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INVITED COMMENTARY

Glucocorticoid metabolism in testicular tissue: a key to fertility?

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Glucocorticoids are well-known as mediators of the systemic stress response. Increased glucocorticoid release and synthesis in response to endogenous or exogenous stressors have been shown to impair testicular hormone production in a variety of species and therefore may affect fertility. However, the physiological involvement of glucocorticoid hormones in tissue maturation and differentiation during both fetal and postnatal life is at least of similar importance as their role in the stress response. A well-regulated balance between beneficial and detrimental glucocorticoid hormone levels is therefore of utmost importance for tissue development as well as maintenance of tissue function. In the issue of the *Asian Journal of Andrology*, Zhou *et al.*¹ have published a study investigating the involvement of 11 β -hydroxysteroid dehydrogenase (11 β HSD) types 1 and 2 in the maturation of rat testis. These isohormones catalyze the interconversion of active glucocorticoids and inert glucocorticoid metabolites at the cellular level. In their article, Zhou *et al.*¹ are able not only to describe the expression pattern of the two isoforms during testicular development in the male rat. They also address the relevance of changes in the endocrine environment obtained by experimental manipulation of luteinizing hormone and testosterone concentrations on 11 β HSD expression and testicular development. In addition, the use of immunohistochemistry allows describing cell-type related differentiation in enzyme expression and its response to the changes in hormone concentration. Not all results presented in this article are new, but it very nicely highlights the complexity of the 11 β HSD system in the male gonad. Due to the fact that research with laboratory animals

allows for collection of tissue samples in a high number of experimental animals and at a variety of maturational stages, a near-complete picture of changes in the expression of the 11 β HSD system can be presented. This is extremely helpful for understanding the physiology and pathophysiology of testicular maturation – most probably not only in the male rat. Results obtained in laboratory animals can only in part be transformed to other mammalian species. However, the presence and relevance of the 11 β HSD system in testicular tissue maturation has been demonstrated not only in laboratory rodent species² but also in humans,³ some domestic animals (i.e., pigs⁴ and horses⁵) and in fish species. An involvement of these enzymes in testicular maturation and function in many other species thus appears feasible. The present results clearly underline the importance of fine tuning of glucocorticoid concentrations in male gonads for the development of fertility. Disturbance of these regulatory pathways may be a key factor to understanding of male infertility.

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