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Improving the Distribution and Therapeutic Efficacy of Anticancer Drugs

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EDITORIAL

Fibrin a component of the Extra Cellular Matrix (ECM), functions as a transport barrier within the core of tumour, restricting blood arteries and producing clots resulting in poor anticancer medication intratumoral distribution. A micro plasmin based thrombolytic ferritin nano cage that efficiently targets and dissolves clots without generating systemic fibrinolysis or destabilizing hemostatic clots was previously developed by our lab. We anticipated that thrombolytic nano cage mediated fibrin clot breakdown in the tumour ECM could result in improved intratumoral drug delivery, particularly for nano sized anticancer medicines. After surgery and chemotherapy fibrin clot deposition intensifies, obstructing drug delivery evens more. In addition, the risk of Venous Thrombi Embolism (VTE) rises. We created fibrinolytic nano cages by combining thrombolytic nano cages with multivalent clot targeting peptides and fibrin degradation enzymes like micro plasmin to disintegrate fibrin in the tumour microenvironment (FNCs). These FNCs selectively and successfully target tumour clots. FNCs effectively remove fibrin clots inside tumour arteries, suggesting that they may help cancer patients avoid VTE. In a syngeneic mouse melanoma model, administration of FNC with doxorubicin increased chemotherapeutic efficacy. Furthermore, the FNCs boosted doxil doxorubicin nanoparticle distribution within mice tumours. These findings show that fibrinolytic co therapy may aid anticancer nano medicine therapeutic efficacy. Because of their hemostatic safety and capacity to home in on the tumour, micro plasmin based fibrinolytic nano cages are potential candidates for this technique. The therapeutic efficacy of anticancer medicines is restricted by cellular reasons of resistance and poor drug distribution within solid tumours. Autophagy is mediated by acidic endosomes in cancer cells, which helps stressed cells, survive and may contribute to treatment resistance. Basic medicines, such as doxorubicin are trapped in acidic endosomes, directing them away from their target DNA and reducing their penetration into distant cells. The effects of the PPI lansoprazole on doxorubicin activity were investigated. Using *in vitro* and mouse models, we investigated the effects of lansoprazole on endosomal pH, doxorubicin spatial distribution and biomarkers reflecting its activity to learn more about its processes. Lansoprazole raised endosomal pH and inhibited doxorubicin endosomal sequestration in cultured tumour cells in a concentration dependent manner. Lansoprazole was not harmful to cancer cells, but it increased the cytotoxicity and penetration of doxorubicin in multi layered cell cultures. Lansoprazole improved the distribution of doxorubicin in solid tumours while simultaneously increasing the expression of drug activity indicators throughout the tumour. When compared to either medication alone, a combination of lansoprazole and doxorubicin was more effective in slowing tumour growth. Lansoprazole and doxorubicin work together to improve doxorubicin's therapeutic benefits in solid tumours by enhancing its distribution and raising its activity. The use of PPIs to promote medication distribution and suppress autophagy is a promising technique for improving anticancer treatment efficacy in solid tumours. In the field of medication development, tumour targeting drug delivery has gotten a lot of attention. Because traditional anticancer treatments are generally nonspecific for tumour cells, tumor targeting drug delivery is a possible way to increase chemotherapy's therapeutic efficacy. PUFAs, such as linoleic acid, linolenic acid, arachidonic acid and docosahexaenoic acid are naturally occurring vital chemicals that play important functions in cell proliferation. PUF As are easily integrated into the lipid bilayer of cells, particularly tumour cells, due to their lipophilic nature.

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