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HORMONE-MEDIATED REGULATION OF HEPATITIS DELTA ANTIGEN EXPRESSION IN HEK 293 CELLS

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Persistent low-level infections of the Hepatitis Delta Virus (HDV) have been suggested to lead to the full development of primary Sjogren's syndrome (pSS). Primary Sjogren's syndrome is a chronic autoimmune disease characterized by decreased tear and/or saliva production, inflammation within salivary gland tissues and development of autoantibodies. Women are 9 times more likely to be diagnosed with pSS and are most often diagnosed during early-stage menopause where progesterone, estrogen and testosterone levels decrease. We hypothesize that differential hormone profiles associated with peri- or post-menopausal women may lead to increased HDV antigen expression and increased risk of pSS development.

Therefore, a study was designed to identify whether changes in concentrations of these hormones affect the expression of the HDV antigen in an in-vitro system. HEK 293 cells expressing the small HDV antigen under control of a tetracycline-inducible promoter (HEK-293-SAG) were cultured in 96-well plates with media containing gradients of hormone concentrations within physiological constraints. Testosterone, progesterone, B-estradiol, and DHEA were selected as study hormones. HEK-293-SAG cells were incubated for 3 days +/- hormone exposure and +/- 0.1 ug/mL tetracycline to activate the tetracycline promoter. The cells were incubated under experimental conditions for 72 hours. At the termination of the study, RNA was isolated, and the antigen copy number was quantified via qPCR. An increased HDV transcript copy number indicates stimulation of antigen transcription while a decreased copy number indicates inhibition.

Based on previous research, it is expected to observe an increase in HDV antigen transcripts with lower hormone concentrations. Further studies are warranted to validate the mechanism of hormone-mediated regulation of HDV antigen expression.