FOOT & ANKLE INTERNATIONAL

Cumulative Industrial Trauma as an Etiology of Seven Common Disorders in the Foot and Ankle: What Is the Evidence?

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ABSTRACT

The concept of cumulative industrial trauma as an etiology of orthopaedic disease has recently generated considerable attention in both the medical and legal communities. To clarify the current state of knowledge about the issue as applied to the foot and ankle, we critically reviewed the literature on the etiology of seven foot and ankle disorders commonly involved in compensation litigation in the practice of the senior author: hallux valgus, interdigital neuroma, tarsal tunnel syndrome, lesser toe deformity, heel pain, adult acquired flatfoot, and foot and ankle osteoarthritis. Koch's postulates were appropriately modified and used as a logistic framework to analyze the potential for cumulative industrial trauma to cause foot pathology.

In none of the disorders analyzed could cumulative industrial trauma reasonably satisfy even one of Koch's three postulates. We conclude there is currently no unequivocal literature support upon which to invoke cumulative industrial trauma as a clear etiology of these disorders of the adult foot and ankle. The superb evolutionary adaptation of the human foot to prolonged ambulation and the absence of industrial demands that significantly differ from this task likely account for this dramatically reduced vulnerability of the foot to industrial repetitive motion disorders compared to the upper extremity.

INTRODUCTION

Insidious work-related diseases originating from repetitive occupational exposure remained uncompensated until relatively recently. Over several decades in the first half of this century, epidemiologists studying the British mining industry established "coal-miners' lung" as the first common and widely compensated occupational disease related to a lifetime's accumulated exposure to dangerous working conditions.[95] Other celebrated and well-documented cumulative occupational diseases have followed, including lung cancer in uranium miners,[107] mesothelioma and asbestosis in shipyard workers,[20] multiple bone infarcts in caisson workers,[17] and "phossy jaw" in match factory workers.[17]

It is only recently, however, that both the medical community and the legal machinery of workers' compensation have taken up the issue of whether or not repetitive mechanical work alone can serve as the basis for musculoskeletal disorders. The term "overuse syndrome" was originally proposed by Fry in 1988 to explain symptoms of pain and weakness in the arms of musicians.[33] A variety of similar clinically imprecise terms have come into use in the legalistic world of workers' compensation, including "cumulative stress disorder," "repetitive stress injury," and "cumulative trauma disorder."

Interest in cumulative industrial trauma has to date been overwhelmingly focused on the upper extremity. Despite frequent warnings of caution in the orthopaedic literature,[84] statistics demonstrate the concept has met with a burst of outright surgical enthusiasm. Of the 240,000 carpal tunnel releases performed annually in the United States, 47% are considered to be work-related according to data from the Centers for Disease Control.[1] No large-scale extension of the concept of cumulative industrial trauma to the foot and ankle has yet been made. Of 31,000 claims of work-related cumulative trauma disorders recently made over a three-year period at a large automobile plant in Detroit, none were submitted involving the lower extremity.[103] Nevertheless, it is our experience that patients and their attorneys have increasingly begun to ask if their foot and ankle disorders could be related to their jobs, a concept perhaps now more familiar to the layman given its currency in the upper extremity. Similarly, employers and their workers' compensation carriers have become more interested in the potential of litigation on the subject. With this in mind, in this paper we critically analyze the available literature on the etiology of several com-

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mon foot and ankle disorders to determine if any scientifically rigorous connection to cumulative industrial trauma can be made.

METHODS

Standards of Proof

A dichotomy has grown between the standards of proof applied by the legal system to workers' compensation cases and those traditionally accepted by the scientific community. Workers' compensation in the United States is administered by the 50 states, and each has subtly different definitions of "injury." These are often terse or intentionally vague.

The existence of these loose and variable legal standards should not necessarily abrogate the responsibility of physicians to consider the more rigorous traditional medical standards of proof of disease causation. We can reasonably expect that the current legal standards will continue to apply in individual cases. However, linkages between the workplace environment and disease are also the appropriate foci of public policy, potential future legislation, and preventative medicine efforts. An understanding of how well these linkages hold up to scientific rather than legal scrutiny is vital.

With this premise in mind, we undertook a literature review to explore individually the etiology of seven common foot and ankle disorders with particular reference to cumulative industrial trauma. The seven disorders together comprise the majority of work-related claims seen in the practice of the senior author.

Although the literature on these disorders is abundant, it is immediately apparent that little reliable epidemiological information is available specific to the work environment. In the absence of sufficient data to use statistical techniques such as metaanalysis, we performed a subjective review by applying the information to the framework of Koch's postulates of the etiology of disease. For practicality, only reports which present data or conjecture relevant to the etiology of the diseases in question are referenced.

Koch's Postulates

Koch's postulates have served for over a century as the logistic basis for the rigorous proof of disease causation. They cannot always be satisfied within the limits of finances, technology, or ethics, but they continue to represent the ideal medical and scientific standards of proof. Although originally proposed for microbial infections, they can be generalized to analyze any proposed etiology of disease. We therefore applied them to each of several common foot and ankle disorders, using the available literature to determine if they were satisfied. Briefly stated, Koch's postulates are:

1. *The etiologic agent must be found in each case of the disease.* This condition of exclusivity is, in fact, rarely satisfied in occupational diseases. More commonly, the prevalence or incidence of a disease is found to be excessive in a class of workers. When this is the case, it is most appropriate to state that the occupational exposure is at best one of several etiologic factors.

2. *The etiologic agent must be able to be isolated.* Koch created this condition to literally mean that a microbe must be able to be grown in culture. Extending this concept to mechanical industrial exposures is challenging. If the disorder is not one that occurs exclusively in subjects with a known, obvious exposure, we must rely on statistical analysis to isolate the exposure from other potential etiologic factors. This is, unfortunately, a complicated affair that relies on statistical techniques such as analysis of variance or regression. To be interpretable, all statistical techniques require that any potentially confounding exposures be known. This is problematic. A laborer working a 40-hour week spends just under 25% of his or her time actually on the job; a patient's leisure activities, habits, and home environment make up a substantially larger percentage of the mechanical environment in which he lives than the workplace.
   In addition, even when the variables are known, confounding interactions between them can easily be overlooked if not carefully sought out.

3. *The etiologic agent should be able to cause disease if a normal human or model animal is exposed to it.* Satisfying this third postulate for occupational disease is obviously inherently difficult. Prospective studies of workers detailing their health before and after entering an occupation are exceedingly rare, and animal models of repetitive mechanical occupational tasks would be very difficult to validate.

Applying Koch's Postulates to Occupational Repetitive Trauma: a model disorder from the upper extremity

Upon careful review, Koch's postulates seem to represent a seemingly unachievable level of rigor to implicate something as nebulous as occupational mechanical exposure in orthopaedic disease. It is indeed reasonable to ask if "cumulative industrial trauma" can satisfy these exacting demands for any orthopaedic disorder: certainly many popular upper extremity diagnoses would come under suspicion if analyzed in this way. There are, however, isolated examples of disorders in

which cumulative industrial trauma can satisfy at least Koch's first two postulates without much debate. Of these, the hypothenar hammer syndrome is perhaps the most clear-cut, and it is useful to keep in mind as an example and model when we expand the analysis to foot and ankle pathology.

The hypothenar hammer syndrome is a rare vascular condition that occurs almost exclusively in jack-hammer operators and carpenters who habitually hammer objects using their hypothenar eminences.[124,54] With time, injury to the ulnar artery in the region of Guyon's canal can occur and, if the patient's radial artery is insufficient to reconstitute the entire palmar arch, digital ischemia on the ulnar aspect of the hand can result. The disorder can be objectively identified through angiography.

It is precisely the rarity of the condition that allows it to satisfy Koch's postulates. Aside from a handful of case reports in avid racquet sportsmen and cyclists,[98] the condition is confined to a small, well-defined group of laborers with similar exposures, thus satisfying Koch's first postulate. The mechanical stimulus of repetitively impacting the hypothenar eminence is so unique that it is easy to isolate it from the rest of the patients' mechanical environment, satisfying Koch's second postulate. As expected, prospectively satisfying the third postulate is problematic, but it is worth noting that in no reported cases have patients reported symptoms prior to beginning their occupation.

RESULTS

The Etiologies of Common Foot and Ankle Disorders

Adult Hallux Valgus

Perhaps more than any disorder of the foot, theories of the initial etiology of hallux valgus abound. Constrictive shoewear is regarded as the primary culprit, but it is appropriately regarded as a necessary but not sufficient condition for the development of the disorder.[87] Among the additional factors blamed in the literature are dynamic muscle imbalance about the joint,[42,43] hereditary or acquired metatarsus primus varus,[29,40,123,21,71] pes planus,[47,55] generalized ligamentous laxity,[94,16] attenuation of the medial sesamoid ligament,[26] and incompetency of the passive restraint offered by the cristae.[26] From a critical point of view, although each of these factors can be identified in at least some populations of patients with hallux valgus, it is impossible to determine if they caused the deformity or developed as a secondary result of it. To date, only retrospective studies are available. Although many opinions have been expressed, none can be backed with a prospective study following an at-risk population as a proportion of them develop the deformity.

There is, however, a common thread binding the various theories of hallux valgus. Essentially all authors note that footwear is a necessary, if not always sufficient, factor. A wealth of epidemiological evidence supports this. Prior to the 18th century, the prevailing style of footwear in the West was an accommodative flat-soled thong. With the subsequent widespread arrival of modern, more constrictive shoes the first mention of hallux valgus in the writings of a variety of French physicians began to appear.[87] Sim-Fook and Hodgson more quantitatively established the link in the1950's, demonstrating that 33% of shoe-wearing Chinese had at least mild hallux valgus compared to only 1.9% of those who habitually went barefoot.[118] Kato and Watanabe noted that hallux valgus was essentially unknown in Japan prior to the introduction of Western style shoes and operations for the deformity were rarities prior to the 1970's.[57] Their data demonstrate a correlation between the number of hallux valgus cases encountered at their institution with a rise in the numerical ratio of Western-style shoes to traditional clogs sold in Japan. Numerous ancient Japanese footprints from the period 6000 – 300 B.C. fail to demonstrate any evidence of the deformity. Studies of unshod populations by MacLennan in New Guinea,[86] Wells in South African,[127] Barnicot and Hardy in West Africa,[3] Engle and Morton in the Belgian Congo,[28] and James in the Solomon Islands[50] have all upheld the relative natural rarity of hallux valgus. In unshod populations the deformity is essentially a painless aberration uniformly accompanied by metatarsus primus varus, and the cases noted in these population studies may indeed represent juvenile rather than adult-acquired bunions.

Notably absent from the literature are any hints of occupational repetitive trauma beyond normal ambulation and footwear as a cause of hallux valgus. The notion is addressed only in a single study, a survey of foot deformities seen in general geriatric inpatients in a Hong Kong hospital ward by Hung et al.[46] They noted an overall 20% incidence of hallux valgus in their population, only a small percentage of which was symptomatic. They also recorded that 7% of their female and 20% of their male population had engaged in occupations requiring "heavy work" and an additional 19% and 26%, respectively, had "used their feet a lot." Using a 20° hallux valgus angle determined on a goniometer-assisted examination as their criterion, they determined 19% of their female and 22% of their male patients had the deformity. Significantly, there was no correlation between the presence of hallux valgus and the patients' work history.

Lesser Toe Deformities

Disorders of the lesser toes are among the most common of foot deformities. Much like hallux valgus, con-

strictive footwear is generally felt to be a major contributor to the development of the deformity in those cases that cannot be directly linked to a known motor neuropathy.[22,23] The toes must buckle to conform to a tight toe box when a constant deforming force is applied. Much like hallux valgus, lesser toe deformity is rarely encountered among unshod populations. Also like hallux valgus, there is a strong predilection for females (5.7:1),[15] and essentially all studies suggest that the deformities develop insidiously and their prevalence increases almost linearly with age.[112, 22, 111]

Several factors have been proposed as additional contributors to the deformity. Intrinsic denervation can certainly cause clawing through intrinsic contracture, but it is not the common thread behind all cases of the disorder. In a series of 68 patients requiring surgical correction, Taylor found no abnormalities of the intrinsic musculature upon gross and histologic examination.[121] The progressive and age-related attenuation of the passive plantar restraints of the metatarsophalangeal joints is likely the most important factor.[112] This may be accelerated by anatomic anomalies which overload the entire forefoot, such as equinous contracture,[111] or those which overload single joints, such as a congenitally long second metatarsal.[122] Dynamic forces may also come into play in cases of anterior tibial weakness in which the EDL is used as an accessory ankle dorsiflexor, causing a secondary dorsiflexion deformity of the MTP.[111]

No epidemiologic data link lesser toe deformities to industrial exposure, and, as in the case of hallux valgus, what little data do exist actually refute the concept. The incidence of lesser toe deformities is roughly the same in a 1938 study of English industrial workers by Lamberinudi[68] and in a survey of foot deformities in English military recruits drawn from all walks of life by Hewitt in 1954.[41] Multiple studies have shown the highest prevalence of the disorder to be not in the population of active industrial workers but in elderly women. In Hung's aforementioned survey of foot deformities among geriatric inpatients in Hong Kong, lesser toe deformities were present in 20% of the population.[46] Only 3% of the patients with deformities reported an occupation requiring "heavy work," a figure below that for the population as a whole.

Interdigital Neuroma

The original description of interdigital neuroma is usually attributed to Thomas Morton, who described the condition in a case series in 1876.[96] Most authors attribute the disorder to entrapment of the interdigital nerve as it passes under the transverse intermetatarsal ligament.[109,92,39] This is primarily a dynamic phenomenon; it likely occurs in terminal stance phase as the toes dorsiflex and stretch the nerve across the plantar surface of the ligament. The condition is aggravated by the use of shoes with a narrow forefoot, which provide a degree of medial-lateral compression as well, and by high heels, which force the toes into dorsiflexion throughout the entire gait cycle.

Histologic evidence supports the concept of a mechanical nerve entrapment as the primary etiology. Both light and electron microscopy of Morton's neuromas demonstrate characteristic changes similar to those seen in other peripheral nerve entrapments, including an increased width of perineurium, increased number of blood vessels per fascicle, demyelination, endoneurial edema, an absence of Wallerian degeneration, and the presence of Renaut's bodies.[73,38,117,39]

Two suggestions as to etiology are increased thickness of the nerve from a contribution of medial and lateral plantar nerves[10] or a smaller volume in the third interspace[81]. Neither of these has been fully proven.

Variant anatomy has been suggested as being involved in generating the disorder. Betts first suggested that the condition is more likely to affect unusually thick digital nerves, and he proposed the nerve to the third web space is commonly thicker than normal because it is made of contributions from the medial and lateral plantar nerves.[10] This has proven a matter of some debate. In a cadaveric study, Levitsky et al. found no difference in thickness between digital nerves formed by confluence of the medial and lateral plantar nerves and those that were not.[81] In fact, the third digital nerve contained contributions from both plantar nerves in only 27% of their specimens. They proposed as an alternative explanation of the predilection of the disorder for the second and third interspaces that the available volume of space between the metatarsal heads is smaller in these areas.

The intermetatarsophalangeal bursa has been suggested as an important factor by some authors[12], but the clinical significance of the bursa remains debatable.

Although most series indicate a preponderance of middle-aged women among patients with the disorder, no other major epidemiological associations have been made. There is no evidence that occupational exposures or mechanical trauma beyond normal ambulation are involved in the etiology of interdigital neuroma.

Tarsal Tunnel Syndrome

Tarsal tunnel syndrome is the homologue to the upper extremity disorder most commonly blamed on occupational cumulative trauma, carpal tunnel syndrome. Like most compressive neuropathies, tarsal tunnel syndrome was not described until relatively recently. Kopell and Thompson first described the condition in 1960,[64] and it was given its name in two independent reports by Keck[58] and Lam[66] in 1962. In most cases, the cause of

the disorder remains obscure.

Cimino recently conducted an extensive review of the literature on the subject and found 24 case series which reported operative findings or associated conditions.[18] These are most notable for their wide variety.[88,36,37] Many of the pathologies seen in association with tarsal tunnel syndrome can be linked to the disorder only by speculation, including hindfoot varus or valgus[27,106] and prior indirect or direct trauma to the foot.[27,67,83] More easily understood are several disorders leading to mass lesions within the tunnel, including ganglions,[93] lipomas,[101] peripheral nerve tumors,[52] venous varicosities,[36] and proliferative tenosynovitis in rheumatoid patients.[8]

Associations with industrial occupations have been proposed, but only on an anecdotal basis. Forst and Hryhorczuk did present a case of apparent tarsal tunnel syndrome in a sewing machine operator who operated a foot pedal for many years. Electrodiagnostic testing was not reported and the patient ultimately continued to be symptomatic despite two tarsal tunnel releases.[31] Lam addressed the issue in his case series, reporting there was "(no) significant common factor concerning occupation."[67] Overuse has been proposed as a possible etiology of tarsal tunnel syndrome among athletes and dancers, but even this data exists at the case report and small series level.[48,110] Lam cited a personal communication with Davies who relayed his belief that jockeys were at increased risk.[67] While anecdotal evidence of this kind can be valuable, there is no formal prevalence data documenting an increased risk for any occupation, athletic or industrial.

Heel Pain

Heel pain in the patient without rheumatologic disease likely represents a variety of subtly different disorders with similar manifestations. Most commonly, it has been attributed to chronic injury and repair resulting from repetitive microtrauma at the origin of the plantar fascia.[35,11,72,78,85,108,119,79,61,51] Additionally, many authors have proposed that subgroups of patients may have nerve entrapment syndromes, including the medial calcaneal nerve[116] and the nerve to the abductor digiti quinti.[5,6]

Objective evidence for both schools of thought exists. Indirect evidence of pathologic change does exist from ultrasonography and magnetic resonance imaging of patients with heel pain, which demonstrate thickening and signal heterogeneity of the plantar fascia, along with the absence of adjacent fluid collections.[56,126,9] Histologic examinations have demonstrated a chronic degenerative process including collagen necrosis and matrix calcification.[119] Some objective evidence for compressive neuropathy can also be found. Schon et al. demonstrated abnormal electrodiagnostic indices in 23 of 38 painful heels with neuritic radiation of the pain.[114] Some histology is also available; Baxter and Pfeffer reported electron microscopy findings consistent with compression neuropathy in two resected first branches of the lateral plantar nerve.[5]

Whatever the anatomic cause of heel pain, the salient issue for this review is whether or not cumulative trauma beyond normal ambulation can be implicated. This is probably plausible in the case of running athletes. Although not borne out by any rigorous data on the incidence of heel pain in running athletes versus a suitable control group, it does represent the clinical consensus in the literature.[7,78,85,119,79] Factors which increase the demand on the passive windlass mechanism (and therefore the plantar fascia) have been implicated as playing a role, including dynamic hyperpronation[7] and diminished extrinsic muscle strength.[61]

Although the high passive stresses and extremes of range of motion associated with running are likely implicated in heel pain, the evidence that the level of activity involved in the vast majority of industrial occupations increases relative risk for the disorder is sketchy at best. Lapidus and Guidotti reported a strong bias toward occupations that required standing and walking in their series of heel pain cases reported in 1965. Unfortunately, this data is rendered largely uninterpretable by the fact that well over half of their patients were drawn from a workers health clinic.[72] In 1996, Gill and Kiebzak reported the results of a survey given to a large population of patients with heel pain. They found a modest but statistically significant difference in the number of patients with heel pain who self-reported that their jobs required large amounts of walking and involved hard working surfaces compared to a control group of patients with other orthopaedic problems. These data, too, are difficult to interpret as they are not objective. The patients themselves, already suffering from heel pain, were asked to assess the demands of their job. It is not possible to separate out the actual working conditions from the patients' perceptions.

Studies also exist that imply negative correlations between heel pain and patients' occupations. Davis et al. reported on a series of 105 non-operatively treated patients of whom 103 were felt by the authors to have sedentary jobs upon chart review.[25] Williams listed the occupations of 44 patients he had seen for the problem, of whom well over half were either retired or had jobs requiring only moderate physical activity.[129]

Thus, what data exist are not only scant but also contradictory. Most studies on heel pain do not address the issue of patient occupation at all. Conclusive evidence for or against an association between heel pain and working conditions is not likely to come except in the form of a study designed specifically to address the question.

Adult Acquired Flatfoot

The linkage of acquired adult flatfoot and dysfunction of the posterior tibial tendon has been elucidated only over the last thirty years.[60] With the exception of arthritic, neuropathic, and traumatic variants, the etiology of the acquired adult flatfoot is, therefore, synonymous with the etiology of posterior tibial tendon dysfunction.

The disorder most commonly affects middle-aged females, but cases in males are not unusual.[34,89,90] A recent study by Holmes and Mann demonstrated an increased prevalence of hypertension and obesity among patients with the disorder compared with the general population.[44] Systemic inflammatory diseases have also been implicated, particularly HLA-B27 associated disorders.[99]

Anatomic factors may play a role. A hypovascular watershed area of approximately 14 mm in length located 40 mm proximal to the insertion of the tendon lies between the proximally and distally-based blood supplies,[32] although rupture commonly occurs distal to this. Increased stress distally from an accessory navicular has also been associated with tears identified by MRI.[115] Several authors have also suggested congenital pes planus (the flexible adolescent flat foot) may put an undue chronic stress on the posterior tibial tendon ulimately leading to pathology.[34,49,60,89,90,53]

No data exist which link repetitive mechanical exposures other than normal ambulation to posterior tibial tendon dysfunction and, by association, acquired adult flatfoot. Only one major study addresses extrinsic trauma as an etiologic factor at all; Holmes and Mann did collect data on isolated traumatic events involving the medial aspect of the ankle in their case series, but found it impossible to analyze in the absence of control data.[44]

Osteoarthritis

The etiology of osteoarthritis remains elusive and complex and has been the subject of a vast amount of research well summarized in other reviews.[45,105,97] Little epidemiologic attention has been focused specifically on osteoarthritis of the foot and ankle, so conclusions about the potential for an etiologic role for occupational exposures must be inferred from data about other joints.

Including mild forms, osteoarthritis of the foot and ankle is extremely common. By radiographic criteria, which naturally include a large number of asymptomatic cases, the National Health and Nutrition Examination II found the prevalence of osteoarthritis of the foot among Americans aged 75 to 79 to be 51%.[77] Unfortunately, beyond this simple prevalence statistic, there are no well-controlled data with which to address whether or not the mechanical environment has an influence on foot and ankle arthritis.

The linkages between mechanical factors and arthritis in other weight-bearing joints have been studied, however. A positive association between osteoarthritis of the knee and obesity has been demonstrated by several studies.[30,24] Some authors have suggested that this association may not be entirely due to increased stress across the joint alone; osteoarthritis of several non-weight-bearing joints has also been shown to be more prevalent in obese persons as well.[59]

Activity-related repetitive trauma has been examined as well. Several studies have found osteoarthritis of the knee to be no more prevalent among amateur runners than among control populations.[70,63,69,102] While carefully done, these studies have been criticized for their retrospective nature; runners who develop symptomatic arthritis are unlikely to continue to participate in the sport. Professional participation in athletic activities has been associated with an increased incidence of lower extremity arthritis, particularly soccer.[62,82,125]

Finally, some associations between osteoarthritis in weight-bearing joints and selected industrial occupations do indeed exist. Arthritis of the hips, knees, and shoulders has been found to be more common in miners than age-matched porters and clerks,[113] and dockworkers have been shown to have a higher prevalence of knee arthrosis than age-matched civil servants.[104] Caution is naturally required in extrapolating these results to the foot and ankle. The distinct mechanical demands placed on the knee may vary dramatically in industrial occupations from those required for ordinary ambulation. Indeed, repetitive occupational knee bending alone has been shown to be a significant contributor to arthrosis.[2]

Thus, the question "Do industrial occupations contribute to osteoarthritis of the foot and ankle?" proves unanswerable on the basis of the currently available literature. There are no data which currently prove they do, but the issue has not been carefully evaluated. The balance of evidence does suggest that mechanical factors such as obesity and participation in certain professional sports or industrial occupations may play a role in knee osteoarthritis, but the distinct mechanical environment of each of the lower extremity joints ultimately mandates that they be examined separately.

DISCUSSION

The Application of Koch's Postulates

"Occupational" cumulative trauma can be an all-encompassing term. There is certainly evidence that, for instance, dancers, military recruits, and professional athletes incur an occupational risk for foot and ankle disorders. Outside of these rarefied worlds, however, there is currently no data suggesting that repetitive

industrial trauma above and beyond normal shoewear and ambulation can satisfy even one of Koch's postulates of disease for any of the common foot and ankle disorders examined. (Table 1)

Consider, for instance, Koch's first postulate: the etiologic agent must be found in each case of the disorder. Even if we invoke the looser standard that the prevalence of the disease must be demonstrated to be excessive in a population, no irrefutable statistical evidence exists to establish this in any of the seven disorders considered.

Koch's second postulate, that the etiologic agent must be able to be "isolated," goes equally unsatisfied. Remember that the typical worker spends only about 25% of his or her time on the job during his or her working years, a figure that translates to less than 15% of that worker's lifetime when childhood and retirement are factored in. Unlike the upper extremity, the activities of the lower extremity for almost all jobs vary minimally from the everyday demands of ambulation. Any additional factors (the quality of the floor surface, steel-toed shoes, etc.) that might be proposed simply run headlong into the inescapable conclusion that they represent strikingly minor quantitative variances in the inexorable stresses of daily ambulation and shoewear. It is, therefore, impossible to find a suitable match in the foot to the example given earlier of hypothenar hammer syndrome, a rare disorder occurring only in subjects with a history of a unique mechanical stress. In fact, it is even impossible to find a correlate for the more tenuous link between repetitive trauma and carpal tunnel syndrome, in which the disorder may not be rare but at least the industrial stresses purported to contribute to it are not part of most people's everyday lives.

Table 1: Summary — Modified Koch's Postulates Applied to Cumulative Occupational Trauma as an Etiology of Common Foot and Ankle Disorders

	1st Postulate (Increased prevalence of the disorder in an industrial population)	2nd Postulate (Unusual mechanical stresses are isolatable in an associated population)	3rd Postulate (Prospective demonstration of the disorder in unaffected workers joining the occupation)
Hallux Valgus	No (some negative data)	No	No
Lesser Toe Deformity	No (some negative data)	No	No
Interdigital Neuroma	No	No	No
Tarsal Tunnel	No	No	No
Heel Pain	No	No	No
Acquired Flatfoot	No	No	No
Osteoarthritis	No*	No	No
Hypothenar Hammer (upper extremity model)	Yes (jackhammer operators, carpenters)	Yes (hypothenar eminence striking)	No

NOTE: "No" implies no supportive data exist in the literature. Specific notation is made where negative data does exist.

* Occupational roles have been documented for OA in other lower extremity weight-bearing joints, particularly in the knee. Occupational involvement in foot and ankle OA has not been studied.

Koch's third postulate, that the etiologic agent must be able to cause disease if a normal host is exposed to it, requires a prospective cohort study if it is to be rigorously met in the context of industrial disease. These simply do not exist for the foot and ankle disorders in question. Indeed, with such scant retrospective data to suggest the culpability of cumulative industrial trauma in these seven disorders, there is little chance the third postulate will soon be satisfied in any form.

The Resilient Foot

Is this paucity of supportive data simply a matter of not looking hard enough? It is true that little effort has been expended to date on the association of foot disorders with the workplace. Some of the disorders analyzed may indeed be ultimately demonstrated to have an association with high-demand jobs; heel pain and osteoarthritis appear to be the leading candidates. Still, the linkages cannot be supported based upon the literature available today.

Upon reflection, this striking absence of proof for cumulative industrial trauma in the etiology of foot and ankle disorders should offer little surprise. With the possible exception of the heart, the foot is perhaps the portion of the human anatomy most subject to repetitive and transient mechanical stress. It is supremely adapted to this role. The peak forces seen by the foot in normal gait are around 110% body weight, and each foot's stance phase represents approximately 62% of the gait cycle.[91] For each foot, this event is repeated almost 1500 times in walking just one mile. When jogging, an activity that the Masai and others in East Africa spend much of their lifetimes doing, the peak forces are up to 240% of body weight, and each foot's stance phase represents approximately 38% of the gait cycle. Thus, approximately twice the force is applied during half the time during jogging. This simple act therefore quadruples the peak impulses applied to the foot. Any proposed additional forces applied to a walking foot in the

industrial setting pale by comparison.

For the seven common disorders mentioned, there is currently no compelling evidence that the mechanical stresses in industrial occupations represent an undue threat to the foot; they simply represent another manifestation of the normal environment for which it is superbly adapted.

EDITORIAL FOOTNOTE:

It is known that occupational cumulative trauma is not a specific medical disorder. There are concerns that Koch's Postulates would not be appropriate to evaluate these. Cumulative trauma in this article is being evaluated as a proposed etiology and not a disorder in and of itself. Therefore, I feel it is reasonable to attempt to apply Koch's Postulates.

This format is also unusual in that a meta-analysis or statistical evaluation would not be appropriate. The subjective analysis in the paper seems to fit an accepted logical framework.

We publish the paper because there is an absence of hard epidemiological evidence in the literature to support cumulative trauma. This paper does not refute its existence; it merely suports the level of evidence for it. My sense is that this paper brings us away from some of the anecdotal aspects of the literature, but still does not completely support or deny the evidence that these disorders are related to cumulative trauma.

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