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Iliotibial Band Impingement Syndrome: An Evidence-Informed Clinical Paradigm Change

Paul R. Geisler, EdD, ATC, and Todd Lazenby, MA, ATC • Ithaca College

Clinical practice in sports medicine is often guided by axioms or paradigms of practice, some of which have persisted over time despite a lack of objective evidence to support their validity. Evidence-based practice compels practicing clinicians to not only seek out and produce evidence that informs their decision-making, but also to challenge existing paradigms of thought and practice, especially when favorable treatment outcomes remain elusive. Insidious, load induced lateral knee pain around the iliotibial band in runners, cyclists, military personnel, rowers, and other athletes has for decades now been conceptualized as *iliotibial band friction syndrome*, a biomechanically based and unsubstantiated paradigm based on Renne's 1975 theory that the iliotibial band slips back and forth over the lateral femoral epicondyle during flexion and extension movements of the knee, primarily irritating the underlying bursa and even the iliotibial band itself. Newer evidence about the anatomy and biomechanics of the iliotibial band, the physiology of the condition, and interventional outcomes is now available to challenge that long-held paradigm of thought for iliotibial band related pathology. Given this plethora of new information available for clinical scientists, *iliotibial band impingement syndrome* is proposed here as a new, evidence-informed paradigm for evaluating and treating this problematic overuse syndrome.

For many practitioners in the field of sports medicine, the specific paradigm for insidious onset, chronic lateral knee pain in runners, cyclists, and rowers has contained the idea that the iliotibial band (ITB) "moves over and back of the lateral femoral epicondyle" with repeated flexion and extension movements of the knee. Further, it has long been accepted that this biomechanically induced and anatomically based "frictional" force aggravates a subtendinous bursa that separates the bony prominence from the undersurface of the tendinous aspect of the ITB, causing localized and debilitating pain from an inflamed bursa, an inflamed ITB, or both with chronic knee motion. Many clinicians know this pathoetiological model as "iliotibial band friction syndrome", a nebulous but widely accepted clinical overuse syndrome first learned in formal education and thought to occur more commonly in running athletes with "tight IT Bands" because of mechanical "slipping" in the band itself. As a result, ITB friction syndrome has long been treated with stretching of the ITB and deep tissue massage to the irritated aspects of the tissue itself, in an attempt to loosen the tight tissue and thus relieve the pain and discomfort. As foundational knowledge of anatomy, physiology, and biomechanics has advanced in recent years, the current evidence supports the approach that stretching and massage to the ITB is, in the least, ineffective at decreasing symptoms but rather most likely to exacerbate the problem.[1] Poor treatment outcomes are perhaps just one indication that our axiomatic conception of the pathoetiology of the syndrome is inaccurate, and thus it is time for a new evidence-informed paradigm for assessing and treating this overuse injury.

A Common Problem for Repetitive Flexion–Extension Athletes

Epidemiologically, ITB-related pain and discomfort occurs primarily in active persons, with approximately 12% of all runners being affected.[2] Though ITB friction syndrome (ITBFS) is widely reported as the "most common cause of lateral knee pain in athletes", its frequency is varied in both distribution and population, with a reported overall incidence range between 1.6% and 52%.[3] Orava found ITBFS as the primary culprit of pain in 6.4% of every 1,000 athletes across various sports, while Linenger stated it comprised 22% of all lower extremity injuries,[4,5] yet Fredericson and Wolf reported it accounted for only 12% of all overuse running injuries.[2] Many additional ITBFS studies have been published focusing on specific populations, as Renne first reported 1–5% of all military recruits suffer from the condition and Noble reported that 52% of long-distance runners presenting with insidious knee pain had ITBFS.[6,7] Devan and his group noted ITBFS to be the most common lower extremity overuse injury in female soccer, basketball, and field hockey athletes, while Holmes et al. reported a 24% occurrence in road cyclists, and Rumball et al. documented it as a very common ailment for competitive rowers.[8–10] In addition, recreational exercisers are not immune to ITB-related lateral knee pain as it has been reported to be problematic in 15% and 7% of adult women and men, respectively, who perform general exercise.[11]

The Current Paradigm of Practice and Thought

As we have been so often taught, the key to understanding complex and troublesome musculoskeletal syndromes is to first have a solid understanding of the relevant pillar knowledge that contributes to the phenomenon at hand—the anatomy, physiology, and biomechanics of the body part, joint, or system under investigation. When knowledge of anatomy, physiology, and biomechanics is well established *and* understood, this clinical science and reasoned approach to problem solving is typically straight forward and follows a general set of somewhat predictable rules that make comprehension productive and intervention effective. However, when proper evidence or knowledge is lacking, particularly in the domains of anatomy, physiology, and biomechanics, the challenges presented to the clinician are magnified because of the increased complexities associated with understanding the *pathophysiological* underpinnings of the matter, and patient outcomes remain bothersome due, at least in part, because the working paradigm guiding clinical practice is incomplete or inaccurate. The anatomy and function of the ITB or iliotibial tract (ITT) exists in somewhat of a "gray zone" for many clinicians, partially because of what many of us were formally taught, due to what was actually known for many years about this long, tough tissue that has its own name. Our clinical education and best practice of the time led us to assess ITB tightness or dysfunction with the Ober's test, and then to treat any tightness with stretches and massage techniques focused on lengthening the tissue. Based on the current evidence, it has become clear that these common practices should be reexamined and refined.

First, a Little History

Sixteenth century anatomist Vasalius first described the tensor fascia lata as a tibial muscle, and given its clear and palpable tibial insertion, this made sense at the time.[12] *Gray's Anatomy,* the much revered text, described the ITB in a rather pedestrian manner—a structure "over the lateral femoral aspect (where) the fascia lata is compacted into a strong iliotibial tract."[13, p. 339] Kaplan concluded in 1958 that although all quadruped animals have gluteus maximus muscles and tensor fascia lata, only humans actually have an ITB because of its developmentally and functionally critical roles in stabilizing the lateral knee joint and assisting with erect posture.[14] More has been learned in the past 10–15 years in the area of fascial anatomy, histology, neurophysiology, and force transmission dynamics alone than in the previous millennia combined, as recent advances in technology have dramatically improved our ability to not only visualize living fascia tissue, but also begin to better understand the function and components of this constituent, three-dimensional tension network. In addition, the establishment of the International Fascia Research Congress has brought clinicians, scientists, anatomists, and physicians from a multitude of different disciplines together to share, discuss, and disseminate their respective research and clinical and anecdotal experiences in the exploration, evaluation, and treatment of fascia tissue function and dysfunction.[15] This has led to a realization of the importance of a greater scientific understanding of this often ignored, dissected, and discarded tissue in the overall purpose of the human form and function. As a

result, our understanding of the anatomy, function, and purpose of the ITT has increased substantially to the point that what many of us were taught about the etiology of ITB dysfunction can now be turned on its ear.

Today, many clinicians have generally come to understand the basic details of the ITB, but perhaps far too simplistically (see Table 1): that the ITB originates from the fascia of the tensor fascia lata (TFL) and gluteus medius muscles and is anchored on the iliac crest, anterior superior iliac spine, and the capsule of the femoroacetabular joint and distally travels down the lateral femur with broad insertions to the linea aspera, is contiguous with fascial tissue that envelopes the thigh, and has one pronounced and well versed insertion at Gerdy's tubercle on the lateral tibial prominence.[16] Indeed, this model is still taught today in many professional programs and, further, properly palpating Gerdy's tubercle (because of its association with the ITB) remains a key component of many oral practical examinations at the professional level. In relating anatomy to biomechanics (and thus, too, to pathomechanics), it has largely been accepted that because the ITB originates from two hip joint muscles and inserts on Gerdy's tubercle, it was simplistically considered as a tendon (in that it provided distal anchoring for the tensor fascia lata muscle), and that it logically "worked" to transmit the muscular forces and energy of the TFL and gluteus medius (in other words, to assist with abduction of the hip, especially in a flexed hip position).

To be sure, the ITB or ITT is a lot of things at once—by name it connects the hip ("ilia") to the lower leg (tibial); by location it runs from the iliac crest, anterior ilium, and anterior superior iliac spine to the linea aspera and Gerdy's tubercle on the tibia; by nature and depending upon what portion one is referring to, it is part fascial thickening, part ligament, and part tendon; by architecture it receives most of the gluteus maximus muscle fibers and all of the tensor fascia lata muscle fibers; and by microscope it is a dense, regular connective tissue composed almost exclusively of regular collagen with a little bit of elastin content, and is largely avascular. As with other tissues in the body, fascia tissue demonstrates an enormous ability to adapt to mechanical stress and, in particular, repetitive demands. The ITB is a structural tissue that is not present when we are born, but develops over time into a dense, collagenous-rich fascia along the outside aspect of the thigh in response to the stress demands placed on the tissue during bipedal locomotion and functions to provide stabilization of the hip when walking, running, and hopping.[17] To point, the stiffness noted in the ITB upon palpation in individuals that walk and/or run regularly is absent in individuals who are sedentary or wheelchair bound. To better illustrate the adaptability of the fascial tissue, el-Labban and his group found the opposite to be true in horseback riders as the fascia along the medial aspect of the thigh has been found to adapt to the stress load patterns associated with saddle contact over time and therefore develop a thicker,

Table 1 Anatomical Components of Two Paradigms for ITB Pathology

ITB Paradigm	Origin	Insertion	Action(s) or Implication
Old (What we used to "know")	Fascial union from TFL and gluteus maximus on the iliac crest and ASIS	– Broad to linea aspera – Gerdy's tubercle on tibia	Transmit force of TFL muscle during hip activity (abduction from a flexed position); acts as "pelvic deltoid", stabilizing to stance stability and "swinging of the knee along with the hip"; restrict excessive hip adduction (if tight)
New (What we now "know")	Fascial union from TFL and gluteus maximus on the iliac crest and ASIS	– Broad to linea aspera – Lateral femoral condyle – Lateral femoral epicondyle – Patella via lateral retinaculum – Gerdy's tubercle (3 layers): superficial, deep, capsular osseous – Fibular head	Passive stability of the hip joint (via gluteus maximus tensioning in the ITB) during loading/deceleration in stance and monopodal gait; passively resists hip adduction and internal rotation during loading/deceleration in stance; passively limit anterior translation and internal rotation of the tibia during loading/deceleration in stance

Abbreviations: ITB = iliotibial band; TFL = tensor fascia lata; ASIS = anterior superior iliac spine.

more dense fascia traversing the medial aspect of the thigh.[18] Functionally, the ITB is also quite multifaceted as it crosses two large ambulatory and weight bearing joints, at times dependent upon position and motion, working proximally to move the hip in manners concordant with the function of the gluteus maximus and tensor fascia lata. The ITB also works distally as a tendon to anchor the aforementioned hip muscles and even as a ligament to help stabilize the patellofemoral and knee joints.[19]

The Origins of a Clinical Paradigm or Axiom

In 1975 and based off early understandings of anatomy and biomechanics, Renne first described the idea of ITBFS after noting a preponderance of insidious onset, lateral knee pain conditions in a population of US marines undergoing high-level physical training.[6] At that time, it was thought that the anatomical insertion of the ITB was solely on the lateral tibial prominence known as Gerdy's tubercle, therefore Renne hypothesized that the ITB would roll over the lateral femoral epicondyle with repetitive flexion and extension movements of the tibiofemoral joint. In full extension, Renne reasoned the ITB would be "anterior of the lateral epicondyle", and once past 30 or so degrees of flexion, the tendinous aspect of the band would be positioned posterior to the bony femoral prominence. With repetition, this movement or "slippage" would presumably create friction on the undersurface of the ITB and corresponding subtendinous bursa, creating inflammation, pain, and degeneration along the superior lateral aspect of the affected knee joint.[16]

Just a few short years later, Noble published a pair of papers that took up Renne's clinical axiom and extended the idea that ITB displacement or slippage was responsible for producing the sharp and pronounced pain at the site of the lateral femoral epicondyle, even coining the now oft used "Noble's Test" for diagnostic purposes.[7,20] According to Noble, increased pain with manual compression at the lateral femoral condyle (LFC) with the knee at 30° of flexion is diagnostic for ITBFS in runners "in all cases" and symptoms are worse with downhill running, stating the source of the pain was a wounded ITB and/or "bursal type tissue" or "fascial tissue", while the effective treatment simply consisted of rest, addressing training errors, steroid injections, and, in recalcitrant situations, corrective surgery. To date there are dozens of papers that have since taken up and extended Renne and Noble's "friction syndrome" paradigm for lateral knee pain and, as such, it has gradually become the axiomatic standard for clinical practice despite the absence of compelling evidence to support the original pathomechanical underpinnings, anatomical foundations, or the various interventional strategies employed.[2,3,21–24]

Our early and limited understandings of the anatomy and function of the ITB can now be appreciated as the root sources of the false clinical axiom that many have helped to promulgate, a paradigm of thought and practice we commonly and habitually know as "iliotibial band friction syndrome". However, more recent anatomical and biomechanical studies reveal that the attachments and functions of the ITB are actually far more complicated and multifactorial than originally thought. Most importantly, and with profound implications for an evidence-informed clinical practice, these new enlightenments shed a brighter light on the clinical axiom surrounding ITBFS that has been carried forth from our education and practice. Thankfully, this new clinical science offers critical evidence and direction for a new paradigm of thought concerning insidious onset lateral knee pain in highly active and running-based individuals.

Updating the Paradigm

In the mid-2000s, John Fairclough and colleagues clearly attempted to alter the working paradigm for ITB-related knee pain with the publication of two papers on the topic. Experimentally, the UK-based group put the ITBFS paradigm predicated upon a slipping tissue and a painful subtendinous bursa to the test with a thought provoking and multi-level study in the *Journal of Anatomy,* and followed that up a year later with an explanatory opinion piece in the *Journal of Science and Medicine in Sport*.[1,25] Using a combination of gross anatomical dissection and histology studies in cadaveric specimens and magnetic resonance imaging (MRI) scans of healthy and "ITB syndrome" positive subjects, Fairclough and colleagues put forth the following claims about ITB anatomy, its function, and its dysfunction: the ITB does not roll over the femoral epicondyle because it is anchored firmly by the fascia lata, rather an illusion of movement is created because of changing tensions in the anterior and posterior fibers of the ITB during flexion and extension, and, respectively, there is no subtendinous bursa, but rather a highly innervated fat pad deep to the ITB.[25] Further, this study showed two distinct regions of

the ITB that implicate a multifactorial function of the tissue, a 'tendinous' part proximal to the lateral femoral epicondyle and a 'ligamentous' portion between the epicondyle and its insertion at Gerdy's tubercle. The authors explained the "movement illusion" further based on a model of sequential "load shifting". As the knee flexes, the tendinous or fascial fibers of the ITB that attach to the patella come under tension as the patella tracks distally in the trochlear groove of the femur while the more posterior ligamentous portions become tensioned as knee flexion increases further. In short, the various ITB fibers are progressively tensioned from the anterior to the posterior during increasing knee flexion and the many fibrous connections of the ITB limit any significant movement in the area of the lateral femoral condyle.[1]

At about the same time, a Brazilian group added to the complexity with another anatomical dissection study of the ITT in an attempt to better define its insertional arrangements and its functional relationship with other structures of the knee.[26] Ten detailed dissections by Vieira and his team[26] revealed three specific distal layers of the ITT (band) that led them to conclude that not only does the ITB have critical connections to the femur, the patella, and the lateral tibia, but that it plays a pivotal role in patella-femoral stability. In addition, its capsular-osseous layer is an important anterolateral knee stabilizer that joins the anterior cruciate ligament in a functional unit, forming a special "horseshoe" system that helps prevent excessive tibial rotation and anterior displacement.

Further complicating the issue are at least three other studies concerning possible anatomical and noxious sources and contributors of ITB syndrome, one involving "normal anatomy", and two concerning benign tissue abnormalities. In 1996, Nemeth and Sanders built upon a 1941 report that detailed a "lateral extension of the synovial pouch" and their own clinical studies to further investigate chronic ITBFS.[27] Although they weren't the first (or the last) to report the presence of this "normal anatomical" feature, they could not resolutely report that the lateral synovial recess (LSR) is *primarily* involved in the pathogenesis of chronic ITBFS, but their study does point the finger at the LSR as at least being partially involved in the chronic lateral knee syndrome we call ITBFS. Based on histological and imaging (MRI) data, Nemeth and Sanders felt comfortable in stating the following; that no bursa exists under the ITB, there is a highly vascularized adipose tissue present between the femur and the ITB, and that histology results of subjects with a history of ITBFS supports the notion that regular "impingement" of the LSR and/or adipose tissue in the lateral spaces of the knee is responsible for the chronic inflammation and synovitis in the ITBFS patients.[27]

Grando and colleagues offered direct and more recent evidence for the role of both extrasynovial tissue and fat pads in ITB-related pathologies in their 2014 article published in *Magnetic Resonance Imaging Clinics of North America*.[28] First, they connect the reader to the utility of fat pads (versus bursa) by succinctly recounting the theoretical work of MacConaill in 1950[29]: "they not only occupy dead space in the joint but also help maintain the joint cavity and promote efficient lubrication by helping to distribute synovial fluid…explains the presence of fat pads in areas of the body exposed to mechanical stresses, such as the knee".[28, p. 725] More critically, they provide further imaging evidence of both normal and pathological adipose and synovial tissue in the lateral recesses of the knee, between the femur and the ITB, and specifically refer to an "impingement zone" in which maximum fat pad and synovial tissue impingement or compression occurs in the early stance phase of running, when the knee flexion angle approximates 30°. From 0–30° of flexion during the loading or deceleration phases of gait (stance), considerable eccentric energy and control is required to stabilize and normalize the forces and movements of the weight bearing lower extremity. This critical zone of stability requires strength, control, and endurance of muscles of the hip and thigh in order for reactive forces on the limb's inert tissue to be minimized. Incidentally, this critical zone mirrors quite well the trouble range or zone that Renne and Noble both described in their initial ITBFS models.

In the third and most recent study, a Korean group published a paper that pinpointed "intra-articular fibroma" in the tendon sheath as a culpable source of pain in ITBFS patients.[30] The authors presented a brief review of the rare condition (intra-articular fibromas) and a case involving a 45-year-old male athlete with recurrent lateral knee pain and a palpable nodule. MRI revealed a thickened ITB, fatty abnormalities deep to the ITB, and the presence of an abnormal nodule in the space between the ITB and the LFC. Arthroscopic resection revealed an inflamed lateral synovial recess and a whitish polypoid intra-articular nodule that was attached to the joint capsule, and histology studies

demonstrated marked hemorrhage, fibrosis, and prominent capillary proliferation in the resected fibrous nodule. Though fibromas of tendon sheaths are rare overall, they more typically develop in the fingers, hand, and wrist joint. Extra-articular fibromas in the knee are exceedingly rare however, (only 7 reported cases), but tendon sheath fibromas in the knee are the most common location for intra-articular lesions in 20–50-year-old males. When they do develop, it is easier to assess clinically than typical ITBFS because of the painless mass (31 %), sense of fullness, and mechanical symptoms that typically present as a result of these fibrotic neoplasm or reactive fibroses (in contrast to ITBFS without fibromas). Table 2 summarizes the "old" and the "new" anatomical and biomechanical evidence that is pertinent to the understanding of ITB-related pathology and dysfunction over time.

This new anatomical and biomechanical information begs for a new paradigm of thought and practice for IT band related knee pain. To be frank, Craig Denegar first made us aware of this paradigm change in 2010 as part of a case study presentation he delivered on a recalcitrant case of ITBFS in an elite female runner. Denegar's evidence-informed experience forced him to fundamentally reexamine the clinical paradigm or axiom from which he was initially working to address this common problem, and the net result of this presentation was that it forced clinicians in the audience to reconsider what they "thought they knew" about ITB-related pathology.[31] There is now a firm belief that the ITB cannot be stretched[32] per se because, as Fairclough states, "the fascia lata, the lateral intermuscular septum, and the distal fibrous bands anchoring the ITB to the femur would *all* need to be stretched for the ITB to be lengthened."[1] The 2006 research of Chaudhry et al. reinforces this notion with a three-dimensional mathematical model claiming that the forces required to produce a 1 % shear and compression in fascia lata are far beyond the physiologic range that manual therapy can induce, leading to the conclusion that the fascia lata remains very stiff under any shearing produced with stretching.[33] They further hypothesized that the anecdotal relaxation changes reported by therapists when massaging fascial tissue may be due to a stimulation of fascial mechanoreceptors that then leads to tonus changes in connected muscle fibers.

Table 2 Summary of Old vs. New Paradigm Clinical Components for ITB Pathology

Syndrome Component	ITB Friction ("Slipping Band")	ITB Impingement ("Compression")
Pathoetiology	Movable ITB, crosses over lateral femoral condyle with knee flexion/extension, especially at 30° of flexion	ITB does NOT move across the lateral femoral condyle, and thus cannot cause friction-related pathology; dynamic valgus collapse due to neuromuscular factors causes impingement of subtendinous tissue during repetitive weight bearing flexion/extension activities
Tissue & anatomy	Subtendinous bursa; ITB; ITB inserts on linea aspera and Gerdy's tubercle	Extra synovial pouch/recess; highly innervated fat pad; benign tendon sheath fibroma; ITB?; ITB has multiple and varied insertions on tibia, patella, lateral retinaculum, and LFE itself
Clinical presentation	History concurrent with ITB presentation, including pain over lateral femoral condyle, overuse, increased mileage, other modifiable risk factors common in runners; positive Ober's test; positive Noble's test	History concurrent with ITB presentation, including pain over lateral femoral condyle, overuse, increased mileage, other modifiable risk factors common in runners; inconclusive Ober's test; positive Noble's test
Treatment	Steroidal injections; stretching of ITB; deep tissue/friction massage; therapeutic modalities; activity modifications; surgery (recalcitrant cases)	Activity modifications; address modifiable risk factors; hip strengthening and neuromuscular training
Outcomes	Not supported	Supported

Abbreviation: ITB = iliotibial band.

Rather than the ITB being "stretched" over a subsiding bursa and slipping fore and aft over the lateral femoral condyle, the new theory put forth for those who suffer from pain and discomfort is that the ITB band *does move medially and laterally* due to the varying tensioning forces. These forces are enough to compress a highly vascularized and innervated fat pad that "works as a bursa would" and sits underneath the IT band and in effect prevents undue forces on vulnerable tissue (as MacConaill described and detailed prior). Because the fat pad between the ITT and the epicondyle is a richly vascularized and innervated connective tissue, it may contain pressure-sensitive Pacinian corpuscles, pain-sensitive nociceptors, and proprioceptive nerve endings that may, through neural feedback, play an important pathoetiological role in the perpetuation of ITB syndrome.[1] Grando emphasizes the importance and under appreciation of fat pads in and around the knee by summarily informing us that fat pads should be appreciated as more than passive, space occupying structures. Rather, fat pads of the knee are critical in that they protect the joint by remodeling their shape according to flexion angles and assist in preserving the various compartments by modulating direct contact. The richly innervated and vascularized fat pads in our knees function as "windows between the synovium and the capsular layers", and therefore "extrasynovial impingement and inflammation syndromes about the knee should be in the differential diagnosis for patients presenting with knee pain".[28, p. 739]

In addition, Vieira's anatomical work has expanded our notion of what the ITB "does" and thus has lent significant credence toward the idea that the ITB (or as he calls it, ITT) is essential to both static and dynamic stability of both the patellofemoral and knee joints. Because the ITB crosses both the hip and the knee joints, proper tensioning and function in the ITB is crucial for normal biomechanical function at each level.[26] Ward et al. recently wrote a very salient literature-review-based editorial suggesting a profound chemico-physical relationship between fat pads and tendon pain, adding some interesting insight to the controversy surrounding the true nature of common tendinopathies (inflammation or degeneration?) at the knee and ankle.[34] They propose that fat pads share an anatomic and functional relationship with adjacent tendons and shared vascularization, innervation, and further that the fat pad's production of inflammatory cytokines contribute to the development of clinical tendinopathies. In citing several histological and imaging findings, Ward and colleagues[34] link the size and location of fat pads around the patellofemoral joint and Achilles' tendon to inflammatory and vascular changes that effectively contribute to, or produce the perception or manifestation of "tendon pain". Hoffa's fat pad (knee) and Kager's fat pad (retro calcaneal) are known to produce greater inflammatory cytokines and to possess a complex network of surface vessels, and structurally they contact the under surface of the proximal tendons (patellar and Achilles', respectively). Together, these properties in effect provide extrinsic blood supply that at times may become the "root" for neovascularization of the tendon to occur as a tendinopathy develops. They term this neovascularization phenomenon as "parainflammation", operationally defined as "chronic, low-grade inflammation associated with repetitive tissue stress".[40] Given this biochemical evidence, Ward et al. hypothesize that "fat pads adjacent to common sites of tendinopathy are a key source of cytokines and influence the pathophysiology of tendinopathy via parainflammation pathways".[34, p. 1492]

Summarily, we now have compelling evidence that (a) the ITB is extremely functional and important to lower quarter mechanics; (b) the ITB has several, multifunctional insertions that contribute to patellofemoral and knee joint stability, including along the linea aspera itself; (c) ITB syndrome is related to compressive or impingement type forces (and not friction, slippage, or tightness of the ITB); (d) there is no subtendinous iliotibial bursa present; and (e) the noxious producing tissue is either a richly innervated fat pad or an extra synovial pouch, or some combination thereof. Given this plethora of new information from basic sciences (anatomy, physiology, and biomechanics), we have the underpinnings for a new working paradigm of ITB pathology for clinicians that treat runners, rowers, soldiers, cyclists, and other athletes with insidious onset, lateral knee pain, one specifically inspired by the intersection of Grando's extrasynovial and fat pad tissue imaging data and Denegar's paradigm change articulation–*ITB impingement syndrome*. Before we move on, however, we need to first consider the "how" and "why" impingement serves as an apt conceptual model for ITB pathology.

Biomechanics and Pathomechanics

If the ITB is not "slipping" over and back of the lateral femoral condyle at the 30° of flexion point with repetitive running-based activities, as Renne first proposed,

then why do some people experience insidious lateral knee pain in their knee, while like activities and volume are immune to it? What is the precise pathoetiology for this troublesome syndrome, and how can it be fixed or prevented? Why doesn't the standard treatment protocol of stretching and massage relieve the pain and discomfort associated with this type of lateral knee condition in runners?[3,32] If we accept Denegar's contention that "understanding pathology informs treatment", we must first understand how or why the synovial pouches and/or fat pads of some runners are impinged, yet are not so in others undertaking the same mode and volume of activity.[31] Perhaps the answer lies in a combination of information from Fairclough's proposed entrapment theory, Vieira's functional and anatomical elaboration, and numerous biomechanical investigations concerning the interconnection between hip musculature and knee function during closed chain activities.[1,26,31]

After debunking Renne's original "friction" or tissue slippage theory, Fairclough and colleagues were obligated to offer an alternative mechanical cause for the pain that ITBFS sufferers reported. If the ITB did not roll over the lateral femoral condyle, and if the subtendinous fat pad was being compressed (rather than a bursa), what was causing this compression in some athletes (but not others)? Pathomechanically, what exactly induces this pain in some people and not in others? Interestingly, Fairclough did not have to look very hard for an answer, because there were already at least two studies published by Fredericson and colleagues that offered an explanation.[2,35] Although he produced no original data of his own making to support his alternative theory, but based on Fredericson's prior work, Fairclough contended that "ITB syndrome is related to impaired function of the hip musculature" and that "resolution of the syndrome can only be properly achieved when the biomechanics of the hip muscle function are properly addressed."[1, p. 315] Not only did Fredericson find that long distance runners with ITBS had weaker hip abduction strength in their affected legs, but also that their conditions resolved and that they returned to preinjury training after achieving improvements in their hip abductor strength.[35] Although not a definitively established cause-effect relationship as of yet (Grau et al. reported in their 2008 study that hip abductor weakness did "not" factor into ITB syndrome[36]), there are indeed several studies documenting at least a role of the hip musculature in causing ITB impingement syndrome (ITBIS), including an award winning 2007 prospective paper in *Clinical Biomechanics* by Noehren, et al., in which they found ITB syndrome to be related to peak hip adduction and knee internal rotation moments, and that treatment interventions should focus on controlling these energy absorbing forces and planar movements during the stance phase of gait.[37]

Like many syndromes, ITB indeed seems to comply with the tenets of "systems theory" in that it likely involves many interconnected factors both etiologically and pathomechanically and, indeed, Louw and Deary's[16] systematic review on the etiology of ITB syndrome in runners revealed exactly this type of segmental interconnectedness or "regional interdependence". Their intensive review of 12 quality studies (out of 1,732 identified) revealed that runners suffering from ITB syndrome display decreased rear foot eversion, tibial internal rotation, and hip adduction angles at heel strike while having greater maximum internal rotation angles at the knee and decreased total abduction and adduction range of motion at the hip during the stance phase, concluding that a "clear biomechanical cause for ITBS could not be devised due to the lack of prospective research". The biomechanical work on ITB strain rate and the pathogenesis of ITBS by Hamill et al. lends further objective support to the notion that stretching a "tight" ITB does not help reduce pain (due to compression or impingement), and that increased dynamic valgus collapse of the knee joint during stance plays a role in the development of ITB syndrome because of the speed in which it induces strain on the ITB.[38] They found that "strain rate" (an indirect measure of tension in the collagen-based viscoelastic tissue) and not the absolute magnitude and increased adduction and internal rotation of the femur and tibia, respectively, were related to the presence of ITBS, especially at midsupport where the knee is flexed to its maximum range (in normal running). They thus concluded that since ITB syndrome subjects exhibited greater strain in their ITB throughout the support period, as compared with their control group, that strain *rate* is a major factor in the development of ITB syndrome.

Lastly, a thoughtful study by Miller et al. in 2007 sought to test the relation between hip musculature performance and ITBS by inducing fatigue in runners to see if their lower quarter mechanics changed as a result.[39] In their eight subjects (runners) with a history of ITB syndrome, an exhausting run caused them

to display increased knee flexion and tibial internal rotation angles at heel strike, and to produce higher ITB strain rates throughout the entire stance phase as compared with healthy control runners. Given that the functional utility of the stance phase of walking, running, and landing gait is to essentially absorb body weight and ground reaction forces through decelerative muscle action (eccentric contractions), exaggerated or excessive joint motions in the lower extremity indicate that the ITBs in susceptible runners' knees are in the "impingement zone" longer, and that endurance runs that fatigue the pelvic muscles further decrease the ability of these vulnerable tissues to effectively absorb the energy loads that are produced, creating a cyclical recipe for insidious onset pathology. In concluding that ITB syndrome pathomechanics appear to be related to changes in knee flexion at heel strike and internal rotation of the leg, the authors suggest that kinematic discriminators for the clinical assessment of ITB syndrome are supported by their findings.

One final overarching consideration is the recent acceptance of the biomechanical concept of tensegrity. The architectural theory of tensegrity was first introduced through the indirectly linked efforts of sculptor, Kenneth Snelson, and system theorist and architect, Buckminster Fuller. Based on the design concepts of Snelson's sculptures, Fuller developed architectural structures that were supported utilizing compression and tension elements, coining the term "tensegrity" in naming the unique designs.[40] As it relates to human form, the compression struts in a "tensegrity" design are represented by bones which in essence float within the structure, they are not continuous with each other and therefore do not transmit compression to adjacent bones. The tension elements are muscles, tendons, and, in essence, the fascia tissue itself, as they directly distribute their tension load to adjacent tension elements.[41] Biotensegrity, coined by Dr. Steven Levin in 1981 and further developed on a cellular level by Dr. Donald Ingber, builds upon Fuller's model and provides an explanation for how dynamic forces and cellular signaling are transmitted throughout the body, which is of utmost importance to address ITB issues from a holistic approach. Biotensegrity provides for not only a structural approach and understanding as to how force is transmitted through the three-dimensional construct of the constituent tissues of the body (the fascial tissue as defined by Guimberteau & Armstrong[42]) but, more importantly, how the body itself changes and adapts to stress through the "sensing" of tension, a process known as mechanotransduction. Adaptation is a product of biotensegrity and mechanotransduction as cells "sense" and/or message each other through the connection each cell has with each adjoining and connected cell.[43] The nucleus of the cells are signaled or stimulated to transcribe DNA leading to adaptations occurring in the tissue.

Once the concepts of biotensegrity and mechanotransduction are better understood, it becomes imperative to not just address the local tissues when evaluating a patient with complaints of lateral knee pain, but also to take multiple other structures into consideration. Biotensegrity dictates that tension and forces from adjacent and, quite plausibly, structures somewhat remote from the presenting dysfunction could be contributing to the dysfunction. In other words, restrictions in tissues away from the site of symptoms and/or dysfunction may actually be the root cause of the problem. In the case of ITBIS, a dysfunction in the thoracolumbar fascia (TLF) may be contributing and/or causing the symptoms being experienced in the lateral knee; this occurs due to the interconnectedness of all tissues in the body. Tension and strain are produced in a muscle or deep fascial tissue and the resulting force is transmitted through the myriad of fascial connections into adjacent tissues up and down the line from the point of origin. Therefore, if there is a restriction in any of the tissues along the line, the transmission of force will be altered leading to abnormal loading of said tissue or adjoining tissues ending in dysfunction and potential pathology. In the case of ITB tissue, pathology can lead to symptoms being experienced in the lateral aspect of the knee. Franklyn-Miller and his group performed a study on fresh cadavers investigating microstrain levels produced in various interconnected tissue groups when a straight leg raise is performed.[44] Their results strengthened the biotensegrity theory of transmission of strain to adjacent structures as there was over a 200% greater strain produced in the ITB, a 146% greater strain produced in the ipsilateral TLF, and a 102% and 100.6% greater strain produced in the lateral compartment of the lower leg and Achilles, respectively, than that produced in the posterior thigh. These results were found on nonliving tissue and if extrapolated to potential in-vivo findings, one can conclude that transmission of force will occur in an active setting as well, with one expecting to find significant, though perhaps lower strain, values, as

living muscle fibers would be able to absorb some of the force transmission. Therefore, ITBIS is likely to be a multifactoral issue and not just a problem in local tissue, so examining other related areas of the body for restrictions and or dysfunctions is indeed warranted.

An Evidence Informed Summary and Conclusion

Indeed, there is a need for high-quality prospective studies to test any new theory or paradigm of thought or axiom for clinical practice if we are to become and be an evidence-informed profession; in this case, there is certainly room for more objective and clear-cut evidence for resolving ITB- related pathology. But, given that there is very little to no evidence to support Renne's original "friction" theory for ITB pain and dysfunction, and very little credible and nonanecdotal evidence to support the way that many of us have been treating this difficult clinical problem in runners, cyclists, and rowers for years, there is certainly room for a new way of thinking and addressing ITB dysfunction if we hope to establish better patient outcomes in those that suffer. New evidence from our pillar sciences of anatomy, physiology, and biomechanics has emerged that directly challenges both the name and the nature of the paradigm many of us regularly use and refer to as iliotibial band friction syndrome. Considerable evidence now exists to help provide a scientific platform for a new and more informed paradigm for ITB-related pathology in running-based athletes. *Iliotibial band impingement syndrome*, or ITBIS, is proposed here as a more apropos paradigm because it is based on new information not only about the basic anatomy of the ITB and surrounding tissues, but also from physiological evidence that implicates extrasynovial tissue and fat pads as the pathologically insulted and painful sources, as well as biomechanical data that implicates neuromuscular dysfunction at the hip, and possibly tissue further away, as a primary etiological contributor to the syndrome (or cocontributor, at least).

Combining this science-based evidence with the clinical science data conducted on the condition in the laboratory provides a tenuous but plausible evidence-informed paradigm for insidious onset, lateral knee pain. Given this information, it is thus recommended that clinicians cease from attempting to stretch and massage the ITB, limit their use of modality application to the ITB itself, and, rather, begin to use a regional interdependence quality assessment approach to treating ITBIS—look at athletes' landing and stance mechanics, assess the biomechanics of femoral and tibial responses to loading, ascertain if athletes with ITB-related lateral knee pain have a pronounced or exaggerated "impingement" period or zone during early to midstance. If so, look up the chain and down the chain, assess and address tissue dysfunctions that can contribute to a change in force transmission as well as neuromuscular issues at the hip to assess and address biomechanical flaws in the foot and ankle, see what kind of outcomes are generated with this relatively common problem, and don't forget to document and share the results. ▮

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Paul R. Geisler and ***Todd Lazenby*** are with the Athletic Training Education Program in the Department of Exercise and Sport Sciences, Ithaca College, Ithaca, NY.

Luke Donovan, PhD, MEd, University of North Carolina at Charlotte, is the report editor for this article.