The broad term of sexual dysfunction includes erectile dysfunction, ejaculatory dysfunction, hypogonadism and low sexual desire, Peyronie’s disease and other penile morphological alterations, and urinary incontinence associated with sexual function. Knowledge of the epidemiology of various sexual dysfunctions is important in designing sexual health programs and allocation of budget and healthcare resources, in patients’ and partners’ education, and in clinical assessment of individual subjects. There are numerous epidemiologic studies in the contemporary medical literature, comprehensively detailing the prevalence of sexual dysfunctions; however, reported epidemiologic data vary greatly. Several factors account for these inconsistent data. The main one is probably the definition used to define a particular sexual dysfunction. For example, in selected high-quality studies reporting on erectile function outcome in the post radical prostatectomy male population, more than 20 different definitions of favorable erectile function were used. Hence, the reported incidence of adequate erectile function varies, ranging from 25 to 78%. Since the definition of sexual dysfunction is not unified, it is not unreasonable to expect variation in sexual dysfunction epidemiologic data; the higher the threshold for normal sexual function, the greater is the incidence of sexual dysfunction. Moreover, sexual dysfunction is commonly assessed using questionnaires. More objective modalities, such as hemodynamic assessment of the penis by Doppler ultrasound of the erect penis to establish a diagnosis of vasculogenic erectile dysfunction, or stopwatch-measured intravaginal ejaculatory latency time for establishing a diagnosis of premature ejaculation, are not commonly employed to define a sexual dysfunction. Not surprisingly, the type of questionnaire used in a certain study may also have an impact on epidemiologic findings. Another issue in sexual function epidemiologic research is the study population, as the prevalence of sexual dysfunction varies greatly according to age, risk factors, demographic population characteristics, and other population-related factors. An important consideration in the epidemiology of sexual dysfunction is the existence of specific risk factors in specific populations. There are many well-studied risk factors for sexual dysfunction. Common risk factors include cardiovascular diseases, obesity, hypertension, hyperlipidemia, smoking, lower urinary tract symptoms, radical pelvic surgery and, of course, diabetes. Radical pelvic surgery has a multifaceted impact on sexual function. For example, radical prostatectomy affects erectile function mainly by disruption of neural pathways, causing un-ejaculation resulting from removal of anatomic structures—the prostate and seminal vesicles—and increasing the risk of urinary incontinence during sexual activity owing to urinary sphincter weakness and even increasing the risk of penile morphologic changes, penile length loss, and Peyronie’s disease. There are also other, less commonly discussed but nonetheless important, factors that may be associated with the epidemiology of sexual dysfunction. It has been suggested that the prevalence of sexual dysfunction may be related to the availability of therapies and interventions for sexual dysfunction. New therapies may increase patients’ and partners’ awareness and hence increase reporting of sexual dysfunction. In the light of these difficulties in measuring and reporting sexual dysfunction, epidemiologic data should be interpreted cautiously.
Erectile dysfunction

Erectile dysfunction is the most commonly researched and discussed sexual dysfunction, and the most prevalent sexual dysfunction in older men. The first landmark study that looked at the epidemiology of erectile dysfunction was the Massachusetts Male Aging Study, by Feldman et al. and published in 1994. This was a community-based observational study, and looked at a random sample of non-institutionalized men aged 40–70 years in the Boston area. Erectile function was assessed by a self-administered questionnaire, and the study’s main findings were that the combined prevalence of minimal, moderate, and complete impotence was as high as 52%; subject age was the variable most strongly associated with impotence; and the prevalence of complete impotence tripled from 5 to 15% between subject ages 40 and 70 years. Another pivotal erectile dysfunction epidemiologic study was the Cologne Male Survey by Braun et al., published in 2000. This study looked at a European population, not a random sample but a representative sample of 8000 men, using a validated questionnaire. Results of this study were based on approximately 4500 evaluable questionnaires yielding a response rate of 56%. The prevalence of erectile dysfunction in this study was 19.2%, with a steep age-related increase (2.3 to 53.4%) and a high rate of conditions comorbid with erectile dysfunction – hypertension, diabetes, pelvic surgery, and lower urinary tract symptoms – corroborating findings of earlier studies of different populations. The most extensively studied risk factors for erectile dysfunction are cardiovascular risk factors, primarily diabetes, and other risk factors, including the metabolic syndrome and its components (abdominal obesity, dyslipidemia, hypertension, and impaired fasting glucose), smoking, ischemic heart disease, and peripheral vascular disease and other cardiovascular risk factors. Recognition of these important risk factors, especially modifiable cardiovascular risk factors, may improve patient knowledge and awareness, and provide a window for cardiovascular disease diagnosis and early intervention in men with newly diagnosed erectile dysfunction, leading to not only better sexual health but also better overall health. The main pathophysiologic link between erectile dysfunction and cardiovascular morbidity is probably endothelial dysfunction. However, there are risk factors other than cardiovascular ones, both organic and psychological, that are associated with increased risk of erectile dysfunction. Among the significant organic non-vascular risk factors is Peyronie’s disease, an under-diagnosed condition in men presenting with newly diagnosed erectile dysfunction. Peyronie’s disease may contribute to the development of erectile dysfunction probably by altering the elastic properties of the penile tunica albuginea. Another not uncommon risk factor for erectile dysfunction is testosterone deficiency (hypogonadism). It is well established that adequate testosterone levels are required not only for the penile vascular response during erection, but also to preserve penile structural integrity. Erectile dysfunction is more prevalent in men with certain non-organic risk factors, such as emotional, couple related, and socioeconomic factors, creating a complex picture when the epidemiology of erectile dysfunction is discussed in certain specific populations. A good example of an emotional risk factor for erectile dysfunction is depression. In a study by Shiri et al. the incidence of erectile dysfunction was 59/1000 person-years in men with depressive mood and 37/1000 person-years in those without depression. Theses authors also found that the association of depression and erectile dysfunction is bidirectional: not only were men with depression at increased risk for erectile dysfunction, but also men with erectile dysfunction were at increased risk for depression. In summary, the prevalence of erectile dysfunction is high and age-dependent, with more than half of men at age of 50 years or older being affected. There are many risk factors for erectile dysfunction, hence epidemiologic data in specific populations should be viewed with careful consideration of the specific characteristics of the population reviewed.

Premature ejaculation

Premature ejaculation is likely the most common sexual dysfunction in men across all age groups and populations, with a worldwide prevalence of approximately 30%. For clinical research purposes the accepted definition of premature ejaculation is an intravaginal ejaculatory latency time (IELT) of 1–2 minutes; a prospectively stopwatch-measured IELT is preferred over self- or partner-reported IELT upon recall. While there is a definition of premature ejaculation for research
purposes, in clinical practice there is no agreed definition. Waldinger et al. surveyed a population of 500 couples who were recruited from five countries, aged 18 years or older, had a stable heterosexual relationship for at least 6 months, with regular sexual intercourse. In their study, the median IELT was 5.4 minutes and the range was 0.55 to 41 minutes. In their study, the median IELT decreased significantly with age, from 6.5 minutes in the 18–30 years group, to 4.3 minutes in the group older than 51 years, while other studies did not show this age-related increase in prevalence of premature ejaculation. Regardless of whether the prevalence of premature ejaculation is clearly age-related or not, it is obvious that in younger men who are less likely to have other sexual dysfunctions such as organic erectile dysfunction, premature ejaculation is the most prevalent sexual dysfunction. In the real-world clinical setting, the use of stopwatch IELT to define premature ejaculation is definitely not a practical approach. Other ways to categorize men as having premature ejaculation are based on men self-reporting low or absent control over ejaculation irrespective of the duration of the ejaculation time, on the resulted distress for them or their sexual partner or both, or on patients’ report that they “climax too soon.” Indeed, in a Canadian web-based study of more than 3800 men, the prevalence of premature ejaculation (PE) ranged from 16% to 24% depending on the definition of PE utilized. The etiology underpinning this high prevalence remains to be clarified, but current evidence reflects a shift from psychogenic theories to more neurobiological bases. While elucidation of the etiology of premature ejaculation is undoubtedly important for development of more effective therapies, it is clear that, whatever the cause of the condition, it is associated with a significant burden on psychological and overall health. Generally, men with premature ejaculation are more likely to self-report other sexual dysfunctions (e.g., anorgasmia, low libido, erectile dysfunction) and psychological disturbances (e.g., depression, anxiety, excessive stress) than men without PE. Similarly, as for other sexual dysfunctions, the epidemiology of PE may vary in special groups. Tang and Khoo showed that PE prevalence varied according to ethnicity. It is worth mentioning that in their study, which included men in a primary care setting not a general population sample, the prevalence of PE was about 40%, significantly higher than in the general population. Shindel et al. looked at the prevalence of PE in another population of great interest, infertile couples, and found that about 50% of men reported that they ejaculated more rapidly than they wished. When men reported PE, their partners agreed with the diagnosis in 47% of cases. Female partners of men who did not report PE, reported PE in 11% of cases. Partner frustration related to PE was reported by 30% of men. Partners agreed that they were frustrated in 43% of these cases. Among the 70% of men who did not report partner frustration from PE, 93% of the partners agreed that they were not frustrated.

In summary, PE is hard to define exactly, yet it is the most prevalent sexual dysfunction and the most significant sexual dysfunction in young men.

**Peyronie’s disease**

Peyronie’s disease is commonly undiagnosed. The main clinical symptom is penile curvature; however, men may have significant Peyronie’s disease, manifested by penile plaques, erectile dysfunction, penile pain, and penile shortening, even without a curvature. Not uncommonly, the underlying cause of erectile dysfunction in poor responders to phosphodiesterase 5 inhibitors is Peyronie’s disease. The diagnosis of Peyronie’s disease in these men is established by physical examination and penile Doppler ultrasound in certain cases. Moreover, in men who are not sexually active or in men who are sexually active without achieving erection and performing penetration, the penile curvature may not be seen and Peyronie’s disease may exist but remain undiagnosed. Therefore, the reported prevalence of Peyronie’s disease depends on the manifestations of this condition, on a high index of suspicion, and on patients, partners, and sexual health care providers’ awareness. Epidemiological data on Peyronie’s disease are limited. Prevalence rates of 0.4–9% have been published, but the majority of the medical literature supports a prevalence rate of 3–8% or 5–8%. In the past, Peyronie’s disease was considered a condition that is limited to older men. However, newer data show that Peyronie’s disease does occur also in younger men and even in teenagers, but the prevalence in these very young men remains unknown. Men younger than 40 years are more likely to present at an earlier stage of Peyronie’s disease, to have diabetes, and to have more than one plaque at the time of presentation.
populations at risk, the prevalence of Peyronie’s disease is far greater. Diabetes, genetic predisposition, trauma of the penis, systemic vascular diseases, smoking, and alcohol consumption are all mentioned in the medical literature as risk factors for Peyronie’s disease.\textsuperscript{40,41} In diabetic men, not only is Peyronie’s disease more prevalent, but also it tends to manifest in older age, and present with longer duration and greater severity: There is greater penile curvature and more pronounced penile deformity, and a greater prevalence of coexisting erectile dysfunction, probably resulting both from penile structural alterations due to Peyronie’s disease itself and from diabetic vascular disease.\textsuperscript{42} Tal et al. looked at the incidence of Peyronie’s disease in a very distinct population consisting of men who had had radical prostatectomy as a monotherapy for prostate cancer, and calculated the 3-year post-prostatectomy incidence to be 16% and a mean time to presentation of 14 months after surgery.\textsuperscript{14} Rhoden et al. conducted a case-control study to shed more light on Peyronie’s disease risk factors, and found that race is a strong risk factor for Peyronie’s disease, with an odds ratio of 8.5.\textsuperscript{43} Interestingly, in this study, higher low-density lipoprotein (LDL)-cholesterol level (>130 mg/dL) and increased waist circumference (>102 cm) actually had a protective effect in Peyronie’s disease, with an odds ratio of 0.5 for both. Moreno and Morgentaler investigated the association of testosterone deficiency and Peyronie’s disease. Their study is of special significance since testosterone is a principal anabolic hormone in men and definitely has a major role in maintaining the health of penile tissues. In their pilot study, the severity of penile curvature correlated significantly with free testosterone level but not with total testosterone level, and a possible important association between testosterone deficiency and Peyronie’s disease was suggested.\textsuperscript{44} Besides the epidemiology of Peyronie’s disease itself, sexual healthcare professionals should be aware of the epidemiology of emotional conditions in Peyronie’s disease, which are under-represented in the medical literature and often under-diagnosed and treated in daily clinical practice. Consistent data from two leading sexual medicine centers in the United States show that the psychological burden in men with Peyronie’s disease is great, possibly greater than in men with other sexual dysfunctions.\textsuperscript{45,46} Overall very high rates of emotional burden and relationship problems attributable to Peyronie’s disease were found: 81% and 54%, respectively, predictors of which were penile length-loss and inability to have intercourse. Using validated instruments, it was demonstrated that 48% of men with Peyronie’s disease had clinically meaningful depression that would warrant medical evaluation. This high level of depression stayed consistent across time since diagnosis, suggesting that most men do not psychologically adjust to their diagnosis of Peyronie’s disease; all men with Peyronie’s disease should be considered for appropriate mental health screening. This high prevalence of psychological morbidity may be attributable to the fact that there is no fully effective treatment for Peyronie’s disease, and men with symptomatic Peyronie’s disease will never regain their pre-morbid penile function, appearance, and length.

In summary, the epidemiology of Peyronie’s disease is intriguing: Its prevalence is higher and its age distribution is broader than previously thought; there are numerous suggested risk factors and a very high risk of associated psychological morbidity, inherent to changes in penile appearance and function, that should not be overlooked.

### Hypogonadism

Hypogonadism (low testosterone) is under-diagnosed and under-treated. The significance of hypogonadism diagnosis and treatment cannot be over-emphasized, as androgen receptors and testosterone activity exist in most body tissues, organs, and systems. Testosterone deficiency effects are not limited to sexual function: besides desire and erection, it may also affect bone health, muscle mass, hematopoiesis, cognitive function, spermatogenesis and seminal fluid production, vascular and cardiac performance, lipid and glucose metabolism, and many other metabolic, primarily anabolic, processes. The key to diagnosis is awareness of the high prevalence of testosterone deficiency, identification of populations at risk, and early symptoms and laboratory evaluation. Worth mentioning is that symptoms of hypogonadism are not specific and can be attributed to other medical conditions, such as hypothyroidism or depression. Therefore, questionnaires are neither sensitive nor specific for hypogonadism screening or diagnosis, and must be combined with measured plasma testosterone levels.\textsuperscript{47,48} Clinically, it was suggested that the findings that are best correlated with late onset hypogonadism
are sexual symptoms (poor morning erection, low sexual desire, erectile dysfunction) and total testosterone level of less than 11 nmol/L (<320 ng/dL). Epidemiologic questionnaire-based studies without testosterone measurement failed to correctly define the true prevalence of hypogonadism, yielding an overestimated prevalence of up to 80%. Combining hypogonadism symptoms and plasma testosterone levels, Araujo et al. found that the overall prevalence of symptomatic testosterone deficiency in a random sample of men from the Boston Area Community Health Survey was 5.6%. In this study, symptomatic testosterone deficiency prevalence increased with age, yielding a prevalence of 18.4% in men over 70 years old. Age-related increased prevalence of hypogonadism was also demonstrated in the Baltimore Longitudinal Study of Aging: the prevalence of hypogonadal testosterone levels was about 20% in men over 60 years old, 30% in men over 70 years old, and 50% in those over 80 years of age, using total testosterone criteria; the prevalence was even greater when free testosterone criteria were employed. The prevalence of testosterone deficiency is increased in men with certain risk factors such as diabetes, cancer, lung disease, other systemic diseases, obesity, the metabolic syndrome, and erectile dysfunction, and in men with reduced mass and function of testicular tissue, for example, infertile men, men having varicocele, and men after unilateral orchiectomy. Diabetes is a well-studied risk factor for testosterone deficiency: in a study by Corona et al. the prevalence of hypogonadism was 24.5% in diabetic men versus 12.6% in non-diabetic subjects, which was statistically significant after adjustment for age and body mass index (BMI). Similar results were recently published by Al Hayek et al.: the prevalence of hypogonadism was 24.3% in diabetic and 8.3% in non-diabetic patients. Hypogonadism also is common in men with erectile dysfunction, with a reported prevalence of approximately 30–40%. Not uncommonly, erectile dysfunction is the presenting symptom. This population of men presenting with erectile dysfunction deserves special attention: it is a unique opportunity to impact men’s health. Correct evaluation and management of testosterone deficiency and other cardiovascular risk factors may actually reduce future cardiovascular morbidity and mortality. Therefore, symptomatic treatment of erectile dysfunction in these men without in-depth risk factor identification should be discouraged.

Less discussed risk factors for hypogonadism include poorly functioning testicular tissue and/or reduced testicular mass. Hypogonadism is more common among infertile men, with an estimated prevalence of 20–30%. Men with non-obstructive azoospermia, especially in Klinefelter’s syndrome, are at even greater risk. Surgical sperm retrieval procedures may cause further testicular tissue insult and reduce Leydig cell mass and testosterone production. While azoospermia is uncommon in men (0.5–2%), varicocele is fairly common, with an incidence of 15% in the general male population and up to 40% in infertile men. Recent studies have shown that men with varicocele are at increased risk for low testosterone levels; furthermore, varicocele repair may increase testosterone levels and may be considered as a definitive treatment for hypogonadism.

In summary, hypogonadism is a treatable cause of sexual dysfunction. While sexual dysfunction may be the presenting symptom, the benefits of testosterone level normalization are much broader. Awareness of the epidemiology of testosterone deficiency, especially in high-risk populations, is the key to diagnosis and treatment.

References


