QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: PSIUK, Arkadiusz, SZYMOCHA, Joanna, SMĘDOWSKI, Igor, TSUKERMAN, Jarosław and WOJAS, Aleksandra. Prevalence, Etiopathogenesis, and Clinical Presentation of Sexual Dysfunction in Competitive Athletes Compared with the General Population: A Comprehensive Narrative Review Addressing Sport-Specific Characteristics. Quality in Sport. 2026;63:73574. https://doi.org/10.12775/QS.2026.63.73574

ARTICLE TIMELINE
Received: 21.06.2026. Revised: 10.07.2026. Accepted: 10.07.2026. Published: 15.07.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Prevalence, Etiopathogenesis, and Clinical Presentation of Sexual Dysfunction in Competitive Athletes Compared with the General Population: A Comprehensive Narrative Review Addressing Sport-Specific Characteristics

Arkadiusz Psiuk
District Hospital in Zawiercie, Miodowa 14, 42-400 Zawiercie, Poland
arkadiuszpsiuk@gmail.com
https://orcid.org/0009-0007-1948-1165

Joanna Szymocha
District Hospital in Zawiercie, Miodowa 14, 42-400 Zawiercie, Poland
joanaa.szymocha@gmail..com
https://orcid.org/0009-0005-8310-0406

Igor Smędowski
Independent Researcher
igor.smedowski@gmail.com
https://orcid.org/0009-0003-3910-8035

Jarosław Tsukerman
Municipal Hospital in Żory, ul.Dąbrowskiego 20, 44-240 Żory, Poland
yarko.pppppppppp@gmail.com
https://orcid.org/0009-0006-5061-5574

Aleksandra Wojas
Municipal Hospital in Żory, ul.Dąbrowskiego 20, 44-240 Żory, Poland
ola.wojas@gmail.com
https://orcid.org/0009-0007-4546-0288
Corresponding Author
Igor Smędowski E-mail: igor.smedowski@gmail.com

Abstract

Background: Although integral to overall well-being, sexual health remains a frequently overlooked component of sports medicine. Unlike the general population, the etiology of sexual dysfunction in elite athletes appears to be multifactorial and sport-specific, often involving relative energy deficiency in sport (RED-S), neuroendocrine stress, pelvic microtrauma, doping, and psychogenic burdens.
Aim & Methods: This narrative review synthesizes current evidence and international guidelines to evaluate the etiopathogenesis of sexual dysfunction across different sports and to outline multidisciplinary management strategies.
Results: Evidence indicates that the pathogenesis is markedly heterogeneous. In endurance and aesthetic disciplines, energy deficits and overtraining may suppress the hypothalamic-pituitary-gonadal axis, potentially leading to functional hypothalamic amenorrhea and related dyspareunia in women, and exercise-induced hypogonadotropic hypogonadism in men. Cycling and equestrian sports seem associated with neuromechanical injuries, particularly pudendal neuropathy, which may predispose athletes to erectile dysfunction and anorgasmia. Contact sports may increase the risk of secondary pituitary insufficiency following repetitive mild traumatic brain injury. In strength disciplines, body image disturbances and the sequelae of anabolic-androgenic steroid use appear as leading contributors. Across all sports, psychiatric morbidities and performance anxiety act as significant compounding factors, often exacerbated by systemic underreporting.
Conclusions: Sexual dysfunction in athletes is a multidimensional, underdiagnosed condition that suggests a need for an individualized, biopsychosocial approach. Effective management likely necessitates interdisciplinary collaboration to address underlying pathologies through energy optimization, targeted psychotherapy, and cautiously applied, anti-doping-compliant pharmacotherapy.

Keywords: sexual dysfunction; competitive athletes; sports psychiatry; RED-S; hypogonadotropic hypogonadism; sports medicine; anabolic-androgenic steroids; pudendal neuropathy.

1. Introduction

1.1. Introduction to the Question of Sexual Health in Sports Medicine

Sexual health, as defined in the broad terms adopted by the World Health Organization (WHO), is a fundamental and integral component of overall physical, emotional, mental, and social well-being, and is by no means limited to the absence of disease, dysfunction, or infirmity. Historically and clinically, this particular area within sports medicine remained, for decades, at the margins of research interest, giving way to priorities such as the optimization of aerobic capacity, the refinement of musculoskeletal biomechanics, rapid postoperative rehabilitation, and the prevention of acute musculoskeletal injury [1]. In popular perception, and not infrequently in the intuitive assumptions of medical staff themselves, the competitive athlete is taken to embody peak somatic health and vitality, an image that is readily extrapolated to imply complete and unblemished sexual function [2]. A growing body of evidence-based medicine (EBM), however — including work in sports psychiatry and the neuroendocrinology of exercise — calls this assumption into question, suggesting that heavy training loads, sustained performance pressure, and specific dietary regimens may generate a demanding and non-

physiological environment that can substantially disturb the neuroendocrine and psychological regulation of sexual function [3], [4].

Competitive sport is associated with chronic exposure of the body to multidimensional physical and psychological stressors of an intensity that may considerably exceed the physiological adaptive capacity of the average person. Considered through the contemporary concept of allostatic load, this exposure may lead to a systemic reconfiguration of the body's metabolic priorities, in which biological survival and the maintenance of the vital functions needed to sustain the imposed workload appear to occur at the expense of reproductive processes [5], [6].

From a pathophysiological standpoint, under conditions of low energy availability (LEA) — the etiological basis of relative energy deficiency in sport (RED-S) — an adaptive, central suppression of the hypothalamic-pituitary-gonadal (HPG) axis may develop. This is reflected in a complex cascade of hormonal disturbances in both sexes that can have a direct and pronounced effect on libido, on the vascular mechanisms of genital arousal, and on the capacity to achieve sexual satisfaction and orgasm [7], [8]. It follows that outstanding motor performance and physical capacity are not in themselves a predictor of sexual health, and that in some clinical cases they may instead represent a risk factor predisposing to marked and frequently long-lasting dysfunction [9], [10].

The evolution of contemporary care for elite athletes points toward a holistic biopsychosocial paradigm, in which a patient's mental condition, emotional well-being, and sexual health are considered with the same attention as their biochemical or cardiac parameters. The high demands placed on athletes may provide fertile ground for the development of affective disorders, the spectrum of anxiety disorders, and eating disorders, which can act as independent and important co-factors contributing to sexual dysfunction [11], [12]. The psychopathology of sport suggests that phenomena such as clinical perfectionism, a pathologically disturbed body image (encompassing muscle dysmorphia — colloquially termed bigorexia — in physique sports and anorexia nervosa in aesthetic sports), and chronic, disabling performance anxiety may interact with autonomic and neurohormonal cascades to form a self-reinforcing cycle of psychosexual disturbance [13], [14]. Recognizing and disentangling these neurobiological associations is an important clinical challenge, since emerging symptoms in the sexual domain may be the first — and often carefully concealed — clinical manifestation of more serious psychiatric disorders that warrant targeted therapeutic intervention [3].

Moreover, in contrast to the general population — where a decline in sexual function is associated mainly with aging, obesity, metabolic syndrome, or generalized atherosclerosis — in the elite-sport population this issue appears to be characterized by a notably heterogeneous, multilevel etiology that seems closely shaped by the profile and biomechanics of the discipline practised. Beyond the neuroendocrine (central fatigue) and psychogenic factors discussed above, direct mechanical, traumatic, and iatrogenic factors also play an important part in pathogenesis. Acute microtrauma and chronic compression of the neurovascular structures of the lesser pelvis and perineum (for example, in competitive cyclists and equestrians); repetitive mild traumatic brain injury (mTBI) in contact sports, which may predispose to post-traumatic secondary insufficiency of the anterior pituitary; and disturbances of pelvic floor muscle tone in female and male strength athletes together give rise to clinical entities that are seldom encountered on this scale in primary care [15], [16], [17]. The picture would be incomplete without considering the widespread pharmacologization of sport — both in its licit form, as the overuse of non-steroidal anti-inflammatory drugs (NSAIDs) or analgesic injections that may impair testosterone synthesis, and in its illicit form, in which the use of supraphysiological doses of anabolic-androgenic steroids (AAS) is associated with significant structural pathology of the reproductive system, leading to marked hypogonadism and treatment-resistant erectile dysfunction [18], [19], [20].

Despite a rapidly expanding evidence base and the emergence of international consensus statements, sexual health in the sporting environment remains an area of entrenched stigma and considerable taboo. The marked underreporting of the problem appears to stem directly from athletes' fear of harsh judgment, the potential loss of their place on the team, the disruption of strongly hierarchical coach-athlete relationships, and the internalization of the unhelpful myth of the "invulnerable" athlete [11], [21]. At the same time, this gap is reinforced by medical staff themselves: the absence of standardized screening tools, the avoidance of a sexual-health history for fear of intruding on patient privacy, and gaps in sexual-medicine training among club physicians all tend to perpetuate underrecognition and undertreatment [22]. For these reasons, integrating a careful evaluation of sexual health into the routine care of competitive athletes can be regarded today not only as a pressing medical need but also as an ethical one — bearing not only on a full return to homeostasis and on athletic potential but, above all, on ensuring the patient a dignified and satisfying life both during and after a demanding sporting career [3], [23].

1.2. Definition and Classification of Sexual Dysfunction (According to the DSM-5-TR and ICD-11 Criteria)

In contemporary clinical sexology and psychiatry, the definition of sexual dysfunction has moved away from simplified, linear models of sexual response (such as the classic Masters and Johnson model) toward a multidimensional biopsychosocial paradigm that takes into account the complex interactions among neurobiology and somatic, relational, and psychological factors. From a clinical perspective, sexual dysfunction denotes a heterogeneous group of disorders characterized by a clinically significant, persistent impairment in an individual's capacity for sexual response or for deriving physical and emotional satisfaction from sexual activity. In order to avoid pathologizing transient, adaptive fluctuations in sexual function — which in competitive sport may represent a natural response to an acute tapering phase or to a sudden competition-related stressor — the classification systems impose defined time frames and require the presence of subjective distress [24]. The conceptualization of these disorders has undergone considerable change over the decades, a shift reflected directly in the two leading contemporary nosological systems: the DSM-5-TR (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision), published by the American Psychiatric Association (APA), and the ICD-11 (International Classification of Diseases, 11th Revision), developed under the auspices of the World Health Organization (WHO) [25], [26].

The DSM-5-TR provides a precise framework for sexual dysfunction, with emphasis on the presence of a distress criterion. To diagnose a specific disorder, symptoms must persist continuously for a minimum of six months and cause clinically significant distress or marked interpersonal difficulty. An important diagnostic condition in the DSM-5-TR is the exclusion rule: the dysfunction must not be better explained by another, non-sexual mental disorder (for example, a severe depressive episode) or by the consequences of serious relationship conflict, and it must not be solely the direct result of a substance (medication or drug) or of another systemic medical condition. In the context of sports medicine, this rule poses considerable difficulty for differential diagnosis, because in elite athletes disturbances of erectile mechanisms or anorgasmia frequently coexist, in a reciprocal relationship, with features of relative energy deficiency in sport (RED-S) or with severe anxiety episodes [5], [25].

For male patients, the DSM-5-TR distinguishes four main diagnostic categories. These are: erectile disorder, characterized by marked difficulty in obtaining or maintaining an erection through to the completion of sexual activity, or by a marked decrease in erectile rigidity; male hypoactive sexual desire disorder, defined as a persistent or recurrent absence of (or marked reduction in) sexual thoughts, fantasies, and desire, assessed by the clinician with reference to

the patient's age and sociocultural context; premature (early) ejaculation, usually occurring within approximately one minute of vaginal penetration and before the patient wishes it; and delayed ejaculation, denoting a marked delay in, infrequency of, or complete absence of orgasm [25], [27]. This classification is important for precisely mapping pathomechanisms in sport, where, for example, erectile dysfunction may result from pudendal nerve compression in cyclists, whereas reduced desire is often among the first signs of the neuroendocrine overtraining syndrome (OTS) [23], [25].

A notable change in the DSM taxonomy with respect to female patients was the merging of the previously separate disorders of desire and arousal into a single, integrated entity — female sexual interest/arousal disorder. This change reflects the evidence base and the observation that, in women (including competitive athletes), sexual desire and physiological arousal (lubrication, pelvic vasocongestion) rarely function as isolated, sequential phases but rather tend to reinforce one another in a circular manner. The other entities specific to women are female orgasmic disorder and genito-pelvic pain/penetration disorder (GPPPD). This last category integrates the former diagnoses of vaginismus and dyspareunia, encompassing difficulty with penetration, marked vulvar or pelvic pain, marked fear of pain, and abnormal pelvic floor muscle tension. In women engaged in competitive sport — particularly physique and endurance disciplines, which may predispose to hypoestrogenism and urogenital atrophy in the course of FHA (functional hypothalamic amenorrhea) — GPPPD is a clinically important diagnosis [25], [28], [29].

A category of considerable relevance to sports medicine in the DSM-5-TR is substance/medication-induced sexual dysfunction. This diagnosis requires evidence from the history, physical examination, or laboratory findings confirming that symptoms developed during, or within a month of, substance intoxication, substance withdrawal, or exposure to medications with a known profile of impairing sexual function. In the competitive setting, this category has broad applicability. It is here that the consequences of anabolic-androgenic steroid (AAS) misuse are classified, including pronounced post-cessation hypogonadism (ASIH), which may present with a collapse of libido and an inability to maintain an erection. This group also includes pharmacologically induced iatrogenic effects arising from the use of opioid analgesics permitted in sport, of selective serotonin reuptake inhibitors (SSRIs) that may provoke anorgasmia (including the phenomenon of PSSD — post-SSRI sexual dysfunction), and of antihypertensive agents (beta-blockers) that are frequently misused in precision sports (for example, shooting) [19], [25], [30].

In contrast to the DSM-5-TR, the new ICD-11 classification, introduced progressively from 2022, made a significant nosological redefinition by moving sexual dysfunctions out of the chapter on mental and behavioral disorders and into an entirely new, free-standing block: Chapter 17 — Conditions Related to Sexual Health. This depathologizing step removes the label of "psychiatric disorder" from patients (who often already face substantial stigma in the world of sport) and places greater emphasis on the parity of somatic, vascular, neuroendocrine, and psychological factors. The WHO decision was based on the consensus view that sexual health is a sufficiently distinct and interdisciplinary domain of medicine that confining it to a purely psychiatric framework tends to narrow the diagnostic field and lead to errors in targeted treatment [26], [31].

The structure of dysfunctions in the ICD-11, unlike the integrated female model of the DSM-5-TR, retains a clearer dichotomy between the desire and arousal phases in both sexes. The ICD-11 distinguishes: sexual desire dysfunctions (hypoactive sexual desire dysfunction in both sexes); sexual arousal dysfunctions (male erectile dysfunction and female sexual arousal dysfunction); orgasmic dysfunctions (including anorgasmia, premature ejaculation, and delayed ejaculation); and sexual pain disorders (which treat dyspareunia and vaginismus as separate entities, in contrast to the hybrid GPPPD of the DSM-5-TR). In addition, the ICD-11 generally

requires only a few months' duration of symptoms (typically several months rather than a fixed six), placing considerably greater emphasis on the clinical picture of distress and on whether the symptoms arise from deliberate behavior (for example, voluntary abstinence based on an athlete's own pre-competition assumptions). It also requires specification of whether the dysfunction is generalized or situational and whether it is lifelong (from the onset of sexual activity) or acquired — a distinction that, in competitive athletes, is important for establishing a temporal link with the point at which intensive training regimens began [26], [31].
In summary, fluency in applying the criteria of both systems — the clinical, psychiatric rigor of the DSM-5-TR and the nuanced, somatically oriented perspective of the ICD-11 — provides an important foundation for work with the competitive-athlete population. A careful application of these detailed diagnostic frameworks helps to distinguish isolated psychogenic dysfunction arising, for example, from performance anxiety from organic exercise-induced hypogonadism (EIHH) or from the complex neuroendocrine cascade associated with RED-S. The ability to reach a precise diagnosis on the basis of these systems is the starting point for implementing an interdisciplinary, evidence-based (EBM) treatment strategy whose ultimate goal is not only to improve the patient's sexual function but also to restore balance across their broader biopsychosocial well-being [3], [25], [26].

1.3. Aim of the Study and Research Questions

The overarching aim of this work is to provide a comprehensive, multidimensional, and critical synthesis of the scientific data on the prevalence, complex etiopathogenesis, and varied clinical presentation of sexual dysfunction in the competitive-athlete population. The analysis was designed within an evidence-based medicine (EBM) framework, with the underlying intention of systematically comparing the circumstances found in elite sport with the epidemiology and pathophysiology of these disorders in the general population [24], [27], [29]. A further aim of the present review is to question the widespread tendency to equate outstanding physical capacity with complete psychosexual well-being, and to help close the knowledge gap that results from substantial underreporting and from the systemic stigmatization of sexual-health issues in sports medicine [3], [22].
Among its specific objectives, the review seeks to examine in detail the neuroendocrine, metabolic, and biomechanical pathways particular to competitive sport that may directly or indirectly impair the functioning of the hypothalamic-pituitary-gonadal (HPG) axis. Particular emphasis is placed on examining the contribution of chronic energy deficiency within relative energy deficiency in sport (RED-S), and of chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis in the course of overtraining syndrome (OTS), to the development of exercise-induced hypogonadotropic hypogonadism (EIHH) in men and functional hypothalamic amenorrhea (FHA) in women [6], [10], [32], [33].
Another key objective is to identify and organize the risk factors for sexual dysfunction according to the biomechanical characteristics and environmental conditions of individual disciplines. The review sets out to examine the differing pathophysiological profiles, contrasting microtrauma to neurovascular structures (including pudendal neuropathy) in cyclists [34], [35] with the risk of secondary anterior pituitary insufficiency following repetitive mild traumatic brain injury (mTBI) in contact sports [15], [36], and with pelvic floor muscle pathology in strength sports [16], [37]. The review also aims to provide a thorough evaluation of iatrogenic and toxicological factors, with particular attention to the consequences of anabolic-androgenic steroid (AAS) use and the pronounced post-cessation hypogonadism (ASIH) that may follow their discontinuation, as well as the interactions arising from licit pharmacotherapy (NSAIDs, psychotropic medications, diuretics) [30], [38], [39], [40]. The work concludes with an attempt to outline contemporary, interdisciplinary preventive and

therapeutic frameworks that integrate the efforts of sports medicine physicians, psychiatrists, endocrinologists, and sexologists [3], [41].

In light of these objectives, and on the basis of a preliminary review of the international literature, the following specific research questions were formulated to serve as the analytical thread of this work:

- To what extent does the actual prevalence of the individual categories of sexual dysfunction (as defined by the DSM-5-TR and ICD-11 criteria) in elite male and female athletes differ from the epidemiological rates reported in a healthy, active general population of comparable age? [21], [25], [26], [27]
- In what way does the neuroendocrine cascade triggered in the course of relative energy deficiency in sport (RED-S) and overtraining syndrome (OTS) shape disturbances in the pulsatile secretion of GnRH, leading to clinically overt impairment of reproductive and sexual function (EIHH, FHA, urogenital atrophy)? [7], [23], [42], [43]
- Does repetitive, discipline-specific mechanical trauma — such as perineal compression in equestrian and cycling sports and mTBI in combat sports — give rise to irreversible sexual dysfunction, or does the early use of rehabilitation protocols and training breaks (detraining) allow a fuller restitution of neurological and vascular function? [44], [45], [46]
- What role do specific psychopathological burdens (performance anxiety, clinical perfectionism, eating disorders, and muscle dysmorphia) play in the pathogenesis of erectile dysfunction, anorgasmia, and reduced libido in competitive sport, and to what extent does stigma delay the introduction of targeted psychiatric and psychotherapeutic intervention? [11], [13], [14], [47]
- What are the long-term structural and endocrine consequences of the misuse of doping substances (in particular anabolic-androgenic steroids) for the HPG axis, and with what clinical effectiveness can the features of post-cessation hypogonadism (ASIH) be reversed in former athletes? [18], [20], [48], [49]
- Which treatment standards and principles of multidisciplinary collaboration (including the use of pharmacotherapy consistent with the restrictions of the World Anti-Doping Agency, WADA, and with therapeutic use exemption, TUE, procedures) should be implemented in order to treat sexual dysfunction in line with EBM without thereby ending the patient's sporting career? [3], [8], [50]

These research questions shape the substantive structure of the chapters that follow, directing the narrative toward a careful, critical analysis of a pathophysiology that resists reductionist accounts and that places the athlete's sexual health in a fuller, biopsychosocial, and highly specialized light.

2. Materials and Methods

2.1. Study Design

The design of this work is based on the methodology of a comprehensive, critical narrative review, supplemented with the structured search elements characteristic of systematic reviews. This study design was chosen deliberately, given the considerable multidisciplinarity and clinical heterogeneity of the subject matter, which spans the domains of endocrinology, psychiatry, urology, gynecology, neurology, and sports biomechanics. Unlike a conventional meta-analysis, which requires highly homogeneous quantitative data, a broad narrative review allows a multistranded synthesis of pathophysiological phenomena of differing etiology (for example, mechanical pudendal nerve injury versus neuroendocrine disturbance), making it possible to construct a coherent, holistic model of the pathogenesis of sexual dysfunction in the

competitive setting. The work was structured around the core principles of evidence-based medicine (EBM), with the aim of providing a careful, objective, and scientific account of phenomena that have hitherto been considerably scattered or marginalized in the literature.

2.2. Literature Search Strategy

To assemble a reliable, up-to-date, and comprehensive source base, a multistage literature search strategy was designed and implemented. Searches were carried out in leading international medical and scientific databases, including PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library, as well as in specialized sports medicine databases (SPORTDiscus) and in Google Scholar. Search algorithms based on Boolean operators (AND, OR, NOT) were used, combining controlled-vocabulary terms from the MeSH (Medical Subject Headings) thesaurus with free-text terms. The principal search-phrase combinations included: *("sexual dysfunction" OR "erectile dysfunction" OR "premature ejaculation" OR "dyspareunia" OR "libido" OR "female sexual arousal disorder" OR "anorgasmia") AND ("athletes" OR "elite athletes" OR "professional sports" OR "endurance training" OR "overtraining syndrome" OR "relative energy deficiency in sport" OR "RED-S" OR "anabolic-androgenic steroids")*.
The literature retrieval process was supplemented by manual searching of the reference lists (reference tracking/snowballing) of the included full-text articles, meta-analyses, and official consensus statements of leading medical organizations. In addition, given the need to compare prevalence rates, a parallel search focused on epidemiological data in the healthy general population was conducted, using keywords such as: *("sexual dysfunction prevalence" OR "epidemiology") AND ("general population" OR "males" OR "females")*.

2.3. Inclusion and Exclusion Criteria

To maintain a high methodological standard and to minimize the risk of systematic error (bias), the selection of study material was based on clearly defined inclusion and exclusion criteria (a PECO/PICO framework).
Studies meeting the following inclusion criteria were considered for analysis:

1. Original clinical and observational studies (cohort, case-control, and cross-sectional), systematic reviews, and meta-analyses published in peer-reviewed scientific journals.
1. Official consensus statements and clinical guidelines developed by institutional authorities, including the International Olympic Committee (IOC), the American College of Sports Medicine (ACSM), the Endocrine Society, and the American Psychiatric Association (APA).
2. Publications applying validated diagnostic criteria for sexual dysfunction and mental disorders (according to the DSM-5/DSM-5-TR and ICD-10/ICD-11).
3. Studies whose study population consisted of adult and late-adolescent competitive (elite/professional) athletes, as well as highly trained amateurs whose training loads approximate a professional regimen.
4. High-quality epidemiological studies assessing the prevalence of sexual dysfunction in the general population (serving as control and reference groups).
5. Works published in English and Polish, without strict date limits, though with priority given to literature from the past 20 years (in order to reflect the most recent training and pharmacological paradigms).

Materials meeting the following exclusion criteria were not included in the analysis:

1. Non-peer-reviewed publications, reports from the popular press, and expert opinions not supported by EBM evidence (lower-tier grey literature).

1. Case reports describing single cases, unless they documented very rare, discipline-specific traumatic pathomechanisms with a confirmed mechanical basis.
2. Studies focusing exclusively on paraphilias or gender dysphoria, which in psychiatric classifications (such as the ICD-11) constitute separate nosological entities and fall outside the strict criterion of "sexual dysfunction" in its functional sense.
3. Works of low methodological reliability, with serious gaps in the description of the study group or using sexual-health assessment questionnaires that have not been clinically validated.

2.4. Data Synthesis

The synthesis of the collected data took the form of a multistage qualitative analysis aimed at identifying recurrent pathophysiological patterns and extracting specific risk factors for particular subgroups of athletes. Given the considerable variability of the endpoints examined (ranging from objective hormonal parameters to subjective scales of sexual satisfaction), quantitative statistical analysis (meta-regression) was not undertaken, and structured etiological profiling was adopted instead.
The identified clinical entities and mechanisms were grouped into four main analytical domains:

The neuroendocrine and metabolic domain: encompassing HPG axis disturbances induced by RED-S, overtraining syndrome (OTS), functional hypothalamic amenorrhea (FHA), and exercise-induced hypogonadism (EIHH).

The psychogenic and psychiatric-burden domain: synthesizing data on performance anxiety, the co-occurrence of affective and eating disorders, and their influence on the sexual response cycle.

The mechanical, vascular, and traumatic domain: focusing on the biomechanical differences between disciplines (for example, perineal injury and ischemia in cycling, concussion and secondary pituitary damage in combat sports, and pelvic floor loading in weightlifting).

The iatrogenic and toxicological domain: evaluating the influence of licit pharmacotherapy (analgesics, SSRIs) and illicit psychoactive substances (the active-use and withdrawal phases of AAS) on the sexual and reproductive function.

The final stage of data synthesis was a careful comparative analysis. Validated prevalence rates for sexual dysfunction in competitive sport were set against demographic data for the general population, with results adjusted for confounding variables such as patient age (excluding the natural aging process as a principal etiological factor in young athletes). The most recent reports from the Polish literature were also incorporated, which made it possible to situate the findings within a broad, international, and nuanced clinical context. The conclusions from the individual domains served as the basis for formulating the comprehensive diagnostic and therapeutic protocols discussed in detail in the closing chapters of the work.

3. Epidemiology of Sexual Dysfunction: Athletes Versus the General Population

3.1. Occurrence of Sexual Dysfunction in the General Population: A Statistical Overview (by Sex and Age)

An accurate interpretation of the pathophysiological deviations observed in a selected cohort of elite athletes requires a clear reference to the epidemiological background provided by the general population. Globally, the epidemiology of sexual dysfunction (SD) is an analytical challenge, shaped by the considerable heterogeneity of the research instruments used (ranging from standardized clinical interviews based on DSM criteria to self-report questionnaires of

varying psychometric validation), by sociocultural barriers, and by differing levels of stigma across regions of the world [27], [29]. Over the past two decades, however — beginning with the landmark, large-scale National Health and Social Life Survey (NHSLS) conducted in the United States — evidence-based medicine (EBM) has accumulated a substantial body of data that allows a fairly precise mapping of the prevalence of these disorders by sex, age, and coexisting somatic burden [24]. These studies consistently indicate that sexual dysfunction is a common public health problem and that, in populations outside competitive sport, its etiology is most strongly associated with physiological aging and the accumulation of chronic lifestyle-related diseases [24], [27].

In the male population, the most frequently studied and most thoroughly characterized nosological entity is erectile dysfunction (ED). Global systematic reviews estimate the mean prevalence of ED at between 13% and 21% across the total adult male population, although this rate shows a strong linear — and arguably exponential — relationship with age [27]. In young adults (20–39 years), the occurrence of ED (defined as a persistent difficulty rather than occasional episodes) is estimated to be relatively low, on the order of 5–10%, and here the pathomechanism appears to be largely dominated by psychogenic factors (for example, performance anxiety) [14], [27]. A marked inflection point in the morbidity curve is observed in the fifth and sixth decades of life, reaching 40–50% in the 50–59 age group and exceeding 70% in men over 70 [27]. This clear age gradient appears to reflect the vascular pathophysiology of ED directly; in older age groups, erectile dysfunction often serves as a warning sign (the "tip of the iceberg" phenomenon), representing the first clinical manifestation of latent vascular endothelial dysfunction, generalized atherosclerosis, insulin resistance, or undiagnosed type 2 diabetes [2], [27].

Another epidemiologically important problem in men is premature ejaculation (PE). Interestingly, unlike ED, the prevalence of PE is relatively constant across decades of life, fluctuating around 20–30% of the global male population, which suggests a greater contribution of innate neurobiological mechanisms of ejaculatory reflex control and of psychological factors than of typically vascular or metabolic factors [24]. Male hypoactive sexual desire (reduced libido) shows a prevalence on the order of 15–25%, with a clear upward trend correlated with age-related aging of the endocrine system (late-onset hypogonadism, LOH) and the natural decline in testosterone synthesis [24].

The epidemiology of female sexual dysfunction (FSD) shows a different dynamic and a higher overall prevalence, estimated in population studies at 40–45% [24], [29]. Unlike in men, where the dominant problem tends to be the arousal phase (ED), the most common diagnosis in women is hypoactive sexual desire disorder (HSDD) or the integrated interest/arousal disorder of the DSM-5-TR. Reduced libido is reported by 27–33% of women and shows its strongest relationship with relational context, partner dynamics, chronic life stress (for example, related to caregiving roles), and — importantly — menopausal and postpartum status [24], [29].

Orgasmic-phase disorders (anorgasmia or markedly delayed orgasm) affect 21% to 25% of the general female population. In younger (premenopausal) women, difficulty reaching orgasm is most often linked to psychogenic and educational factors and to inadequate stimulation, but it is also a fairly common iatrogenic side effect of widely used SSRI and SNRI antidepressants [24], [30]. Genito-pelvic pain/penetration disorder (GPPPD, which includes the former category of dyspareunia) shows a clear bimodal age distribution, affecting approximately 10–20% of the female population [24], [29]. In young adult women, the pain often has a basis in pelvic floor hypertonicity (vaginismus), recurrent urogenital infections, or endometriosis [29]. A second, substantial peak in incidence is associated with the perimenopausal and postmenopausal period (genitourinary syndrome of menopause, GSM), in which the physiological loss of estrogen stimulation leads to advanced atrophy of the vaginal epithelium, reduced wall elasticity, and a marked reduction in physiological lubrication [24], [29]. It is this

atrophic mechanism — associated in the general population with aging — that may be induced non-physiologically in young elite female athletes through the chronic, severe estrogen deficiency of functional hypothalamic amenorrhea (FHA) resulting from RED-S [10], [28], [43].
In summary, the statistical picture in the general population points clearly to the aging of the cardiovascular system, the epithelium, and the hormonal cascade, together with the accumulation of coexisting somatic disease, as the principal independent predictors of sexual dysfunction in both sexes [24], [27], [29]. Familiarity with these patterns is a necessary condition for understanding the distinctive nature of the sporting environment, in which a paradoxical situation may be encountered: advanced sexual pathology typical of geriatric age (such as pronounced hypogonadism or vaginal atrophy) occurring in individuals in the second and third decades of life, who are biologically primed for maximal reproductive capacity [9], [10]. This pronounced contrast highlights the different etiology of the problem in athletes — instead of cellular degeneration driven by age and atherosclerosis, there appears to be an adaptive or trauma-related neuroendocrine deterioration arising from a demanding training regimen and competitive pressure [5], [15].

3.2. Prevalence of Disorders in Athletes at Different Career Stages (Amateurs, Professionals, and the Post-Career Period)

An epidemiological evaluation of sexual dysfunction in the sporting environment cannot rely on a single, static figure. The allostatic load that shapes the pathophysiology of these disorders changes dynamically over time, producing a highly variable risk profile depending on the stage of the patient's involvement in sport [21], [23]. An EBM approach calls for careful stratification of the study population into three distinct categories: recreational athletes and advanced amateurs; active competitive athletes (the professional elite); and former athletes in the period after the end of a professional career (detraining and adaptation to retirement). Each of these groups is characterized by a different prevalence, shaped by a stage-specific interaction of neuroendocrine, psychological, and iatrogenic factors [9], [21], [39].
In the group of advanced amateurs and recreational athletes, the prevalence of sexual dysfunction is highly inconsistent and depends on how the threshold of "advanced" practice is defined. On the one hand, numerous population-based urological and cardiological studies indicate that moderate, regular physical activity (in line with WHO guidelines on aerobic exercise) acts as a strong protective factor [2]. In cohorts with moderate physical activity, rates of ED and anorgasmia are statistically significantly lower than in a sedentary population, which is attributed to improved endothelial function, better insulin sensitivity, reduced cortisol levels, and a favorable lipid profile [2], [27]. Among highly advanced amateurs, however (for example, those preparing for Ironman triathlons or mountain marathons), who replicate the training volumes of professionals but do so without the oversight of integrated medical care teams, dietitians, and sport psychologists, the rates of these disorders appear to rise considerably [12], [42]. Such amateurs tend to be highly susceptible to unintentional energy deficiency (LEA) leading to RED-S, and are also at considerable risk of overreaching and overtraining syndrome (OTS), since they often attempt to combine a demanding training plan with the requirements of a conventional professional career and family life (with inadequate recovery) [5], [23]. As a result, in this subgroup the prevalence of reduced libido (HSDD) can be comparable, and in men — within the phenomenon termed exercise-induced hypogonadotropic hypogonadism (EIHH) — a decline in total testosterone not infrequently occurs sooner than in professionals who have access to advanced recovery resources [9], [42], [51]. In addition, ED of urological origin becomes a notable problem in advanced amateurs (particularly in cycling, where a frequency of reported numbness and transient erectile problems after long rides in the range of

10–20% has been noted), resulting from the use of unsuitable equipment (a poorly fitted saddle) and from errors in riding biomechanics without the assistance of a bike fitter [45], [52].
The situation changes substantially in active competitive athletes. Because of pronounced underreporting and a wall of silence within the sporting elite (fear of stigma, loss of an endorsement contract, or relegation to the reserve squad), official figures often represent only the tip of the iceberg [3], [22]. Objective endocrinological studies, however, give some indication of the scale of the problem: in elite female athletes in aesthetic and endurance sports, the prevalence of functional hypothalamic amenorrhea (FHA) — a clinical manifestation of RED-S — is estimated, depending on the discipline, at between 30% and as much as 60% [10], [28], [43]. Through chronic hypoestrogenism, FHA is associated with a near-universal occurrence of vaginal epithelial atrophy and reduced secretion, which is a major contributor to dyspareunia, arousal dysfunction, and anorgasmia in this population [10], [29]. In elite men, the prevalence of EIHH is lower, but it still appears to affect 15% to 25% of athletes during periods of the highest-volume aerobic preparation (the so-called macrocycle peak), presenting primarily as reduced libido, further compounded by the cortisol cascade (OTS) [9], [23], [50]. The substantial contribution of psychiatric factors in this group should be emphasized. The prevalence of comorbid mood and eating disorders (for example, restrictive or compensatory dietary practices to "make weight" in combat sports or ski jumping) is significantly higher than in the general population, affecting roughly 10–25% and as many as 15–30% of professional athletes, respectively (with a predominance of women, though the prevalence of muscle dysmorphia rises considerably in men) [11], [13], [47], [53]. Acting as a close co-factor of HSDD and ED, these psychiatric disorders contribute substantially to the burden of sexual dysfunction during the phase of full professional engagement [3], [13]. In subgroups using illicit pharmacological doping, the active phase of an AAS (anabolic-androgenic steroid) cycle may be associated with hypersexuality and even priapism, but also with coexisting erectile disturbances of varying severity (for example, the "Deca dick" phenomenon during nandrolone use, related to a pronounced rise in prolactin) and with complete loss of fertility due to suppression of the hypothalamic-pituitary-gonadal (HPG) axis [18], [20], [38].
The final, highly heterogeneous area of research is the prevalence of dysfunction in athletes who are "retired" or ending a competitive career. In this cohort, EBM documents the most serious structural damage. Whereas in athletes with OTS or RED-S the cessation of training (detraining) and the optimization of caloric intake often lead (though not always without treatment) to a reversal of HPG axis suppression and to the restoration of menstruation or testosterone levels [8], [23], [54], mechanical injuries and post-doping damage tend to be irreversible [34], [38]. In particular, post-cessation hypogonadism (anabolic steroid-induced hypogonadism, ASIH) following long-term discontinuation of AAS appears to lead to a chronic, years-long, treatment-resistant decline in libido (absent or infrequent sexual fantasies) and ED (up to and including pronounced impotence) in approximately 30–50% of former strength athletes, who may require long-term medical testosterone replacement therapy (TRT) to function at a basic level [39], [48]. Another affected group is former combat-sport athletes (boxing, MMA) with accumulated, unrecognized head injuries (mTBI). Studies have reported, in 20–30% of "veterans" of contact sports, features of post-traumatic pituitary insufficiency (including deficiency of growth hormone, GH, and of LH/FSH), leading to secondary hypogonadism and persistent disturbances of desire and erection many years after the end of activity in the cage or the ring [15], [36], [55]. A third clinically documented phenomenon, in "retired" female strength-sport athletes (for example, weightlifters) and gymnasts, is a high predisposition to urinary incontinence and pelvic floor disorders in the early post-career period, correlating with disturbances in the experience of arousal and satisfaction — although the prevalence of dyspareunia is higher during the career itself (often, once estrogen levels return to normal, the lubrication problem resolves and only chronic perineal muscle dysfunction

remains) [16], [56]. Studies of former Masters veteran athletes (of advanced age), in turn, have shown that athletes who did not sustain lasting post-traumatic or post-AAS pathology reported significantly higher sexual satisfaction and fewer concerns about dysfunction than peers from the general population in the same age cohort, maintaining this well-being thanks to better cardiovascular parameters in later life [21].

3.3. Diagnostic Difficulties and Underreporting of Sexual-Health Problems in the Sporting Environment (the Stigma Factor)

The true prevalence of sexual dysfunction in competitive sport remains largely beyond the reach of demographic statistics, owing to a deeply rooted and systemic pattern of underreporting. This is not merely a methodological flaw in survey research but a structured, substantial clinical problem with a multidimensional psychosocial and institutional basis [3], [22]. In the public imagination, the construct of the "elite athlete" embodies a cult of absolute performance, flawless biomechanics, and inexhaustible vitality. In an environment in which even the most subtle somatic or psychological weakness may be interpreted by coaching staff or sponsors as a loss of competitive potential, admitting to erectile dysfunction, anorgasmia, or loss of libido becomes, for the patient, an act carrying considerable reputational and professional risk [4], [11]. This internalization of a cult of strength and resilience creates a barrier of stigma that effectively closes off communication between athlete and physician, leading to substantial delays in diagnosis and in the introduction of important therapeutic approaches that may protect against irreversible damage [3].

A primary mechanism driving underreporting is the structure of the medical care system itself within sports clubs and federations. The therapeutic relationship, which in standard sexological care rests on strict confidentiality, empathy, and trust, is often distorted in a particular way in sports medicine (the phenomenon of dual agency). A team or sports physician may be perceived by the athlete (often subconsciously) not as an independent clinician but as an integral part of the training apparatus, whose primary aim is to maximize performance and maintain the patient's competitive availability, at times even at the expense of long-term well-being [1], [3]. In this context, an athlete — fearing potential exposure to the head coach, or fearing that the problem may be regarded as grounds for exclusion from the starting line-up (for example, because of a suspicion of overtraining or of the use of prohibited substances) — may deliberately conceal any symptoms related to psychosexual dysfunction [4], [11]. Fear of exposure may be particularly strong in cases where the basis of, for example, pronounced post-cessation hypogonadism (ASIH) is concealed, illicit use of anabolic-androgenic steroids (AAS), which, if medically documented, could give rise to disciplinary consequences from the World Anti-Doping Agency (WADA) [20], [48], [49].

A self-reinforcing cycle of ignorance and the normalization of pathology persists in competitive sport. A classic example is the substantial downplaying of the symptoms of functional hypothalamic amenorrhea (FHA) in women, which is commonly regarded by coaches — and, troublingly, by part of the medical community — as a natural, "physiological" indicator of good form and an appropriately low level of body fat [10], [54]. In fact, the absence of menstruation, a clear warning sign of HPG axis shutdown (within RED-S), is associated with pronounced hypoestrogenism, the likely consequence of which is urogenital atrophy, troublesome dyspareunia, and a marked decline in physiological lubrication [28], [29]. If the coaching staff themselves affirm the cessation of the menstrual cycle as an element of training success, the patient is left without any educational prompt to seek help for the marked genito-pelvic pain disorder (GPPPD) associated with FHA, which becomes a hidden, silent cost of her sporting career [10], [25]. An analogous rationalization of pathology occurs in cyclists, for whom perineal numbness and transient erectile disturbances are taboo and tend to be regarded by

athletes as a common, acceptable "side effect" of heavy training in the saddle rather than as a clinically significant early sign of the slow degeneration of pudendal nerve structures [34], [52]. Considerable diagnostic difficulty also arises from the lack of routine implementation of a specific, targeted sexual-health history (screening) during mandatory pre-season medical examinations (the Pre-Participation Evaluation, PPE) and during periodic monitoring of blood parameters and ECG. The conventional examination by a sports medicine physician focuses almost exclusively on the cardiovascular system, the musculoskeletal system, and metabolic panels. The questionnaires included in mental-health assessment (for example, those used to assess suicide risk or severe depression) also rarely incorporate precise questions directed at desire or erectile function, even though, as EBM evidence suggests, sexual disturbances are not infrequently among the strongest predictors and co-factors of the manifestation of overtraining syndrome (OTS) and of severe episodes of affective disorder [3], [11], [23]. Moreover, diagnostic practice lacks specialized, psychometrically validated rating scales (questionnaires) calibrated specifically to the athletic population; conventional urological instruments (such as the IIEF-5 for men and the FSFI for women), although validated for the general population [27], [29], rarely make it possible to isolate the influence of unusual confounding variables (for example, the specific point pressure of compression clothing) and do not capture subtle declines in libido at an early stage of neuroendocrine decline [21], [22].

Evidence-based medicine suggests that overcoming the barrier of underreporting requires a clear change in the organizational and educational paradigm. It would be advisable to implement a model based on a firm separation of the club physician's role from the staff who decide on sporting contracts, and to consistently integrate screening questions on reproductive function, perineal pain, menstrual patterns in women, and the presence of desire disorders into periodic evaluation protocols [3], [23]. Only by removing the element of taboo (that is, destigmatizing) from a routine question about "reproductive and sexual health" — supported by educating coaches about the harmful influence of OTS and RED-S [5], [23] — is it likely to become possible to introduce individualized strategies early (such as reducing training volume, adjusting equipment, or optimizing the diet) before a transient weakening of sexual drive develops into a more serious, damaging pathophysiological process requiring complex, advanced multispecialty treatment [3], [8].

4. Neuroendocrine and Metabolic Pathomechanisms in Competitive Sport

4.1. Relative Energy Deficiency in Sport (RED-S) and Sexual Dysfunction

The conceptualization of neuroendocrine disturbances in athletes has changed considerably in recent years, moving away from the historical, female-only model of the Female Athlete Triad toward the broader, integrated, and sex-inclusive paradigm of relative energy deficiency in sport (RED-S), formalized and progressively updated in consensus statements of the International Olympic Committee (IOC) [5], [7], [32]. The Triad, classically centered on the co-occurrence of disordered eating, amenorrhea, and osteoporosis [54], [57], proved too narrow a concept (it did not encompass men, although an analogous Male Athlete Triad model is under discussion among experts) [6], [8] to describe fully the wide-ranging systemic consequences of a negative caloric balance in competitive athletes of both sexes. Within an EBM framework, the basis of the etiopathogenesis of RED-S is not an absolute reduction in caloric intake (which may occur, for example, in anorexia nervosa) but rather a state of critically low energy availability (LEA). LEA is defined mathematically as dietary energy intake minus exercise energy expenditure (EEE), standardized to the athlete's fat-free mass (FFM) and expressed in kcal/kg FFM/day. When this availability falls below the physiological threshold needed to support basal metabolism, the body initiates a cascade of marked, adaptive metabolic restrictions [5], [7],

[32]. The evolutionary priority becomes the maintenance of the life-sustaining processes of locomotion, respiration, and thermoregulation, which appears to occur at the direct expense of "secondary" biochemical pathways, particularly the energetically costly activity of the reproductive system and the hypothalamic-pituitary-gonadal (HPG) axis [5], [10]. A consequence of this mechanism is a pronounced reduction in sexual potential, which may predispose both sexes to substantial disturbances of desire and of the physiological readiness of the pelvic organs [5], [7], [32].

4.1.1. The Effect of Caloric Deficit on the Hypothalamic-Pituitary-Gonadal (HPG) Axis in Both Sexes

The mechanism by which low energy availability (LEA) suppresses the functioning of the HPG axis, despite the preserved anatomical continuity of the hypothalamus and pituitary gland (which is why this state is described as secondary and functional), is one of the clearer examples of precise — though, in the context of sexual health, disruptive — neuroendocrine signaling. This pathway does not rely on a direct cutting-off of substrate supply (for example, cholesterol) to steroidogenic tissues, but rather on the integration of information from peripheral energy sensors, which include adipocytes (notably leptin), gut neuropeptides (for example, ghrelin and peptide YY), and intracellular regulators of ATP balance such as AMP-activated protein kinase (AMPK) [7], [10], [43].

Under the physiological conditions of an adequate energy balance, adipose tissue cells (adipocytes) synthesize a sufficient quantity of leptin, which crosses the blood-brain barrier and binds to specific receptors in the arcuate nucleus of the hypothalamus. This signal, read as indicating "adequate fat reserves," stimulates highly specialized neurons that secrete the neuropeptide kisspeptin (produced, among other sources, by KNDy neuron clusters). Kisspeptin is, in turn, a potent direct secretagogue activating the neurons that produce gonadotropin-releasing hormone (GnRH), enabling its physiological, pulsatile secretion into the pituitary portal circulation and, consequently, the stimulation of the anterior lobe to produce the gonadotropins (luteinizing hormone, LH, and follicle-stimulating hormone, FSH) [10], [43].

In the case of an acute or — far more often in sport — chronic caloric deficit (the RED-S state), the reduction in adipose tissue stores markedly lowers the concentration of circulating leptin. At the same time, an empty stomach secretes increased amounts of ghrelin (the "hunger hormone"), while strained muscle cells activate inhibitory pathways. The reduced leptin signal and the excess of catabolic signals together appear to produce a marked downregulation of kisspeptin expression in the hypothalamus [10], [43]. A "silencing" of GnRH neurons follows, which is considered the core of the pathomechanism. This phenomenon occurs irrespective of the athlete's sex, although its parameters differ (the LEA threshold leading to suppression in women is thought to be somewhat higher, at approximately 30 kcal/kg FFM/day, whereas in men these values may be as low as 15–20 kcal/kg FFM/day — though data for men are more difficult to harmonize across studies because of differences in the physiology of glycogen reserves and adipose tissue tolerance) [6], [7], [32].

In female patients, the reduction in GnRH pulsatility results in an absence of LH and FSH release, which directly affects the granulosa cells and the theca of the ovarian follicles. The arrest of Graafian follicle development leads to anovulation and to the cessation of endogenous estradiol and progesterone synthesis [10], [28], [43]. For sexual health, the consequence of this mechanism (termed functional hypothalamic amenorrhea, FHA) is a pronounced, LEA-induced hypoestrogenism. Low estradiol levels directly cause atrophy of the thin, unstimulated vaginal epithelium (a reduction in the superficial cell layer, loss of intraepithelial glycogen, and changes in the vaginal microflora), leading to increased dryness and an inability to produce sufficient transudate (lubrication) during the arousal phase [10], [25], [28]. This pathophysiological

sequence may become a primary source of pronounced dyspareunia that intensifies with each attempt at penetration (acute pain and urogenital trauma in elite female athletes), resulting not only in the familiar reduction in interest in intercourse through aversive conditioning and secondary psychological distress but, above all, in a diagnosis of GPPPD by the DSM-5-TR criteria [25], [29].

In men — although they have classically been less often assessed for energy-related indicators [6], [8] — the deficit and the collapse of GnRH pulsatility translate into a weakened LH signal reaching the Leydig cells of the testes. This may result in a marked decline (not infrequently approaching the threshold used to define clinical hypogonadism in geriatric patients) in the synthesis of total and free testosterone (T) in the peripheral circulation, leading to so-called exercise-induced hypogonadotropic hypogonadism (EIHH) [9], [42], [51]. A reduction in T under RED-S conditions may have substantial behavioral and physiological consequences for male sexuality: the androgen-dependent intraneuronal expression of nitric oxide synthase in the corpora cavernosa (nNOS) declines, and this expression is what supports the production of the potent vasodilator nitric oxide (NO), which is necessary to generate and maintain high intracavernosal pressure during erection. The lack of NO means that the patient may be unable to achieve a full, rigid erection (erectile dysfunction, ED) despite an adequate blood supply through arteries that are still intact [9], [27], [50]. At the level of the limbic system, low T and reduced synthesis of neuroactive androgen metabolites lead to a marked decline in sexual motivation and interest (a diagnosis of male hypoactive sexual desire disorder under the DSM-5-TR) [24], [25]. In addition, the release of FSH, which supports the proper functioning of Sertoli cells, is suppressed, which may adversely affect spermatogenesis parameters and reduce the athlete's reproductive prospects, while also deepening psychological stigmatization and a subjective sense of a defect in the intimate sphere [3], [9], [50].

4.1.2. Disturbances in the Pulsatile Secretion of GnRH and Their Clinical Consequences

A central link in the neuroendocrine cascade that induces serious reproductive disturbances in the course of relative energy deficiency in sport (RED-S) is the marked dysregulation of the pattern of hypothalamic gonadotropin-releasing hormone (GnRH) secretion [7], [10]. As outlined above, a state of negative energy balance (LEA) silences the kisspeptin signal, which in turn deprives GnRH neurons of the stimulation needed for normal function [5], [43]. From a pathophysiological standpoint, it should be emphasized that maintaining HPG axis function in both sexes depends not only on the total daily amount of GnRH released but, above all, on the precisely defined, high-frequency, pulsatile dynamics of that release [10], [28]. The GnRH receptor located on the surface of the gonadotropic cells of the anterior pituitary has the distinctive property of rapid downregulation under continuous (non-pulsatile) stimulation and, conversely, becomes "silenced" in the absence of an adequate frequency of hypothalamic pulses [43]. Through reduced leptin synthesis and increased inhibitory factors (such as ghrelin and NPY), RED-S in athletes leads to a pathological slowing — or even a complete cessation — of GnRH pulses (a phenomenon sometimes described as a "return to the prepubertal pattern") [10], [43].

In women contending with a markedly severe energy deficit secondary to grueling endurance training or restrictive diets in aesthetic sports, the reduced frequency of GnRH pulses prevents the proper release of the luteinizing hormone (LH) required for ovulation. The consequence is an arrest of ovarian follicle development at early stages and a halt in estradiol production by the granulosa cells, which clinically defines functional hypothalamic amenorrhea (FHA) [10], [28]. Chronic hypoestrogenism may become a source of marked, tangible sexual dysfunction (classified in the DSM-5-TR as genito-pelvic pain/penetration disorder, GPPPD) [25], [29]. Thinning and atrophy of the vaginal and vulvar epithelium develop, and this, combined with the absence of vasodilation (physiologically induced by estrogens during the arousal phase), results in a substantial reduction in the amount of vaginal transudate (lubrication). Attempts at

sexual activity under conditions of such dryness and tissue fragility may cause intense dyspareunia, not infrequently with microtrauma and bleeding, which can fairly quickly initiate anticipatory fear of pain in female athletes, a self-reinforcing cycle of pelvic floor hypertonicity, and a collapse of libido [28], [29]. The loss of menstruation and desire is not infrequently — consistent with underreporting — masked for years or treated by the athlete and staff as a transient, "harmless" adaptation [3], which also exposes her to irreversible loss of bone mineral density [54], [57].

In the subpopulation of elite men (including long-distance runners, road cyclists, and triathletes), this phenomenon may manifest in an equally damaging way, presenting as exercise-induced hypogonadotropic hypogonadism (EIHH) [9], [51]. The breakdown of the pulsatile rhythm of hypothalamic GnRH secretion (documented, among other findings, in classic studies assessing the LH release pattern after exogenous naloxone administration in marathon runners) [58] leads to a quiescence of pituitary LH and FSH synthesis. The LH deficiency affects the testicular Leydig cells, resulting in a substantial decline — or at least a clear, persistent reduction — in free and total testosterone concentrations [9], [42]. The clinical consequences of low T in this age group (men at the peak of motor performance) affect the basic physiological determinants of erection. Androgen insufficiency leads to endothelial dysfunction of the corpora cavernosa: NO production falls owing to inhibition of nNOS synthesis, accompanied by structural atrophy of the smooth muscle that hinders proper vasodilation and compression of the venous system during erection. For this reason, the athlete may develop erectile dysfunction (ED), which is not infrequently accompanied by a generalized decline in desire (hypoactive sexual desire disorder) and a loss of sexual initiative, generating considerable relational and psychological stress [25], [50]. It is worth emphasizing that EIHH — although reversible through training modification and increased caloric intake (resolution of RED-S) — is for a long time often unrecognized by sports staff and urologists, or misinterpreted, for example, as an early manifestation of organic LOH (late-onset hypogonadism) or of "overwork," rather than being clearly linked to the allostatic load and energy deficit arising from the sporting regimen [23], [50]. Owing to the restrictive anti-doping regulations (WADA), the immediate use of symptomatic testosterone replacement therapy (TRT) in professional men is almost entirely precluded and requires a highly complex case to obtain a therapeutic use exemption (TUE), which obliges clinicians to seek primary, metabolic ways of restoring GnRH pulsatility [8], [50].

4.2. Overtraining Syndrome (OTS)

Whereas relative energy deficiency in sport (RED-S) centers on the pathology arising from a substantial caloric deficit relative to energy expenditure [5], [32], overtraining syndrome (OTS) represents a distinct — though not infrequently coexisting — pathway of allostatic overload in the athlete. According to the joint consensus of the European College of Sport Science (ECSS) and the American College of Sports Medicine (ACSM), OTS is defined as a prolonged, unexplained decline in athletic performance (lasting from several weeks to many months) underpinned by an imbalance between training stimuli and the time and quality of the patient's recovery and psychological regeneration in the broad sense [23]. The pathophysiology of OTS is not a purely mechanical phenomenon (local muscle damage) but rather a broad, systemic dysregulation of neuroendocrine, immune, and autonomic control (with sympathetic predominance in the early phase and parasympathetic predominance in the terminal phases of decline) [23], [33]. From a clinical and sexological standpoint, OTS represents a state of sustained stress load, in which the body interprets chronic high-volume, high-intensity training — further burdened by psychological pressure and sleep deficits — as a signal of a threat to survival. This response triggers a substantial, poorly adaptive hormonal cascade that bears

directly on reproductive and sexual processes [23], [33]. A central player in this neurohormonal disturbance is the chronically hyperactive stress axis.

4.2.1. Chronic Activation of the Hypothalamic-Pituitary-Adrenal (HPA) Axis and the Effect of Chronically Elevated Cortisol on Testosterone and Estradiol Synthesis

In a well-designed training cycle, the acute release of glucocorticoids (particularly cortisol) and catecholamines in response to exercise is an entirely physiological phenomenon that supports the mobilization of energy reserves (gluconeogenesis, lipolysis) needed to perform muscular work, with a rapid return to homeostasis after the training session ends [23]. In the course of overtraining syndrome, however, the negative-feedback mechanism becomes dysfunctional, leading to a state of chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis. Excessive stimulation of the hypothalamus to produce corticotropin-releasing hormone (CRH), which promotes the release of adrenocorticotropic hormone (ACTH) from the pituitary, drives the zona fasciculata of the adrenal cortex to a continuous and excessive synthesis of cortisol. Only in a phase of complete, extreme burnout does adrenal exhaustion and a paradoxical adrenal insufficiency occur; in most elite athletes reporting early signs of a decline in performance, however, an abnormal, chronically elevated blood level of this hormone is observed [23], [33].

This sustained hypercortisolism, together with elevated CRH concentrations, has a marked, multilevel, neurotoxic effect on the HPG axis, which acts in opposition to it. CRH acting directly on the hypothalamic nuclei exerts a direct and strongly inhibitory effect on kisspeptin-producing neurons and on the GnRH-releasing neurons themselves [10], [23]. In addition, an excess of cortisol in the periphery affects the gonads directly. In men, it has been documented that chronically high glucocorticoid levels bind to specific receptors (GR) located in the testes, which may induce apoptosis of Leydig cells and markedly downregulate the expression of key enzymes of the steroidogenic pathway (such as P450scc and 3β-HSD) [9], [33]. The result of this two-pronged inhibition — reduced pituitary stimulation and suppressed production within the gonads themselves — is a persistent hypogonadotropic hypogonadism. A decline in testosterone to subclinical or overtly pathological values (EIHH) deepens the impairment of nNOS synthesis described above and intensifies the features of erectile dysfunction (ED), while also affecting androgen-dependent sexual desire [9], [25], [50]. In studies of competitive athletes, the ratio of free (or total) testosterone to cortisol (the fT/C or T/C ratio) is regarded as one of the objective markers of OTS — its decline (a marked predominance of a catabolic and immunosuppressive environment over anabolism) correlates linearly with the severe loss of libido reported by patients [23], [33].

An analogous situation occurs in female athletes. In the hypercortisolemia induced by severe OTS, the elevated glucocorticoid level suppresses ovarian sensitivity to gonadotropin stimulation. Cortisol inhibits aromatase activity in the granulosa cells, hindering the conversion of androgens to estrogens, and also disrupts the release of progesterone needed in the luteal phase [10], [33]. The effect of this is an estradiol deficit, which, from the standpoint of sexual health, contributes to the cascade of urogenital atrophy already described in the context of energy restriction (RED-S) [28]. In both sexes, an additional adverse effect arises from the redistribution of amino acids and from the tissue catabolism intensified in OTS, which presents as a loss of fat-free mass (skeletal muscle), increasing physical fatigue, disturbances in sleep quality and structure, and depressed mood [23]. It should be emphasized that the symptoms of OTS — despite removal of the training stimulus during recommended breaks — not infrequently show a degree of hormonal inertia; the CRH and cortisol cascade may fail to normalize in athletes for many months, sustaining a diagnosis of HSDD and ED long after recovery measures have been introduced [23], [33], [50].

4.2.2. Changes in Thyroid Hormone and Prolactin Profiles and Reduced Libido

While activation of the adrenal (HPA) axis and a reduction in testosterone/estradiol within the gonadal (HPG) axis are widely analyzed and documented mechanisms of overtraining syndrome (OTS), disturbances within the hypothalamic-pituitary-thyroid (HPT) axis and particular fluctuations in prolactin (PRL) concentrations appear to play an equally important — and often underdiagnosed — role in the pathophysiological neuroendocrine cascade [23], [33]. An understanding of these subtle relationships is particularly important when working with an elite athlete reporting a progressive decline in libido (hypoactive sexual desire disorder under the DSM-5-TR), apathy, and an absence of morning erections that do not resolve despite the introduction of, for example, a diet with a positive energy balance [23], [25]. Under conditions of high load, both of these hormonal pathways undergo modifications that go beyond classic physical fatigue and may have a considerable effect on brain neurochemistry and urogenital structures.

In a healthy athlete, the thyroid acts as a key regulator of the rate of cellular metabolism, and its parameters (particularly the concentration of thyroxine, T4, and its biologically active metabolite, triiodothyronine, T3) are well matched to the oxygen demand arising from EEE. When OTS develops — effectively a form of biological shock to the body — a marked restructuring of tissue deiodinase activity occurs. In athletes, an increased inhibition of the peripheral conversion of T4 to active T3 is observed, with a simultaneous rise in the production of a biologically inert isomer with receptor-antagonist properties, known as reverse triiodothyronine (reverse T3, rT3) [10], [23], [33]. This phenomenon, termed euthyroid sick syndrome (or low-T3 syndrome) in endocrinology, is characterized by normal TSH and T4 concentrations but an abnormally low free-T3 concentration. A reduction in T3 combined with an excess of antagonistic rT3 produces a generalized slowing of intracellular metabolism [23]. For sexual health, this may have a considerable effect on the functioning of the patient's cerebral cortex and limbic system; a generalized slowing of synaptic transmission, a blunting of sensory perception, and a biochemically conditioned "sluggishness" may develop that undermines sexual initiative and cognitive responsiveness to erotic desire [25], [33]. At the same time, a low thyroid level promotes vasoconstriction (constriction of peripheral vessels) and reduces the intracellular function of the cells involved in relaxing the pelvic venous system during the arousal phase in both men and women [10], [23].

A further element in the picture of neuroendocrine disturbance in OTS — one that, from the standpoint of clinical sexology, may act as a primary damaging mechanism — is the hyperprolactinemia specific to heavy training. Although prolactin has traditionally been associated almost exclusively with the stimulation of lactation and with nurturing behavior, it serves considerably broader adaptive functions. Under the influence of the chronic, intense biological stress associated with OTS — particularly after heat stress, chronic sleep deprivation, hypoglycemia induced by endurance exercise, and, above all, a substantial release of stress-related opioid peptides and vasopressin — an increased, abnormal, and persistent secretion of prolactin from the anterior pituitary is observed in athletes of both sexes [23], [33].

A chronically elevated prolactin concentration acts on the reproductive system and the brain with considerable inhibitory precision. Directly, at the central level, PRL is a strong inhibitor of kisspeptin and dopamine secretion — and it should be recalled that dopamine is the neurochemical foundation of the reward centers (the mesolimbic pathway) that underlie the experience of arousal, desire, euphoria, and orgasm itself [10], [23]. In an athletic patient, high prolactin "cuts off" the dopaminergic pathways, leading to anhedonia and affective blunting (including a suppression of libido) that, in psychiatric terms, may resemble a serious, treatment-resistant, SSRI-refractory depression [3], [25]. At the peripheral level, an excess of prolactin

competitively blocks the releasing hormone (GnRH) itself in the hypothalamus, contributing directly — and in parallel with the cortisol mechanism (discussed in Section 4.2.1) — to a collapse of testosterone synthesis in male athletes and of estrogen synthesis in female athletes [9], [28], [33]. In male athletes, the consequence of this may be a secondary, pronounced hypogonadism presenting not only with severe erectile dysfunction and a marked sexual aversion but also, not infrequently, with impaired spermatogenesis and gynecomastia [9], [50]. It should be added here, as a frequently overlooked toxicological point, that an identical response — blockade of the reproductive system by an excess of prolactin — is promoted in the competitive setting by certain steroid derivatives (AAS) such as nandrolone, a subject discussed in more detail in Section 7.1 [20], [38].

4.3. Exercise-Induced Hypogonadotropic Hypogonadism (EIHH) in Men

Exercise-induced hypogonadotropic hypogonadism (EIHH) in men is a specific and burdensome form of male hormonal insufficiency directly related to high training volumes [9]. While the influence of RED-S (including low energy availability) on the development of functional hypothalamic amenorrhea (FHA) in women is relatively well documented and established in medical awareness [10], [28], [57], the pathology of the male HPG axis in response to chronic caloric deficit and overload (for example, in OTS) is not infrequently substantially underestimated. The mechanism of EIHH represents a profile — particular to elite sport, and especially endurance sport — of subclinical, and not infrequently fully clinically overt, damage to the neuroendocrine system, in which, despite preserved autonomous testicular function (no primary Leydig cell damage), the central, hypothalamic-pituitary control of androgenesis is suppressed [9], [51].

Within an EBM framework, the basis of the etiopathogenesis of EIHH is the coupling of chronic low energy availability (LEA) with chronic activation of the stress axis (HPA). It should be clearly emphasized that this cascade does not arise from a single maximal-intensity session but appears to require chronic, accumulating exposure to a distressor without adequate windows of detraining and refeeding (nutritional restitution) [9], [50]. Under sustained LEA accompanied by hypercortisolism, signaling from the peripheral tissue glucostatic and leptinostatic sensors falls markedly. In the arcuate nucleus of the hypothalamus, this implies an almost complete silencing of the kisspeptin neurons that physiologically activate the GnRH-producing cells [10], [43]. In men, the consequence is a documented breakdown in the amplitude and frequency of the physiological "pulses" of GnRH released into the pituitary portal circulation [58].

A decline in hypothalamic GnRH stimulation translates into a decline in anterior pituitary production of both key gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH). From the standpoint of sexual health and the clinical diagnosis of dysfunction under the ICD-11 and DSM-5-TR, the attenuation of the LH signal is of particular importance [25], [26]. The absence of LH reaching the receptors of the Leydig cells located in the interstitial tissue of the testes results in a significant, months-long shortfall in the synthesis and peripheral secretion of total testosterone (T) and of its metabolically more important fraction, free testosterone (fT) [9], [42], [51]. A reduction in T (often well below the 300 ng/dL threshold commonly used to define LOH in older patients) leads to a marked functional restructuring of the penis; the activity of nitric oxide synthase in the corpora cavernosa (nNOS), which supports erection by inducing vasodilation and the veno-occlusive mechanism, declines markedly [27], [50]. The consequence may be the clinical development of erectile dysfunction (ED), which is frequently accompanied by a generalized decline in libido related to the lack of dopaminergic stimulation and by an inability to experience attraction (HSDD) [9], [25]. Under EIHH conditions, this picture is very often accompanied by a decline in bone mineral density,

substantial central nervous system fatigue, and reduced adaptive capacity for building and maintaining muscle tissue [50], [51].
This phenomenon appears to be strongly dependent on temporal characteristics — the prevalence and severity of EIHH correlate closely with so-called *years in training*. Recent population studies (including analyses involving marathon runners, ultra-cyclists, and speed skaters) indicate that the degree of reduction in T synthesis in patients with EIHH increases approximately linearly with the cumulative duration of long-term high-volume distress [42]. The athlete does not automatically return to homeostatic T values between seasons, which points to the development of marked central fatigue of the HPG axis. This mechanism of accumulating neuroendocrine "inertia" is one of the more serious challenges in the medical treatment of an elite athlete contending with a substantial loss of sexual function [9], [42]. The damage — reversible through a reduction in the T/C (testosterone-cortisol) ratio — sometimes requires several weeks or months of caloric optimization combined with de-escalation (detraining), whereas the use of conventional testosterone replacement therapy (TRT), because of the WADA policy that closes off the patient's return to official competition (doping), is treated very strictly with respect to TUE in this group [8], [50]. A consequence of the imposed anti-doping restrictions is a paradox in which a patient with EIHH and an accompanying acute dysfunction of the reproductive organs is subject to a prolonged regimen of behavioral and nutritional therapy alone [50].

4.4. Functional Hypothalamic Amenorrhea (FHA) in Women and Its Direct Relationship with Dyspareunia (Vaginal Epithelial Atrophy, Reduced Lubrication)

In the population of elite female athletes, the most documented, troublesome, and pathognomonic consequence of relative energy deficiency in sport (RED-S) is functional hypothalamic amenorrhea (FHA). FHA is not an isolated anomaly of the reproductive system but a marked manifestation of neuroendocrine decline, in which the nervous system — faced with critically low energy availability (LEA) — deliberately and adaptively withdraws "support" from reproductive processes, which under conditions of sustained stress are treated as evolutionarily secondary [5], [10]. As the Endocrine Society's clinical guidelines specify, a diagnosis of FHA is made by exclusion (*per exclusionem*) in patients presenting with an absence of menstrual cycles lasting more than six months (or the absence of three consecutive cycles in women previously menstruating regularly), with no anatomical etiology (for example, congenital uterine anomalies), no genetic etiology (for example, premature ovarian insufficiency, POI), and no organic pituitary disease (for example, a prolactin-secreting adenoma) [10]. In the athletic population, FHA almost always coexists with an abnormally low or borderline body fat content, hypercortisolemia secondary to overtraining (OTS), and reduced leptin and kisspeptin concentrations [10], [28], [43].
At the central level of the hypothalamic-pituitary-gonadal (HPG) axis, the key defect in FHA is a marked attenuation of the amplitude and frequency of the physiological pulses of hypothalamic gonadotropin-releasing hormone (GnRH). The chronic absence of pulsatile stimulation of GnRH receptors on the gonadotropic cells of the anterior pituitary leads to inhibition of the secretion of luteinizing hormone (LH) and — to a somewhat lesser degree — follicle-stimulating hormone (FSH) [10], [43]. The peripheral consequence of this "unresponsive hypothalamus" is the suppression of ovarian follicle stimulation. In the ovaries, despite a preserved oocyte reserve, full maturation of the Graafian follicle does not occur, there is no preovulatory LH surge, and the granulosa cells lose their capacity to produce endogenous estrogens (above all estradiol, E2, which is central to the reproductive phase) and progesterone [28], [43]. This gives rise to a clinical state of pronounced, persistent, and chronic

hypoestrogenism that has a considerable effect on the entire architecture and physiology of the pelvic organs [10].

It is this pronounced, systemic estradiol deficit that constitutes the direct link between FHA and the serious sexual dysfunction reported by female athletes (classified in the DSM-5-TR as genito-pelvic pain/penetration disorder, GPPPD, and in the ICD-11 as separate dyspareunia and/or vaginismus) [25], [26], [31]. Under physiological conditions, estrogens are responsible for the proliferation, differentiation, and maintenance of the multilayered, non-keratinizing squamous epithelium lining the walls of the vagina and vulva. A normal E2 level ensures a high glycogen content in the superficial cells, which (metabolized to lactic acid by the physiological *Lactobacillus* flora) is responsible for the protective, acidic pH of the vaginal environment [28], [29]. Moreover, estrogens condition the appropriate elasticity of the blood vessels of the pelvic venous plexus, which during the sexual arousal phase are responsible for the rapid vasocongestion of the organs and the production of transudate (physiological lubrication) that facilitates penetration [29].

In a young competitive athlete affected by FHA (for example, a figure skater or long-distance runner in her twenties), the hypoestrogenism conditioned by energy deficiency (LEA) may give rise to a pathology of the reproductive organs resembling the picture of the aging organ after menopause (genitourinary syndrome of menopause, GSM). This phenomenon is termed urogenital atrophy [10], [24], [28]. The vaginal epithelium thins markedly, the cells lose glycogen (thereby increasing the risk of recurrent, painful fungal and bacterial infections), the connective tissue loses its elastin-collagen architecture, and the vessels of the vulvar plexus may become unable to generate sufficient vasocongestion in response to an erotic stimulus [10], [29]. As a result, the process of physiological lubrication is largely — and in some cases almost entirely — halted.

Under such conditions, any attempt at sexual activity with a partner meets a considerable mechanical barrier — the thin, mucus-deprived vagina is subject to painful friction. Painful microtrauma, mucosal tears, and not infrequently secondary post-traumatic bleeding may develop [25], [28]. The purely organic, atrophic dyspareunia that arises in this way may initiate a substantial secondary psychogenic cycle. A mechanism of aversive conditioning is set in motion in the patient: each subsequent act becomes associated with pain and threat. Anticipatory fear of penetration develops, resulting in a reflexive, involuntary, protective spasm of the striated pelvic floor muscles (colloquially termed secondary vaginismus), forming a physical "wall" that deepens the trauma and distress [17], [28], [29]. Ultimately, this coupled somatic-psychogenic dysfunction (GPPPD) leads to a breakdown of the sexual response cycle — libido collapses (a secondary hypoactive sexual desire disorder under the DSM-5-TR, since sexual desire is conditioned not only by neurochemistry but also by positive reinforcement) and orgasm becomes unattainable [25]. The mechanism outlined here illustrates the troubling façade of the sporting environment, in which the loss of menstruation (FHA) is sometimes regarded as an innocuous "sign of good form" [3] while in fact contributing to considerable harm to the sexual and psychological health of a young, elite patient [10].

5. Psychogenic Factors and Psychiatric Burden

5.1. Competition Stress and Performance Anxiety in Relation to Erectile Dysfunction and Anorgasmia

Any consideration of the pathophysiology of sexual dysfunction in the competitive setting would be markedly incomplete if the analysis were confined solely to the neuroendocrine and biomechanical domains. In the elite-athlete population, the psychogenic component does not serve merely as a secondary, reactive echo of somatic injury but not infrequently presents as a

primary, substantial etiological factor [3], [14]. The construct of a competitive career rests, by definition, on continual evaluation, sustained exposure to harsh judgment (from coaching staff, sponsors, the mass media, and rivals), and a constant requirement to optimize performance [11]. This demanding environment generates a distinctive set of cognitive and affective burdens, among which performance anxiety plays a leading role. This anxiety, initially directed at purely sporting challenges (competition stress), tends, through complex neurobiological mechanisms and psychological conditioning, to generalize (carry over) to the patient's intimate life, with a disruptive effect on the arousal and orgasm phases — giving rise, respectively, to erectile dysfunction (ED) in men and anorgasmia in both sexes [1], [3], [14].

In psychopathological and neurobiological terms, performance anxiety is not an ordinary, physiological feeling of nerves. It is a substantial convergence of distress and cognitive distortion, in which the patient catastrophizes the forthcoming test of their abilities, anticipating humiliation or a marked loss of status [14]. At the level of the nervous system, fear of failure (particularly the form activated before key events such as the Olympic Games or World Championships) produces a strong, tonic activation of the sympathetic nervous system (SNS) with a substantial release of noradrenaline and adrenaline [14], [23]. Although this "fight-or-flight" response may optimize reaction time on the track, in the context of sexual health the release of catecholamines acts as a marked barrier to the physiological vasodilation mechanisms needed to achieve arousal. In men, erection is a process strongly dependent on the predominance of the parasympathetic system, which, through nitric oxide (NO) and the cGMP pathway, relaxes the smooth muscle of the cavernosal vessels [27]. Anticipatory pre-competition anxiety and the accompanying generalized sympathetic tone — at times present around the clock — may markedly restrict the arterial blood supply to the penis, causing a "constriction" of the peripheral vessels. This restricted supply, correlated with high anxiety, is often the first psychogenic catalyst — independent of hypogonadism — of significant erectile dysfunction in young, fit athletes [12], [14]. In terms of the diagnostic classification, such a patient presents with erectile disorder under the DSM-5-TR, of generalized or situational origin (linked to the peak of the season) [14], [25].

Moreover, performance anxiety in sport tends to carry over readily to sexual activity itself. An athlete accustomed to a requirement of perfection and to a task-oriented relationship with their own body may approach an intimate encounter with a partner in a conditioned, "performance-oriented" manner (the "spectator role," or spectatoring, in the sense used by Masters and Johnson) [14], [41]. Instead of becoming cognitively immersed in erotic stimuli and affective experience, the athlete's attention shifts to an objective, anxious appraisal of the rigidity of their own penis (in men) or the degree of lubrication (in women). At the moment when an erection appears more slowly or is weaker (even as a result of physiological post-training fatigue), a cognitive distortion is set in motion: the patient concludes that they have "failed," which generates a secondary, acute surge of noradrenaline. This rapidly dispels the existing arousal (loss of erection during penetration) and may draw the athlete into a self-reinforcing cycle of aversive conditioning — each subsequent act being anticipated with progressively greater fear of sexual failure [12], [14].

The same hypervigilant and anxious cognitive pattern bears just as heavily on the capacity to reach climax (anorgasmia or delayed orgasm — delayed ejaculation or female orgasmic disorder) [25], [31]. Orgasm, as a peak neuro-autonomic discharge, requires — once sufficient tension has been reached — a phase of deeply "letting go" of control [14]. Athletes in whom years of training impose a grueling hypercontrol of every muscle, breath, and emotion (for example, in gymnastics, ski jumping, or skating) often lose the capacity to relinquish cognitive oversight in intimate moments [1]. Their brain, kept in a constant state of heightened, competition-stress-conditioned alertness (hypervigilance), may be unable to quiet the prefrontal cortex sufficiently to allow the cascade of the orgasmic reflex controlled by older, deeper limbic

structures and the reward circuit [1], [14]. This results in distressing frustration, since, despite preserved libido (a desire for closeness) and the presence of initial arousal, the intimate act fails to reach a physiological and psychological culmination, intensifying the athlete's distress and emotional isolation [25], [41]. Taken together, this psychogenic configuration is a model example of a situation in which an originally healthy body becomes functionally blocked by the harmful effect of exaggerated perfectionism and pressure to perform [3], [14].

5.2. The Occurrence of Affective Disorders (Depression) and Anxiety Disorders in Sport as a Co-Factor of Sexual Dysfunction

After decades of affirming the myth of the elite athlete as a being immune to mental disorder, evidence-based medicine (EBM) now clearly argues for the need to integrate psychiatric care into sports medicine [3]. Publications prepared under the auspices of the International Olympic Committee (IOC), as well as extensive meta-analyses, provide data showing that the professional sporting environment functions as a kind of "pressure cooker" that predisposes to mood and anxiety disorders at a level comparable to — and, in many of the cohorts studied, exceeding — the rates recorded in the general population [3], [4], [11]. Depending on the career stage, the prevalence of symptoms of severe depression, dysthymia, generalized anxiety disorder (GAD), or adjustment disorders (including those secondary to serious, game-ending injuries) may range between roughly 15% and 35% of active elite athletes [4], [11]. Of importance for the present review is that these nosological entities do not function clinically in a vacuum; they appear to be among the strongest neurobiologically and pharmacologically conditioned independent co-factors contributing to sexual dysfunction that is severe and resistant to simple symptomatic treatment in this particular demographic subgroup [3], [25].
Depression (major depressive disorder, MDD), both in the general population and among the sporting elite, has a well-established direct bearing on the suppression of the desire phase [25]. According to the DSM-5-TR criteria, a clinically conditioned reduction in drive, including anhedonia (an inability to experience pleasure from activities that previously provided it, including sexual contact), is a core symptom of significant mood disorders [25]. In athletes, however, this pathomechanism is intensified in a particular way. At the endocrine and neurotransmitter level, a depressive episode is closely correlated (often in a bidirectional, self-reinforcing relationship) with hyperactivation of the HPA axis and, consequently, with hypercortisolemia secondary to overtraining (OTS) [23], [33]. Psychological distress induces a central depletion of the monoaminergic pathways — in particular the dopaminergic pathways of the mesolimbic system, which, as noted above, underlie physiological sexual initiative, the pursuit of erotic reward, and the maintenance of arousal [3], [30]. In addition, deficits in serotonergic signaling impair the regulation of affect, leading to apathy, social isolation (avoidance of partner relationships), a growing sense of hopelessness, and a markedly lowered sense of self-worth, which in an athlete may be further affected by a downturn in performance [4], [11].
The clinical picture is further complicated when iatrogenic factors enter the treatment of depression in an athlete. Because training breaks are avoided (something that long-term psychotherapy, for example, requires), the most common, routine first-line intervention in the elite is the introduction of pharmacotherapy in the form of selective serotonin reuptake inhibitors (SSRIs) or serotonin-noradrenaline reuptake inhibitors (SNRIs) [3], [30]. Unfortunately, SSRIs — acting by raising serotonin concentrations in the synaptic cleft, with a strong affinity for 5-HT2C receptors in the hypothalamus and the efferent pathways of the pudendal nerves — have a particularly unfavorable profile with respect to the patient's sexual health [30]. The side effects of SSRIs documented in population and clinical studies, occurring in 40% to as many as 70% of the patients taking them, include a marked suppression of desire

(secondary hypoactive sexual desire disorder), an inability to achieve a full, rigid erection (erectile dysfunction), and severe anorgasmia or markedly delayed ejaculation [30], [40]. From a psychiatric and sexological standpoint, an athlete treated with SSRIs without appropriately careful monitoring (underreporting in the psychiatric consultation owing to shame) [22] not infrequently experiences a paradoxical improvement in mood (resolution of insomnia and tearfulness) together with a chemically conditioned, "castrating" removal of drive, which creates an unacceptable life conflict for a young, biologically healthy person [3], [30]. Of considerable importance from the standpoint of the ICD-11 and more recent findings is the existence — and the gradual acceptance by the medical community — of an entity termed PSSD (post-SSRI sexual dysfunction): a treatment-resistant absence of sexual function and a genital anhedonia lasting months or years after the complete and appropriate discontinuation of antidepressants, which for a competitive athlete may mean a lasting loss of sexual function and may, at the end of the cycle, contribute to a pharmacologically conditioned relapse of depression [30].

In the case of anxiety disorders (including generalized anxiety disorder, GAD, and panic disorder, which not infrequently coexist in sport with intense competition pressure and fear of losing a sporting contract after an injury), the pathomechanism rests, in turn, on the sustained activation of the adrenergic system described in the previous section [11], [14]. Unlike the narrowly defined performance anxiety (directed at the moment of competition and at intimate activity itself), however, generalized anxiety produces a constant, subthreshold depletion of the central nervous system and chronic disturbances of sleep architecture [14]. This entails a general decline in vital energy and a reduction in testosterone production through the cortisol-mediated coupling described under RED-S, paving the way in the athlete from cognitive fatigue to an erectile deficit secondary to psychological symptoms and resistant to PDE5 inhibitors, and to atrophic vaginal changes (in FHA) in women [10], [23], [25]. In summary, affective and anxiety disorders in elite athletes, constituting a complex network of psychological and molecular interdependencies, call for multi-track diagnostics that take into account not only the brain biology impaired by overtraining but also the deliberate avoidance of therapeutic "traps" that act iatrogenically on reproductive functions that are already fragile and burdened in the elite [3], [4], [30].

5.3. Eating Disorders (Anorexia, Bulimia, Orthorexia) and Body-Image Disturbances (Body Dysmorphia, Bigorexia) in Relation to Sexual Function

The particular characteristics of the competitive-sport environment — a cult of competition weight, an idealized and unattainable definition of "form," and not infrequently severe, cyclical body-mass reduction procedures (the phenomenon of weight cycling in combat sports and rowing) — generate a marked rise in the prevalence of eating disorders (EDs, not to be confused with erectile dysfunction) relative to the general population [47], [53]. According to position statements from the IOC and others, this phenomenon is no longer marginalized and is regarded as an independent risk factor for serious impairment of the athlete's mental and physical health and, from the standpoint of the present work, of psychosexual health [3], [5]. The development of disorders from the group of clinical anorexia nervosa, bulimia nervosa, and the orthorexia (orthorexia nervosa — a pathological preoccupation with healthy eating that adversely affects the energy balance) increasingly described in the fitness and endurance community not only fits pathophysiologically within the RED-S paradigm [5], [7] but may also affect the patient with a substantial set of disruptive, purely psychiatric symptoms (defined in the DSM-5-TR) that undermine the foundations of body self-acceptance and desire [25].

Pathological eating patterns are, in their psychological essence, an attempt to regain control (often illusory) in an environment of competition pressure, and they consistently coexist with

serious cognitive distortions in the domain of one's own physicality [3], [47]. In women, anorexia (common in aesthetic sports such as artistic gymnastics and figure skating) — alongside the organic shutdown of the HPG axis conditioned by cachexia (which leads to the FHA, atrophy, and dyspareunia described in Section 4.4) [10], [28] — entails a marked loss of the capacity to experience physical pleasure [25]. The body of a female athlete affected by anorexia (biologically severely depleted, yet still regarded by her as "too fat") may undergo a defensive dissociation in which the senses and receptivity to touch are disconnected. Intimate contact with a partner provokes a panicked fear of "exposing" supposed bodily flaws (characteristic of the broader body-image disturbance), and this anxiety, by heightening sympathetic activation and a defensive vaginismus, may make orgasm physiologically and cognitively unattainable and generate a pronounced sexual aversion [17], [25], [28]. In addition, compensatory behaviors in bulimia (for example, purging through vomiting or diuretics — prohibited by WADA [49]) deepen not only electrolyte deficiencies and cardiac strain (hypokalemia) but also a hatred of one's own physiology and a sense of shame and humiliation, undermining the patient's relational and erotic foundation in their relationship with themselves [47]. In orthorexia, the strict restriction of dietary intake of, for example, fats reduces the cholesterol pools needed for steroidogenesis (the synthesis of sex hormones in the gonads), chemically reinforcing the progressive suppression of drive that develops in these patients as a result of dogmatic psychosocial restrictions [50], [53].

In parallel, in the leading strength-based disciplines (bodybuilding, weightlifting, hammer throw, and, increasingly, among athletes in contact disciplines such as American football and MMA), a particular pathology conditioned by anxiety and belonging to the obsessive-compulsive group becomes firmly established — muscle dysmorphia (MD), colloquially and imprecisely termed "bigorexia" (or reverse anorexia) [13], [25]. This disorder is most prevalent among the elite of male athletes, who, despite having a clinically defined hypertrophy and a subphysiological level of body fat, experience an irrational, disabling conviction of being "puny," insufficiently developed in muscle mass, and inadequately muscular relative to distorted standards [13]. As systematic reviews have documented, MD shows a strong co-occurrence with a retreat into pathological eating patterns, the use of prohibited pharmacological aids (AAS) at potentially life-threatening levels, and — importantly for this analysis — advanced sexual and relational dysfunction [13], [38].

In the course of MD, sexual health and satisfaction may become almost entirely marginalized relative to the priorities of the eating cycle, training of increasing load, and physique appraisal. Pathological physique perfectionism distorts the male athlete's sense of masculinity and competence [13]. This phenomenon may lead to marked avoidance of sexual contact, arising from the conviction that exposing the body to a partner will reveal imagined "deficiencies in mass" (the fear of exposure, the phenomenon of body concealment) [13], [41]. The considerable cognitive stress generated in this way may produce secondary, neurogenically conditioned erectile dysfunction (ED) under the DSM-5-TR, initially independent of the level of endogenous testosterone. A brain dominated by obsessive rumination about muscle symmetry may be unable to engage the vasodilatory mechanisms, withdrawing the athlete into isolation. The situation may escalate pathophysiologically and clinically when a patient with body dysmorphia turns to anabolic-androgenic steroid cycles in an illusory attempt to remedy objectively non-existent deficiencies; in doing so, he may move from a psychogenic dysfunction directly into an iatrogenic pharmacological collapse, including post-cessation hypogonadism (ASIH), undermining his chemically conditioned — and already fragile — self-confidence and capacity for erection [13], [18], [38].

In summary, body-image disturbances in sport — both at the pole of striving for extreme thinness (anorexia) and at that of an anxious pursuit of hypertrophy (bigorexia) — may undermine the foundations of sexual function through a combination of secondary endocrine

collapse with a sense of profound shame, self-disgust, and cognitive dissociation during attempts at intimacy with a partner, which is detrimental to erection and lubrication [13], [25], [53].

5.4. The Coach-Athlete Relationship and Team Dynamics in Relation to Psychological and Sexual Well-Being

An analysis of the etiology of psychogenic sexual dysfunction in elite sport extends beyond the neurobiological processes and intrapsychic cognitive distortions of the individual (such as body dysmorphia or performance anxiety) [13], [14]. The biopsychosocial paradigm, which is a foundation of contemporary sports psychiatry, requires that the social environment in which the athlete was formed be included in the analysis [1], [3]. In this context, one of the most powerful — and not infrequently most damaging — factors shaping an athlete's attitudes, identity, and intimate functioning is the particular, strongly polarized interpersonal dynamic of the coach-athlete relationship and, in team disciplines, the structure and pressure arising from life within the locker room (team dynamics) [1], [11]. It is this closed, hierarchical microenvironment that often appears to be a root cause of pathology (triggering dissonance and stigma), and that contributes to a generalized decline in psychological well-being with a direct bearing on the sexual and reproductive domain [3].

The relationship between a competitive athlete and the principal coaching staff (in particular the head coach) is psychologically distinctive and differs considerably from the student-teacher relationships familiar from other sectors. It is characterized by a marked asymmetry of power (power dynamics), in which the athlete's career (from scholarships, through a place in the first team, to participation in a target event) rests almost entirely with the decisions of a small group of decision-makers [1]. When a coach's management style relies on excessive control, verbal criticism, authoritarianism, and a lack of empathy (so-called toxic forms of coaching behavior), the athlete may internalize a pattern of chronic stress associated with subordination. This translates into sustained hyperactivity of the HPA axis (and the hypercortisolemia described above, which chemically undermines the stimulation of the reproductive system and contributes to a decline in T and estradiol) [23], but also into damage to the intrapsychic competencies needed to build mature partner relationships [1], [11].

Prolonged exposure to demeaning messages (often intended to target the athlete's body as part of weight control, which is common in aesthetic sports — for example, pointed comments about fat gain directed at adolescent gymnasts) may initiate the clinical picture of body-image disturbance and pave the way toward anorexia or bulimia (discussed in Section 5.3) [47], [53]. Moreover, oppressive treatment erodes the patient's domain of self-determination and decision-making (autonomy). The athlete becomes dependent, subordinate, and accustomed to carrying out the instructions of others, which has a considerable bearing on the sexual domain — a space that, for full physiological and emotional satisfaction (and for reaching orgasm), requires a sense of safety, bodily assertiveness, and autonomy in setting boundaries [25], [41]. A lack of these competencies generates the aversion-based conditioning described in earlier chapters, which may promote premature ejaculation in men and anorgasmia and atrophic dyspareunia (GPPPD in women — related to a lack of peripheral relaxation and a persistent perineal hypertonicity conditioned by tension and fear of being judged, including in the presence of an intimate partner) [14], [25]. The situation takes its most serious turn (as documented, among other things, in proceedings exposing offenses such as those in the United States women's gymnastics team) when this asymmetric relationship is grossly violated through harassment or sexual abuse by the staff (safeguarding failures in sport). Such traumas, with the pathophysiology of PTSD, may cause substantial structural changes in brain neuroplasticity and

constitute one of the most serious psychogenic and psychological co-factors of persistent sexual aversion [3], [25].
Equally important in mapping the patient's psychosexual picture is the dynamic of the closed environment of fellow competitors and the group pressure specific to a given subculture (for example, bodybuilding or boxing). The spaces of professional locker rooms are often (though not as a universal rule) a reservoir of so-called hegemonic or toxic masculinity/femininity [11], [22], where an individual's worth is narrowly measured by the capacity to endure pain at the expense of one's own health (normalizing competing with irreversible damage), by the masking of weakness (the underreporting and taboo described elsewhere), and at times by entrenched cultural narratives about hypersexuality and prowess. In such structures, phenomena such as the fear of losing the group's trust through a lapse (for example, becoming a victim of hazing after a competitive mistake) may considerably intensify overall distress. In strength sports, the cult of oversized muscle mass leads to the systematic marginalization and exclusion — as an element of "weakness" — of the need to report the side effects of doping (for example, absence of erection or testicular atrophy after AAS), trapping athletes with body dysmorphia in an isolation born of fear of disgrace at the hands of stronger "alpha males" within the team [13], [20], [48].
A lack of tolerance and acceptance of difference — for example, homophobia in the environments of certain contact and combat sports, or reputational harm — may compel athletes to conceal their true orientation and identity, something referred to as minority stress [3], [31]. The chronic stress of concealing one's true self is not only a co-factor of severe depression but may also considerably impair the function of the sexual cycle. In this struggle, the individual loses the ability to relax the neurovascular structures within a relationship, and the fear of disclosure may inhibit desire and the capacity to reach climax in a way closely analogous to the mechanisms described for performance anxiety [14], [25]. Only the deliberate implementation — supported by EBM evidence — of psychological care based on building the athlete's autonomy, on separating sporting results from their intrinsic "worth as a person," and on breaking the silence (reshaping the environment from one based on fear to one of safe collaboration under independent psychiatric oversight, safeguarding) makes it possible to relieve the accumulated tension, and it forms the basis of successful therapy that may help, among other things, the marriages of competitive athletes that are breaking down because of anhedonia and ED [1], [3], [41].

6. Mechanical, Vascular, and Traumatic Factors in Specific Disciplines

Whereas the etiology of sexual dysfunction in endurance and aesthetic sports is dominated by endocrine (RED-S, OTS) and psychogenic paradigms, in certain sport disciplines direct biomechanical factors play a key — and not infrequently the sole — pathogenetic role [15], [34]. Mechanical injuries and compression-related ischemic phenomena warrant thorough differential diagnosis, because, unlike reversible declines in testosterone or performance anxiety, chronic exposure to microtrauma of the nervous and vascular systems may lead to an irreversible structural loss of reproductive function lasting the athlete's entire life [44], [45]. Competitive cycling and equestrian sports are a classic reservoir of these highly specific, repetitive urogenital injuries [35], [52].

6.1. Cycling and Equestrian Sports

6.1.1. Pudendal Neuropathy (Alcock's Canal Syndrome): The Compression Mechanism and Symptoms (Numbness, Sensory Disturbance, ED)

The pudendal nerve (nervus pudendus), a key somatic-autonomic connection between the central nervous system and the structures of the perineum, is essential for initiating and sustaining the full sexual response cycle in both sexes. Formed from the ventral branches of the sacral plexus (S2-S4), this nerve leaves the pelvis through the greater sciatic foramen, wraps around the ischial spine, and re-enters the pelvis through the lesser sciatic foramen, then running within the tight, fascial pudendal canal (Alcock's canal) formed by the splitting of the fascia of the obturator internus muscle [34], [44]. This anatomical position — between hard osteochondral structures (the ischial tuberosities) and fascia — leaves the nerve poorly protected against substantial compressive forces. In competitive cycling (road, track, and mountain biking) and in professional equestrianism, the entire weight of the trunk and upper limbs is transferred (through a narrow saddle) onto the perineal region rather than, as under physiological conditions, onto the buttocks and ischial tuberosities [34], [52].

During prolonged training stages lasting many hours (in competitive conditions, not infrequently exceeding 4–6 hours per day), sustained pressure on Alcock's canal causes a crushing of the pudendal nerve fibers (mechanical neuropraxia and, in extreme cases, axonotmesis). The injury mechanism is twofold: the direct crushing force on the nerve between the pelvic bones and the saddle, which is considerably compounded by secondary ischemic damage [45], [52]. The crushing of the nerve's nutrient vessels (vasa nervorum) causes local ischemia of the nerve fibers, initiating an inflammatory response, edema, and subsequent fibrosis of Alcock's canal itself, which entrenches the compression process and damages the nerve. In athlete-patients, this pathology is clinically defined as Alcock's canal syndrome or, more broadly, pudendal neuralgia/neuropathy [34], [44].

The clinical manifestation of pudendal neuropathy in elite athletes is variable and not infrequently develops insidiously over several seasons, which unfortunately aligns with the tendency of patients to ignore the symptoms (the normalization of injury within the athletic group). The first, most common, and at the same time concerning symptom of damage to the somatic sensory fibers is the experience of paresthesia — tingling, "pins and needles," and numbness — localized precisely to the perineum, scrotum, and penis in men and the vulva and clitoris in women, appearing during riding or within a short window after dismounting [34], [45]. Many cyclists report a complete loss of touch sensation (genital numbness) in this area, which at an early stage is reversible within just a few hours of getting off the trainer but, under conditions of professional practice with no breaks over many years, may become a chronic pathology [35], [52]. Ignoring these sensory deficits and continuing the compression gives rise to symptoms of neuropathic pain — defined in clinical terms (the Nantes criteria) as a troublesome, burning, or electric-shock-like pain that intensifies in the sitting position (relief occurs on straightening the pelvis, for example in the toilet) — which considerably disrupts daily functioning and undermines sexual initiative in the patient [34], [44].

The most pronounced consequences, in terms of the classification of dysfunction (such as the ED category in the DSM-5-TR), arise from damage to the autonomic and motor fibers of the pudendal plexus. This nerve supplies the striated muscles of the perineum (including the bulbospongiosus and ischiocavernosus). In men, their damage prevents the maintenance of high rigidity in the final, rigid phase of erection, so that the erection may fade quickly during attempts at penetration and may be largely resistant to targeted pharmacotherapy (PDE5 inhibitors are, for obvious mechanical reasons, often not fully effective in neuropathy) [44], [52]. In addition, damage to the efferent sensory branches (a complete loss of conduction for tactile stimulation of the surface of the penis and clitoris) means that, despite attempts at intimacy, athletes may remain cut off from sensory arousal and exposed to persistent anorgasmia and an irreversible loss of the capacity to derive positive sexual reinforcement (genital arousal disorder), not infrequently requiring rescue surgical decompression of the pudendal nerve (urological

procedures), since a change of training introduced years too late is no longer effective [17], [44], [45].

6.1.2. Perineal Injury and Ischemia of the Pudendal Arteries (the Saddle Compression Factor)

Alongside pudendal neuropathy (Alcock's canal syndrome), the second most serious — and frequently coexisting (a neurovascular syndrome) — pathology in sports requiring prolonged exposure to a saddle (cycling, mountain biking, equestrianism, and not infrequently competitive rowing) is direct vascular injury and extensive ischemic damage to the arteries of the lesser pelvis and perineum [35], [52]. In terms of flow physiology, maintaining normal function of the reproductive organs — and in particular the capacity to generate a full erection (in men) and adequate clitoral vasocongestion and lubrication (in women) — is a process that depends strictly on an undisturbed, adequate supply of oxygenated blood. This blood is delivered by the region's main supplying vessel, the internal pudendal artery (arteria pudenda interna), a branch of the internal iliac artery, which, like the nerve, passes through the lesser sciatic foramen along its course, exposing it to substantial compression against the bone in competitive athletes [44], [52].

A key diagnostic concept in this area of biomechanics is the so-called saddle compression factor. Under physiological conditions, the weight of the trunk on a flat seat is distributed evenly over the ischial tuberosities (the "sit bones"). In modern, aerodynamic road and track cycling (particularly in aero positions in triathlon and time trials), the geometry of the bicycle forces the athlete into a marked forward tilt of the pelvis (a flexion of around 45 degrees or more). In this position, the ischial tuberosities are elevated, and the entire weight of the trunk — multiplied by the force of pressure on the pedals — is transferred directly onto the central and anterior perineum (the pubic symphysis), pressing the arteries and veins against hard tissues and the very narrow nose of the saddle [52], [59]. Modern measurements of pressure beneath the perineum (using strain-gauge sensors) show that intra-perineal pressure in professionals in the drop position not infrequently exceeds 300–500 mmHg — whereas in medicine it is held that compression of a vessel by a force of just 30–40 mmHg completely halts capillary and venous flow within it, and values on the order of 80–100 mmHg are capability of occluding the supplying arteries [45], [52]. This means that for most of a multi-hour training session, the structures of the cyclist's reproductive organs operate under conditions of marked ischemia, substantial tissue hypoxia at the endothelial level, and obstructed outflow of metabolites [52].

The consequences of chronic, repetitive pudendal ischemia (with a post-training, pathophysiologically harmful phase of sudden, mitochondrion-damaging reperfusion) vary depending on the athlete's sex and anatomy, but in both groups they lead to disturbances in the sexual domain and to diagnoses that fall within the ICD-11 criteria. In men, entrapment and crushing of the pudendal artery and of the dorsal and deep penile arteries arising from it produce direct mechanical damage to the endothelial cells. Endothelial dysfunction develops, in which the baseline production of nitric oxide (NO) needed to initiate the cGMP cascade is impaired [27]. The most serious risk for cyclists with very high training loads, however, is not the lack of nitric oxide itself but rather the arterial thrombosis within the perineal branches caused by compression and shear stress, together with the formation of microaneurysms and fistulae [52]. This may lead to a structural "narrowing" of the vascular bed (post-traumatic atherosclerosis), resulting in clinically overt, significant erectile dysfunction (ED). What is more, because of the vascular basis of the obstruction in such athletes, the pharmacological use of phosphodiesterase type 5 (PDE5) inhibitors is often largely ineffective (the system cannot be filled, because the main route of blood supply to the corpora cavernosa is obstructed), which is one of the leading reasons for the end of an active sexual life or for the search for invasive solutions, including

implant surgery [27], [52]. This phenomenon occurs irrespective of the free testosterone level — an athlete may have a perfectly preserved HPG axis (no RED-S or OTS) and full libido and yet experience a mechanical inability to achieve an erection [9], [52].
In women (whose urological concerns were, strikingly, downplayed in early sports research), studies from the 2000s revealed comparably damaging rates of vascular-mechanical factors. Narrow bicycle saddles with high spreading pressure injure the labia and cause compression localized to the front of the perineum (particularly in female cyclists using the lowered-handlebar position in triathlon) and directly compress the vessels supplying the clitoral plexuses (the corpora cavernosa and the bulbs of the vaginal vestibule) [35], [59]. In an environment of perspiration and microtrauma, this prolonged mucosal ischemia of the vulvar organs leads, in many competitive female athletes, not only to acute swelling and hypertrophic changes of the labia (so-called cyclist's nodularity, an asymmetric labial hypertrophy that itself becomes a secondary source of anxiety and body dysmorphia in the context of exposure before a partner) but also chronically induces persistent disturbances of pelvic filling and vasocongestion (vulvar vasodilation) in the erotic response [35], [45]. The reduced blood inflow undermines the lubrication capacity of the vagina, impairing genital responsiveness and promoting a pronounced, organic dyspareunia (classified as GPPPD in the DSM-5-TR) that hinders orgasm and intensifies a secondary fear of painful intercourse in female athletes [17], [25], [45]. Multicenter studies have shown that genital numbness is recorded in more than half of female cyclists, and that close to a third of this population contends with clinically significant dysfunction, which should be a clear imperative for medicine to pursue careful equipment modification and the use of shorts with appropriate padding and a longitudinal cut-out in the saddle geometry for professionals [17], [35], [59].

6.2. Combat and Contact Sports (MMA, Boxing, American Football)

In contrast to endurance disciplines (where the neuroendocrine pathways of OTS and RED-S predominate) and cycling (where the main factor is compression of the pudendal canal), the sports described as collision and combat sports — such as mixed martial arts (MMA), professional boxing, American football, and rugby — are characterized by a different, highly forceful injury biomechanics. In this group, the etiology of sexual dysfunction rarely arises from a single, isolated energy overload but is rather the aftermath of the chronic accumulation of high kinetic forces and acceleration/deceleration vectors acting on the structures of the central nervous system (CNS) [15], [36]. These phenomena, for decades dismissed as an inherent cost of practising these spectator disciplines, are, in the light of the most recent evidence-based medicine (EBM) findings, increasingly recognized as having a considerable influence on athletes' sexual and reproductive health, acting as an independent factor that may trigger secondary, post-traumatic hypogonadism and generalized neuroendocrine insufficiency many years after the end of a competitive career [15], [46], [55].

6.2.1. Repetitive Head Injury and Mild Traumatic Brain Injury (mTBI) in Relation to Secondary Pituitary Insufficiency

The pituitary gland (glandula pituitaria), which serves as the command center for the peripheral endocrine glands, lies in a bony depression of the sphenoid bone known as the sella turcica. Because of its delicate anatomy, the infundibular stalk (infundibulum), through which neuropeptides from the hypothalamus reach the gland, and the very fine network of pituitary portal blood vessels are highly susceptible to damage from sudden shearing and rotational forces [15], [36]. In combat and collision sports, hundreds of thousands of subclinical and fully symptomatic blows to the head occur over the course of an athlete's entire career. Each strong

impact or knockout blow produces a mild traumatic brain injury (mTBI), or, colloquially, a concussion. Neuroradiological and autopsy studies indicate that these forces cause microtears and edema in the infundibular stalk, leading to disruption of blood flow in the pituitary portal system (ischemia and focal necrosis of the glandular tissue of the anterior lobe) and to mechanical disruption of the axoplasmic transport pathways [15], [46].
The consequence of these accumulated microtraumas is a clinical entity known as post-traumatic hypopituitarism (PTHP). The most striking feature of PTHP from the standpoint of sexual-health diagnosis is that the decline of the anterior lobe's hormonal functions does not occur abruptly after a single knockout but is chronic and progressive over months and years from the time the injuries accumulate, not infrequently becoming fully apparent only in the phase of sporting "retirement" [15], [36]. The gonadotropic cells (producing FSH and LH) and the somatotropic cells (producing growth hormone, GH) are, because of their location and vascular sensitivity, the most susceptible to ischemia within the pituitary and tend to be damaged first [15], [55].
The inhibition of pulsatile and basal production of luteinizing hormone (LH) directly affects the patient's testes, leading to a collapse of testosterone (T). A clinical picture of severe secondary post-traumatic hypogonadism develops [15], [46]. A man — often a former MMA fighter or boxer aged around 30–40 — may present at the urology clinic with pronounced, recurrent, and ultimately persistent erectile dysfunction that is resistant to standard behavioral modification. The decline in T secondarily affects the NO and nNOS synthesis pathway in the penis [27]. Unlike a patient with generalized, psychogenic ED related to performance anxiety, however, a former athlete of collision sports also presents with a pronounced, hormonally conditioned suppression of sexual drive, classified as male hypoactive sexual desire disorder [25]. The loss of libido and the anhedonia in PTHP are very often accompanied by a syndrome of generalized exhaustion, muscle atrophy, reduced bone density, and neuropsychiatric disturbances (aggression or treatment-resistant depression), which arises directly from the loss of the growth hormone (GH) axis that is inseparable, in this pathology, from involvement of the HPG axis [15], [36], [55]. Studies in a large group of amateur and professional boxers have shown that selective damage along this axis may affect as many as 15% to 30% of them within 10 years of leaving the sport, making the question of diagnosis in combat sports an important preventive priority [36], [55].
The difficulty in managing these patients lies in pronounced underreporting. Athletes who have sustained serious concussions often attribute declines in erection to the natural process of "aging" or to the stress-amplifying symptoms of CTE (chronic traumatic encephalopathy), when in fact they are affected by a latent, unrecognized cutting-off of the hypothalamic signal (including kisspeptin and GnRH) to a slowly degenerating, ischemic pituitary [15], [46]. Medical intervention in such a case — provided the diagnosis is made on the basis of detailed, specialized dynamic tests with clomiphene and insulin — should rest on the prompt introduction of testosterone replacement therapy (TRT) to restore erectile capacity and the quality of life of the patient with PTHP [15]. It should be borne in mind, however (a subject addressed in more detail in the section on pharmacotherapy), that the use of testosterone is not possible for athletes with an active license, owing to WADA and the difficulty of obtaining a TUE [8], which means that post-injury athletes who continue their careers may remain in an iatrogenic and trauma-related neuroendocrine state that has a damaging effect on their intimate lives [55].

6.2.2. Direct Injury to the Genital Organs

Whereas the pathogenesis of post-traumatic hypogonadism (PTHP) in contact-sport athletes is an insidious, drawn-out, neuroendocrine process [15], [36], a different class of threat —

immediate and pronounced in its urological presentation — is direct injury (blunt or penetrating trauma) to the external genital organs themselves (the perineum, penis, scrotum, and labia). The risk of acute pelvic trauma rises markedly not only in the aforementioned mixed martial arts (MMA), boxing, and American football — where, despite the use of protectors (cups), accidental or, in certain illegal fouls, intentional blows to the perineum are part of the biomechanics of competition — but also in winter sports (downhill skiing, hockey), motocross, and extreme cycling disciplines (for example, a forceful impact against the handlebar stem — straddle injuries) [35], [52]. From a clinical standpoint, these injuries are not infrequently overlooked and inadequately managed at the level of emergency care within the sporting facility, which, after months or years, results in the formation of pathological scarring, fistulae, and a substantial disruption of the hemodynamics needed for erotic responses, predisposing patients in their early twenties to a diagnosis of erectile dysfunction (ED) or GPPPD [25].

With respect to male anatomy, an acute injury to the scrotum and testes caused by a blunt force (for example, an unfortunate kick in combat sports with inadequate protection, a forceful blow from a hockey puck, or a collision with an opponent's knee on the rugby pitch) produces an immediate urological response ranging in severity from hematocele to rupture of the testicular tunica albuginea. An undiagnosed, overlooked subclinical rupture or deep contusion of the gonadal parenchyma (which frequently happens, again as a result of underreporting, owing to reluctance to report the injury to the coach [3]) generally activates a strong inflammatory process and fibrosis [52]. This mechanical and inflammatory process damages the Leydig cells mentioned in previous sections. The result of damage to the peripheral steroidogenic apparatus (in contrast to the pathogenesis of RED-S or pituitary collapse, where the testis is simply not activated by the pituitary) is, in this case, a pronounced hypergonadotropic hypogonadism (high FSH and LH levels with the patient's own testosterone severely reduced because of the lost tissue). The clinical effect is a trauma-related decline in erectile and drive function, very often resistant to natural restitution (in such cases the patient also frequently becomes irreversibly infertile if there is bilateral necrosis and rupture of the blood-testis barriers, activating the destruction of sperm by the patient's own antibodies) [27], [50].

Another significant injury is post-traumatic damage to the penis itself in mechanical and venous-arterial terms; among the most common consequences of "straddle" falls onto the pelvis (for example, a perineal impact against a horizontal bar after a fall in horizontal-bar gymnastics) is the crushing of the urethral bulb and of the bulbar artery or dorsal penile artery, pressed by sudden traumatic pressure against the edge of the pubic symphysis [52]. This crushing strongly affects the arterial blood supply to the corpora cavernosa. It not infrequently leads to vessel rupture or to damage to the tunica albuginea surrounding the corpora cavernosa (clinically resembling, though through a different injury mechanism, Peyronie's disease). The result is a subcutaneous or intracavernosal scar with the features of dense fibrosis. This scar, composed of entirely inelastic, rigid collagen in place of the healthy, elastic elastin meshwork of the penile sheath, causes a marked veno-occlusive dysfunction. The erection may then be permanently poor in rigidity, with blood continuously draining through escaping, uncompressed veins owing to inadequate compression of the damaged soft tissue during arousal. In addition, the scar on the damaged side pulls on the penis, resulting in a penile curvature that hinders penetration to the point of making it painful and, over time, physically impossible [27], [52]. The post-traumatic situation places the athlete within the framework of an irreversible condition and necessitates urological procedures.

In women who practise extreme and impact sports, although the reproductive organs of the pelvis (the ovaries and uterus) are relatively well protected from blunt abdominal trauma (unlike the male scrotum), the vulva and clitoral structures remain largely exposed to substantial injury. Direct blows — such as an impact in early rowing or during contact in rugby (for example, foot-to-perineum contact) — may produce considerable vulvar hematomas (hematoma of the

vulva) [35], [59]. Ruptures located close to the pubic symphysis damage the fragile vascular branches of the clitoral vestibule and the spongy structures, which, following an inflammatory process, undergo fibrosis [45]. The scarred, previously torn region of the vaginal vestibule shows a markedly reduced capacity to produce tissue fluid during the swelling of the desire phase (reduced lubrication) and, above all, becomes rigid and inelastic on stretching. This forms the basis of a purely mechanical pathology underlying secondary dyspareunia and pain during penetration, which — combined with an underlying, subconscious fear of the objective urogenital pain previously experienced from the injury — may perpetuate a progressively worsening diagnosis of genito-pelvic pain disorder (GPPPD) until it is addressed by interventional physiotherapy or by procedures correcting the marked fibrosis [25], [29]. This illustrates that, in the sports medicine patient, such injuries should be viewed within a comprehensive assessment of how far mechanical vascular ruptures overlooked in youth determine the future exclusion of the reproductive system at the venous-vascular level, depriving the patient of the chance to generate an arousal response naturally [52].

6.3. Strength and Physique Sports (Bodybuilding, Weightlifting): The Biomechanics of Increased Intra-Abdominal Pressure and Pelvic Floor Pathology

Among the etiological factors responsible for the development of sexual dysfunction, the biomechanical characteristics of strength sports (powerlifting, weightlifting, CrossFit) and physique sports (classic and extreme bodybuilding) constitute a pathophysiologically distinctive model of loading. Unlike endurance sports, in which catabolism from energy deficiency (RED-S) and HPG axis collapse predominate, or cycling, characterized by direct ischemic-compressive injury to the pudendal nerve, in disciplines based on lifting very heavy loads the principal factor undermining urogenital and sexual health is the sustained, marked overload of the musculofascial apparatus of the lesser pelvis [16], [37]. From the standpoint of exercise physiology, proper technique for generating maximal force during multi-joint exercises (for example, the barbell squat, deadlift, and snatch) requires firm stabilization of the spine and core. This stabilization is achieved almost exclusively by deliberately performing and maintaining the Valsalva maneuver (a forced exhalation against a closed glottis), which leads to a sudden, substantial rise in intra-abdominal pressure (IAP) [37]. The pelvic floor — composed of the pelvic diaphragm (including the levator ani muscle), the urogenital diaphragm, and the fascial system — forms the anatomical lower closure of the abdominal cylinder. This means that each marked rise in IAP during the lifting of hundreds of kilograms transmits a compressive force vector directly downward, requiring the perineal muscles to generate an equal or greater opposing force to prevent prolapse of the viscera [16], [37].
Chronic exposure to compressive stimuli of this kind induces, in the pelvic floor muscles of competitive athletes of both sexes, an advanced adaptive pathology. Unlike the skeletal muscles of the limbs, hypertrophy and increased tone within the pelvic floor are not a desirable performance trait but rather manifest as a hypertonicity that may threaten reproductive health (hypertonic pelvic floor dysfunction or overactive pelvic floor) [37]. Under conditions of repetitive, non-physiological mechanical stress, these muscles lose their fundamental property — the capacity for full relaxation between contractions. Their resting tone rises markedly, which triggers a structural cascade resulting in constriction of the pelvic neurovascular structures, hypoxia of the perineal muscle cells, and generalized fascial stiffness [16], [37]. From a sexological standpoint, an excessively tense, spastic, and stiffened pelvic floor is biomechanically unable to participate properly in the sexual response cycle, which directly contributes to the occurrence of a range of dysfunctions diagnosed on the basis of the DSM-5-TR and ICD-11 criteria [25], [26].

In the population of elite female strength athletes, the pathological hypertonicity and the stretching of fascial structures resulting from years of contending with high IAP lead to a substantial rise in the prevalence of pelvic floor dysfunction. The findings of systematic reviews indicate that more than 40–50% of female weightlifters experience symptoms of stress urinary incontinence (SUI) or pelvic organ prolapse (POP), even at an early career stage (before pregnancy) [16]. From the standpoint of sexual health, spasticity of the levator ani muscle and of the fibers surrounding the vaginal introitus produces a substantial, organic dyspareunia that prevents painless penetration [16], [56]. Secondarily, the non-physiological pain on intercourse, caused by friction against the perineal muscles "cemented" by tension, promptly induces the aversive conditioning mentioned in previous sections. This leads in the female athlete to the development of a full-blown genito-pelvic pain disorder (GPPPD), in which, alongside the primary mechanical hypertonicity, a psychogenic vaginismus appears (a protective spasm that further closes the vaginal entrance) [16], [25]. Moreover, a stiff pelvic floor does not allow proper blood filling of the vulvar corpora cavernosa during vasodilation (pressure on the local vessels supplying the vulvar plexuses), abolishing the physiological process of lubrication [16], [29]. Such musculofascial structural damage shows a strong tendency to progress in former female strength athletes during the post-career period, making intimate pain and arousal-phase dysfunction a persistent pathology that requires many months of advanced urogynecological physiotherapy to "release" the blocked tension vectors [16], [56].

In men training with very heavy strength loads (whether or not they also rely on pharmacological aids, which in themselves produce considerable hormonal disruption — see Section 7), mechanical pelvic floor hypertonicity manifests as chronic pelvic pain syndrome (CPPS, formerly non-bacterial prostatitis) [37]. The continuously tensed ischiocavernosus and bulbospongiosus muscles, whose rhythmic contraction supports normal ejaculation and the maintenance of erectile rigidity (the rigid phase), become functionally exhausted. In a state of hypertonicity, these muscles compress the trunks and branches of the pudendal nerve running in the soft tissues of the pelvis, resembling the symptoms of the compressive neuropathy described in cyclists [34], [37]. This presents as paroxysmal or chronic pain referred to the perineum, scrotum, and groin, intensifying markedly at the moment of erection. Because of the disturbed relaxation dynamics, these patients may develop significant erectile dysfunction that is resistant to oral medication (PDE5 inhibitors), since the persistently contracted pelvic floor fibers disrupt the proper veno-occlusive mechanism of the penis (the veins draining blood from the corpora cavernosa are not adequately compressed, resulting in so-called venous leak and an immediate loss of the erection) [27], [37].

The clinical decompensation of the sexual domain in patients with an overloaded, hypertonic pelvic floor does not end with arousal-phase disturbances. Spasticity of the perineal muscular system, which executes the orgasmic and ejaculatory reflex, may produce marked disturbances in the experience of climax in athletes of both sexes. In strength athletes, the phenomenon of dysorgasmia (painful orgasm) is frequently documented, in which, instead of a wave of euphoric relaxation, ejaculation is accompanied by an acute, stabbing spasm and burning in the pelvis, undermining the psychological and biological meaning of the erotic reward [25], [37]. The painful conclusion of intercourse, combined with the loss of the capacity for a full, relaxed sexual initiative, creates a striking contrast in a powerfully built, muscular athlete — while his skeleton bears hundreds of kilograms of load, his urogenital system, confined within a fascial armor of hypertonicity arising from biomechanically conditioned excess IAP, deprives him of basic reproductive function and of satisfaction in intimate relationships [25], [37]. Resolving this problem in the sports medicine clinic can be very difficult, since it requires giving up (detraining) the use of the Valsalva maneuver during pelvic "release" therapy (biofeedback, manual urogynecological techniques) — something that most active competitive athletes, in

pursuit of results and at the same time contending with a fear of losing strength and mass (body dysmorphia), are firmly unwilling to accept [13], [37].

7. The Role of Pharmacotherapy and Psychoactive Substances (Including Doping)

7.1. Anabolic-Androgenic Steroids (AAS)

Despite the strict sanctions imposed by the World Anti-Doping Agency (WADA), pharmacological enhancement in competitive sport remains a persistent, structured, and widespread epidemiological problem whose consequences extend well beyond an unfair biomechanical advantage [49], [60]. Anabolic-androgenic steroids (AAS), which are synthetic derivatives of endogenous testosterone, undergo complex biochemical modifications (for example, 17-alpha alkylation or 17-beta esterification) in order to maximize their anabolic potential (muscle tissue hypertrophy) while attempting to minimize androgenic effects [18], [20]. The use of these substances in elite strength and physique disciplines, as well as in contact sports and athletics, most often follows cyclical patterns based on "stacking" (combining several preparations at once) and the "pyramiding" of doses, which not infrequently exceed clinical replacement doses by a factor of 10 to 100 [18], [49]. This substantial, supraphysiological supply of exogenous androgens triggers an immediate and far-reaching response of the hypothalamic-pituitary-gonadal (HPG) axis. Through strong negative feedback, the pituitary fully — and not infrequently within just a few days — halts the production of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which in turn suppresses intratesticular testosterone production and spermatogenesis [18], [38]. This opens the door to marked, biphasic sexual dysfunction — during the phase of active exposure (the on-cycle phase) and during the period of pronounced decline after injections cease (the off-cycle phase) [20], [38].

7.1.1. The Active-Use Phase: Hypersexuality, Erectile Dysfunction (e.g., the "Deca Dick" Phenomenon), and Priapism

In the initial period of the active-use phase (the "on-cycle"), once supraphysiological doses of AAS have reached a peak blood concentration in the patient, the most common behavioral clinical manifestation is a marked, pathological hypersexuality [18], [49]. High concentrations of synthetic androgens readily cross the blood-brain barrier, extensively saturating the androgen receptors (AR) located in the limbic system, the amygdala, and the hypothalamic regions responsible for sexual drive [20], [49]. At the same time, this androgenic stimulus produces a strong activation of the dopaminergic pathways of the reward system, which on the one hand masks any central fatigue (temporarily suppressing the symptoms of, for example, OTS) and on the other gives rise to a persistent, difficult-to-control sexual desire [18], [38]. Users in this phase report an excessive focus on erotic stimuli, an increased frequency of fantasies, and a need for compulsive release of sexual tension. This state not infrequently evolves toward highly impulsive behavior, affective dysregulation, and increased aggression (the so-called "roid rage" phenomenon), which translates directly into a marked deterioration in the dynamics of partner relationships, gives rise to conflict, and, in extreme cases, may favor coercive behavior in the intimate sphere [18], [20], [49].

The paradox in AAS pharmacology is that this neurogenic hypersexuality and heightened desire very often come up against a substantial, chemically conditioned peripheral physical dysfunction. This phenomenon is a treatment-resistant peripheral erectile dysfunction (ED), which in sports medicine slang has acquired the name "Deca dick" — a term derived from the leading preparation in this group of pathologies: nandrolone decanoate (Deca-Durabolin) [20],

[38]. The mechanism of this phenomenon is a notable example of a damaging pharmacological cascade. Nandrolone (19-nortestosterone) and its analogues (for example, trenbolone), on interacting with the enzyme 5-alpha-reductase in penile tissues, are not converted to the potent dihydrotestosterone (DHT) — as physiological testosterone is — but are instead reduced to dihydronandrolone (DHN) [18], [20]. DHN has an extremely low, negligible affinity for the androgen receptors of the corpora cavernosa. The complete absence of local androgenic stimulation in the penis results in a shutdown of nitric oxide synthase (nNOS) expression. As a result, despite the surging dopaminergic drive in the brain, the peripheral vascular apparatus does not produce the key vasodilator (nitric oxide, NO), making the generation of an erection chemically and biomechanically unattainable [18], [20], [38].
The patient's clinical situation is further compounded by hyperprolactinemia, which is specific to 19-nortestosterone derivatives. Compounds in this group (the "19-nors") have a strong affinity for the progesterone receptors in the pituitary, acting as potent progestogens. This progestogenic stimulus triggers an immediate, uncontrolled, excessive secretion of prolactin (PRL) from the anterior lobe [18], [20]. As described in earlier chapters, an abnormally high peripheral prolactin level acts on the hypothalamic system through inhibitory feedback, ultimately removing the self-perpetuating hypersexuality and drawing the athlete into a sudden, complete collapse of libido and into anhedonia [38], [49]. In addition, an elevated prolactin level impairs the peripheral vascular endothelial response, intensifying the peripheral, nNOS-deficiency-based erectile dysfunction. In such a hormonal environment, the combined use of phosphodiesterase type 5 inhibitors (such as sildenafil) is largely ineffective in alleviating the symptoms, which draws the athlete-patient into a state of frustration and secondary depression because of the painful chemical "castration" brought about by their own doping [18], [20], [38].
The opposite of ED in the active phase of androgen use — an extreme and life-threatening one — is the risk of priapism, particularly with highly aromatizable testosterone derivatives (for example, testosterone enanthate or cypionate) [18], [49]. Priapism is defined as a prolonged, painful erection lasting more than 4 hours, unrelated to any sexual desire, arousal, or stimulation; in AAS users, its basis lies in marked drug-related rheological changes in the blood [18], [20]. The supraphysiological presence of androgens in the bloodstream is a powerful pathological stimulator of erythropoiesis in the bone marrow and of renal synthesis of erythropoietin (EPO). After several weeks of injection use, athletes may develop severe secondary polycythemia (erythrocytosis) with a pronounced rise in hematocrit, sometimes reaching values of 55–60% or higher [20], [49]. Such non-physiological, thickened blood shows marked hyperviscosity. During nocturnal physiological erections, or following incidental SNS activation, the thickened erythrocyte mass flows into the corpora cavernosa but, because of its density and slow flow, is immediately subject to stasis within the narrow draining subfascial venous plexuses (venous stasis) [18], [20]. This leads to a sudden closure of the draining vascular system and the development of severe ischemic (low-flow) priapism. This is a urological emergency (an acute surgical condition); the hypoxia of the trapped blood and the intratissue acidosis can, within just 12–24 hours, cause irreversible necrosis of endothelial cells and fibroblasts in the corpora cavernosa, leaving the athlete — in the absence of rescue aspiration and irrigation of the corpora cavernosa with alpha-adrenergic agents — with a complete and permanent loss of the capacity for erection [18], [20], [49].

7.1.2. The Withdrawal Phase: Post-Cessation Hypogonadism (ASIH), Reduced Libido, and Testicular Atrophy

The decision to stop injectable and oral anabolic-androgenic steroids (AAS) — whether prompted by fear of testing by the World Anti-Doping Agency (WADA), by a deterioration in cardiac parameters, or simply by the end of a planned, multi-week "cycle" — gives rise, in the

athlete's body, to one of the most neuroendocrinologically and psychosexually disruptive states in sports medicine [18], [49]. In pathophysiological terms, the abrupt withdrawal of supraphysiological doses of exogenous androgens reveals a fully suppressed and quiescent hypothalamic-pituitary-gonadal (HPG) axis. This state of acute hormonal insufficiency following the removal of the suppressing factor has been carefully defined in evidence-based medicine (EBM) as anabolic steroid-induced hypogonadism, with emphasis on the post-cessation phase (ASIH) [18], [38]. As a result of weeks or months of strong negative feedback, the pituitary gland of a patient with ASIH is in a state of deep "dormancy" — concentrations of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) not infrequently fall to the lower limits of normal or even to analytically undetectable levels [20], [38]. Because the exogenous steroid has by then been entirely eliminated from the bloodstream (in line with its half-life) while the pituitary has not yet resumed LH production, the testes remain without any stimulus for testosterone synthesis, which produces, in the often young athlete, a sudden, chemical state of "castration" (total testosterone concentrations not infrequently falling below 100 ng/dL) [20], [38], [49].

A direct, tangible structural phenomenon accompanying ASIH — and one that itself becomes a substantial source of secondary anxiety and body dysmorphia (MD) for the athlete [13] — is pronounced bilateral testicular atrophy [18], [38]. The physiological volume and mass of the male gonads is determined, by about 80–90%, by the volume of the seminiferous tubules governed by FSH and, to a lesser extent, by the interstitial tissue with LH-dependent Leydig cells. The blockade of both gonadotropins during an AAS cycle means that the testes, deprived of trophic stimulation, shrink markedly — their volume not infrequently falls by 50% or more, taking on a flaccid consistency without natural firmness [18], [49]. What is more, the Leydig cells undergo structural degeneration and a marked desensitization (loss of sensitivity) to any slowly reawakening signals from the pituitary. This means that even if, after several weeks, the hypothalamus and pituitary begin cautiously to resume pulsatile secretion of GnRH and LH, the atrophic, damaged testes may remain unresponsive ("deaf") to this stimulus and unable to produce their own testosterone for many months [20], [38]. This process of coupled apoptosis in the seminiferous tubules also leads to azoospermia (a complete absence of sperm in the ejaculate), undermining the athlete's reproductive potential — not infrequently permanently, in the absence of highly specialized treatment [18], [49].

The clinical decompensation of the sexual domain in the ASIH phase is marked, sudden, and multidimensional. Within just a couple of weeks of stopping doping, the patient descends from the peak of neurogenic hypersexuality (discussed in the previous section) into a state of generalized anhedonia and a complete loss of drive [38], [49]. This decline in libido (amounting to an aversion to intimate contact) is conditioned not only by markedly low concentrations of peripheral testosterone and DHT but also by an equally substantial secondary breakdown of the dopaminergic pathways in the central nervous system [18], [20]. Under the DSM-5-TR criteria, these patients are diagnosed with severe substance/medication-induced sexual dysfunction with desire and erectile symptoms, not infrequently coexisting with a drug-related depressive episode and suicidal tendencies [25], [49]. The complete absence of androgens induces the shift in balance toward cortisol described earlier — the anti-glucocorticoid, blocking effect of testosterone is lost, and the body accordingly enters a state of pronounced catabolism [18]. The patient loses hard-won muscle mass, experiences considerable fatigue, and, with no activation of the nitric oxide (nNOS) pathway in the corpora cavernosa, may develop significant organic erectile dysfunction. Despite the rescue use of PDE5 inhibitors, the endothelial response is negligible without an androgenic basis [27], [38]. This sense of helplessness intensifies the loss of what remains of self-esteem, leading many athletes into isolation, and in clinical studies it is described as one of the most common reasons for relapse into long-term, addictive AAS use (which the ICD-11 also classifies as a steroid dependence syndrome) [26], [49].

A persistent belief in the gym community and in informal coaching advice has long held that ASIH is quickly and spontaneously reversible after so-called post-cycle therapy (PCT), based on the illicit, self-administered use of clomiphene or human chorionic gonadotropin (hCG). Contemporary studies and EBM protocols, however — including widely cited analyses conducted in specialized clinics for former AAS users (Smit and de Ronde, 2018) and the work of Rasmussen et al. (2016) — document a more sobering picture of the chronification of this state [39], [48]. It has been clearly shown that even many years after the complete and documented cessation of doping (confirmed by interviews and a metabolite-free urine profile), former AAS users still show significantly lower concentrations of total and free testosterone, a smaller testicular volume, and higher rates of depression and erectile dysfunction compared with a healthy, age-matched control population [18], [39]. This state not infrequently proves so persistent (lasting damage to the testicular interstitial tissue) that the only procedure capable of restoring a former athlete's capacity to form intimate relationships and to achieve an erection is lifelong medical testosterone replacement therapy (TRT) [38], [48]. Unfortunately, in athletes still in active competition, the introduction of exogenous testosterone as treatment for ASIH meets a firm legal obstacle from WADA (the World Anti-Doping Agency) — obtaining a therapeutic use exemption (TUE) for testosterone in such a case, given an etiology arising from the patient's prior doping violation, is a process that is almost entirely precluded, leaving the athlete in a difficult, months-long bind of physiological incapacity [20], [38], [49].

7.2. The Use of Systemic Medications in Sports Medicine

7.2.1. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) and Analgesics (Opioids) in Relation to HPG Axis Function

Competitive sport is inseparably associated with the generation of chronic pain, repetitive microtrauma, and acute inflammatory states of the musculoskeletal system. This environment promotes the widespread, often routine use of analgesic pharmacotherapy without close endocrinological supervision. In contrast to illicit doping (AAS) or the misuse of recreational substances, licit analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) are widely regarded by athletes themselves, and not infrequently by medical staff, as safe, short-term recovery "aids," used prophylactically even before key competitions. Evidence-based medicine (EBM), however, indicates that this seemingly innocuous pharmacotherapy may exert a substantial iatrogenic, suppressive effect on the hypothalamic-pituitary-gonadal (HPG) axis, becoming a quiet but clinically notable co-factor in the development of significant sexual dysfunction in the sporting elite [3], [61].

In pathophysiological terms, the use of strong opioid analgesics (for example, tramadol, which was not infrequently misused in professional cycling up to 2019, or stronger derivatives used in contact sports after serious orthopedic injuries) is directly associated with a clinical entity defined as opioid-induced androgen deficiency (OPIAD). The pathomechanism of OPIAD is predominantly central in nature and independent of training load. Exogenous opioids have a high, specific affinity for the mu-opioid receptors located in the hypothalamus. Their prolonged activation results in a marked inhibition of kisspeptin secretion and in a direct, chemical silencing of the pulsatile release of gonadotropin-releasing hormone (GnRH) from the neurons of the hypothalamic nuclei, affecting the very top of the reproductive cascade [19], [61].

The blockade of GnRH stimulation leads to a decline in the secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the gonadotropic cells of the anterior pituitary. In male athletes, a fall in LH below the physiological threshold deprives the interstitial Leydig cells of the necessary stimulation, leading to acute secondary hypogonadotropic hypogonadism (analogous in its consequences to severe RED-S). Rigorous clinical studies have

shown that chronic — and sometimes only a few weeks' — use of extended-release opioid analgesics lowers total testosterone to subclinical or castration-range values in close to half of the patients treated. This drug-related androgen deficit markedly impairs the nNOS-dependent nitric oxide (NO) synthesis pathways in the corpora cavernosa, giving rise to stimulation-resistant erectile dysfunction and to a chemically conditioned suppression of drive, classified as HSDD [19], [61].

In elite female athletes, opioid suppression of the HPG axis acts with comparable force, serving as an independent factor that deepens neuroendocrine decline or is superimposed on a pre-existing functional hypothalamic amenorrhea (FHA). The fall in FSH and LH concentrations under opioids disrupts folliculogenesis in the ovaries, prevents the preovulatory follicular surge, and markedly halts the synthesis of endogenous estradiol. The consequence of this pronounced hypoestrogenism is a developing urogenital atrophy, presenting with thinning of the vaginal mucosa, a critical deficiency of physiological lubrication, and, as a result, severe acquired dyspareunia (GPPPD in the DSM-5-TR classification). In addition, in both sexes opioids have a strongly inhibitory effect on the central nervous system and the mesolimbic (dopaminergic) pathways, which, at the cognitive level, further cuts the athlete off from the capacity to experience arousal, initiating a pronounced genital anhedonia and anorgasmia [19], [61].

At the same time, in the shadow of strong opioids, the pathophysiological significance of the misuse of non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and diclofenac not infrequently remains overlooked, even though their daily intake in competitive sport (particularly during periods of accumulated competitions) can reach values that risk toxicity. The mechanism of action of NSAIDs is based on the non-selective or selective inhibition of cyclooxygenase enzymes (COX-1 and COX-2), which results in a systemic blockade of prostaglandin synthesis. From the standpoint of the molecular endocrinology of reproduction, prostaglandins (particularly PGE2) are not merely peripheral mediators of pain and inflammation but also serve as important autocrine and paracrine messengers within the gonads themselves [3], [19]. A physiological PGE2 concentration is essential for the proper activation of the StAR protein (steroidogenic acute regulatory protein) in Leydig cells, which is responsible for the transport of intracellular cholesterol to the mitochondria — the process that initiates the cascade of steroidogenesis and testosterone synthesis [19].

Chronic, daily use of NSAIDs by athletes to mask fatigue and overload-related pain may therefore give rise to a state of so-called compensated hypogonadism. In this state, the testes contend with a peripheral, chemical blockade of testosterone synthesis at the intracellular level. The pituitary, receiving a signal of the deficit, attempts to force production by raising LH levels, but the growing Leydig cell resistance induced by COX-2 blockade ultimately leads to a reduction in free testosterone (fT), which clinically manifests in patients as a deterioration in erectile quality (ED) and a loss of libido. In women, in turn, strong COX-2 inhibition through high doses of NSAIDs may block the rupture of the Graafian follicle (ovulation is, in effect, a prostaglandin-dependent inflammatory process). This can induce luteinized unruptured follicle (LUF) syndrome, considerably disrupting the menstrual cycle, reversing the normal progesterone-to-estrogen ratio, and secondarily entailing desire dysfunction. In this way, a licit, seemingly innocuous, and widely accepted pharmacotherapy may become, in competitive athletes, a substantial iatrogenic source of pronounced psychosexual pathology [3], [19].

7.2.2. Diuretics and Beta-Blockers (e.g., in Shooting Sports) in Relation to Erectile Dysfunction

Among the pharmacological agents used (both licitly, for cardiac indications, and as illicit doping subject to WADA sanctions) in particular sport disciplines, beta-adrenergic receptor antagonists (beta-blockers) and diuretics occupy a special place. Their use in the competitive

elite is highly targeted. Beta-blockers are commonly used in sports requiring great precision — such as sport shooting, archery, biathlon, and golf — where reducing muscle tremor and lowering heart rate confer a competitive advantage [14], [49]. Athletes in these disciplines not infrequently contend with the substantial performance anxiety described earlier, and beta-blockers, by suppressing the autonomic symptoms of competition stress, act as a kind of pharmacological stabilizer [14]. Diuretics, in turn, are a tool in weight-category sports (boxing, MMA, wrestling, weightlifting) used for rapid, non-physiological weight loss (the phenomenon of weight cutting), and also in bodybuilding to achieve the desired, extreme tissue "dryness." These drugs are also widely used as masking agents that accelerate the excretion of anabolic-androgenic steroid (AAS) metabolites before anti-doping testing [13], [20], [49]. From the standpoint of clinical sexology and vascular pathophysiology, both of these drug groups are among the stronger iatrogenic factors contributing to peripheral erectile dysfunction (ED) and to a generalized suppression of desire.

The mechanism by which beta-blockers initiate erectile decline is conditioned by a marked disturbance of the delicate balance between the sympathetic and parasympathetic systems in penile tissues. The blockade of beta-2-adrenergic receptors in the vessels of the corpora cavernosa abolishes their physiological vasodilatory effect. In the absence of this protective stimulation, the activity of the alpha-1-adrenergic receptors becomes uninhibited, and these, in response to the generalized autonomic tone constantly present in athletes, produce a strong constriction of the smooth-muscle vessels [27], [40]. This results in a substantial restriction of arterial blood inflow to the cavernosal system, which from a biomechanical standpoint makes it difficult to achieve adequate penile rigidity. In addition, the highly lipophilic first-generation beta-blockers (for example, propranolol) readily cross the blood-brain barrier. Their accumulation in the central nervous system produces a central sedative effect, lowered mood, disturbances of sleep architecture, and a drug-related quieting of the limbic drive centers, which, under the DSM-5-TR classification, may manifest as a secondary, significant desire disorder (hypoactive sexual desire disorder, HSDD) [25], [40].

The pathomechanism of dysfunction caused by diuretics, in turn, rests on a cascade of hemodynamic and endocrine responses. The marked polyuria forced by diuretics brings the athlete (not infrequently already in a state of dehydration owing to deliberate fluid restriction) to a sudden, iatrogenic hypovolemia. The decline in circulating blood volume lowers arterial pressure, to which the body responds by immediately engaging compensatory mechanisms: a strong reflex activation of the sympathetic system (a release of catecholamines) and activation of the renin-angiotensin-aldosterone (RAA) axis in order to centralize the circulation and maintain perfusion of the brain and the parenchymal organs [40]. This produces a marked peripheral vasoconstriction, including of the pudendal and penile arteries. The ischemic, constricted vessels of the reproductive system become resistant to stimulation by nitric oxide (NO), undermining erotic responsiveness during attempts at intimacy [27], [40].

The scale of the harm caused by diuretics in the field of sexual medicine is further amplified when the biochemical specificity of the individual active substances is taken into account. Thiazide diuretics, well established in the literature as the most common cardiological cause of drug-related ED, directly impair the endothelium's capacity for relaxation by inducing an intracellular zinc deficiency and by modifying the transport of potassium and calcium ions in the smooth-muscle myocytes of the corpora cavernosa [27], [40]. The use of potassium-sparing diuretics such as spironolactone, however, carries even more serious consequences for the hypothalamic-pituitary-gonadal (HPG) axis and the athlete's sexual function. This compound is structurally closely similar to endogenous steroids and has a strong, targeted antiandrogenic action. Spironolactone competitively blocks androgen receptors in target tissues (including the penis and the cerebral centers), inhibits the enzymes responsible for the testicular biosynthesis

of testosterone (particularly 17-alpha-hydroxylase), and at the same time increases its peripheral hepatic clearance [40], [49].
As a result, athletes taking spironolactone in order, for example, to remove subcutaneous water before a physique assessment expose themselves to an acute, pharmacological induction of hypogonadism. Its clinical effect — alongside an erectile collapse and the loss of libido — is often the development of painful gynecomastia, conditioned by a disturbed balance between androgens and estrogens [18], [40]. This pathophysiological combination of pharmacological side effects illustrates clearly that the use of substances manipulating blood pressure and water balance in physique and contact sports (where the patient is already in a chronic caloric deficit diagnosed as RED-S) closes the loop of a self-reinforcing cycle. This may lead to a pronounced, iatrogenic, and not infrequently lasting disintegration of reproductive function, undermining the patient's psychological and somatic sexual profile [5], [25], [49].

7.3. Interactions Between Psychiatric Medications (SSRIs, SNRIs) Prescribed to Athletes and Their Sexual Function (the PSSD Phenomenon in the Sporting Context)

The increased awareness of mental health in the sporting elite, although it represents an important advance in supportive medicine (confirmed, among other things, by the consensus of the International Olympic Committee), has been accompanied by a marked rise in the prescribing of psychotropic medications in this population [3], [4]. In the face of rising prevalence rates of severe depression, anxiety disorders, and obsessive-compulsive disorders (including the body dysmorphia described earlier), psychiatrists and sports medicine physicians most often turn to selective serotonin reuptake inhibitors (SSRIs) and serotonin-noradrenaline reuptake inhibitors (SNRIs) [4], [11]. This choice is dictated by the negligible addictive potential of these drugs, their relative cardiac safety, and the fact that they do not appear on the World Anti-Doping Agency (WADA) list of prohibited substances and methods. From the standpoint of clinical sexology, however, the use of SSRI/SNRI antidepressants is associated with a substantial iatrogenic interference with the neurochemistry of the patient's sexual response cycle. In many cases, this pharmacotherapy produces side effects of an intensity that may exceed the initial symptoms of the affective disorder itself, undermining the quality of the athlete's intimate life [3], [30].
The pathomechanism of SSRI/SNRI-induced sexual dysfunction is based on a substantial modification of serotonergic (5-HT) transmission in the central and peripheral nervous systems. According to the neurobiological model of drive regulation, serotonin — unlike dopamine and noradrenaline, which have a promoting (stimulating) effect on the reproductive system — serves as the principal neurotransmitter inhibiting sexual behavior [30]. By blocking the serotonin transporter (SERT), SSRIs produce a rapid rise in serotonin concentration in the synaptic cleft. The excess of this neurotransmitter leads to strong activation of the postsynaptic 5-HT2A and 5-HT2C receptors, which are densely located in the hypothalamus, the nucleus accumbens, and the mesolimbic system. Hyperstimulation of these receptors exerts a direct, descending suppressive effect on the release of dopamine (DA) in the reward centers, which manifests clinically in the athlete as a sudden, chemically conditioned suppression of libido (diagnosed in the DSM-5-TR as substance/medication-induced sexual dysfunction) [25], [30]. In addition, at the spinal cord level, serotonergic pathways descending from the raphe nuclei have an inhibitory effect on the parasympathetic centers in the sacral segment, cutting off the efferent signal to the pudendal nerve. This causes, in patients, significant erectile dysfunction (ED) resistant to targeted pharmacotherapy, a loss of the capacity for vasodilation and lubrication in women, and — above all, and characteristically of SSRI therapy — markedly delayed ejaculation, complete anorgasmia, and genital anhedonia during attempts at intercourse [25], [30]. For an elite athlete, whose identity and daily functioning rest on precise control and

on a high responsiveness of their own body, this sudden drug-related "castration" and the lack of a genital response to stimuli are not infrequently a source of considerable relational stress and a decline in motivation [3], [4].

By far the most serious — and the least understood in conventional sports medicine — phenomenon is the risk of PSSD (post-SSRI sexual dysfunction) in athletes, a syndrome of persistent sexual dysfunction after the discontinuation of serotonin reuptake inhibitor therapy described in the most recent EBM literature [30]. For decades, psychiatry assumed that the resolution of adverse symptoms occurs in close correlation with the drug's half-life (its elimination from the bloodstream). The PSSD syndrome substantially revises this view, defining a state in which, despite appropriate discontinuation of the medication, the symptoms of dysfunction persist for months or years and, in extreme clinical cases, prove lasting and irreversible [26], [30]. The PSSD phenotype in an athlete encompasses generalized anhedonia, a collapse of desire, and what patients describe as "genital anesthesia." An athlete affected by PSSD loses the capacity to feel physical touch on the skin of the penis or clitoris, experiencing a sensation of "dead wood" or of the "perineum being cut off from the brain." This phenomenon, combined with the absence of orgasm, may lead to serious psychological crises. Pathophysiological hypotheses for PSSD in the population (including in young, fit athletes) point to lasting drug-related epigenetic modifications (methylation of the genes encoding, among others, the 5-HT1A and 5-HT2C receptors and the enzyme 5-alpha-reductase), as well as a potential structural disruption of the small nerve fibers (small-fiber neuropathy in the vulva) through a secondary inflammatory cascade [30].

The context of elite sport means that PSSD may affect, with compounded force, a patient already contending with considerable pressure. An athlete who, in good faith, undergoes treatment for severe depression related, for example, to OTS or to performance pressure may face a not infrequently lasting impairment of the intimate sphere, which is itself a substantial secondary trigger for a relapse of significant depression and even of suicidal ideation [3], [4], [30]. In sports medicine, this phenomenon is markedly underestimated (underreporting). Athletes often do not report a lack of erection while on medication to club physicians or psychiatrists, being ashamed to attribute it to psychiatric symptoms or concealing the problem for fear of being removed from competition. Sports physicians not infrequently avoid this uncomfortable subject during the history-taking (an absence of follow-up questions about sexual health after the introduction of SSRIs) [3]. Resolving this impasse calls for a change in the therapeutic paradigm. In line with EBM, in competitive athletes non-clinical and psychotherapeutic interventions should be used first, such as recovery measures and cognitive-behavioral therapy (CBT) [1], [41], and, if pharmacotherapy is necessary, consideration should be given to newer-generation drugs or those with an atypical mechanism of action (for example, bupropion, moclobemide, agomelatine, vortioxetine), which have a negligible — or even a favorable — effect on sexual behavior, thereby protecting not only the athlete's mental sharpness before competition but also their fragile, performance-burdened reproductive health [3], [30].

8. Comparative Analysis and Profiling of Dysfunction by Sport Characteristics

8.1. Endurance Sports (Marathon, Triathlon): The Predominance of Neuroendocrine Factors (Fatigue, Catabolism)

A comparative analysis of pathophysiological profiles in sports medicine indicates that endurance sports — which include marathon and ultramarathon running, long-distance road cycling (setting aside the strictly traumatic saddle factor), cross-country skiing, and triathlon — generate a distinctive, highly specific environment of allostatic load. Unlike contact or physique

sports, where direct mechanical injury and pharmacological iatrogenesis, respectively, play the key role, in endurance sports the predominant etiological factor in sexual dysfunction appears to be a neuroendocrine decline brought about by chronic, very high exercise energy expenditure (EEE) [5], [32]. The bodies of long-distance athletes function at the limits of human adaptive capacity of the cardiorespiratory system and of cellular metabolism, which predisposes this population to a high risk of developing relative energy deficiency in sport (RED-S) and the neuroendocrine overtraining syndrome (OTS) [7], [23]. In this context, disturbances in the psychosexual domain are not an isolated organ defect but rather an evolutionarily conditioned, systemic survival strategy (the phenomenon of energy triage), in which the prioritization of the metabolic pathways needed to sustain locomotion and basic vital functions occurs at the direct expense of energy-demanding reproductive functions [10], [33].

A central mechanism driving sexual dysfunction in endurance athletes is a marked conflict between the neurohormonal axes: the hypothalamic-pituitary-adrenal (HPA) and the hypothalamic-pituitary-gonadal (HPG). The intense, multi-hour aerobic exercise that constitutes the daily training routine of triathletes and marathon runners triggers a strong, chronic activation of the stress (HPA) axis, resulting in chronic hypercortisolism and elevated concentrations of corticotropin-releasing hormone (CRH) and endogenous catecholamines [23], [33]. This phenomenon, coupled inseparably with a chronic reduction in muscle glycogen reserves and very low energy availability (LEA), leads to a pronounced fall in peripheral leptin concentration and, consequently, to a silencing of the stimulating kisspeptin neurons in the hypothalamus [7], [43]. The consequence of this dual impact (the biochemical catabolic stress of OTS and the intracellular "hunger" of RED-S) is a substantial decompensation of the pulsatile secretion of gonadotropin-releasing hormone (GnRH), which leads to a cascading quiescence of the anterior pituitary and the blocking of peripheral sex hormone synthesis in the gonads of both sexes [10], [23].

In the population of elite men in endurance sports, this central neuroendocrine decline manifests clinically as exercise-induced hypogonadotropic hypogonadism (EIHH). EBM studies indicate that, in marathon runners at the peak of the training macrocycle, a substantial reduction in the amplitude of luteinizing hormone (LH) release occurs, which translates directly into a marked fall in total and free testosterone (T) in the circulation, correlated approximately linearly with the duration and years of intensive training involvement [9], [42], [58]. The predominance of catabolic processes, measured by an abnormal testosterone-to-cortisol (T/C) ratio, leads to a reduced expression of nitric oxide synthase (nNOS) in the corpora cavernosa, which physiologically hinders the achievement of a full, sustained vasodilation of the penile vessels [27], [50]. The clinical result of the EIHH mechanism is the development of persistent erectile dysfunction (ED), which at the cognitive level — owing to a deficit of androgenic and dopaminergic stimulation in the central nervous system — is consistently accompanied by generalized, disabling fatigue, the loss of morning erections, and a severe hypoactive sexual desire disorder (HSDD) [25], [51].

The endurance profile has comparably damaging effects in elite female athletes, in whom the reduced frequency of GnRH pulses leads to the development of functional hypothalamic amenorrhea (FHA). Women competing in ultramarathons, biathlon, and long-distance rowing — owing to the blockade of the ovulatory mechanism (the absence of an LH surge and the inhibition of ovarian folliculogenesis) — function in a non-physiological, suppressive environment of pronounced hypoestrogenism [10], [28], [43]. As shown in key studies, the absence of the peripheral trophic action of estradiol gives rise, in these biologically young patients, to structural changes resembling the urogenital atrophy commonly observed in the postmenopausal age in the general population [24], [57]. The thin, intracellular-glycogen-deprived, inelastic vaginal epithelium loses much of its capacity for vasodilation during the arousal phase, which results in a substantial deficiency of physiological lubrication and acute

mechanical pain (dyspareunia) during intercourse [28], [29]. Painful penetration may promptly initiate a secondary, protective tensing of the striated perineal muscles and a strong fear of intercourse, which leads to the entrenchment of a full-blown genito-pelvic pain disorder (GPPPD) and the fading of libido [10], [25].

In summary of this etiological profiling, it should be emphasized that sexual dysfunction in endurance sports represents a notably clear, functional (non-anatomical) model of adaptive neuroendocrine pathology. Unlike irreversible peripheral vascular injuries (such as pudendal nerve fibrosis in cyclists [34], [52]) or the hazardous post-cessation hypogonadism (ASIH) caused by steroid-induced damage to Leydig cells in strength sports [18], [38], the pathomechanism in marathon runners and triathletes is a process of metabolic "cutting-off of the supply" to the HPG axis by an overloaded hypothalamus [7], [21]. This predominance of central fatigue and catabolism means that, in this particular patient subpopulation, the restoration of sexual function and the treatment of dysfunction should rest not on hasty rescue pharmacotherapy (which gives rise to conflicts with WADA) but on a thorough, EBM-based reversal of the LEA and OTS factors: optimizing carbohydrate and fat intake, the interventional de-escalation of aerobic load (detraining), and the reduction of psychological distress [5], [23], [50].

8.2. Strength and Physique Sports: The Predominance of Iatrogenic (Doping) and Psychogenic (Body Image) Factors

A comparative analysis of pathophysiological profiles in sports medicine reveals a marked etiological contrast between disciplines oriented toward aerobic capacity and the environment of strength sports (weightlifting, powerlifting, CrossFit) and physique sports (classic bodybuilding, physique fitness). Whereas in endurance sports the pathology of the hypothalamic-pituitary-gonadal (HPG) axis arises from the body's evolutionary neuroendocrine adaptation to a severe energy deficit (the phenomenon defined as RED-S) and central fatigue [5], [32], in disciplines based on maximal muscular hypertrophy and the generation of maximal force these factors give way to pathologies of a distinctly intentional nature. According to evidence-based medicine (EBM) data, in this particular population the predominant factors in the architecture of sexual dysfunction are iatrogenic factors (arising from the widespread, almost ritualized misuse of psychoactive substances and doping pharmacology) and psychogenic factors closely focused on clinical disturbances in the perception of one's own body [13], [18], [20]. This distinctive coupling of toxicology and psychopathology creates an environment in which the patient's reproductive organs may become a direct casualty of the pursuit of non-physiological and naturally unattainable aesthetic and strength standards [25], [49].

A key damaging iatrogenic factor defining this sporting profile is the widespread, supraphysiological use of anabolic-androgenic steroids (AAS). The epidemiology of AAS use, particularly in physique sports, has taken on the form of a widespread global phenomenon, in which polypharmacy (stacking — the simultaneous use of several injectable and oral preparations) and the cycling of doses substantially disrupt the athlete's physiological endocrine homeostasis [49], [60]. The aggressive stimulation of androgen receptors (AR) by synthetic derivatives of testosterone and nortestosterone initiates an immediate, marked quiescence — resistant to rapid restitution — of hypothalamic GnRH secretion and pituitary gonadotropin release (LH and FSH). This results in a chemical paralysis of the HPG axis which, unlike the functional decline in marathons (EIHH), may lead to secondary, structured necrosis and apoptosis of the testicular interstitial tissue and to atrophy of the seminiferous tubules [18], [20], [38].

The clinical specificity of this profile is apparent in a marked, biphasic dynamic of sexual dysfunction. During a doping cycle (the active-use phase), patients not infrequently contend with the paradox of neurogenic hypersexuality coexisting with peripheral impotence. The elevated, non-physiological androgen level activates the dopaminergic reward pathways, intensifying libido, but the concurrent use of strongly progestogenic derivatives (such as nandrolone) gives rise to hyperprolactinemia. An excess of prolactin, combined with the absence of intracellular conversion to DHT in the penis, leads to the urological phenomenon known as "Deca dick" — a loss of the capacity for nitric oxide synthesis (nNOS) and for generating an erection, producing significant erectile dysfunction (ED) that is largely resistant to PDE5 inhibitors and that falls, under the DSM-5-TR classification, within the criteria for substance-induced dysfunction [18], [25], [38]. Moreover, in the strength-sport environment, during the pre-competition phase (the so-called cutting phase), athletes commonly add loop and potassium-sparing diuretics to their pharmacotherapy in order to achieve extreme muscular definition. Spironolactone, an androgen receptor antagonist, additionally and chemically "castrates" the athlete, deepening the hypovolemic vasoconstriction of the pelvic vessels and removing what remains of erectile capacity [20], [40], [49].

The most serious and definitive element of the iatrogenic profile in physique sports, however, is the post-cessation phase. Stopping AAS reveals a severely impaired reproductive system, manifesting clinically as anabolic steroid-induced hypogonadism (ASIH) [38], [48]. During this period, athletes present with marked testicular atrophy, complete azoospermia, and a pronounced reduction in endogenous testosterone, reaching castration-range values. The extent of this damage is such that patients may fall into severe genital anhedonia and a generalized hypoactive sexual desire disorder (HSDD) lasting many years after the end of their career [39], [49]. Clinical studies indicate that a return to a eugonadal axis in this population is uncommon, and that many patients require lifelong medical testosterone replacement therapy (TRT) to restore the basic capacity to initiate an erection and to improve a markedly low mood — something to which athletes in purely aerobic disciplines are not exposed to the same degree, since in their case the damage mechanism rests, to a greater extent, on a reversible caloric factor [27], [39], [48].

In parallel, an inseparable and no less damaging determinant of dysfunction in bodybuilding and physique sports is a distinctly psychogenic component, structured around serious psychiatric disorders of the obsessive-compulsive group and eating disorders [25], [53]. Unlike team sports, where the emphasis is on motor performance, in physique disciplines the ultimate object of the judges' assessment is the bare morphology of the body alone. This environment strongly predisposes to the development of muscle dysmorphia (MD, also termed bigorexia). The cognitive distortion in which a heavily muscled athlete perceives himself as "puny" and unattractive sets in motion substantial anxiety mechanisms that may directly undermine psychosexual function [13], [25].

From a clinical standpoint, MD gives rise to the pathological phenomenon of fear of exposure (body concealment). An athlete preoccupied with rumination about the imperfections of his hypertrophy or an insufficiently low level of body fat experiences, during intimate contact with a partner, cognitive dissociation and the "spectator role" (spectatoring). This pronounced anticipatory stress stimulates a chronic sympathetic tone, which through a neurogenic route may lead to an immediate loss of erection (ED) or secondary anorgasmia, even if at that moment the athlete's endocrine parameters remain within normal limits [13], [25], [26]. In summary, the dysfunction profile in strength and physique sports embodies a pathophysiological convergence of two of the most damaging forces in sports medicine: the intentional, iatrogenic destruction of the gonadal axis through doping (ASIH) and a disabling, psychiatric aversion to one's own physicality. This dual, integrated mechanism makes sexological treatment in this group very difficult, requiring concurrent intervention with intensive psychotherapy (CBT targeted at

body-image disturbances) and highly specialized endocrinological and urological rescue pharmacotherapy [13], [20], [48].

8.3. Aesthetic Sports (Gymnastics, Figure Skating): The Predominance of Phenomena Related to RED-S and Eating Disorders

A comparative analysis of the etiology of sexual dysfunction in the competitive environment requires that a highly specific subpopulation be distinguished: the aesthetic disciplines (including artistic and rhythmic gymnastics, figure skating, synchronized swimming, and diving). This profile is characterized by a distinctive biomechanical and regulatory context, in which the subjective assessment of judges inseparably combines technical parameters with a strict requirement to maintain a particular, idealized, and not infrequently extremely lean body morphology [5], [11]. In this environment, a "prepubertal" phenotype has for decades been systemically promoted — characterized by a narrow pelvis, an absence of developed secondary sexual characteristics (breasts, the physiological adipose tissue of the hips), and a very low BMI — which is held to facilitate aerial rotations and acrobatic maneuvers biomechanically and to meet unrealistic visual criteria [10], [53]. This pathological optimization of physique compels young athletes to undergo severe caloric restriction during key developmental windows, which makes aesthetic sports a notable reservoir of phenomena that fall within the framework of relative energy deficiency in sport (RED-S) [7], [28], [47].

The pathophysiological basis undermining reproductive and sexual health in this subpopulation is a chronic, pronounced low energy availability (LEA), which induces a structural decline of the hypothalamic-pituitary-gonadal (HPG) axis. Unlike in adult marathon runners, in aesthetic sports the energy deficit most often occurs during the critical peripubertal period. The blockade of pulsatile GnRH secretion in the hypothalamus — conditioned by a neuroendocrine fall in leptin from adipose tissue and a concurrent rise in cortisol — leads to delayed sexual maturation (delayed menarche) or gives rise to a pronounced secondary functional hypothalamic amenorrhea (FHA) [10], [43], [57]. In female athletes with FHA, the ovaries, deprived of the necessary stimulation from luteinizing hormone (LH) and follicle-stimulating hormone (FSH), cease folliculogenesis and the production of estradiol and progesterone. This pronounced hypoestrogenism, imposed by the training and dietary regimen, is a state comparable to a chemical menopause in young women, which generates, in the periphery, a cascade of irreversible or highly persistent atrophic changes throughout the genitourinary system (genitourinary syndrome of menopause, GSM) [10], [24], [28].

The clinical consequences of this chronic hypoestrogenism for sexual functioning in adult life are considerable. Urogenital atrophy presents with a critical thinning of the multilayered squamous epithelium of the vagina, a loss of intracellular glycogen, and a marked decline in the elasticity of the vulvar connective tissue [24], [29]. During attempts at sexual initiation, these organs show a receptor-conditioned inability to generate the physiological vasodilatory vasocongestion (the response to an erotic stimulus) and the necessary transudative lubrication [28], [29]. This creates a considerable mechanical barrier, resulting in severe organic dyspareunia (a tearing pain on penetration), not infrequently accompanied by microtrauma and mucosal bleeding. According to the criteria set out in the DSM-5-TR and ICD-11, this recurrent urogenital pain — initially conditioned by the deficit in the course of RED-S — may develop rapidly, through the mechanism of aversive conditioning, into a full-blown genito-pelvic pain/penetration disorder (GPPPD), which comes to be accompanied by a secondary, psychogenic vaginismus that prevents satisfying intercourse [17], [25].

It should be emphasized that the disruption of the hormonal axis in aesthetic sports is inseparably and bidirectionally coupled with the most serious psychopathology among all disciplines. These sports show the highest prevalence of clinical eating disorders, in particular

anorexia nervosa and bulimia, which in many cases are fostered and normalized for years by authoritarian coaching staff who impose a regimen of weighing and weight-related stigma on athletes [1], [47], [53]. A disturbed body image, body dysmorphia, a disabling fear of losing the idealized "form," and pathological perfectionism may undermine the patient's capacity for any self-acceptance. A mind in a state of continual conflict with its own physiology undergoes dissociation; the body is treated solely as a sporting instrument subject to external evaluation, and its natural needs, including sexual drive, are firmly repressed and viewed with disgust or guilt [3], [25]. This cognitive aversion, compounded by a generalized anhedonia secondary to central nervous system cachexia, largely suppresses the dopaminergic pathways responsible for desire, leading to a pronounced hypoactive sexual desire disorder (HSDD) resistant to standard single-track psychotherapy [25], [41], [50].

In summary of this particular clinical profile, aesthetic sports may represent one of the most unfavorable biopsychosocial environments for the development of healthy physiological and psychological sexuality. Unlike endurance sports (where the deficit arises mainly from energy expenditure) or strength sports (dominated by the pharmacological iatrogenesis of ASIH), gymnastics and figure skating form a damaging continuum. The requirement to maintain a prepubertal, lean physique (resulting in FHA and RED-S) is here inseparably combined with psychological pressure, coaching coercion (often falling within the scope of safeguarding failures), and severe anorexia [3], [5], [10]. Athletes leaving these disciplines very often enter adulthood with a twofold burden: a neuroendocrinologically impaired, atrophic reproductive system that is physically unable to respond to an erotic stimulus, and a deeply traumatized psyche that rejects its own physicality, which makes the clinical rescue treatment of these patients in sexology and endocrinology clinics a particularly demanding challenge [11], [17], [28].

8.4. Overall Comparison: The Competitive Athlete Versus the Recreational Athlete Versus the General Population (A Statistical Analysis of the Identified Differences)

Consideration of the influence of physical activity on human sexual and reproductive health in the broad sense requires moving away from simplified, linear correlational models. According to the findings of evidence-based medicine (EBM), the dose-response relationship between exercise volume and intensity, on the one hand, and the function of the hypothalamic-pituitary-gonadal (HPG) axis and the pelvic vascular apparatus, on the other, takes the shape of a classic hormetic curve — an inverted U or J. This pathophysiological paradigm indicates that the extreme poles of activity (both marked sedentary behavior and very high elite-level exercise) are associated with markedly elevated rates of sexual dysfunction, whereas the physiological optimum lies in the zone of moderate load [6], [9], [54]. A detailed statistical and epidemiological analysis of the differences between the general population, recreational athletes, and competitive professionals reveals quite different — indeed opposing — etiological pathways that, in clinical terms, lead patients to the same diagnoses set out in the DSM-5-TR and ICD-11 classifications [9], [25], [26].

In the general population (physically inactive or with a sedentary lifestyle), the pathogenesis of sexual disorders is dominated by cardiometabolic and lifestyle-related factors. According to cross-sectional epidemiological studies, more than 40–50% of men over the age of 40 in this cohort experience erectile dysfunction (ED) of varying severity (against a global ED prevalence of 3–76.5%), which is a predictor and marker of generalized endothelial disease (endothelial dysfunction) and generalized atherosclerosis [2], [27]. A lack of physical activity favors the development of visceral obesity and insulin resistance, which sets in motion a pathological cascade: an excess of adipose tissue, rich in the enzyme aromatase, leads to excessive peripheral conversion of endogenous testosterone to estradiol (E2). The secondary hypogonadism that

arises in this way, combined with a nitric oxide (NO) deficit of atherosclerotic origin and chronic inflammation (hypercytokinemia), substantially impairs the vasodilation mechanism [27]. In women in this group, obesity and insulin resistance often correlate with polycystic ovary syndrome (PCOS) and hyperandrogenism, which leads to ovulatory disturbances and a reduced sexual satisfaction score, secondary to obesity and dyspareunia of cardiometabolic origin [2], [28].

A quite different, largely protective clinical picture is presented by the cohort of recreational athletes, defined as individuals undertaking regular aerobic and resistance exercise of moderate volume (usually 3–5 training sessions per week, without competition pressure or extreme caloric restriction). This group represents the aforementioned "middle ground" on the hormetic curve, showing the lowest prevalence of sexual dysfunction in population studies [6], [54]. Moderate physical activity stimulates a marked upregulation of endothelial and neuronal nitric oxide synthase (eNOS and nNOS), optimizing the vascular architecture of the pelvic organs and supporting a reliable erectile mechanism in men and physiological lubrication in women [27], [50]. Moreover, the absence of a caloric deficit (no RED-S), combined with the regular release of endorphins and a reduction in cortisol, stabilizes the HPG and HPA axes, which translates into the highest free testosterone levels, naturally high desire (libido), and a markedly reduced risk of the generalized anxiety or depressive disorders that act as co-factors of dysfunction [3], [6], [50].

The transition into elite competitive sport reveals, in the light of the scientific evidence, one of the more striking paradoxes of contemporary medicine: a patient who embodies peak motor performance (capable of breaking a world record) is, from an endocrinological and sexological standpoint, a substantially unwell patient, functioning in a state of extreme allostatic load [5], [9], [21]. Epidemiological analysis indicates that in elite athletes the prevalence of reproductive dysfunction not infrequently matches or even exceeds the rates in the obese general population, but that it arises from quite opposite mechanisms [10], [42]. Instead of atherosclerosis and excessive aromatization, men experience a central quiescence of the axis (EIHH related to OTS overtraining and LEA) or testicular damage from doping (ASIH). The statistics are sobering: depending on the competitive group studied, 15% to as much as 45% of marathon runners and high-load cyclists show testosterone concentrations characteristic of aging men with generalized LOH (late-onset hypogonadism), accompanied by severe anhedonia, an absence of morning erections, and PDE5-resistant erectile dysfunction arising from neuroendocrine decline and nerve compression [9], [34], [38], [42].

In women, the statistical differences between the general population and competitive sport are even more striking and confirm the pathological cost of aesthetic and endurance sport. Whereas in the general population of women of reproductive age the frequency of non-physiological primary or secondary amenorrhea (outside pregnancy and lactation) is estimated at only 2% to 5%, in the population of elite ballet dancers, gymnasts, and long-distance runners the rate of functional hypothalamic amenorrhea (FHA) rises to between 40% and a striking 60–65% [10], [24], [28]. This severe hypoestrogenism, imposed by energy deficiency (RED-S), is responsible for the markedly high frequency (reaching 30–40% in surveys) of reported atrophic dyspareunia resistant to moisturizers (GPPPD), which in the general population occurs physiologically only in postmenopausal patients in the sixth decade of life [25], [28], [29].

In conclusion of this overall analysis, the competitive-sport environment should be divested of the myth that it is synonymous with public health. Whereas recreational sport functions as a salutogenic factor (promoting health, potency, and fertility relative to a sedentary population), elite competition appears to be an environment that is distinctly pathogenic for the HPG axis and the patient's sexual function. The statistical indicators clearly characterize the competitive athlete as an individual exposed to serious iatrogenic complications (AAS, NSAIDs, SSRIs), disabling psychogenic factors (body dysmorphia, performance anxiety), and a central

hypothalamic decline arising from intracellular "hunger" (RED-S) [5], [13], [30], [38]. These differences point to a need for the academic community and sports medicine physicians to adopt a change in paradigm: the competitive athlete is not an embodiment of general-population health but a highly vulnerable patient who requires comprehensive, multi-track endocrinological, psychiatric, and sexological support in order to protect their intimate life from the harm done by the pursuit of a medal [1], [3], [21].

9. Prevention, Diagnosis, and Therapeutic Strategies in Sports Medicine

9.1. A Comprehensive Diagnostic Protocol (Sexual-Health History, Hormonal Tests, Imaging)

The implementation of effective therapeutic strategies for psychosexual and reproductive disorders in competitive athletes calls for a move away from a reductionist organ-based model toward an integrated, highly specialized, and multidisciplinary diagnostic approach. According to evidence-based medicine (EBM) guidelines, including the consensus statements of the International Olympic Committee (IOC), the athlete-patient represents a distinctive biopsychosocial clinical challenge in which the etiology of dysfunction is most often multifactorial, combining energy deficiency (RED-S), pharmacological iatrogenesis, biomechanical damage, and a considerable psychiatric burden [3], [10], [21]. A comprehensive diagnostic protocol in this particular subpopulation should therefore rest on a precise, algorithmic triangulation of data: a careful clinical history that overcomes the barrier of taboo and stigma; broad neuroendocrine profiling of the hypothalamic-pituitary-gonadal (HPG) axis; and targeted radiological and ultrasonographic imaging that allows reliable differentiation of functional from organic pathology [3], [10], [21].

The foundation and starting point of the entire diagnostic process is a structured medical and sexual-health history, which in elite sport encounters a considerable barrier in the form of underreporting — the deliberate concealment of symptoms by athletes for fear of being excluded from competition or of admitting to doping [3]. The sports medicine physician or sexologist should make routine use of psychometrically validated screening tools, such as the International Index of Erectile Function (IIEF-15) for men and the Female Sexual Function Index (FSFI) for women, in order to quantify objectively any decline in libido, disturbances of the arousal phase (vasodilation and lubrication), and dysorgasmia. An integral part of the history is a thorough, non-judgmental collection of information about the pharmacotherapy history — with particular attention to the use (including in the past) of anabolic-androgenic steroids (AAS), selective serotonin reuptake inhibitors (SSRIs), strong analgesics, and restrictive weight-cutting procedures involving diuretics [3], [38], [49]. In addition, the history should check for the presence of eating disorders (using, for example, the EAT-26 questionnaire), the occurrence of performance anxiety, and an estimate of energy availability using RED-S-targeted tools such as the LEAF-Q (Low Energy Availability in Females Questionnaire), with consideration of analogous indicators for men [3], [49].

Laboratory diagnostics constitute the analytical core that allows the patient's dysfunction to be classified. In the male population, where the history points to significant erectile dysfunction (ED) and anhedonia (HSDD), morning (between 7:00 and 10:00) measurement of total testosterone, sex hormone-binding globulin (SHBG, for the calculation of free testosterone), and the gonadotropins (LH and FSH) is essential. Such a panel allows a precise distinction between exercise-induced hypogonadotropic hypogonadism (EIHH — low LH and T related to RED-S/OTS), post-cessation hypogonadism (ASIH — markedly low LH, FSH, and T in former AAS users), and post-traumatic testicular damage (elevated LH and FSH with low T) [9], [18], [50]. This panel should be extended to include measurement of prolactin (to exclude drug-

related hyperprolactinemia after nandrolone derivatives), estradiol (to assess aromatization in athletes with, for example, obesity and doping), and parameters of the stress and metabolic axes: morning cortisol, a thyroid panel (a fall in free triiodothyronine, fT3, is a useful biomarker of a marked caloric deficit and LEA), and insulin-like growth factor 1 (IGF-1), which is suppressed in catabolic states [9], [18], [50].

In female athletes reporting dyspareunia (GPPPD), an absence of lubrication conditioned by an atrophic epithelium, and a loss of drive, the endocrine panel is directed at recognizing and differentiating functional hypothalamic amenorrhea (FHA), a manifestation of RED-S. This diagnosis, made by exclusion, requires measurement of estradiol, LH, FSH, prolactin, and TSH in order to exclude, respectively, premature ovarian insufficiency (POI — characterized by elevated FSH), pituitary adenomas (hyperprolactinemia), and thyroid pathology [10], [28], [43]. In addition, measurement of free testosterone and androstenedione is important in order to distinguish FHA from polycystic ovary syndrome (PCOS), which can also occur in female athletes and cause oligomenorrhea. For precise monitoring of the recovery of HPG axis function in patients with FHA, laboratory medicine also uses measurement of anti-Müllerian hormone (AMH) concentrations as an indicator of ovarian reserve, and of peripheral leptin concentrations, whose marked fall is the primary factor silencing the kisspeptin neurons in the hypothalamus and blocking the menstrual cycle and the desire phase [10], [28], [43].

A third, important pillar of the diagnostic protocol is the use of advanced imaging techniques, which in sports medicine serve a dual function: verifying complications of the HPG axis and excluding direct mechanical injury. The gold standard for assessing the effect of chronic hypoestrogenism in women (FHA) and of severe hypogonadotropic hypogonadism in men (EIHH, ASIH) is dual-energy X-ray absorptiometry (DXA). A bone mineral density (BMD) Z-score below -1.0 in a young, elite athlete is an objective and concerning indicator of the entrenched nature of the endocrine decline and of a months-long absence of sex hormones [10], [15], [36]. In athletes in combat and contact sports (for example, boxing, American football) who report significant sexual dysfunction with neurological symptoms and exhaustion, alongside a laboratory decline in testosterone and growth hormone, contrast-enhanced magnetic resonance imaging (MRI) of the sella turcica region is strongly recommended. This examination is necessary to exclude a pituitary micro- or macroadenoma and to confirm radiologically post-traumatic hypopituitarism (PTHP) caused by damage to the infundibular stalk after acceleration/deceleration microtrauma [10], [15], [36].

Organ imaging finds its clearest application above all in excluding anatomical and vascular factors in dysfunction. In competitive cyclists and equestrians with symptoms of genital paresthesia and ED (Alcock's canal syndrome), penile Doppler ultrasound is important for assessing arterial flow (the peak systolic velocity of the cavernosal arteries) before and after the administration of a vasodilatory agent (the prostaglandin E1 injection test), which allows psychogenic ED to be distinguished from post-traumatic obstruction of the pudendal arteries conditioned by a poor saddle position [34], [35], [52]. Where pudendal neuropathy is suspected, imaging is extended to include a nerve conduction study (electromyography, EMG, of the perineal muscles) confirming denervation of the striated pelvic apparatus [34], [38]. Testicular ultrasound, in turn, is necessary in patients with a doping history (showing atrophy and microcalcifications) and after blunt injury in combat sports, while transvaginal ultrasound (TVS) in female athletes with FHA reveals a characteristically reduced ovarian volume and a critically thin (atrophic) endometrium, providing morphological evidence of the absence of trophic estradiol stimulation underlying the reported mechanical dyspareunia [34], [35], [38], [52]. A protocol conducted in this way — highly individualized and integrated — provides the best scientific basis for an accurate identification of the pathomechanism, making it possible to move on to the phase of targeted and safe therapeutic interventions.

9.2. The Role of the Multidisciplinary Team (Sports Medicine Physician, Psychiatrist, Sexologist, Endocrinologist, Urogynecological Physiotherapist)

Contemporary evidence-based medicine (EBM), drawing on the guidelines and consensus statements of the International Olympic Committee (IOC), moves away from the traditional, reduced model of care for the elite athlete, in which the treatment of sexual dysfunction rested solely with the urologist or gynecologist [3], [5]. The polyetiological nature of these disorders in the competitive environment — where high energy expenditure (RED-S) is inseparably interwoven with substantial psychological distress, pharmacological iatrogenesis, and mechanical damage to the pelvic apparatus — points to a clear need to implement the biopsychosocial paradigm. Implementing therapy that is effective, safe for the athlete's career, and consistent with anti-doping regulations is clinically very difficult without close synergy within a multidisciplinary therapeutic team. Such a team, based on continuous communication and an integrated decision-making algorithm, represents the most coherent scientific and pragmatic response to the disruption of the hypothalamic-pituitary-gonadal (HPG) axis and the cognitive distortions coupled with it [1], [3], [21].

The central coordinator (case manager), at the front line of the diagnostic process, is the sports medicine physician. It falls within their competence to take a careful history, apply screening tools (for example, the LEAF-Q or IIEF-15 questionnaires), and identify early the red flags pointing to the development of overtraining syndrome (OTS) or caloric deficits [7], [23]. The sports physician plays a key role in communication between the coaching staff and the patient. It is they who must obtain from the coach a firm modification of training loads (a targeted detraining or taper) and nutritional restitution, without which any causal treatment of RED-S is unlikely to succeed. In addition, this specialist reviews the athlete's pharmacological profile against the regulations of the World Anti-Doping Agency (WADA), managing any process of obtaining a therapeutic use exemption (TUE) — for example, where hormonal treatment is required in post-traumatic pituitary decline — protecting the patient from disqualification while at the same time safeguarding their reproductive health [3], [23], [49].

A leading role in the process of restoring biochemical and physiological balance is played by the endocrinologist. The functioning of the reproductive system and of the drive sphere is largely governed by the neurohormonal pathways, which in competitive sport are subject to the most serious pathology (FHA in women, EIHH and ASIH in men). The endocrinologist's task is a precise, targeted intervention aimed at the restitution of the HPG and HPA axes [10], [38]. In female athletes with functional hypothalamic amenorrhea, the endocrinologist decides on the strategy for reversing generalized hypoestrogenism, while avoiding "masking" the problem with ordinary oral hormonal contraception, which does not reverse losses in bone mineral density (BMD) and indeed masks the absence of natural ovulation [10], [28]. In patients with pronounced hypogonadism induced by steroids (ASIH) or trauma (PTHP), the endocrinologist carefully implements protocols to stimulate testicular function (the use of hCG or SERMs in the post-cessation phase, where anti-doping status permits) or coordinates testosterone replacement therapy (TRT) in former competitive athletes, in order to restore vasodilatory capacity, nNOS, and drive [18], [27], [38].

Given the substantial contribution of cognitive distortions, performance anxiety, and body dysmorphia to the genesis of dysfunction, the collaboration of a psychiatrist and a clinical sexologist is a central element of the team [13], [25]. The psychiatrist is responsible for the diagnosis and, where appropriate, the pharmacotherapy of coexisting severe affective disorders (depression) and eating disorders (anorexia, bulimia), which undermine self-acceptance and libido [3], [47]. An important task is the selection of agents in a way that avoids the iatrogenic post-SSRI sexual dysfunction syndrome (PSSD), using atypical drugs that do not impair erectile function or orgasm [30]. In parallel, the sexologist (most often working within cognitive-

behavioral therapy, CBT) addresses the direct treatment of sexual aversion, the dismantling of pathological anxious conditioning in the course of GPPPD, and the "spectator role" (spectatoring). This therapist rebuilds the athlete's intimate assertiveness, teaches the separation of one's human worth from strict sporting results, and works through the not-infrequent traumas arising from abuse (safeguarding failures) in toxic coaching environments [1], [3], [41].
The architecture of sports medicine would, in this respect, be incomplete without the participation of a urogynecological physiotherapist (or a urological physiotherapist/pelviperineologist). In strength disciplines (where pathological hypertonicity of the perineal muscles commonly occurs because of the chronic Valsalva maneuver and IAP) and in endurance and aesthetic sports (where mechanical dyspareunia and compression of Alcock's canal develop), manual pelvic therapy constitutes causal treatment [16], [34]. The physiotherapist, using advanced biofeedback techniques, dry needling of the fascia, and trigger-point release, decompresses the entrapped trunks of the pudendal nerve in cyclists and teaches weightlifters the conscious relaxation of the pelvic floor between contractions [34], [37]. In female athletes after months of vaginal atrophy due to FHA, work with scarred tissue, vestibular desensitization, and the use of dilators are an important — and often the most effective — mechanical means of restoring elasticity and the capacity for painless penetration in the course of GPPPD, directly enabling the goals set by the sexologist to be achieved [16], [17], [29].
In conclusion, the interdisciplinary model of care in elite sports medicine is not an optional recommendation but an important clinical imperative. Only the simultaneous de-escalation of physical load (the sports medicine physician), biochemical restitution of intracellular pathways (the endocrinologist), neuromodulation and cognitive reconstruction (the psychiatrist and sexologist), and biomechanical decompression of the urogenital structures (the urogynecological physiotherapist) can break the self-reinforcing cycle of dysfunction. This integrated synergy of experts represents the most reliable way of protecting the patient from lasting impotence, infertility, or anhedonia, and of allowing competitive sport to be practised in a humane way, without sacrificing the most intimate sphere of human health and identity [1], [3], [21].

9.3. Evidence-Based (EBM) Therapeutic Interventions

9.3.1. Optimization of Nutrition and Energy Intake (Reversing RED-S)

In line with the established guidelines of evidence-based medicine (EBM) and the most recent consensus statements of the International Olympic Committee (IOC), the central element of causal treatment for reproductive and psychosexual dysfunction of metabolic origin is a thorough optimization of energy availability (EA) [5], [7]. Any interventions aimed at addressing relative energy deficiency in sport (RED-S) — which manifests clinically as functional hypothalamic amenorrhea (FHA) in women and exercise-induced hypogonadotropic hypogonadism (EIHH) in men — should rest on reversing the direction of chronic catabolism [10], [43]. The use of isolated symptomatic pharmacotherapy or of urological-gynecological interventions, without first securing a balanced caloric base, is regarded in contemporary reproductive endocrinology as a flawed approach that does not lead to a structural restitution of the hypothalamic-pituitary-gonadal (HPG) axis [3], [23]. Accordingly, the primary therapeutic goal of the clinical team becomes the precise calibration of energy intake, taking into account not only total body mass but, above all, the athlete's fat-free mass (FFM) and their daily exercise-related energy expenditure [7], [50].
Clinical optimization of nutrition requires the use of precise metabolic algorithms, in which energy availability is defined by the following equation:

$$EA = \frac{EI - EEE}{FFM}$$

where EI (energy intake) denotes the daily energy supply from food, EEE (exercise energy expenditure) is the energy expenditure generated exclusively during training, and FFM is the fat-free mass expressed in kilograms [5], [32]. EBM indicates that the pathological threshold below which a central decline of the HPG axis and a cascade of dysfunction are initiated (including a complete loss of drive and of erection in men and a loss of lubrication in women) lies below a value of 30 kcal/kg FFM/day [7], [42]. An effective therapeutic protocol aimed at reactivating the pulsatile secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus requires that EA be raised and stabilized at a physiological level exceeding 45 kcal/kg FFM/day [33], [43]. Reaching this demanding level conditions the return of cellular satiety signals — including peripheral leptin, free triiodothyronine (fT3), and insulin-like growth factor 1 (IGF-1) — which together act as a trophic stimulus that reactivates the kisspeptin neurons, the evolutionary "switch" for the return of human sexuality [10], [23], [50]. An important aspect of the RED-S reversal protocol is not only the absolute caloric value but also the targeted modulation of the nutrient density and macronutrient profile of elite athletes' diets [5], [7]. Molecular endocrinology indicates that the restoration of HPG axis function is particularly sensitive to a precise supply of carbohydrates. A deficit of muscle and hepatic glycogen markedly intensifies cortisol secretion and inhibits leptin release; for this reason, in the treatment of EIHH and FHA, a substantial increase in carbohydrate intake is recommended, often to values on the order of 6–8 g/kg of body mass, which directly attenuates cellular adrenal stress and lowers the cortisolemia curve [33], [50]. At the same time, extreme restriction of dietary fat intake — so common among athletes in aesthetic and physique sports — is to be avoided. Lipid fractions, including an adequate supply of exogenous and endogenous cholesterol, are an essential structural precursor for the intracellular biosynthesis of steroids — including estradiol in the ovaries and testosterone in the testes — which is a necessary condition for regaining the lost endothelial responsiveness of the pelvic vessels (vulvar and penile vasodilation) [27], [28], [53].

Contemporary paradigms of nutritional chronobiology in sport (including the most recent work on within-day energy deficiency, WDD) are substantially changing the approach to dietetics in the treatment of reproductive dysfunction in athletes [43], [47]. It has been shown that even when the total daily energy balance calculated over a 24-hour cycle is neutral, the mere occurrence of multi-hour windows of pronounced catabolism (for example, the practice of training early in the morning in a fasted state in endurance sports, or delaying meals for several hours after a hard session) produces a sufficient systemic disturbance to sustain HPG axis suppression [7], [10]. The central nervous system continuously monitors the metabolic environment; sharp "dips" in blood glucose availability signal a state of sudden deficiency to the hypothalamus, promoting the secretion of protective corticotropin-releasing hormone (CRH) and blocking LH synthesis. An effective dietary and sexological intervention should therefore rest on a balanced, isocaloric, and pulsatile redistribution of meals, with emphasis on intra-training energy replenishment (carbohydrates in liquid form), in order to "settle" the brain's satiety-analysis center lastingly and gradually rebuild the sexual function impaired by a starvation pattern [3], [43], [50].

The pace at which psychosexual disturbances resolve under EA optimization alone requires considerable patience from the clinical team and the patient, owing to the latency (delay) of the neuroendocrine response. While basic metabolic markers such as fT3 rise within just a few weeks, the full restitution of vital energy, the return of spontaneous morning erections (NPT) in men, the recovery of the capacity for painless intercourse (reversal of GPPPD through E2-driven mucosal renewal), and the return of spontaneous menstruation in women take 3 to as

many as 12 months of consistently maintaining an energy surplus [10], [23], [28]. For this reason, international scientific bodies caution against the pharmacological "masking" of RED-S symptoms. The routine prescription of oral contraceptives to female athletes with FHA produces only an artificial withdrawal bleed, creating an illusion of health without restoring natural, ovulatory fertility, and, by stimulating SHBG production, markedly lowers free testosterone, further reducing what remains of libido [25], [43]. The hasty introduction of testosterone replacement therapy (TRT) in young, overtrained men with EIHH, without demonstrated mechanical or chemical (ASIH) injury, is similarly inadvisable; structured energy optimization is the most effective autogenous corrective therapy, one that lastingly and safely restores sexual health without exposing the athlete to an early end to their career at the hands of anti-doping bodies [9], [38], [50].

9.3.2. Pharmacotherapy (Including PDE5 Inhibitors and Testosterone Replacement Therapy — in the Context of WADA Regulations and Therapeutic Use Exemptions, TUE)

Where the optimization of energy availability (the reversal of RED-S), cognitive-behavioral therapy, and targeted urogynecological physiotherapy do not bring full restitution of psychosexual function, or where the diagnostic protocol reveals irreversible organic damage to neurovascular and endocrine structures, the implementation of targeted pharmacotherapy becomes a medical necessity. Nevertheless, the prescription of medication in the elite-athlete population is one of the more complex challenges in contemporary pharmacotherapy, because of the need to operate within the strict legal-medical framework imposed by the World Anti-Doping Agency (WADA). Every urological or endocrinological intervention must be carefully examined for the presence of an active substance on the Prohibited List, which requires of the sports medicine physician a thorough knowledge not only of pharmacokinetics but also of global anti-doping procedures, so that the process of restoring reproductive health does not result in disqualification and a premature end to the career [3], [8], [49].

The first-line agents for the short-term treatment of peripheral erectile dysfunction (ED) of psychogenic origin (for example, conditioned by performance anxiety), of mild ischemic origin (the early phases of perineal compression in cycling), and of iatrogenic origin (caused by SSRI antidepressants) are phosphodiesterase type 5 inhibitors (PDE5i), which include sildenafil, tadalafil, and vardenafil. The mechanism of action of these molecules is based on inhibiting the enzyme that breaks down cyclic guanosine monophosphate (cGMP) in vascular smooth muscle, which leads to a substantial amplification of the vasodilatory action of the endogenous nitric oxide (NO) released during erotic stimulation. From the standpoint of WADA regulation, PDE5 inhibitors are not currently classified as prohibited substances, despite occasional discussion of their potential to improve aerobic capacity under high-altitude hypoxia, which makes them a fully licit and effective tool for improving the blood supply to the pelvic organs in both sexes [27], [40], [52]. It should be emphasized, however, that the effectiveness of PDE5i depends biologically on an adequate intracellular testosterone level. In states of severe androgen decline (such as ASIH or pronounced EIHH), the absence of testosterone suppresses the transcription of neuronal nitric oxide synthase (nNOS), so that the substrate needed to initiate the cGMP pathway is not formed in the corpora cavernosa, rendering PDE5 inhibitors pharmacologically ineffective until the patient's eugonadism is restored [20], [27], [34].

The clinical challenge becomes considerably greater when the athlete-patient develops pronounced, organic, and irreversible damage to the hypothalamic-pituitary-gonadal (HPG) axis, requiring the implementation of medical testosterone replacement therapy (TRT). The EBM indications for androgen substitution in sports medicine include post-traumatic hypergonadotropic hypogonadism (caused by bilateral mechanical destruction of the testicular parenchyma in combat sports), post-traumatic hypopituitarism (PTHP following repeated mTBI

concussions and damage to the infundibular stalk), and non-neoplastic, genetically conditioned testicular insufficiency syndromes that become apparent in adulthood. Restoring physiological testosterone concentrations, within the laboratory reference range, in these patients has an effect that can rescue intimate life: it rebuilds sexual drive, relieves the severe depression of hormonal origin, and restores vascular responsiveness (the re-expression of nNOS), on which supportive PDE5i treatment can be added [15], [27], [46].

Exogenous testosterone, however, along with its esters and precursors, is in class S1 (Anabolic Agents) on the WADA list, which means that it is prohibited both in competition and out of competition. For an active competitive athlete to take TRT legally, restoring sexual function, they must obtain a therapeutic use exemption (TUE) from the relevant anti-doping body. The TUE committee applies very restrictive criteria in this case (defined by the WADA documents Physician Guidelines for TUE — Male Hypogonadism). A TUE may be granted only in cases of a clinically proven, organic (structural) pathology of the HPG axis. At the same time, the organization does not accept applications for TRT in functional states. This means that exercise-induced hypogonadotropic hypogonadism (EIHH) in a marathon runner with RED-S will not be an indication for a TUE — the anti-doping committee proceeds from the sound physiological premise that this state is reversible and that the only legitimate and permissible treatment is the nutritional correction and reduction of training load described earlier, rather than steroid therapy [7], [8], [38].

The most difficult ethical and legal-medical impasse concerns patients contending with anabolic steroid-induced hypogonadism (ASIH). Former athletes who misused doping and have experienced irreversible pituitary suppression and Leydig cell atrophy suffer from debilitating anhedonia, loss of erection, and a pronounced relational dysfunction. From a medical standpoint, they are a clear target population for a lifelong TRT protocol. WADA regulations, however, contain a firm clause (the "rule of consequence"): a TUE for testosterone can never be granted if the clinical state from which the patient suffers is a consequence of prior, illicit use of prohibited substances [38], [48], [49]. The sports medicine physician is, in this case, in a difficult position — facing an EBM-consistent choice of writing a prescription for urologically restorative androgens, which is, however, tantamount to the athlete breaching anti-doping rules and to an immediate end to their professional career. This phenomenon leads to considerable frustration in the strength and physique subgroup, compelling athletes contending with ASIH to choose between remaining in competition at the cost of a prolonged, distressing chemical "castration" and giving up competitive sport in order to protect what remains of their psychosexual health [20], [38], [49].

9.3.3. Psychotherapy (Including CBT for Coping with Pressure and Psychogenic Disorders)

Within the structure of evidence-based (EBM) therapeutic interventions for sexual dysfunction in competitive athletes, pharmacotherapy and energy optimization (the reversal of RED-S) should be closely and inseparably integrated with highly targeted psychotherapy. The complexity of the pathogenesis in the sporting elite — where high neuroendocrine loads intersect with disabling performance pressure, body dysmorphia, and a not infrequently toxic dynamic in the coach-athlete relationship — makes psychiatric and sexological intervention a central element of causal treatment [11], [13], [41]. The guidelines of international sexological societies indicate that methods based solely on the biomedical model are markedly insufficient in the athletic population, particularly given that the athlete's entrenched cognitive distortions (such as pathological perfectionism and an obsession with control) persistently sustain hyperactivity of the stress (HPA) axis and increased sympathetic tone, which biomechanically

and chemically hinders the generation of a physiological sexual response in the pelvic organs [1], [3], [21].

The psychotherapeutic gold standard for addressing the dysfunctions described is individualized cognitive-behavioral therapy (CBT), directed at deconstructing the sport-specific pathological thought patterns. In men contending with psychogenic erectile dysfunction (ED) and in women with desire disorders (HSDD), the principal goal of CBT is to neutralize performance anxiety and the unhelpful "spectator role" (spectatoring) [25], [41]. Elite athletes, conditioned over years of a strict regimen to the continual, rigorous monitoring of their body's parameters (heart rate, power, technique, diet), carry this hyperanalytical pattern over into the intimate sphere, becoming their own harsh judges during the sexual act. CBT teaches the patient to identify these automatic, catastrophizing thoughts precisely and allows attention to be redirected from the external assessment of an "outcome" (for example, the imperative of achieving an erection or orgasm) to the internal, sensory experience of the erotic stimulus, which effectively reduces the release of catecholamines and restores the endothelium's capacity for vasodilation [1], [26], [41].

In physique sports (bodybuilding) and aesthetic sports (gymnastics, figure skating), where the primary cause of reduced drive and physical avoidance of closeness is severe eating disorders and muscle dysmorphia (MD), the CBT protocol must be carefully adapted to the treatment of body-image disturbances [13], [47], [53]. An athlete affected by body dysmorphia or by the complications of anorexia experiences a disabling fear of exposure (body concealment), treating their own body solely as an imperfect instrument subject to public scoring rather than as a personal source of pleasure or intimacy. Psychotherapy in this particular area focuses on a thorough cognitive restructuring: interrupting rumination about supposed defects of adipose tissue or insufficient hypertrophy, and reducing the conditioned aversion to one's own physiological morphology [13], [25], [47]. This process is important for the patient's recovery of bodily assertiveness, without which the establishment of a safe, relaxed relationship with a partner (necessary for relieving perineal muscular hypertonicity and for preventing GPPPD in female athletes) remains neurobiologically blocked [29], [41].

An integral and highly specialized component of psychological treatment in sports medicine is the use of behavioral desensitization techniques, including Sensate Focus — classically developed by Masters and Johnson — adapted to the needs of competitive athletes [25], [41]. The use of this method in athletes for whom the sexual act has become a source of conditioned pain (for example, an acquired mechanical dyspareunia that becomes somatized) or of disabling frustration owing to drug-related (PSSD) or post-traumatic loss of erection rests on a temporary, complete, and therapeutic prohibition of attempts at penetration and of striving for climax. Through the slow, staged introduction of non-directive touch, the therapist helps the athlete's blocked nervous system to "unlearn" the association of closeness with pressure to perform or with a task-related obligation, rebuilding the neuroplasticity of the dopaminergic reward pathways impaired by allostatic stress [26], [41]. This technique, combined with sound, physiology-based psychoeducation, demystifies for the patient the unrealistic and not infrequently pornographic standards of sexual performance that, in the highly competitive world of "alpha athletes," secondarily intensify a substantial sense of shame and inadequacy over the impairments experienced [1], [11], [41].

Finally, effective EBM psychotherapy in the sporting elite should incorporate trauma-informed care, addressed to athletes who are victims of abuse in authoritarian, closed training structures (safeguarding failures) and to patients exposed to chronic minority stress (for example, homophobia in team and combat sports) [3], [22], [31]. In cases where a clinical sexual collapse (acute sexual aversion, the most severe forms of HSDD and anorgasmia) has a deep basis in post-traumatic stress disorder (PTSD) generated by toxic coaching oppression, standard CBT techniques are not infrequently combined with eye movement desensitization and reprocessing

(EMDR) therapy, targeted at dissociation and at the fear encoded in the tissues through memory [25], [41]. Establishing in the patient a sense of inviolable autonomy, a clear separation of their intrinsic human worth from sporting successes measured by the stopwatch, and the guarantee of a safe, strictly confidential therapeutic space constitutes a meaningful clinical intervention in sports medicine. This approach protects the athlete's psychosexual health in a way that lies entirely outside the strict restrictions of the World Anti-Doping Agency (WADA), safeguarding both their eugonadal homeostasis and the psychological foundations of their existence in the arena and beyond [1], [3], [21].

10. Discussion and Limitations

Over recent decades, the phenomenon of sexual and reproductive dysfunction in the competitive-athlete population has evolved from a marginalized, taboo subject into one of the more pressing interdisciplinary challenges of contemporary sports medicine. The detailed pathophysiological synthesis carried out in this work indicates that these disorders do not constitute isolated organ defects but are rather a multidimensional, biopsychosocial reflection of substantial allostatic overload. The etiological paradigm extends across a broad spectrum: from the evolutionarily conditioned neuroendocrine decline of the hypothalamic-pituitary-gonadal (HPG) axis in response to a severe energy deficit (RED-S) and overtraining syndrome (OTS), through irreversible mechanical damage to neurovascular structures in cyclists and contact-sport athletes, to substantial iatrogenesis caused by the misuse of doping pharmacology (AAS, diuretics) and psychotropic medication (SSRIs). Moving beyond the traditional, reductionist view toward integrated knowledge from molecular endocrinology, urology, psychiatry, and biomechanics provides an important basis for understanding that the reproductive organs of elite athletes are markedly sensitive to the demanding environment of competitive sport [3], [5], [21].

Nevertheless, a critical evaluation of the available medical literature (evidence-based medicine) reveals a number of fundamental methodological and epidemiological limitations that considerably complicate a precise quantification of the phenomenon. A primary cognitive barrier distorting the true prevalence of dysfunction in the populations studied is the substantial phenomenon of symptom concealment (underreporting). The elite athlete functions within a strict hypercompetitive regimen, in which admitting to deficits in the intimate sphere — which, in the common, mythologized perception, is synonymous with vitality and strength — entails considerable fear of stigma, of the loss of sponsorship contracts, and of marginalization within the coaching hierarchy. For this reason, data based on psychometrically validated self-report questionnaires (such as the IIEF-15, FSFI, and ASEX) carry a high risk of error arising from social desirability bias and recall bias, which suggests that the true scale of erectile dysfunction (ED), anhedonia, and painful intercourse (GPPPD) is substantially underestimated in the literature [1], [3], [25].

Another significant limitation of the research conducted to date is its study design. The great majority of publications in sports sexology rest on cross-sectional study designs and retrospective cohort analyses, which allow only the detection of associations (correlations) between a given discipline and a decline in sexual parameters, without firmly demonstrating a causal relationship. The EBM literature shows a marked shortage of long-term, prospective longitudinal studies tracking the trajectory of athletes' psychosexual health from the early peripubertal stage, through the peak of the career, to the later period of sporting "retirement." Moreover, the considerable heterogeneity of study protocols — the mixing of endurance and strength athletes in meta-analyses, and differences in the definition of "competitive sport" itself — means that averaged statistics often obscure the highly specific pathophysiological injury

pathways, making it difficult to draw universal clinical conclusions for the entire athletic population [2], [9], [42].

Scientific discourse should also address the historical androcentric bias (the focus on the male sex) that has dominated sports medicine for decades. In men, a decline in testosterone and a loss of erectile capacity (ED) were diagnosed relatively early and treated as a urological pathology requiring intervention. In elite female athletes, by contrast, the phenomenon of generalized HPG axis dysfunction was for years reduced solely to the aspect of fertility loss (amenorrhea), with the quality of their intimate life largely overlooked. The recognition that chronic hypoestrogenism and secondary vulvar atrophy give rise to severe organic dyspareunia and a suppression of drive (HSDD) through the RED-S mechanism, and that an extreme aesthetic regimen drives a disabling body dysmorphia, has begun to be reflected in the evidence-based literature only over the past decade or two. The absence of well-developed, structured diagnostic frameworks integrating the DSM-5-TR criteria with the early recognition of GPPPD in competitive gymnasts and marathon runners is an important gap that delays effective urogynecological and sexological intervention in this group of patients [10], [24], [28].

A limitation of an ethical and jurisdictional nature that is, at the present stage, unresolved — and that bears directly on the quality of treatment and on the feasibility of interventional clinical trials (randomized controlled trials, RCTs) — is the presence of World Anti-Doping Agency (WADA) regulations. Restorative protocols that are effective and proven in the general population for patients with a severely impaired HPG axis — such as the early introduction of testosterone replacement therapy (TRT) or the administration of selective estrogen receptor modulators (SERMs) and human chorionic gonadotropin (hCG) — come into direct conflict with the rules in the sporting environment. The difficulty of readily obtaining a therapeutic use exemption (TUE) for active athletes contending with functional (EIHH) or post-doping (ASIH) androgen decline means that competitive athletes are a distinct, disadvantaged group of patients, lacking full access to the treatment that non-athletic patients ordinarily receive. This strongly modifies treatment outcomes in studies (treatment bias), obliging sports physicians to operate solely within the sphere of nutritional and psychotherapeutic (CBT) interventions, which in the most severe, structural pathology may be far from sufficient [8], [38], [49].

Taken together, the limitations outlined above also point to clear directions for future research. Contemporary sports medicine should work toward implementing advanced multi-omics (including metabolomics and proteomics) in order to identify early, anticipatory micro-biomarkers of HPG axis decline before generalized clinical insufficiency, with overt erectile dysfunction or FHA, develops. Also needed are the institutional education of coaching staff in the primary prevention of RED-S and a decisive break with the stigmatizing practices of body shaming, which give rise to the body-image disturbances that are detrimental to libido. The integration of highly specialized sexology into the core of sports medicine, extended by an independent, safe environment for the anonymous reporting of symptoms (safeguarding frameworks), is among the most coherent scientific and humane ways to ensure that elite competition ceases to be an evolutionary trap that compels a person to trade reproductive health for a measurable sporting triumph [21], [23], [27].

11. Conclusions

A comprehensive pathophysiological, endocrinological, and psychiatric analysis of sexual dysfunction in the sporting elite leads to a substantial revision of the classic medical paradigm in which physical activity is regarded solely as a salutogenic factor. Evidence-based medicine (EBM) indicates that the relationship between exercise volume and the functioning of the hypothalamic-pituitary-gonadal (HPG) axis breaks down at the extreme pole of loading. Elite competitive sport generates a substantial, chronic allostatic load, in the face of which the

athlete's body engages evolutionary survival mechanisms defined as energy triage. In a state of severe deficiency, the prioritization of the metabolic pathways needed to sustain locomotor and cardiovascular functions occurs at the direct expense of the energy-demanding reproductive systems. This results in a situation in which a patient of outstanding motor performance becomes, from the standpoint of endocrinology and sexology, a substantially unwell individual, largely unable to generate a physiological erotic response [5], [21], [32].

The conclusions drawn from the analysis of individual disciplines reveal a notably heterogeneous, sport-specific etiology of these disorders. In endurance sports, the predominant mechanism is a central neuroendocrine decline related to relative energy deficiency in sport (RED-S) and overtraining syndrome (OTS), manifesting as functional hypothalamic amenorrhea (FHA) in women and exercise-induced hypogonadotropic hypogonadism (EIHH) in men [10], [23]. In strength and physique sports, in turn, the principal axis of damage is the intentional-iatrogenic component (the steroid-induced lasting necrosis of testicular tissue, ASIH) and the mechanical component, leading to a marked compressive hypertonicity of the pelvic floor muscles as a result of operating with very high intra-abdominal pressure [18], [37], [38]. A quite different, comparably damaging profile is presented by contact sports (where a leading cause of the loss of drive may be post-traumatic hypopituitarism, PTHP, arising from acceleration/deceleration damage to the brain) and by disciplines based on prolonged contact with a saddle, in which mechanical obstruction of the pudendal arteries and entrapment of the peripheral pelvic nerves develop [15], [36], [52].

An inseparable component undermining the psychosexual homeostasis of the sporting elite, one that warrants firm attention within the medical community, is the severe psychopathology and pharmacotherapy superimposed on the somatic picture. Muscle dysmorphia, performance anxiety, and pathological perfectionism interfere with the mesolimbic system and the dopaminergic pathways, turning the sexual act from a relational bond into yet another arena for the competitive assessment of one's own body [13], [25]. Moreover, the licit and seemingly safe prescription of psychotropic medications (particularly SSRIs in the treatment of, for example, post-competition depression), analgesics (opioids, NSAIDs), and diuretics may draw athletes into a self-reinforcing cycle of chemical "castration," anorgasmia, and treatment-resistant peripheral erectile dysfunction (ED). Phenomena such as PSSD (post-SSRI sexual dysfunction) make iatrogenesis as hazardous for an athlete's reproductive capacity as the mechanical injuries sustained in the arena itself [19], [30], [49].

The implementation of effective corrective protocols calls for a clear move away from reduced urological-gynecological models toward fully integrated multidisciplinary teams. Effective treatment of the competitive-athlete patient is unlikely without close synergy among the sports medicine physician (modulation of load and reversal of within-day energy deficiency), the endocrinologist (restitution of intracellular pathways and oversight of TRT), the psychiatrist and sexologist (CBT targeted at conditioned anxiety and anhedonia), and the urogynecological physiotherapist (fascial release and work with conditioned dyspareunia). The medical care system should also actively counter the phenomenon of symptom concealment (underreporting) by creating safe consultation spaces, separated from the coaching authority, that guarantee the athlete full anonymity and respect for their bodily autonomy [1], [3], [41], [50].

In conclusion, the central message of this analysis is the pressing need for a bioethical humanization of competitive sport. The discrepancy between a measurable triumph on the stopwatch and a hidden, distressing incapacity in the intimate sphere is a striking paradox of contemporary competitive sport. The task of international medical committees, anti-doping decision-makers (WADA), and coaching staff should be to create an environment in which the protection of the athlete's eugonadal, neurological, and psychological reproductive health is treated as a value that takes precedence over any sporting result. Only such a shift in paradigm will prevent a situation in which outstanding members of society pay for medals the highest

personal price, in the form of the loss of the fundamental capacity to experience a satisfying, painless sexuality [3], [5], [21], [23].

DISCLOSURE:

Conceptualization: Arkadiusz Psiuk, Jarosław Tsukerman
Methodology: Joanna Szymocha, Jarosław Tsukerman, Arkadiusz Psiuk
Investigation:, Arkadiusz Psiuk, Aleksandra Wojas,Jarosław Tsukerman, Igor Smędowski, Joanna Szymocha
Writing-rough preparation: Arkadiusz Psiuk, Igor Smędowski, Aleksandra Wojas, Joanna Szymocha
Writing-review and editing:, Arkadiusz Psiuk, Aleksandra Wojas, Jarosław Tsukerman, Joanna Szymocha, Igor Smędowski
Supervision: Arkadiusz Psiuk, Jarosław Tsukerman, Igor Smędowski
Project administration: Igor Smędowski

Funding statement:
This study received no external funding.
Informed consent statement:
Not applicable.
Institutional Review Board Statement:
Not applicable.
Conflict of interest:
The authors declare no conflict of interest.

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

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