DM-2 in different cell lines as follows: Lane 1, MCF-10-2A; lane 2, BMRPA1; lane 3, AG13145 fibroblasts; lane 4, TUC-3; lane 5, MIA-PaCa-2; lane 6, MCF-7; lane 7, A-2058. The first three cell lines are untransformed; the remainders are different cancer cell lines. Each bar graph shows the percentage of HDM-2 in whole cell lysate that is present in the membrane of each cell line listed above. The numbers on the X axis of the bar graphs correspond to the lane numbers shown in the blots." (Safaraz-Yazdi E, Bownwe WB, 2010)

Studies and Trials on PNC-27 and PNC-28

Electron microscopy was utilized to observe the levels of HDM2 in the cell membrane (Wang, et al 2010). Parts of the membrane were isolated, and whole cell lysates from several different cancer and untransformed cell lines are shown in the figure below. The cells were blotted for HDM-2 (Figure 4). "In the lower section in the table, it can be seen that all whole cell lysates had positive results for the blotting procedure for HDM-2. On the upper section for the blots shown in the figure, the membrane fraction of each cancer cell line (lanes 4–7) is seen to contain significant levels of HDM-2. In contrast, the three untransformed cell lines (lanes 1–3) were discovered to contain low levels of HDM-2 in the region22 of the membrane. The percentage of whole cell lysate of HDM-2 present in the membrane fractions of the cell lines is shown in the lower section of the bar graph." (Safaraz-Yazdi E, Bownwe WB, 2010) The graph illustrated that the fractions of HDM2 present in the membranes of the cancer cell lines have an increased level of four-fold to nine-fold.

When the PNC-27 peptide was tested, researchers noted pain levels drop in the average time of a week. Three weeks following the administration of the peptide, the subjects sometimes developed flu-like symptoms. This is an indicator that the immune system had the ability to recognize and react to the death of cancer cells (Sookraj, Ka, Bowne WB 2010). Additionally, when the cancer killing peptide PNC-27 was administered after tumor growth had occurred at a different site from the original tumor, the tumor decreased in size, followed by a gradual increase in tumor growth that was "significantly slower than growth in the presence of control peptide." If the cancer killing peptide is immediately senttouro to the tumor, researchers concluded that the peptide would be successful in treating cancer. (Prendergast, Jaffee, 2007). At six weeks, the researchers noted an increase in lactate dehydrogenase and bilirubin levels.

At ten weeks, a large amount of tumor breakdown is noticeable. At the same time, the tumors become softer and pliable. In addition, some increase in the size of the tumor itself occurred simultaneously. However, this can often be attributed to inflammation because of immune system response. At about three months, researchers saw that the subjects of the study exhibited better energy levels and less cancer-related symptoms.

A study published in the International Journal of Cancer (2006) writes that researchers discovered that PNC-28 was able to reduce the rate of cancer cell growth in an organism at a quicker pace. Research tested PNC-28 to analyze its function to halt the replication of cancer cells. When PNC-28 was given over a two-week timeframe, the PNC-28 caused complete destruction of these tumors. (Science Daily, 2006) When administered simultaneously with tumor implantation, PNC-28 blocked the tumor growth entirely in the duration of the two-week period of administration, as well as two weeks after treatment, followed by weak tumor growth that leveled off at

low tumor size (Bowne WB, Sookraj KA, 2015). The trials on both drugs supported the theory that immunotherapy is able to eliminate cancer cells without causing damage to the human system.

All methods and studies in this paper have been completed, and the drug is currently being sold out of the United States. It is in trial stages to be sold in the United States as well. PNC-27 and PNC-28 are novel drugs that bring much promise to the field of cancer research, and promises an alternative to the other harmful treatments being used today. However, larger studies should be conducted to reduce the side effects that are experienced by patients taking the drug. There were no attempts in proving that PNC-27 is harmful for ingestion, unless contaminated by an outside source. There are multiple factors involved in creating immunotherapeutic peptides as treatment to cancer. Other approaches to improving the drug would be to further elucidate the mechanism by studying various models of the disease. By examining different models, this will hopefully give a deeper perspective and aid the efforts for a cure.

Conclusion

Cancer is a disease that has caused an upward trend in mortality rates. Although some progress has been made in cancer research for a cure, there are many drawbacks to the current treatments available. The current treatments are chemotherapy and radiation. While both of these treatments can successfully eradicate tumors and cancer cells, they also tend to damage healthy cells. There is also potential for a relapse because not all cancer cells are targeted during treatment, as some cancer cells may drift to other organs and rebuild there. As a result, many cancer patients succumb to the toxic effects of cancer treatments rather than the cancer itself. With wealth of evidence in support of immunotherapy, this alternative treatment shows great promise because it does not adversely impact healthy cells. This is because the underlying concept of immunotherapy is to invest the body's healthy cells with resources to be able to naturally combat the cancer cells. This is done by PNC-27 and PNC-28 peptides. Through research and study on animal subjects, the PNC-27 and PNC-28 peptides have been shown to be highly effective in selectively targeting a wide variety of specific forms of cancer, including pancreatic cancer, breast cancer, leukemia, melanoma, and additional cancer lines. Since PNC-27 is non-toxic, patients can get rid of the cancer in a healthy manner, while aiming to follow a lifestyle based on holistic health and wellness. When properly administered, PNC-27 and PNC-28 puts cancer on the defensive, resulting in outcomes that include significant pain reduction and, in many cases, lengthening of life. This research supports the evidence that PNC-27 and PNC-28 are one of the most effective ways to treat cancer.

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