

## Prevalence of Premature Ejaculation: A Global and Regional Perspective

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### ABSTRACT

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The effort to determine the global burden of premature ejaculation (PE) has been impeded by the lack of a clear and universally accepted definition of the condition. Current diagnostic criteria are variable and rely, to a large degree, on subjective measurements. Moreover, the absence of a clear consensus on what constitutes a normal ejaculatory latency has impeded research into the prevalence of PE, although evidence is gradually accruing that may make this definition easier. Perception of “normal” ejaculatory latency varies by country and can differ when assessed either by the patient or their partner. Despite these limitations, information from the Global Study of Sexual Attitudes and Behaviors and other sources suggests a global prevalence of PE of approximately 30% across all age groups. The etiology underpinning this prevalence remains to be clarified, but current evidence reflects a shift from psychogenic theories to more neurobiological bases. While elucidation of the etiology of PE will undoubtedly aid the 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.

**Key Words.** Premature Ejaculation; Sexual Dysfunction; Serotonin

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### Introduction

It is becoming increasingly common for practitioners such as urologists and primary care physicians to be presented with cases of premature ejaculation (PE). In part, this represents an acknowledgment by many men that PE is a medical condition. The increasing workload engendered by this shift in attitude has aroused much interest in the topic of PE. For this reason, it is important to define the workload by assessing the prevalence of PE. Previously, the prevalence of PE has been poorly defined, but recent research has provided a clearer indication of the global and regional prevalence of the condition and is starting to illuminate the differences in regional PE prevalence.

### Defining “Normal” in Premature Ejaculation

Rosen (2000) highlighted how the lack of clear diagnostic criteria has led to problems in defining the prevalence of PE and that prevalence values varying from 25% to 60% have been reported, depending on the criteria used. However, despite the lack of diagnostic acuity, Rosen confidently states that PE is the most frequent sexual complaint in men [1].

An accurate determination of the prevalence of any condition depends on an accurate definition of what is “normal.” Such a definition has been lacking for PE. In many respects this reflects the multifactorial influences on ejaculatory latency, and therefore defining normal ejaculatory latency is difficult. First, there is a wide variation in men’s,

and their partners', perception of what is normal. In the Multi Country Concept Evaluation and Assessment of PE incidence study (conducted on behalf of the Alza Corporation) the perception of how long it takes for the "average" man to ejaculate varies between 7 and 14 minutes (Figure 1) [2]. These estimates show substantial geographical variation in perceptions. Among the countries included in this study, the perceived average latency time for German men was only 7 minutes, but was over 13 minutes for men from the United States. The perceived average for men in the United Kingdom, France, and Italy was similar, at around 9.6 minutes.

Generally, women's estimates of the "average" ejaculatory latency were similar to men's, albeit a slight underestimation. The most striking disparity was observed between U.S. men and women, with female partners consistently estimating lower ejaculatory latencies than their partners (11.2 and 13.6 minutes, respectively). Conversely, German women were more generous in their estimation of "average" time to ejaculation than their partners (7.4 vs. 6.9 minutes, respectively).

The difficulties in defining "normal" ejaculatory latency are mirrored in the absence of a universally accepted clinical definition of *premature* ejaculation. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) [3] and the International Classification of Diseases, Tenth Edition (ICD-10) [4] are probably the most widely used definitions of PE and both are circumspect in their classification. The ICD-10 definition includes describing PE as "an inability to delay ejaculation sufficiently to enjoy lovemaking," while the DSM-IV definition states that PE is "persistent or recurrent ejaculation with minimal stimulation before, on, or shortly after penetration and before the person wishes it." Both definitions acknowledge three core components:

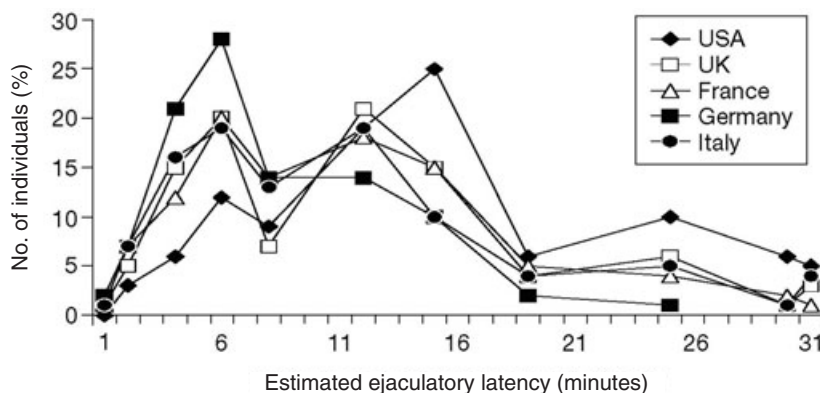
- short ejaculatory latency;
- lack of control over ejaculation;
- lack of sexual satisfaction.

The most obvious limitation of such definitions lies in the subjectivity of the diagnostic criteria, which individually do not provide a basis for either the definition of the condition or the study of the effects of treatment thereupon. This problem was highlighted by Rowland et al. (2001) who emphasized the wide variations in the definitions of PE used in some 45 studies conducted between 1963 and 2000 [5]. Quantifiable ejaculatory measures (e.g., latency, number of thrusts) or subjectively assessed PE criteria, such as perceived lack of control, were reported in less than 50% of studies. Furthermore, while intravaginal ejaculatory latency time (IELT) was the most commonly used index of PE, the cut-off points for latency varied from 1 to 5 minutes.

While most men who present to practitioners with a PE problem have accurately self-diagnosed their condition, it is less apparent that men, collectively, are aware that PE is a medical and therefore a treatable condition. This accents the need for studies that will define attitudes toward PE. Such studies should generate a clearer definition of PE and facilitate development of validated tools to measure ejaculatory control and the associated levels of sexual satisfaction.

#### Prevalence of PE in the Global Study of Sexual Attitudes and Behaviors

In a recent article, Nolzco et al. reported a high prevalence of sexual dysfunction among Argentinean men [6]. The study analyzed 2,715 sexual health questionnaires completed by men attending a prostate awareness campaign. The results indicated a 28.3% prevalence of PE based upon the



**Figure 1** Perceived normal time to ejaculation varies by country. Estimates of how long the "average" man in the United States, United Kingdom, France, Germany, and Italy takes to ejaculate. Percentage of men (y axis) plotted against their estimate of ejaculatory latency in minutes (x axis) [2].

self-report of the study participants. This figure is very much in line with the results of the Global Study of Sexual Attitudes and Behaviors (GSSAB), which has surveyed the attitudes, beliefs, and overall health of people in sexual relationships from 29 countries [7].

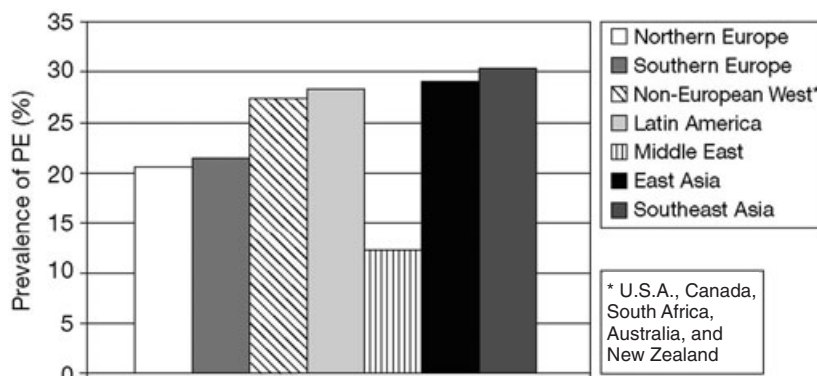
The GSSAB collected data from more than 27,000 participants aged 40–80 years (13,882 women and 13,618 men) via face-to-face and telephone interviews and self-completed mailed questionnaires. The data have provided information on the prevalence of a number of sexual dysfunctions, as well as the treatment-seeking behavior of people who suffer these conditions. The 29 countries in which surveys were taken have been grouped into the seven regions shown in Figure 2 [7].

Notably, and in agreement with Nolzco et al., the GSSAB reported that the prevalence of PE in Latin America was 28.3% (Figure 2) [6]. Overall, although the GSSAB showed that approximately one-third of all men may have PE, there were significant geographical differences. The lowest prevalence was reported in the Middle East (12.4%), while the highest recorded prevalence occurred in Southeast Asia (30.5%). Importantly, the GSSAB assessed the prevalence of self-reported PE by including only those patients who reported experiencing PE “sometimes” or “frequently.” Therefore, these men, who presented as self-diagnosed as PE sufferers, do not represent a population with a transient condition, but rather a cohort who have a chronic complaint that is often associated with significant quality-of-life issues for the couple [7].

### Factors Influencing Regional Variation in the Prevalence of PE

Several well-established factors may account for the regional variation in the prevalence of PE. For example, the popularity of circumcision in certain regions could affect the prevalence of PE, as this procedure is associated with glans penis keratinization and desensitization [8]. The variations may also partly reflect religious and cultural influences. For example, the attitudes to, and recognition of PE may be different among “puritan” Protestant and “liberal” Catholic populations, and it is interesting that in many demographic studies, Northern and Southern Europe are considered as distinct entities. Additionally, the rates of PE are likely to be greater in those regions where sex has special importance, such as East Asia—which is heavily influenced by the Tantra philosophy—and in Central and South America, where sex is regarded as very important and female sexuality is fully accepted. However, compared with Europe, the Middle East is represented by predominantly Muslim, patriarchal societies, where females occupy a lower social position. In these communities, PE may be viewed less as a medical condition than as a sign of virility.

Studies from the United States have shown equivocal data on the association of ethnicity with the prevalence of PE. The National Health and Social Life Survey (NHSLs), conducted in 1992, surveyed 1,410 men between the ages of 18 and 59 years and found that the prevalence of PE among Caucasian, black, and Hispanic men was



**Figure 2** Overall prevalence of premature ejaculation (PE) by region. Regional prevalence of PE in seven geographical regions. Percentage of patients who reported suffering from PE “sometimes” or “frequently.” Data from the Global Study of Sexual Attitudes and Behaviors of 13,618 men aged 40–80 years [7]. The presence of sexual problems was assessed with the following question: “During the last 12 months have you ever experienced any of the following for a period of 2 months or more when you: reached climax (experienced orgasm) too quickly?”

19%, 34%, and 27%, respectively. However, this finding of an apparently higher incidence among black men was not supported by a more recent study that reported a corresponding prevalence of 16%, 21%, and 29% in Caucasians, blacks, and Hispanics, respectively [9]. However, this study was conducted in an older population (40–80 years).

Data from the NHSLs have also shown that, unlike erectile dysfunction (ED), the incidence of which increases with age, the prevalence of PE is not associated with age [10]. Indeed, the prevalence is essentially constant over the 18–59 years age range (Figure 3). This highlights the fact that, in contrast with ED, PE often occurs in younger individuals. This places a particular importance on the discovery of effective management strategies as PE often occurs at a time when the instigation, development, and maintenance of relationships is a critical part of the life experience.

Overall, the GSSAB found few definitive correlates for the prevalence of PE. For instance, a preliminary association between vascular disease and PE was negated after controlling for the simultaneous presence of ED—a likely consequence of confusion about the two conditions among respondents. However, the GSSAB suggested that education is negatively associated with PE. For instance, in Central/South America and the Middle East, men with no college education were twice as likely to report PE [odds ratio (OR) 2.3–2.6,  $P < 0.05$  and OR 2.2–2.3,  $P < 0.05$ , respectively], compared with men who had received “at least some college” education. Other factors associated with PE included financial problems, although this was statistically significant only

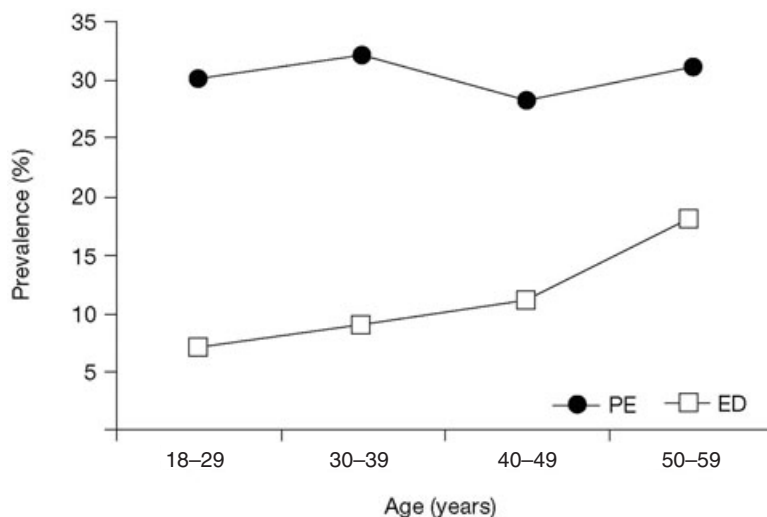
in the Middle East (OR 1.8,  $P < 0.05$ ) and, perhaps unsurprisingly, a trend for association of PE with infrequent sex [7].

### Etiology of PE

The etiology of PE remains to be fully elucidated. However, the last 10 years have witnessed a change from the belief that the condition is of a purely psychological nature to one based on the findings of neurobiological research.

### A Historical Perspective

Discussions on the etiology of PE have been ongoing for more than a century. As outlined by Waldinger, the historical timeline for the evolution of theories on the etiology of PE can be divided into four phases [11]. The first phase (1887–1917) can be seen as beginning with the first mention of “rapid ejaculation” in the medical literature, by Gross in 1887; von Krafft-Ebing added a second paper to the literature in 1901. The second period (1917–1950) began with the description by Abraham of “ejaculatio praecox,” a name that drew attention to ejaculation that was rapid, rather than premature [12]. Abraham developed a psychodynamic theory for the etiology of PE, believing the condition was the adult manifestation of unresolved and excessive narcissism during infancy, which resulted in an exaggerated importance being placed on the penis [13]. However, Abraham provided little evidence in support of his theory, and other researchers since have been similarly unable to generate an evidential base for this narcissism hypothesis [14]. Throughout this period, psychoanalysis was the predominant treat-



**Figure 3** Prevalence of premature ejaculation (PE) and erectile dysfunction (ED) with age. Prevalence (percent) of patients reporting PE (filled circles) and ED (open squares) over a 4-decade age range. Data from the NHSLs study of 1,410 U.S. men, aged 18–59 years, showing that, unlike ED, whose incidence increases with age, the prevalence of PE is not associated with age [10].

ment approach. In 1943, Schapiro defined PE as a psychosomatic disturbance caused by a combination of a “psychologically overanxious constitution and a weak ejaculatory system” [14].

Despite Schapiro’s ascribing equal importance to psychological and physiological components, behavioristic therapies dominated the third phase (1950–1990), with the biological component of PE advocated by Schapiro largely overlooked. This change of emphasis was largely prompted by the claims of success for the “squeeze” technique advocated by Masters and Johnson [15]. The perception of PE as learned behavior, with initial rapid intercourse leading to habituation and performance anxiety, gradually subsided as publications on the treatment of PE with psychoactive drugs began to appear. Such studies initiated the fourth phase (1990 to present), in which the efficacy of clomipramine and selective serotonin reuptake inhibitors (SSRIs) in the treatment of PE was established and recognized [16]. This recognition also led to the development and acceptance of theories of a neurological basis for PE involving dysfunctional central serotonergic neurotransmission.

#### *Current Knowledge of PE Etiology and Risk Factors*

Present-day theories of PE etiology focus on the combination and interaction of psychological and organic factors. Although, to date, no single etiological theory has universal acceptance, there is a general shift toward the acceptance of the condition as one in which psychologically mediated processes exacerbate an underlying organic component. The identification of a common cause of PE is nevertheless complicated by the fact that most researchers differentiate between two forms of PE: a primary (lifelong) and secondary (acquired) form, which may have distinct etiologies.

Psychological causes of PE may include sexual conditioning as described above and described by Masters and Johnson [15]. Anxiety too could also play a major role. Indeed, patients with PE often cite anxiety as a coexistent feature of the condition. As autonomic sympathetic nerves play a key role in ejaculation and anxiety increases the activity of the sympathetic nervous system, there is a plausible physiological connection between an increased sympathetic activity and a lowered ejaculatory threshold [17]. However, the evidence for a casual connection between male sexual problems and anxiety has not been proven [18,19], and some experts believe that anxiety is more likely to be a consequence of PE, rather than the cause [11].

Support for an organic etiology for PE originated largely with the discovery that PE has a genetic component. Schapiro first reported a familial predisposition to PE in 1943 [14]. More recently, Waldinger et al. showed that 10 of 14 first-degree relatives of men with lifelong PE also suffered PE, with an IELT of less than 1 minute [20]. The genetic component to PE, as well as the discovery that the condition is associated with disturbances in the central serotonin (5-HT) signaling system, led Waldinger to hypothesize that PE represented a point on a normally distributed population continuum of ejaculatory latency [21]. Therefore, the etiology of PE is based on a genetic predisposition and, although cognitive and emotional influences interact with these primary defects, the psychological disturbances associated with PE may be secondary phenomena [22]. Moreover, this genetic predisposition could underpin the other organic theories for the etiology of PE, which include penile hypersensitivity, a hyperexcitable ejaculatory reflex, and central 5-HT receptor sensitivity (Table 1).

Penile hypersensitivity has been proposed by many authors to be a component of the organic etiology of PE [23–26]. It has been postulated that men with penile hypersensitivity either reach the ejaculatory threshold more rapidly or have a lower threshold compared with men with normal ejaculatory latency. However, it is worth noting, by way of caveat, that this theory would not account for the existence of secondary PE and, moreover, other research has failed to support the hypothesis even in patients with primary PE [27,28].

Another theory suggests that PE is the result of a defective or hyperexcitable ejaculatory reflex leading to a faster emission and/or expulsion phase of ejaculation. The bulbocavernosus muscle surrounds the urethral bulb and is one of several key muscles involved in the expulsion phase of ejacu-

**Table 1** Organic theories for the etiology of premature ejaculation (PE)

Penile hypersensitivity [23–26]	<ul style="list-style-type: none"> <li>• Reach ejaculatory threshold more rapidly</li> <li>• Lower ejaculatory threshold</li> </ul>
Hyperexcitable ejaculatory reflex [23,29]	<ul style="list-style-type: none"> <li>• Faster emission and/or expulsion phase</li> <li>• Faster bulbocavernosus reflex</li> </ul>
Genetic predisposition [14,20]	<ul style="list-style-type: none"> <li>• Higher incidence of PE in first degree relatives</li> </ul>
Central 5-HT receptor sensitivity [21]	<ul style="list-style-type: none"> <li>• Lower 5-HT transmission in PE?</li> <li>• 5-HT<sub>2C</sub> receptor hyposensitivity?</li> <li>• 5-HT<sub>1A</sub> receptor hypersensitivity?</li> </ul>

lation. A study by Colpi et al. suggested that PE sufferers have a hyperexcitable bulbocavernosus reflex (BCR) [23]. Godpodinoff et al. showed that men with primary PE had a shorter BCR latency compared with men with either acquired PE or with normal ejaculatory function [29]. However, the study also suggested that men with acquired PE had a longer BCR latency than normal controls. Therefore, it is doubtful that a hyperexcitable BCR has a universal role in the etiology of PE [29].

Extensive data from animal studies have shown that serotonin receptors are pivotally involved in the central control of the ejaculatory sequence [21]. The 5-HT<sub>2C</sub> and 5-HT<sub>1A</sub> receptor subtypes appear to be particularly important: stimulation of 5-HT<sub>2C</sub> receptors in male rats has been shown to delay ejaculation, while activation of 5-HT<sub>1A</sub> receptors facilitates ejaculation [30]. The hypothesis of Waldinger and Olivier is predicated on the view that PE is caused by hypersensitivity of 5-HT<sub>1A</sub> and/or hyposensitivity of 5-HT<sub>2C</sub> receptors. From this perspective, PE is associated with a threshold IELT that is genetically "set" at a lower point, and is determined by the extent of imbalance between the 5-HT<sub>1A</sub>/5-HT<sub>2C</sub> systems [11,21]. Confirmation of this theory will depend on the development of agents that selectively target these receptor subtypes.

The clinical experience of practitioners suggests that, in at least some patients, PE may be multifactorial with a combination of organic causes. This conclusion is based on the fact that many patients are extremely difficult to treat and fail to respond to any existing medical therapies or behavioral interventions. Moreover, the disappointing success rate of current PE therapeutic strategies emphasizes the need for the development of more efficacious regimens.

Clearly, there are various psychological risk factors associated with PE. The most cited are such factors as sexual inexperience, infrequent sexual intercourse, and/or fear and anxiety [15]. It is also well established that PE is associated with relationship problems, and it is a logical correlate that any comprehensive management strategy for PE should aim to involve the sexual partner of the patient. A poor level of understanding of sexual responses may also exacerbate PE, and patient education may play an essential role in improving this situation. In many countries, the provision of sex education is suboptimal and many individuals miss the opportunity to benefit from lessons that cover all aspects of sexual relationships.

PE is also associated with a range of physiological comorbidities. Those in poorer general health are at a higher risk for all forms of sexual dysfunctions [31], and PE is associated with the presence of urinary tract infections [32] and with diabetes [33]. Ongoing patient pharmacotherapy can also predispose individuals to PE. Drugs such as opiates, trifluoperazine, and even over-the-counter cold remedies containing sympathomimetics have been associated with PE [34]. Furthermore, PE is often present in individuals with a history of drug abuse.

Probably the most therapeutically significant comorbidity, existing in up to 30% of men with PE, is ED [35]. However, individuals in whom these two conditions coexist form a separate population from the PE sufferer who is able to maintain a strong erection, and the two patient subtypes may require modifications to a common basic therapeutic strategy.

## Conclusions

Ascertaining the true prevalence of PE is difficult. This is not surprising considering the absence of a clear definition of normal, let alone premature, ejaculatory latency. Furthermore, there is no universally accepted clinical definition of PE, and validated measures of IELT and sexual satisfaction are lacking. However, it is clear that PE is highly prevalent and, globally, approximately 30% of all men across all age groups can be considered to have the condition. The sensitive provision of appropriate information will assure men that PE is a medical condition for which they should be seeking advice from a medical practitioner. This may be particularly important in facilitating an improvement in the quality of life not only of the patient, but also of the partner.

While the exact etiology of PE remains to be fully elucidated, it is increasingly clear that the disorder constitutes a neurophysiological condition that involves dysfunctional serotonergic neurotransmission in the central nervous system. As such, the most effective treatments for PE are likely to arise from a combination of pharmacological agents that act on this system and other interventions that reduce the impact of the psychological and physiological risk factors.

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