

Anatomy of an Injury: A spectrum of systemic challenges

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Headline

Various biomechanical (physical), biochemical (metabolic), and neuro-psychological stressors drive a wide spectrum of sports injuries. Asymptomatic pre-injuries frequently precede detectable signs of neuromuscular and other dysfunctions. Consequently, localized joint or muscle pain is often a secondary manifestation of complex systemic imbalances in the brain, metabolism, or gut, or from early overtraining. Complete recovery encompasses acute and chronic physiological stages, concluding only when the underlying causes of dysfunction are resolved, allowing the athlete to safely return to full training and competition.

Aim

This paper explores the full spectrum of injuries, emphasizing an integrated approach to identify performance impairments before symptom onset. Because physical dysfunctions rarely occur in isolation, they require brain-body system evaluations to facilitate targeted treatment and recovery. For example, a compromised gut microbiome—often driven by excess refined carbohydrates—can trigger systemic inflammation and skeletal muscle dysfunction, which can promote joint pain. Ultimately, this holistic approach is essential for preventing injuries, averting recurrences, and optimizing physical performance.

Injury is physiological damage causing localized or systemic dysfunction. In addition to joint pain, other examples include cardiac injury (heart damage), brain injury (neurological dysfunction), gut injury (microbiome disruptions), and burn injury. Most athletic injuries involve multifactorial interactions linked to underlying stressors or risk factors. Even simple issues, such as knee pain, can be part of a larger kinematic chain of events. For example, impairment in the ankle can create dysfunction and pain in the knee (1). The ankle may remain asymptomatic, while the knee develops acute inflammation—a biochemical response to localized stress and a key part of healing. Furthermore, the mental-emotional stress of pain, lack of rest, or continued training can worsen an injury or trigger a new one. Pain can be highly deceptive; by the time symptoms appear, the injury process may have been ongoing for days, weeks, or months, often starting silently in another part of the body. Whether simple or complex, recovery is achieved only when the underlying physiological dysfunctions are resolved, and the athlete can return to unhindered training and peak competition.

To compensate for an injury, the body relies on complex biomechanical, biochemical, and neurological activities. These adjustments alter movement patterns to maintain stability while attempting to heal the injury and prevent further damage. Consequently, these compensations often lead to noticeable training and performance impairments, including:

- Elevated submaximal heart rate (HR).
- Reduced eye-hand/foot coordination.
- Impaired motor control and reaction times.
- Gait irregularity.

- Changes in mood and focus (2, 3).

This model of sports injuries builds upon Hans Selye's classic work on stress (4). Three key stressors—biomechanical, biochemical, and the brain/mental-emotional—elicit a shared physiological response. Because they frequently occur together, these stressors can initiate a vicious cycle that triggers and sustains injuries while preventing complete recovery. Traumatic injuries share this same model, influencing the severity of the trauma, the rate of recovery, and the risk of chronicity and recurrence (5).

Preinjury: Asymptomatic dysfunction

Even before an obvious injury with accompanying symptoms, underlying risk factors or "preinjuries" can produce detectable signs of functional impairment (6). These range from subtle, localized abnormal joint movements to global conditions like irregular gait or autonomic dysfunction, all of which reduce optimal performance. In some cases of early overtraining, however, an abnormally elevated sympathetic state can temporarily increase muscle function, producing a sudden, noteworthy surge in performance.

A preinjury often results from cumulative stressors that can include daily training load, single high-intensity events, or a lengthy competitive season. This predisposes athletes to further damage that ultimately leads to classical signs and symptoms. Common examples of preinjuries include:

- **Improper biomechanics:** Poorly fitting or overly supportive shoes that impair normal foot mechanics.
- **Systemic inflammation:** Body-wide, low-grade chronic inflammation.
- **Inadequate recovery:** Poor recovery protocols between training sessions or competitions.
- **Neuromuscular imbalances:** Asymptomatic imbalances in the foot and leg.

Neuromuscular imbalance may be the most prevalent preinjury, combining abnormal inhibition (an overstretched or inhibited muscle) and an over-facilitated (tight) muscle (7, 8). These mechanisms are part of the neuromusculoskeletal system—an integrated network of muscles, bones, and nerves sustained by metabolic processes and regulated by the central nervous system (9). Muscles actuate bony levers via tendons while stabilizing joints, ligaments and other soft tissues. When neuromuscular activation or coordination is impaired even slightly, a preinjury state can occur.

While often asymptomatic, the subjective feeling of a preinjury may be vague including mild discomfort, achiness, or a sense that something is "just not right." It can reduce ranges of motion, impair poor eye-hand/foot coordination, promote inflammation, and other subtle changes. The use of non-steroidal anti-inflammatory or other drugs can mask

these sensations. Though difficult to differentiate from normal training stress, these dysfunctions often resolve with extended rest. Additional rest becomes a key therapeutic and preventive strategy, otherwise impaired physical function and performance can continue. Neuromuscular imbalances are typically assessed through manual muscle testing, gait analysis, or similar evaluations (10).

Global Injuries

While any localized neuromusculoskeletal imbalance can ultimately affect the whole body, systemic or global athletic injuries can manifest as full-body impairment. Examples include chronic fatigue, myalgia, and generalized muscle soreness. These complaints are often difficult to explain, with standard medical tests revealing little to no abnormalities, yet they can significantly reduce athletic performance.

Overtraining

A primary injury affecting virtually all bodily systems can include the overtraining syndrome (OTS), a complex, systemic

condition resulting from accumulated physical, biochemical, and brain (mental-emotional) stress. Classically known to impair immunity, increase inflammation, and adversely affect gut and adrenal functions, OTS ultimately reduces all athletic performance (11, 12). Initial symptoms can be vague or nonspecific, presenting as persistent physical and mental fatigue and irregular appetite, with a common early sign being elevated resting, training, and competitive HR (13). Neuromuscular imbalance can also lead to a gradual progression or rapid onset of physical injuries. In some cases, a sudden, temporary surge in performance is an early indicator of overtraining. This occurs when an athlete transitions from functional to nonfunctional overreaching, where excess sympathetic tone briefly enhances neuromuscular function. Continued training and competition with insufficient recovery worsens OTS leading to more classical signs and symptoms.

Table 1 outlines a three-stage model of the OTS, whose onset coincides with the transition from healthy to unhealthy training. Recovery times are estimates based on assessment, treatment, and adherence to training and lifestyle recommendations.

Table 1. The overtraining syndrome

Overtraining:	Stage 1	Stage 2	Stage 3
Alternative terms:	Nonfunctional over-reaching; Acute stress.	Sympathetic OT; Excess stress.	Parasympathetic OT; Chronic OT
Performance response:	↑ Training stress and or ↓ recovery.	↓ Training and or competitive performance.	All performance greatly diminished.
Symptoms:	Fatigue, hunger, soreness; ↑ need for recovery & sleep.	↑ Fatigue, pain, daytime sleepiness, depression/ anxiety; crave sweets, caffeine.	Reduced training and competitive motivation; worsening symptoms.
Signs:	↓ Submax performance. ↑ resting HR; poor sleep quality & quantity	↑ Cortisol & sympathetic tone; hormone imbalance; ↑ resting and exercise HR. ↑ Illness and injury.	↑ Injury, illness; ↓ sympathetic and parasympathetic tone. ↓ Resting HR.
Actions:	↓ Training intensity and or volume; ↑ sleep/ recovery; address diet and other stressors.	Cease high intensity training and competition; ↓ training volume; ↑ rest & sleep; address diet and other stressors.	Cease competition & training except for walking; sleep as needed; strictly address all stressors.
Recovery:	~1-3 weeks.	~1-3 months.	Months to years.

Vitamin D

As a vital prohormone, vitamin D is required for optimal athletic performance, yet insufficiency and deficiency remain highly prevalent among athletes (14). Primarily synthesized in the skin via ultraviolet radiation, endogenous production is frequently limited by clothing, sunscreen, darker skin, adiposity, and indoor training. Inadequate vitamin D can reduce muscle strength, power, and endurance, impair immunity, compromise bone health to increase stress fracture risks, and elevate susceptibility to inflammatory injuries. A standard blood test is necessary to accurately quantify vitamin D status.

Oral health

Systemic and oral health are strongly connected through underlying health and fitness conditions, which often manifest first as dental problems (15). Athletes experience unusually high rates of poor oral health, which can serve as a red flag for systemic issues impacting performance and increasing injury risk (16). A primary driver of poor oral health is the excess intake of refined carbohydrates (beyond ergogenic needs):

- **Direct local effects:** Liquid sugars aggressively trigger acid production, promote oral bacterial fermentation, reduce pH, and dissolve enamel, leading to cavities and gum inflammation.
- **Indirect systemic effects:** Excess refined carbohydrates can depress fat burning, increase fat storage, raise respiratory quotient (RQ), promote chronic inflammation, and reduce salivary pH.

Ergogenic carbohydrate should be carefully timed around training and competition. However, many athletes consume excess amounts throughout the day, displacing nutrient-dense whole foods. After measuring salivary pH for over 20 years in thousands of athletes, I observed that chronically low oral pH closely correlated with increased refined carbohydrate intake and injury rates, and reduced performance (17). Oral pH is easily monitored using standard pH paper available through most major retailers.

Brain injury

In addition to traumatic brain injuries, dysfunction caused by biochemical issues like impaired metabolism can occur. Reduced cellular energy can disrupt the brain's ability to neu-

rologically drive physical movement, causing specific deficits in perceptual-motor tasks like visual-motor coordination (18, 19). This mental fatigue can result from metabolic inflexibility—the body's inability to obtain sufficient energy from glucose, fat, and ketones, and the brain's inability to utilize glucose and ketones. Early signs of inflexibility include excess body fat, insulin resistance, and overconsumption of refined carbohydrates, and can be measured by RQ. When blood glucose falls during training or competition, the body's reduced ability to burn stored fat or ketones causes muscle fatigue, while the brain's inability to use ketones increases mental fatigue. Resulting elevated compensatory stress hormones, such as cortisol, when chronic, can significantly impair performance. Psychologically, mental fatigue degrades performance by increasing perceived exertion and decreasing an individual's motivation and willingness to work harder (20).

Practical applications

Ongoing assessment of an athlete's brain-body signs and symptoms is a highly effective strategy in the prevention, treatment, and monitoring of sports injuries.

Health survey:

- Subjective information on mental and physical energy, moods, discomfort, pain.
- Objective information about sleeping quantity and quality.
- Gut function and digestion: bloating, excess intestinal gas, indigestion, and other abnormal signs and symptoms during rest, training, and competition.
- Diet: Assess regular meals and snacks (whole foods), training/competitive-related products (sports foods), and hydration.
- Drugs: Caffeine, alcohol, over-the-counter and prescription drugs, others.
- Footwear: Training, competitive, casual—fit and comfort. Feet: discomfort, callousing, blisters, blackened toenails.
- Regular comprehensive care from health practitioner(s) including dentist, with blood, urine, and other laboratory evaluations.

Fitness evaluations:

- Regular submax HR evaluations, such as the MAF Test (13).
- Morning/resting HR.
- HR variability.
- Waist-to-height ratio.
- Oral pH.
- Training and competition performance monitoring.

Conflict of interests

The author declares no conflict of interests.

References

1. Sciascia A, Cromwell R. Kinetic chain rehabilitation: a theoretical framework. *Rehabil Res Pract*. 2012;2012:853037. doi: 10.1155/2012/853037.
2. Patel M, Hasoon J, Diez Tafur, et al. The Impact of Chronic Pain on Cognitive Function. *Brain Sci*. 2025 May 24;15(6):559. doi: 10.3390/brainsci15060559.
3. Bensalma F, Hagemeister N, Cagnin A, et al. Biomechanical markers associations with pain, symptoms, and disability compared to radiographic severity in knee osteoarthritis patients: a secondary analysis from a cluster randomized con-

trolled trial. *BMC Musculoskelet Disord*. 2022;23:896. doi: 10.1186/s12891-022-05845-1.

4. Selye H. A syndrome produced by diverse nocuous agents. *J. Neuropsychiatry Clin. Neurosci*. 1998;10:230–231. doi: 10.1176/jnp.10.2.230a.

5. Halcomb E, Davidson P. Using the illness trajectory framework to describe recovery from traumatic injury. *Contemp Nurse*. 2005 Jul-Aug;19(1-2):232–41. doi: 10.5172/conu.19.1-2.232.

6. Sahrman S, Azevedo DC, Dillen IV. Diagnosis and treatment of movement system impairment syndromes. *Braz J Phys Ther*. 2017. Nov-Dec;21(6):391–399. doi: 10.1016/j.bjpt.2017.08.001.

7. Maffetone P. The assessment and treatment of muscular imbalance – The Janda Approach. *J Bodyw Mov Ther*. 2010; 14(3):287288. doi:10.1016/j.jbmt.2009.11.003.

8. Khanmohammadi R, Qanbari S, Hejazi, HS. Central and peripheral factors moderating pain effects on motor control and disability in chronic lumbosacral radicular pain. *J Neuro Engineering Rehabil* 23, 12 (2026). doi.org/10.1186/s12984-025-01824-4.

9. Buchanan TS, Lloyd DG, Manal K, Besier TF. Neuromusculoskeletal modeling: estimation of muscle forces and joint moments and movements from measurements of neural command. *J Appl Biomech*. 2004 Nov;20(4):367–95. doi: 10.1123/jab.20.4.367.

10. Conroy VM, Murray BN, Alexopoulos QT, McCreary J. (2022). *Kendall's muscles: Testing and function with posture and pain* (6th ed.). Wolters Kluwer.

11. Brooks K, Carter J. Overtraining, Exercise, and Adrenal Insufficiency. *J Nov Physiother*. 2013 Feb 16;3(125):11717. doi: 10.4172/2165-7025.1000125.

12. Maffetone PB, Laursen PB. (2015). Athletes: Fit but Unhealthy? *Sports Med Open*;2:24. doi: 10.1186/s40798-016-0048-x.

13. Maffetone P. Enhancing the aerobic system and brain health to optimize athletic performance. *Sport Performance & Science Reports*. 2026. <https://sportperfsci.com/>

14. Ip TS, Fu SC, Ong MT, Yung PS. Vitamin D deficiency in athletes: Laboratory, clinical and field integration. *Asia Pac J Sports Med Arthrosc Rehabil Technol*. 2022 Jul 2;29:22–29. doi: 10.1016/j.asmart.2022.06.001.

15. Kapila YL. Oral health's inextricable connection to systemic health: Special populations bring to bear multimodal relationships and factors connecting periodontal disease to systemic diseases and conditions. *Periodontol 2000*. 2021 Oct;87(1):11–16. doi: 10.1111/prd.12398.

16. Schulze A, Busse M. Sports Diet and Oral Health in Athletes: A Comprehensive Review. *Medicina (Kaunas)*. 2024 Feb 13;60(2):319. doi: 10.3390/medicina60020319.

17. Maffetone P. Functional Testing and Interpretation. In: *Complementary Sports Medicine*. Champaign: Human Kinet-

ics; 1999. p. 69-84.

18. McAllister TW, Stein MB. Effects of psychological and biomechanical trauma on brain and behavior. *Ann N Y Acad Sci.* 2010 Oct;1208:46-57. doi: 10.1111/j.1749-6632.2010.05720.x.

19. Maffetone P, Laursen PB. Refined carbohydrates and the overfat pandemic: implications for brain health and public health policy. 2025. *Front. Public Health* 13:1585680. doi: 10.3389/fpubh.2025.1585680.

20. Schiphof-Godart L, Roelands B, Hettinga FJ. Drive in sports: how mental fatigue affects endurance performance.

Front Psychol. 2018;9:1383. doi: 10.3389/fpsyg.2018.01383.

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