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Hypermobility Spectrum Disorders and Sport Participation: When Movement Helps and When It Harms – a Narrative Review of the Medical Evidence

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ABSTRACT

Background: Joint hypermobility (JH), the hypermobility spectrum disorders (HSD) and hypermobile Ehlers–Danlos syndrome (hEDS) are common, under-recognised connective-tissue phenotypes in which the same ligamentous laxity that may favour performance in aesthetic disciplines can also predispose to injury, chronic pain and deconditioning.

Aim: To critically synthesise the current medical evidence on how sport and structured exercise can both harm and help hypermobile individuals, and to derive practical, risk-stratified implications for athletes, clinicians and physiotherapists.

Material and methods: Narrative review of systematic reviews, meta-analyses, prospective cohorts, case–control studies and intervention trials addressing JH, HSD and hEDS in the context of sport, injury and rehabilitation.

Results: The Beighton score remains the standard screen but is an imperfect, upper-limb-weighted measure of generalised JH. Meta-analyses indicate that generalised JH increases knee/anterior cruciate ligament and shoulder injury risk, especially in contact sport, whereas ankle risk is largely unchanged; several prospective cohorts in football, soccer and dance found no overall excess of injury. Symptomatic, not asymptomatic, hypermobility drives adverse outcomes. Conversely, individualised proprioceptive, stabilisation and even

supervised heavy-resistance programmes reduce pain and kinesiophobia and improve function and quality of life, whereas inactivity perpetuates a self-reinforcing symptom cycle.

Conclusions: Movement is therapeutic when graded, supervised and symptom-guided, yet potentially harmful when laxity is symptomatic or syndromic. Management should be individualised, multidisciplinary and stratified by symptom burden and EDS subtype, with vascular EDS representing an extreme high-risk exception.

Keywords: joint hypermobility; hypermobility spectrum disorder; Ehlers–Danlos syndrome; sports injury; Beighton score; rehabilitation; physical activity; proprioception

1. Introduction

Joint hypermobility (JH) describes the capacity of a joint, or a group of joints, to move passively or actively beyond the limits considered normal for the person's age, sex and ethnic background. It is not in itself a diagnosis but a clinical descriptor, observed either on history or on physical examination [1,2]. When the trait is confined to one or a few joints it is termed localised JH; when it is present simultaneously at the four limbs and the axial skeleton it is termed generalised joint hypermobility (GJH) [1]. A further peripheral pattern, limited to the hands and feet, and a historical pattern, in which previously generalised laxity has regressed with age, are also recognised [1]. The distinction between these patterns matters greatly for sport, because a single hyperextensible joint and a body-wide pattern of connective-tissue laxity carry entirely different implications for performance, injury risk and clinical management.

For most of the twentieth century, symptomatic hypermobility was labelled (benign) joint hypermobility syndrome and was thought to be confined to the musculoskeletal system, distinct from heritable disorders of connective tissue. The 2017 international revision of the Ehlers–Danlos nosology dismantled this assumption. It replaced the older terminology with a spectrum running from asymptomatic GJH, through the hypermobility spectrum disorders (HSD) — GJH plus one or more secondary musculoskeletal manifestations such as trauma, chronic pain or disturbed proprioception — to hypermobile Ehlers–Danlos syndrome (hEDS), the only one of the thirteen recognised EDS subtypes that still lacks a confirmatory genetic marker and is therefore defined purely clinically [1,3]. Asymptomatic JH, the HSDs and hEDS are now best understood as points on a single continuum, and an individual may move along that continuum as symptoms evolve over a lifetime [1]. This conceptual shift is central to any discussion of sport, because the evidence reviewed below repeatedly shows that it is symptomatic, rather than asymptomatic, hypermobility that is associated with harm.

Epidemiological estimates vary widely with the population studied, the instrument used and the cut-off applied. The reported prevalence of GJH ranges from roughly 2–57% in adults and 7–36% in children, with consistently higher figures in girls, in younger age groups and in Asian and African populations [1,2,4]. A meta-analysis of childhood and adolescent data places overall prevalence near 34%, with girls (about 32%) substantially more affected than boys (about 18%) [5,6]. Among college-aged adults the prevalence of GJH has been reported between roughly 11% and 26% [1], while the combined diagnosed prevalence of HSD and hEDS was about 1 in 500 in a national electronic cohort in Wales, with a mean diagnostic

delay differing between the sexes by some years; expert opinion regards both conditions as substantially under-diagnosed [1,2]. Within sport, the prevalence of GJH is markedly higher in disciplines that select for or cultivate flexibility — reaching roughly 65–72% in professional dancers and high values in gymnasts [1,7,8,9]. Because organised youth sport coincides with precisely the developmental window in which hypermobility is most common, clinicians frequently encounter young athletes in whom laxity is simultaneously an asset and a potential liability.

The question that frames this review is not new. Whether joint hypermobility is, for the athlete, an asset or a liability has been debated since the early observation that ballet dancers combine exceptional mobility with a propensity to injury [7,10]. The modern literature has not so much resolved this question as refined it, showing that the answer depends on which joint, which sport, which symptom status and which EDS subtype is in view. The same laxity that allows a dancer to achieve an extreme line, or a gymnast to absorb a landing, can leave a footballer's knee or an overhead athlete's shoulder dependent on active stabilisation that fails under load; and the same exercise that rehabilitates one hypermobile patient can endanger another with vascular EDS. A review that treats hypermobility as a single, uniform exposure will therefore inevitably produce contradictions; one that disaggregates by joint, symptom and subtype can recover a coherent and clinically usable picture, which is the aim pursued here.

The purpose of this narrative review is to bring together the medical evidence on this dual character of movement in hypermobile people, and to do so critically rather than descriptively. We first address terminology, classification and the problem of measurement, including a detailed appraisal of the Beighton score; we then summarise the natural history, pathophysiology and multisystem burden relevant to sport; we examine in turn "when movement harms" (injury risk across the knee and anterior cruciate ligament, the ankle, the shoulder, dance, gymnastics and team sports, and the asset-versus-liability debate); we examine "when movement helps" (proprioceptive, stabilisation and resistance training, multidisciplinary and paediatric programmes, physical-activity patterns and the psychological dimension); and we consider special situations — surgical management of the hypermobile shoulder, the neuroconnective phenotype, the relationship between trained flexibility and constitutional laxity, and the extreme case of vascular EDS. Throughout, the emphasis is comparative: the literature is heterogeneous, frequently under-powered, and dominated by an inconsistent definition of the very exposure it seeks to study.

Because the field is heterogeneous, this review is deliberately structured around the question that organises it — whether, and when, movement helps or harms — rather than around a single sport or intervention. We draw on the highest available levels of evidence (systematic reviews and meta-analyses), on prospective cohorts where they exist, and on case–control, cross-sectional and intervention studies where they do not, treating each according to its design and limitations. We make no claim to be exhaustive; rather, we aim to present a critical and comparative synthesis that an athlete, physiotherapist or sports physician could use to reason about an individual patient. The recurring analytical moves are three: to disaggregate hypermobility by joint, by symptom status and by EDS subtype; to weigh meta-analytic estimates against prospective cohorts and to explain their discrepancies; and to read the

"harm" and "help" literatures together, since the same pathophysiology that creates vulnerability also defines the therapeutic target.

2. Terminology, classification and the problem of measurement

2.1 From benign syndrome to a spectrum

The 2017 framework subdivides HSD into generalised (G-HSD), peripheral (P-HSD), localised (L-HSD) and historical (H-HSD) variants, each requiring JH of the relevant distribution plus at least one secondary musculoskeletal manifestation, and each requiring that hEDS be excluded [1,3]. The secondary manifestations include macro- and micro-trauma (dislocations, subluxations, soft-tissue injury), chronic pain, disturbed proprioception with muscle weakness, and other musculoskeletal traits such as pes planus, valgus deformities and mild scoliosis [1]. hEDS itself is diagnosed against three criteria: generalised JH by the Beighton score (with age-adjusted thresholds), a constellation of systemic connective-tissue features (unusually soft or hyperextensible skin, atrophic scarring, striae, arachnodactyly, recurrent hernias, pelvic-floor prolapse, mitral-valve prolapse or aortic-root dilatation), a positive family history, and recurrent dislocations or chronic widespread pain; alongside the mandatory exclusion of other heritable or acquired connective-tissue disorders [3]. In primary care the practical consequence is that the diagnosis is clinical and requires no laboratory confirmation; a positive family history may, by itself, shift a patient from G-HSD to hEDS [3].

A simple five-part questionnaire (the 5PQ) is widely used as a screening adjunct, asking whether the patient can place the hands flat on the floor with straight knees, bend the thumb to the forearm, contort the body or do the splits, has had a kneecap or shoulder dislocate more than once, or considers themselves double-jointed; two or more affirmative answers carry a sensitivity of about 84% and specificity of about 85% for the historical presence of JH [1,3]. For the sports clinician this taxonomy is more than pedantry: hEDS sits at the more symptomatic, multisystem edge of the spectrum, and identifying it changes the threshold for caution during training and competition, just as identifying a non-hypermobile EDS subtype (above all the vascular form) changes it absolutely [3,11].

2.2 The Beighton score: standard, but not a gold standard

Across virtually every study reviewed here, GJH is operationalised with the nine-point Beighton score — passive fifth-metacarpophalangeal dorsiflexion beyond 90°, thumb-to-forearm apposition, elbow and knee hyperextension beyond 10° (each scored bilaterally) and forward flexion of the trunk with the palms flat on the floor [2,4]. Generalised JH is conventionally indicated by a score of at least 4 in adults under 50, with the cut-off adjusted upward in children (≥6) and downward in older adults (≥4 over 50) [4]. Its strengths are real: it is quick (about five minutes), requires no equipment beyond a goniometer for equivocal joints, and is known and used worldwide [4]. Yet two careful appraisals argue that its routine diagnostic use is problematic, and their critique is directly relevant to the inconsistency of the sports-injury literature [2,4].

The score originated in 1964 as an epidemiological screening tool for an association between laxity and congenital hip dislocation, was modified by Beighton and colleagues in 1973 to study an African population, and has remained unchanged since, with no evidence-based

justification for either the joints selected or the cut-off applied [2]. Two-thirds of the items assess the upper limb; the shoulder, hip, patellofemoral joint and ankle — precisely the joints most often symptomatic in athletes — are not examined; only sagittal-plane motion is captured; and the system is "all-or-nothing", recording the presence but not the severity of laxity [2,4]. Malek and colleagues document that the score does not correlate with hypermobility of the shoulder (often the most troublesome joint in hEDS and a frequent first presentation through dislocation), shows inconsistent correlation with knee, ankle and temporomandibular-joint laxity, and correlates better only with the thumb and wrist [2]. The forward-flexion manoeuvre is further confounded by hamstring length and by muscle retractions, so that in one series 84% of confirmed hypermobile patients were unable to perform the very test that would have awarded them a point — an item whose sensitivity is so low (around 14%) that it arguably adds nothing to the score [2].

Reliability is only moderate: intra- and inter-rater kappa values cluster between 0.4 and 0.8, and a best-evidence synthesis using formal measurement standards rated the strength of evidence supporting a positive Beighton score as "limited to conflicting" [2,4]. Concurrent validity is judged poor to fair [4]. The score is also sensitive to circumstantial factors — dominance, prior warm-up, ambient temperature and menstrual-cycle phase — that can shift a borderline result [2]. The clinical implication, advanced most forcefully by Malek and colleagues, is that a negative score should not be used to exclude GJH, nor a positive score to confirm it, because the instrument neither directly samples the joints that define a generalised pattern nor distinguishes localised from generalised laxity; they illustrate the danger with a family in whom classical EDS was eventually confirmed molecularly despite Beighton scores of only 3 and 4 [2]. Engelbert and Rombaut reach a similar verdict, recommending that the score be supplemented by assessment of additional joints and by functional measures of stability, strength, proprioception, pain and participation, and noting that more comprehensive instruments such as the Upper and Lower Limb Assessment tools sample more joints in more planes [4].

The pattern of joint-by-joint validity is itself informative. The score correlates moderately with thumb and wrist laxity, inconsistently with the knee and ankle, and with spinal intervertebral mobility — though the forward-flexion item used to infer spinal hypermobility is trainable, as ballet dancers demonstrate, and is confounded by hamstring length [2]. For the temporomandibular joint, despite a recognised clinical association between GJH and temporomandibular disorders, most studies find no direct correlation between a positive Beighton score and measured joint hypermobility, plausibly because repeated subluxation eventually limits range and converts hypermobility into restriction [2]. The shoulder, the joint most often symptomatic in hEDS and a frequent first presentation through dislocation, is not assessed at all, and patients with strong clinical suspicion of EDS may have glenohumeral abduction 20° above normal yet a negative Beighton score [2]. These observations support the conclusion that the score functions best as an initial screen, after which the shoulder, hip, ankle and remaining digits should be examined directly before generalised hypermobility is either confirmed or excluded [2,4].

This measurement problem is not academic. Much of the apparent disagreement in the sports-injury literature is plausibly an artefact of how the exposure was defined: Pacey and

colleagues identified seven different objective measures of GJH, with ten differing measurement methods and several cut-offs, across the eighteen studies in their meta-analysis [12]. When the same threshold can capture quite different phenotypes, and when the instrument systematically ignores the shoulder and lower limb, the resulting heterogeneity is unsurprising — a theme to which we return throughout.

2.3 Natural history: laxity is dynamic, not fixed

Hypermobility is not static, and a single cross-sectional measurement can mislead longitudinal management. A two-year longitudinal study of 126 Nigerian schoolchildren used goniometry alongside the Beighton score and showed that the proportion classified as hypermobile (Beighton 7–9) fell from 25% to 13% to 6% over successive years, driven by reductions in elbow and knee range rather than in the fingers, whose range did not change [5]. The pattern of decline was very similar in boys and girls, and neither baseline muscle strength nor proprioception predicted the change, suggesting that natural tissue maturation rather than physical conditioning governs early childhood mobility [5]. Importantly, 19% of children actually became more mobile over the two years, a finding the authors flag as exceptional and as a possible warning sign warranting closer follow-up, since increasing rather than decreasing range may signal an underlying connective-tissue disorder [5]. The same study underscored the value of supplementing the dichotomous Beighton classification with goniometric "excess range of motion", which proved a more sensitive measure of change and revealed localised hypermobility in 28% of children classified as normally mobile [5].

Across the lifespan the dominant trend is one of decreasing laxity, classically described in three phases — a "hypermobility" phase in the first decade, marked by frequent sprains and strains; a "pain" phase in the second, characterised by widespread chronic pain with increasing instability but decreasing range; and a "stiffness" phase later, with a dramatic fall in joint mobility and diminished quality of life [2]. A cut-off age of around 33 has been proposed, beyond which many genuinely hypermobile patients no longer reach a Beighton score of 4 [2]. For longitudinal athlete management this means that a score obtained at one age cannot be assumed to hold at another, and that the trajectory of symptoms matters more than any single cross-sectional measurement — a point with direct bearing on whether and when sport helps or harms.

2.4 Distinguishing hypermobility from related and dangerous conditions

A diagnosis of HSD or hEDS is, by definition, one of exclusion, and for the sports physician the most important task is to rule out the heritable connective-tissue disorders in which hypermobility is accompanied by life-threatening features [1,3]. Marfan syndrome, Loeys–Dietz syndrome, osteogenesis imperfecta and — above all — vascular EDS share joint laxity but carry risks of aortic dilatation and dissection, arterial and organ rupture, or bone fragility that fundamentally alter the safety of sport [1,3,11]. Physical signs that should prompt referral and further investigation rather than reassurance include marked skin hyperextensibility or fragility, atrophic scarring, arachnodactyly or an arm-span-to-height ratio above 1.05, mitral-valve prolapse or aortic-root dilatation on echocardiography, and a family history of sudden death or arterial events [3]. Where such features are present, genetic evaluation is warranted, because the difference between hEDS — in which exercise is broadly beneficial — and vEDS

— in which it can be fatal — is not a matter of degree but of kind [11]. The clinical discipline of establishing the subtype before counselling on sport cannot be overstated, and it is the reason a careful systemic examination must accompany any Beighton score.

2.5 Epidemiology of hypermobility in athletic populations

Sport both selects for and may cultivate hypermobility, and prevalence figures in athletes therefore differ markedly from those in the general population. In a study of 132 elite adolescent athletes, GJH prevalence varied with the cut-off applied — 27.3% at Beighton ≥4, 15.9% at ≥5 and 6.8% at ≥6 — and with the discipline, being far higher in ballet dancers (68.2% at ≥4) and TeamGym gymnasts (24.6%) than in team-handball players (13.2%) [13]. Armstrong's comparison of 378 participants found GJH (Beighton ≥5 plus secondary manifestations) in about 70% of female dancers, 25% of netballers, 22% of female controls, 19% of female rugby players, but under 4% of male rugby players and under 2% of male controls, demonstrating both a strong sex effect and an activity effect [10]. Whether the high prevalence in dance reflects selection of hypermobile individuals into the discipline, or adaptation of the joints to its demands, remains unresolved and would require prospective study from childhood [10]. The practical message is that a Beighton score must be interpreted against the expected values for the athlete's sex and sport, and that training-induced range of motion can inflate the score without indicating systemic connective-tissue laxity [2,10].

3. Pathophysiology and the multisystem burden relevant to sport

3.1 Connective tissue, muscle and tendon

The mechanisms linking laxity to symptoms are multifactorial and incompletely understood, but several threads recur across the literature. At the tissue level, abnormal synthesis or organisation of collagen and other extracellular-matrix proteins — and in some cases of non-collagenous proteins such as tenascin-X — reduces the tensile strength of capsules, ligaments and tendons, increasing the demand placed on the active muscular stabilisers [1,3,14]. Increased tendon laxity may impair transmission of muscle-generated force, contributing to the reduced muscle strength and functional performance reported in some hypermobile cohorts, and two studies comparing asymptomatic GJH with symptomatic forms found decreased lower-limb joint momenta in the symptomatic group, necessitating greater force to maintain equilibrium [1]. Repetitive biomechanical overloading and silent micro-trauma of lax joints are thought to generate localised arthralgia (nociceptive pain) and, over time, diffuse musculoskeletal pain [1,3].

The genetic basis of these tissue differences is partly understood and partly not, with direct clinical implications. Most of the rarer EDS subtypes arise from identified mutations affecting fibrillar collagens (types I, III and V) or the enzymes that process them and the proteoglycans of the matrix, allowing molecular confirmation; hEDS, by contrast — the subtype most relevant to sport — has no known genetic marker and is diagnosed clinically, while both HSD and hEDS appear to follow a weak autosomal-dominant pattern with variable penetrance [1,3]. The absence of a molecular test for hEDS is why clinical assessment, family history and the exclusion of dangerous subtypes carry such weight, and why the Beighton score, for all its limitations, remains embedded in the diagnostic criteria [2,3]. The environmental contribution

is thought to involve localised biomechanical overloading and chronic soft-tissue micro-injury secondary to laxity and instability, which together drive the transition from intermittent arthralgia toward diffuse, centrally sensitised pain in susceptible individuals [1].

3.2 Proprioception and neuromuscular control

Proprioceptive impairment is among the most consistent findings, although not universal. A cross-sectional comparison of 83 adults reported significantly greater joint-position-sense error at the elbow and knee in hypermobile than in non-hypermobile participants (for example mean elbow error at 60° of 4.05° versus 2.48°), while grip strength and closed-kinetic-chain functional stability did not differ — a pattern the authors interpret as evidence of compensatory neuromuscular strategies that preserve gross function despite a sensorimotor deficit [15]. A meta-analysis cited within that work confirmed reduced joint proprioception in benign joint hypermobility syndrome, and a higher proprioceptive detection threshold at the knee has been linked to biomechanically unsound joint positions and thereby to injury risk [15]. In elite adolescent athletes, GJH was associated with significantly larger centre-of-pressure path length on every balance test, especially with the eyes closed (implicating proprioception rather than vision), even though injury rates, function and quality of life were not different from those of non-hypermobile peers [13]. The reconciliation is that a measurable deficit need not translate into adverse outcomes in a highly trained population whose conditioning compensates for it [13].

A crucial interpretive distinction, developed most clearly by Hanzlíková and colleagues, separates asymptomatic from symptomatic hypermobility. Studies that distinguish the two consistently find that neuromuscular and biomechanical differences — altered muscle activation during stair climbing, greater corticospinal excitability, poorer balance, biochemical markers of collagen degradation — are pronounced in symptomatic but not in asymptomatic individuals [16]. Asymptomatic hypermobile athletes appear to adapt, whereas symptomatic individuals carry the additional burden of pain that itself alters movement and reduces activity [16]. This dichotomy is arguably the single most important interpretive key to the field: pooling symptomatic and asymptomatic participants, as most older studies do, blurs precisely the contrast that determines whether movement helps or harms, and it explains why studies that restrict themselves to asymptomatic athletes so often find no excess risk [13,16].

3.3 Pain mechanisms: from nociception to central sensitisation

Pain in hypermobility is heterogeneous in mechanism. Nociceptive pain arises from structural change in joints, muscle and connective tissue; neuropathic features are reported in a substantial minority; and central sensitisation — an amplification of neural signalling that produces hypersensitivity to normal or sub-threshold input — appears to underlie the generalised hyperalgesia common in the syndrome [17,18]. Quantitative sensory testing in hEDS has demonstrated an increased wind-up ratio without somatosensory nerve damage, consistent with central rather than peripheral sensitisation, and the pain neuromatrix is thought to be overactive [17]. Patients with central sensitisation report more severe pain, poorer quality of life and greater disability, depression and anxiety than those with

predominantly nociceptive pain [17]. Chronic pain is itself the dominant and most disabling symptom of EDS, reported in roughly 90% of patients, frequently widespread, and a leading reason for seeking care; it does not feature among the diagnostic criteria yet profoundly degrades quality of life [18]. The clinical corollary, developed in the rehabilitation literature, is that interventions must address the central and cognitive dimensions of pain — body awareness, fear of movement, catastrophising — and not only peripheral tissue, a principle that shapes the "help" side of this review [17,18,19].

Olszak and colleagues describe how the pain of EDS takes several forms — nociceptive pain from structural change in joints, muscle and connective tissue; neuropathic pain from disturbed proprioception and muscle weakness; and central sensitisation — and how cognitive factors, excessive focus on negative sensations and unhealthy boom-and-bust activity patterns contribute to its chronicity [18]. They note that panic and anxiety increase the perceived pain burden, that successful management requires a multidisciplinary approach optimising treatment, education and lifestyle, and that physiotherapy, transcutaneous electrical nerve stimulation, trigger-point injection, low-dose naltrexone and cognitive-behavioural therapy have shown benefit [18]. Crucially, low-resistance exercise that increases core and limb muscle tone and improves joint stability is presented as a means of preventing the primary symptoms, reinforcing from the pain literature the same conclusion the rehabilitation literature reaches from the function literature: appropriately dosed movement is part of the treatment of pain, not a threat to be avoided [18].

3.4 Extra-articular and multisystem features

Because the connective-tissue defect is systemic, the burden of hypermobility extends far beyond the joints, and these features shape exercise tolerance directly. Autonomic dysfunction — orthostatic intolerance and postural orthostatic tachycardia syndrome (POTS) — is reported in a high proportion of patients (commonly cited at 40–75%, with autonomic symptoms in up to 78% in some series), and orthostatic intolerance limits upright and high-intensity exertion [1,18,20]. Functional gastrointestinal disorders (reflux, abdominal pain, constipation, early satiety, irritable-bowel symptoms), fatigue (with a prevalence of around 77–84%), sleep disturbance, pelvic-floor and bladder dysfunction, and a well-documented association with anxiety and depression are all over-represented [1,3,18]. Olszak and colleagues catalogue the breadth of organ involvement — neurological (migraine, craniocervical instability, Chiari malformation, tethered cord), respiratory (dyspnoea, pneumothorax in the vascular subtype), cardiovascular (mitral-valve prolapse, varices, easy bruising, orthostatic hypotension), gastrointestinal and obstetric — and emphasise that successful management of this multisystem disease is a major challenge requiring multidisciplinary collaboration [18]. For the sports physician, the practical point is that a hypermobile athlete reporting fatigue, dizziness, palpitations or gut symptoms is not necessarily presenting with unrelated complaints but may be manifesting the systemic phenotype, and that exercise prescription must account for autonomic and fatigue limitations rather than treating the joints in isolation [18,20].

3.5 Muscle strength, fatigue and the deconditioning cycle

Whether hypermobility entails a true strength deficit is contested, and the answer again depends on whether participants are symptomatic. Some studies of symptomatic GJH report reduced muscle strength and impaired functional performance, attributable in part to the difficulty of transmitting muscle-generated force through lax tendons and to protective avoidance of dynamic loading [1,21]. The cross-sectional analysis of functional consequences by Scheper and colleagues found measurable reductions in strength and physical function in symptomatic GJH, whereas comparisons of asymptomatic GJH with controls have frequently shown no difference [15,21]. Akaras and colleagues, studying 83 young, physically active adults, found that hypermobile participants performed no worse on grip strength or on closed-kinetic-chain upper- and lower-extremity stability tests, despite their proprioceptive deficit, and even showed a positive correlation between Beighton score and grip strength — a finding the authors interpreted cautiously as reflecting compensatory neuromuscular adaptation in a young, active sample rather than an inherent strength advantage [15]. The reconciliation, once more, is that conditioning and symptom status, not laxity in the abstract, determine functional capacity.

Fatigue compounds the strength question. Fatigue is among the most prevalent symptoms of HSD and hEDS, reported in roughly three-quarters of patients, and it both reduces activity and, through deconditioning, worsens the very weakness and instability that provoke symptoms [18]. In hypermobile dancers, fatigue levels are higher than in non-hypermobile dancers, and neuromuscular control, proprioception and functional stability deteriorate after fatigue, suggesting that the lax athlete operates with a narrower reserve and is more vulnerable late in training or competition [10]. The clinical consequence is a self-reinforcing cycle: pain and fear reduce activity; inactivity reduces strength, aerobic fitness and proprioceptive acuity and worsens fatigue and autonomic symptoms; and these in turn deepen avoidance [1,22,23]. Recognising this cycle reframes the therapeutic goal — not to protect the lax athlete from movement but to rebuild the strength, endurance and confidence that interrupt the spiral — and it explains why graded, supervised exercise is so consistently beneficial in the intervention literature [22,24,25].

4. When movement harms: hypermobility and sports injury

The intuition that lax joints are vulnerable joints is old, and it is partly correct. Dislocations, subluxations and sprains are reported more often in people with GJH, and it is reasonable to expect that physically demanding activity magnifies that risk [1,12]. Yet the empirical picture is markedly joint-specific and confounded by the heterogeneity of exposure definitions, sports and injury criteria discussed above. We consider the knee and anterior cruciate ligament, the ankle, the shoulder, dance and gymnastics, and team and contact sports in turn, and then the broader asset-versus-liability debate and the quality of the underlying evidence.

4.1 Knee and anterior cruciate ligament

The strongest and most coherent signal concerns the knee. The systematic review and meta-analysis by Pacey and colleagues screened 4,841 studies, included 18, and pooled data using a standardised Beighton cut-off; it found a significantly increased risk of knee-joint injury in hypermobile participants during contact sport, with a combined odds ratio of 4.69 (95% CI

1.33–16.52), while no increased risk was found for the ankle [12]. The categorisation of participants into not hypermobile, hypermobile and extremely hypermobile showed a statistically significant gradient for knee injury during contact sport ($p < 0.001$) [12]. The authors explained the knee–ankle contrast anatomically: the ankle is restrained by both active musculotendinous structures and passive ligaments and by the bony congruency of the talocrural joint, whereas the knee — particularly against rotational, valgus and varus forces — relies far more heavily on passive ligamentous and capsular restraint, leaving it disproportionately exposed when that restraint is lax [12]. A separate systematic review by Sundemo and colleagues concluded that GJH increases ACL injury risk and is associated with inferior outcomes after ACL reconstruction, including higher rates of graft failure and revision [26].

Prospective cohort data, however, temper this picture and illustrate the limits of the evidence. In a 4.5-year study of 287 female team-sport athletes (basketball, floorball, ice hockey, volleyball), Pasanen and colleagues recorded 23 magnetic-resonance-confirmed non-contact ACL injuries and found that GJH was not associated with risk (odds ratio 1.10, 95% CI 0.80–1.51); neither anterior–posterior knee laxity, genu recurvatum, femoral anteversion, hamstring extensibility nor navicular drop predicted injury on univariate logistic regression [27]. The investigators noted that some earlier studies — for example the military cohort of Uhorchak and a youth basketball and handball cohort — had reached differing conclusions, that only a minority used prospective designs, and that their own study was powered only for moderate-to-strong associations, so that detecting small risk associations would have required roughly 200 injury cases rather than 23 [27]. This explicit acknowledgement of under-power is instructive: ACL injuries are rare events, individual cohorts are frequently under-powered, and pooled estimates are sensitive to how laxity is defined and which sports are included. The honest synthesis is that GJH probably raises knee and ACL injury risk in contact sport, but the effect is modest, joint- and context-specific, and easily obscured in any single cohort.

The biomechanical reasoning that Pacey and colleagues advanced deserves emphasis because it makes the joint-specificity intelligible rather than arbitrary [12]. At the knee, there is little bony congruency and the surrounding musculature offers minimal active restraint to rotation and to varus–valgus force; most knee injuries occur at the end of extension range or involve unrestrained rotation, valgus or varus, all of which depend almost entirely on the passive cruciate, collateral and capsular restraints that hypermobility weakens [12]. When the foot is fixed to the ground — as in studded footwear during contact sport — the internal torques generated at the knee exceed those at the ankle because of the greater distance from the ground, further predisposing the lax knee to injury [12]. The hypermobile athlete may rely more heavily on dynamic muscular control to stabilise lax lower-limb joints than non-hypermobile peers, placing them at greater risk of both musculotendinous and capsuloligamentous injury when that control is overwhelmed [12]. This mechanistic account also predicts the therapeutic target: strengthening and neuromuscular control to replace the deficient passive restraint, precisely the intervention the “movement helps” evidence validates [12,24].

The implications of GJH for the surgically reconstructed knee deserve separate mention, because they extend the harm beyond the index injury. The systematic review by Sundemo

and colleagues concluded not only that GJH raises the risk of ACL injury but also that it is associated with inferior outcomes after reconstruction, and the football cohort observed that patients undergoing revision reconstruction carry higher generalised-laxity scores on average than those undergoing primary surgery [26,28]. Other reports cited within these studies link greater knee hyperextension and laxity to graft failure and to poorer postoperative function, and one prospective cohort found that hypermobile patients were less likely to return to sport and showed less symmetrical knee-extension strength two years after reconstruction [16,26,28]. For the hypermobile athlete this means that the consequences of an ACL rupture may be more prolonged and the rehabilitation more demanding than for a non-hypermobile peer, strengthening the case for prevention through neuromuscular and landing training, and for realistic counselling about return-to-sport timelines after reconstruction [16,26].

4.2 Ankle and chronic ankle instability

Consistent with Pacey's meta-analysis, the weight of evidence does not support an excess of ankle injury attributable to GJH, and a systematic review of ankle-sprain predictors found that it was reduced — not increased — ankle dorsiflexion range that predicted sprains [12]. A case–control study of 47 Japanese high-school volleyball players did find that athletes with a history of ligament injury scored higher on the Beighton and Horan joint-mobility index (mean 2.40 versus 1.24, $p = 0.006$), and that those with multiple or recurrent injuries scored higher still than those with a single injury (3.18 versus 1.95, $p = 0.02$) [29]; but almost all injuries in that retrospective sample were ankle sprains, ankle laxity is not part of the index used, and the cross-sectional design cannot establish the direction of causation [29]. The authors themselves concluded only that hypermobile athletes may be more prone to recurrent sprains and might benefit from a prevention programme, and called for prospective study [29].

Critically, generalised laxity does not appear to compromise rehabilitation of the ankle. In a prospective cohort of 40 patients with chronic ankle instability randomised by hypermobility status, those with GJH achieved equal or even better outcomes after a three-month supervised balance-training programme than non-hypermobile patients: a lower re-sprain ratio (11.1% versus 23.5% immediately after training, and 16.7% versus 29.4% at three months), greater gains in sport-specific function and plantar- and dorsiflexion strength, and comparable improvements in postural control [30]. The investigators concluded that GJH is not a contraindication to conservative management and that balance training should be offered before surgery is considered — directly contradicting the assumption that lax ankles cannot be rehabilitated and reinforcing the "movement helps" theme [30].

4.3 Shoulder

For the shoulder the association with injury is robust, and the data simultaneously expose the inadequacy of the Beighton score. A meta-analysis of six observational studies (2,335 athletes, 34% female, mean age about 20 years) reported threefold higher odds of shoulder injury in athletes with JH (odds ratio 3.25, 95% CI 1.64–6.43) [31]. The magnitude depended entirely on how hypermobility was defined: using GJH the odds ratio was 1.97 (95% CI 1.32–2.94), whereas using a shoulder-specific measure of joint hypermobility it rose to 8.23 (95% CI 3.63–18.66), and exposure definition explained the great majority of the substantial between-study heterogeneity [31]. Studies with low risk of bias showed the strongest associations [31].

This dissociation is an indictment of the Beighton score, which does not examine the shoulder and therefore captures local glenohumeral laxity only by the questionable assumption that all joints are equally affected [2,31]. The clinical inference is to assess the shoulder directly — for example with an apprehension or relocation test, an external-rotation range beyond 85°, or self-reported prior instability — rather than relying on a global GJH score when counselling overhead and contact athletes [31]. The overall quality of evidence was nonetheless rated low, so future research may revise the estimate [31].

Not every shoulder pathology, however, follows laxity, and the contrast is illuminating. In a study of 124 patients with subacromial impingement syndrome or adhesive capsulitis, neither condition was associated with GJH or with benign joint hypermobility syndrome; indeed the adhesive-capsulitis group had significantly lower Beighton scores than controls, prompting the speculation — consistent with an earlier report — that a degree of laxity might even protect against the capsular fibrosis of frozen shoulder [32]. The plausible reconciliation is that hypermobility predisposes specifically to instability-type injury (dislocation, subluxation, labral and capsular failure), not to the fibrotic or impingement pathologies that arise from stiffness; mixing these distinct mechanisms under a single heading of "shoulder pain" would obscure a real and clinically important specificity [31,32].

4.4 Dance and aesthetic disciplines

Dance is the paradigm of hypermobility as an asset, and the evidence here is genuinely two-sided. Prevalence of GJH in elite and pre-professional dancers is high — around 65–72% in several series, and the highest of any group in comparative studies — and both selection of hypermobile individuals into dance and adaptation of the joints to its extreme range have been proposed [7,8,10,13]. Aesthetic demands may favour the selection of hypermobile dancers, and within ballet a high range of motion is explicitly prized [10]. Yet the largest and best-designed prospective cohorts question whether laxity translates into injury. Van Rijn and Stubbe followed 185 pre-professional contemporary dancers across four annual cohorts, screening musculoskeletal function at entry and collecting monthly health questionnaires with a 90% response rate; the one-year injury incidence was high (67.6% for all complaints, 54.6% for time-loss injuries), but across four different Beighton cut-offs GJH was not associated with any injury definition in either univariate or multivariate analysis [7]. The only consistent predictor was a previous long-lasting injury (odds ratios around 2.6–3.2), leading the authors to recommend that injury history, not the Beighton score, guide screening of dancers [7]. Their finding aligns with an earlier prospective study by Roussel and with Armstrong's conclusion that a total Beighton score is only a weak predictor of time-loss injury in dancers [7,10].

Armstrong's comparison of rugby players, netballers, dancers and controls (378 participants) found that dancers had by far the highest Beighton scores (mean 4.89) and GJH prevalence (about 70%), the highest mean count of hEDS criteria, and the highest prevalence of pain and dislocation or subluxation among hypermobile participants; five women across the whole sample met criteria for hEDS, three of them dancers [10]. The same author's work on young gymnasts similarly framed hypermobility as carrying implications for both injury and performance, and noted that hypermobile dancers stand with hyperextended joints that make

maintaining a position difficult, that fatigue levels are higher in hypermobile dancers, and that neuromuscular control, proprioception and functional stability deteriorate after fatigue — pointing to work-to-rest ratios and conditioning rather than range of motion as the appropriate focus of injury prevention [9,10]. These findings have generated a critical literature questioning whether the health risks of GJH are adequately understood within the classical-ballet narrative, where dancers may continue to perform in pain and so deny injuries the opportunity to heal [10,33].

The prevalence data themselves carry a caution about screening in dance. In a cross-sectional study of elite Australian dancers, the prevalence of generalised and syndromic hypermobility varied widely with the cut-off applied, and the wide range reported across the dance literature — from roughly 2% to over 80% depending on the Beighton threshold and the population — reflects more about methodology than about biology [7,8]. This variability matters because a screening programme that classifies the great majority of a ballet company as hypermobile conveys little prognostic information, and because a single cut-off cannot serve a population in which extreme range is the professional norm [2,8]. The recommendation that emerges is to assess injury history and symptom burden, and to examine joints beyond the Beighton battery, rather than to rely on a global score that, in dancers, may be positive in most and predictive in few [7,8].

Reassuringly for the long term, a five-year follow-up of professional ballet dancers reported that JH did not increase the risk of developing hip pain, cartilage defects or premature retirement [34]. The tension between high pain and dislocation prevalence on the one hand, and preserved careers and unproven injury excess on the other, captures the asset-versus-liability paradox at the heart of aesthetic sport: in an elite, highly trained and self-selected population the immediate symptomatic burden of laxity is high, yet its long-term articular consequences may be limited, and the trait may be a genuine performance enabler [10,34].

4.5 Gymnastics and other flexibility-dependent sports

Gymnastics resembles dance in selecting for flexibility, and Armstrong's study of young gymnasts framed joint hypermobility as having dual implications for injury and performance [9]. In the elite-adolescent cohort of Schmidt and colleagues, TeamGym gymnasts had a GJH prevalence (24.6% at Beighton ≥4) intermediate between dancers and team-handball players, and the authors speculated that hypermobility may be advantageous in younger gymnasts, for whom the trampoline and tumbling are less demanding, but may cease to be an asset as the requirements for muscular strength, power and stability increase with age and competitive level [13]. This developmental nuance — laxity helpful early, potentially limiting later — mirrors the natural-history data showing that range of motion declines through adolescence and that the demands of high-level performance shift toward active stabilisation [2,13].

4.6 Team and contact sports

In team and contact sport the evidence is again mixed and frequently null, particularly when participants are asymptomatic and well conditioned. A two-year prospective cohort of 73 NCAA Division I American-football players found that a pre-season diagnosis of GJH (present in 9.6%) did not increase the number of injuries, musculoskeletal issues, treatment

episodes, days unavailable or surgeries; there were no significant between-group differences in injuries per athlete (3.0 versus 4.1) or per game, and the authors concluded that no specific pre-participation risk counselling was warranted for football players with GJH [28]. The study also produced an intriguing observation: quarterbacks had the highest mean Beighton score (2.8) and GJH rate (60%) while no linemen were hypermobile, and athletes with GJH actually appeared in significantly fewer games than their peers — hinting that laxity tracks with playing position and the physical demands a player self-selects into, and that position is a confounder of any crude association between laxity and injury [28]. The authors situated their null finding within a divided older literature in which some contact-sport studies (soccer, rugby, martial arts) reported increased injury and others (lacrosse, netball) did not [28].

In adolescent female volleyball, a study that explicitly separated asymptomatic hypermobility from symptomatic forms — excluding syndromic and symptomatic participants — compared 22 asymptomatic hypermobile and 28 non-hypermobile players on the Landing Error Scoring System, single-leg dynamic balance on a force plate, and limb-symmetry indices for single and triple hops [16]. No significant differences emerged on most measures; the only group difference favoured the hypermobile players, who showed greater single-leg-hop symmetry, and a weak negative correlation suggested slightly better medio-lateral balance with higher Beighton scores [16]. The authors concluded that, in the absence of symptoms, hypermobility alone may not necessitate specialised injury-prevention training, and emphasised that symptomatology is the important differentiating factor in neuromuscular control [16]. This is perhaps the cleanest demonstration in the literature that the conflation of symptomatic and asymptomatic laxity, not laxity itself, drives the appearance of risk.

Soccer provides a further null example that is often overlooked. A prospective cohort of elite female soccer players found no effect of generalised joint hypermobility on injury risk, and this result is cited across the subsequent literature as evidence that, in a conditioned female cohort, laxity is not an independent predictor of injury [27,28,30]. Combined with the football and volleyball findings, it builds a consistent picture for the conditioned team-sport athlete: where participants are screened for symptoms, well trained and studied prospectively, generalised hypermobility tends not to emerge as a meaningful risk factor, in contrast to the elevated pooled estimates derived from heterogeneous, often retrospective samples that mix symptomatic and asymptomatic individuals [12,27,28]. The reconciliation, once more, is that the conditioned, asymptomatic athlete has adapted, and that the risk concentrated in meta-analyses is carried disproportionately by symptomatic and syndromic individuals and by the specific vulnerability of the knee and shoulder in contact and overhead disciplines [12,16,31].

Among the 132 elite adolescent athletes of Schmidt and colleagues, injury frequency, health-related quality of life and functional muscle performance did not differ between those with and without GJH, even though the hypermobile group swayed significantly more on every balance test [13]. The authors hypothesised that the high training volume of elite athletes (6.5–10.5 hours per week) may be protective, buffering the theoretical vulnerability conferred by laxity and even, in some studies, conferring superior balance through a protective training effect [13]. Taken together, the team- and contact-sport data suggest that asymptomatic GJH in a conditioned athlete is a weak and inconsistent risk factor, and that symptom status,

training load, playing position and joint-specific laxity are far more informative than a global Beighton score.

Rugby and netball illustrate the variability across team sports. In rugby, where physical contact and tackling are intrinsic, hypermobility has been proposed as a risk factor, and Armstrong noted that male rugby players carried the highest prevalence of peripheral joint hypermobility and a high frequency of pain and dislocation among those with GJH, plausibly reflecting the injury exposure of a collision sport [10]. In netball, a non-contact but high-demand sport, a trend toward impaired movement control has been reported in players with GJH, and peripheral hypermobility of the fingers and thumbs may even constitute a performance adaptation for handling the ball [10]. Across Armstrong's cohort the dominant signals were a strong sex effect — GJH far more common in women — and an activity effect, with dancers most affected; the author concluded that decisions on injury prevention, training load and even sport selection should consider the athlete's sex and predominant activity, so that a Beighton score is interpreted against expected values within that domain rather than as an absolute risk marker [10]. This call for sport- and sex-specific interpretation is a recurring and well-justified theme.

4.7 The asset-versus-liability debate and the quality of the evidence

The recurring inconsistency in this literature is not merely empirical noise; it reflects genuine methodological limitations that deserve explicit appraisal. First, the exposure is defined differently across studies — seven instruments and multiple cut-offs in Pacey's review alone, with upper-limb-weighted scales such as Beighton and lower-limb-weighted scales such as Nicholas raising questions about the face validity of either as a measure of whole-body mobility [12]. Second, most cohorts are under-powered for rare outcomes such as ACL rupture, so that null prospective results (in football, in female soccer, in contemporary dance) coexist with positive meta-analytic estimates without genuine contradiction [7,27,28]. Third, the symptomatic–asymptomatic distinction, which the pathophysiology identifies as decisive, is rarely made [16]. Fourth, retrospective and cross-sectional designs predominate in some joints (the volleyball ankle data, for instance), precluding causal inference [29]. Fifth, definitions of "injury" range from arthroscopically confirmed ligament rupture to self-reported time loss, further fragmenting comparability [12]. Sixth, sports differ widely in contact demand, and Pacey's data suggest GJH may even be protective in some limited-contact and non-contact activities while raising knee-injury risk in contact ones [12].

The honest synthesis is therefore conditional rather than categorical. Generalised laxity raises knee and shoulder injury risk in contact and overhead sport, especially when it is symptomatic or syndromic and when shoulder-specific laxity is present, but it is a weak or absent risk factor for the ankle and for asymptomatic, well-trained athletes, and it may even be advantageous for performance in flexibility-dependent disciplines. This nuanced, joint- and symptom-specific conclusion is far more defensible than any blanket statement that hypermobility is dangerous in sport, and it sets up the complementary and more uniformly positive evidence on the therapeutic value of movement.

Table 1. Selected studies of hypermobility and sports-injury risk.

Study [ref.]	Population / design	Exposure	Key finding
Pacey et al. [12]	18 studies, meta-analysis	GJH (standardised ≥4/9)	Knee injury OR 4.69 in contact sport; no excess ankle risk
Liaghat et al. [31]	6 studies, 2,335 athletes, meta-analysis	GJH / shoulder JH	Shoulder injury OR 3.25; GJH OR 1.97 vs shoulder-specific OR 8.23
Sundemo et al. [26]	Systematic review	GJH	Higher ACL injury risk; inferior outcome after reconstruction
Pasanen et al. [27]	287 female athletes, 4.5-y prospective	GJH (Beighton)	No association with non-contact ACL injury (OR 1.10)
Nicolay et al. [28]	73 NCAA football, 2-y prospective	GJH ≥4/9 (9.6%)	No excess injury; quarterbacks most hypermobile; fewer games
Sueyoshi et al. [29]	47 volleyball, case–control	Joint-laxity index	Injured/recurrently injured scored higher; mostly ankle sprains
Hanzlíková et al. [16]	50 volleyball, cross-sectional	Asymptomatic GJH	No worse landing/balance/symmetry; better hop symmetry
Atici et al. [32]	124 shoulder patients, case–control	GJH / BJHS	No association; adhesive capsulitis had lower Beighton scores
Schmidt et al. [13]	132 elite adolescent athletes	GJH (3 cut-offs)	Greater sway; no difference in injury, function or HRQoL
van Rijn & Stubbe [7]	185 pre-professional dancers, prospective	GJH (4 cut-offs)	GJH not associated with injury; prior injury predictive
Armstrong [10]	378 athletes/controls, cross-sectional	GJH / hEDS criteria	Dancers highest Beighton (4.89) and GJH (~70%); 5 met hEDS criteria

5. When movement helps: exercise, rehabilitation and physical activity

If the harm side of the ledger is genuinely two-sided, the help side is more uniformly positive. Across HSD and hEDS, physical activity and structured rehabilitation are repeatedly associated with reduced pain, improved function, lower kinesiophobia and better quality of life, while inactivity — often driven by fear of injury — perpetuates deconditioning, fatigue and symptom amplification [22,23,25]. The challenge is therefore not whether to prescribe movement but how to dose, supervise and individualise it, and how to dismantle the fear that keeps hypermobile people sedentary.

5.1 Proprioceptive and neuromuscular training

Because impaired proprioception and deficient active joint stabilisation are core deficits, the most consistently recommended interventions target muscle control and balance rather than further stretching of already lax joints [15,22]. A systematic review of physical therapy for hEDS identified muscle training to enhance proprioception as the dominant strategy, with the goal of restoring lower-limb motor control and balance; every trial examining pain or

proprioception reported benefit in the intervention group, and benefit was seen independently of the specific technique used, suggesting that the act of structured, supervised exercise matters more than its precise form [25]. In chronic ankle instability complicated by GJH, supervised balance training improved postural control and muscle strength as effectively in hypermobile as in non-hypermobile patients, with a lower re-sprain ratio in the hypermobile group, indicating that laxity does not blunt the response to sensorimotor rehabilitation [30].

The GoodHope Exercise and Rehabilitation (GEAR) programme exemplifies a structured, individualised model designed specifically for EDS and generalised HSD [22]. Delivered by physiotherapists and kinesiologists, it begins with a two-hour assessment, prescribes an eight-week individualised home programme using conventional frequency–intensity–time–type parameters and a modified rating of perceived exertion, and crucially teaches neuromuscular stabilisation before resistance work — progressing from closed to open kinetic chain, from non-weight-bearing to weight-bearing, from bilateral to unilateral, and from supported to unstable surfaces [22]. It explicitly screens for orthostatic symptoms before prescribing aerobic activity, embeds self-management education to reduce kinesiophobia and manage pain flares, and links patients to community resources to sustain the gains beyond the programme [22]. A systematic review of exercise interventions in EDS, cited within the GEAR description, identified eleven studies showing benefit for muscle strength, proprioceptive acuity and quality of life, providing the evidential rationale for such structured care [22].

The content of such programmes reflects their stabilisation-first philosophy. In the GEAR model, neuromuscular exercises are taught and trained before resistance work to ensure effective muscle activation, joint stabilisation and posture, and patients are quickly progressed to resistance training as control develops [22]. Aerobic prescriptions emphasise endurance-building activity within the patient's orthostatic tolerance — walking, hydrotherapy, recumbent cycling, swimming — while high-impact, stop-and-go activities are limited where lower-limb arthritis is present [22]. Resistance exercise progresses from supportive postures with body weight or light resistance toward more demanding positions and greater load, always using neuromuscular principles to avoid aberrant movement such as joint hyperextension, and stretching is encouraged only within normative range as a warm-up rather than as an end in itself [22]. This explicit avoidance of end-range loading in already lax joints, combined with graded strengthening and proprioceptive challenge, captures the consensus of the rehabilitation literature and stands in deliberate contrast to the flexibility-seeking culture of the aesthetic sports [22,25].

5.2 Resistance and heavy-strength training

A persistent clinical belief holds that heavy resistance training is inappropriate, even dangerous, in hypermobile people, for fear of pain flares, injury or joint damage. The best available data directly contradict this belief. Henriksen and colleagues delivered a twelve-week supervised, progressive heavy-resistance programme (leg press, calf raise, leg extension, leg curl, forward lunge, performed to failure) to sixteen young women with knee-joint hypermobility and knee pain [24]. The intervention was well tolerated, with excellent adherence (all completed at least 88% of sessions) and no major adverse events; pain during the most aggravating activity fell by a mean of 32.5 mm on a 100-mm scale (95% CI 21.4–

43.6), comfortably exceeding the 15–20 mm regarded as clinically important [24]. Every participant reported less pain, and the improvements were accompanied by gains across all strength measures (39–82% on five-repetition-maximum tests), in knee proprioception (mean joint-position error reduced by 55%, to values comparable with non-hypermobile populations) and in patellar-tendon stiffness, alongside reductions in kinesiophobia on the Tampa scale [24]. Several participants ultimately lifted very heavy loads — more than 200 kg on the leg press in some cases [24].

Several details of the Henriksen programme are worth drawing out because they overturn specific clinical fears. Acute pain flares induced by each session were low and did not increase systematically over the twelve weeks; the few participants who developed transient knee or hip pain were managed by temporary, case-by-case reductions in range or intensity, after which the prescribed load was restored, usually within a single session [24]. Patellar-tendon stiffness rose in eleven of sixteen participants by about 11% on average, and rate of torque development — a protective compensatory factor against symptomatic laxity — improved in thirteen of sixteen [24]. The participants and supervising clinicians consistently identified supervision and encouragement as important to adherence and confidence [24]. The study cannot, as a case series, prove efficacy, and the authors call for randomised comparison with usual care; but it provides strong proof of tolerability and a clear signal of benefit in exactly the population — young women with symptomatic knee-joint hypermobility — in whom heavy loading is most feared [24]. The practical message is that fear of resistance training in hypermobility is largely unwarranted, provided loading is supervised, progressed sensibly and individualised.

The mechanistic rationale is coherent: increasing maximal strength, rate of force development and tendon stiffness enhances the active stabilisation on which lax joints disproportionately depend, improving control of limb position and force transmission [24]. Consistent with this, narrative and review syntheses now argue that resistance training is not only feasible but beneficial in hEDS and HSD, provided it is supervised, progressed cautiously to avoid micro-trauma in weakened tissue, and combined with cognitive-behavioural strategies to address fear of movement; the literature even suggests that individuals with hypermobility strengthen at rates similar to controls [25]. A randomised comparison of wrist-stabilising exercise versus hand-orthosis use in 169 adults with hEDS found that both interventions produced comparable benefits for pain, paraesthesia, grip strength and quality of life, indicating that structured stabilisation exercise is a legitimate alternative to passive support [25]. This convergence — that supervised strengthening, including heavier loading where tolerated, is safe and effective — is one of the clearest and most clinically useful conclusions in the field.

5.3 Multidisciplinary and paediatric programmes

In children the evidence is more sobering and underlines how much remains unproven. The randomised Bendy Study compared an eight-week individualised multidisciplinary, multisite programme (bespoke physiotherapy and occupational therapy in clinic, home and school) with standard care — advice, education and therapy referral as needed — in 119 children with symptomatic hypermobility [35]. Both groups improved significantly in child- and parent-reported pain, coordination and grip strength over twelve months, but the intensive

intervention conferred no additional benefit over standard care, and the response was independent of the degree of hypermobility, with baseline pain rather than Beighton score predicting improvement [35]. The authors concluded that, for the majority of children, education and positive, exercise-promoting self-management are sufficient, and that more complex and costly intervention is not routinely required; they speculated that reduced anxiety following diagnosis and reassurance may itself drive much of the improvement [35].

These paediatric findings should not be misread as evidence that exercise is ineffective in children. Both arms of the Bendy Study improved, and the comparison was between an intensive multidisciplinary package and an active standard-care arm that itself included education, advice and therapy referral — not against no treatment [35]. The interpretation is that the essential ingredients (education, reassurance, encouragement of healthy activity and self-management) are delivered effectively in standard care, and that the additional resource-intensive components do not add measurable benefit for most children [35]. Earlier work cited within these reviews similarly found that children improve in pain over follow-up regardless of the particular management strategy, and that quality-of-life scores in symptomatic hypermobile children, while affected, often remain within the normal reference range, suggesting that the condition tends not to limit overall well-being to the degree clinicians might fear [14,35,36]. The constructive message is that simple, accessible, exercise-promoting care should be the default for children, that intensive intervention should be reserved for the subgroup who do not respond, and that diffusing unhelpful beliefs about disability is itself therapeutic [35,36].

A systematic review of physical and mechanical therapies for lower-limb problems in hypermobile children reinforced this caution: it identified only two randomised trials (86 participants total), found no clear benefit of generalised over targeted physiotherapy nor of exercising into the neutral versus the hypermobile range of knee extension, and noted that mechanical therapies — orthoses, splints, taping, footwear — had never been evaluated in a randomised design despite being used commonly in practice [14]. Sample sizes at completion fell below the numbers required by the trials' own power calculations, placing them at high risk of attrition bias [14]. These paediatric data are a caution against over-treatment and a call for adequately powered trials, but they do not undermine the general message that movement and education help; rather, they suggest that simple, low-cost approaches may suffice for most children and that the burden of proof for intensive intervention has not been met [14,35].

For adults, a neurocognitive rehabilitation approach focused on body awareness and pain perception — drawing on the principles of narrative medicine and the recognition of central sensitisation — produced statistically significant reductions in pain (numerical rating scale and McGill questionnaire), fatigue, kinesiophobia and pain-associated disability in eighteen hEDS patients with chronic low-back pain, with a minimal clinically important pain reduction achieved by 88.9% of participants [17]. This pilot illustrates that interventions addressing the central, sensorimotor and cognitive dimensions of pain — and not only peripheral strengthening — have a place in management, and that helping patients to "re-perceive" movement can reduce the fear that drives avoidance [17]. More broadly, syntheses of rehabilitation in EDS conclude that tailored programmes combining proprioceptive and stabilisation exercise with adjuncts such as kinesiotaping or compression garments reduce

pain and improve function; that strength training should be introduced with particular caution because of prolonged tissue-healing times; that exercise can safely reduce the symptoms of co-existing POTS when individualised; and that a nine-week rehabilitation programme improved six-minute-walk distance, reduced kinesiophobia and increased energy, with benefits sustained at six months [25].

5.4 Physical-activity patterns, fatigue and quality of life

Objective data confirm that hypermobile young people are less active than their peers, with consequences for health. Using thigh-worn accelerometry over seven days, Schubert-Hjalmarsson and colleagues compared 37 adolescents with HSD/hEDS and 45 healthy controls and found that the hypermobile group spent significantly more time sedentary and less time in moderate-to-vigorous physical activity (median 20 versus 32 minutes per day) and moved significantly more during sleep; neither group met the recommended 60 minutes of daily moderate-to-vigorous activity [23]. The hypermobile adolescents reported far more fatigue and pain catastrophising and near-universal chronic pain [23]. Although the exploratory and probably under-powered design could not confirm an association between fatigue and daytime activity, it did demonstrate a strong relationship between fatigue and pain catastrophising, pointing to the psychological mediation of inactivity and to the importance of addressing catastrophising, sleep and anxiety alongside physical conditioning [23].

The relationship between autonomic dysfunction and exercise adds a further layer. Co-existing POTS, common in hEDS, is itself responsive to carefully graded aerobic reconditioning, and exercise training has been shown in non-EDS populations to bring the majority of POTS patients into remission; rehabilitation programmes for HSD/hEDS increasingly aim to extend these benefits while screening for orthostatic symptoms before and during exertion [22,25]. This means that the fatigue and exercise intolerance of the hypermobile patient are not necessarily fixed barriers but are themselves partly treatable through the same graded activity that addresses the joints, provided the programme respects autonomic limits and progresses slowly [20,22,25]. The therapeutic logic is therefore unified: graded movement simultaneously rebuilds strength, refines proprioception, reconditions the autonomic system and reduces fear, attacking the deconditioning cycle from several directions at once.

That the field still lacks robust activity guidance is made explicit by a registered scoping-review protocol, which observes that previous reviews focused narrowly on physiotherapy-based exercise and excluded structured sport, unstructured play and household activity, younger children and older adults, and the influence of female hormonal transitions (puberty, the menstrual cycle, pregnancy, menopause) on symptoms and injury risk [37]. The recurring clinical picture across these sources is a vicious cycle: pain and fear of injury reduce activity, inactivity worsens deconditioning, fatigue and orthostatic intolerance, and these in turn reinforce avoidance and further reduce participation [22,23,37]. Breaking this cycle — through graded, confidence-building, supervised activity — is the central therapeutic task, and it is precisely what the intervention evidence above supports.

5.5 The psychological dimension and kinesiophobia

Psychological factors are not peripheral to exercise prescription in this population; they are central. A systematic review of psychological interventions in HSD/EDS examined six studies (628 enrolled, 343 analysed, predominantly female) and found that the most effective approaches were those delivered alongside physiotherapy, which improved both the physical and the psychological components of pain — pain intensity, interference, pain-related fear and catastrophising — and, through them, quality of life [19]. An isolated positive-psychology programme improved affect and life satisfaction, and benefited most those patients who helped design their own intervention, but had only a short-term effect on physical disability [19]. Inpatient programmes were less effective than outpatient or community delivery, suggesting that intensive residential care is neither necessary nor cost-effective [19]. Pain catastrophising and pain-related fear emerged as the most modifiable targets — one programme reduced catastrophising by about 32% with sustained effect at five months — and reductions in kinesiophobia accompanied improvements in function across several studies [17,19].

The diagnostic journey itself shapes the psychological state in which a hypermobile athlete arrives at rehabilitation, and clinicians should be alert to it. Patients with HSD and hEDS frequently describe long, distressing diagnostic odysseys — sometimes spanning many years from the onset of childhood symptoms — during which they are dismissed, misdiagnosed and mistreated, and during which trust in healthcare providers erodes [18,19]. Attending physiotherapy can itself provoke or heighten anxiety when previous interactions were unhelpful or when exercise was not individualised [19]. Recognising this history matters because the therapeutic alliance, goal-setting, reconceptualising beliefs about the body, and fostering self-efficacy are integral to successful management, and because a patient who has been told for years that their pain is unexplained may need active reassurance that movement is safe before they will engage with it [19]. The corollary for sport is that returning a hypermobile athlete to activity is as much a matter of rebuilding confidence as of rebuilding tissue, and that dismissive or fear-inducing messaging from clinicians and coaches can itself become a barrier to participation [18,19].

This evidence justifies the now-standard recommendation that exercise for hypermobile patients be “psychologically informed”: graded, confidence-building and explicitly designed to dismantle fear of movement rather than simply to load tissue [19,22]. The review’s authors call for psychologically informed physiotherapy and for greater training of physiotherapists in cognitive-behavioural and behaviour-change techniques, while acknowledging that the evidence base rests on small studies, only one of which was a randomised trial, and that adequately powered trials with long-term follow-up are needed [19]. The broader lesson, consistent with the pain-mechanism data, is that chronic pain in EDS is a biopsychosocial phenomenon, that roughly 90% of patients live with it, and that its management demands multidisciplinary collaboration rather than pharmacology alone [18].

5.6 Quality of life, body awareness and the burden of inactivity

Beyond pain and injury, hypermobility affects quality of life and the lived experience of movement, and these dimensions inform why activity matters. A longitudinal study of school-aged children reported that generalised JH had a measurable impact on quality of life and on

physical-activity participation, reinforcing the picture from accelerometry that hypermobile young people are less active and more symptomatic than their peers [38]. In young adults with HSD, investigation of body awareness, fatigue, physical fitness and musculoskeletal problems found these domains to be interrelated, with altered body awareness and fatigue accompanying reduced fitness and more frequent complaints [39]. Such findings matter for exercise prescription because they identify the targets — body awareness, confidence, fatigue management — that a purely biomechanical programme would miss, and they align with the neurocognitive and psychologically informed approaches that the intervention evidence supports [17,19,39]. The consistent thread is that inactivity is not a neutral default in hypermobility but an active contributor to morbidity, and that restoring participation is itself a therapeutic outcome [23,38].

5.7 What is the safe dose? The gap in physical-activity guidance

Despite the consistent message that movement helps, the literature is candid about how little is known regarding the optimal type, intensity and volume of activity, particularly outside the physiotherapy clinic. The registered scoping-review protocol of Golden and colleagues was motivated precisely by this gap: prior reviews concentrated on therapeutic exercise within physical therapy and neglected structured sport, occupational activity, unstructured play and household movement, as well as age-specific considerations across the lifespan and the influence of female hormonal transitions [37]. The protocol frames safety in terms of adverse events — falls, strains, sprains, subluxations, dislocations, cardiovascular and orthostatic events — mapped against benefits across the domains of body function, activity and participation [37]. Until such syntheses mature, the practical default supported by the existing evidence is graded, supervised, individualised activity that begins at low intensity, prioritises stabilisation and proprioception, progresses cautiously, and is adjusted for pain, fatigue and orthostatic tolerance — an approach that the GEAR model and the rehabilitation reviews operationalise in detail [22,25,37]. The honest position is that movement should be encouraged and dosed conservatively, not prescribed by a precise, evidence-based formula that does not yet exist.

5.8 Mechanical adjuncts and self-management

Alongside active exercise, a range of mechanical and self-management adjuncts is used to support lax joints, although the evidence for them is weaker than for exercise itself. Bracing, splinting, neuromuscular taping and kinesiotaping are employed to stabilise joints, reduce arthralgia and subluxation, and provide additional proprioceptive input, and compression garments may help both joint support and the orthostatic intolerance of co-existing autonomic dysfunction [1,3,25]. Orthotic shoe inserts can improve lower-limb alignment and proprioception and reduce the risk of sprains in some patients [3]. Within structured programmes such as GEAR, splints, braces and gait aids are incorporated selectively for patients with significant instability, and patients are taught pain-relieving positions, energy-conservation strategies and counter-pressure manoeuvres for orthostatic symptoms as part of self-management education [22]. Stasiak and colleagues note that adjuncts such as kinesiotaping and compression garments can alleviate pain, improve function and mitigate

instability-related symptoms when combined with exercise [25]. The important caveat, established by the paediatric systematic review, is that mechanical therapies — orthoses, splints, taping and footwear — have never been evaluated in randomised trials in hypermobile children despite their common use, so their role rests on clinical rationale rather than high-level evidence and should complement, not replace, active rehabilitation [14]. Self-management education aimed at reducing fear, pacing activity and managing flares is, by contrast, a consistent feature of the programmes that work [22,25,35].

Table 2. Selected studies of exercise and rehabilitation in hypermobility.

Study [ref.]	Design / population	Intervention	Main outcome
Mittal et al. [22]	Programme description (GEAR)	Multi-component rehab + education	Model for individualised, supervised, graded exercise care
Henriksen et al. [24]	Case series, 16 women, knee JH + pain	12-wk heavy resistance	Pain −32.5 mm; ↑strength, proprioception, tendon stiffness; well tolerated
Hou et al. [30]	Prospective cohort, 40 CAI patients	3-mo balance training	GJH group equal/better; lower re-sprain (11% vs 24%)
Bale et al. [35]	RCT, 119 children	Multidisciplinary vs standard care	Both improved; no added benefit of intensive programme
Celletti et al. [17]	Pilot, 18 hEDS + low-back pain	Neurocognitive rehabilitation	↓pain, fatigue, kinesiophobia, disability
Peterson et al. [14]	Systematic review, 2 RCTs, 86 children	Physical/mechanical therapies	No clear benefit of any single approach; mechanical therapies untested
Schubert-Hjalmarsson et al. [23]	37 HSD/hEDS vs 45 controls, accelerometry	Observational	More sedentary, less MVPA (20 vs 32 min/day), more sleep movement
Clark et al. [19]	Systematic review, 6 studies	Psychological ± physiotherapy	Combined approach most effective for pain and QoL
Stasiak et al. [25]	Narrative review	Exercise / rehabilitation in EDS	PA improves QoL, pain, function; resistance feasible with caution

6. Special considerations and high-risk situations

6.1 The hypermobile shoulder and the limits of surgery

When conservative, proprioceptively focused management of the unstable hEDS shoulder fails, surgery is considered, but the evidence base is strikingly thin and the results sobering. A systematic review of glenohumeral surgery for instability and pain in hEDS concluded that standard procedures effective in the general population — Bankart repair and the Latarjet coracoid transfer — give only good short-term results and questionable long-term durability in hEDS, because the inherent tissue laxity favours recurrent instability and the Latarjet addresses only anterior instability while hEDS patients typically present with multidirectional instability [40]. Soft-tissue insufficiency, structural skin instability, intra-operative bleeding, post-operative haematoma and delayed wound healing attributable to fibroblast dysfunction all complicate surgery in this population [40]. The review surveyed a range of alternatives — arthroscopic capsular plication, posterior and circumferential bone-block augmentation, Achilles- and tibialis-tendon allograft reconstruction, open capsular shift, 270° capsulorrhaphy, thermal capsulorrhaphy (now largely abandoned because of chondrolysis), arthroplasty and, as a last resort, arthrodesis — but found no studies on large hEDS cohorts and could not identify a clearly superior technique [40].

The arc of surgical strategy in the hEDS shoulder reflects this uncertainty. Because soft-tissue procedures fail more often when the capsule and ligaments are intrinsically lax, attention has shifted toward techniques that augment bony restraint or replace deficient tissue — bone-block augmentation that increases the articular surface without relying on soft-tissue stabilisation, and allograft reconstruction that substitutes lax, scarred capsule with more durable collagenous tissue — while thermal capsulorrhaphy, once promoted as minimally invasive, has been largely abandoned because of severe complications including chondrolysis [40]. Even these alternatives carry technique-specific drawbacks, from graft resorption and donor-site complications (to which hEDS patients are particularly prone) to the persistence of multidirectional instability that an anterior-only procedure cannot address [40]. Salvage operations such as arthroplasty and arthrodesis are reserved for repeated failure and severe joint deterioration, and even arthroplasty in hEDS, while feasible, carries a substantial complication rate [40]. The cumulative impression is of a field improvising in the absence of high-level evidence, which is itself the strongest argument for exhausting conservative, exercise-based management and for concentrating surgery in experienced hands [40].

The authors emphasised that the rarity of hEDS, the absence of long-term controlled trials and the documented excess of surgical failure and complications mean that management must be individualised, undertaken by surgeons experienced with connective-tissue disease, and preceded by informed consent that acknowledges the heightened surgical risk [40]. The clinical message reinforces the rehabilitation literature: because surgery is uncertain and complication-prone, prevention and conservative, stabilisation-focused management — improving scapulohumeral coordination, correcting scapular dyskinesia, and strengthening the periscapular and rotator-cuff musculature — are preferable wherever possible, and recurrent dislocation should not lead reflexively to operation [25,40]. This is a domain where “movement” (in the form of targeted exercise) is not merely safer than surgery but, for many patients, the better-evidenced option.

6.2 The neuroconnective phenotype: hypermobility, dysautonomia and neurodevelopment

An emerging body of work links GJH and hEDS to neurodevelopmental and autonomic conditions, of direct relevance to athletes presenting with unexplained somatic symptoms. A systematic review of the attention-deficit/hyperactivity disorder (ADHD)–hypermobility connection summarised studies reporting markedly increased prevalence of GJH in people with ADHD — for example 32% versus 14% in one early study, 74% versus 13% in another, and an odds ratio of about 4.7 in a large case–control comparison that rose to 6.9 when symptomatic GJH was considered — and a nationwide cohort confirming increased psychiatric morbidity in people with EDS or hypermobility relative to their non-affected siblings [20]. The review proposed a unifying "neuroconnective endophenotype" in which joint hypermobility, autonomic dysfunction and neurodevelopmental traits co-occur [20]. Proposed mechanisms include collagen abnormalities affecting central-nervous-system structure (for example white-matter integrity and the corpus callosum), autonomic dysregulation such as POTS (present in 40–50% of hEDS patients) amplifying inattention, fatigue and anxiety, altered proprioceptive feedback degrading body awareness and motor coordination, chronic pain and sensory hypersensitivity impairing attention, and shared dopaminergic dysfunction affecting motor control, attention and impulsivity [20].

For the sports physician this matters because such athletes may report fatigue, brain fog, orthostatic intolerance, sleep disturbance and anxiety alongside their joint complaints, and because recognising the cluster encourages an integrated rather than a purely orthopaedic approach, including screening hypermobile patients for autonomic dysfunction when unexplained somatic symptoms are present [20]. The clinical value is in connecting symptoms that might otherwise be treated in isolation, and in tailoring exercise to accommodate autonomic and cognitive features — for example pacing, attention to orthostatic tolerance, and the use of routine and structure — rather than prescribing exercise as though the joints were the only system involved [20].

6.3 Vascular EDS: the extreme of "when movement harms"

At the far end of the risk spectrum lies vascular EDS (vEDS), where the calculus of movement is fundamentally and qualitatively different. vEDS accounts for roughly 5% of EDS, is caused by heterozygous pathogenic variants in COL3A1 encoding type III collagen, and weakens the walls of arteries and hollow organs; about half of patients suffer a major arterial complication in their lifetime, more than 40% experience recurrent vascular events, and median life expectancy is roughly 40–50 years, with arterial rupture the leading cause of death [11]. A narrative review of exercise- and sport-related injury in vEDS analysed reported cases and found that both competitive sport and seemingly trivial activity — casual arm stretching, recreational basketball, wrestling, rowing, general play and even passive hamstring stretching during post-stroke rehabilitation — have precipitated catastrophic, sometimes fatal vascular events, frequently in previously undiagnosed adolescents and young adults (median age 16, predominantly male) [11]. Emergency surgery was required in the majority, two patients died, and one survivor underwent above-elbow amputation [11].

The individual cases collated in that review make the danger concrete. They include an eleven-year-old girl who ruptured a popliteal-artery aneurysm while playing; a young student-athlete who developed exercise-induced myocarditis after intense rowing-machine exercise; a twelve-year-old boy who suffered active haemorrhage from an axillary-artery tear after casual arm stretching, ultimately requiring above-elbow amputation; an eighteen-year-old man who died of thoracic-aortic dissection after a minor fall in recreational basketball; a sixteen-year-old who developed a subclavian-artery pseudoaneurysm during wrestling; a fifteen-year-old who died of a vascular catastrophe after feeling a "pop" while playing basketball; and a stroke patient who sustained intramuscular haemorrhage during routine passive hamstring stretching in rehabilitation [11]. In the majority the diagnosis of vEDS was unknown at the time of the event, and emergency surgery was complicated by extreme tissue fragility [11]. These cases underline two points: that even supervised, low-intensity rehabilitation manoeuvres can precipitate catastrophe in vEDS, and that early diagnosis and individualised emergency planning are not optional refinements but life-saving necessities in this subtype [11].

The implication is not that vEDS patients should be entirely sedentary — appropriate, supervised, low-load activity may support function and quality of life — but that exercise prescription must prioritise safety and individualisation [11]. Acceptable activities include walking at a conversational pace, gentle cycling and supervised swimming; high-intensity interval training, competitive and contact sport, sprinting, endurance events, heavy resistance and isometric exertion are discouraged because of the transient blood-pressure surges that can precipitate aneurysm expansion or dissection [11]. Pharmacological blood-pressure control (commonly beta-blockers, including celiprolol) and individualised emergency planning, including a "vEDS passport" documenting the diagnosis, are central to care [11]. vEDS thus stands as the clearest counterpoint to the rest of this review: in most hypermobility, movement is therapeutic and should be actively encouraged, but in this rare subtype the very same activity can be lethal, and the distinction between EDS subtypes is therefore not academic but life-determining [11]. It is precisely for this reason that establishing the subtype must precede any counselling on sport.

6.4 Trained flexibility versus constitutional laxity

Because hypermobility and trained flexibility are easily conflated, it is worth distinguishing them, particularly in dancers and gymnasts whose acquired range can inflate a Beighton score [2,10]. Flexibility is one of the basic physical qualities; it develops most intensively up to roughly 14–15 years of age, with sensitive periods for passive flexibility around 9–10 and for active flexibility around 10–14 years, and it can be improved by stretching even into middle age, whereas pathological hypermobility reflects constitutional connective-tissue laxity rather than trainable muscle and tendon length [41,42]. Systematic stretching increases range of motion and is associated with increased muscle strength and endurance, reduced and prevented muscle pain, better posture and coordination, and accelerated recovery, but ballistic stretching carries a higher injury risk and is generally reserved for advanced, professional contexts [41]. A controlled study of an annual cycle of stretching classes in adolescent girls produced statistically significant gains in active flexibility and in cervical, thoracic and lumbar spinal mobility, demonstrating that flexibility is genuinely trainable and that its development is an element of injury prevention [42].

The relevance to hypermobility is twofold. First, training-induced flexibility (as in dancers, gymnasts and stretching-trained students) can produce high Beighton scores without indicating systemic laxity, complicating screening and reinforcing the argument that the score be interpreted against sport- and sex-specific expectations rather than as a fixed diagnostic threshold [2,10,42]. Second, in a genuinely hypermobile athlete the therapeutic emphasis should shift away from further stretching of already lax joints — which adds little and may aggravate instability — and towards strengthening, proprioceptive training and active stabilisation, exactly the interventions the "movement helps" evidence supports [3,22,24]. The proper role of stretching in this population is targeted at tight muscles, not lax joints, a distinction repeatedly stressed in the management literature [3,18].

6.5 Sex, age and hormonal influences

Hypermobility is consistently more prevalent in females and, in athletic medicine, the female athlete is over-represented among the hypermobile [2,5,10]. The reasons are partly hormonal: ligamentous laxity varies across the menstrual cycle and increases around puberty, and these fluctuations may influence both symptom severity and injury risk [2,37]. Female athletes are also the group in whom ACL injury is most studied, and although the largest prospective cohort found GJH unassociated with non-contact ACL injury in women, the broader literature on sex differences in knee laxity and injury keeps the question open [27]. The scoping-review protocol of Golden and colleagues makes the influence of female sex hormones — across puberty, the menstrual cycle, pregnancy and menopause — an explicit research priority, noting that some women report more frequent injury and worsening pain during hormonal transitions and improvement after menopause, yet no previous review of exercise in HSD/hEDS had addressed it [37]. Age modifies the picture as well: laxity declines through adolescence and adulthood, the demands of high-level sport shift from flexibility toward strength and stability with maturation, and the same trait that aids a young gymnast may limit an older one [2,13]. Sex- and age-specific counselling is therefore appropriate, even though the evidence to make it precise is still developing [37].

6.6 Return to sport, load management and prevention

Although no large trial has tested a hypermobility-specific return-to-sport protocol, the principles that emerge from the reviewed evidence are coherent. Because injured hypermobile athletes may heal more slowly and may sustain greater tissue damage before an injury is reported, a cautious, criterion-based progression rather than a time-based one is prudent, with particular attention to restoring proprioception and active stabilisation before return [10,29]. Because fatigue degrades neuromuscular control disproportionately in hypermobile athletes, load and work-to-rest ratios deserve explicit management, especially late in training and competition when most injuries occur [10]. Because shoulder-specific and knee laxity carry the clearest risk, prevention should target these joints directly — scapular and rotator-cuff stabilisation for the overhead athlete, neuromuscular and landing training for the cutting athlete — rather than applying a generic programme [31,40]. And because asymptomatic, well-conditioned athletes show little excess risk, blanket restriction of sport is unwarranted and may itself be harmful by promoting deconditioning [13,16,28]. The overarching aim is to

keep hypermobile athletes active and progressing while managing the specific, joint- and symptom-defined risks that the evidence actually supports.

7. Practical implications for athletes and clinicians

Several practical principles follow from this synthesis. First, screening should not rest on the Beighton score alone; a negative score does not exclude clinically relevant laxity, and the shoulder, hip, patellofemoral joint and ankle — together with the presence and pattern of symptoms — should be assessed directly, particularly in overhead and contact athletes, since shoulder-specific laxity carries far higher injury odds than a global GJH classification [2,4,31]. Second, risk stratification should be guided by symptom status and EDS subtype rather than by laxity per se: asymptomatic, well-conditioned athletes carry little excess injury risk and rarely need specialised programmes, whereas symptomatic or syndromic individuals warrant closer attention and may benefit from targeted prevention [13,16,28]. Third, exercise should be prescribed, not withheld; the dominant therapeutic strategy is graded, supervised proprioceptive and stabilisation training, with progressive resistance — including heavier loading where tolerated — introduced cautiously, all delivered within a psychologically informed framework that explicitly targets kinesiophobia [19,22,24,25].

Fourth, management should be multidisciplinary and individualised, integrating physiotherapy, pain management and, where relevant, psychological and autonomic care, because pain, fatigue, autonomic and neurodevelopmental features cluster and interact and cannot be managed in isolation [18,19,20]. Fifth, the EDS subtype must be established before any counselling on sport, since vascular EDS demands restriction of high-intensity, contact and isometric activity that would be entirely appropriate, indeed beneficial, for most other hypermobile people [11]. Sixth, in children, simple education and self-management appear sufficient for the majority, and intensive multidisciplinary intervention has not been shown to add benefit, so that over-treatment should be avoided [14,35]. Seventh, stretching should be directed at tight muscles rather than at already lax joints, and the therapeutic emphasis in hypermobile athletes should be active stabilisation rather than further mobilisation [3,41].

It may help to render these principles as a sequence the clinician can follow. The first step is to establish whether hypermobility is present and generalised, using the Beighton score as a screen but examining the shoulder, hip, patellofemoral joint and ankle directly and taking a careful history of dislocations, pain and family connective-tissue disease [2,4]. The second step is to exclude the dangerous heritable disorders — vascular EDS above all, but also Marfan and Loeys–Dietz syndromes and osteogenesis imperfecta — by looking for skin fragility, arachnodactyly, cardiovascular signs and a family history of arterial events, and by referring for genetic evaluation when such features are present [3,11]. The third step is to determine symptom status, since asymptomatic, well-conditioned athletes need reassurance and general conditioning rather than restriction, whereas symptomatic individuals need targeted assessment of the painful or unstable joints [13,16,28]. The fourth step is to prescribe graded, supervised, stabilisation-first exercise, progressing to resistance and sport-specific training as control improves, within a psychologically informed framework and with attention to fatigue and orthostatic tolerance [19,22,24,25]. The fifth step is to monitor over time, recognising that laxity and symptoms change with age and that the trajectory matters more

than any single measurement [2,5]. This sequence operationalises the review's central message: individualise by subtype and symptom, encourage movement, and manage the specific risks the evidence supports.

These recommendations are necessarily provisional. They rest on a literature dominated by small, heterogeneous and frequently under-powered studies, by inconsistent definitions of the exposure, and by a paucity of adequately powered randomised trials — a gap acknowledged across the field, from paediatric physiotherapy and surgical management to physical-activity guidance [14,35,37,40]. Where the evidence is strongest — the joint-specificity of injury risk, the tolerability and benefit of supervised strengthening, the centrality of proprioception and of fear of movement, the danger of vascular EDS — it converges on a coherent and clinically usable picture. Where it is weakest — optimal exercise dosing across subtypes and ages, the role of mechanical adjuncts, the influence of hormonal transitions — the appropriate response is cautious individualisation rather than prohibition, and the systematic collection of better data.

8. Methodological appraisal and research priorities

A narrative review of this field would be incomplete without an explicit reckoning with the quality of its evidence, because the inconsistencies catalogued above are largely a product of that quality. Four limitations recur. The first is the definition of the exposure. Generalised JH has been measured with at least seven instruments and numerous cut-offs, with upper-limb-weighted scales such as the Beighton score sitting alongside lower-limb-weighted scales such as the Nicholas index, and with the same numerical threshold capturing materially different phenotypes [2,12]. The Beighton score itself, as detailed in Section 2, lacks validated cut-offs, ignores the shoulder and most lower-limb joints, samples only the sagittal plane, and shows only moderate reliability, so that a study's conclusions may depend as much on its measurement choices as on biology [2,4]. Standardising and broadening the assessment of hypermobility — and reporting it transparently — is the single most important methodological reform the field requires.

The second limitation is statistical power. Serious sports injuries, above all ACL rupture and shoulder dislocation, are rare, and individual cohorts are frequently too small to detect the modest effects that meta-analyses suggest. Pasanen and colleagues were explicit that their 287-athlete cohort, with 23 ACL injuries, was powered only for moderate-to-strong associations and would have needed roughly 200 cases to detect smaller ones [27]. Null prospective findings in football, female soccer and contemporary dance therefore do not refute positive pooled estimates; they reflect the difficulty of studying rare outcomes in finite cohorts [7,27,28]. The third limitation is the conflation of symptomatic with asymptomatic hypermobility. The pathophysiology and the cleanest studies agree that symptom status is decisive, yet most older work pools the two, blurring the very contrast that determines harm [16]. The fourth is design and outcome heterogeneity: retrospective and cross-sectional studies predominate for some joints, definitions of injury range from arthroscopic confirmation to self-reported time loss, and intervention trials are small, often non-randomised and short in follow-up [12,19,35].

From these limitations a research agenda follows directly. Future studies should adopt a standardised, multi-joint assessment of hypermobility and report it in full; should separate symptomatic from asymptomatic phenotypes a priori; should power their cohorts adequately for the rare injury outcomes they seek, ideally through multicentre collaboration; should favour prospective designs with objective, consensus injury definitions; and should test exercise interventions — including resistance dosing, proprioceptive programmes and psychologically informed care — in adequately powered randomised trials with long-term follow-up across the relevant subtypes and age groups [14,19,27]. Specific gaps deserving attention include the influence of female hormonal transitions on injury and symptoms, the still-untested role of mechanical adjuncts such as orthoses and bracing, hypermobility-specific return-to-sport criteria, and the long-term articular and surgical outcomes of hypermobile athletes [14,37,40]. Only with such data can the conditional, individualised recommendations offered here be placed on firm ground.

9. Conclusions

Hypermobility occupies an unusual position in sports medicine because the same trait can be, simultaneously, an asset and a liability. The evidence reviewed here resolves much of the apparent contradiction by being specific rather than categorical. Movement harms chiefly when laxity is symptomatic or syndromic, when it affects the knee or the shoulder in contact and overhead sport, and — in the extreme and rare case of vascular EDS — when it stresses fragile vessels; it is a weak or absent risk factor for the ankle and for asymptomatic, well-trained athletes, and it may even enable performance in flexibility-dependent disciplines. Movement helps, more uniformly, when it is graded, supervised and symptom-guided: proprioceptive and stabilisation training, progressive and even heavy resistance, balance training after ankle injury, and psychologically informed rehabilitation reduce pain and kinesiophobia and improve function and quality of life, whereas inactivity perpetuates a self-reinforcing cycle of pain, fear, fatigue and deconditioning.

Two further conclusions deserve emphasis because they cut across the harm-and-help dichotomy. The first concerns measurement: until the assessment of hypermobility is standardised and broadened beyond the Beighton score, and until studies separate symptomatic from asymptomatic phenotypes, the field will continue to generate apparent contradictions that are artefacts of method rather than reflections of biology. The second concerns the subtype distinction: the gulf between hypermobile EDS, in which movement is overwhelmingly beneficial, and vascular EDS, in which the same movement can be fatal, means that no recommendation about sport can be made responsibly without first establishing which condition is present. These are not peripheral caveats but central to the safe and effective management of the hypermobile athlete, and they should guide both clinical practice and the design of future research [2,11].

The Beighton score, though indispensable as a screen, is too narrow — too upper-limb-weighted, too insensitive to the shoulder and lower limb, too binary — to carry the diagnostic and prognostic weight often placed on it, and much of the field's inconsistency is traceable to how the exposure has been defined and to the routine conflation of symptomatic with asymptomatic laxity. The most defensible reading of the literature is therefore conditional and

individualised. The practical conclusion for athletes and clinicians is neither encouragement nor prohibition in the abstract, but individualised, multidisciplinary, subtype- and symptom-stratified care — a clinical posture in which the operative question is not whether hypermobile people should move, but how, how much, and under what supervision. Future research should standardise the assessment of hypermobility, separate symptomatic from asymptomatic phenotypes, power its cohorts for rare injury outcomes, and test exercise dosing across subtypes and across the lifespan, so that the still-provisional recommendations offered here can be placed on firmer ground.

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