LUTEINIZING HORMONE RECEPTOR EXPRESSION IN THE FEMORAL HEAD SUBCHONDRAL BONE OF THE CANINE COXOFEMORAL JOINT

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Gonadectomy has been associated with a 1.5-2 fold increase in the occurrence of canine hip dysplasia independent of breed or concurrent obesity.[1] In intact dogs, luteinizing hormone (LH) is secreted from the adenohypophysis to stimulate the synthesis of gonadal hormones, which negatively feedback to regulate LH secretion. Conversely, in gonadectomized dogs, no negative feedback occurs and LH concentrations are up to 30 times greater.[2] LH receptors (LHR) are present in non-reproductive tissues and their activation is associated with several long-term health conditions in dogs (e.g. urinary incontinence).[3] Our objective was to use immunohistochemistry to determine if LHR were present in the femoral head subchondral bone. We hypothesized that LHR would be present and that gonadectomy would increase LHR expression. The femoral head was removed from canine cadavers postmortem, formalin-fixed, decalcified in Cal-Ex for 14 days, paraffin-embedded, and sectioned (6 μm) onto charged slides. Epithelium from a separate dog was treated in the same manner as a positive control. All slides were deparaffinized, rehydrated, and subjected to heat-induced epitope retrieval. Endogenous peroxidase activity was inactivated with 3% hydrogen peroxide. Nonspecific binding was blocked with 1% rabbit serum. Goat polyclonal anti-human LHR antibody was applied at a 1:50 dilution. Negative controls from each tissue were treated in the same manner without primary antibody. Slides were then reacted with biotinylated rabbit anti-goat IgG and incubated with preformed avidin-biotin-peroxidase complex followed by Nova Red Peroxidase substrate. Slides were counter-stained with hematoxylin, dehydrated, and mounted. LHR immunoexpression was detected using bright-field microscopy at 400X magnification and osteocytes with visible nuclei were counted. Although the number of positive cells varied, LHR were present in all dogs studied. There was a trend for intact dogs to have more LHR positive cells (52.4±40.8%; n=5) than gonadectomized dogs (27±25.4%; n=10; p=0.079). The etiopathogenesis for the increased incidence of hip dysplasia in gonadectomized dogs remains unknown, but the presence of LHR in combination with increased serum levels of LH suggests that persistent LHR activation in this structural support tissue may play a role. LHR activation is known to stimulate nitric oxide release, which in predisposed breeds could result in excessive joint laxity and the onset of hip dysplasia.[4]

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