# The relationship between jump load and risk of jumper’s knee in elite men’s volleyball

## Background

Overuse knee problems are common in men’s indoor volleyball.1 They may lead to reduced player availability and impaired performance: Among 75 elite men’s volleyball players, 33% experienced knee problems that moderately or severely reduced training volume or volleyball performance.REFSkazalski2022 Patellar tendinopathy, also known as jumper’s knee, is the most frequent type of knee complaint.2 Jumper’s knee, like most overuse injuries, has a gradual onset without an identifiable inciting event.3 Symptom severity can fluctuate across a season, and players may also experience asymptomatic periods. Jump load—the cumulative long-term exposure to jumping—may increase the risk of jumper’s knee and contribute to such fluctuations.4 5

From biomechanical rationale, consecutive jumping imposes mechanical load on the knee tissue, and in response to pressure and strain, the tissue undergoes a process of repair and adaptation.6 If the jump load exceeds the capacity for the tissue to repair, it may weaken and eventually develop stiffness and pain6; the symptoms of jumper’s knee. Understanding the relationship between jump load and symptomatic jumper’s knee can be an important step in developing prevention interventions.7

Despite this potential, research on how jump load affects jumper’s knee is limited. Time-loss and medical attention injury definitions of previous studies8 9 do not fully capture overuse problems.1 Most studies have assessed the risk of all injuries,5 9 not jumper’s knee specifically, or considered total training load,10 not jump load specifically. General injury and training load definitions might introduce noise to analyses, and it is currently recommended to choose training load measures and injury definitions that are biomechanically tied.11

## Vision

Ideally, volleyball coaches have the knowledge to plan training programs that reduce the risk of symptomatic jumper’s knee, and sports physiotherapists can recommend jump load levels that reduce risk of symptom relapse.

## Aim

Determine the relationship between jump load and symptomatic jumper’s knee in elite men’s volleyball.

## Study design

Prospective cohort

Causal inference (not prediction)

## Research questions

**Main questions:**

1. How much does jump height and jump frequency affect the risk of receiving symptomatic jumper’s knee, respectively?
2. Does relative change in jump height or jump frequency affect the risk of symptomatic jumper’s knee?
3. How much of the effect of jump height and jump frequency can be explained by relative jump height and jump frequency?

**Secondary questions**

1. How does the effects of jump height and jump frequency change depending on distance in time since they were performed/sustained?
2. Does match congestion affect the risk of increased jumper’s knee symptoms?

Congestion = periods of frequent volleyball and jump load exposure followed by periods of low exposure.

1. Is there an increase of pain occurrence in the pre-season, even after adjusting for the amount of jump load?  
   This will indicate whether the pre-season itself has characteristics which increases risk, or whether previous findings suggesting an increased risk is only due to mediation from the time in exposure, as it is always higher in the pre-season.

## Variables needed

Player ID  
Team ID  
Date of activity  
Competition system / match week type (if registered; otherwise, can be extrapolated from team ID)  
Preseason (yes/no)  
Match (yes/no)  
Match contribution (%)  
Age (ideally date of birth)  
Playing position  
Height  
Weight  
Jump frequency  
Jump height  
Jump max height  
Jump % of max height  
OSTRC-O response

## Analysis

We will consider this from a time-to-event perspective. We can then take into account that some participants are more “robust” than others, and time is inherently adjusted for.

We will use Cox regression with a frailty term on individual. Jump intensity and jump frequency will be modeled with a distributed lag non-linear model (DLNM) to account for past jump load.12

Jump intensity will be measured with the jump height, and jump frequency with the number of jumps.

Based on the assumptions of the causal pathways visualized in the DAG, there are several potential strategies for approaching unbiased estimates of the effect of jump load on the risk of jumper’s knee. We choose adjustment in regression modelling to block backdoor pathways if possible, and stratification only if necessary. If we can choose between a single covariate over multiple covariates to block a backdoor pathway, we will prioritize that to improve statistical power. However, we will also prioritize constructs with intuitive measures of low measurement errors over constructs that are multidimensional or have high degrees of uncertainty in the measurement.

Our list of potential covariates are therefore age, fitness, past injury, past jump load, playing position, match yes/no, time since previous match, weight.

Note that weight is only a potential deconfounder for jump height (jump intensity), not jump frequency. Time since previous match and past jump load, is only a potential deconfounder to jump frequency, not jump intensity. However, we will assess the effects of past jump load as part of the exposure of interest.

Also note that wellness and past injury are not sufficiently measured, but the backdoor pathways through these constructs could not be blocked through use of other measures. The implications of these missing variables must be considered and discussed.

### Model summary

In total, six Frailty models. The outcome is time to jumper’s knee symptoms (yes/no). The exposure approaching an unbiased estimate (given the assumptions of the DAG holds true) is in bold.

Models 1 and 2 answers research question 1 + 4.

1. **Jump height**, age, weight, playing position, match (yes/no), max jump height (fitness) + frailty term
2. **Jump frequency**, age, max jump height (fitness), playing position, match (yes/no), n days since previous match + frailty term

Model 3 answers research question 2.

1. jump height, jump frequency, **relative jump frequency, relative jump intensity** + frailty term

Model 4 and 5 answers research question 3.

1. **Jump frequency**, **relative jump frequency**, age, max jump height (fitness), playing position, micro-cycle day (match, training, recovery), n days since previous match + frailty term
2. **Jump height**, **relative jump height**, age, weight, playing position, micro-cycle day (match, training, recovery), max jump height (fitness) + frailty term

Models 6 and 7 answers research question 6. The change in effects size between model 6 and 7 should show whether the effect of pre-season is mostly mediated through the jump load variables, or if there is an effect from pre-season outside of this meditation. That could be through relative jump load or past jump load (unmeasured).

1. **Pre-season** + frailty term
2. **Pre-season**, jump height, jump frequency + frailty term

Model 8 answers research question 5.

1. **Time since previous match**, match week type (2,3,4 or 5 matches per week) + frailty term

### Details on model choice

#### Main jump load exposures

**Jump frequency.** The number of jumps per day.

**Jump intensity.** The height of each jump is a measure of intensity. All jump heights will be summed per day.

**Age, height, weight, playing position, pre-season (yes/no), training program, match (yes/no)**. Will be adjusted for by including the variables in the model.

**Past injury (previous symptoms)**. Will be adjusted for with a frailty term.13 Might have to use sequential stratification14 to avoid collider stratification bias15 if we want to look at this more closely.

**Previous jump load**. Will be adjusted for by using DLNM.

**Fitness.16** To adjust for fitness, we will include the max jump height as a covariate. If necessary, we will use the % of jump height instead of the absolute jump height for a more statistically efficient model. However, with that method, we can’t separate the effect of jump height from the effect of fitness (which has causal pathways to jumper’s knee outside of jump height).  
  
**Wellness.** To adjust for wellness, we may include the % match contribution, which describes whether they were a substitute or not. If they are a substitute, they are likely to not be as ready for the match. This is the only measure of wellness we have, so we’ll have to discuss this in the paper. We think this effect will be very small, so if we have problems with overfitting, statistical power etc. then we will remove this first.

#### Secondary jump load exposures

**Relative jump load.** To answer how much of the effect of jump load can be explained by relative jump load (mediation), we need to have at least 2 models. One where we have jump height and number of jumps, adjusted for all the confounders. This will show the total effect of these two external load measures. We will then have another model where, in addition to the original model specification, relative jump height and relative jump frequency is added in. When (if) the coefficients for jump height and number of jumps change, that change tells us how much of the effect is explained by the respective relative jump load variables. We will initially look at only relative jump frequency, and depending on time, we can also look at relative jump height.

Q: How to calculate? In the literature, 1 week vs. 3 to 4 weeks is used. Is this arbitrary for volleyball? 1 day to previous day too granular? Micro-cycle to micro-cycle relative? This is still unclear. Will consider and discuss.

**Match congestion.** Measured by time since previous match. We assume this affects the global fatigue of an athlete.

**Pre-season.** We will have one model unadjusted for jump load, showing the association with pre-season. We will then have one with the absolute jump load variables to determine whether pre-season still has an increase in risk. This will show whether there is still an association between pre-season training and jumper’s knee, or whether the causation is through mediation through jump load. If the result is that there is no increased risk after adjustment, then that’s a sign that the increased risk seen during the pre-season is simply because they do more jumping in the pre-season.

According to the DAG, to determine an unbiased estimate of pre-season’s effect of jumper’s knee, no adjustment is required (other than frailty term). Then, all adjustment is required to see how much of the pathway is through jump load and jump intensity.

### Assumptions and pitfalls

**Overfitting/Power.** We will model jump frequency and jump height in separate models. They are not involved in the causal pathways of each other, and according to the 6-step process performed on the DAG, this can be done safely. In addition, jump frequency does not to adjust for weight for an unbiased estimate (given the DAG) so that model can be a bit more efficient.   
  
If we have problems of overfitting, %-match contribution will be the first to go. Might have to look at just relative frequency and not relative jump height to answer research question 2 and 3. For jump intensity, we can save power by adjusting for BMI instead of height and weight separately, but only if we have to. Another option is the % of max jump height in stead of the absolute jump height for studying jump intensity.

UPDATE: We ended up creating a compound jump load measure instead of looking at jump frequency and height separately.

**Non-linearity** Jump load performed further back in time might have a different effect than current jump load. Non-linearity will be assessed using restricted cubic splines (aka natural splines) in the DLNM crossfit.17

**Missing data** Imputed with multiple imputation.18 Assumed to be MAR, as the company improved the VERT devices over the years. More missing will be in the earlier years/seasons than in later years/seasons. This variable is not in any of our regression models. We can therefore include this variable in the imputation model.

**Autoregression.** Dealt with using a frailty term.

**Proportional hazard.** If violated, an alternative is the Royston-Parmar model.19

**Multicollinearity.** We can struggle with the jump load variables being correlated with each other. Will check using Variance Inflation Factor (VIF) or other criteria. The biggest concern is relative jump frequency vs. absolute jump frequency. The worst case scenario is we won’t able to answer research questions 2 and 3 with any certainty. UPDATE: We ended up creating a compound jump load measure.

**Overly influential outliers.** Checked with DFbeta and set to be the highest level in the natural range of the variable if any are detected.20 Any value above 120 cm in jump height is considered an error (Skazalski et al. 2018).

## Limitations

We have no measures on internal training load – neither psychological nor physiological. This means we cannot determine how much of the effect of jump height and jump frequency is through mechanical load, physiological load, and psychological load, respectively. We do suspect that mechanical load plays the biggest role, because the pressure that stems from the landing is most likely what causes tissue changes (not heart rate etc. that comes from the act of jumping).

We don’t have a concrete measure for wellness, which is the only confounder left after model adjustment. Since the backdoor pathways of wellness are through physiological load and psychological load, we believe this will have little impact on the model.

We don’t have total injury surveillance. Other injuries, such as ankle sprains, are competing events. Implication: If we find an effect of jump load, the real effect may be (even) larger, as competing events are natural breaks towards developing further jump load strain/microdamage.

The questionnaire captures all knee complaints, and ca. 10% may not be jumper’s knee, but other complaints. Implication: Likely to be small, as jump load affects these other injuries as well, but may affect them a bit differently. I.e. maybe height and frequency is more or less important for these injuries than they are for jumper’s knee, respectively. May add some uncertainty (broader CIs) to the estimates of jump height and jump frequency.  
  
Each team from the different countries belong to different competition systems. The number of matches in these systems differ, from 2 to 5 matches per week. We cannot separate the effect of competition system from country, if there are any population effects.

We do not have information of previous jumper’s knee symptoms at baseline. We can only adjust for this during the course of the longitudinal study, but we thankfully have data collection spanning several years. Correction: 1 team collected baseline injury data.

Coaches and support staff were instructed to collect weekly OSTRC questionnaires. Sometimes these were collected late, and it is unclear whether the OSTRC responses then pertain to the previous week (which it was supposed to), or the last 7 days, meaning that the responses could overlap with the next OSTRC.

# Changes during analyses work

Due to interval-censored data, we could not do everything that was initially planned. We could not look at match congestion. We could not use Cox regression. We also could not adjust for match or time since previous match.

Due to negative findings, we didn’t go into more detail with relative training load. We also did not separate our compound jump load measure at any point, since there was too much uncertainty to look at the jump load measures individually.

# References

1. Clarsen B, Bahr R, Heymans MW, et al. The prevalence and impact of overuse injuries in five Norwegian sports: Application of a new surveillance method. *Scandinavian Journal of Medicine & Science in Sports* 2015;25(3):323-30. doi: <https://doi.org/10.1111/sms.12223>

2. Lian ØB, Engebretsen L, Bahr R. Prevalence of Jumper's Knee among Elite Athletes from Different Sports: A Cross-sectional Study. *The American Journal of Sports Medicine* 2005;33(4):561-67. doi: 10.1177/0363546504270454

3. Bahr R, Krosshaug T. Understanding injury mechanisms: a key component of preventing injuries in sport. *British journal of sports medicine* 2005;39(6):324-29.

4. Bahr MA, Bahr R. Jump frequency may contribute to risk of jumper's knee: a study of interindividual and sex differences in a total of 11 943 jumps video recorded during training and matches in young elite volleyball players. *British Journal of Sports Medicine* 2014;48(17):1322-26. doi: 10.1136/bjsports-2014-093593

5. de Leeuw A-W, van der Zwaard S, van Baar R, et al. Personalized Machine Learning Approach to Injury Monitoring in Elite Volleyball Players. *European Journal of Sport Science* 2021:1-14.

6. Verheul J, Nedergaard NJ, Vanrenterghem J, et al. Measuring biomechanical loads in team sports – from lab to field. *Science and Medicine in Football* 2020;4(3):246-52. doi: 10.1080/24733938.2019.1709654

7. Moran LR, Hegedus EJ, Bleakley CM, et al. Jump load: capturing the next great injury analytic. *British Journal of Sports Medicine* 2019;53(1):8-9. doi: 10.1136/bjsports-2018-099103

8. Taylor JB, Barnes HC, Gombatto SP, et al. Quantifying External Load and Injury Occurrence in Women's Collegiate Volleyball Players Across a Competitive Season. *J Strength Cond Res* 2022;36(3):805-12. doi: 10.1519/jsc.0000000000004212 [published Online First: 2022/02/19]

9. Timoteo TF, Debien PB, Miloski B, et al. Influence of Workload and Recovery on Injuries in Elite Male Volleyball Players. *Journal of strength and conditioning research* 2018

10. Visnes H, Bahr R. Training volume and body composition as risk factors for developing jumper's knee among young elite volleyball players. *Scandinavian journal of medicine & science in sports* 2013;23(5):607-13.

11. Kalkhoven JT, Watsford ML, Coutts AJ, et al. Training load and injury: causal pathways and future directions. *Sports Medicine* 2021:1-14.

12. Gasparrini A. Modeling exposure–lag–response associations with distributed lag non‐linear models. *Statistics in medicine* 2014;33(5):881-99.

13. Ullah S, Gabbett TJ, Finch CF. Statistical modelling for recurrent events: an application to sports injuries. *Br J Sports Med* 2014;48(17):1287-93.

14. Gran JM, Røysland K, Wolbers M, et al. A sequential Cox approach for estimating the causal effect of treatment in the presence of time‐dependent confounding applied to data from the Swiss HIV Cohort Study. *Statistics in medicine* 2010;29(26):2757-68.

15. Shrier I, Stovitz S, Wang C, et al. Methods Matter: Beware of collider stratification bias when analyzing recurrent injuries. 2021

16. Visnes H, Aandahl HÅ, Bahr R. Jumper's knee paradox—jumping ability is a risk factor for developing jumper's knee: a 5-year prospective study. *British Journal of Sports Medicine* 2013;47(8):503-07. doi: 10.1136/bjsports-2012-091385

17. Bache-Mathiesen LK, Andersen TE, Dalen-Lorentsen T, et al. Not straightforward: modelling non-linearity in training load and injury research. *BMJ Open Sport & Exercise Medicine* 2021;7(3):e001119. doi: 10.1136/bmjsem-2021-001119

18. Bache-Mathiesen LK, Andersen TE, Clarsen B, et al. Handling and reporting missing data in training load and injury risk research. *Science and Medicine in Football* 2021:null-null. doi: 10.1080/24733938.2021.1998587

19. Royston P, Parmar MKB. Restricted mean survival time: an alternative to the hazard ratio for the design and analysis of randomized trials with a time-to-event outcome. *BMC Medical Research Methodology* 2013;13(1):152. doi: 10.1186/1471-2288-13-152

20. Harrell FE. Regression modeling strategies. *BIOS* 2017;330:2018.