2. Μη τεχνική περίληψη του έργου

Current chemotherapeutic agents, in sufficient concentrations, are potent enough to kill cancer cells. Nonetheless, failure of standard chemotherapies for many cancers (e.g. breast, colon and pancreatic cancers and various sarcomas) is primarily because these agents will never reach cancer cells in amounts sufficient to cause complete cure. In solid tumors, blood vessels are compressed and sometimes collapsed, drastically reducing perfusion and resulting in insufficient delivery of any blood-borne therapeutic agent. Vessel compression is an effect of physical forces developed within the tumor due to unchecked cancer cell proliferation in the confined space of the host tissue. Alleviation of these forces has the potential to reopen compressed vessels and improve tumor perfusion. Increased perfusion, in turn, would enhance delivery of therapeutic agents and improve the efficacy of the treatment leading to increased overall survival. In this project, the hypothesis to be tested is that modification of the tumor microenvironment with common anti-fibrotic drugs, already approved for clinical use, has the potential to alleviate forces in highly desmoplastic and hypo-perfused tumors and thus, enhance chemotherapy. To explore this hypothesis, *in vivo* studies in immunodeficient mice will be performed to indentify the anti-fibrotic drug that more effectively alleviates forces and reopens tumor vessels. Subsequently, this anti-fibrotic drug will be combined with chemotherapy to prove that combined treatment of an anti-fibrotic drug with chemotherapy is more effective than chemotherapy alone in improving the overall survival in mice bearing tumors.