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# A Review of the Function of the Quadratus Plantae

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The Foot and Ankle Online Journal 2 (11): 5

*There has been very little scientific evidence of any well defined function of the quadrates plantae (QP). It has been the focal point of multiple theories. This paper discusses four of those theories: the QP as a counter to the oblique force of the flexor digitorum longus (FDL), QP as a pronator of the foot, the QP as a plantarflexor of the lesser digits and lastly the QP as a stabilizer of the lumbrical muscles. No single theory has received unanimous support. The following is a presentation of the support and opposition which has surfaced for each theory.*

**Key Words**: Quadratus plantae, plantar foot muscles, biomechanics.

**Accepted:** October, 2009 **Published:** November, 2009



The quadratus plantae (QP) muscle is located in the second layer of the plantar musculature and consists of two heads separated by the long plantar ligament. (Fig. 1) The medial head originates from the medial surface of the plantar calcaneus, while the lateral head originates from the lateral border of the calcaneus. The muscle inserts into the posterolateral margin of the flexor digitorum longus (FDL) tendon as it crosses the plantar foot obliquely from posteromedial to anterolateral. This insertion falls at the level of the origin of the lumbrical muscles.[1] As for the biomechanical function of the QP, there is a relative lack of evidence supporting any one particular theory. At least four theories of its function have been proposed, however very little has been demonstrated through empirical research. The goal of this paper is to address these four theories, along with a discussion of the consenting and dissenting voices of each.

*The QP Countering the Oblique Pull of the FDL*

Certainly, one of the most widely accepted theories states that the QP functions to balance out the oblique pull of the FDL on the lesser digits. Again, the FDL tendon courses obliquely across the plantar foot in a direction that would tend to pull the lesser digits, particularly the fourth and fifth digits, into an adductovarus position, if it were not for this straightening effect.[1-8] Although many have simply accepted this theory and published it as fact, others have tried to provide direct evidence. Manoli and Weber reported on three cases which provided fairly convincing evidence in support of this theory.[9]

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ISSN 1941-6806 doi: 10.3827/faoj.2009.0211.0005

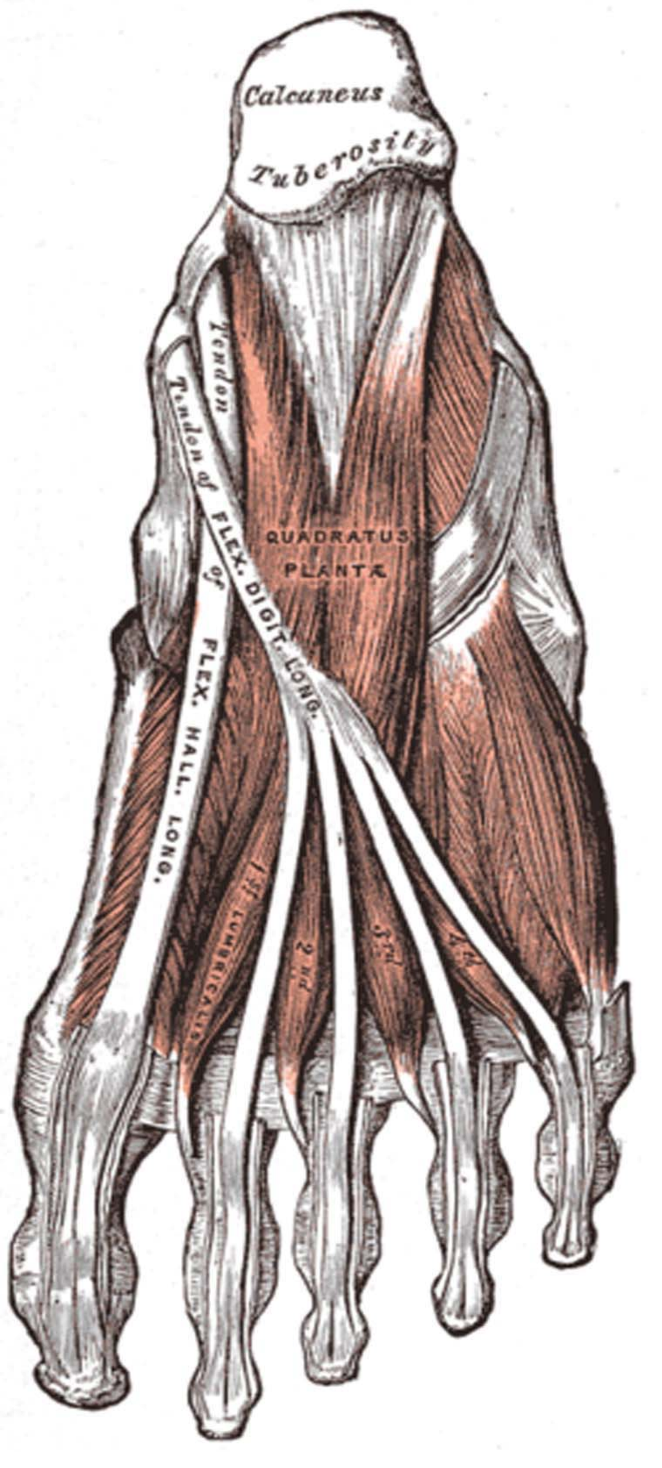


**Figure 1*** The qudratus plantae muscle resides in the second muscle layer of the foot. It's origin is from the calcaneus and inserts at the origin of the lumbrical muscles.

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Their intention was to demonstrate that the QP was in its own separate plantar compartment, the calcaneal compartment. Each of the cases consisted of a calcaneal fracture which healed uneventfully, but with lesser digit adductovarus contractures developing in each of the patients within 8-13 months. They also found that none of the deformities were reducible with ankle plantarflexion, indicating intrinsic muscular contracture. They hypothesized that the contractures were due to an undetected calcaneal compartment compression damaging the neurovascular supply to the QP. They concluded therefore that the QP functions to prevent adductovarus toe contractures of the lesser digits.

Interestingly however, the effect of the damaged QP in this study was most prominent in the second and third digits. If the QP was damaged and the oblique angle of the FDL was exaggerated on the toes, wouldn't the fourth and fifth receive the most oblique force? The results didn't appear completely consistent with the theory.

Not everyone has accepted this theory. Basing much of their argument on the anatomic presence of the flexor tendon sheath, others claim that an oblique pull on the toes would be impossible.[10,11] The well-defined flexor sheath on the plantar side of each digit begins proximal to the metatarsal phalangeal joint. The tendon of the FDL runs in this sheath and its resultant force could only be in line with the sheath. Furthermore, the interphalangeal joints are hinge joints and any force applied to them could only result in uniplanar motion.[10,11] Kaplan took his argument one step further with a cadaver experiment. He applied multidirectional forces to the cadaver FDLs and the result in all cases was straight toe flexion. He did not find that the QP was required to produce straight flexion of the digits.[10]

*The QP as a Pronator*

This experiment conducted by Kaplan contributed to the development of the theory that the primary function of the QP is to pronate the foot. In his cadaver experiments, the FDL was pulled on both a fixed calcaneus as well as an unfixed calcaneus. When the FDL was pulled on the unfixed calcaneus, it resulted in straight toe flexion with obvious adduction of the foot and supination of the lateral border. When the calcaneus was unfixed and a force was applied to the FDL and QP, the resultant motion was again straight toe flexion, but this time the foot pronated with no adduction. Therefore the QP seemed to be a pronator of the foot. Kaplan also dissected the feet of two infant cadavers which were born with congenital club foot. In further support of the theory, there were no QP muscles present in either one.[10]

Reeser, et al., challenged this theory with electromyographic studies of the foot. The study revealed no difference in the activity of the QP when the foot was inverted versus everted. Their conclusion was that the QP does not necessarily aid more in pronation or supination. They theorized that a pronator muscle would fire more during pronation than supination, which apparently was not the case. This study was based on four participants.[11]

*The QP as a Lesser Digit Plantarflexor*

Reeser, et al.,'s study brought up a point in support of yet another theory. They found that the only time that the QP consistently fired in each of the patients was during plantarflexion of the digits. The other movements incorporated with the toe plantar flexion did have some impact on the strength of the contraction, but ultimately combinations of motion which included plantarflexory toe movements demonstrated firing of the QP.

They concluded that this muscle was intimately involved with toe plantarflexion. They proposed that the QP functions to counteract active insufficiency of the FDL in plantarflexion, as well as create more force at the interphalangeal joints when needed.[11] The previously discussed case series presented three cases of supposed QP damage followed by claw toes of the second and third digits.[9] A contracture of the QP may have been the force pulling the distal phalanges into plantarflexion. It was interesting however that the fourth and fifth digits were not the primary digits affected.

*The QP as a Lumbrical Stabilizer*

Anatomic location is the basis for the next theory proposed by Root, et al.[2] They stated that due to the location of insertion of QP on the FDL, it could possibly stabilize the lumbricales. The lumbricales originate on the FDL near the insertion of the QP.[2] This theory has not met with the same opposition that the others have. This probably provides a small piece of the complete puzzle, whatever that may be.

## Conclusion

No single theory about the function of the QP has been unanimously supported or rejected by the scientific community. There is indeed a lack of evidence available to support any one line of thinking. The reality of this controversy is that a combination of these theories, and some yet to be discovered, make up the true function of the QP muscle. Certainly more research is warranted. If the first theory discussed holds true, and a weak QP causes adductovarus deformity of the fourth and fifth digits, then a method of strengthening the QP could be developed to prevent such cases. Likewise the involvement of the QP in the other functions discussed could lead to advances in the prevention and correction of some supinatory deformities and contracted digits.

Insight into the etiology of this deformity, and others like it, can lead to prevention. The impact of the QP needs to be determined and its function isolated to lead to these important steps of foot control and function.

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