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So prostate cancer currently rates as the second most diagnosed cancer in men worldwide. While the primary tumour is fairly innocuous, patients with the more advanced prostate cancer face limited treatment options and high mortality rates due to additional comorbidities. These reasons highlight the necessity to understand and identify biological phenomena and biomarkers that induce the metastatic phenotype for future treatment options.

Caveolin-1 is an important biomarker for cancer progression. In healthy human cells, caveolin-1 is co-expressed and co-localized with putative tumour suppressor, cavin-1, to evoke their canonical function. However, in the case of many cancer types, caveolin-1 is overexpression without corresponding binding partner, which has been linked to many of the hall marks of cancer and cancer progression. Interestingly, knocking down caveolin-1 or adding cavin-1 to a cancer cell line that exerts this activity reduces the oncogenic phenotype induced by the caveolin.

We’ve used this information previously on the advanced prostate cancer cell line, PC3. This cell lines lacks cavin-1 expression, but over expression on caveolin is thought to contribute to many of the aggressive processes. When transfected with cavin-1, our lab determined that this transforms the usually aggressive PC3 cell line to a more placid form by nutralising caveolin-1. BY using this model, we can compare between the cell lines, between the aggressive prostate cancer cells and less aggressive cell lines to identify the pathways that caveolin-1 is involved with but also the processes that lead to the increase mortalities associated with advanced prostate cancer.

Using this model, we focused on the role of extracellular vesicles in these cell lines. These vesicles compartmentalise material from the host cell and transport it to the recipient cell, which the content is then absorbed into the endogenous population to evoke their canonical function. This is an import mode of intracellular communication that has been of resent focus in cancer research. Here, the EVs secreted contain content from the host tumour cell that initiate modifications of the microenvironment and establishment of premetastaic niche when absorbed into the recipient. Primarily this was believed to be due to proteomic content, however our work found that the expression of cavin-1 modulates EV specific concentrations of microRNAs.