Bone metastasis is the most common complication derived from advanced prostate cancer formation. While the primary tumour can be treated and removed efficiently resulting in almost 100% survival, patients inflicted with metastatic prostate cancer possess a reduced 5-year survival rate of 29.3%. This highlights the necessity to identify therapeutic targets and underlying biological phenomena that induce the metastatic phenotype.

Caveolin-1 has been linked to prostate cancer metastasis and has been a speculated biomarker for cancer progression. This protein usually functions as a cholesterol transporter, where its interaction with cytoplasmic protein Cavin-1 initiates the formation of specific lipid microdomains on the plasma membrane called caveolae. These proteins are co-expressed and co-localised in healthy human tissue, however in the case of many cancer types only caveolin-1 is expressed. Increased proliferation, migration and differentiation are a result of the aberrant caveolin-1 expression, yet, the mechanism that links caveolin to these phenotypes is still actively being investigated. Additionally, ectopic expression of functional cavin-1 in caveolin-1 only expressive cells, such as the PC3 advanced prostate cancer cells, truncates many of the pro-tumorigenic properties observed by these cell lines. This is a common tool used to assess the pathogenicity of overexpressed caveolin-1.

Earlier work from our lab utilizes the caveolin-1/cavin-1 system to investigate the role of caveolin-1 in prostate cancer. Interestingly, the cellular modification inflicted by comparing between PC3 and PC3 cavin-1+ cells modified extracellular vesicle content, a pathway unrelated to the function of caveolin or cavin-1. Recently, particular interest into EVs has sparked due to its speculated role in cancer metastasis. Secreted membrane-bound vesicles, consisting of exosomes and microvesicles, collectively called extracellular vesicles (EV) are important mediators of intercellular communication (Figure 1). Exosomes are defined as 40-100nm diameter extracellular vesicles which are released upon fusion of the multivesicular bodies with the plasma membrane (Gu *et al.* 2014). Whilst similar in function and biochemical markers, microvesicles (≥100nm) differ from exosomes by being released from budding of the plasma membrane (Minciacchi *et al.* 2015). EV cargo consists of cytoplasmic material, functional RNA and proteins, where this content can differ between the subpopulations (Stoorvogel 2015). Secretion and uptake of the extracellular vesicles has been reported to influence a range of biological processes, such as the selective export of cytokines in immunological responses and establishing a pre-metastatic niche in cancer progression (Campos *et al.* 2015; De Toro *et al.* 2015). #Need to shorten this bit. However, studies have emerged that determined cancer-derived EVs absorbed into recipient cells induce establishment of the pre-metastatic niche. Primarily this is attributed to the proteomic EV content being introduced into the endogenous population of the target cell, such as introduction of beta-catenin, epidermal growth factor and major elements of the MAPK pathway. However, more intriguing is the reports of microRNA export associated with this function.

MicroRNAs (miRNAs) have been found to be involved in most developmental and pathological processes due to its ubiquitous gene regulatory function. The functional miRNA sequences (~19-24 nt) are derived from longer transcripts that undergo processing and shuttling events to give rise to functional mature sequences, known to induce RNA degradation (Ha and Kim 2014). Typically, the mature miRNA sequence interact with the 3’ untranslated region (3’-UTR) of its target transcripts and guides a multi-protein RNA induced silencing complex (RISC) to destine these molecules for degradation or translational inhibition (Djuranovic et al. 2012). A 2009 estimate predicted that approximately 60% of the mammalian genome is able to be directly mediated by the microRNA RISC mechanism. This instigates the necessity of tight temporal and spatial control over miRs to prevent dysregulation of vital pathways. Indeed, this is maintained by the high content of RNases in the extracellular space which would rapidly degrade any miRNAs that attempt to translocation across extracellular space. However, lipid-bound miRNAs are able to bypass this degradation and thus allows for the absorption of these molecules into recipient cells, thus evoking their canonical function in a potentially diverse cell type.