Endocrine disrupting chemicals (EDCs) produce adverse effects associated with numerous pathologies: neurological disorders, metabolic diseases, infertility and cancer for example. How EDCs interfere with the development of hormone-sensitive tissue is a major question in biology. In this study, we propose that the model of prostate organoids can be used to study the effects of EDCs on the development of the prostate gland. Indeed, the differentiation of adult stem cells (ASC) isolated from prostate into organoids recapitulate quite faithfully the development of the gland. We isolated ASC from mouse prostate (7 to 9 weeks) using flow cytometry. We cultured them in matrigel with a specific organoid medium containing dihydrotestosterone (DHT). These culture conditions allowed the proliferation and the differentiation of ASC. This resulted in a 3D cellular structure composed of an external basal cell layer, an internal luminal cell layer and a central cavity containing luminal cell secretions. Once developed, organoids could be stained and imaged using confocal microscopy in order to analyze phenotypes induced by EDCs. DHT is known as the main regulator of the prostate gland differentiation, we first explored the kinetics of DHT regulation. We showed that prostate organoid development was divided in two phases. The first one was a hormone-independent phase from day 0 (D0) to D4, during which a beta-catenin gradient was formed along organoids radius. This gradient could control early differentiation of adult stem cells into luminal-like cells. The second phase, from D5 to D9, was DHT dependent and induced the late differentiation into secretory luminal cells leading to a lumen formation in the center of organoids. We investigated the effect of anti-androgenic compound DDE on lumen formation in prostate organoid. We used mathematical approaches to increase the robustness of our model and evaluate the actions of EDCs on normal differentiation and development of organoids. Moreover, we hypothesized that luminogenesis may involve ion channels whose expression or functionality are likely to be regulated by hormones, as previously reported in intestinal organoids. The interaction of DHT and DDE and the cystic fibrosis transmembrane conductance regulator (CFTR) is currently studied in prostate organoids. Thus, we present here the organoid model as a new screening platform to study the effects of EDCs on the prostate gland by combining an analysis of organoids morphological features with mathematical modeling.