Fatty acids and prostate cancer: a journey from the lab to the clinic

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Cancer cells have different metabolic requirements compared to normal cells, as they need to survive in an altered microenvironment and are often highly proliferative. Data generated from tumour explants identify that the phospholipidome can inform on tumour responsiveness to front-line therapeutic use. For example, changes, or lack thereof, in the phospholipidome following treatment with anti-androgen correlate with proliferation. This correlation may be linked to one of the most common metabolic alterations in cancer cells, being increased lipid synthesis (lipogenesis), which is regulated by androgens. However, little attention has been paid to the metabolism of extracellular-derived fatty acids, including those secreted from local adipocytes and how this many influence tumour behaviour. We have observed that adipocyte lipolysis (triglyceride breakdown) is stimulated by prostate cancer cells, which in turn take up adipocyte-derived fatty acids and exhibit enhanced proliferation. This supports a model in which prostate cancer cells mobilize and utilize fatty acids from adipocytes. Future experiments will test where phospholipid species can be used as non-invasive detection modalities to predict and/or track tumour response to therapy.