Commentary

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The Delivery of Commercialized Nanomedicines, But Not Small Molecule Drugs, Into Solid Tumors in Terms of Tumor Accumulation

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Description

In solid tumors, cancer stem-like cells rely heavily on hypoxia, making it difficult to develop commercially available nanomedicines. For the first time, we used hyperbaric oxygen therapy to help commercialized nanomedicines like Doxil and Abraxane get rid of CSCs in stroma-rich solid tumors like pancreatic ductal adenocarcinoma and triple negative breast cancer for effective cancer treatment. We demonstrated that HBO directly suppressed CSCs and cancer metastasis by disrupting hypoxia in solid tumors. More importantly, we discovered that HBO normalized tumor blood vessels structurally and functionally by depleting excessive extracellular matrix, such as collagen and fibronectin. In order to effectively eradicate CSCs and inhibit tumor growth, HBO increased the delivery of commercialized nanomedicines, but not small molecule drugs, into solid tumors in terms of tumor accumulation, deep penetration, and cellular internalization. These findings suggest that the combination of HBO and commercialized nanomedicines for the treatment of hypoxic solid tumors in clinics is promising because it makes it possible for HBO to eliminate CSCs in stroma-rich solid tumors. Nanomedicine has emerged as a result of the significant opportunities that nanotechnology has presented for healthcare, including novel diagnostic and therapeutic possibilities. Social science research has paid more attention to this new and complicated field in conjunction with these technological advancements. However, a systematic and coherent understanding of nanomedicine as a distinct socio-technical system is still lacking.

Drug Accumulation in Tumors and Reduce Toxicity

We examine the field of nanomedicine and identify key thematic areas in which it is developing by charting the existing literature and drawing on insights from science, innovation, technology, organizational studies, and other fields. As a whole, our research contributes to a better understanding of why and how nanomedicine can be best understood as a unique setting for new social science research questions. Through the enhanced permeability retention effect, anticancer nanomedicines aim to increase drug accumulation in tumors and reduce toxicity by reducing drug accumulation in normal organs through long systemic circulation. However, the differing efficacy and safety of nanomedicines in cancer patients and preclinical cancer models has sparked discussion regarding the design criteria for nanomedicines. Using five clinically used nanomedicines, we investigate the design criteria for better clinical translations of their observed clinical efficacy/safety than free drug or clinical micelle formulation in three types of preclinical cancer models. Long- and short-circulating nanomedicines improved tumor accumulations in subcutaneous and orthotopic breast cancer models, but they did not enhance tumor accumulation by EPR effect in transgenic spontaneous breast cancer models, regardless of their size or composition, when compared to drug solution or clinical micelle formulation. However, regardless of the breast cancer models, when tumors were compared to normal breast tissue, clinical micelle formulation, drug solution, and nanomedicines demonstrated enhanced tumor accumulation.

In addition, long-circulating nanomedicines did not generally reduce drug accumulations in normal organs or further increase tumor accumulation in transgenic mouse spontaneous breast cancer; They might have altered the clinical efficacy or safety by decreasing or increasing accumulation in various organs. Contrarily, the clinical efficacy and safety of short-circulating nanomedicines are correlated with their decreased blood concentration and altered drug distribution in normal organs. To ensure consistent clinical translation for the purpose of increasing their clinical efficacy and safety in cancer patients, the current design criteria for nanomedicines must be reevaluated. One method reportedly used to prevent anticancer nanomedicines from being delivered off-target was intralipid, a lipid emulsion that is currently in use in clinical settings; additionally, intralipid significantly enhanced drug delivery to tumors and enhanced therapeutic effects. However, there has not yet been a comprehensive report on the mechanisms involved—the why and how—in Intralipid's facilitation of nanomedicine delivery to tumors. When using three anticancer nanomedicines, including the clinically approved drug doxorubicin (Doxil), we investigated Intralipid and discovered its beneficial effects. A polymeric nanoprobe used in photodynamic therapy was absorbed 40% less by the liver after intralipid

pretreatment, and nanomedicine accumulation in tumors increased 1.5 times. Using Doxil, this finding was confirmed that this increased accumulation resulted in significantly improved therapeutic effects.

Nanomedicines Off-Target

Intralipid pretreatment significantly extended the plasma half-life of nanomedicines in healthy mice, but not in tumor-bearing mice. This interesting finding suggests that when liver delivery is suppressed, tumors may become an alternative delivery channel for nanomedicines. Fluorescence angiography revealed a significant increase in tumor blood flow as well as a significant decrease in blood viscosity following Intralipid pretreatment. All of our findings show that intralipid treatment not only reduced the reticuloendothelial system's ability to deliver nanomedicines off-target, but it also increased nanomedicine delivery to tumors by increasing tumor blood flow, which is essential for effective drug delivery due to its enhanced permeability and retention effect. The combination of intralipid pretreatment and the use of nanomedicines resulted in significantly improved therapeutic outcomes. As the master sensors and effectors of apoptosis, ATP production, reactive oxygen species management, mitophagy/autophagy, and homeostasis, mitochondria network together; the pharmaceutical industry can easily manipulate this organelle. Many diseases are caused by mitochondrial dysfunction. For instance, Amyloid has been shown to prevent mitochondrial protein import and cause apoptosis in Alzheimer's disease, while dysfunctional mitochondrial PINK1 and Parkin proteins are linked to some forms of Parkinson's disease. From cardiovascular disease to cancer, mitochondrial medicine can be used to treat a variety of diseases. The difficulty of targeting a subcellular target with therapies in mitochondrial medicineThe clinical translation and efficacy of mitochondrial medicine can greatly benefit from strategies based on nanotechnology and mitochondrial targeting. Methods for delivering drugs through mitochondria and their potential applications are the subject of this review. Methods from nanomedicine have the potential to bring mitochondrial therapies' clinical success to the forefront. The distribution and accumulation of nanomedicines in the tumor are slowed down by obstructed blood flow and erratic blood supply. Therefore, for effective drug delivery, it is essential to improve these conditions. In order to improve the accumulation of nanomedicines in the tumor, we created and synthesized a novel BK copolymer conjugate based on N-(2 hydroxypropyl)methacrylamide. This copolymer conjugate possessed sufficient systemic stability and tumor-selective action.

An acid-cleavable hydrazone bond (P-BK) was used to attach Levulinoyl-BK (Lev-BK) to an HPMA-based polymer. P-BK alone showed below 10% BK activity after intradermal application, indicating a decrease in BK's vascular permeability activity when attached to the polymer carrier. An acid-responsive release of Lev-BK was observed from P-BK.P-BK pre-treatment increased the tumor accumulation of pegylated liposomal doxorubicin (PLD) by approximately threefold and improved blood flow in the tumor tissue by 1.4–1.7 times, both of which were sustained for more than 4 hours. In addition, tumor-bearing mice's survival was significantly increased and P-BK pretreatment enhanced PLD's antitumor activity. P-BK's negligible vascular permeability enhancing activity required the release of BK from P-BK in the tumor's acidic environment for P-BK to work. Together, P-BK pretreatment resulted in a significant reduction in tumor growth by increasing tumor accumulation of nanomedicine and improving intratumoral blood flow. As a result, these results show that P-BK could be used in conjunction with other medications to improve nanomedicine delivery to tumors.