

HORMONE RESPONSES, KNEE JOINT LAXITY AND KNEE JOINT MOMENTS DURING MOVEMENT IN HEALTHY FEMALES

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INTRODUCTION

Hormonal influences on joint laxity and the ACL have been suggested as a strong potential factor which may increase ACL injury risk such as knee joint moment[1]. However, hormone level changes and their contribution to knee joint laxity and knee joint mechanics are known to vary between subjects[2]. Therefore, the objective of this study was to determine whether subsets of women exist who demonstrate or do not demonstrate changes in knee joint laxity in response to circulating hormone levels and their relationship with knee joint moment as a female ACL injury mechanism.

METHODS

Physically active college females (n=26, 22.7±3.3yrs, 170.1±7.1cm, 65.0±9.3kg), who have a normal menstrual cycle (29.22 ± 3.1days) and do not take oral contraceptive agents, were recruited for the study. Serum estradiol, and progesterone levels, right knee joint laxity at a load of 89N (KT-2000, Medmetric Corp., San Diego, CA) and lower extremity kinematics (Motion Analysis Corp., Santa Rosa, CA) and kinetics (Kistler AG, Winterthur, Switzerland) during a jump stop movement were collected three times (follicular, ovulation and luteal phase) during the subjects' menstrual cycles. The first measurements were taken between day 3 and 7 of the menstrual cycle (follicular phase). The second data collection (ovulation) occurred within 24 to 48 hours after estrogen surge based on results from an ovulation predictor kit (Clearblue, Bedford, UK). The third data collection (luteal phase) occurred approximately 7 days later following the peak estrogen surge. All the measurements were performed by one experimenter to eliminate interater differences. Based on a threshold of 0.4mm (determined from repeatability tests of the knee laxity measurement) subjects were divided into responders and non-responders depending on whether changes in knee joint laxity were detected from phase to phase. Changes in hormonal levels and knee joint moments were compared from phase to phase within each group.

RESULTS AND DISCUSSION

14 subjects (responders) demonstrated at least a 0.4mm increase in laxity from the follicular phase to ovulation while 12 subjects (non-responders) showed less than 0.4mm of increased or decreased laxity over the same intervals (Table 1). However, there were no significant differences in hormonal levels between the responders and non-responder subsets; meaning there was no group effect (Table 1). In the relationship between knee joint laxity and knee joint kinetics, responders who increase knee joint laxity from the follicular phase to ovulation showed increased peak knee external rotation and adduction moment (Figure 1). Therefore, findings indicated that the influence of hormones on knee joint laxity was subject specific, possibly explaining why some females are more prone to ACL injury risk than others. Also, we conclude that increased knee joint laxity during the menstrual cycle leads to greater knee joint moments in specific females during selected high risk movements.

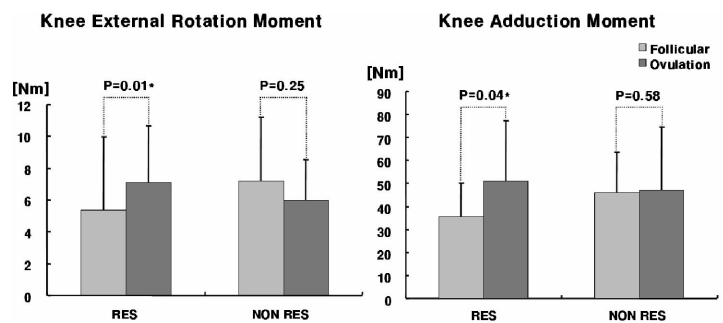


Figure 1: Changes in knee joint moment from the follicular phase to ovulation in each group (*indicates $P < 0.05$)

REFERENCES

1. Park S-K, et al. *Br J Sports Med* Online first, 2008
2. Park S-K, et al. *Am J Sports Med* In press, 2009

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Table 1: Summary of responders (n=14) and non-responders (n=12) from the follicular phase to ovulation

Outcome Measure	Group	Follicular Phase [mean(SD)]	Ovulation [mean(SD)]	Phase Effect F(df) [P]	Group Effect F(df) [P]	Phase by Group Effect F(df) [P]
Laxity at 89N (mm)	Responders	4.42 (1.39)	5.69 (1.53)	7.992(1,24)[0.009*]	0.190(1,24)[0.667]	59.073(1,24)[0.000*]
	Non-responders	5.07 (1.97)	4.48 (1.72)			
Estradiol (pg/ml)	Responders	43.77 (21.44)	78.32 (46.23)	22.697(1,24)[0.000*]	0.204(1,24)[0.749]	0.065(1,24)[0.801]
	Non-responders	45.33 (27.18)	83.79 (34.39)			
Progesterone (ng/ml)	Responders	0.99 (0.52)	3.18 (2.14)	23.579(1,24)[0.000*]	0.023(1,24)[0.881]	0.022(1,24)[0.885]
	Non-responders	1.00 (0.40)	3.32 (2.71)			

(Responders: subjects who increased laxity (greater than 0.4mm) from the follicular phase to ovulation. Non-responders: subjects who did not increase laxity from the follicular phase to ovulation. Two-way repeated measure (one between subject factor of 2 levels, namely-group, and one within subject factor of 2 levels, namely-phase) was conducted at $\alpha=0.05$. Bolded Bold numbers* indicate $P < 0.05$)