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ABOUT THE COVER

The inhibition of androgen signaling is a major therapeutic strategy in prostate cancer; however, response is often transient, and patients ultimately relapse on therapy giving rise to a currently incurable condition known as castrate-resistant prostate cancer (CRPC). McCourt and colleagues show elevated expression of the androgen-regulated antiapoptotic protein c-FLIP in prostate cancer, which is further elevated in CRPC. Repression of c-FLIP induced apoptosis in non–castrate-resistant and CRPC cells and potentiated sensitivity to AR-targeted therapy, indicating that prostate cancer cells require c-FLIP to maintain viability. Consequently, targeting c-FLIP may represent a novel strategy to improve therapeutic response to the novel antiandrogen strategies under clinical development in CRPC. For details, see the article by McCourt and colleagues on page 3822 of this issue.