A STOCHASTIC BIOMECHANICAL MODEL FOR OF THE RISK AND RISK FACTORS FOR NON-CONTACT ACL INJURY

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INTRODUCTION

Anterior cruciate ligament (ACL) injury is one of the most common injuries in sports. The majority of ACL injuries are non-contact injuries. To prevent non-contact ACL injuries, modifiable risk factors have to be identified. Although tremendous efforts have been made in the last decade to identify risk factors for non-contact ACL injuries, little scientific evidence have been revealed to support any proposed risk factors. The limited progress in research on ACL injury preventions is largely due to limitations in traditional epidemiological research methods that are expensive and labor intensive, and lack ability to establish cause-and-effect relationship between injury and associate factors. Stochastic biomechanical modeling is a method of studying the random outcome of human movements. This method has been successfully applied in many studies on musculoskeletal system injury preventions. The purpose of this study was to develop and validate a stochastic biomechanical model for studying the risk and risk factors of sustaining non-contact ACL injury.

METHODS

An ACL loading model was instrumented to a Monte Carlo simulation to express the risk of sustaining non-contact ACL injury in a stop-jump task as a function of selected sagittal plane and non-sagittal plane biomechanical factors. The total ACL loading at the peak posterior ground reaction force was decomposed as the loadings from sagittal and non-sagittal plane biomechanics. ACL loading from sagittal plane biomechanics was expressed as an inverse dynamic function of knee flexion angle, posterior ground reaction force, tibial tilting angle, the location of center of pressure relative to ankle joint center, and hamstring and gastrocnemius forces. Lower extremity anthropometry data were obtained from literature and normalized to standing height and body weight. ACL loading from non-sagittal plane biomechanics was expressed as a regression function of knee valgus-varus and internal-external rotation moments. The relationships of the ACL loadings with non-sagittal biomechanics were determined using the *in vitro* ACL loading data. The density distribution of each independent variable was determined from the *in vivo* data of 40 male and 40 female college aged recreational athletes in a stop-jump task. A non-contact ACL injury was defined as the peak ACL loading was greater than 2250 N for males and 1800 N for females. Monte Carlo simulations were performed to determine non-contact ACL injury rate for each gender. Each Monte Carlo simulation had 100,000 iterations. Ten Monte Carlo simulations were performed for each gender. Female-to-male non-contact ACL injury rate ratio was determined and the contributions of sagittal and non-sagittal plane biomechanics to ACL injury loading in each simulated injury case were recorded.

RESULTS AND DISCUSSION

The female-to-male non-contact ACL injury rate predicted by the stochastic biomechanical model was 4.96 to 1, which was essentially the same as the injury rate ratio of 4.59 to 1 in the basketball players reported in literature using traditional epidemiological methods. The stop-jump task is frequently performed in the sports of basketball. Non-conntact ACL injuries frequently occur in the stop-jump and similar tasks. The similarity of the predicted and reported relative injury rates supports the validity of the stochastic biomechanical model developed in this study.

The validity of the stochastic biomechanical model developed in this study was also supported by some predicted biomechanical characteristics of non-contact ACL injuries. The predicted knee flexion angle at the peak ACL loading in the simulated injury trials was 22.0 ± 8.0 degrees for male subjects and 24.9 ± 5.6 degrees for female subjects, which were consistent with those reported in literature. The predicted contribution of the sagittal plane biomehanics to ACL injury loading was 1839.86 ± 894.81 N for males and 1773.27 ± 603.83 N for females, which were also consistent with the literature if ACL elevation angle is considered in the literature as this study did.

The mean contribution of sagittal plane biomechanics to ACL injury loading were 1839.86 ± 894.81 N for males and 1773.27 ± 603.83 N for females, respectively. The mean contribution of non-sagittal plane biomechanics to ACL injury loading were 926.83 ± 666.45 N for males and 501.20 ± 388.81 N for females. These results suggest that the contribution of sagittal plane biomechanics to ACL injury loading was 82% of the strength of the ACL for males and 99% for females, while the contribution of non-sagittal plane biomechanics to ACL injury loading was 41% to the strength of the ACL for males and 28% for females. These results indicate that the sagittal plane biomechanics alone can generate sufficient force to injure the ACL while the non-sagittal plane biomechanics alone may not be able to in the stop-jump task.

SUMMARY/CONCLUSIONS

A stochastic biomechanical model has been validated and applied to predict the relative risk and risk factors for the non-contact ACL injury in the stop-jump task. Although most of the non-conntact ACL injuries involve both sagittal and non-sagittal plane biomechanics, sagittal plane biomechanics may play a major role in the risk for non-contact ACL injury in the stop-jump task. Future studies are needed to apply the stochastic biomechanical model to quantify the effects of detailed risk factors on the risk for non-contact ACL injury.

REFERENCES

Agel et al. (2005). Am J Sports Med, 33:524-530.
Markolf et al. (1995). J Orthop Res, 13: 930-935.
McLean et al. (2003). Clin Biomech, 19: 828-838.
Olsen et al. (2004). Am J Sports Med, 32: 1002-1012.
Stapleton et al. (1998). Am J Sports Med, 26: 442-445.