
NEW TRANS-THEORETICAL APPROACHES TO PREMATURE EJACULATION DISORDER

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Abstract

This paper explores the pathophysiology of chronic and acquired premature ejaculation. Through a detailed analysis of multiple studies and research findings, we present a comprehensive description of the factors associated with this condition, bringing forth the latest information from the scientific literature. Thus, we outline a detailed picture of the neurological, endocrine, urological, and psychological mechanisms involved in the development and persistence of premature ejaculation, highlighting the complex interactions between them. The theoretical approach of this paper provides a solid perspective on the progress in understanding premature ejaculation, contributing to the foundation of future research and the optimization of therapeutic intervention strategies. By synthesizing existing information, this work aims to be an essential guide for healthcare professionals and researchers involved in the study of premature ejaculation.

Key words: premature ejaculation, pathophysiology, sex disorders, chronic premature ejaculation, acquired premature ejaculation, risk factors, physiological mechanisms, psychological mechanisms

INTRODUCTION

Premature ejaculation (PE) is regarded as one of the most common male sexual disorders with profound implications on both quality of life and interpersonal relationships (Metz & McCarthy, 2003). In an effort to synthesize advances in the current understanding of the pathophysiology of premature ejaculation, this paper focuses on its main subtypes, specifically the chronic and acquired forms (Jannini et al., 2013). With a perspective grounded in scientific literature, we investigate the organic and psychogenic mechanisms that underlie this sexual dysfunction, thus developing a comprehensive description of the risk factors associated with chronic and acquired forms of premature ejaculation. This theoretical

analysis not only illustrates the progress in understanding the pathophysiology but also outlines possibilities for future research and therapeutic intervention strategies. Through a comprehensive approach to the subject, this work aims to be an essential tool for healthcare professionals and researchers interested in expanding the knowledge in this complex field of male sexual health.

Historical development of premature ejaculation definition and classification

In the 1960s, Masters and Johnson defined premature ejaculation as the condition where in a man ejaculates before his partner achieves orgasm in over 50% of his sexual interactions (Masters & Johnson, 1970). Over time, defini-

tions have evolved, shifting the focus from the partner's orgasm to other aspects deemed crucial for this dysfunction: duration of penetration, frequency of occurrence, individual control over ejaculation, and the degree of individual and/or relational impairment. Presently, according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), premature ejaculation is defined as a persistent or recurrent pattern of ejaculation that occurs during sexual activity with a partner approximately 1 minute after vaginal penetration and before the individual desires it (APA, 2013). A major contribution in the field of premature ejaculation was Bernard Schapiro's distinction between premature ejaculation types A and B (Schapiro, 1943), later renamed as primary and secondary premature ejaculation (Godbodino, 1989). Despite this distinction, all DSM versions have maintained a single, general definition of premature ejaculation. This persistence in retaining a unified definition is unfortunate but understandable since, for decades, the clinical characteristics of the two subtypes had not been systematically investigated. However, the situation changed with the introduction of selective serotonin reuptake inhibitors (SSRIs) in the early 1990s. Consequently, data leading to the new definition of primary premature ejaculation emerged from SSRIs' clinical studies in the mid-1990s (McMahon et al., 2008). Regarding the definition of secondary or acquired premature ejaculation, it remains an ongoing process. While defining premature ejaculation based on intravaginal ejaculation latency time (IELT) was relatively straightforward, objective measurement parameters for secondary premature ejaculation have not been identified to date. Thus, presently, this subtype of ejaculation can be identified by its occurrence after a period of satisfactory ejaculatory control (Jannini et al., 2013).

Intravaginal ejaculation latency time (IELT) as an objective parameter in defining premature ejaculation

In order to establish an evidence-based operational definition of premature ejaculation, intravaginal ejaculation latency time

(IELT) was introduced as an objective clinical measure. IELT represents the time elapsed from intromission to intravaginal ejaculation (Waldinger et al., 1994). Waldinger also proposed the use of masturbatory ejaculation latency time (MELT), oral ejaculation latency time (OELT), and anal ejaculation latency time (AELT) as tools for assessing ejaculatory performance in both homosexual and heterosexual men (Waldinger, 2007). However, to date, only IELT has been utilized in premature ejaculation research involving heterosexual men.

Clinical studies indicate that the majority of men suffering from primary premature ejaculation ejaculate within one minute of penetration (McMahon, 2002). Regarding men with secondary premature ejaculation, we currently lack objective IELT-related data, though there is no reason to suspect notable differences in intravaginal latency compared to the primary subtype (Jannini et al., 2013).

In addition to this new definition of primary premature ejaculation, Waldinger et al. proposed a new classification based on IELT duration (Table 1). In this classification, there are four subtypes of premature ejaculation. Alongside primary and secondary, based on clinical and epidemiological stopwatch data, Waldinger postulated the existence of two additional subtypes: normal (variable) premature ejaculation and subjective premature ejaculation (Waldinger & Schweitzer, 2008).

Men suffering from primary premature ejaculation report consistently shorter IELTs of about one minute in the majority of sexual interactions, starting from puberty or adolescence. In the case of men suffering from secondary or acquired premature ejaculation, the etiology may be organic - thyroid disorders, inflammatory prostatitis - or psychogenic, namely individual or relational issues (Serefoglu et al., 2010). Variable or normal premature ejaculation means that men may occasionally experience very short IELTs whereas in subjective premature ejaculation, men have a normal or even higher than normal IELT, yet still perceive themselves as experiencing premature ejaculation. While very short IELT values in men with primary premature ejaculation are assumed

Table 1. The new classification of the four subtypes of premature ejaculation and their characteristics based on intravaginal ejaculation latency time (Waldinger & Schweitzer, 2008)

Primary PE	Acquired PE	Normal/variable PE	Subjective PE
Rapid ejaculation occurs in almost every sexual encounter with almost every partner. Since the onset of sexual activity. In 80% of cases men ejaculate within 1 minute (generally within 30 seconds). In 20% of cases between 1-2 minutes. PE is permanent.	PE emerges at some point in a man's life. Generally occurs at a later age, after a period of normal ejaculatory function. Ejaculation takes place within 1-2 minutes as a result of urological, neurological, endocrine and psychological problems.	Inconsistent and irregular rapid ejaculation pattern. Present from the onset of sexual activity. IELT can be short or normal, with a weak or absent ability to control ejaculation time. It represents the normal and natural variability of ejaculatory function.	The subjective perception of rapid ejaculations. Diminished or absent ability to delay ejaculation. Present from the onset of sexual activity or it occurs later in life. IELT is normal or even longer. PE here is subjective, not an actual sexual dysfunction.

to result from neurobiological processes and genetic factors, subjective premature ejaculation is believed to be strongly associated with psychological and cultural factors. In this case, the intravaginal ejaculation latency is time normal or higher than normal, however the perception of their sexual performance is distorted. Nevertheless, there is currently no general consensus on this new classification of the four subtypes of premature ejaculation (Waldinger & Schweitzer, 2008). In clinical practice, the adoption of these subtypes provides a valuable framework that goes beyond the traditional understanding of premature ejaculation. By recognizing the nuanced variations in its presentation, healthcare practitioners gain a more comprehensive understanding of PE. This heightened awareness, in turn, facilitates a more efficient and tailored approach to treatment. This classification enables practitioners to tailor interventions, whether they involve sex therapy, pharmacotherapy, psychoeducation, or psychotherapy, based on the specific subtype and underlying factors associated with an individual's premature ejaculation (Metz & McCarthy, 2003).

Algorithm for the diagnosis of premature ejaculation

The diagnostic algorithm (Figure 1) for premature ejaculation involves a thorough assessment of the patient's medical history and sexual behaviors. The diagnosis begins with an open discussion about the patient's symptoms

and sexual experiences. Factors such as the frequency of premature ejaculations, overall medical history, any associated sexual disorders, and recent psychological or stressful events are analyzed. Physical examinations and laboratory tests may also be conducted to exclude possible organic causes. In the case of primary premature ejaculation, occurring from the onset of sexual activity, and secondary premature ejaculation, which develops later in life, differential diagnosis will help identify underlying factors such as hormonal disorders, other sexual disorders such as erectile dysfunctions, or psychological issues. Through this algorithm, a comprehensive and personalized assessment can be made in order to guide the patient toward an appropriate treatment plan.

Pathophysiology Of Primary Premature Ejaculation

The pathophysiology of primary premature ejaculation explores the physiological aspects underlying this sexual dysfunction, focusing on neurobiological processes and genetic factors that may contribute to the early onset of ejaculation in the context of a man's sexual life. McMahon defines primary premature ejaculation as a male sexual dysfunction characterized by ejaculation that consistently or almost always occurs before or within approximately one minute of vaginal penetration. This is coupled with the inability to delay ejaculation in all or almost all vaginal penetrations, resulting in negative personal consequences

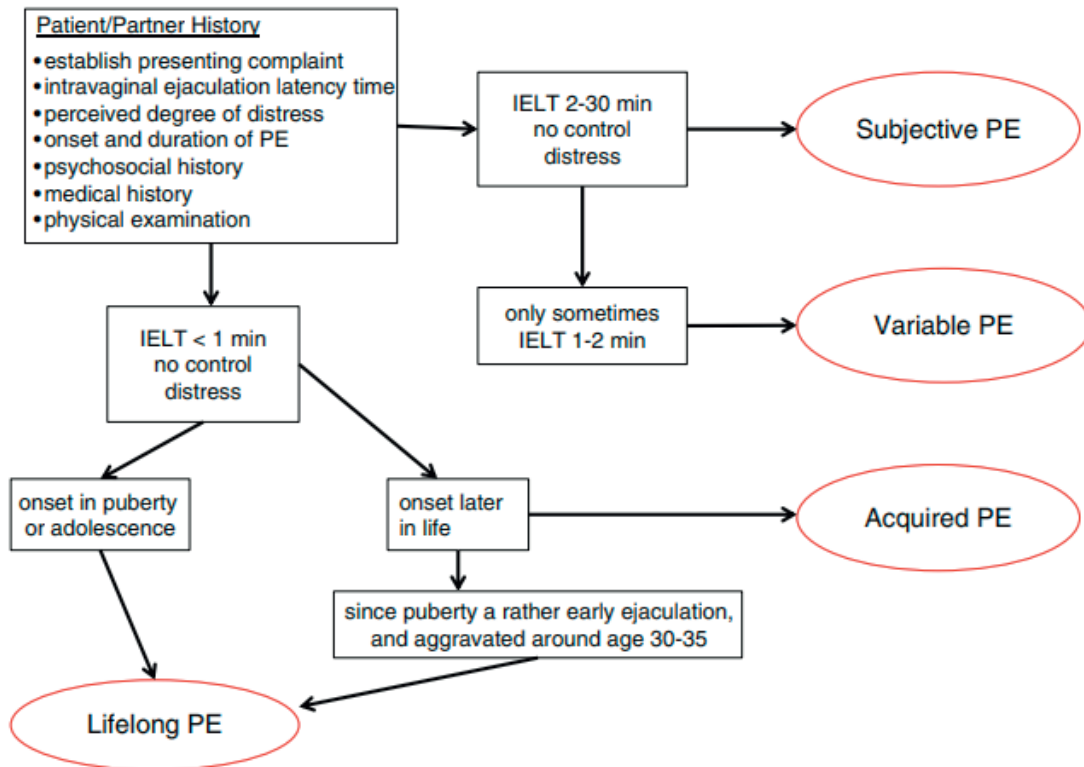


Figure 1. Algorithm for the diagnosis of premature ejaculation
(Waldinger & Schweitzer, 2006; Waldinger & Schweitzer, 2008; Waldinger, 2008)

such as discomfort, disturbance, frustration, and/or avoidance of sexual intimacy (McMahon et al., 2008). Concerning its prevalence, research suggests that around 2.5% of men in the general population experience primary premature ejaculation (Waldinger et al., 2005). This statistic underscores the significant but relatively limited occurrence of this subtype among men at large.

Neurobiology and genetic polymorphism in primary premature ejaculation

Based on the scientific literature, it is speculated that the variability of intravaginal ejaculation latency time (IELT) in primary premature ejaculation is caused by neurobiological and genetic factors. Rapid ejaculation is associated with low serotonin (5-hydroxytryptamine or 5-HT) levels, hypersensitivity of 5-HT_{1A} receptors, and/or suboptimal functioning of 5-HT_{2C} receptors (Waldinger et al., 1998). Thus, from a neurobiological standpoint, primary PE may be linked to dysfunction of 5-HT_{1A} and

5-HT_{2C} receptors in specific brain regions involved in ejaculation (Waldinger et al., 1998). Genetically, there is preliminary evidence that short IELTs in men with primary PE may be due to genetic polymorphism in serotonergic transmission (Jannini et al., 2013). It is essential to emphasize that a characteristic of primary premature ejaculation is a man's inability to control the duration of his intercourse, namely his IELT. Regardless of his actions, techniques, or thoughts, his intravaginal ejaculation latency time is almost always a matter of a few seconds. These men are permanently affected, experiencing this condition throughout their entire lives, without the capacity to change it. At the moment, the precise etiology of primary premature ejaculation remains unknown (Jannini et al., 2013).

Regarding the hereditary aspect, genetic research has revealed that most human traits are, to some extent, genetically influenced (Schapiro, 1943). Recently, increased attention has been given to investigating the genetic eti-

ology of ejaculatory disorders. Studies have observed that premature ejaculation appears to run in families (McMahon et al., 2008), with an increased likelihood of PE occurrence among relatives of men with PE (McMahon et al., 2008; Waldinger, 1994). Furthermore, a 2009 study on the Dutch population indicates that the prevalence of LL, SL, and SS genotypes in primary PE is comparable to the normal general population (Janssen et al., 2009). However, men with the LL genotype ejaculated 100% faster ($p < 0.027$) than men with the SS and SL genotypes (Janssen et al., 2009). This finding aligns with pharmacological knowledge, suggesting that reduced serotonergic neurotransmission facilitates premature ejaculation (Jannini et al., 2013).

Serotonergic modulation of the spinal ejaculatory reflex

Evidence derived from animal studies indicates a prominent role of the central serotonergic system in adjusting the spinal ejaculatory reflex (Truitt & Coolen, 2002). This modulation of the reflex can result in either premature or delayed ejaculation, with the actual act being influenced not only by the serotonergic system but also by other neurotransmitter systems situated within the spinal cord (Waldinger, 2011). Despite being a crucial aspect of the central serotonergic system, this modulation is rarely explored in scientific literature, although it holds vital importance for the genetic research of primary premature ejaculation. The modulation of ejaculation in men may vary, for example. It can be strong or weak. In cases of absence, the serotonergic situated in the brainstem might fail to regulate the ejaculatory reflex in the lower spinal cord, hindering a man's ability to alter his IELT. Even with SSRI pharmacotherapy, individuals in this category may still not be able to change their intravaginal ejaculation latency time. Even though it has not been systematically investigated, it is clinically well known that a subset of men shows no IELT improvement on any SSRI treatment (Waldinger, 2011). We can speculate, therefore, that for these men, the serotonergic system may be unable to affect the ejaculation reflex (Waldinger, 2011). The prem-

ise that serotonin has the ability to modulate ejaculation can have significant implications for genetic and pharmacological research, suggesting that a certain group of men may lack the ability to modulate ejaculation, regardless of the presence or absence of the functional serotonergic polymorphisms (Waldinger, 2011). Therefore, these men will not show any change in the duration of IELT if the modulation of ejaculatory latency is not entirely associated with such serotonergic polymorphisms.

Clinical profile of primary premature ejaculation

Conceived as a clinical syndrome, primary premature ejaculation is identified by a set of distinctive features (Waldinger, 2007). The symptomatic presentation includes:

1. Premature ejaculation is present from the first or nearly the first sexual encounters.
2. It occurs with almost every female partner in more than 80-90% of sexual encounters.
3. The intravaginal ejaculation latency time remains relatively constant as individuals age or worsens in 25-30% of patients around the ages of 30-35.
4. Premature ejaculation occurs within the range of 30-60 seconds after vaginal penetration in almost every sexual encounter for most affected men. For 10% of men with primary PE ejaculation occurs within 1-2 minutes.

Pathophysiology Of Acquired Premature Ejaculation

The pathophysiology of acquired premature ejaculation explores the organic and psychological changes associated with the onset of this sexual dysfunction. Unlike its primary counterpart, this form of premature ejaculation develops over time and can be influenced by factors such as stress, relational issues, or health problems. The scientific community lacks consensus on a specific definition for acquired premature ejaculation due to the absence of objective clinical data regarding Intravaginal Ejaculation Latency Time (IELT) (Jannini et al., 2013). Nevertheless, this subtype of ejaculation

is identified by its occurrence at some point, emerging after a period of normal functioning. The clear implication here is that primary premature ejaculation is likely inherent, whereas the acquired form typically stems from an organic and/or psychogenic origin, impacting the complex mechanism of ejaculation. Once acquired, this subtype of PE does not necessarily manifest in every sexual encounter, nor does it have an objectively established IELT. However, there is no reason to suspect a notable difference in IELT values compared to primary PE (Jannini et al., 2013).

In terms of prevalence, numerous studies (McMahon et al., 2008; Waldinger, 2007; Waldinger, 1998) estimate that approximately 20-30% of the male population may be affected by acquired premature ejaculation at some point in their lives. However, it is crucial to consider the methodological limitations of these studies when evaluating the quality of information. While the prevalence of premature ejaculation has been repeatedly reported around 20-30% of the male population, these percentages indicate the percentage of men dissatisfied with their own ejaculatory latency, not the percentage of clinically diagnosed men with PE. Only 8-10% of the male population is actually diagnosed with either primary or acquired PE (Jannini et al., 2013).

Organic risk factors

Attempts to explain the etiology of premature ejaculation (EP) involve a diverse range of organic and psychological factors. The organic causes of premature ejaculation are often categorized into genetic, neurological, endocrine, and urological subsets (Godpodinoff, 1989). Organic risk factors can significantly influence ejaculation, thus, these risk factors can contribute to the etiology of this sexual dysfunction, either catalyzing its onset, maintaining it, or worsening it. Understanding how these risk factors affect the functioning of the nervous system and ejaculatory reflexes is essential for identifying treatment and intervention options for affected patients. By exploring the connection between organic risk factors and sexual dysfunction, we can develop more per-

sonalized and effective therapeutic approaches that improve the quality of life for patients and contribute to the prevention or management of acquired EP.

Neurological risk factors

Neurological pathologies can exert a significant impact on the functioning of the ejaculatory system. Traumatic brain injuries, cerebrovascular diseases, Parkinson's disease, and epilepsy are critical factors that can influence the neural circuit responsible for ejaculatory control. These pathologies can disrupt the delicate balance between excitation and inhibition at the cerebral and spinal levels, thus contributing to the development of acquired premature ejaculation.

Premature ejaculation is not uncommon in populations with brain pathologies. Studies on sexual dysfunctions arising from these pathologies have identified that 17–36% of men report various post-traumatic ejaculatory issues, including premature ejaculation (Serefoglu et al., 2011; Waldinger, 2006), with an incidence of 9% in PE (Waldinger, 2002). Cerebrovascular accidents can affect sexuality in several ways, including reducing libido, causing erectile and ejaculatory dysfunction, and decreasing the frequency of sexual activity (Balercia et al., 2007; Cohen, 1997; Pirke et al., 1979). Issues with ejaculation are common in men after having suffered a stroke. While most men were able to ejaculate before a stroke, only 29% retained proper ejaculatory function afterward (Morelli et al., 2004), and similar findings have been reported by other authors (Corona et al., 2008).

Sexual dysfunction is common even in men diagnosed with Parkinson's disease (Keleta et al., 2007; Mancina et al., 2005). In one study (Mancina et al., 2005), 40.6% of Parkinson's patients were diagnosed with premature ejaculation, while another research identified PE in 8% of patients (Carosa et al., 2002). The mechanism behind PE in Parkinson's disease is not well understood at the present moment.

Another risk factor for the ejaculatory function is epilepsy. It is estimated that between 38–71% of men who suffer from epilepsy expe-

rience various sexual issues (Bilezikian & Loeb, 1983; Williams et al., 1977; Polikar et al., 1990). These issues include different types of sexual impairment such as erectile dysfunction, premature ejaculation, orgasmic dysfunction, and decreased sexual desire. The prevalence of PE among those with epilepsy varies between 2 and 66.7% (Jannini et al., 2000; Jannini et al., 2005; Corona et al., 2006).

Penile hypersensitivity is another risk factor in PE, although there is an ongoing debate in the scientific community. The hypothesis suggesting penile hypersensitivity as a potential risk factor for PE is grounded in the scientific literature. Studies have shown that the genital amplitudes of somatosensory-evoked potentials (SEPs) in the cortical area were significantly more elevated in men experiencing PE compared to those with normal ejaculatory function (Fanciullacci et al., 1988; Xin et al., 1997). This suggests a larger cortical representation of sensory stimuli from the penis, indicating that the ejaculation center may play a role in PE. Another study supports the hypothesis of penile hypersensitivity (Xin et al., 1996). This study further strengthens the notion that heightened penile sensitivity could play a significant role in the occurrence of premature ejaculation (PE). Another element that support this theory is the use of desensitizing agents in PE patients, such as topical sprays with lidocaine-based anesthetic agents. The use of these numbing agents is associated with improved IELT, significantly increasing the time to ejaculation (Choi et al., 1999; Busato & Galindo, 2004; Dinsmore & Wyllie, 2009), adding further weight on the hypersensitivity theory. On the other hand, certain studies reject the role of hypersensitivity as a risk factor in PE. For example, Perretti and his colleagues failed to demonstrate faster nerve conduction along the pudendal sensory pathway or larger cortical representation of sensory stimuli from the genital area in patients with PE (Perretti et al., 2003).

Endocrine risk factors

While it is widely accepted that male reproduction is hormonally regulated (Bhasin

& Basson, 2008), research on the endocrine control of the ejaculatory process and intravaginal latency time is still in its early stages. A recent proposition suggests that gonadal hormones, along with thyroid and pituitary hormones (oxytocin and prolactin), play a role in regulating the ejaculatory process and its latency to varying extents. However, the current body of evidence is limited, with only a few clinical studies available (Corona et al., 2004; Carani et al., 2005; Althof et al., 2010; Jannini et al., 2009). The correlations between erectile dysfunction (ED), premature ejaculation (PE), hypothyroidism, and hyperthyroidism have been extensively documented, even in animal studies (Corona et al., 2004; Cihan et al., 2009; Carani et al., 2005). Hyperthyroidism induced in rats leads to an increased frequency of seminal vesicle contractions and contractile activity of the bulbospongiosus muscle, suggesting that hyperthyroidism affects both the emission and expulsion phases of the ejaculation process. Similar results have been obtained in several clinical studies. In a consecutive series of 755 men presenting with sexual dysfunctions, a prevalence of hyperthyroidism was found to be twice as high among men with premature ejaculation (Corona et al., 2004). Carani and colleagues demonstrated that 50% of patients with hyperthyroidism also suffer from PE, a prevalence that was significantly reduced (15%) by treating the underlying thyroid disease, with a doubling of ejaculatory latency upon remission (Carani et al., 2005). Thus, based on these studies, we can conclude that hyperthyroidism should be considered a new and reversible etiological factor in premature ejaculation, regardless of subtype.

Urological risk factors

Several studies have identified a number of urological risk factors that may predispose a man to acquired premature ejaculation (Chia, 2002; Laumann et al., 2005; Porst et al., 2007; Jannini et al., 2005). The main risk factors include prostate pathologies, chronic pelvic pain syndrome (Liang et al., 2004; Screponi et al., 2001; Sonmez et al., 2010; Zohdy, 2009), and varicocele (Jarow, 2001) (Lotti et al., 2009).

Two of the most common urogenital conditions that can significantly impact a man's overall quality of life are chronic prostatitis (CP) and chronic pelvic pain syndrome (CPPS) (Bartoletti et al., 2007; Davis et al., 2009). These conditions involve urogenital pain, pain and/or discomfort during ejaculation, urinary dysfunction, and, of course, sexual dysfunction. Numerous studies on patients with prostatitis or chronic pelvic pain syndrome (CPPS) have found that rapid ejaculation is a frequent complaint among these patients, occurring in 26,2-77,3% of cases (Liang et al., 2004; Liang et al., 2010; Nickel et al., 2001; Gonen et al., 2005). In the specialized literature, chronic prostatitis is closely linked to premature ejaculation (Liang et al., 2004; Screponi et al., 2001; Shamloul & el-Nashaar, 2006). While these pathologies often coexist, a causal relationship has not been established to date. Screponi and colleagues were among the first to ascertain the prevalence of chronic prostatitis in men with PE (Screponi et al., 2001). In their study, a cohort of 46 men with premature ejaculation was compared to a control group of 30 healthy men of similar ages. The frequency of CP in the group of patients was notably greater compared to the control group. In a comparable study, Shamloul and colleagues aimed to validate these findings using a larger cohort comprising 153 men with PE (94 with primary PE and 59 with acquired PE) and found that 63.3% had prostatitis based on urine analysis and prostatic secretion examination (Shamloul & el-Nashaar, 2006). A notable aspect is that only 12.4% of PE patients showed signs of prostate infection. The research also revealed a considerably elevated prevalence of CP in men experiencing acquired PE (91.5%) in contrast to those with primary PE (45.7%). Moreover, the literature strongly supports the beneficial effect of antibiotic treatment on improving ejaculatory function (Shamloul & el-Nashaar, 2006). Together, these findings strongly support the idea that chronic prostatitis (CP) can be a common cause of acquired premature ejaculation, which should be ruled out, especially in men with associated pelvic pain and/or concurrent urinary symptoms.

Another urological risk factor in acquired PE is the presence of varicocele, namely the abnormal dilation of veins in the pampiniform plexus due to retrograde venous flow, significantly impacting sexual function. Varicoceles are prevalent in urology, with estimated occurrences ranging from 15% in the general population to 35% in men facing primary fertility challenges (Jarow, 2001). The potential link between varicocele and acquired premature ejaculation (PE) has been recently proposed as an etiological factor. In a cross-sectional study conducted in Italy, Lotti et al. investigated 2,448 patients with sexual dysfunction to determine the prevalence of varicocele (Lotti et al., 2009). Their comparison of individuals with varicocele to those without revealed a significant difference in PE status (29.2% vs. 24.9%, respectively) after adjusting for factors like age, anxiety levels, and prolactin levels. The researchers established a correlation between the severity of varicocele observed in Doppler ultrasound analysis and seminal levels of interleukin-8, serving as a surrogate marker for non-bacterial prostatitis. These findings led to the hypothesis that PE could manifest as a clinical symptom of an underlying inflammatory condition triggered by varicocele and/or prostatitis. Consequently, the presence of varicocele has been associated with elevated inflammation levels in the pelvic region.

Psychological risk factors

Unlike organic risk factors, the psychological influence in the manifestation and persistence of premature ejaculation is often underestimated, although it holds significant importance. Performance-related anxiety, early sexual experiences, frequency of sexual acts, and relationship issues are just a few psychological components that can contribute to the onset or maintenance of premature ejaculation. By exploring the complex relationships between mental and behavioral aspects, we gain a comprehensive perspective on psychogenic factors that can affect ejaculation and the quality of our sexual encounters (Rowland, 1999; Rowland et al., 2000). The inquiry into whether men experiencing premature ejaculation dif-

fer fundamentally from other men has been a longstanding point of interest. Initial research proposed that these men were narcissistic and apathetic toward their partners (Masters & Johnson, 1970), a notion that has found little to no backing in recent studies (Rowland et al., 2004).

Anxiety, a central construct in various psychological disorders, has been suggested as a pivotal element in psychogenic sexual dysfunctions, including premature ejaculation. Recent studies have identified depression, anxiety, and neuroticism as key dimensions that distinguish men with sexual dysfunctions from control subjects (Quinta Gomes & Nobre, 2011; Rowland et al., 2011; Costa et al., 1992). Concerning premature ejaculation, although the research is not as extensive, a similar pattern appears to be emerging. Men with premature ejaculation, on average, report higher levels of anxiety and depression than the general population (Munjak et al., 1978; Corona et al., 2004).

Additional research indicates that men with premature ejaculation approach psychosexual stimuli with an overall more negative affect than control subjects. Specifically, men with premature ejaculation report elevated levels of embarrassment/guilt and worry/tension (Rowland et al., 2003). The psychological burden on men with premature ejaculation is corroborated by several studies. These men report increased levels of personal distress and interpersonal difficulties, a less positive self-image, and diminished quality of life compared to control subjects (McCabe, 1997; Rowland et al., 2007; Patrick et al., 2005; Rosen & Althof, 2008). Moreover, men with premature ejaculation exhibit higher levels of general emotional stress, feelings of inadequacy, self-esteem challenges, and disappointment compared to control subjects (Rowland et al., 2007; Revicki et al., 2008; Tondo et al., 1991). Additionally, Hartmann and his colleagues characterized men with premature ejaculation as preoccupied with thoughts related to orgasm control, anxiously anticipating possible failure, experiencing thoughts of shame, and concerning themselves with maintaining their erection (Hartmann et al., 2005).

Intrapsychic risk factors

In the comprehensive exploration of risk factors associated with premature ejaculation, our attention is also directed towards the intrapsychic domain, delving into the influences and interactions within an individual's mental sphere. These intrapsychic factors are particularly relevant in understanding the etiology and persistence of premature ejaculation, highlighting aspects related to emotions, anxiety, performance fears, and other elements within the emotional and cognitive realm that can play a significant role in the dynamics of this sexual dysfunction. Understanding the relationship between emotional states and premature ejaculation is pivotal in comprehending this condition. The affective dimension of sexual response, particularly anxiety, has long been suspected to play a part in premature ejaculation (Kaplan, 1974; Masters & Johnson, 1970; Barlow, 1986; Strassberg et al., 1990). Despite its commonly perceived association with a general inhibition of erectile response, anxiety might, in reality, have the effect of heightening arousal (Beck & Barlow, 1986; Rowland et al., 2011). Therefore, the interconnections among anxiety, emotional and sexual arousal, and the ejaculatory threshold (the point of ejaculatory inevitability) can serve as crucial elements in understanding and addressing PE. Another study theorizes that excessive anxiety related to sexual performance may divert men's attention from the prodromal sensations preceding ejaculatory inevitability (Kockott et al., 1980; Vandereycken, 1986) making it more challenging for them to control their ejaculation. In this context, specific affective states related to the dysfunctional sexual response may either be part of the dysfunction or represent a reaction to the failed ejaculatory control, subsequently intensifying the issue (Bancroft, 1989).

Psychophysiological research offers deeper insights into the impact of emotions on sexual responses. In comparison to men with normal sexual function, those experiencing sexual dysfunctions exhibit heightened levels of negative emotions and reduced levels of positive emotions when exposed to erotic stimuli (Rowland

et al., 2011; Rowland & Heiman, 1995; Rowland et al., 1996). Moreover, individuals with premature ejaculation differ from individuals without PE on specific positive and negative emotional dimensions during visual sexual stimulation (Rowland et al., 2003), as well as in their emotional responses to pharmacotherapy. Confirming earlier studies, men with premature ejaculation reported elevated levels of negative emotions, including shame/guilt, stress/worry, and anger/irritation, both before and during visual sexual stimulation compared to the control group. While certain negative emotions decreased in men with PE who exhibited improved Intravaginal Ejaculation Latency Time (IELT) through clomipramine treatment, levels of guilt/shame and anger/irritation remained elevated when compared to the control group (Rowland et al., 2003). The fact that effective pharmacotherapeutic treatment of PE leads to improved overall emotional well-being, indicates that the initially lower positive affect in men with PE is a result of their sexual dysfunction rather than an inherent negative personality trait. Higher levels of sexual functioning correspond to greater positive affect. Conversely, the persistence of elevated negative emotions in men with PE, even among those benefiting from pharmacotherapy, suggests that increased negative affect may contribute to the perpetuation (and possibly the origin) of the dysfunctional response. Additional research is necessary to explore whether specific negative emotions linked to sexual dysfunction might resist the positive effects of ejaculation-delaying medications. There is a possibility that certain negative emotional states are more dispositional than response-related to the unsuccessful sexual response in men with PE, a concept with limited support in the scientific literature (Cooper et al., 1993; Tondo et al., 1991).

Within the intrapsychic domain, psychosexual skills deficiency represents another risk factor for PE. In the context of premature ejaculation, this deficiency refers to the absence of basic sensory skills in managing the body during sexual arousal (Metz & Pryor, 2000). Some men may experience lifelong premature

ejaculation due to the limited development of these skills. These individuals appear to lack skills related to courtship and romantic relationships, as well as knowledge and specific abilities concerning physiology and sexuality. Men with premature ejaculation encounter challenges in focusing on their own sensations, contend with anticipatory anxiety, struggle with body relaxation, fail to engage their pubococcygeal muscles in controlling ejaculation, and tend to be hyper focused on their partner's body (Metz & Pryor, 2000). Additionally, these men experience cognitive distortions related to sexual performance and harbor negative core beliefs about themselves, significantly affecting their cognitive-behavioral involvement during sexual activity, thus contributing to sexual dysfunction (McCarthy, 1988; Zilbergeld, 1992).

Relational risk factors

The quality of romantic and/or sexual relationships is generally negatively impacted by PE (Rosen & Althof, 2008). Both single men and those in relationships, along with their partners, experience adverse effects. However, it is important to note that relational conflict can also act as a catalyst for premature ejaculation or at least act as an aggravating factor. The influence of premature ejaculation on the quality of life varies greatly depending on the individual affected - either the man with PE or his partner. A qualitative study (Symonds et al., 2004) involving 28 men with PE, aged 25 to 70, unveiled the repercussions of sexual dysfunction on self-image, sexual life, partner relationships, and daily living. Around 50% of men avoid romantic relationships. Those who are already in a romantic relationship experienced distress due to the inability to satisfy their partners, while others were concerned about potential partner infidelity stemming from sexual dissatisfaction. Additionally, a majority of men (68%) reported diminished confidence in their sexual abilities. Worries about controlling ejaculation, anxious anticipation of possible failure, feelings of shame, and maintaining an erection after ejaculation are integral aspects of the subjective experience for men with PE, as indicated by another study (Hartmann et al.,

2005). Moreover, in contrast to men with PE who harbor negative thoughts about sex, men without sexual dysfunctions tend to emphasize arousal and sexual satisfaction, focusing on the positive aspects of sexuality.

Research on men dealing with premature ejaculation (PE) and their partners consistently reveals a link between PE and negative psychosocial and quality of life outcomes, including adverse effects on the relationship itself (Rosen & Althof, 2008). One particular study compared men with PE and their partners to men without PE and their partners (Rowland et al., 2007). The group comprising men with PE and their partners exhibited lower levels of sexual functioning, diminished sexual satisfaction, heightened personal distress, and more pronounced interpersonal difficulties compared to men without PE and their partners. Moreover, men with PE generally reported a lower quality of life than their counterparts without PE. This highlights the substantial psychological strain that premature ejaculation imposes on affected men, their partners, and the overall relationship. Additionally, Shabsigh and Perelman note that 55% of individuals without PE expressed being “very” or “extremely” satisfied with their quality of life, whereas only 38% of those with PE shared the same sentiment. When asked about the enjoyment of their most recent sexual encounter, 78% of individuals without PE described recent sexual experiences as “very” or “extremely” enjoyable, whereas only 54% of those with PE felt the same way (Shabsigh & Perelman, 2005). In a study on male and female sexual dysfunction, McCabe and his colleagues reveal that men with PE experience the most significant deficits in quality of life (McCabe, 1997). Using validated scales to assess various facets of intimacy (emotional, social, sexual, recreational, and intellectual) and satisfaction, they found that men who experience premature ejaculation tend to have lower scores compared to their counterparts without this issue.

Another significant association observed in men with premature ejaculation is related to sexual dysfunction in women. A 2005 study revealed a high incidence of sexual dysfunction

among the partners of men with PE (Riley & Riley, 2005). Specifically, 22% of women presenting with sexual desire disorder, 29% of women experiencing lubrication problems during arousal, 43% of women with anorgasmia, and 48% of women who did not enjoy sex had partners suffering from premature ejaculation. The treatment of PE using clomipramine not only led to a substantial delay in ejaculation but also enhanced sexual satisfaction in couples and facilitated orgasm during sexual intercourse for women (Althof et al., 1995). Another study delved deeper into the experiences of partners of men with and without PE (Hobbs et al., 2008). Hobbs and colleagues administered multiple questionnaires and inventories to 138 women partners of men with PE and 89 women whose partners did not have PE. The findings revealed that 78% of partners of men with PE experienced at least one sexual dysfunction, in contrast to 42% of partners of men without PE. Among partners of men with PE, 55% reported arousal-related issues, while 52% reported orgasm-related problems. They also scored significantly lower than partners of men without PE across all measures, indicating lower life satisfaction, a less fulfilling sexual relationship with their partners, reduced sexual satisfaction, and more difficulties related to desire, arousal, and orgasm than partners of men without PE (Hobbs et al., 2008).

CONCLUSION

This paper represents an effort to explore the pathophysiology of both primary and acquired premature ejaculation. Through a detailed analysis of numerous relevant studies and research findings, we have managed to outline the main risk factors associated with this condition, highlighting the complexity of neurological, endocrine, urological, and psychological interactions involved in the development and persistence of premature ejaculation. By synthesizing existing information, this paper aims to be a significant reference point in the field of premature ejaculation, facilitating a deeper understanding and providing a solid

foundation for future research and treatment strategies.

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