Patellar tendinopathy

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Publication date:
2012

Chapter 3

Is the jumper's knee a lander's knee? A systematic review of the relation between take-off and landing biomechanics and patellar tendinopathy

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Abstract

Patellar tendinopathy (jumper's knee) is a common injury in sports that comprise jump actions. This article systematically reviews the literature that examines the relation between patellar tendinopathy and take-off and landing biomechanics, in order to uncover risk factors and potential prevention strategies. A systematic search of the Pubmed, Embase and Amed databases was conducted, and nine articles that met the inclusion criteria were identified. The identified studies were diverse in methods used, jump actions studied and in populations. A synthesis of the literature suggests that a flexible movement pattern during jumping reduces the risk for patellar tendinopathy and that patellar tendinopathy is related to landing more so than take-off. Accordingly, employing a flexible movement pattern, especially during landing, seems an expedient strategy to reduce the risk for developing/redeveloping patellar tendinopathy. Together, these findings indicate that improving kinetic chain functioning, performing eccentric exercises and changing landing patterns are potential tools for preventive and/or therapeutic purposes.

Introduction

Patellar tendinopathy (PT), also known as jumper's knee,[1] is a common injury in sports that involve repetitive jumping like basketball and volleyball. Among elite and recreational basketball players the prevalence is 32% and 12% respectively, and among elite and recreational volleyball players 45% and 14% respectively.[2, 3] Prevention of this injury is important because symptoms can last for years, can affect sports and work participation, and can even be a reason to end a sports career.[4, 5] Although several treatments have been described, treatment results are variable.[6] Knowledge of risk factors is necessary in order to develop preventive measures.[7] Many risk factors have been suggested in the literature and it appears that PT has a multifactorial etiology. Suggested factors for which there is most evidence that they play a role in the onset of PT are weight, body mass index, waist-to-hip ratio, leg-length difference, arch height of the foot, quadriceps flexibility, hamstring flexibility, quadriceps strength, vertical jump performance and training volume.[8, 9] The high prevalence of PT in sports that involve jump actions suggests that PT is caused by jumping – that is, by take-off and/or landing. Hence to understand the etiology of PT one must at least understand the relation between PT and take-off and landing. Indeed, a number of biomechanical studies have investigated how take-off and landing may be related to PT. However, as we will see in the current review, these studies are diverse in their adopted research methods, jump actions studied, and populations. Furthermore, the causality in the relation between jump biomechanics and PT is often ambiguous. The aim of this systematic review is to come to a better understanding of how PT may be related to take-off and landing biomechanics. Studying both jump phases may provide more insight into the development of PT, and also addresses the question of whether take-off and landing pose an equal risk for developing PT. In this way risk factors may be uncovered which can be used to identify take-off and/or landing patterns which predispose athletes for developing PT. Potential means for prevention of PT can be subsequently developed through, for example, adaptation/training of these patterns.

Methods

Search strategy and inclusion criteria

A computerised search of the Pubmed, Embase and Amed databases was conducted in October 2011. The following terms were used: *patella(r) tendon, jumpers knee, jumper's knee, patella(r) tendinopathy, patella(r) tendinosis, patella(r) tendinitis, patella(r) tendonitis, patella(r) apicitis, patella(r) apex syndrome, patella(r)*

Search terms:

patella(r) tendon, jumpers knee, jumper's knee, patella(r) tendinopathy, patella(r) tendinosis, patella(r) tendinitis, patella(r) tendonitis, patella(r) apicitis, patella(r) apex syndrome, patella(r) tip syndrome, patella(r) tenosynovitis AND jump, jumping, land, landing, take off, touchdown

	Pubmed	Amed	Embase
	↓	↓	↓
Studies identified	73	11	42
	↓		
Total studies identified	126		
	↓		
After checking for duplicates	97		
	↓		
After excluding reviews/letters/abstracts	80		
	↓		
After reading articles	8		
	↓		
After reference checking	9		

Figure 1. Literature search

tip syndrome, patella(r) tenosynovitis combined with *jump, jumping, land, landing, take off, touchdown* and plural forms. The search was restricted to articles in English. Reference lists of the included studies as well as other relevant studies were checked for additional references. Studies were included if they met the following three criteria: 1) it was an empirical study that investigated jump and landing characteristics of real jumps in relation to PT; 2) kinematics, kinetics or energetics of these jumps were collected; and 3) a comparison was made in that study between a control group and a group with (a)symptomatic PT. Titles and abstracts were screened independently by two authors to determine inclusion or exclusion. If it was not clear whether the study should be included, the full text was screened.

Data extraction

Data on study population, investigated factors and jump tasks were extracted and summarised from the included studies. Investigated factors were categorised into *kinematics*, *kinetics* and *energetics*.

Table 1. Characteristics of included studies

Author (year)	Factors	Analysed jump phase	Jump action	Population	N	Groups (N)
Lafortune (1985)	Kinematics (Hip, Knee)	TL	Vertical jump	Male college basketball players	7	control (5) previous PT (asymptomatic) (2)
Richards et al. (1996)	Kinematics and kinetics (Knee)	TL	Block jump and spike jump	Men of the Canadian National volleyball team	10	control (7) symptomatic PT (3)
Richards et al. (2002)	Kinematics and kinetics (Ankle)	TL	Spike jump	Men of the Canadian National volleyball team	10	control (7) symptomatic PT (3)
Bisseling et al. (2007)	Kinematics, kinetics and energetics (Hip, Knee, Ankle)	L	Drop jump landings from 30, 50 and 70 cm height	Male elite volleyball players	24	control (8) previous PT (asymptomatic) (7) symptomatic PT (9)
Bisseling et al. (2008)	Kinematics, kinetics and energetics (Knee, Ankle)	TL	Spike jump	Male elite volleyball players	15	control (8) previous PT (asymptomatic) (7)
Siegmund et al. (2008)	Kinematics (Hip, Knee, Ankle)	TL	Standing countermovement jumps, running layup jumps	Male basketball players, elite and recreational	24	control (12) symptomatic PT (12)
Edwards et al. (2010)	Kinematics, kinetics and EMG (Hip, Knee, Ankle)	L	Stop jump	Male athletes from team sports involving repetitive landing	23	controls (16) PTA, no previous or current symptoms (7)
Sorenson et al. (2010)	Kinematics, kinetics and energetics (Knee)	T	Spike jump	Male elite volleyball players	13	controls (7) PT without self-reported activity limitations (6)
Souza et al. (2010)	Kinetics (Hip, Knee, Ankle)	TL	Hopping	Male elite volleyball players	14	controls (7) PT without self-reported activity limitations (7)

L = landing phase; T = take-off phase

Results

Results of literature search

Nine articles that investigated the relation between patellar tendinopathy and jumping biomechanics were included in the review (figure 1). The literature search yielded 8 articles,[10-17] and one additional study was found after reference-checking.[18]

Description of studies

Characteristics of the included studies are shown in Table 1. Five studies compared subjects with clinically diagnosed symptomatic PT with a control group.[10, 11, 14, 16, 17] The study by Bisseling et al. (2007) compared a group of controls with an asymptomatic group with previous PT and a symptomatic group with current PT.[12] The same authors conducted another study with only the first two groups.[13] This last comparison was also made in another study.[18] Edwards et al. included subjects without present or previous symptoms, but with patellar tendon ultrasonographic abnormality (PTA), and compared them with subjects without ultrasonographic abnormalities.[15] The presence of PTA increases the likelihood of the onset of PT.[19-21] Studying a group with PTA provides an opportunity to study subjects without symptoms but with a high risk for developing PT. In the remainder of this article, the term 'asymptomatic' refers to both subjects with PTA and subjects with previous PT.

Jump tasks investigated were a vertical jump task,[18] the volleyball spike jump,[10, 11, 13, 16] the volleyball block jump,[10] a drop jump,[12] a standing countermovement jump,[14] a running layup jump,[14] a stop jump task,[15] and hopping.[17] Six of the nine studies looked at take-off and landing,[10, 11, 13, 14, 17, 18] two only analysed the landing,[12, 15] and one study solely examined the take-off.[16] So both jump phases are represented fairly evenly in the included studies.

Methodological quality of the studies

No prospective studies were found during the search; all included studies had a cross-sectional design. Drawing conclusions about causality is therefore impossible. Three studies reported that the subject characteristics (e.g. age, height, weight) of the control group and the (a)symptomatic group were comparable,[15-17] one study reported that groups were matched (on height, weight, position, experience and frequency of play) but did not support this with statistical testing,[14] and the five remaining studies made no statements about the comparability of groups.

Table 2. Differences in jump kinematics between controls and other study groups

	Variable	Reference	Analysed jump phase	Task	Groups (compared to controls) PT-S	PT-P	PTA
Ankle	Ankle flexion at maximal VGRF	[12]	L	Drop jump	<		
	Ankle flexion at TD	[13]	L	Spike jump		<	
	Maximum dorsiflexion angle	[14]	L	Layup jump	<		
	Ankle inversion at maximal PTF	[15]	L	Stop-jump vertical phase			>
Knee	Knee ROM	[18]	L	Vertical jump		<	
	Knee ROM	[10]	L	Spike jump	>		
	Knee ROM	[13]	L	Spike jump		<	
	Knee ROM	[15]	L	Stop-jump horizontal phase			<
	Knee flexion at TD	[15]	L	Stop-jump horizontal phase			>
	Knee flexion velocity at TD	[15]	L	Stop-jump horizontal phase			<
	Knee angular velocity	[12]	L	Drop jump		>[1]	
	Maximum knee flexion acceleration	[14]	L	Standing counter movement jump	<		
	Time to maximum knee flexion	[14]	T	Standing countermovement jump	>		
	Internal knee rotation	[15]	L	Stop-jump horizontal phase			>
Hip	Hip ROM	[18]	L	Vertical jump		<	
	Hip extension velocity at TD	[15]	L	Stop-jump horizontal phase			>
	Hip abduction at maximal VGRF	[15]	L	Stop-jump horizontal phase			>
	Hip external rotation velocity at maximal patellar tendon force	[15]	L	Stop-jump horizontal phase			>
	Hip flexion velocity at maximal VGRF	[15]	L	Stop-jump vertical phase			>
	Maximum hip flexion angle	[14]	T	Standing countermovement jump	>		
	Maximum hip flexion acceleration	[14]	T	Standing countermovement jump	<		
Interaction	Between knee and ankle ROM	[13]	L	Spike jump		<	
	Between knee angular velocity during eccentric phase of take-off and landing	[13]	TL	Spike jump		>	

1 = compared to PT symptomatic; > = a higher value than controls; < = a lower value than controls; **L** = landing phase; **PTA** = patellar tendon ultrasonographic abnormalities; **PTF** = patellar tendon force; **PT-P** = previous patellar tendinopathy; **PT-S** = symptomatic patellar tendinopathy; **ROM** = range of motion; **T** = take-off phase; **TD** = touchdown; **VGRF** = vertical ground reaction force

Table 3. Differences in jump kinetics between controls and other study groups.

	Variable	Reference	Analysed jump phase	Task	Groups (compared to controls)		
					PT-S	PT-P	PTA
General	Maximal VGRF	[10]	T	Spike jump	>		
		[16]	T	Spike jump	<		
	Maximal VGRF	[10]	T	Block jump	>		
	Loading rate VGRF	[10]	T	Spike jump	>		
	Loading rate VGRF	[12]	L	Drop jump		>	
	Loading rate VGRF	[15]	L	Stop-jump vertical phase			<
Ankle	Right foot inversion moment (in right knee PT)	[11]	L	Spike jump	>		
	Loading rate ankle moment	[12]	L	Drop jump	<	>	
Knee	Peak loading rate knee extensor moment	[10]	L	Spike jump	>		
	Peak tibial external rotation moment	[10]	T	Spike jump	>		
	Peak tibial external rotation moment	[10]	T	Block jump	>		
	Peak knee moment	[12]	L	Drop jump	<		
	Loading rate knee moment	[12]	L	Drop jump		>[1]	
	Loading rate knee moment (eccentric phase)	[13]	T	Spike jump		>	
	Knee contribution to total support moment	[17]	TL	Hopping	<		
Hip	Hip contribution to total support moment	[17]	TL	Hopping	>		

1 loading rate was significantly higher than the symptomatic group, with trend towards being significantly higher than the control group
> = a higher value than controls; < = a lower value than controls; **L** = landing phase; **PT** = patellar tendinopathy; **PTA** = patellar tendon ultrasonographic abnormalities; **PT-P** = previous patellar tendinopathy; **PT-S** = symptomatic patellar tendinopathy; **ROM** = range of motion; **T** = take-off phase; **VGRF** = vertical ground reaction force

Table 4. Differences in jump energetics between controls and other study groups.

Variable	Reference	Analysed jump phase	Task	Groups (compared to controls)		
				PT-S	PT-P	PTA
Peak knee power	[12]	L	Drop jump	<		
Knee joint work	[12]	L	Drop jump	<		
Knee net joint work (eccentric phase)	[16]	T	Spike jump	<		
Positive-to-negative knee net joint work ratio	[16]	T	Spike jump	>		
Average knee net joint power (eccentric phase)	[16]	T	Spike jump	<		
Positive-to-negative average knee net joint power	[16]	T	Spike jump	>		

> = a higher value than controls; < = a lower value than controls; **L** = landing phase; **PTA** = patellar tendon ultrasonographic abnormalities; **PT-P** = previous patellar tendinopathy; **PT-S** = symptomatic patellar tendinopathy; **T** = take-off phase

For these five studies it is possible that differences between study groups are not the result of the presence or absence of PT, but are related to other variables that differed between groups.

Jump biomechanics

Differences between groups for take-off and landing were found for a number of kinematic (Table 2), kinetic (Table 3) and energetic (Table 4) variables. Although take-off and landing were investigated by almost the same number of studies (7 vs. 8), differences between groups, which are possible risk factors, were found more often for the landing (see Tables 2-4).

Kinematics

Take-off

Symptomatic subjects showed greater maximal hip flexion, lower angular accelerations for hip and knee flexion, and a longer time to maximal knee flexion compared to controls.[14]

Landing

Symptomatic subjects showed more range of motion (ROM),[10] and lower angular velocities for the knee during landing.[14] It was also found that symptomatic subjects have a smaller maximal ankle dorsiflexion angle than controls.[14] Compared to controls, subjects with previous PT and PTA showed smaller ankle plantar flexion at touchdown (TD) and larger knee angle at TD,[13, 15] smaller ROM for knee and hip,[13, 15, 18] and greater angular velocities in the hip.[15] They also showed differences for kinematics in the frontal and axial planes, such as more ankle inversion, internal knee rotation and hip abduction and greater hip external rotation velocity.[15]

Differences between groups

Symptomatic subjects generally showed a flexible movement pattern with more joint flexion and took more time for their take-off and landings, except for the finding that they have a smaller maximal ankle dorsiflexion angle than controls during landing. Subjects with asymptomatic PT, on the other hand, showed a stiff jump and landing pattern, opposite to that of symptomatic PT subjects – that is, with less joint flexion and higher velocities during take-off and landing. They also showed more translation in the frontal and axial planes.

Kinetics

Take-off

One study found a higher maximal vertical ground reaction force (VGRF) and loading rate of the VGRF for symptomatic subjects compared to controls for the spike jump take-off and only a higher vertical ground reaction force (VGRF) for the block jump take-off,[10] whereas another found that the maximal VGRF was lower for the spike jump take-off in symptomatic subjects compared to controls.[16] The loading rate of the knee moment during spike jump take-off was higher in subjects with previous PT compared to controls.[13] Richards et al. (1996) showed that the tibial external rotation moment was higher in symptomatic subjects compared to controls during both block jump and spike jump take-off.[10]

Landing

The inversion moment of the foot was higher in symptomatic subjects compared to controls.[10,11] For moments in the sagittal plane it was found that the peak knee moment was lower in symptomatic subjects than in controls (drop jump landing),[12] whereas the peak loading rate of the knee moment was higher (spike jump landing),[10] and the knee contributed less to the total support moment.[17] Subjects with previous PT showed higher loading rates of VGRF and knee and ankle moments than controls,[12] whereas the only study that compared subjects with PTA with controls found that the loading rate of the VGRF was lower in the first group.[15]

Differences between groups

The observed differences in kinetics between groups were inconsistent across studies. The only clear difference was that for moments in the transverse plane which were higher in symptomatic subjects than in controls.

Energetics

Take-off

Differences in energetics were found between controls and symptomatic subjects during spike jump take-off, with symptomatic subjects showing less knee net joint work and average knee net joint power than controls.[16] Symptomatic subjects also showed a higher positive-to-negative ratio of knee net joint work and knee net joint power during this action.[16]

Landing

During landing of a drop jump symptomatic subjects also had lower knee power and knee joint work than controls.[12]

Differences between groups

Overall, symptomatic subjects generated less energy than controls, especially during eccentric movements in take-off and landing.

Discussion

The aim of this systematic review was to come to a better understanding of how PT may be related to take-off and landing biomechanics. The review revealed a number of differences in kinematics, kinetics and energetics between subjects with (a)symptomatic PT and controls. Some of these differences may play a role in the onset of PT, whereas others may be the result of PT. As no prospective studies are available in the literature it is impossible to discern causes and effects. These results as currently available from literature do not allow for firm conclusions regarding causality in the relation between these differences in jump biomechanics and PT. Furthermore, because of the heterogeneity of the studies the results were difficult to cluster, hence a direct comparison between studies is unfeasible. This heterogeneity resided in the diversity of the adopted methods, the employed jump actions and the studied populations.

The joints together with the bones, muscles and tendons form a kinetic chain. During take-off this kinetic chain acts to overcome gravity in order to propel the body into the air, while during landing this kinetic chain acts to dissipate kinetic energy, by muscles and tendons, to withstand collapsing. Both take-off and landing are achieved by interplay between the components of the kinetic chain, and the role of each component can vary. For example, a stiff landing will put more load on the skeletal system, whereas a flexible landing (with greater flexion angles) will put more load on the muscles.[23] As we will argue, kinetic chain function plays an important role in developing PT.

Based on the kinematics of subjects with symptomatic PT it can be hypothesised that they used a tendon load-avoiding movement pattern to minimise pain, a pattern characterised by greater maximal flexion, greater ROM and lower velocities.[12] Devita and Skelly (1992) found that a flexible (soft) landing pattern, with large maximum knee flexion angles, led to an increased absorption of kinetic energy by the muscles, thereby putting less stress on the other tissues.[23] Compared to subjects with symptomatic PT, subjects with previous PT and PTA generally showed a less flexible (i.e. stiff (Devita & Skelly 1992)) jumping and landing pattern with smaller ROM and greater angular velocities. Such kinematics indicate that subjects with previous PT likely revert to a pattern that caused their PT in the past now that the pain has disappeared. Because PTA is thought to be a precursor of symptomatic PT,[19-21] subjects with PTA constitute a high-risk population for developing PT and are likely to show riskful jump biomechanics. Hence despite the absence of prospective studies, from studies examining asymptomatic groups (i.e. subjects with previous PT and subjects with PTA) we may hypothesise that whereas the symptomatic group used a flexible movement pattern to minimize pain, subjects with previous PT and PTA show a stiff pattern that puts them at risk for developing or re-developing PT. The results regarding the kinetics could be interpreted less straightforwardly than the kinematics. Subjects with previous PT showed higher loading rates than controls,[12, 13] while this was not the case for subjects with PTA.[15] Studies that measured the VGRF of controls and symptomatic subjects came to contradictory results.[10, 16] In general, symptomatic subjects showed larger joint moments in the transverse plane.[10, 11] They also seemed to shift the load away from the knee.[17] The reviewed results regarding the energetics showed that symptomatic subjects generated less knee joint power and work,[12, 16] which is also in line with a pain-minimising strategy. Also, symptomatic subjects may simply be unable to generate more power with the knee because of degenerative changes in the tendon structure. However, this is unlikely because if this were the case a loss of power would also have been expected for subjects of the PTA group, who also show degenerative changes. It is also known that the fibril morphology is abnormal in tendinopathy but the mechanical properties of the tendon aren't.[24] Anyway, the symptomatic subjects examined in the reviewed studies showed a movement pattern (probably due to the associated pain) that is most likely a result of PT and not a cause of it.

Take-off vs. landing

According to one of the main pathophysiological theories about tendinopathy, micro-injuries in the tendon resulting from repeated overload can eventually lead to matrix and cell changes as well as altered mechanical properties of the tendon.[25] Especially eccentric loads are thought to produce micro-injuries because these can be much higher than concentric loads.[26] In contrast with this idea, a study that measured peak patellar tendon torques during (concentric) take-off and (eccentric) landing of a maximal vertical jump found no differences in peak torque between the two phases in healthy subjects.[27] The present review does indicate that differences between groups were found more frequently for the landing phase than the take-off phase, which suggests that subjects who are unable to cope with these peak eccentric patellar tendon torques during landing may be more prone to developing PT. When landing is concerned, there is a difference between landing from a vertical jump, where only vertical deceleration has to be achieved, and landing from a forward jump, where also horizontal deceleration must be achieved. One study that compared the horizontal landing phase (after forward acceleration) and the vertical landing phase during a stop-jump task found that the control group and the PTA group differed more during the horizontal landing phase.[15] Together with the finding that the peak force in the patellar tendon is higher during the horizontal landing phase than the vertical landing phase,[22] this suggests that the horizontal phase may play an important role in the onset of PT. This may also explain why prevalence of PT is highest in volleyball players,[2, 3] because although similar movements are performed in sports like basketball and soccer, the volleyball net forces players to reduce the horizontal velocity to zero during the horizontal landing phase, leading to high loads exerted on the patellar tendon.

Clinical relevance

The present review suggests that risk factors for developing PT are in general 1) flexion angles (small ankle plantar flexion angle, large knee flexion angle) at touchdown that reduce the available ROM, 2) small post-touchdown ROM in the joints, and 3) high post-touchdown joint angular velocities. The landing of a jump also appears to pose a greater threat for developing PT compared to the take-off, a threat that is especially high during horizontal landing after a forward acceleration. This may be relevant for the prevention of this injury, since it suggests that employing a more flexible jumping and landing pattern may reduce the risk of developing PT. This may be achieved in two ways. First, it has been shown that reduced flexibility of the kinetic chain, such as that of the upper leg muscles and reduced dorsiflexion range,[28-30] are related to tendinopathy. For this reason, optimising kinetic chain function (by addressing strength, flexibility and joint function) – one of the main elements of a patellar tendon rehabilitation program according to Kountouris and Cook (2007)[31] – may also be valuable towards preventing patellar tendinopathy. Second, changing stiff landing patterns towards more flexible ones is another preventive option. Indeed, it has been shown that it is possible to modify jump technique by verbal instruction or videotape feedback.[32,33] Before applying such interventions, obviously 'riskful' take-off and landing patterns will have to be detected first. A first step may be to investigate whether experts like trainers and coaches are able to visually recognise 'riskful' take-off and landing techniques, or whether they can be trained to recognise them. Taken together, prevention strategies should focus on kinetic chain function and on changing stiff take-off, especially landing patterns.

The current notion that PT relates to landing technique (involving eccentric loading) more so than the jump take-off (involving primarily concentric loading) supports the idea of using eccentric training in the rehabilitation of PT.[31] To adapt the tendon to eccentric forces may reduce the detrimental effect of such forces. Eccentric exercises may thus also be investigated for their potential use as a preventive measure in addition to their use in rehabilitation.[20]

Finally, future research focusing on risk factors for PT should preferably use a prospective design where data of jump biomechanics are collected at baseline in asymptomatic subjects, who are then followed longitudinally, which will enable us to gain more insight into the causality question. Furthermore, though the subject of study is labelled as a knee problem, joints are evidently connected. Hence in line with the kinetic chain function approach in PT rehabilitation,[31] studying the coordination between joints (see e.g. Hughes et al. (2008), Yeow et al. (2011))[34,35] may provide valuable information about jumping patterns in relation to developing and, accordingly, preventing PT.

Conclusion

We studied the literature for the relation between take-off and landing biomechanics and PT. Although the identified literature was diverse in methods used, jump actions studied and in populations, a synthesis of the literature suggests that PT is mainly caused by factors related to landing rather than take-off. This may raise the question of whether a more appropriate label for this injury would be ‘lander’s knee’ rather than ‘jumper’s knee’. Employing a flexible landing pattern may also be an expedient way to reduce the risk for PT in athletes who take part in sports that involve jump actions. We propose to investigate kinetic chain functioning, eccentric training and particularly changing landing patterns as possible ways to achieve this.

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