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Resumo	Diabetic bladder dysfunction (DBD) is the most prevalent complication of diabetes mellitus (DM), affecting >50% of all patients. Currently, no specific treatment is available for this condition. In the early stages of DBD, patients typically complain of frequent urination and often have difficulty sensing when their bladders are full. Over time, bladder function deteriorates to a decompensated state in which incontinence develops. Based on studies of diabetic changes in the eye, kidney, heart, and nerves, it is now recognized that DM causes tissue damage by altering redox signaling in target organs. NADPH oxidase (NOX), whose sole function is the production of reactive oxygen species (ROS), plays a pivotal role in other well-known and bothersome diabetic complications. However, there is a substantial gap in understanding how NOX controls bladder function in health and the impact of NOX on DBD. The current review provides a thorough overview of the various NOX isoforms and their roles in bladder function and discusses the importance of further investigating the role of NOXs as a key contributor to DBD pathogenesis, either as a trigger and/or an effector and potentially as a target.
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