**Introduction:** The hormone relaxin, found in pregnant and non-pregnant females, has been shown to have a collagenolytic effect on ligamentous tissue. Relaxin receptors have recently been identified on human female anterior cruciate ligaments (ACL). This study evaluated whether the administration of recombinant relaxin and estrogen or relaxin alone will lead to a significant increase in ACL laxity in the guinea pig model.

**Materials and Methods:** Guinea pigs were administered 20 ug/hr of recombinant relaxin ± 5 ug/hr of estradiol using separately implanted osmotic pumps. ACL laxity was tested by implanting radio-opaque markers in the femur and tibia of each leg. After applying a standard force (22N) anteriorly translating the tibia, the distance between markers was measured radiographically at day 0 and day 21 compared to controls. The animals were then sacrificed and the ACLs were analyzed for load-to-failure using a material testing machine.

**Results:** Animals treated with relaxin and estrogen (n=4) showed a significant (p=0.02) increase in ACL laxity under an applied force compared to controls (n=4). Animals only treated with relaxin (n=4) also showed a significant (p=0.04) increase in ACL laxity under an applied force compared to controls (n=4). Load-to-failure testing showed hormone treated ACLs (Relaxin + Estrogen μ=32.7 N) (Relaxin only μ= 40.4 N) were significantly weaker than controls (μ=64.1 N) (p=0.000).

**Discussion:** This data suggests that relaxin and estrogen significantly alter the mechanical properties of the ACL in an animal model. The effects of relaxin may contribute to the etiology of female non-contact ACL injuries.