Oncogene Addiction: How Cancer Cells Become So Aggressive

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Abstract

Bone cancer is an aggressive, yet rare form of cancer that makes up less than 1% of all people who are diagnosed with cancer. Patients with bone cancer face significant challenges when it comes to cancer diagnosis and treatment due to the tumor's rich microenvironment and heterogeneity. This review provides a comprehensive overview of bone cancer, starting with foundational mechanisms like DNA replication and the cell cycle, detailing how specific genes like tumor suppressors and oncogenes create errors and dysregulation in these processes, aiding in the formation of malignant tumors. With 500,000 people battling this complex cancer, it is important to understand the mechanisms of how it can form and metastasis in order to figure out new ways of diagnosing and treating patients more effectively.

An Overview of Primary Bone Cancers: Focus on Osteosarcoma and Related Malignancies

In 2025 alone, 2.04 million new cancer cases were reported, and an estimated 618,120 currently diagnosed patients are expected to die from this disease (1). Cancer is the uncontrolled or abnormal growth of cells that can occur anywhere in the body. Cancerous tumors can be found in various states, such as solid tumors, found within an organ, or blood cancers, found within the lymphatic system or bone marrow (2). Cancer is most prevalent in areas with a high tissue concentration, such as the breast and skin, but is least common in compacted and highly structured cells, like those found within the bones. Bone cancer makes up only 1% of the cancer diagnoses worldwide, making cases extremely rare, with a limited number of cases to study.

Osteosarcoma is the most prevalent type of bone cancer, with around 3,770 cases diagnosed each year (3). This cancer primarily affects those between the ages of 10 - 30, and also tends to affect more males than females. This age group is most affected because osteosarcoma is found during the early formation of bone cells, a process that declines with age

(4). While the mechanism behind affecting males more than females is not entirely understood, this preference may be linked to the rapid growth rates of males during this period, increasing the likelihood of a mutation within the bone. Osteosarcoma occurs primarily in the body's larger bones, like the femur, tibia, and humerus.

While osteosarcoma is the most prevalent, two other types of bone cancer also have a high diagnosis rate, called Chondrosarcoma and Ewing tumors. Chondrosarcoma is the second most common bone cancer and originates in the cartilage, which is the connective tissue at the ends of the bones that acts like a cushion (Fig. 1). The risk of chondrosarcoma increases with age and develops most commonly in the pelvic, leg, and arm bones. The other prevalent bone cancer is the Ewing tumors, which primarily affects people between the ages of 10 and 30 and is mostly found in the pelvis, ribs, shoulder blades, and spine (5). These tumors invade the bones' interior and weaken the bone overall. These different types of bone cancers are scarce, complex, and unknown, leaving a large room for research and review on this topic.

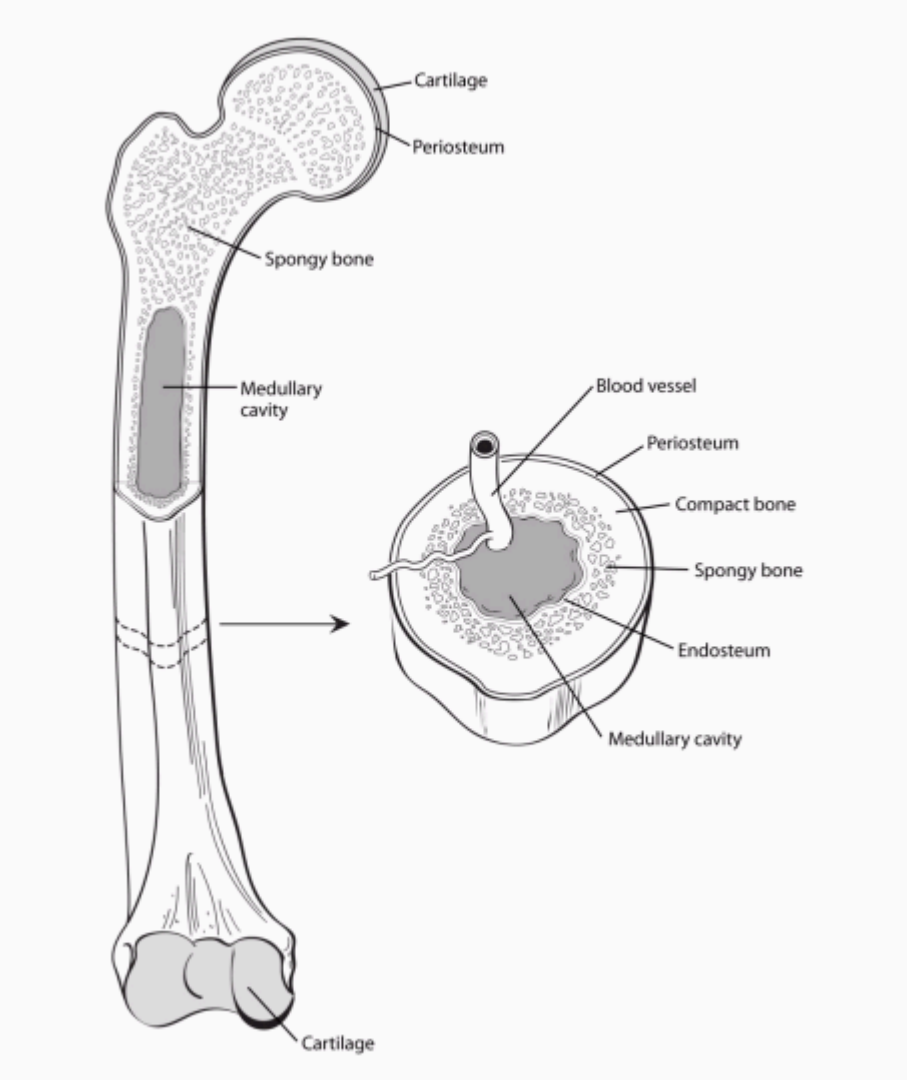

Figure 1. Anatomical Diagram of a Human Long Bone (10)

Diagnostic Approaches and Therapeutic Strategies in Bone Cancer

Though bone cancer is sporadic, the tools behind diagnosing and treatmenting bone cancer have continued to adapt, paving the way for better survival for each patient. There are many different ways to diagnose bone cancer, the majority of them using an imaging technique, in parallel with a biopsy to confirm the diagnosis. Imaging techniques like X-rays, CT scans,

Magnetic Resonance Imaging (MRI), and bone scans can all be used to identify any abnormalities in the bone first. X-rays and CT scans use ionizing radiation that absorbs differently into different tissues in the body to take cross-sectional images and get a detailed view of the tumor. Imaging techniques like MRI use magnetic radio waves to create detailed pictures of the bone and soft tissue, thus revealing size, shape, and invasion of tumor tissue (Fig. 2).

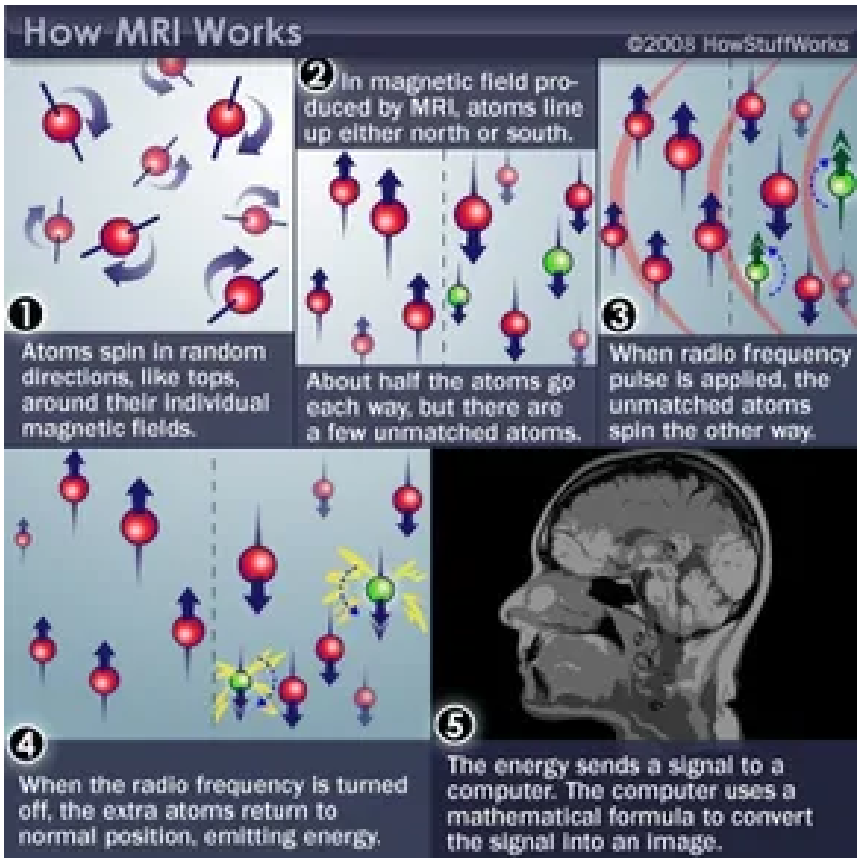

Figure 2. MRI technology works using a magnetic field, radio pulse, and the ability to capture the energy to generate detailed internal images (29).

Lastly, bone scans use a radioactive tracer designed to accumulate in areas of increased bone activity. They can detect the osteoblastic response, the body's building of new bone to repair damage (20). However, the only definitive way to diagnose the presence of cancer is through a biopsy, which removes a sample of the suspected tumor and is thoroughly examined under a microscope. There are two ways to do this for bone cancer, one via a needle and one

through a surgical procedure. Using local anesthetic, the procedure is straightforward where a needle is inserted through the skin and into the bone to collect a sample. A surgical biopsy uses general anesthesia and creates an incision at the tumor site to take a larger sample (21). These two techniques ensure the diagnosis and help decide the best treatment options.

Treatment for bone cancer varies greatly depending on factors like location, size, and stage of the tumor. These factors play a massive role in the order of operations when trying to eradicate the cancer. The most common form of treatment is surgery to remove the tumor from the bone entirely, which can then be paired with chemotherapy and radiation afterwards to ensure that the cancer can not reform or metastasize to other parts of the body. Chemotherapy is another form of treatment that is systemic administration of toxic drugs to attack the tumor and disrupt or inhibit cell division or prevent DNA damage repair. This process uses pharmaceutical drugs taken either by pill or intravenously, targeting fast-growing and dividing cells. The most common chemotherapy drugs for bone cancer are cisplatin, doxorubicin, ifosfamide, and vincristine. These drugs all either damage the cancer cell's DNA, inhibit DNA and RNA synthesis or disrupt cancer cell division (Table 1, 30).

Drug	Type	What it does	Usual Drug Combinations
Cisplatin	Platinum-based: uses platinum to act as an alkylating agent	Forms adducts with DNA, disrupting structure and function, leading to apoptosis.	**MAP** - methotrexate, cisplatin, & doxorubicin **CISPDOXO** - cisplatin & doxorubicin
Doxorubicin	Anthracycline: derived from bacteria to inhibit many cancer mechanisms	Inserts themselves between base pairs disrupting the structure and topoisomerase 2 inhibition; a crucial enzyme for DNA replication & repair.	**MAP** - methotrexate, cisplatin, & doxorubicin **CISPDOXO** - cisplatin & doxorubicin **VDC/IE** - vincristine, doxorubicin, cyclophosphamide; altered with ifosfamide & etoposide
Ifosfamide	Alkylating agent: adds an alkyl group to a	Alkylation prevents DNA helix from linking properly,	**VDC/IE** - vincristine, doxorubicin, cyclophosphamide; altered

	guanine base	breaking the DNA.	with ifosfamide & etoposide
Vincristine	Vinca alkaloids: derived from the Madagascar Periwinkle plant	Binds to a protein called tubulin, preventing microtubule's assembly and function; ultimately halting cell division.	**VDC/IE** - vincristine, doxorubicin, cyclophosphamide; altered with ifosfamide & etoposide

Table 1. Shows the different types of chemotherapy drugs for bone cancer, their type and what mechanisms they inhibit.

While this treatment can shrink the cancerous tumor, there are severe side effects that accompany this, like extreme fatigue, nausea, hair loss, and other permanent damage to the body (22). Lastly, radiation treatment is another treatment that may be used in certain types of bone cancer. This can only be used in certain cases of bone cancer due to the radioresistance of the cancer cells, meaning that a higher dose of radiation is needed which is extremely harmful to healthy cells. This treatment uses X-ray radiation in the form of X-ray beams to target tumors and kill them with excessive amounts of energy, which can destroy genetic materials that control how the cell grows and divides. Modern-day radiation is close-to precise, but only works on certain types of cancers and has severe side effects like chemotherapy as well (23). However, to truly understand cancer, we have to look towards its roots; how mutations in DNA disrupt the genes that regulate the cell cycle, leading to uncontrolled growth and tumor formation. By examining these foundational processes, we gain insight into how malignancies begin and why targeting these early disruptions is essential for effective treatment.

The Molecular Foundations of Cancer: Cell Cycle Regulation and DNA Replication

While the origins of osteosarcoma are unknown and rare, the best way to understand its inner workings is through the basics, by reviewing the processes of DNA replication and the formation of cells through the cell cycle. DNA replication is a process to create an exact copy of a cells DNA. It is a semiconservative process because each new molecule created consists of

one original and one newly synthesized strand (6, Fig. 3). The first step is performed by an enzyme called helicase, which “unzips” the DNA double helix strand by breaking the attractive forces within the hydrogen bonds that keep DNA together. This enzyme requires ATP, adenosine triphosphate, to initiate DNA replication. Bacterial helicase unzips the DNA in the 5’ to 3’ direction while eukaryotic helicase unzips from the 3’ to 5’ direction (7). Helicase starts unzipping the DNA at a place called the origin. It continues to unwind the DNAuntil it reaches the enzyme topoisomerase. This ensures the DNA strands are not supercoiled or tangled. The next step involves an enzyme called DNA polymerase, which is responsible for rebuilding new DNA strands from the parent strand.

At this stage, an enzyme called primase aids the DNA polymerase by indicating where to start rebuilding the strand. This process runs in the 5’ to 3’ direction because that is the only way DNA polymerase can build. However, this creates an issue in rebuilding the strands of DNA, resulting in what are known as leading and lagging strands. The leading strand is the quickest to rebuild because the original strand goes in the 3’ to 5’ direction, meaning that the DNA polymerase does not have to keep stopping while rebuilding. However, in the lagging strand, the helicase unzips the DNA quicker than the polymerase can build because that strand goes in the 5’ to 3’ direction. After this strand is completed, another enzyme called Ligase glues the lagging strand fragments, also called Okazaki fragments, back together to create one full strand of DNA.

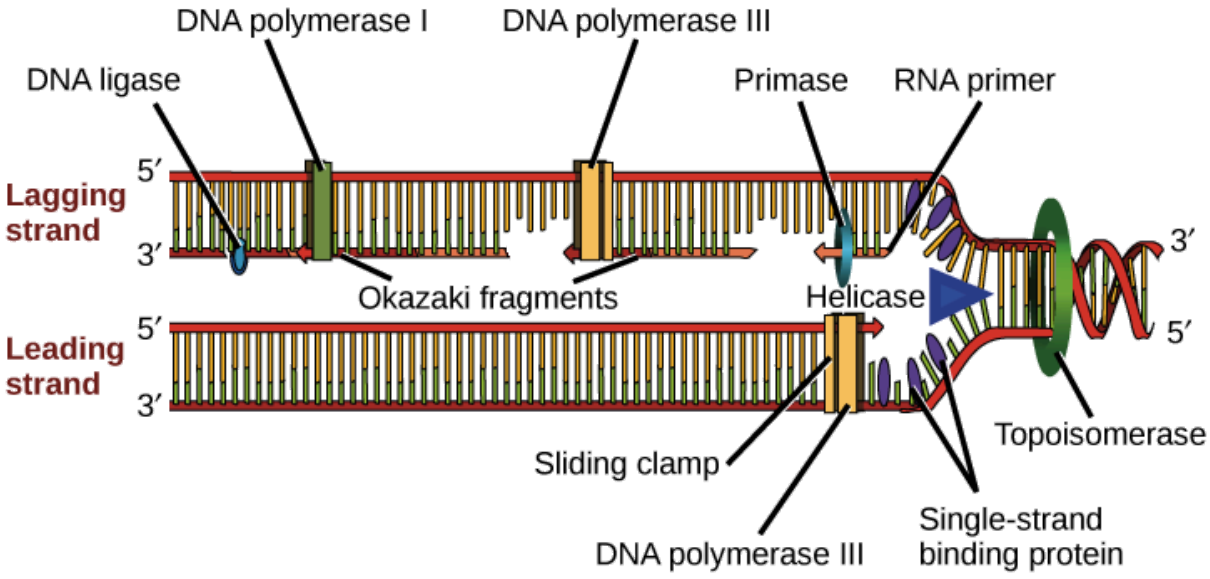

Figure 3. DNA Replication, Highlighting Key Enzymes in the Process of Rebuilding Both Strands (11)

DNA replication occurs during a phase in the cell cycle, which is a crucial cycle for normal cellular division, and can play a role in cancer formation. The cell cycle comprises four phases: G1, S, G2, and M (Fig. 4). G1, S, and G2 phases are sorted into another phase called interphase, where the cell spends most of its time. Additionally, the cell cycle comprises many checkpoints within each phase to ensure that all processes run smoothly and that no mutations have occurred. The cell begins in the G1 phase, which is when the cell starts to grow in size and creates all the necessary organelles and proteins for DNA replication. This is where the cell undergoes a checkpoint in G1 to ensure everything is properly functioning. If the checkpoint discovers anything wrong with the cell, it can hold it in G1 for longer or send it to G0 for apoptosis/programmed cell death (8). After passing the checkpoint, the cell moves into S phase, where the cell's DNA is replicated, creating two identical copies of each chromosome (Fig. 2). Next, the cell passes into the G2 phase, where the cell continues to grow and produces necessary proteins for cell division. The next checkpoint occurs at the end of G2/ start of M. This is to ensure that the replication process went well and that there are no errors before cell division starts.

If everything is correct, the cell moves to M phase, which includes prophase, metaphase, anaphase, telophase, and cytokinesis. In prophase, the chromatin breaks down, the nuclear envelope breaks down, and the mitotic spindles start to form. Next, in metaphase, the chromosomes get in line at the metaphase plate at the center of the cell, and the mitotic spindles attach to each chromosome. This is also where the last checkpoint of the cell cycle occurs, and it checks to ensure that the spindles are correctly connected to ensure that each daughter cell receives the same number of chromosomes. Then there is Anaphase, where the sister chromatids are pulled apart to opposite cell poles. Lastly is telophase, where the chromatids are pulled to each pole and a new nuclear envelope is formed around the new cell. Another phase of cell cytokinesis also occurs after telophase, when the cytoplasm fully divides, officially creating two new daughter cells (9).

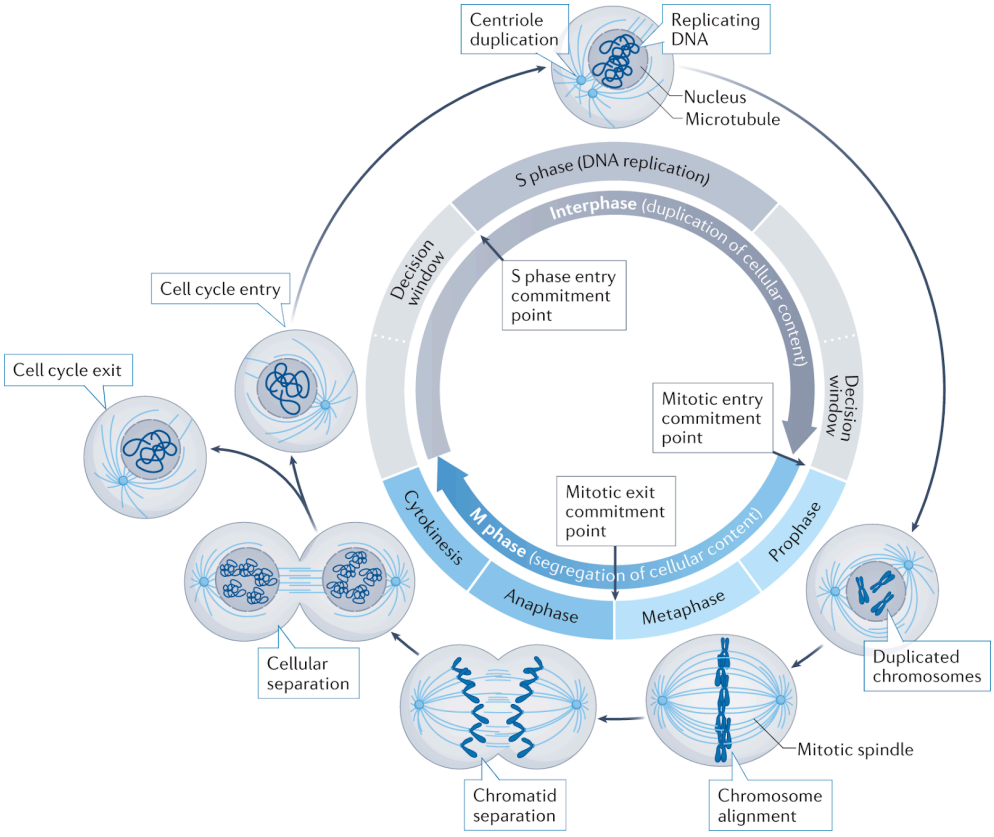

Figure 4. The eukaryotic cell cycle highlights key phases, checkpoints, and chromosomal events involved in accurate cell division. (12)

When Cellular Safeguards Fail: Oncogenes, Tumor Suppressors, and Defective DNA Repair

Various factors like CDK, cyclin, checkpoints, growth hormone, and environmental conditions collectively help regulate the cell cycle. These factors, especially CDKs and cyclins play a huge role in the DNA repair process. CDKs and cyclins are proteins that work together to create complexes that act as molecular switches to control the cell cycle and its progression. The cyclins bind to the CDKs to fully activate them, and when binding the complex the phosphorylates of certain target proteins to drive the cell cycle forward. These complexes are used at certain transitions between phases like G1 to S and G2 to M, as the degradation of the cyclins ensures that these complexes are only active in certain stages of the cell cycle.

Furthermore, specific genes, called proto-oncogenes and tumor suppressor genes, help regulate the cell cycle. These genes code for proteins that aid in regulating cell growth, differentiation, or signaling for apoptosis. Proto-oncogenes are proteins that have mutated from oncogenes, increasing their number of functions. With this mutation these genes now act like the cell cycle's gas pedal, meaning they help push along each cell to ensure all processes are working correctly and that division is going smoothly. Conversely, tumor suppressor genes act like brakes within the cell cycle, acting as brakes to stop the cell cycle if mutations are detected before continuing along their journey in the cell cycle. These two genes work hand in hand to regulate each cell cycle step. However, once one becomes mutated or dysfunctional, that is when cancer can start to form. Proto-oncogenes turn into oncogenes, which can speed up the cell cycle's pace, shortening the cell's time to replicate. Tumor suppressor genes, many of which regulate cell cycle checkpoints, stop working and allow the cell cycle to continue replication dramatically, causing uncontrolled cell growth and cancer (15).

The most prevalent tumor suppressors in osteosarcoma are TP53 and RB1. When these genes are mutated, they can lead to uncontrolled cell growth and disruption within the cell cycle. TP53 is a gene that provides instructions for making the p53 protein. This protein acts as a brake and regulates cell division to prevent uncontrolled cell growth. This protein binds to the DNA and stimulates the production of the p21, (a cyclin-dependent kinase inhibitor, meaning it can block the activity of CDKs, an enzyme that play a crucial role in regulating the cell cycle) that can bind and react with a cell division stimulating protein called Cdk2 (Fig. 5). With these two proteins together, any cell with a mutation within their genetic code that affects a protein that regulates the cell cycle will not be allowed to pass through to the next stage of the cell cycle, prohibiting any uncontrolled cell growth or mutations to occur. However, p53 cannot bind to the DNA when this protein mutates, allowing the cell to continuously go through the cell cycle, forming tumors (13). The next protein is the RB1 protein, which plays a role in DNA replication. This protein produces pRB, which stops other proteins from triggering DNA replication, allowing cell growth regulation. However, when mutated, the protein becomes dysfunctional, allowing cells to continue DNA replication without the proper regulation (14).

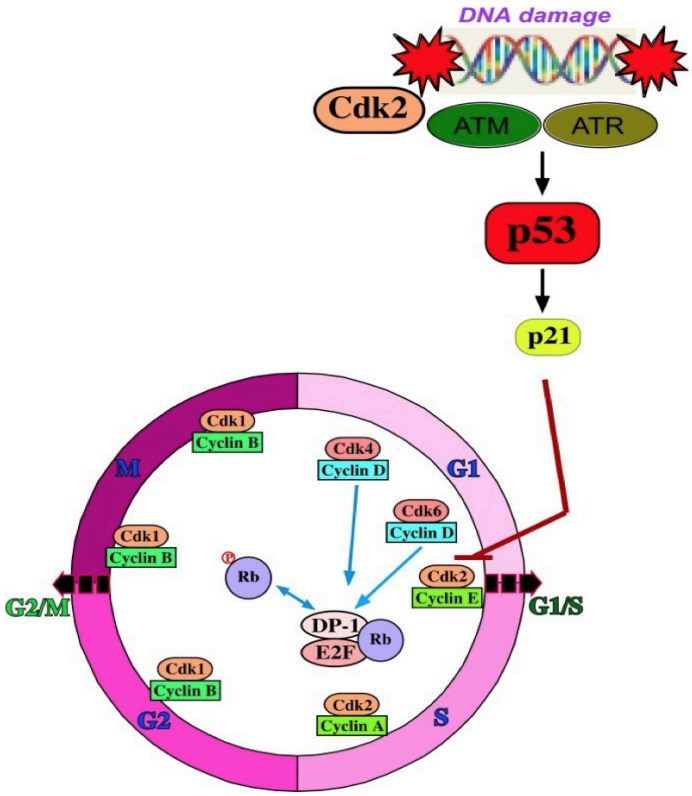

Figure 5. Regulation of the cell cycle by p53 and p21 in response to DNA damage, highlighting inhibition of CDK2 at the G1/S checkpoint (Figure adapted from 16).

The Role of the Bone Microenvironment in Tumor Initiation, Growth, and Metastasis

Although the dysregulation of oncogenes and tumor suppressor genes is fundamental in the initiation and progression of cancer, the tumor's microenvironment also plays a significant role in influencing tumor development and behavior. A tumor's microenvironment refers to the ecosystem surrounding the cancer in the body, which is composed of the immune cells, the extracellular matrix, blood vessels, and other cells like fibroblasts (17). This environment enables the cancer to grow by feeding it nutrients and giving it the space to continue to expand. In osteosarcoma, the continuous process of recycling and creating bone cells provides the perfect environment for cancerous cells to grow and thrive. This constant process of bone remodeling, where molecules like osteoclasts and osteoblasts are utilized, allows old damaged bone tissue to be replaced with new tissue, allowing for an environment full of growth hormones,

bone marrow niches, and continuous blood flow, thereby allowing the tumor to thrive and metastasize more easily.

Growth hormones stimulate the bone to produce more cells, but when redirected to the tumor, they encourage the creation of additional cells. Then the bone marrow niches, like endosteal and perivascular, support the hematopoietic stem cells, which can differentiate into any blood cell. Tumors can then migrate and utilize these stem cells not only for survival but also for drug resistance. Additionally,the bone remodeling cell osteoclasts (bone-resorbing) and osteoblast (bone-forming), are easily manipulated. This allows the process to be easily overtaken by the cancerous cells, which can cause bone destruction, also called osteolytic lesions, leading to a vicious cycle where the bone is slowly broken down to release more growth factors for the tumor to use to expand (18). Lastly, due to the vascular-rich environment in the bone, it's extremely easy for any bone cancer to create new blood vessels from existing ones that run within the bone, which is a process called angiogenesis. This allows the tumor to manipulate existing blood vessels to run through the tumor, making the cancer more independent overall. This process enables a tumor to take oxygen directly from the circulatory system, giving it more nutrients to grow and a pathway to metastasize to other body parts (19).

Bone metastases are a process where a cancerous tumor develops from a primary spot in the body but then spreads and invades into the bones. While it is infrequent for bone cancer to metastasize to other body parts or organs, it is relatively common for people with solid malignancies in some regions of the body to develop bone metastases. This process is most commonly found in cases of prostate, breast, and renal cancers. The likelihood of developing bone metastases in prostate cancer is 88.74%, for breast cancer it is 53.71% and for renal cancer it is 38.65% (38). Additionally, with bone metastases, the survival rate significantly decreases, and is very dependent on the location of the tumor before metastasis. As for people with lung cancer that spreads to the bone, their survival rate is less than 10%. In contrast, people with prostate or breast cancer who had spread were closer to around 50% (25). These statistics highlight the critical need for early detection and targeted treatment strategies, as bone metastases not only indicate advanced disease but also drastically reduce a patient’s chances of survival. One promising avenue of treatment involves targeting epigenetic changes in genes, which may help reverse malignant behavior and improve therapeutic outcomes in metastatic bone cancers.

Epigenetic Regulation in Osteosarcoma: Silencing Tumor Suppressors and Enabling Resistance

Epigenetic modifications play a critical role in the development and progression of cancers, especially osteosarcoma. These modifications can regulate gene expression without altering the actual DNA sequence (Fig. 6). Epigenetic modifications are modifications that can silence tumor suppressor genes or active oncogenes, ultimately promoting uncontrolled cell growth. One of these processes,called DNA methylation, is a process where an extra methyl group is added to a DNA molecule, and the effect on gene expression varies depending on the site of this addition. If added in a promoter region, the gene is usually silenced, while methylation in the gene body creates a more dense form of chromatin, making the process of gene transcription harder (34). Another epigenetic process is one that affects histones, which are what DNA is wrapped around to form chromatin, affecting the activity of genes. These histone modifications cause chemical changes to the histone proteins, can be things like acetylation, where an acetyl group is added, increasing gene expression, or methylation, where a methyl group is added and can either activate or repress gene expression based on the specific amino acid. Additionally, phosphorylation, where a phosphate group is added to a molecule, can ultimately affect the chromatin structure (35). Lastly, another modification in epigenetics is the usage of non-coding RNAs. These segments of genetic material that do not code for proteins but play a crucial role in regulating gene expression at both the transcriptional and post-transcriptional levels.These segments affect chromatin remodeling by reacting with different complexes to change gene expression, and post-transcriptional regulation, where these RNAs can bind to mRNA and affect its ability to translate proteins (36).

However, with these epigenetic modifications, there has been a rise in certain treatment options that may help regulate the effects of these modifications. Especially in osteosarcoma cases, using inhibitors like DNMT (DNA methyltransferases) and HDAC (histone deacetylase) has shown some promise. For DNMT it primarily works against DNA methylation by being able to restore the silencing of certain genes and aid in restoring expression in tumor suppressor genes. In addition, HDACs counteract histone modifications, promote apoptosis, and help

reverse epigenetic alterations that drive tumor progression, making them promising therapeutic targets in cancer treatment. However, despite these advances, the effectiveness of many therapies remains limited due to the growing challenge of drug resistance, a major barrier in the treatment of aggressive cancers like osteosarcoma.

The Rise of Resistance: Why Cancer Treatments Lose Effectiveness

With the use of drug-related therapies, the rise of a new problem, known as drug resistance, has become an issue for patients with cancer. Bone cancers, in particular, have many different properties that allow them to evade drugs and inhibit the drugs from performing correctly. Bone cancers have a rich microenvironment where they reside and have access to molecules that aid in specific processes to promote drug resistance. Bone cancers, due to the bone's rich and unique environment, have more subpopulations of cancer stem cells compared to other types of cancers. These cells possess stem-cell-like properties, including self-renewal and differentiation, which all aid in tumors' survivability, growth, and metastasis. Additionally, these cells show significant resistance to conventional drug therapies, paving the way for the cancer to resist the effects of drug treatments. This is shown in the various mechanisms these cells use to evade or inhibit chemotherapy drugs from working (Fig. 7). Mechanisms like Epithelial-mesenchymal transition (EMT), a process where epithelial cells lose their adhesion properties allowing for increased motility and invasiveness, thus allows for the formation of the cancer stem cells (CSCs) and for the maintenance of CSCs during treatment. Furthermore, these CSCs also have high levels of detoxification proteins like: ALDH, ABCG2, and NRF2, creating an overexpression of these proteins and the inability for chemotherapy drugs to properly work (33).

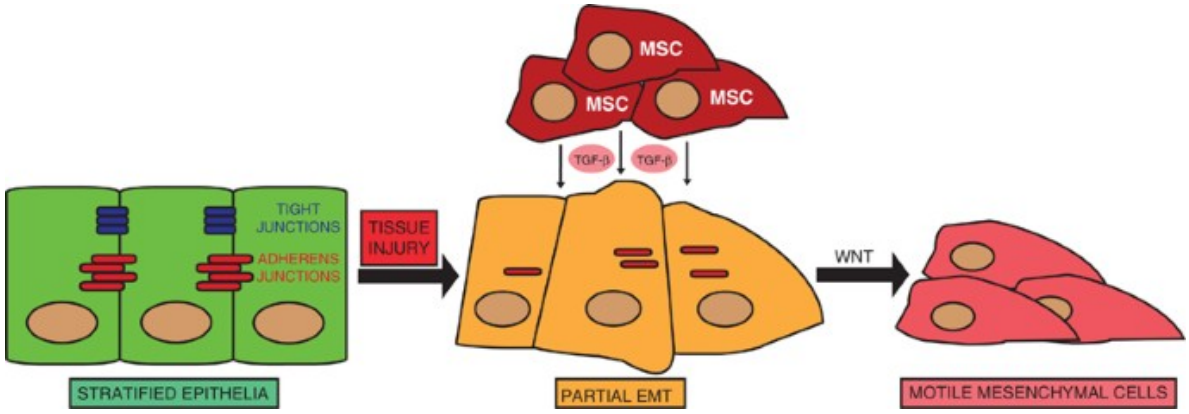

Figure 7. Schematic representation of epithelial-to-mesenchymal transition (EMT) following tissue injury (39).

Lastly, bone cancers have high tumor heterogeneity, which is the difference between each tumor cell. These differences include drug transport, metabolism, and other pathways that can help with drug resistance. The most common mechanisms of drug resistance shown in bone cancer include increased drug efflux and enhanced DNA repair. The efflux is the ability for the tumor to pump out the chemotherapy drugs more effectively, preventing them from reaching the target. Then, enhanced DNA repair is when the tumor activity can repair its DNA faster and more efficiently than the chemotherapy drug can work, making the tumor less susceptible to chemotherapy treatments (24). However, due to the increased issue of drug resistance, more chemotherapy drugs and medicines are being altered to combat this resistance and help the patients who have any cancer.

Future Directions in Bone Cancer: Innovations in Treatment and Research

Ongoing research and innovation are exploring novel approaches to bone cancer treatment. There have recently been a few new and innovative ways of helping patients that have been tested through research and clinical trials, which may be a breakthrough for treating bone cancer. The identification of new biomarkers in the early stages of bone cancer and bone metastases promises quicker diagnosis, more personalized treatment plans, and the integration

of new therapeutic strategies. Additionally, these biomarkers like BALP, TRACP, and OPN have already been identified as potential markers for the formation of bone cancer, and the increased detection of these biomarkers will help inform clinicians to add or reduce therapy and contribute to future clinical trial designs while also aiding in the patient's prognosis. The majority of emerging drugs and research in this field focus on two key areas: toxicity reduction and immunotherapy. These approaches aim not only to overcome drug resistance and target the tumor more effectively but also to offer less toxic and burdensome treatment options for patients (26).

In addition to biomarkers, there are also new forms of treatments in immunotherapy. This is a process where the patient's immune system is either enhanced to fight off the cancer on its own, or the cancer becomes more susceptible to the immune system attack. This process usually tries to stimulate the T cells to become more active in recognizing and destroying cancerous cells more efficiently. This is seen in the use of PD-1 and CTLA-4 inhibitors, as these pathways are easy for tumors to exploit and use to their advantage to avoid the immune system attacks, as PD-1 regulates cell activity and CTLA-4 regulates T cell early activation. However, by using inhibitors to block these pathways, it creates a way for the immune system to effectively attack the cancerous cells (31). Another immunotherapy on the rise is CAR-T cell, a process where a patient's T cells are collected and genetically modified to express a certain receptor called chimeric antigen receptor that not only targets and destroys cancer cells but also boosts the patient's immune system to recognize cancerous cells more easily (32). However, with these treatments come their limitations when it comes to treating bone cancer, like the poor immunogenicity of osteosarcoma cells, meaning that it's extremely difficult for a substance to trigger an immune response. Additionally, the bone's microenvironment has limited infiltration of immune cells, meaning it would be difficult for an attack to even reach the tumor environment, leading to decreased ability of the immune therapy to work.

Another new form of treatment administration is using nanoparticles for targeted drug delivery. Nanoparticles are extremely small molecules that have very unique properties that enable them to be used for new applications. They have an increased surface area compared to their volume, significant quantum effects like electrical and magnetic properties, and they have different optical properties as they exhibit unique colors and light-scattering properties. These

properties allow nanoparticles to be manipulated for accurate and precise drug delivery for chemotherapy drugs; minimizing the harmful effects of anticancer drugs on healthy cells while enhancing the efficacy and selectivity of the drugs, toward cancer cells, through loading, targeting, and controlled release, offering advanced capabilities in drug delivery (27). This usage of nanoparticles allows for the control and precision unavailable in regular chemotherapy drugs and would eliminate the many limitations and side effects of the current drug delivery system for chemotherapy drugs. This new technique and usage of a small particle show a significant advancement in treatment options for cancer patients.

Lastly, another newly founded treatment option is phototherapy. This treatment is an external and minimally invasive technique using specific light wavelengths that can induce photothermal conversion. Light energy is converted into heat in this process, leading to tumoricidal effects. This happens because the increase in heat denatures and affects the tumor cells, leading to dysfunction and the discontinuation of growth. Additionally, the temperature rise sends signals to the immune system to attack and kill the invading cells in the area, helping get rid of the tumor (28). Overall, these cutting-edge innovations, like biomarker detection and advanced therapies like nanoparticle drug delivery and phototherapy, show significant progress in the fight against bone cancer and other cancers. As research continues to evolve, these technologies hold great potential to transform cancer treatment into a more personalized, effective, and less invasive treatment. With continued scientific discovery and clinical advancements, the future of cancer care remains full of possibilities.

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