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DEGREE:	Philosophiae Doctor
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DEPARTMENT:	Department of Biosciences
AREA OF EXPERTISE:	Prostate cancer
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DISSERTATION TITLE:	*The transcriptional role of c-Myc in prostate cancer*

c-Myc is a notorious protein involved in the initiation and progression of multiple malignancies. However, surprisingly little was known about its role in prostate cancer. Stefan J. Barfeld aimed to tackle this issue and elucidated in his PhD work the role of c-Myc in the most common cancer in men.

Every year, about 4,000 men die as a result of prostate cancer in Norway alone. Decades of research have improved detection and treatment options but nonetheless, prostate cancer often remains a fatal disease. Dysregulation of the androgen receptor, a testosterone-binding protein that controls cell proliferation, has been identified as the main cause and driver of the disease. However, a range of other proteins have been shown to play vital roles in prostate cancer. Amongst these, c-Myc is a factor that has been extensively studied in various cancers, especially lymphomas. Surprisingly, the contribution of c-Myc to prostate cancer development and androgen receptor activity has not been thoroughly examined yet.

In his PhD work, Stefan J. Barfeld identified c-Myc to be the driver of a variety of supporting metabolic pathways in prostate cancer, including purine *de novo* biosynthesis. This pathway is crucial for rapidly dividing cancer cells, which need large quantities of purines to sustain proliferation. Notably, disrupting this pathway slowed cancer cell growth and induced a cellular stress response.

Moreover, the impact of elevated c-Myc levels on androgen receptor activity was examined. Strikingly, c-Myc overexpression dysregulated androgen receptor activity and suppressed a range of well-established androgen receptor targets. These

findings have potential implications for future prostate cancer patients as they provide novel insights into the molecular basics of the disease, which could offer treatment alternatives to current therapies.