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Targeting ribosome biogenesis and function to treat prostate cancer

Prostate epithelium is exquisitely sensitive to the overexpression of the proto-oncogene MYC which causes neoplastic transformation. Indeed, MYC protein is almost universally overexpressed in metastatic castration resistant prostate cancer (CRPC) making targeting MYC an attractive option for treating advanced stage disease. Unfortunately, the development of therapeutic agents directly targeting MYC has been largely unsuccessful, thus emphasizing the need to indirectly target MYC activity through inhibition of downstream cellular processes it regulates. One of the main effects of MYC in cancer cells is to accelerate proliferative growth via stimulation of high levels of ribosome biogenesis. Accordingly, the control of protein synthesis rate has emerged as the “Achilles’ heel” of a wide array of tumors. MYC also regulates and cooperates with PIM kinases to increase the activity of the eIF4F translation initiation complex and MYC-driven tumors are addicted to eIF4E. Here, we investigate the efficacy of a single and dual approach targeting ribosome biogenesis and function to treat prostate cancer (PC).