Hillary Myears, Biochemistry

University: University of Missouri-Columbia
Year in School: Senior
Hometown: Springfield, Missouri
Faculty Mentor: Dr. Grzegorz Sowa, Medical Pharmacology & Physiology

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Identifying the role of caveolin-1 and caveolin-2 in cancer cell proliferation and cancer drug resistance

Hillary Myears and Grzegorz Sowa

Caveolae are flask-shaped invaginations of the plasma membrane. Caveolins are the major protein component building these caveolae. Caveolins are involved in cell signaling and play a role multiple subcellular processes, such as endocytosis. Of the three types of caveolins (numbered 1, 2, and 3), most research has been performed on caveolin-1. For example, in the field of cancer, it has been found that caveolin-1 has the ability to either suppress or encourage growth of cancer cells (depending on the type of cancer). Interestingly, it has been reported that the level of both caveolin-1 and -2 increases in drug resistant cancer cell lines. However, it is unknown if such increases are coincidental or actually play an important role in the development of drug resistance. The major goal of this study is to better understand the role of caveolins-1 and -2 in the growth and drug resistance of A549 lung adenocarcinoma. For this purpose, the levels of caveolin-1 and -2 were dramatically reduced (knocked down) using small interfering RNA (siRNA) stably expressed in A549 cells. The efficient reduction of caveolin-1 and -2 levels by siRNA has been confirmed by Western Blot. By using colorimetric assay and cell count, we are currently determining the possible differences in growth of A549 cell populations expressing control siRNA and caveolin-1 or -2 siRNA in the absence or presence of different concentrations of two anticancer drugs, i.e., taxol and etoposide. We hypothesize that in the absence of drugs, cells with a knockdown of caveolin-1 or -2 will grow equally as fast as or faster than control cells. Conversely, we expect that knockdown of caveolins will increase sensitivity of A549 to anti-cancer drugs and limit the possibility of developing drug resistance. Such results would be a first direct evidence that caveolin-1 and/or -2 are involved in acquiring drug resistance. If the latter is true, targeting caveolins in lung adenocarcinomas and other cancers could limit or even prevent the development of drug resistance and thus increase the efficacy of anti-cancer drugs.