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The influence of foot positioning on ankle sprains

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1. Introduction

Ankle sprains are arguably the most common sports injury. They are the most common injury in soccer (Ekstrand and Gillquist, 1983; Maehlum and Daljord, 1984b), volleyball (Schafle, 1993), and all sports combined (Garrick, 1977; Maehlum and Daljord, 1984a). Ankle sprains are not trivial, as they account for up to one sixth of all time lost from sport due to injury (Garrick, 1977). Many ankle sprains occur when performing a lateral cutting movement, or landing on an irregular surface, such as the foot of another player (Garrick, 1977). Landing on an irregular surface or a poorly executed cut can result in a large supination torque which supinates the foot excessively, overloading and damaging the lateral ankle ligaments including the anterior talo-fibular (ATF) and the calcaneo-fibular (CF) ligaments.

Individuals with a history of ankle sprains are more susceptible to ankle sprains than others. Ankle sprains are associated with increased susceptibility to subsequent

sprains, or chronic ankle instability, in between 20 and 50% of all sprain sufferers (Freeman et al., 1965; Smith and Reischel, 1996), and it has been found that 47% of ankle sprains occurs in ankles that had been previously sprained (Ekstrand and Gillquist, 1983). Therefore, in previously sprained ankles, there must be some factor that causes the ankle to be excessively supinated more frequently.

There are numerous factors and mechanisms that are thought to contribute to this increased ankle sprain occurrence (Lentell et al., 1995). One of these factors is the inability to accurately position the foot prior to touchdown. Once the foot has touched the ground in a potential ankle sprain situation, it is questionable whether the ankle pronating muscles can react quickly enough to prevent an injury-causing excessive supination (Isakov et al., 1986). However, the position of the foot as it first touches the ground may influence the sprain frequency. If the foot is already supinated at touchdown, the ground reaction force moment arm about the subtalar joint may be greater, causing excessive supination (Fig. 1). Furthermore, if the foot is plantarflexed at touchdown, it may also increase the ground reaction force moment arm about the subtalar joint (Fig. 2) (Barrett and Bilisko, 1995; Shapiro et al., 1994). This inappropriate foot positioning prior to touchdown has been hypothesized to be a fundamental cause of ankle sprains

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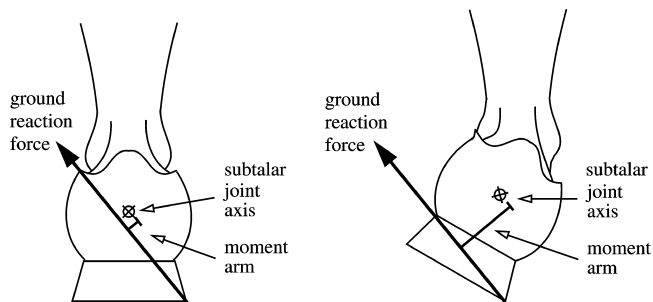


Fig. 1. A view of the foot and ankle from behind at touchdown when performing a cutting or side-shuffle movement. The moment arm of the ground reaction force about the subtalar joint when the foot is flat (left) is much smaller than the moment arm when the foot is supinated (right).

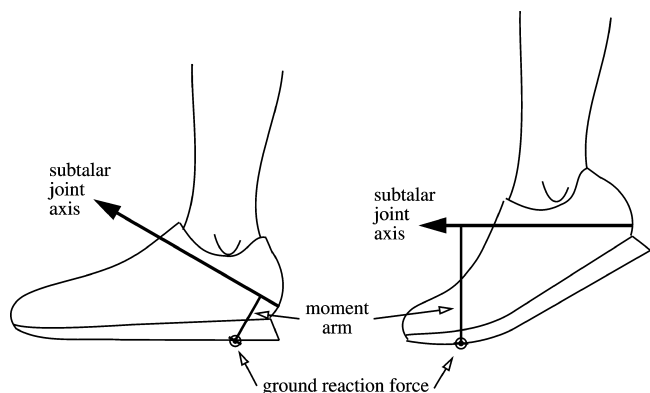


Fig. 2. A view of the foot and ankle in the sagittal plane at touchdown when performing a cutting or side-shuffle movement. The moment arm of the horizontal component of the ground reaction force about the subtalar joint when first contact is made with the heel (left) is much smaller than the moment arm when the foot is plantarflexed and first contact is made at the toe (right).

(Robbins et al., 1995), but this hypothesis has not yet been tested.

For inappropriate foot positioning to cause increased susceptibility to subsequent sprains in previously sprained ankles, the initial sprain would have to cause subsequent inappropriate foot positioning. A history of ankle sprains or chronic instability has been associated with impaired foot position awareness (Lentell et al., 1995). This may be due to damage to the proprioceptive sensors in the ligaments of the ankle (Freeman et al., 1965). Alternatively, inappropriate foot positioning may be caused by disturbances in motor control, which have been observed in ankle sprain patients (Bullock-Saxton et al., 1994). Postural stability, an indirect measure of a combination of joint position sense and motor control, has also been associated with a history of sprains (Tropp et al., 1984). Therefore, this inappropriate foot positioning is a potential cause of the increased sprain susceptibility following an initial sprain.

If inappropriate foot positioning is an underlying cause of increased ankle sprain susceptibility, then limiting foot position may help prevent ankle sprains. Ankle taping and bracing have been shown to reduce the incidence of respraining (Surve et al., 1994), and may do so by preventing inappropriate foot positioning. Just prior to touchdown when the ankle is not weight bearing, the externally applied torque at the ankle is zero. Therefore, even a small stiffness in the tape or brace may return the ankle to a more appropriate position, even though the tape or brace strength may be too small to directly prevent ligament overloading by bearing the load after the foot starts to bear weight (Ashton-Miller et al., 1996). Alternatively, since ankle taping improves kinesthetic awareness, or joint position sense, in the unloaded ankle (Heit et al., 1996), the tape may aid in active foot repositioning by the leg muscles prior to touchdown. There are, however, other possible mechanisms by which ankle taping or braces may reduce the sprain frequency, so the hypothesis that inappropriate foot positioning at touchdown contributes to sprain occurrence must be tested.

There have been many clinical and experimental studies that have attempted to identify the underlying causes of increased ankle sprain susceptibility and to identify the mechanism by which ankle taping and bracing prevent ankle sprains. However, these past studies did not examine side-cutting movements, and many studies examined the ankle only when it is already weight bearing (Ashton-Miller et al., 1996; Isakov et al., 1986; Johnson and Johnson, 1993; Konradsen et al., 1997). One study did examine the touchdown phase of a movement, but it did not examine touchdown foot position (Springings et al., 1981). Additional shortcomings of *in vivo* studies include the inability to carefully control muscle activity, and that direct injury observation is not possible for ethical reasons. Experimental ankle sprain investigations *in vitro* have been performed (Dias, 1979; Leonard, 1949), but muscle activities are difficult to simulate, and repeated tests to failure with the same specimen were not possible. Forward dynamics computer simulation does not have the same limitations that *in vivo* and *in vitro* experiments have. A model has been developed that has been shown to be suitable for ankle sprain studies (Wright, 1999) and was available for the study of the influence of touchdown foot positioning on sprain occurrence.

Therefore, the purpose of this study was to determine the influence of foot positioning at touchdown on simulated ankle sprain susceptibility. It was hypothesized that increased plantar flexion and increased inversion at touchdown would cause an increase in inversion sprain susceptibility. This information will help identify factors that contribute to ankle sprain occurrence, and will help identify the mechanism by which ankle taping and braces prevent respraining.

2. Methods

2.1. Forward dynamics simulation

The forward dynamics simulation model was developed using DADS (version 8.5, CADSI, Coralville, IA) based on the work of Delp (1990), and has been described in detail previously (Wright, 1999). The model consisted of rigid bodies representing the segments of the right leg of a 180 cm tall, 75 kg male. The torso, head, arms and the other leg were represented by a visceral mass and a rest-of-body segment. The subtalar joint and talocrural joint were represented by revolute joints aligned with joint axes based on Inman (1976). Passive non-linear joint stiffnesses were applied as moments about the subtalar and talo-crural joint axes to represent the effects of passive soft tissue and bony constraints on movements about these axes (Chen et al., 1988). For the knee, the centre of rotation of the shank relative to the thigh moved in the sagittal plane as a function of knee flexion angle. Adduction/abduction and internal/external rotation were permitted at the knee and were limited by passive moments. The hip joint was modelled as a spherical joint.

The model was actuated by 14 muscle groups, each representing one or several functionally similar muscles. These muscle groups were the gluteus maximus, gluteus medius, adductor magnus, iliopsoas, rectus femoris, hamstrings, vasti, gastrocnemius, soleus, flexor digitorum, tibialis posterior, tibialis anterior, extensor digitorum and peroneals. Each muscle had an origin and insertion fixed relative to the model segments, and several muscles had additional points through which the muscle passed to prescribe more anatomically accurate muscle paths (Delp, 1990). The muscle force-length-velocity-activation characteristics were modelled using a Hill-based model controlled by square-wave stimulation patterns. The stimulation onset was permitted to be before the simulation started so that the muscle activation could be increasing before touchdown.

The contact between the foot and the ground was represented by 66 discrete independent contact elements, each representing the mechanical properties of a region of a running shoe sole and the underlying soft tissue.

2.2. Ankle sprain simulations

Forward dynamics simulations of the first half of stance phase of a side-shuffle movement were performed. Initial kinematic conditions for the simulations were taken from the mean measured limb segment positions, orientations and velocities at touchdown of 10 subjects performing a side shuffle movement (Neptune et al., 1999). Muscles stimulations were selected that minimized the difference between the measured and simulated

movements for the flat surface condition for one subject as described previously (Wright, 1999; Wright et al., 1998,2000). These same muscle stimulation patterns were then used to perform simulations for 10 subject-specific sets of initial conditions on 50 irregular floor conditions, with 11 different values for the peroneus strength and 11 different onset times for peroneus stimulation (for a total of 11 000 simulations). For each simulation maximum supination angle and maximum supination torque were calculated. The torque values determined were those applied to the passive resistance at the subtalar joint and did not include the load on the muscles. In real subjects, these torques would be resisted by bony constraints and ligaments.

In the simulations, landing on an uneven surface was assumed to be the cause of ankle sprains. The floor in the computer simulation was divided into four quadrants, with the intersection of the four quadrants located beneath the middle of the shoe (at touchdown). The 50 irregular floor conditions were generated by varying the height of each quadrant with a uniform random distribution over ± 10 cm. The height of the simulation model was adjusted so that the foot was just touching the highest point on the floor at the beginning of the simulation (touchdown).

The subtalar and talocrural joint angles were manipulated independently in the range $\pm 15^\circ$ from subject-specific touchdown values. For each altered joint angle configuration, simulations were performed on each of the 50 irregular floor conditions.

A sprain was said to have occurred for any given simulation when the torque about the subtalar joint exceeded some given value, or when the angular displacement at the subtalar joint exceeded some given value. The absolute magnitude of the threshold at which an injury occurred would have been difficult to determine and was likely to vary considerably between subjects. Therefore, a range of threshold values was used and the number of simulations which resulted in maximum torques or displacements greater than these threshold values was determined.

3. Results

Increased initial supination angle caused an apparent small increase in the occurrence of sprains while a decrease in the initial supination angle (i.e. an increase in the initial pronation) angle caused an apparent small decrease in the occurrence of sprains (Fig. 3). The results were the same whether a supination torque threshold or a supination angle threshold was selected as the criterion for sprain occurrence. This apparent influence was most evident when the threshold (either the angle or torque above which a sprain was considered to have occurred) was low. There was almost no influence of initial

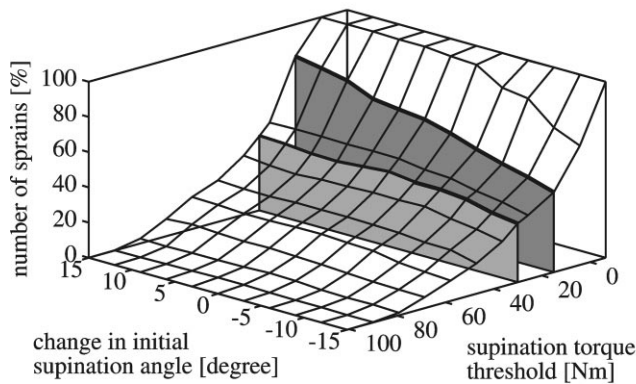
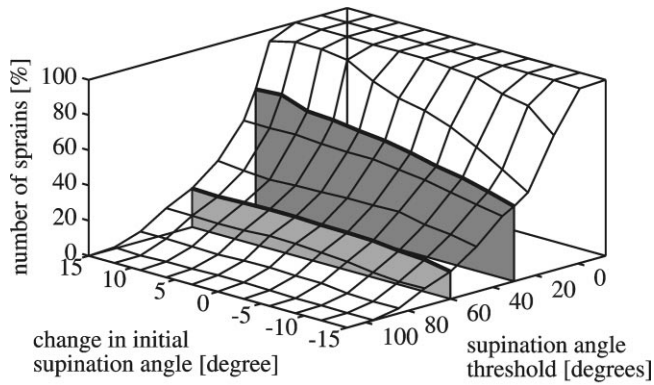


Fig. 3. The number of sprains (percent) as a function of change in initial supination angle and supination angle threshold (top) or supination torque threshold (bottom). Sprains are defined as occurrence of either supination angle (top) or passive supination torque (bottom) exceeding the threshold value. The dark and light shaded supination angle threshold values are 30 and 60°, respectively. The dark and light shaded supination torque threshold values are 15 and 30 Nm, respectively.

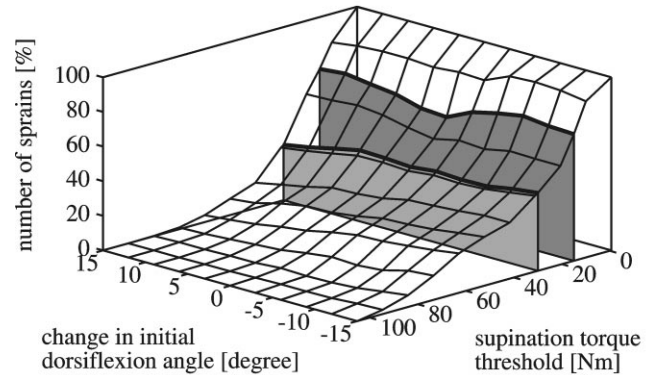
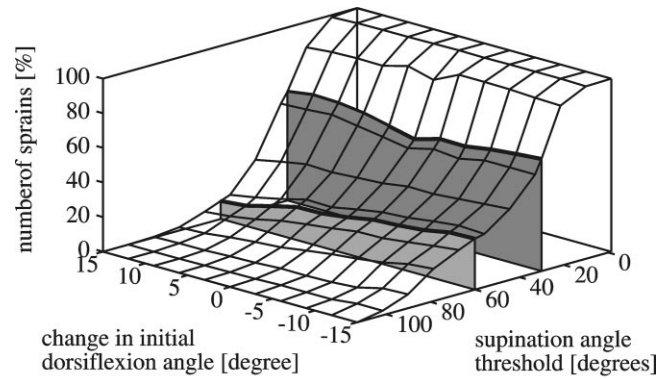


Fig. 4. The number of sprains (percent) as a function of change in initial dorsiflexion angle and supination angle threshold (top) or supination torque threshold (bottom). Sprains are defined as occurrence of either supination angle (top) or passive supination torque (bottom) exceeding the threshold value. The dark and light shaded supination angle threshold values are 30 and 60° respectively. The dark and light shaded supination torque threshold values are 15 and 30 Nm, respectively.

supination angle on sprain occurrence when the threshold angle or torque was relatively large.

For larger torque or supination angle thresholds, an increase in the initial dorsiflexion angle caused a decrease in the sprain occurrence, while a decrease in the initial dorsiflexion angle (i.e. an increase in the initial plantar flexion angle) caused an increase in the occurrence of sprains (Fig. 4). This effect was not observed when the torque or angle thresholds were low. The mean peak supination angle (across all subject-specific sets of initial conditions and all floor conditions) increased with increasing initial supination angle (Fig. 5a) as did mean peak supination torque (Fig. 5b). Mean peak supination angle decreased with increasing initial dorsiflexion (Fig. 5c) as did mean peak supination torque (Fig. 5d).

4. Discussion

For low threshold values, changes in the initial supination angle appeared to have an influence on sprain occur-

rence. However, at low threshold values, the effect of the initial supination angle on the number of excessive supinations was likely to have been exaggerated. The irregular floor conditions used to cause sprains in this study included irregular floor conditions that may have caused pronation of the foot upon touchdown rather than supination. In these instances, the maximum supination angle would have simply been equal to the initial supination angle. Therefore, when the threshold angle was less than the maximum initial supination angle plus the largest change examined ($17.3^\circ + 15^\circ = 32.3^\circ$), some simulations that were counted as sprains may not actually have had supination of the ankle following touchdown, and would not have resulted in an injury to a real subject. This effect was also revealed by the plot of the mean maximum supination angle vs. the initial joint angle (Fig. 5a). The change in the mean maximum supination angle was smaller than the range of change in the initial supination angle. To be certain that the influence of the initial supination angle on sprain frequency was not exaggerated in this way, only the sprain

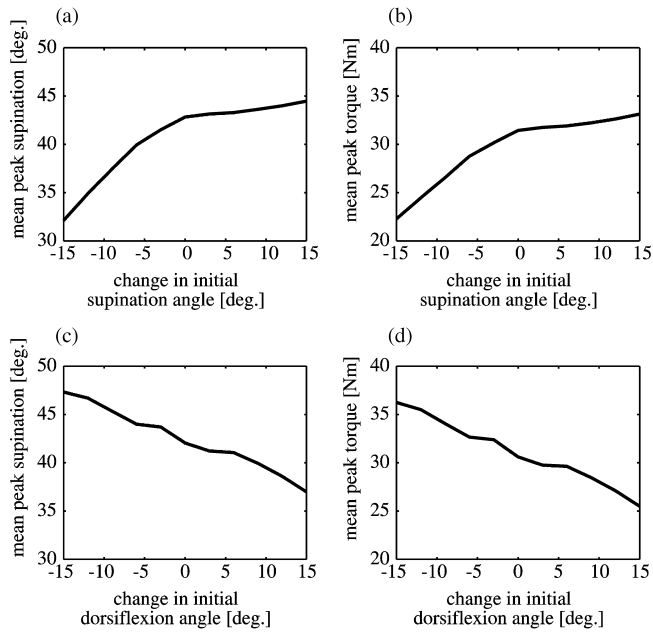


Fig. 5. Mean (across 500 simulations) peak supination angles vs. change in initial supination angle (a), mean peak supination torques vs. change in initial supination angle (b), mean peak supination angles vs. change in initial dorsiflexion angle (c) and mean peak supination torques vs. change in initial dorsiflexion angle (d).

occurrences when the supination threshold was greater than 32° should be considered. In this region (Fig. 3), the influence of the initial supination angle on sprain frequency was small. Taking this into consideration, the mechanism of sprain occurrence was shown not to agree with the situation depicted in Fig. 1. These current findings did not support the hypothesis that initial supination of the foot can contribute to sprain occurrence.

The influence of initial dorsi/plantar flexion on sprain occurrence was not subject to the same exaggeration as described above for the initial supination angle. When the initial dorsi/plantar flexion angle was manipulated, the initial supination/pronation angle was held constant, and so there was no biasing of the supination angles based on the initial conditions. Also, for varying dorsi/plantar flexion angle, the influence on sprain occurrence was small when the threshold angle or torque was small, but was more noticeable when the threshold was greater. This meant that the more plantarflexed the foot was at touchdown, the greater the incidence of excessive supination. This result was further supported by the findings presented in the literature. Inversion sprains often occur when the foot is plantarflexed (Leonard, 1949; Renstrom and Konradsen, 1997), and the ATF ligament, which is loaded when the foot is plantarflexed and supinated, is the most frequently sprained ligament of the ankle (Leonard, 1949; Saunders, 1980). Therefore, it appears that susceptibility to sprains is increased by initial plantar flexion.

In addition to the predicted increase in sprain frequency with increased touchdown plantar flexion, the mean peak torque decreased with increasing dorsiflexion (Fig. 5c). This result suggested that increased plantarflexed position of the foot at touchdown increased not only the sprain susceptibility, but also the severity of the sprains. However, to the authors knowledge, there is no clinical support for this finding.

In the current investigation, only a side-shuffle movement was investigated. Sprains occur during a variety of movements. The findings of the current study cannot be extrapolated to all movement conditions. Also, there are several other factors that may contribute to sprain occurrence that have not been investigated here. To investigate other movements and other possible sprain causing factors, additional simulations would have to be performed.

In real subjects, the torque or load required to cause an injury may change with foot position. This was not taken into consideration in the current study. As the ankle is plantarflexed, the ATF ligament becomes more stretched (Nigg et al., 1990). The ATF ligament is the weakest of the lateral ligaments of the ankle (Siegler et al., 1988), and may sustain damage at lower load levels. If this is true, then the threshold supination torque required to cause an ankle sprain injury may be lower as the foot is plantarflexed thereby increasing the influence of increased initial plantar flexion on sprain occurrence.

Ankle taping and bracing may influence the position of the unloaded foot, decreasing the tendency of the foot to be plantarflexed. Taping and bracing stiffen the ankle in dorsi/plantar flexion as well as supination/pronation (Bruns et al., 1996; Siegler et al., 1997). Siegler et al. suggested that “the largest effects of the ankle braces on the passive flexibility of the ankle complex occurred near the neutral position of the ankle.” Therefore the braces not only limit the range of motion, but may return the unloaded ankle to a neutral position. Since the foot was initially plantarflexed an average of 10° s at touchdown during the simulated side-shuffle movements (Neptune et al., 1999), returning the foot to a neutral (zero dorsiflexion position) would tend to decrease the sprain frequency. Both ankle taping and bracing have been shown to improve foot position awareness in the unloaded foot (Heit et al., 1996). Therefore, in addition to passive ankle repositioning, taping and bracing may improve active ankle repositioning (by the muscles) in response to proprioceptive signals, thereby reducing the occurrence of excessive plantar flexion at touchdown. The findings of the current study would therefore suggest that repositioning of the foot prior to touchdown may be the means by which taping and bracing reduce ankle sprain frequency.

This proposed mechanism by which taping and bracing prevent sprains is consistent with the finding that taping and bracing reduce the frequency of sprains in previously sprained ankles more than in unsprained

ankles (Surve et al., 1995). An unsprained ankle presumably has sound proprioception and motor control, so is less likely to land with inappropriate foot positioning. However, a recently sprained, or chronically unstable ankle may have impaired proprioception or motor control, and be more likely to land in a more plantarflexed position. The tape or brace, either by improving foot position awareness or mechanically repositioning the foot in the unloaded situation prior to touchdown, returns the foot to a neutral position, thereby counteracting the effects of the previous injury, but not improving a healthy ankle.

The mechanism of increased ankle sprain occurrence investigated in this study was one of many factors that may contribute to ankle sprains. In this study, it was found that touchdown plantar flexion angle may have a greater influence on sprain occurrence than touchdown supination angle. However, no conclusions can be drawn about the relative importance of this factor compared to other possible sprain causing factors, such as changes in mechanical strength or length of the damaged ligaments or changes in strength of the peroneus longus and brevis muscles for example. Since many of these factors tend to co-occur in ankles that have been sprained (Lentell et al., 1995), it has been impossible to examine any of these factors independently. The methods used in the current study permit the examination of some of these factors independent of one another. Additional studies of several other factors thought to contribute to ankle sprains will have to be completed before conclusions can be made about which of these factors are the most significant contributors to sprain occurrence.

In conclusion, a tendency of the foot to be in a more plantarflexed position at touchdown may cause an increased occurrence of ankle sprains. This may be the mechanism which causes ankles with a history of ankle sprains to have an increased susceptibility to subsequent sprains. This may also reveal a mechanism by which taping of a sprained ankle or the application of an ankle brace leads to decreased ankle sprain susceptibility.

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