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In the Spotlight

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Early Risk Factors for Prostatic Hyperplasia

Prostate cancer constitutes a major health concern, as it is the second most commonly diagnosed cancer in Western males, with 1 in 36 American men dying of this disease.[1] In the United States alone, the estimated total economic burden of prostate cancer for 2010 was US$11.5 billion.[2] Therefore, identification of modifiable risk factors predisposing to prostate cancer is essential to avoid the adverse prostate outcomes and to lessen the burden this disease causes on the society. Since prostate development occurs many decades before disease presentation, it has been suggested that environmental exposures during fetal and early childhood developmental stages may contribute to the predisposition of prostate cancer later in life.[3]

Benesh and coauthors have previously shown, while developing a murine precancer model, that maternal diet-induced obesity stimulated prostate hyperplasia in the offspring later in life and that it could present as a candidate for a modifiable risk factor for prostate cancer initiation.[3] Now, in the present issue of *Reproductive Sciences*, the authors expand their studies further to show that additional environmental factors contribute to the prostate hyperplasia observed previously.[4]

The authors use a generalized linear model to show that aging and maternal diet-induced obesity each correlate with prostatic hyperplasia. However, their results evidence that prostate hyperplasia does not correlate with the length of the maternal diet-induced obesity. Instead, the authors find that cage density is positively correlated with both offspring body weight and prostate hyperplasia. To further understand these associations, Benesh et al studied the expression of the glucocorticoid receptor in the prostate and observed that it too is positively associated with cage density and negatively correlated with the age of the animal. Thus, taken together, the results presented by Benesh and colleagues suggest that the murine prostate tissue is negatively impacted by maternal overnutrition during fetal development and that it is susceptible to further alterations by environmental factors, such as overcrowding, which may lead to competitive behaviors, increased corticosteroid hormone signaling, subsequent overeating, and increased body weight.

Thus, the present study not only adds to list of factors impacting prostate development[5,6] that can lead to predisposition to disease but also highlights the importance of modifiable environmental factors in the development of precancerous phenotypes, leading to an interesting avenue of future studies. Additionally, it raises awareness for population dynamics in the interpretation of findings using caged animals, as animal health exhibits plasticity in response to exploitation of resources and the physical restraint of crowding, which may undesirably influence the results obtained.

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