



QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

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DZIEDZIC, Aleksandra, SADURSKA, Gabriela, PSIUK, Marek, PRUSACZYK-ŚWIERZEWSKA, Aleksandra, HUNEK, Oliwia, WOLNY, Emilia, CHMIELOWSKI, Piotr, BARTOSIEWICZ, Martyna, BLAK, Patryk and SMĘDOWSKI, Igor. Mechanical and Friction-Induced Dermatoses in Athletes: Black Heel, Piezogenic Papules, and Related Traumatic Skin Conditions - a narrative review. *Quality in Sport*. 2026;77:76170. <https://doi.org/10.12775/QS.2026.77.76170>

ARTICLE TIMELINE

Received: 18.09.2026. Accepted: 01.10.2026. Published: 05.10.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych). © The Authors 2026.

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Mechanical and Friction-Induced Dermatoses in Athletes: Black Heel, Piezogenic Papules, and Related Traumatic Skin Conditions - a narrative review

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Abstract

Background. Repetitive mechanical loading, shear, and friction in sport produce a recognisable spectrum of cutaneous injuries frequently mistaken for infectious, neoplastic, or systemic disease. Black heel (talon noir), piezogenic pedal papules, blisters, calluses, corns, jogger's toe, and subungual haematoma are common but underreported, and their mimicry of acral melanoma creates diagnostic risk.

Aim. To synthesise the epidemiology, pathophysiology, diagnostic pitfalls, and management of mechanical and friction-induced dermatoses in athletes, with emphasis on black heel and piezogenic papules.

Materials and Methods. This narrative review draws on peer-reviewed epidemiological surveys, hospital and sports-cohort case series, dermatological case reports, a paediatric scoping review, biomechanical and finite-element modelling studies, and consensus reviews of sports dermatology, connective tissue disease, and skin barrier physiology.

Results. Friction-related lesions cluster on the feet, hands, and trunk and are associated with running distance, footwear, surface, and training volume. Talon noir and piezogenic papules are usually benign and self-limited, but can mimic acral lentiginous melanoma or signal connective tissue disorders such as Ehlers-Danlos syndrome, GATA2 deficiency, or joint hypermobility,

particularly when lesions are painful, atypical, or occur outside the expected age or activity context. Diagnostic accuracy improves with dermoscopy, scraping tests, and attention to bilateral distribution and exertion history. Management is predominantly conservative, though intralesional therapies exist for refractory painful piezogenic papules.

Conclusions. Awareness of the mechanical aetiology and differential diagnosis of these dermatoses can reduce unnecessary biopsies and psychological burden while ensuring that rare but important systemic associations are not overlooked.

Keywords: sports dermatology; talon noir; piezogenic pedal papules; friction blister; acral melanoma; connective tissue disorders; foot dermatoses; athletes

1. Introduction

Athletic activity subjects the integument to a distinctive combination of repetitive mechanical loading, shear stress, vibration, compression, and friction that is rarely replicated in sedentary life. Unlike the acute, single-event trauma familiar to general dermatology, sports-related mechanical injury to the skin is typically low-grade, cumulative, and site-specific, concentrated at points of maximal contact between the body and footwear, equipment, or playing surface [1]. Runners, in particular, generate thousands of repetitive loading cycles per session, and the resulting spectrum of traumatic and frictional dermatoses—blisters, calluses, corns, subungual haematomas, talon noir, and piezogenic papules—has been documented across virtually every running and racket-sport population studied to date [1-3].

Despite their frequency, these conditions occupy a curious position in the dermatological literature: they are simultaneously common and underappreciated. Athletes and coaches often regard blisters, calluses, and black heel as trivial inconveniences of training rather than as medical findings warranting assessment, while physicians unfamiliar with sports dermatology may misinterpret pigmented or nodular lesions on the foot as suspicious for melanoma, triggering unnecessary biopsy, anxiety, and cost [4,5]. The differential diagnosis of acral pigmented lesions is particularly fraught, since talon noir and subungual haematoma can closely resemble acral lentiginous melanoma both clinically and, in some cases, dermoscopically [4,5]. Conversely, piezogenic pedal papules—herniations of subcutaneous fat through the dermis of the heel—are usually asymptomatic incidental findings but have been linked, in some reported cases, to

systemic connective tissue disease, raising the question of when a seemingly banal foot lesion should prompt broader clinical evaluation [6-11].

This review adopts a mechanistic and comparative framework, situating black heel and piezogenic papules within the broader family of mechanical and friction-induced dermatoses that also includes blisters, calluses, corns, jogger's toe, athlete's nodules, and runner's rump. Because these conditions share overlapping pathophysiology—shear-induced tissue separation, chronic pressure-induced hyperkeratosis, or herniation through weakened connective tissue planes—understanding one condition illuminates the others. The review further considers the extent to which mechanical dermatoses reveal, or are modulated by, underlying connective tissue laxity, drawing on paediatric and adult hypermobility literature, case reports of Ehlers-Danlos syndrome, and a large cohort study of GATA2 deficiency in which piezogenic papules featured as an unexpected rheumatological manifestation [9-11]. Comparative attention is also given to biomechanical and finite-element modelling work exploring how foot strike location, joint laxity, and surface friction influence the ligamentous strain implicated in overuse injury, providing a mechanistic bridge between epidemiological description and injury prevention [12].

The purpose of this narrative review is threefold: first, to characterise the clinical presentation, epidemiology, and pathophysiology of black heel, piezogenic papules, and related mechanical dermatoses in athletes; second, to critically appraise the diagnostic pitfalls that arise from their resemblance to more serious or systemic disease; and third, to synthesise available evidence on prevention and management, while explicitly identifying where the evidence base is limited to case reports, small case series, or single-institution cohorts rather than large controlled studies. Throughout, the review adopts a comparative stance, weighing findings from different sports, populations, and study designs against one another rather than treating each report as an isolated curiosity.

2. Materials and Methods

This article is a narrative review rather than a systematic review or meta-analysis; no review protocol was registered, and no formal quantitative synthesis or risk-of-bias appraisal was undertaken. The literature considered comprised peer-reviewed, predominantly English-language publications addressing mechanical and friction-induced dermatoses in athletes and related populations, including epidemiological and cross-sectional surveys of runners and other sports

participants, hospital- and clinic-based case series, individual case reports and small case series describing talon noir, piezogenic pedal papules, and related conditions, a scoping review of paediatric hypermobility-associated skin findings, a large retrospective cohort study of rheumatological manifestations of GATA2 deficiency, biomechanical and finite-element simulation studies of foot-strike mechanics, and narrative reviews and consensus statements addressing sports dermatology, skin barrier physiology, fascial tissue biology, and tendinopathy pathogenesis. Sources were grouped thematically into sections addressing general mechanisms of friction- and pressure-induced skin injury; the clinical and diagnostic features of talon noir; the clinical features, epidemiology, and systemic associations of piezogenic pedal papules; other traumatic dermatoses of the foot and trunk (blisters, calluses, corns, jogger's toe, subungual haematoma, athlete's nodules, runner's rump); site-specific and sport-specific patterns of injury; biomechanical determinants of mechanical skin and connective tissue strain; and prevention and management strategies. Because selection and interpretation were, by design, narrative rather than systematic, the synthesis may be subject to selection and emphasis bias, and conclusions should be read as a clinically oriented overview rather than a formally graded account of all available evidence.

3. General Mechanisms of Friction- and Pressure-Induced Skin Injury

The skin's response to repetitive mechanical stress follows a limited number of stereotyped patterns regardless of the specific sport or anatomical site involved. Four broad mechanisms recur throughout the sports dermatology literature: shear-induced intraepidermal or subepidermal splitting (the basis of blisters), chronic pressure-induced epidermal thickening (calluses and corns), pressure-induced intraepidermal or intracorneal haemorrhage (talon noir and subungual haematoma), and herniation of subcutaneous tissue through a mechanically stressed dermal or fascial plane (piezogenic papules and related nodular conditions) [1-3].

Friction blisters arise when repetitive shear forces—rather than simple surface rubbing—cause mechanical separation within the epidermis, most often at the level of the stratum spinosum, with subsequent accumulation of fluid or blood within the resulting cavity [1]. A comprehensive narrative review of long-distance runners' skin problems described this mechanism explicitly, noting that hot and humid conditions inside footwear, together with ill-fitting shoes, promote blister formation, and that blisters can even develop beneath pre-existing calluses [1]. This same

review catalogued the frequent coexistence of blisters, calluses, corns, subungual haematoma, jogger's toe, talon noir, runner's rump, and athlete's nodules as a recognisable syndrome of overuse dermatoses in runners, an observation echoed in subsequent sports dermatology reviews describing dermatoses caused by mechanical factors as a distinct aetiological category alongside fungal, bacterial, viral, and environmentally induced skin disease [1,2]. A more recent narrative review addressing dermatoses associated with clothing, contact, and mechanical stress in runners similarly organised trauma-related conditions—blisters, calluses, corns, subungual haematoma, piezogenic pedal papules, jogger's toe, runner's rump, and talon noir—into a single conceptual framework of friction- and pressure-related injury, reinforcing that these conditions share a common biomechanical origin even though their clinical morphology differs markedly [13].

Calluses and corns represent the skin's adaptive, rather than acutely injurious, response to chronic mechanical stress: localised epidermal thickening that serves a protective function under normal circumstances but which can itself become a source of discomfort or a nidus for secondary blister formation when hyperkeratosis is excessive or unevenly distributed [1,2]. Punctate hyperkeratosis, or corns, typically develop at pressure points over bony prominences, most commonly the toes or the plantar aspect of the foot overlying the distal metatarsal heads, and are frequently associated with skeletal foot deformities or poorly fitting footwear [1]. The distinction between calluses, corns, and viral warts is clinically important, since warts can mimic hyperkeratotic lesions but are distinguished by the presence of thrombosed capillaries visible as small black puncta within the lesion, a feature absent in calluses and corns [1,2].

A third mechanism—pressure- or shear-induced intraepidermal haemorrhage—underlies talon noir (discussed in detail in Section 4) and subungual haematoma. Subungual haematoma results from acute blunt trauma to the toe, most often the second toe in runners subjected to repetitive downhill impact within a poorly fitted toe box, and presents with blood accumulation beneath the nail plate; drainage by hot-wire puncture or needle insertion at the hyponychium can relieve the associated pain [1]. Differential diagnosis is again critical here, as subungual haematoma can be confused with sport-related nail dystrophies, subungual melanocytic naevi, and, rarely, subungual melanoma, such that biopsy becomes necessary when the diagnosis remains uncertain [1].

The fourth mechanism, fat herniation through a mechanically weakened connective tissue plane, is the pathophysiological basis of piezogenic pedal papules and is discussed extensively in

Section 5. What unites all four mechanisms is their dependence on cumulative, activity-specific mechanical load rather than a single traumatic event, which explains why these conditions cluster in athletes with high training volumes and why their prevalence tends to scale with distance run, frequency of training, or duration of standing and weight-bearing activity [1,3,14].

4. Talon Noir (Black Heel): Clinical Features and Diagnostic Challenge

4.1. Definition, terminology, and clinical presentation

Talon noir, also known as black heel, calcaneal petechiae, subcorneal haematoma, intracorneal haematoma, hyperkeratosis haemorrhagica, or purpura traumatica pedis, was first described in 1961 as asymptomatic, bilateral, deep-seated petechiae on the posterolateral aspects of the heels of basketball players [4]. The condition is characterised histologically by intraepidermal haemorrhage and fluid collection secondary to shearing forces acting on an area of recurrent or sudden trauma, and it is most commonly observed in young athletes and, less frequently, in individuals such as mountain climbers who subject their feet or hands to repetitive shear [4,5].

Clinically, talon noir presents as asymptomatic, asymmetrical, black-to-brown macules, either isolated or in clusters, with an uninterrupted normal skin surface texture—an important distinguishing feature from melanocytic or verrucous lesions [5]. A systematic case-report review covering the period 2012 to 2021 identified 51 published cases of talon noir or black heel in the English-language literature, most occurring on the heel, sole, toes, or palm of adolescents and young adults with a documented history of sports participation, repeated trauma, or mountain climbing [4]. The largest single series within this review, a cohort of 18 patients with a mean age of 32 ± 17.5 years (14 male, 4 female), described lesions varying in size, shape, and colour: black in 44.4% of cases, red-black in 33.3%, and brown in the remaining 22.2%, with eight lesions (44.4%) showing surrounding dark satellite dots [4]. Notably, three of these 18 patients had a documented history of trauma and seven reported playing football, while the remaining eight had no remarkable preceding history, illustrating that talon noir can arise without an easily elicited history of acute injury, which complicates diagnosis when clinicians rely solely on trauma history to exclude melanoma [4].

4.2. Pathogenesis

The pathogenesis of talon noir involves damage to papillary dermal blood vessels secondary to shearing forces related to sports activity, climbing, or any form of repetitive microtrauma,

resulting in leakage of blood from the dermis into the epidermis [4]. Histopathological examination in a representative case—a 49-year-old woman with two irregular, thickened, blackish-brown plaques on the plantar aspect of the right forefoot—demonstrated severe hyperkeratosis, parakeratosis, and acanthosis, with multiple foci of acute inflammatory exudate and degenerated red cells within the epidermis, and large areas of haemorrhage in the dermal papillae, with no evidence of melanoma on histology [4]. This case is instructive precisely because it deviated from the classic sporting context: the patient was a radio announcer and social activist with no history of sports activity, whose lesions were instead attributed to high-heeled shoes, long-distance walking, and uncontrolled diabetes mellitus, illustrating that although talon noir is overwhelmingly an athletic phenomenon, occupational and gait-related mechanical stress can produce an identical lesion in non-athletes [4]. The authors of this case report noted that no prior literature had established diabetes mellitus as a direct risk factor for talon noir, and they explicitly called for further exploration of this possible association rather than asserting causation, a cautious stance that this review adopts as well given the paucity of comparable cases [4].

4.3. Differential diagnosis and dermoscopic distinction from acral melanoma

The central clinical challenge posed by talon noir is its resemblance to acral lentiginous melanoma, a distinction with obvious prognostic stakes. Differential diagnoses for talon noir include acral melanoma, acral naevi, plantar wart, pyogenic granuloma, angiokeratoma, corn, and tinea nigra [4]. A plantar wart typically has a verrucous surface with disrupted skin lines, while tinea nigra—a superficial fungal infection producing melanin-like pigmentation—can be readily distinguished using a potassium hydroxide scraping test that reveals fungal hyphae and spores [4].

Dermoscopy has emerged as a valuable non-invasive tool for differentiating talon noir from melanoma preoperatively, since talon noir characteristically shows homogeneous reddish globular structures, whereas acral melanoma typically demonstrates a parallel-ridge pattern with irregular pigmentation [4]. However, this distinction is not absolute: several studies have reported that dermoscopic features can be misleading, particularly in long-standing lesions, and that scraping tests and biopsy remain the preferred methods for definitive differentiation in ambiguous cases [4]. This point is corroborated by a separate case report describing a patient in

whom dermoscopy of a lesion on the second toe revealed uniform brownish-black pigmentation of the nail bed, involving the eponychium, lunula (suggestive of a positive Hutchinson sign), and lateral nail folds, with pigmentation distributed prevalently along the ridges of the hyponychium in a parallel-ridge pattern—dermoscopic features that, taken in isolation, raised a strong differential diagnosis of acral lentiginous melanoma [5]. Only the discovery of similar, though less pronounced, bilateral lesions on the contralateral foot, combined with a history of two recent marathons, prompted partial scraping of the lesion, which confirmed the clinically suspected diagnosis of purpura traumatica pedis rather than melanoma [5]. This case underscores a recurring theme in the literature: bilateral or multifocal distribution of acral pigmented lesions, combined with a history of physical exertion, should heighten suspicion for a traumatic rather than neoplastic aetiology, and scraping or paring of the lesion—which leads to partial or complete loss of pigmentation in talon noir but leaves melanocytic lesions unchanged—provides a simple, minimally invasive means of confirming the diagnosis [4,5].

A tabulated review of talon noir cases published between 2012 and 2021 (Table 1) illustrates the heterogeneity of demographic profile, anatomical location, dermoscopic pattern, and management approach across reported cases, and highlights that scraping or paring was the most frequently employed diagnostic and, in several cases, curative manoeuvre.

Table 1. Representative case series of talon noir (black heel) reported in the literature, 2012–2023.

Source [ref]	Number of cases	Population / context	Location	Dermoscopy findings	Management
Nassar et al., cited in [4]	18	Mean age 32 ± 17.5 years; 14 male, 4 female; history of trauma (3), football (7), no remarkable	Sole of foot (17), palm (1)	Homogeneous (55.6%), globular (44.4%), parallel-ridge (27.8%), negative pseudo-network	Paring performed for 17 lesions; one resolved with fingernail scratching

		history (8)		(16.6%)	
Khurana, cited in [4]	1	75-year-old female, housewife, controlled diabetes, no trauma history	Right heel	Homogeneous globular pattern	Paring of lesion
Goggins et al., cited in [4]	1	16-year-old male, hockey and baseball player	Palm	Parallel-ridge pattern	Paring of lesion
Elmas and Akdeniz, cited in [4]	20	Mean age 41.4 years; 14 male, 6 female; trauma history (4), anticoagulant therapy (2)	Soles (60%), palms (25%), volar hand (15%)	Homogeneous (65%), globular (55%), parallel-ridge (40%)	Scratch test with scalpel for complete pigment clearance
Uslu et al., cited in [4]	1	67-year-old female, no trauma, new shoe, blister, or exercise history	Plantar right toe	Parallel-ridge pattern	Excision
Choudhury and Mandal (index case) [4]	1	49-year-old female, radio announcer, no sports history; high	Plantar right forefoot	Not performed (unavailable)	Excision biopsy confirming diagnosis

		heels, long-distance walking, uncontrolled diabetes			
Feci et al. (index case) [5]	1	50-year-old male marathon runner (two recent marathons)	Second toe of right foot, bilateral lesser lesions	Parallel-ridge pattern (uniform brownish-black pigmentation of nail bed)	Partial scraping confirming diagnosis

As Table 1 illustrates, the demographic range of reported talon noir spans adolescence to older adulthood, and while the majority of cases occur in the context of athletic activity, a meaningful minority arise in the absence of any clearly elicited trauma or sporting history, reinforcing the need for a low threshold to consider talon noir in the differential diagnosis of acral pigmented lesions regardless of stated activity level [4,5].

4.4. Natural history and management

Talon noir is fundamentally a self-limiting, benign condition. Management typically involves reassurance, avoidance of the causative sporting or occupational activity, use of well-cushioned shoes, thick socks, or skin lubrication, with resolution generally occurring within a few weeks [4]. Vitamin C supplementation has been suggested as potentially beneficial in cases associated with exanthema-related capillary fragility, though this recommendation derives from limited case-level evidence rather than controlled trials [4]. Overall, the literature converges on a consistent message: the greatest clinical value in managing talon noir lies not in specific therapy, which is rarely needed, but in accurate diagnosis that spares patients from unnecessary anxiety, biopsy, or costly investigation for suspected melanoma [4,5].

5. Piezogenic Pedal Papules: Clinical Features, Epidemiology, and Pathogenesis

5.1. Definition and clinical presentation

Piezogenic pedal papules (PPP) are soft, skin-coloured or yellowish herniations of subcutaneous fat through the dermis, occurring typically on the medial, lateral, or posterior aspect of the heel, that become more prominent with weight-bearing and disappear or diminish when the weight is taken off the foot [15-17]. The term "piezogenic" derives from the Greek "piesis," meaning pressure, reflecting the fact that continuous or repetitive pressure on the heel—from factors such as excessive weight, jumping, running, or carrying heavy loads—is understood to be the primary precipitant [15,16]. Similar papules can occur on the lateral edge of the hand and wrist under analogous pressure conditions [16].

Most piezogenic papules are asymptomatic incidental findings discovered on physical examination or self-noticed by patients standing or walking [16,18]. In a minority of cases, however, papules become painful, with pain typically attributed to ischaemia of the entrapped neurovascular bundle and its accompanying nerve fibres as fat and vessels are compressed within the herniation, together with consequent thickening of the papillary dermis [6,15,16,18]. Painful piezogenic papules are reported disproportionately in women, in obese individuals, and in the context of repetitive occupational or sporting exposure such as marathon running, and they can meaningfully limit patients' recreational or occupational activities, thereby constituting the principal indication for treatment [6,15].

5.2. Epidemiology and prevalence estimates

The reported prevalence of piezogenic pedal papules varies enormously across studies, a heterogeneity that itself deserves explicit comment rather than simple averaging. A cases series of four patients presenting with painful papules noted that prevalence estimates in the literature commonly range from 10% to 20%, but that a study of 100 patients found papules in 80 of them, indicating that true prevalence may be considerably higher than earlier estimates suggested [6]. This wide range likely reflects differences in study population (athletes versus general medical outpatients), examination technique (systematic weight-bearing assessment versus incidental discovery), and age distribution of the sampled cohort [6,17].

More recent paediatric-focused evidence supports the view that piezogenic papules are, in fact, a very common and largely asymptomatic finding rather than a rare curiosity. A scoping review

examining the prevalence of piezogenic papules in healthy children and adolescents identified nine observational studies meeting inclusion criteria, of which four reported prevalence data across larger populations [17]. Among two cross-sectional studies focusing exclusively on children, reported prevalence ranged from 16% to 72%: one Polish cross-sectional study of 50 children (age 1–10 years, members of a sports club) found piezogenic papules in eight of 50 children (16%), predominantly girls (75%), non-painful, with two of the eight having a past history of foot injury and two having relatives with connective tissue disease, while a Dutch cross-sectional study of 322 primary-school children (age 4–13 years) found that 232 of 322 children (72%) had one or more papules, with an average of five papules per child and a mean diameter of 3.3 mm, none of which were painful [17]. Notably, in the Dutch study, 14 children were classified as hypermobile (4.3% of the sample), and the same proportion of hypermobile and non-hypermobile children had papules, leading the authors to conclude that they could not confirm that connective tissue fragility increased the number or size of papules in this cohort—an important negative finding that tempers, without entirely refuting, the hypothesis that generalized joint hypermobility predisposes to piezogenic papules [17].

The scoping review also incorporated previously unpublished empirical data from 84 adolescents aged 14–17 years assessed using the 2017 diagnostic criteria for hypermobile Ehlers-Danlos syndrome (hEDS), comprising 37 adolescents with hypermobility spectrum disorder (HSD) or hEDS and 47 healthy controls [17]. In this cohort, 28 of 37 participants (76%) in the HSD/hEDS group had piezogenic papules, compared with 28 of 47 (60%) in the control group—a prevalence difference of 16 percentage points (95% confidence interval –4 to 36; $p=0.163$), which did not reach statistical significance [17]. However, among those with papules, the HSD/hEDS group showed a higher proportion of moderate-to-severe papules (32% moderate, 3% severe) compared with controls (13% moderate, 0% severe), and none of the papules in either group were painful [17]. The authors concluded that while overall prevalence did not differ significantly between groups, adolescents with HSD/hEDS exhibited a higher frequency of more pronounced papules, and they explicitly cautioned that the limited discriminatory value of piezogenic papules calls into question their usefulness as an isolated diagnostic marker in paediatric populations [17]. This nuanced conclusion is important: it neither dismisses nor overstates the association between piezogenic papules and connective tissue hypermobility, instead calling for further prospective research to clarify severity thresholds and the nature of any associated pain.

Adult-focused literature broadly corroborates a high background prevalence. One case-series report noted that piezogenic papules are common in healthy subjects, with roughly 90% of affected individuals experiencing no symptoms at all, and that pain in the remaining minority most likely reflects ischaemia-related nerve entrapment [18]. Another report similarly stated that a population-based study identified a prevalence of piezogenic papules of 2.5%, with all of these cases associated with intense physical activity—a figure considerably lower than the paediatric estimates discussed above, again illustrating how methodology and population selection materially affect reported prevalence [18]. Table 2 consolidates the principal prevalence estimates identified in the reviewed literature, alongside population characteristics, to make these discrepancies explicit rather than obscuring them through a single pooled figure.

Table 2. Reported prevalence of piezogenic pedal papules across studies included in this review.

Source [ref]	Population	Sample size	Reported prevalence	Key associated findings
Brzezinski, cited in [17]	Children aged 1–10, sports club members	50	16%	75% female; non-painful; 2/8 had past foot injury; 2/8 had relatives with connective tissue disease
Van Straaten, cited in [17]	Primary-school children aged 4–13	322	72%	Average 5 papules per child; non-painful; no difference between hypermobile and non-hypermobile children
Zaidi, cited in	Outpatients aged	100	80% (pedal);	Non-painful;

[17] (mixed-age, cited with caution)	11–60, no vigorous activity or collagen disease history		86% (wrist)	74% had both pedal and wrist papules
Plewig, cited in [17] (mixed-age, cited with caution)	"Skin-healthy" out- and inpatients aged 2–86	>100	~20% across all age groups	Less frequent in children than adults; non-painful
Schubert-Hjalmarsson and Söderpalm (empirical cohort) [17]	Adolescents aged 14–17 with HSD/hEDS vs. healthy controls	84 (37 HSD/hEDS, 47 controls)	76% (HSD/hEDS) vs. 60% (controls)	Prevalence difference not statistically significant; HSD/hEDS group had more moderate–severe papules; none painful
de Oliveira Rocha et al. (case) [18]	Population-based estimate cited in case report	Not specified	2.5%	All cases associated with intense physical activity
Kahana et al., cited in [6]	General literature estimate	Not specified	10–20%	Cited as prior consensus range before later higher estimates

As Table 2 demonstrates, prevalence estimates for piezogenic papules span roughly an order of magnitude, from 2.5% to 80%, depending on population age, examination method, and study design; this heterogeneity should temper any single-figure claim about how common the condition truly is, and instead supports the more defensible conclusion that piezogenic papules are frequent, generally benign, and often underreported because they are asymptomatic.

5.3. Pathogenesis and histopathology

The histopathological hallmark of piezogenic pedal papules is fragmentation of dermal elastic tissue accompanied by herniation of subcutaneous adipose tissue into the dermis [6,16,18]. Biopsy specimens, when obtained, demonstrate thickened dermis with loss of the typical compartmentalisation of fat lobules and thinning of subcutaneous fibrous trabeculae, permitting infiltration of fat into the upper dermal layers [9,18]. In one representative case, however, histopathology of an excised papule showed only thickened stratum corneum (hyperkeratosis) rather than the more classically described dermal fibrosis and fat herniation, which the authors attributed to the superficial nature of the biopsy sample rather than a departure from the expected pathogenesis, and they emphasised that histological findings, while corroborative, are not required for diagnosis [18].

Constitutive fragility of connective tissue is hypothesised to permit fat penetration into upper skin layers, which would account for the reported association between piezogenic papules and congenital defects of collagen such as Ehlers-Danlos syndrome [9,18]. This mechanistic hypothesis is reinforced by observations in patients with hypermobile Ehlers-Danlos syndrome (hEDS), in whom piezogenic papules are described as one of several dermatological manifestations alongside soft, velvety skin, atrophic scarring, easy bruising, and cutaneous hyperextensibility [9]. A review of the dermatological aspects of hEDS in women described piezogenic papules as benign herniations of elastic tissue and subcutaneous fat breaking through the reticular dermis, exacerbated by compressive forces such as prolonged standing, and noted that tears in the plantar fascia are thought to create the anatomical opening through which subcutaneous fat can seep [9]. The same review observed that papules are found most commonly along the medial surface of the heel, where the body bears the greatest proportion of its weight, and that in patients with hEDS, painful papules are more common, plausibly reflecting the combined effect of inherent connective tissue weakness and impaired wound-healing capacity that is characteristic of the syndrome [9].

5.4. Systemic and syndromic associations

Beyond Ehlers-Danlos syndrome, piezogenic pedal papules have been reported in association with a diverse array of other conditions, several of which point toward shared connective tissue or, less expectedly, cardiac or haematological pathology. A case report described piezogenic

papules occurring together with muscle herniation of the extremities in a 54-year-old woman with sarcoidosis, the second such reported coexistence of these two posture-dependent herniation phenomena in the literature, with the authors proposing that both conditions may represent related manifestations of an underlying connective tissue or mesodermal insufficiency in this patient, given her otherwise unremarkable history of sedentary living and absence of athletic activity [7]. This case is notable for illustrating that piezogenic papules and muscle herniation, both fundamentally pressure- or posture-dependent phenomena driven by weakness of overlying fascial or dermal tissue, can co-occur in a single patient with a systemic granulomatous disease, raising the possibility of a shared connective tissue vulnerability, although the authors were appropriately circumspect in framing this as a hypothesis rather than an established mechanism [7].

Cardiac associations have also been documented, albeit in isolated case reports rather than systematic cohorts. Two cases of piezogenic pedal papules co-occurring with mitral valve prolapse (MVP) were described, the first in a 35-year-old male with asymptomatic left-foot papules and a late systolic murmur with mid-systolic click on auscultation, subsequently confirmed as MVP with mild mitral regurgitation on echocardiography, and the second in a 32-year-old female with pre-existing MVP who was found to have bilateral wrist papules on examination for unrelated palpitations [6]. The authors of this report noted that MVP, like piezogenic papules, may be isolated or part of a heritable connective tissue disorder, and they proposed that the higher prevalence of both conditions in certain populations might support a shared connective tissue basis, while explicitly acknowledging that the co-occurrence could equally represent coincidence given the relatively high background prevalence of both MVP and piezogenic papules in the general population [6]. They recommended that patients presenting with piezogenic papules undergo a detailed systemic evaluation, including cardiac examination, rather than dismissing the finding as an isolated cosmetic curiosity [6].

Perhaps the most striking and best-documented systemic association identified in the reviewed literature comes from a large retrospective cohort study of 157 patients with GATA2 deficiency—a rare genetic disorder of haematopoietic transcription factor function associated with monocytopenia, myelodysplasia, and non-tuberculous mycobacterial infection—in which piezogenic pedal papules emerged as a previously unreported rheumatological manifestation, identified in six patients (21% of those with rheumatological findings, and part of a broader

cohort of 28 patients with rheumatological manifestations out of the total 157) [10]. In this cohort, piezogenic papules frequently co-occurred with joint hyperextensibility: a 22-year-old female with a history of congenital absence of one kidney and cytomegalovirus-induced haemophagocytic lymphohistiocytosis presented with numerous piezogenic papules, 10-degree hyperextension of the knees bilaterally, and a high-arched palate, while a 21-year-old female with atraumatic fusion of three thoracic vertebrae also had piezogenic papules without overt inflammatory findings, and her brother, likewise affected by GATA2 deficiency, had piezogenic papules together with increased range of motion in both shoulders and osteopenia refractory to calcium and vitamin D supplementation [10]. Notably, the unaffected mother of this sibling pair, who did not carry the GATA2 mutation, did not have piezogenic papules, an observation the authors highlighted as circumstantial support for a genotype-linked connective tissue phenotype rather than a purely mechanical or incidental finding [10]. A further mother-daughter pair with GATA2 deficiency and psoriasis both exhibited piezogenic papules, with the mother also showing elbow and knee hyperextension and the daughter demonstrating hypermobile joints, pes planus, and chondromalacia patellae [10].

The authors of this GATA2 study proposed that the observed piezogenic papules, considered alongside joint hypermobility, might reflect an underlying abnormality of collagen architecture in GATA2 deficiency, hypothesising a possible mechanistic link through GATA2's known role in the transcriptional regulation of matrix metalloproteinase-2 (MMP-2) in endothelial cells—an enzyme that participates in the degradation and remodelling of collagen in connective tissue—although they were careful to state that no study had directly demonstrated a collagen abnormality in GATA2 deficiency and that this remained a hypothesis warranting further investigation [10]. This finding is important for the present review because it extends the differential diagnosis of piezogenic papules well beyond classical connective tissue disorders such as Ehlers-Danlos syndrome into a rare immunodeficiency and myelodysplasia syndrome, illustrating that an apparently trivial dermatological sign can, in the right clinical context, prompt investigation for a serious underlying haematological or genetic condition [10].

Piezogenic papules have also been reported in rheumatoid arthritis and rheumatic heart disease, though the causal versus coincidental nature of these associations remains debated in the literature. A case of unusually large piezogenic papules—termed "piezogenic pedal tumours of obesity" because the lesions measured 1 to 3.5 cm in diameter, considerably larger than the

typically described 0.2 to 2 cm range—occurred in a 55-year-old woman with a 25-year history of rheumatoid arthritis and a body mass index of 30.1 kg/m², who described painful nodules on standing and walking that became less prominent on relieving pressure [8]. The authors of this report explicitly noted that it remained unclear whether the association with rheumatoid arthritis was causal or spurious, and they proposed that obesity, by increasing sustained pressure on the heels, might explain the unusually large size of the lesions in this patient, since an increase in papule diameter with increasing body weight had previously been reported in children, though this factor had not previously been implicated in adults [8]. This case is a useful illustration of appropriately cautious clinical reasoning: rather than asserting a firm causal link between rheumatoid arthritis and papule size, the authors proposed obesity-related mechanical loading as the more parsimonious explanation while acknowledging that the rheumatoid arthritis association could not be entirely excluded.

5.5. Management of painful piezogenic pedal papules

Because the overwhelming majority of piezogenic papules are asymptomatic, treatment is required only in the minority of cases where pain limits activity, and management follows a graduated approach from conservative measures to more invasive interventions when conservative treatment fails [6,9,15,18]. Conservative measures include reduction of standing time, weight loss where relevant, use of compression stockings, foam-fitting plastic heel cups, and activity modification to avoid prolonged weight-bearing or high-impact loading [6,15].

When conservative management is insufficient, several injection-based and physical therapies have been described, albeit largely in case reports and small series rather than controlled trials. Intralesional injections of betamethasone combined with bupivacaine have been used with reported success in reducing pain, with effects sustained over a five-year follow-up period in one case, and this steroid-anaesthetic combination has been proposed as a reasonable non-surgical option for symptomatic patients [9,18]. Electroacupuncture has been attempted in some patients but with variable and often unsatisfactory results; in one case series, a combination of intralesional triamcinolone and lidocaine was used for larger lesions but was not successful in all cases, and the authors concluded that treatment outcomes for painful piezogenic papules are frequently unsatisfactory regardless of modality chosen [6,18].

A more recent and apparently effective approach involves injection lipolysis using deoxycholic acid (DCA), a minimally invasive technique originally developed and approved for reduction of submental fat [15]. A case report described successful treatment of painful piezogenic pedal papules in a 34-year-old obese woman using DCA injection: after disinfecting the treatment area, a 1% DCA-based cosmetic solution was injected with a 30-gauge needle perpendicular to the skin, with 0.05 to 0.1 mL injected into each nodule depending on its size, and ice applied before and after injection to minimise discomfort [15]. At two-week follow-up, the previously treated nodules had completely disappeared and the patient experienced no pain during standing or walking, with no adverse effects such as swelling, bruising, or tenderness reported [15]. The authors interpreted the rapid resolution of both the nodules and the associated pain as supporting evidence that the papules represent fat herniation rather than true fat accumulation, since a genuine adipose deposit would be expected to respond more slowly to lipolytic injection, and they proposed that this minimally invasive technique could be extended to the treatment of other small, localised fat herniations in various anatomical sites [15]. Nevertheless, the authors themselves acknowledged that this represented, to their knowledge, the first reported case of DCA treatment for painful piezogenic papules, and they explicitly called for larger-scale and longer-term trials to confirm both efficacy and safety before the technique could be recommended more broadly [15]. Surgical excision of the papules with sealing of the underlying fascial defect remains an option for patients who fail conservative and injection-based therapies, and has been reported to be more effective than conservative methods in some series, though it carries the attendant risks and recovery burden of surgery on a weight-bearing structure [6,16].

6. Blisters, Calluses, and Corns: Prevalence and Prevention in Runners

Although blisters, calluses, and corns are less diagnostically fraught than talon noir or piezogenic papules, they represent by far the most prevalent friction-induced dermatoses in athletic populations and merit dedicated attention given their impact on training continuity and performance. A cross-sectional study of 76 road runners in Southern Brazil, assessed by questionnaire, interview, and clinical examination, found that the most prevalent sports-related dermatoses were blisters (50%), chafing (42.1%), calluses (34.2%), onychomadesis (31.5%), sunburn (19.7%), tinea pedis (18.4%), onychocryptosis (14.5%), and cheilitis simplex (14.5%), with piezogenic pedal papules identified in 3.9% of participants and calcaneal petechiae (talon noir) in 2.6% [14]. This study is particularly valuable because it explicitly tested for an

association between weekly running distance and dermatosis prevalence, stratifying participants into three distance categories (≤ 24 km, 25–64 km, and ≥ 65 km per week) and finding that calluses, jogger's nipple, and cheilitis simplex were all significantly more common among athletes running the greatest weekly distances, with linear trend testing confirming that the likelihood of calluses ($p=0.024$), onychocryptosis ($p=0.034$), onychomadesis ($p=0.030$), tinea pedis ($p=0.011$), jogger's nipple ($p=0.002$), and cheilitis simplex ($p=0.034$) all increased with weekly running distance [14]. This dose-response relationship provides some of the strongest available evidence that cumulative mechanical load, rather than incidental or one-off trauma, drives the development of these dermatoses in runners, and it stands in useful contrast to isolated case reports, which by their nature cannot demonstrate such a relationship.

Notably, sunburn in this Brazilian cohort occurred across all distance categories without significant difference between groups, a finding the authors attributed to the predominance of training before 10 a.m. or after 4 p.m. and to widespread use of sun-protective clothing, illustrating that not all sports dermatoses scale uniformly with training volume—some, like sunburn, are more strongly determined by environmental exposure timing than by cumulative mechanical stress [14]. This distinction is useful for the present review because it reinforces that mechanical dermatoses specifically (blisters, calluses, jogger's nipple) show the expected dose-response relationship with training load, whereas environmentally driven conditions do not necessarily follow the same pattern, supporting the mechanistic framework proposed in Section 3.

Comparative data from other populations corroborate the high prevalence of friction blisters and calluses while illustrating substantial variability by cohort and sporting discipline. A Twin Cities Marathon study spanning twelve years found an average blister rate of approximately 20% among participants, a figure lower than the 50% prevalence reported in the Brazilian road-runner cohort, likely reflecting differences in race distance, climate, and footwear practices between populations [1,14]. Reviews of sports-related dermatoses have similarly reported blister prevalence ranging from 0.2% to 39% across different studies, an extraordinarily wide range that underscores how strongly reported prevalence depends on study design, definition of blister severity, and population characteristics rather than reflecting a single "true" prevalence figure [1].

Prevention of blisters centres on minimising friction through moisture-wicking socks, application of petroleum jelly or similar lubricants, ensuring properly fitted footwear, and, in some protocols, hardening the skin through tannic acid soaks or double-layering socks with powder, although these latter strategies are described as less commonly employed than basic friction-reduction measures [1]. When blisters do occur, most are managed conservatively by leaving the roof intact whenever possible, since the overlying epidermis functions as a natural biological dressing; for larger or more uncomfortable blisters, early puncture to release trapped fluid while preserving the roof is recommended, sometimes supplemented with adhesive taping, moleskin pads, or hydrocolloid dressings to reduce discomfort and protect against infection [1]. Management of calluses is generally conservative and often unnecessary from a therapeutic standpoint, since the lesion typically resolves once the causative repetitive activity ceases, though the clinical necessity of active callus removal remains a point of some debate in the literature, with some authors regarding calluses as an adaptive rather than pathological response that should be left undisturbed unless symptomatic [1,2].

7. Jogger's Toe, Runner's Rump, and Other Trauma-Related Foot and Trunk Dermatoses

Beyond the four core mechanisms discussed above, a constellation of related traumatic dermatoses recur throughout the sports dermatology literature, several of which share pathophysiological overlap with talon noir and piezogenic papules but merit separate description because of distinct clinical morphology or anatomical location.

Jogger's toe—also termed runner's toe or tennis toe—results from repetitive microtrauma to the toenail, most commonly the hallux or second toe, caused by shearing forces applied to the nail plate during repetitive forward thrusting of the foot within the toe box, an effect that is particularly accentuated during downhill running [1]. The affected nail characteristically shows thickening, ridging, and discolouration, and preventive strategies focus on cautious, straight-line trimming of the nail to a short length, ensuring proper shoe fit with tight lacing to minimise excessive foot movement within the shoe, and, in some cases, use of protective sponge toe covers [1]. Regular application of an emollient cream to enhance nail-plate flexibility has also been suggested as a means of reducing the risk of splitting under repetitive pressure [1]. The pathophysiology of jogger's toe closely parallels that of subungual haematoma, differing principally in chronicity: whereas subungual haematoma tends to arise from a single episode of

blunt trauma, jogger's toe reflects cumulative repetitive impact over a training cycle, again illustrating the spectrum from acute to chronic mechanical injury that characterises this family of conditions [1].

Runner's rump is a distinct trauma-related dermatosis characterised by hyperpigmentation over the superior aspect of the gluteal cleft in long-distance runners, arising from repeated ecchymotic contact between the buttocks during each running stride [1]. This condition is purely cosmetic in nature and typically resolves spontaneously with a period of reduced running activity, requiring no specific medical intervention beyond reassurance [1]. It is included in this review not because it poses diagnostic difficulty comparable to talon noir, but because it exemplifies how the same underlying principle—repetitive mechanical contact leading to localised dermal vascular trauma—recurs at anatomical sites well beyond the foot.

Athlete's nodules, also known as pilar sheath papilloma or collagenoma, represent a further variant of chronic pressure-induced dermal change, presenting as soft, flesh-coloured nodules with a keratinised surface, most commonly located on the peroneal aspect of the ankle or the dorsum of the foot, and thought to be stimulated by chronic, repetitive trauma to the affected area [1]. These lesions require differentiation from dermatofibromas, which can appear clinically similar but arise through a distinct pathophysiological process [1]. The eponymous variation across sports—"knuckle pads," "surfer's nodules," "jogger's toe," "tennis toe," "turf toe," "runner's rump," "rower's rump," "skier's shin," and football players' ankle nodules—reflects the fact that essentially the same histopathological process (deposition of collagen bundles in a haphazard pattern within the dermis, in response to friction or recent trauma at a sport-specific point of contact) manifests under different names depending on the anatomical site and sporting context in which it occurs, a taxonomic peculiarity noted explicitly in a review of dermatological eponyms linking skin conditions to sports [19]. This same review catalogued several other named phenomena directly relevant to the present discussion, including mogul skier's palm (ecchymosis of the hypothenar eminence in skiers who repeatedly plant ski poles into snow) and stringer's fingers (hypertrophic papules on the fingertips of tennis players who habitually adjust their racquet strings, producing a callus-like thickening of the stratum corneum), both of which further illustrate the principle that virtually any repetitive point of mechanical contact between an athlete's skin and their equipment can generate a site-specific, named dermatosis [19].

Jogger's nipple, a form of irritant contact dermatitis affecting an estimated 2% to 16.3% of runners across different studies, arises from friction between the nipple-areolar complex and clothing, producing painful erythematous erosions with crusting that are exacerbated by tight-fitting shirts made from coarse, slow-drying cotton fabrics [1,14]. Women who train without sports bras and overweight male runners appear to be at particularly elevated risk, and severely affected runners may develop bloody staining of their garments around the nipples during long training sessions [1]. Preventive measures focus on friction reduction through adhesive dressings or petroleum jelly applied directly to the nipples prior to running, and topical antibiotics may be used for secondarily infected or severely eroded lesions [1]. As noted in Section 6, jogger's nipple demonstrated one of the clearest dose-response relationships with weekly running distance in the Brazilian road-runner cohort, reinforcing its classification as a cumulative mechanical dermatosis rather than a chance occurrence [14].

8. Skin Structure, Barrier Function, and the Biological Basis of Mechanical Vulnerability

Understanding why the skin is vulnerable to mechanical and friction-induced injury requires brief consideration of normal cutaneous anatomy and barrier physiology. The skin comprises three principal layers—epidermis, dermis, and subcutaneous tissue—of differing embryological origin and mechanical function, with the epidermis composed of stratified keratinocytes organised into four or five sublayers, and the dermis providing tensile strength and elasticity through its network of type I and type III collagen fibres interwoven with elastic fibres [2]. The thickness of the epidermis and the underlying dermal collagen architecture vary considerably by anatomical site, with the plantar skin of the foot being among the thickest in the body owing to constant weight-bearing demands, yet this same thickness does not confer complete protection against shear-induced blister formation or pressure-induced fat herniation, both of which depend more on the integrity of specific dermal and subcutaneous connective tissue planes than on epidermal thickness alone [1,2].

The dermis is structurally dominated by type I collagen (approximately 80% of dermal collagen) and type III collagen (approximately 20%), with collagen content and cross-linking quality declining with age at a rate of roughly 1% per year, a process accelerated by environmental factors including ultraviolet exposure, smoking, and pollution [2]. This age-related decline in collagen quality plausibly contributes to the observation, discussed in Section 5, that piezogenic

papules occur with somewhat different frequency and severity across age groups, since a dermis with reduced collagen integrity may be more susceptible to fat herniation under a given mechanical load, though direct evidence linking age-related collagen decline specifically to piezogenic papule development was not identified in the reviewed sources [2,17].

Beyond its structural collagen framework, the skin functions as an active barrier organ that responds dynamically to repeated mechanical and environmental stress through changes in hydration, surface pH, lipid composition, and microbial ecology, with endurance exercise inducing measurable, if transient, shifts in each of these parameters [20]. A narrative review of skin barrier dysfunction in endurance athletes described how prolonged, repetitive mechanical loading, sweat and hyperhydration, occlusion by clothing and footwear, and thermal and water exposure combine to generate a recurrent pattern of friction blisters, chafing, intertrigo, and related dermatoses, emphasising that these conditions typically arise not from a single causal factor in isolation but from the simultaneous stacking of several subclinical insults—new footwear, wet socks, elevated ambient heat, salt accumulation on the skin surface, and delayed post-exercise hygiene—that collectively exceed the skin's capacity to compensate [20]. This "barrier reserve" concept, in which a healthy athlete may tolerate a given training load without symptoms until several compounding stressors accumulate simultaneously, offers a coherent explanatory framework for why identical training volumes can produce markedly different dermatological outcomes between individuals, and it is broadly consistent with the dose-response relationship between running distance and callus or jogger's nipple prevalence documented in the Brazilian cohort described in Section 6 [14,20].

The same review noted that friction blisters are increasingly understood to arise from repetitive shear deformation—driven by the relative motion between bone, soft tissue, and the shoe-foot interface—rather than from simple surface rubbing, a refinement of the classical mechanistic account that has direct implications for prevention strategy, since interventions aimed solely at reducing surface friction (for example, lubricants) may be less effective than interventions that also address underlying shear mechanics, such as footwear fit, lacing technique, or gait modification [20]. This more nuanced mechanistic understanding aligns with, and extends, the description of blister pathogenesis presented in Section 3, reinforcing that shear rather than pure friction is the operative biomechanical insult in most cases [1,20].

9. Connective Tissue Fragility, Joint Hypermobility, and Cutaneous Manifestations

Several of the conditions discussed in this review—piezogenic papules most prominently, but also, more speculatively, talon noir and athlete's nodules—have been proposed to occur with greater frequency or severity in individuals with underlying connective tissue fragility, particularly hypermobile Ehlers-Danlos syndrome (hEDS) and hypermobility spectrum disorder (HSD). This section synthesises the paediatric and adult evidence on this relationship, an area where the literature is characterised by genuine uncertainty rather than settled consensus.

A scoping review of age-related symptom clustering in paediatric hypermobility spectrum disorders, synthesising 27 studies spanning 1974 to 2025, identified piezogenic pedal papules as one component of a broader "immune dysregulation and connective tissue clues" symptom cluster that also includes soft, hyperextensible skin, easy bruising, atrophic scars, recurrent hernias, and mast cell activation syndrome-type symptoms such as urticaria, flushing, and pruritus [11]. This review explicitly noted that a combination of seemingly unrelated mild skin and tissue signs—rather than any single finding in isolation—should raise clinical suspicion for an underlying hypermobility-related disorder, and that piezogenic papules, when they occur in a paediatric patient with other suggestive features, should not be dismissed as an incidental cosmetic finding [11]. The same review proposed an age-related evolution of symptom clusters in paediatric hEDS and HSD, in which musculoskeletal pain and joint instability typically dominate from age 0 to 5 years, joint pain and early proprioceptive abnormalities become more prominent from ages 5 to 9, disorder of gut-brain interaction symptoms and fatigue emerge between ages 9 and 12, and the full multisystem picture—including dysautonomia, chronic widespread pain, and psychosocial impact—crystallises during adolescence [11]. Piezogenic papules were not assigned a specific age of peak emergence within this framework, which itself reflects the limited and heterogeneous evidence base regarding their developmental timing, but their inclusion within the broader connective tissue symptom cluster reinforces the plausibility, without proving the certainty, of a mechanistic link to hypermobility [11,17].

A separate review of pain symptomatology and management in paediatric Ehlers-Danlos syndrome similarly listed painful piezogenic pedal papules among the cutaneous manifestations that can contribute to chronic pain in this population, situating piezogenic papules alongside chronic musculoskeletal pain, headache, and abdominal pain as recurrent but underrecognised

sources of discomfort in children with hEDS [21]. This review is instructive in illustrating that even when piezogenic papules are not the primary presenting complaint, their presence can meaningfully contribute to cumulative pain burden and reduced quality of life in affected children, an observation with direct clinical relevance for paediatricians and sports medicine physicians assessing young athletes with hypermobility [21].

Taken together, the paediatric literature converges on a moderately confident but explicitly qualified conclusion: piezogenic papules are common in both hypermobile and non-hypermobile children, occur with somewhat greater severity (though not necessarily greater overall prevalence) in children with HSD or hEDS, and should be interpreted within the context of a broader constellation of connective tissue and mechanical findings rather than as an isolated diagnostic marker [11,17]. This nuanced position stands in contrast to earlier, more assertive claims in some adult case reports that piezogenic papules are essentially synonymous with underlying connective tissue disease, and it illustrates how systematic paediatric evidence has appropriately tempered claims derived from isolated adult case reports [6,8,18].

10. Site-Specific Patterns: Foot, Hand, and Trunk Dermatoses in Different Sports

The anatomical distribution of mechanical and friction-induced dermatoses varies systematically with the specific biomechanical demands of different sports, a pattern that has direct implications for targeted prevention counselling. In distance running, the foot bears the overwhelming majority of mechanical load, and consequently blisters, calluses, onychomadesis, tinea pedis, onychocryptosis, piezogenic pedal papules, and calcaneal petechiae (talon noir) were all documented at meaningful frequencies within the same 76-runner cohort, reflecting the concentration of repetitive impact, shear, and moisture exposure at the plantar and dorsal foot [1,14]. The same cohort also documented non-foot dermatoses—chafing, jogger's nipple, and cheilitis simplex—illustrating that even within a single sport, mechanical dermatoses distribute across multiple anatomical sites according to where clothing, equipment, and repetitive limb motion generate the greatest localised stress [14].

Racket sports and skiing generate a distinct pattern of hand- and finger-based mechanical dermatoses that contrasts with the foot-dominant pattern of distance running. Stringer's fingers, arising from habitual manual adjustment of tennis racquet strings, produces hypertrophic, callus-like thickening of the fingertip stratum corneum, while mogul skiers develop ecchymosis of the

hypothenar palm from repeated forceful planting of ski poles into snow [19]. These hand-centred conditions parallel, in mechanism if not in location, the foot-centred conditions discussed throughout this review, again underscoring that the specific anatomical distribution of mechanical dermatoses is dictated primarily by the point of maximal repetitive contact between the athlete's body and their sport-specific equipment or surface, rather than by any inherent site-specific vulnerability of the skin itself [1,19].

Figure skating provides a particularly well-documented example of how footwear design interacts with joint biomechanics to generate a predictable cluster of mechanical dermatoses, and merits detailed discussion because it is the only source in this review to provide systematic prevalence data stratified by skater experience level. A cross-sectional study of 95 figure skaters of varying ages and skill levels, evaluated through questionnaire, clinical examination, and instrumented measurement of foot and boot characteristics, found that retrocalcaneal bursitis was the most common ongoing condition, affecting at least one foot in 34% of skaters, followed by posterior heel skin calluses (29%) and superficial calcaneal bursitis (28%) [22]. Piezogenic pedal papules were noted incidentally in some elite skaters when reviewing photographs taken during the evaluation, though the authors explicitly stated that these papules were not formally diagnosed during the structured physical examination because affected skaters reported no associated pain, and the papules could only be detected while the skater was weight-bearing—an observation that reinforces the point made throughout Sections 5 and 9 that piezogenic papules frequently escape clinical detection precisely because they are asymptomatic and position-dependent [22].

This same figure skating study found that the prevalence of nearly all documented lower-extremity overuse conditions was substantially higher among elite skaters than non-elite skaters, with retrocalcaneal bursitis, superficial calcaneal bursitis, toe corns, and lateral ankle skin abrasion all showing prevalence increases of 20 to 32 percentage points in elite compared with non-elite skaters [22]. Critically, the study also identified that boot-foot length mismatch—oversized skating boots relative to actual foot length—was present in 68% of skaters and was significantly associated with increased risk of superficial calcaneal bursitis and lateral ankle skin abrasion, while poor in-skate ankle dorsiflexion (present in 49% of skaters) was associated with retrocalcaneal bursitis in non-elite skaters [22]. This finding is directly relevant to the broader theme of this review because it demonstrates, through a rigorously conducted biomechanical and clinical study, that ill-fitting equipment is a modifiable and quantifiable risk factor for mechanical

dermatoses, analogous to the well-established relationship between running shoe fit and blister formation described in Section 6, and it extends the evidence base beyond simple case reports to a study with formal risk-factor analysis using logistic regression [1,22]. The authors concluded that boot retailers should be better educated to fit skaters with appropriately sized boots, and that boot stiffness selection should account for skater body weight rather than skill level alone, recommendations with clear translational value for injury and dermatosis prevention in this sport [22].

11. Cutaneous Manifestations at the Interface of Rheumatology and Sports Dermatology

The reviewed literature reveals a recurring theme that mechanical dermatoses of the foot occupy an unusual position at the interface between dermatology, sports medicine, and rheumatology, since several conditions discussed in this review—most notably piezogenic papules but also, to a lesser extent, athlete's nodules and talon noir—have been documented as incidental findings during rheumatological evaluation for unrelated joint or systemic complaints. This section synthesises the evidence bearing specifically on this intersection.

The large GATA2 deficiency cohort study discussed in Section 5.4 is the most substantial contribution to this literature, not only because of its relatively large sample size (157 patients, with 28 exhibiting rheumatological manifestations) compared with the case-report-dominated remainder of this field, but also because it explicitly framed piezogenic papules within a broader catalogue of rheumatological findings that included early-onset osteoarthritis, ankylosing spondylitis, seronegative erosive rheumatoid arthritis, psoriatic arthritis, and osteopenia [10]. The study's authors constructed a summary figure depicting the full spectrum of infectious, myelodysplastic, and rheumatological disease manifestations observed in GATA2 deficiency, explicitly marking piezogenic papules, ankylosing spondylitis, seronegative erosive rheumatoid arthritis, and several other findings as being reported for the first time in their cohort, which underscores how a relatively small but systematically documented case series can meaningfully expand the recognised phenotypic spectrum of a rare disease [10]. Importantly, the study also performed lymphocyte immunophenotyping and found that patients with rheumatological manifestations (including piezogenic papules) had significantly lower proportions of CD3+CD4+ helper T cells and naïve CD4+ T cell subsets, and significantly higher proportions of CD8+ cytotoxic T cells, compared with GATA2-deficient patients without rheumatological

manifestations, suggesting a possible immunological correlate of the musculoskeletal and dermatological phenotype, though the authors appropriately noted that their retrospective design precluded any claim about the direction of causality between immune dysregulation and connective tissue findings [10].

The authors of this GATA2 study were explicit about the limitations of their retrospective, single-institution methodology, stating clearly that their descriptive approach precluded statistical inferences that could be generalised to the broader population of patients with GATA2 deficiency, and that only 12 of the 28 patients with rheumatological findings underwent comprehensive in-person rheumatological assessment, meaning the true prevalence of piezogenic papules and other findings in this population was likely underestimated [10]. This candid acknowledgment of methodological limitation is a useful model for interpreting the piezogenic papule literature as a whole: even the most systematically conducted study identified in this review explicitly flagged its own findings as preliminary and hypothesis-generating rather than definitive, a caveat that applies with even greater force to the numerous single-patient case reports that constitute the majority of the evidence base discussed in Sections 5 and 9 [6-8,10,15,18].

12. Biomechanical Determinants of Mechanical Skin and Connective Tissue Strain

A distinct but complementary strand of evidence, drawn from computational biomechanics rather than clinical case description, offers mechanistic insight into how specific movement patterns and foot-strike locations influence the strain experienced by connective tissue structures during athletic activity, providing a bridge between the clinical dermatological literature reviewed above and the biomechanical principles underlying injury and dermatosis prevention.

A finite-element in-silico modelling study simulated a "stepping on obstacle" scenario relevant to cross-country running, incorporating ground reaction forces and muscle activation to evaluate ligament strain across 27 simulated conditions varying joint laxity (none, medium, high), foot placement (central, medial, lateral), and surface friction (low, medium, high) [12]. The model found that foot placement was the dominant factor influencing ligament strain, with medial foot strikes producing the lowest anterior tibiotalar ligament strain (11.5%), which increased by 1.75-fold with central foot strikes and 1.73-fold with lateral foot strikes [12]. Friction level showed a negative correlation with strain, such that higher surface friction reduced anterior tibiotalar

ligament stress, while joint laxity was positively correlated with plantar fascia strain, with the medium-laxity simulation group experiencing greater strain than the no-laxity group [12].

Although this modelling study addresses ligamentous rather than cutaneous strain directly, it is included in this review because it offers a rigorous, quantitative illustration of the same underlying biomechanical principle that governs cutaneous mechanical dermatoses: namely, that the specific location and orientation of repetitive mechanical contact—whether at the ligament-bone interface or at the skin-shoe interface—determines the magnitude of resultant tissue strain far more than any single global parameter such as total training volume [1,12,20]. The finding that joint laxity increased plantar fascia strain in this computational model is also directly relevant to the discussion in Sections 5 and 9 regarding the possible role of connective tissue hypermobility in piezogenic papule development, since increased laxity of the plantar fascia—the very structure through which subcutaneous fat is thought to herniate in piezogenic papules—provides a plausible, if indirect, biomechanical corroboration of the hypothesis that connective tissue laxity predisposes to fat herniation under repetitive load [9,12,17]. The authors of the biomechanical modelling study explicitly noted that their in-silico approach allowed evaluation of extreme conditions that could not ethically be tested in human subjects, and that their findings supported medial foot placement and higher surface friction as protective strategies against overuse injury, offering a mechanistic rationale that complements the largely descriptive clinical evidence reviewed elsewhere in this article [12].

More broadly, fascial tissue research addressing the mechanobiology of connective tissue adaptation to mechanical loading provides additional context for understanding why repetitive loading can produce divergent outcomes—adaptive strengthening in some tissues, pathological herniation or degeneration in others—depending on loading magnitude, frequency, and the baseline mechanical competence of the tissue in question [23]. A consensus statement on fascial tissue research in sports medicine described how fascial tissues respond to chronic mechanical overloading by increasing collagen synthesis and stiffness, while chronic underloading produces the opposite adaptation, and emphasised that molecular crosstalk between extracellular matrix components and resident fibroblasts governs this dynamic remodelling process [23]. Although this consensus statement was focused primarily on tendon and myofascial tissue rather than on the specific dermal and subcutaneous planes implicated in piezogenic papules, its general principle—that connective tissue responds to mechanical load through a graded continuum from

adaptive strengthening to pathological failure—offers a plausible unifying framework for understanding why the same repetitive heel-loading that most individuals tolerate without incident produces overt fat herniation in a minority of predisposed patients [9,17,23].

Similarly, work on the pathogenesis of tendinopathy has articulated an "iceberg" model in which cumulative microtrauma from repetitive mechanical loading progressively overwhelms tissue healing capacity, producing a spectrum from asymptomatic microscopic damage to overt clinical pathology, a conceptual framework directly analogous to the progression from asymptomatic to painful piezogenic papules, or from tolerable training load to overt blister and callus formation, described throughout this review [24]. This tendinopathy literature reinforces that mechanical dermatoses and connective tissue injuries share a common underlying logic: tissue damage accumulates below the threshold of clinical symptoms until either cumulative load or reduced tissue resilience (whether from connective tissue laxity, ageing, or systemic disease) tips the balance toward overt pathology [12,23,24].

13. Recovery, Skin Physiology, and Systemic Modulators of Mechanical Tissue Tolerance

A further body of evidence, somewhat more tangential to the direct clinical description of mechanical dermatoses but relevant to understanding factors that modulate an athlete's tolerance to repetitive mechanical stress, addresses the physiological changes induced by intense exercise and recovery interventions that may influence connective tissue resilience and inflammatory responsiveness.

Endurance exercise has been shown to produce measurable, transient changes in systemic inflammatory markers, oxidative stress, and gut microbiota composition, several of which plausibly interact with connective tissue healing capacity, although direct evidence linking these systemic changes to mechanical dermatosis risk was not identified in the reviewed sources [25,26]. A study examining the time-course of inflammatory response after whole-body cryotherapy following a simulated 48-minute trail-running exercise found that a single session of cryotherapy significantly attenuated the postexercise rise in interleukin-1 beta and C-reactive protein compared with passive recovery, suggesting that recovery modalities can modulate the inflammatory cascade triggered by exercise-induced tissue microtrauma [26]. While this study focused on systemic muscle damage markers rather than cutaneous or connective tissue dermatoses specifically, it is included here because it illustrates that the inflammatory response to

repetitive mechanical loading—the same underlying process implicated in the pathogenesis of overuse dermatoses—can be pharmacologically or physically modulated, raising the theoretical possibility that recovery interventions could influence the development or resolution of mechanical dermatoses, though this specific hypothesis has not been tested in the dermatological literature reviewed here [1,20,26].

A separate review of whole-body cryotherapy in athletes similarly described consistent anti-inflammatory and analgesic effects following cold exposure, including reductions in creatine kinase and inflammatory cytokine levels after repeated exposures, alongside more variable effects on interleukin-6 depending on the specific exercise protocol studied [27]. The relevance of this literature to mechanical dermatoses is indirect but not negligible: to the extent that talon noir, blisters, and piezogenic papules represent localised inflammatory and haemorrhagic responses to mechanical microtrauma, systemic or local anti-inflammatory strategies used for muscle recovery might plausibly influence the clinical course of these cutaneous conditions as well, although this remains speculative in the absence of direct evidence [1,26,27].

Endurance exercise has also been documented to induce measurable shifts in gut microbiota composition and function, with several studies suggesting that microbiota composition may modulate systemic inflammatory tone, oxidative stress, and even hydration status during prolonged exercise [25]. While no direct evidence was identified linking gut microbiota to the specific mechanical dermatoses discussed in this review, this literature is included for completeness because it represents part of the broader physiological context of endurance athlete health within which localised mechanical skin injury occurs, and it illustrates the multisystem nature of the physiological adaptations that accompany the training loads implicated in mechanical dermatosis development [1,14,25].

14. Talon Noir, Piezogenic Papules, and the Broader Differential of Pigmented and Nodular Foot Lesions

Given the centrality of misdiagnosis avoidance to this review, it is useful to consolidate the differential diagnostic considerations for pigmented and nodular acral lesions discussed across preceding sections into an integrated framework, since clinicians evaluating an athlete with a foot lesion must simultaneously consider talon noir, piezogenic papules, subungual haematoma,

plantar warts, and, critically, acral melanoma and precalcaneal congenital fibrolipomatous hamartoma.

Precalcaneal congenital fibrolipomatous hamartoma (PCFH), though not itself an acquired athletic dermatosis, features prominently in the differential diagnosis of piezogenic pedal papules and merits brief discussion because two case reports specifically addressed this distinction. PCFH is a benign lesion typically presenting at birth or within the first months of life as bilateral, solitary, skin-coloured, non-tender nodules on the medial plantar aspect of the foot, and is distinguished from piezogenic papules by its onset in infancy rather than in association with weight-bearing activity, its typically solitary rather than multiple presentation, and its lack of the classical pressure-dependent appearance and disappearance that characterises piezogenic papules [28]. A case report of a healthy 1-year-old girl with bilateral asymptomatic plantar nodules explicitly worked through this differential, correctly identifying PCFH rather than piezogenic papules, juvenile plantar fibromatosis, calcified nodules, or nevus lipomatosus superficialis, on the basis that piezogenic papules are pressure-induced and appear only upon standing, whereas the nodules in this case were present at all times regardless of weight-bearing status [28]. A second case report of a 3-year-old girl with rubbery, non-tender bilateral heel nodules present since birth similarly reached a clinical diagnosis of PCFH without biopsy, noting that skin biopsy in young children can be traumatic, painful during subsequent ambulation given the plantar location, and productive of lasting aversion to medical care, and recommending that PCFH be diagnosed clinically whenever the presentation is classical, reserving biopsy or imaging for cases of genuine diagnostic uncertainty [29]. These two paediatric case reports reinforce a broader theme running throughout this review: that the age of onset, the relationship (or lack thereof) between lesion prominence and weight-bearing, and the number and distribution of lesions are together more diagnostically informative than lesion colour or size alone when distinguishing among the various benign nodular and pigmented conditions of the paediatric and adult foot [17,18,28,29].

Talon noir, piezogenic papules, subungual haematoma, and PCFH thus form a partially overlapping but mechanistically and clinically distinguishable group of foot lesions, unified by their benign natural history and their capacity, individually or collectively, to be mistaken for more serious pathology when clinicians are unfamiliar with their characteristic presentation, age of onset, and relationship to mechanical loading. Table 3 summarises the principal distinguishing

clinical features of these conditions as documented across the reviewed sources, intended as a practical reference for the differential diagnosis of nodular and pigmented lesions of the athlete's foot.

Table 3. Distinguishing clinical features of the principal mechanical and friction-induced foot dermatoses discussed in this review.

Condition [ref]	Typical age of onset	Relationship to weight-bearing	Typical morphology	Key differential to exclude
Talon noir [4,5]	Adolescence to adulthood; occasional older-adult cases	Not position-dependent; related to cumulative shear	Black-to-brown macules, uninterrupted skin surface, may show satellite dots	Acral lentiginous melanoma
Piezogenic pedal papules [6,9,15-18]	Any age, including young children; often noted in adolescence/adulthood	Prominent and enlarged on standing; disappears or diminishes on non-weight-bearing	Soft, skin-coloured or yellowish papules on medial/lateral/posterior heel	Precalcaneal congenital fibrolipomatous hamartoma; muscle herniation
Subungual haematoma [1]	Any age with repetitive downhill running or acute toe trauma	Not position-dependent	Blood accumulation beneath nail plate, painful	Subungual melanocytic naevus; subungual melanoma
Precalcaneal congenital	Birth to early infancy	Present regardless of	Bilateral, solitary, skin-coloured, non-	Piezogenic pedal papules;

fibrolipomatous hamartoma [28,29]		weight-bearing status	tender nodules on medial plantar heel	juvenile plantar fibromatosis; calcified nodules
Plantar wart (verruca) [1,4]	Any age	Not position-dependent	Firm, endophytic/exophytic plaque with visible thrombosed capillaries (black dots)	Talon noir; corn

15. Prevention Strategies Across Mechanical Dermatoses: A Synthesis

Having reviewed the individual conditions and their specific management in preceding sections, it is useful to synthesise the prevention literature into a coherent set of principles that apply, with varying degrees of specificity, across the entire spectrum of mechanical and friction-induced dermatoses discussed in this review.

The single most consistently emphasised preventive principle across the reviewed literature is appropriate footwear selection and fit, a theme that recurs from the discussion of blister prevention in runners through to the figure skating boot-fit analysis discussed in Section 10 [1,14,22]. The figure skating study's finding that 68% of skaters wore boots with a significant length mismatch, and that this mismatch was independently associated with increased risk of superficial calcaneal bursitis and lateral ankle skin abrasion in a formal logistic regression model, provides some of the strongest quantitative evidence in this review for the causal role of ill-fitting equipment in generating mechanical dermatoses, and stands in useful contrast to the largely anecdotal footwear recommendations found in the running-focused literature [1,14,22].

Friction and moisture management constitute a second recurring preventive theme, spanning the use of moisture-wicking socks and lubricants to reduce blister formation, the application of adhesive dressings or petroleum jelly to prevent jogger's nipple, and the broader "barrier reserve" concept in which cumulative moisture, heat, and friction stressors combine to overwhelm the skin's tolerance [1,14,20]. This principle extends, with appropriate caution, to the biomechanical

modelling evidence discussed in Section 12, which found that higher surface friction was associated with reduced ligamentous strain in a simulated obstacle-stepping scenario—an apparently counterintuitive finding in the context of cutaneous friction injury, since higher friction is generally associated with increased risk of skin blistering, but which the modelling authors explained by noting that increased friction improves foot-ground stability and thereby reduces the compensatory ligamentous strain that would otherwise occur under slippery conditions [12]. This apparent tension between the cutaneous and ligamentous literature—friction as a risk factor for blistering versus friction as protective against ligamentous strain—is worth making explicit rather than glossing over, since it illustrates that "friction" is not a single unidimensional variable with uniformly beneficial or harmful effects, but rather a parameter whose optimal level likely differs depending on which tissue and injury mechanism is under consideration [1,12,20].

A third preventive principle, particularly relevant to piezogenic papules and other conditions with a plausible connective tissue component, involves activity modification and load management in predisposed individuals, including reduction of prolonged standing time, weight management in obese patients, and avoidance of repetitive high-impact loading where painful lesions have already developed [6,8,15]. Unlike footwear and friction management, which apply broadly across the athletic population, this third category of prevention is most relevant to the minority of patients who develop symptomatic lesions or who have identified predisposing connective tissue laxity, reinforcing the risk-stratified approach to prevention that emerges from synthesising the paediatric hypermobility literature discussed in Section 9 [11,17].

Finally, patient and clinician education regarding the benign natural history and characteristic morphology of these conditions constitutes an underappreciated but recurrent theme across nearly every source reviewed, since the principal harm associated with talon noir, piezogenic papules, and related mechanical dermatoses in clinical practice appears to derive not from the conditions themselves but from diagnostic uncertainty, unnecessary biopsy, and patient anxiety generated by unfamiliarity with their appearance [4-6,18,28,29]. Several case reports explicitly framed their purpose as raising awareness of an underrecognised or rare condition specifically to prevent future unnecessary invasive investigation, a stated goal that this review, in synthesising these individually modest contributions into a coherent overview, seeks to advance [5,7,8,15,18,28,29].

16. Limitations of the Evidence Base

Several limitations qualify the conclusions that can be drawn from the literature synthesised in this review. First, and most fundamentally, the evidence base for piezogenic papules, talon noir, and several related conditions consists overwhelmingly of individual case reports and small case series, with only a small number of studies—the paediatric hypermobility scoping review, the GATA2 deficiency cohort, the Brazilian road-runner survey, the figure skating cross-sectional study, and the biomechanical modelling study—providing systematically collected data from more than a handful of patients [1,10,12,14,17,22]. This case-report predominance means that reported associations between piezogenic papules and conditions such as mitral valve prolapse, sarcoidosis, or rheumatoid arthritis cannot be interpreted as established causal relationships, and several of the authors of these case reports explicitly acknowledged this limitation themselves, framing their findings as hypothesis-generating rather than confirmatory [6-8].

Second, reported prevalence estimates for piezogenic papules varied by roughly thirty-fold across studies (from approximately 2.5% to 80%), a degree of heterogeneity that likely reflects genuine differences in study population, examination technique, and age distribution, but which also means that no single figure can be confidently cited as the "true" prevalence of this condition, and clinicians should be cautious about relying on any individual prevalence estimate when counselling patients [6,17,18]. Similarly, prevalence estimates for blisters ranged from 0.2% to 50% across different cohorts, again reflecting substantial population and methodological heterogeneity that limits direct comparison between studies [1,14].

Third, the majority of sources addressing prevention and management—particularly for painful piezogenic papules—derive from single-patient or small-series reports of specific interventions such as deoxycholic acid injection, intralesional steroid-anaesthetic combinations, or electroacupuncture, with no controlled trials identified comparing these modalities directly or establishing their efficacy against placebo or observation alone [6,15,18]. The authors of the deoxycholic acid case report explicitly called for larger and longer-term trials before their approach could be more broadly recommended, a caveat that applies equally to essentially all treatment approaches discussed in this review [15].

Fourth, several sources included in this review addressed topics adjacent to, rather than directly focused on, mechanical dermatoses—including gut microbiota and endurance exercise, whole-

body cryotherapy, fascial tissue mechanobiology, tendinopathy pathogenesis, and wearable sensor technology for orthopaedic sports medicine—and their inclusion, while providing useful mechanistic or contextual background, means that the strength of direct evidence linking these adjacent bodies of literature to the specific cutaneous conditions discussed in this review is necessarily more attenuated than the evidence drawn from sources focused explicitly on sports dermatology [23,25-27,30]. Readers should therefore weight the conclusions drawn from these adjacent sources with appropriate caution, treating them as plausible mechanistic context rather than as direct clinical evidence regarding the dermatoses under review.

Finally, this review is a narrative rather than a systematic synthesis: source selection and thematic organisation were undertaken by the reviewing authors according to judgment regarding topical relevance and clinical importance, rather than according to a pre-registered protocol with formal risk-of-bias assessment, and the review is consequently subject to the selection and emphasis biases inherent to any narrative synthesis. The relative predominance of case reports over larger cohort or controlled studies in the available literature on talon noir and piezogenic papules is itself a finding of this review, and it should be interpreted as a call for more systematic epidemiological and interventional research in this field rather than as a reflection of any deliberate exclusion of higher-quality evidence, none of which was identified during the literature synthesis process for these specific conditions.

17. Discussion

Read as a whole, the literature reviewed here supports several consistent conclusions while also revealing important areas of genuine uncertainty that merit explicit acknowledgment rather than false consensus. The most robust and reproducible finding across the reviewed sources is that mechanical and friction-induced dermatoses of the athlete's foot and trunk arise through a limited number of shared biomechanical mechanisms—shear-induced epidermal splitting, chronic pressure-induced hyperkeratosis, pressure-induced intraepidermal haemorrhage, and connective tissue herniation—and that these mechanisms manifest as clinically distinct entities (blisters, calluses and corns, talon noir and subungual haematoma, and piezogenic papules, respectively) depending on the specific anatomical structure and tissue plane affected [1,2,4,18]. This mechanistic unification, first articulated systematically in the foundational sports dermatology literature and reinforced by the more recent skin barrier physiology and fascial mechanobiology

reviews, provides a coherent conceptual framework that helps explain why these conditions so frequently co-occur in the same athlete and why prevention strategies targeting friction, pressure, and mechanical load reduction show benefit across multiple conditions simultaneously [1,14,20,23].

A second consistent theme is that diagnostic accuracy for pigmented and nodular acral lesions improves substantially when clinicians attend to specific historical and morphological clues: bilateral or multifocal distribution, a history of recent physical exertion, position-dependence of lesion prominence (present on standing, absent when non-weight-bearing), and age of onset (infancy for precalcaneal congenital fibrolipomatous hamartoma versus later childhood, adolescence, or adulthood for piezogenic papules and talon noir) [4,5,17,28,29]. Where these clues are absent or ambiguous, dermoscopy and scraping tests offer valuable but imperfect discriminatory power, and the reviewed case reports converge on the recommendation that biopsy, while sometimes unavoidable, should be reserved for genuinely uncertain cases rather than applied reflexively to every pigmented or nodular acral lesion in an athlete [4,5,18,28]. This measured, stepwise diagnostic approach—rather than either uncritical reassurance or uniform biopsy—emerges as the most defensible clinical stance across the synthesised literature.

A third theme, more tentative than the first two, concerns the relationship between piezogenic papules and systemic connective tissue or immunological disease. The paediatric scoping review and the associated empirical cohort study represent the most methodologically rigorous evidence bearing on this question, and their conclusion—that piezogenic papules are common in both hypermobile and non-hypermobile children, with somewhat greater lesion severity but not necessarily greater overall prevalence in the hypermobile group—provides a more measured and defensible position than the earlier, more assertive case-report literature suggesting a straightforward causal link to Ehlers-Danlos syndrome [6,9,17,18]. The GATA2 deficiency cohort study extends this cautious empiricism to a genuinely novel disease association, but its authors' explicit acknowledgment of incomplete case ascertainment and the retrospective, single-institution nature of their data collection should temper any inclination to treat piezogenic papules as a reliable diagnostic marker for GATA2 deficiency or any other systemic condition [10]. Taken together, this body of evidence supports a nuanced conclusion: piezogenic papules warrant a brief systemic and connective-tissue-oriented history and examination when they are unusually painful, severe, or occur alongside other suggestive features such as joint hyperextensibility, skin

hyperextensibility, or a family history of connective tissue disease, but they should not, on their own, trigger extensive systemic work-up in an otherwise asymptomatic patient [6-10,17,18].

A fourth and more speculative theme concerns the extent to which biomechanical modelling and mechanobiology research can inform the prevention of mechanical dermatoses. The finite-element modelling study demonstrating that medial foot placement and higher surface friction reduce ligamentous strain provides an elegant, quantitative illustration of a general biomechanical principle, but its direct applicability to cutaneous injury prevention remains inferential rather than directly demonstrated, since the model addressed ligament strain rather than skin blistering or fat herniation specifically [12]. Similarly, the fascial mechanobiology and tendinopathy literature offers a plausible conceptual bridge between cumulative mechanical loading and connective tissue pathology that is consistent with, but does not directly test, the pathogenesis of piezogenic papules [23,24]. These adjacent bodies of evidence should therefore be read as supportive context that strengthens the plausibility of mechanistic explanations proposed elsewhere in the sports dermatology literature, rather than as independent proof of those explanations.

Finally, the overall weight of evidence in this field is characterised by an imbalance between the apparent clinical frequency of these conditions—as evidenced by prevalence figures reaching 50 to 80% for some conditions in some populations—and the depth of the available research base, which remains dominated by isolated case reports and a small number of cross-sectional surveys [1,14,17,22]. This imbalance itself constitutes an important finding of the present review: mechanical and friction-induced dermatoses of the athlete's foot are neither rare nor trivial, yet they have received disproportionately little systematic epidemiological or interventional research attention relative to their apparent burden, a gap this review hopes to highlight for future investigators.

18. Conclusions

Mechanical and friction-induced dermatoses are among the most common yet most underappreciated cutaneous findings in athletic populations, encompassing a spectrum of conditions unified by shared biomechanical mechanisms of shear, pressure, and connective tissue herniation. Talon noir and piezogenic pedal papules occupy a particularly important position within this spectrum because their benign natural history contrasts sharply with their capacity to mimic more serious pathology—acral lentiginous melanoma in the case of talon noir, and, in the

case of piezogenic papules, an underlying connective tissue disorder, immune dysregulation syndrome, or cardiac abnormality in a minority of patients.

On the basis of the evidence reviewed, several principal conclusions can be offered. First, talon noir and subungual haematoma should be distinguished from acral melanoma primarily through attention to bilateral or multifocal distribution, history of recent exertion, and, where available, dermoscopy demonstrating homogeneous reddish globular structures rather than a parallel-ridge pattern, with scraping tests and biopsy reserved for genuinely ambiguous cases. Second, piezogenic pedal papules are common, typically asymptomatic, and require no treatment in the overwhelming majority of patients, but painful or unusually severe papules—particularly when accompanied by joint hyperextensibility, other connective tissue signs, or unexplained systemic findings—warrant a broader clinical evaluation that considers hypermobile Ehlers-Danlos syndrome, hypermobility spectrum disorder, and, in the appropriate clinical context, rarer associations such as GATA2 deficiency. Third, blisters, calluses, corns, jogger's toe, jogger's nipple, and runner's rump collectively represent a dose-dependent consequence of cumulative training load that scales with distance run and is amenable to prevention through appropriate footwear fit, friction and moisture management, and gradual load progression, a principle equally applicable to figure skating boot fit as to running shoe selection. Fourth, the evidence base for these conditions remains heavily weighted toward individual case reports and small case series, with only a handful of systematically conducted cross-sectional, cohort, or biomechanical modelling studies providing quantitative, generalisable data; this imbalance between clinical frequency and research depth represents the principal gap that future work in this field should address, through larger prospective cohorts, controlled treatment trials for painful piezogenic papules, and further biomechanical modelling linking specific movement patterns to both connective tissue and cutaneous injury risk. Until such evidence accumulates, the pragmatic clinical priority remains straightforward: recognise these conditions for what they usually are—benign, mechanically driven, and self-limited—while maintaining a low threshold to investigate further when clinical features deviate from the expected pattern.

DISCLOSURE:

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Project administration: Igor Smędowski

Supervision: Igor Smędowski

Funding statement: This study received no external funding.

Informed consent statement: Not applicable.

Institutional Review Board Statement: Not applicable.

Conflict of interest: The authors declare no conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process. In preparing this work, the authors used Claude for the purpose of checking grammar and improving readability. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication

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