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Cancer Nano Medicine: Recent Advances in Tumor Targeting Using the EPR Effect

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Due to a leaky vasculature and poor lymphatic drainage in solid tumours, nanocarriers preferentially concentrate in the tumour through passive targeting. The permeability of a disordered vascular and Tumour Microenvironment (TME) combined with retention can result in a 70-fold increase in macromolecule accumulation in the TME. The EPR effect is enabled by the leaky and faulty vasculature generated as a result of the fast vascularization required for the maintenance of malignant tumours, along with poor lymphatic drainage. EPR-based drug delivery is influenced by a number of parameters, including circulation time, targeting, and the capacity to overcome obstacles, all of which are influenced by the drug particles' size, shape, and surface features. The majority of passive-targeting relies on a diffusion process. As a result, in the EPR-dependent delivery procedure, size is critical. According to studies, nanoparticles with a size range of 40 nm to 400 nm are appropriate for ensuring lengthy circulation duration and increased accumulation in tumours with decreased renal clearance. Because of the unique interaction of nanoparticles with phagocytic cells, the presence of phagocytic cells might promote an increase in nanoparticle concentration in the tumour microenvironment's vasculature.

While generating hypertension has been demonstrated to be a viable therapeutic technique, enhancing the EPR impact by a strategy that changes the patient's physiological condition poses problems since it might have a whole-body effect. The patient group for generating hypertension is confined to individuals who are not currently taking antihypertensive medication. Targeting the tumour microenvironment is another way to boost the EPR effect. Repairing faulty tumour vasculature has been found to promote the formation of tiny nanoparticles by lowering the tumor's interstitial fluid pressure.

Many small molecular anticancer agents are often ineffective at detecting or treating cancer due to their poor pharmacokinetics. The use of nanoparticles as carriers can help with this since their small size lowers clearance and increases retention inside tumours, but it also delays their rate of transfer from circulation into the tumour interstitium. When a molecular contrast agent and engineered nanoparticle undergo *in vivo* molecular assembly within tumours, the combination of rapid influx of the smaller component and high retention of the larger component provides rapid tumour accumulation of a fluorescent contrast agent, 16 and 8 fold faster than fluorescently labelled macromolecule or nanoparticle controls achieved.

Nanomedicine development is highly reliant on the preclinical models on which they are tested. It is widely documented that these models, particularly tumour xenografts, exhibit the EPR effect independent of tumour subtype, which has resulted in nanomedicines exhibiting significant advances in cancer imaging and therapy. Unfortunately, preclinical results are not replicated, or to the same extent, in patients. It is uncertain if preclinical models correctly represent all components of the EPR effect and whether they are also present in patients with diverse cancer kinds and stages, and further research is needed to determine this. A deeper clinical knowledge of the EPR effect is required to discover why various nanomedicines have failed clinical trials and how these difficulties may be addressed. Furthermore, understanding how the EPR effect changes with tumour heterogeneity intratumorally as well as between different tumour kinds and stages is required in order to design therapeutically relevant animal models and nanomedicines for those specific circumstances.

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