2002). В опытах in vivo на мышиной модели РПЖ показано, что сверхэкспрессия β-катенина вызывает выраженную интраэпителиальную неоплазию и резистентность к антиандрогенной терапии (Yu et al., 2009).

Изложенные данные из литературы показывают, что pRb и β-катенин имеют множественные точки синергичного и антагонистичного влияния на продукцию AR и прогрессирование РПЖ. Дальнейшее изучение механизмов сочетанной роли этих факторов может раскрыть новые стороны патогенеза локализованного РПЖ и его перехода в КР-РПЖ.

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Образцы ткани предстательной железы получали от пациентов, подвергнутых радикальной простатэктомии по поводу рака ПЖ в отделении урологии Ленинградского областного клинического онкологического диспансера им. Л.Д. Романа. Забор материала проводили согласно протоколу № 19, утвержденному биоэтической комиссией Института цитологии РАН 4 мая 2023 г. и согласованному с отделом клинических исследований онкологического диспансера им. Л.Д. Романа. Экспериментов с участием животных авторы не проводили.

КОНФЛИКТ ИНТЕРЕСОВ

Авторы заявляют об отсутствии конфликта интересов.

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INTERACTION OF pRb AND beta-CATENIN IN CANCER AND NORMAL TISSUE IN THE HUMAN PROSTATE

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Prostate cancer (PCa) is one of the most common oncological diseases, which goes through two stages in its development. The first stage, localized prostate cancer, can proceed indefinitely in a dormant form that does not require active medical intervention, or suddenly turn into an aggressive metastatic form with lethal outcome. The pathogenesis of the transition of the dormant form of PCa to the metastatic form remains not fully understood. The signaling pathways of the tumor suppressor pRb and the proto-oncogene β-catenin are probably the most involved in the pathogenesis of PCa but the role of their interaction in the pathogenesis of prostate cancer has not been studied. The publication on the pathogenesis of tumors in other tissues suggests that pRb may lose some properties of a tumor suppressor at the initial stage of PCa development due to its interaction with β-catenin that enables tumor cells to gain competitive advantages for reproduction. In this work, we have shown that the *RB* and β-catenin (*CTNNB1*) genes are well expressed in tumor and normal prostate tissue. Unlike β-catenin, pRb is not detected by immunoblotting in tumor and normal prostate tissue, but is easily determined in this way in extracts of control T98G cells. Co-immunoprecipitation with antibodies to pRb from extracts of tumor and normal prostate tissue makes it possible to detect this protein and β-catenin by subsequent immunoblotting, which indicates the physical interaction of these proteins in prostate tissue. On the other hand, immunoprecipitation of β-catenin with antibodies to its C-terminal fragment does not detect this protein in prostate extracts by subsequent immunoblotting using the same antibody. In contrast to prostate tissue, β-catenin is readily detected by immunoprecipitation combined with immunoblotting in T98G control cell extracts. The obtained data suggest that pRb and β-catenin physically interact with each other in cells of different tissue specificity. In T98G cells, this interaction probably occurs through the C-terminal fragment of β-catenin, but in prostate cells it occurs in a different way, since the C-fragment of β-catenin is shielded from such interaction, possibly due to its physical association with pRb.

Keywords: localized prostate cancer, signal pathway, pRb, β-catenin, interaction