

Stress Modulatory Effect of Estrogen on Reproduction Mediated by Alterations in the Expression of Estrogen Receptor Alpha

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Animals are frequently exposed to various kinds of environmental stressors and estrogen is known to play important role in stress response besides its crucial role in regulation of cellular proliferation, metabolic activity and reproduction. Over past two decades work has been done to examine the role played by estrogen on reproduction and corticosterone in stress response of birds [1,2]. It has been observed that elevated stress increases corticosterone but reduces estradiol in birds thereby affecting its reproduction. Exogenous treatment of estradiol and progesterone suppresses the elevated level of corticosterone [3]. Estradiol is also known to play an important role in expression of anti-oxidant enzymes [4]. Several experimental approaches performed by RT-PCR show that the expression of ER α mRNA is present in each compartment of the reproductive tissue in quail; the expression of ER α mRNA in the granulosa layer of the largest follicle indicates the involvement of estrogen in the biosynthesis of inhibin/activin, progesterone and yolk peri-vitelline layer protein [5]. A very high expression of ER α in the shell gland has been related to the role of estrogen in cell proliferation and protein synthesis in the oviduct. Estrogen induced proliferative and reproductive effect has been observed in the shell gland mediated by its receptors during different photoperiodic conditions [6]. Estrogen mediated cell proliferative effects are induced by the action of estrogen receptor alpha (α) in shell gland. Abundant expression of ER α is observed by immuno-fluorescence in the shell gland of mature egg laying birds while stressors down-regulate its expression [7]. Taking into account the widespread localization of ER α in shell gland, it can be suggested that estrogen produced locally is the relevant hormone involved in down-regulation of egg-laying during stress. Thus, the reproductive potential of birds decreases under the influence of stressors mediated by estrogen and its receptor alpha.

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