Considerations for a Better Definition of Male Orgasmic Disorder in DSM V

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DOI: 10.1111/j.1743-6109.2009.01683.x

ABSTRACT

Introduction. All of the Diagnostic and Statistical Manual of Mental Disorders, 4th Ed., text revision (DSM-IV-TR) criteria for sexual disorders have been criticized on multiple grounds, including that the criteria lack precision, that the requirement of marked distress is inappropriate, and that the specification of etiological subtypes should be deleted. Aim. The goal of this article is to review evidence relevant to diagnostic criteria for male orgasmic disorder published since 1990. Methods. Medline searches from 1990 forward were conducted using the terms male orgasmic disorder, anorgasmia, delayed ejaculation, retarded ejaculation, ejaculatory delay, and ejaculatory disorder. Early drafts of proposed alterations in diagnostic criteria were submitted to advisors. Main Outcome Measure. Evidence reviewed was judged by current usage of terminology, evidence allowing precise definition of the syndrome, and evidence concerning separation of the syndrome from distress. Results. The literature search indicated minimal use of the term male orgasmic disorder and minimal knowledge concerning psychogenic ejaculatory problems. Conclusions. It is recommended that the term male orgasmic disorder be replaced with the term delayed ejaculation. Duration and severity criteria are recommended. Since many ejaculatory problems are idiopathic, it is recommended that the etiological subtypes due to psychological or due to combined factors be eliminated. Segraves RT. Considerations for a better definition of male orgasmic disorder in DSM V. J Sex Med 2010;7:690–699.

Key Words. Orgasm; Ejaculation; Orgasmic Disorder; Delayed Ejaculation

The criterion for the psychiatric diagnosis of male orgasmic disorder in DSM IV TR is a persistent or recurrent delay in, or absence of, orgasm, following a normal sexual excitement phase. The clinicians should take into account the patient’s age and whether the stimulation is adequate in focus, intensity, and duration. The disturbance must cause marked distress or interpersonal difficulty. The orgasmic dysfunction is not better accounted for by another axis I disorder (except another sexual dysfunction) and is not due to the direct physiological effect of a substance (including medication) or to a general medical condition [1]. It should be noted that DSM IV TR specifically excludes ejaculatory difficulties of known organic etiologies from being diagnosed as a psychiatric disorder.

These diagnostic criteria have remained fairly constant in the various editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM) since DSM III [2]. In DSM IV, the name was changed from inhibited male orgasm to male orgasmic disorder [3]. This name change was retained in DSM IV TR. In Diagnostic and Statistical Manual of Mental Disorders, 3rd Ed., revised (DSM-III-R), subtyping (lifelong vs. acquired; generalized vs. situational; due to psychological factors vs. due to combined factors) was added [4]. The criteria were also modified to exclude orgasmic dysfunction secondary to substance use or a general medical condition in DSM IV.

A literature review indicates several issues which should be considered in the definition of male orgasmic disorder in DSM V. These issues...
include (i) the appropriateness of the diagnostic label; (ii) increased knowledge concerning organic etiologies of ejaculatory delay; (iii) the relatively little information that is known about psychological etiologies of male orgasmic disorder; (iv) that in the absence of known organic etiologies, minimal information is available concerning differentiation of orgasmic delay due to psychogenic as opposed to biogenic etiologies; (v) the lack of operationalized criteria sets for this diagnosis; and (vi) whether the DSM IV TR subtypes of acquired vs. lifelong, global vs. situational, and due to psychological vs. due to combined factors should be retained, and the scope of this psychiatric diagnosis. These issues will be addressed in this article.

Diagnostic Terminology

One of the first issues to be considered for these diagnoses is the appropriateness of the name. A Medline search from 1990 to 2008 indicated relative infrequent usage of terminology including orgasm as opposed to terminology specifying ejaculation for male disorders. For example, a Medline search performed on June 18, 2008, indicated only 25 references linked to the term male orgasmic disorder, only five citations linked to male orgasm disorder, and only 125 linked to male anorgasmia. In contrast, there were 590 citations linked to delayed ejaculation, 131 to anejaculation, 106 to retarded ejaculation, and 207 to ejaculatory delay. This suggests that serious consideration be given to a name change in order for the official diagnostic nomenclature to be congruent with current usage.

The DSM IV TR term implies an emphasis on the subjective experience of orgasm, whereas most clinical research appears to be concerned with ejaculation.

The explanation for the minimal use of the current term “male orgasmic disorder” is clear upon reflection. Orgasm and ejaculation usually occur simultaneously in men, although they are two separate phenomena [5]. Ejaculation occurs in the genitals, whereas the sensation of orgasm is a cerebral event [6,7]. The choice of the current terminology can perhaps be best understood in historical context. An assumed parallelism between male and female sexual disorders was introduced by Masters and Johnson [8], which was reflected in the various editions of the DSM [9]. For example, the DSM IV TR contains two parallel diagnoses: female orgasmic disorder and male orgasmic disorder. For female orgasmic disorder, emphasis to date is focused on the subjective sensation of orgasm. In males, emphasis has been primarily on the genital response (i.e., whether or not ejaculation occurs in a timely manner). Unfortunately, the terminology in DSM IV TR does not reflect these sex differences. These sex differences were not fully appreciated by prior DSM committees because of the persistent emphasis on parallelism between the sexes and the small number of cases of male orgasmic disorder seen in clinical practice [10,11]. This author feels strongly that the terminology and criteria sets should be modified to reflect current usage by clinicians and investigators.

Organic Etiologies

In the last three decades, our knowledge of organic contributions to orgasmic difficulties has increased [7]. Organic contributions to ejaculatory problems can be appreciated by a brief review of the neurophysiology of ejaculation. The efferent nerves that cause seminal emission and bladder neck closure are sympathetic fibers from thoracolumbar levels, T10-L2, which travel through the sympathetic ganglia, hypogastric plexus, and peripheral pelvic nerves. Damage to any of these nervous pathways can interfere with ejaculation. Sensory input from the genitals also travels through the dorsal nerves of the penis and contribute to the ejaculatory process. Damage to these fibers could also be expected to affect ejaculation [12]. The most common cause of neurogenic problems with ejaculation is spinal cord injuries. Other causes are nerve damage during retroperitoneal lymph node dissection, periaortic surgery such as aortic aneurysm surgery, and pelvic surgery such as radical prostatectomy [6]. Diseases that cause peripheral neuropathies, such as diabetes mellitus, multiple sclerosis, and alcoholic neuropathies, can also result in ejaculatory dysfunction [13,14]. Many pharmacological agents have been reported to be associated with ejaculatory dysfunction, especially delayed ejaculation [15]. These drugs include many antihypertensive and antiadrenergic agents. Most antidepressants, including tricyclic antidepressants, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, and serotonin-norepinephrine reuptake inhibitors, have been reported to cause ejaculatory delay. Antidepressants with minimal ejaculatory delay include bupropion, mirtazapine, and nefazodone [16]. Antipsychotic drugs such as haloperidol, olanzapine, quetiapine, and risperidone have also been reported to cause ejaculatory delay [17].
There is conflicting data concerning whether the incidence of male orgasmic dysfunction increases with age [7,18,19]. If an age-related increase in the incidence of orgasmic dysfunction exists, it could be partially related to decreased penile sensitivity with age or to an increase in the ejaculatory threshold with age [20,21].

**Psychogenic Orgasmic Disorders**

Complaints of difficulty with orgasm, other than those caused by psychotropic agents, are rarely seen by mental health professionals [22]. Thus, there is minimal information available concerning either etiology or treatment. Most of the available data consists of clinical impressions or case reports and should be considered as anecdotal. Some therapists have emphasized enhancing sexual arousal by the use of either erotic materials and/or use of vibrators. Perelman [23,24] has suggested that some men with orgasmic dysfunction may have histories of idiosyncratic masturbatory techniques, and thus have been conditioned to respond to the masturbatory technique rather than the sensation of vaginal intercourse. Others have commented that such men have histories of strict religious upbringing. Stewart and Ohl [25] suggested that discrepancy between reality of the sexual partner and the sexual fantasy used in masturbation may contribute to the development of retarded ejaculation. They suggest the use of self-stimulating techniques to achieve incremental increases in arousal. Apfelbaum [26] suggests that men with orgasmic disorder have an autosexual orientation and are overly concerned about pleasing their partners to the extent that they become distracted from their own sensations. Rowland et al. [27] reported a study comparing men with complaints of retarded ejaculation with normal controls. Out of a sample of 1,400 men who had been evaluated in the psychophysiology laboratory at Erasmus Medical Center, 25 men were identified as having a primary diagnosis of idiopathic retarded ejaculation. These men had decreased penile erectile response to visual sexual stimulation combined with concomitant vibrotactile stimulation than normals. They also reported less subjective sexual arousal. Relationship satisfaction did not differentiate these men from normals. Subjective sexual arousal was higher in men who previously reported highly arousing sexual partners and less fear of sexual failure.

Waldinger and Schweitzer [7] have introduced a new conceptualization of male orgasmic disorder. They have proposed that both male orgasmic disorder and premature ejaculation be regarded as neurobiological variants of the ejaculatory threshold and that psychological factors may be of minimal import in the genesis and maintenance of this disorder. Thus, ejaculatory latency could be normally distributed in the general population, and premature ejaculation and male orgasmic disorder would be extreme variants of this distribution.

In view of the limited information available concerning the etiology of male orgasmic disorder, the use of the current subtype by etiology (e.g., due to psychological factors and due to combined factors) presupposes a level of knowledge that frequently does not exist.

**Differentiation of Psychogenic from Biogenic Orgasmic Disorder**

Anecdotal reports characterize men with orgasmic dysfunction as being able to ejaculate with masturbation but not coitus [26], having prolonged erections without ejaculation [7], and as having nocturnal emissions although incapable of intravaginal ejaculation [28]. There is an absence of information concerning differentiation of male orgasmic disorder as a psychological diagnosis from retarded ejaculation as a nonpsychiatric diagnosis. In practice, the psychiatric diagnosis of male orgasmic disorder is only considered after the exclusion after all known organic etiologies. In the absence of strict criteria for assigning organic etiologies to delayed ejaculation, one cannot assume that clinical series of men receiving psychological interventions and diagnosed as having male orgasmic disorder are composed totally of psychogenic cases. A report of being able to ejaculate relatively easily with masturbation but not during coitus would be strongly suggestive of a psychological component to the problem. Similarly, certain diseases such as multiple sclerosis and injury to the nervous innervation of the genitalia indicate a probable organic etiology.

**Operational Criteria**

If knowledge is to advance concerning this relatively infrequently encountered disorder, it will be necessary to develop more precise criteria sets. We lack data on average ejaculatory latency in men complaining of ejaculatory delay. However, we do have epidemiological data that might be
utilized to develop some criteria sets. For example, in the Global Study of Sexual Attitudes and Behaviors [29], approximately 9% of men in Northern Europe aged 40–80 complained of inability to reach orgasm for two of the preceding 12 months. However, this number represents the aggregate of men stating this was an occasionally, periodically, or frequently a problem. This author would argue that occasional and periodic problems would not reach the threshold for diagnosis. One could arbitrarily define frequently as occurring on 75% or more of occasions. The prevalence of frequent problems in various world regions were as follows: Northern Europe, 1.6%; Southern Europe, 1.6%; non-European West, 2.7%; South America, 1.9%; Middle East, 1.1%; East Asia, 2.8%; Southeast Asia, 2.1%. Another area in which epidemiological data may be helpful concerns duration of complaint. For example, a national probability study [30] found that 5.3% of men experienced being unable to reach orgasm for a month the previous year. However, only 0.7% reported this problem as lasting at 6 months. Adding a requirement of a 6-month duration prior to diagnosis would also serve to help to define a more homogenous population. Waldinger and his colleagues [31] reported mean ejaculatory times as measured by a stopwatch in a population survey of men in Turkey, the United Kingdom, the United States, Spain, and the Netherlands. Intravaginal ejaculatory times ranged from 55 seconds to 44.1 minutes, with a median intravaginal latency time of 5.4 minutes. If one used a cutoff of 2 standard deviations beyond the mean, a value of 22.36 minutes would be the cutoff for the diagnosis of male orgasmic disorder. There are some clear limitations with using a 22-minute cutoff. Some men with a 22-minute intravaginal ejaculatory latency may have been purposely delaying ejaculation, while other men may not have been able to ejaculate. Also, some men become distressed if they are unable to ejaculate after 5–10 minutes of active thrusting and then cease sexual activity at that point. It is also unclear if a similar time limit would apply to sexual activity other than vaginal intercourse.

There is minimal evidence concerning the emotional reaction of men with delayed ejaculation and whether the requirement of distress is appropriate. As in other psychiatric disorders, the requirement for “marked distress” in order to make a diagnosis complicates research by possibly combining the patient’s emotional reaction to a condition with the condition being diagnosed.

Specific Recommendations

It is recommended that the category of male orgasmic disorder be renamed delayed ejaculation to reflect current usage. It is also recommended that specific severity (e.g., 75% of sexual occasions) and duration of complaint criteria (e.g., 6 months) be introduced into the definition in order to define more homogenous groups for clinical research. An exception for the duration criteria would be instances in which there is a clear and proximate evidence of a cause for the disorder such as interruption of the nervous innervation of the genitalia by surgery or trauma.

At this point, there does not appear to be sufficient information to designate an absolute time delay for the diagnosis of ejaculatory delay. It also should be noted that the little information available pertains to intravaginal ejaculation. There is a paucity of information concerning other sexual activities or sexual activity in homosexual males.

For all the sexual dysfunctions, DSM IV TR requires the designation of subtypes (global vs. situational; lifelong vs. acquired; due to psychological vs. due to combined factors). Clinicians have reported that the subtypes of lifelong vs. acquired and global vs. situational are clinically useful. Given the paucity of information concerning this disorder, it is recommended that these descriptive subtypes be retained. The current use of etiological implies a knowledge of etiology which often does not exist unless there is a known organic etiology. It is strongly recommended that the designation of a psychological or combined etiological subtype be deleted.

In place of etiological subtypes, it is recommended that a designation of associated features should be considered. The designation of associated features should facilitate a better understanding of illness or events associated with delayed ejaculation. Associated psychological features might include such things as lack of attraction to current partner, sexual fantasies markedly different from current sexual activities, and interpersonal discord. Associated medical features might include diseases, medications, and surgical procedures. As mentioned above, in cases where there is an organic etiology that totally explains the ejaculatory problem, it would not be coded as a psychiatric disorder. A psychiatric diagnosis concerning ejaculation would be restricted to cases in which psychological factors are suspected to contribute to the etiology or maintenance of the problem, cases in which the etiology is unknown, and cases.

J Sex Med 2010;7:690–699
suspected to have a predominantly psychological etiology.

It is recommended that the syndrome of delayed ejaculation be defined independently of the psychological state associated with the difficulty. This should help in defining more homogenous groups for research. As for other psychiatric disorders, a clinical diagnosis of delayed ejaculation would only be made only if the patient experiences discomfort with the problem. As with the other male sexual dysfunctions, it is recommended that emotional discomfort be utilized rather than marked distress.

Treatment progress could be monitored by denoting the frequency of ejaculatory failure (e.g., always, almost always, frequently). Alternatively, one could measure reduction in the average time required to ejaculate during sexual activity or the relative difficulty with which ejaculation is achieved.

An overriding issue in the definition of this syndrome is how the DSM and International Classification of Diseases and Related Health Problems (ICD) systems will decide to handle organic conditions with similar syndromal presentations as conditions with unknown or presumed psychogenic etiologies. Historically, the ICD system has separated behavioral syndromes (which are often of psychological or of idiopathic etiology) from medical disorders with known organic etiologies. This presents considerable difficulty in defining criteria sets in certain sexual disorders such as male orgasmic disorder. The issue is that one is trying to define criteria that may or may not prove applicable independent of etiology.

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Conflict of Interest: None.

References


J Sex Med 2010;7:690–699
Male orgasmic disorder (MOD) is not a psychiatric condition but is within the diagnostic criteria of the DSM IV TR. It describes a persistent or recurrent delay in, or absence of, orgasm, following a normal excitement period. There is an inherent expectation of sufficient, if not complete, sexual arousal with both physical erection and psychic stimulation. However, even when this is assessed as sufficient, it is stated that the condition specifically excludes ejaculatory difficulties of known organic etiologies. We argue that at this stage, the processes of ejaculation and orgasm should be separated, although for most men they are synchronous. By uncoupling the processes, the differentiation of ejaculation and orgasm can be considered as individual components. Classification of any factors affecting either process should be considered separately.

The authors indicate only limited references linked to MOD over 18 years in Medline. In part, this is because many people with orgasmic disorder do not seek advice from clinicians. Few cases are written up as clinical papers. Men who do seek help with the “orgasmic” part of the sexual response are often actually complaining about ejaculatory function, and as such, these are published with appropriate key words. There is strong justification for two terms—MOD and ejaculatory disorder (EjD).

Sensation of orgasm is not restricted to cerebral experiences, and for many men, it is a whole body experience. Acquisition of skills to experience multiple orgasms in sequence identifies a technique inherent in tantra. While the focus of pleasure is usually attributed to expansion and expulsion of semen along the urethra with subsequent spurt from the meatus, many of the sensations within the pelvis, extending into the lower limbs from muscular activity, is evident for those men who can experience orgasm ahead of any ejaculatory event. This will preclude MOD being entirely dependent on psychological processes as there is a clear somatic component to the experience. So, MOD is not a wholly psychiatric disorder anymore greater than EjD is entirely an organic disorder.

Many of the cases of rapid (premature) and delayed (inhibited) ejaculation are not the consequence of organic disease within the prostate or genitourinary tract, and so, argument that the process of ejaculation is attributable to physiological factors is as flawed as MOD being wholly psychiatric (or psychic) in origin. The lack of consistency of age-related changes affecting EjD and MOD require much more extensive investigation. The emerging argument that this is a variance of the neurobiological threshold for ejaculation (and orgasm) requires greater consideration through observation and measurement.
We do not, however, accept that MOD is the extreme variant of rapid ejaculation as this assumes ejaculation and orgasm to be coupled. Many men with rapid ejaculation will have very substantial orgasmic pleasure, so it should not be argued that time of ejaculation necessarily equates with the magnitude of orgasmic experience. As with many medical conditions, the role of psychological factors may be considerable and cannot be taken in isolation when trying to apportion the extent these factors may have in the etiology or maintenance of the condition (or problem).

Regarding operational criteria, we agree that a frequency of the problem (EjD or MOD or both) in three out of four sexual events persistently over a period of 6 months is a reasonable suggestion. However, we disagree that an IVET of 2 standard deviations beyond the mean could be the cutoff point for MOD. First, MOD is not directly linked with timing of ejaculation. Second, the time of 22.36 minutes quoted is substantial, and many men or their partners will want to stop sexual penetration activities before this time for a number of reasons such as boredom, personal or partner discomfort, etc. To operationalize delayed ejaculation on time factors alone is erroneous and could lead to flawed diagnoses being applied if penetrative sexual activity is curtailed. Subsequent masturbatory activity (self or by partner) may lead to ejaculation soon thereafter, regardless of the time previously spent on penetrative activity.

In summary, we believe MOD should be separated into the two processes of orgasm and ejaculation. As such, MOD should be reclassified into EjD and (male) orgasmic disorder. EjD can be subdivided into rapid (premature), delayed (inhibited), and retrograde. Severity (75%) and duration (6 months and beyond for a diagnosis of persistent disorder) are reasonable parameters to define in the absence of obvious neurological pathology. While the recent International Society for Sexual Medicine definition of rapid (premature) ejaculation has been reached by consensus, we do not feel that there is sufficient evidence to operationalize a time to define delayed ejaculation. Furthermore, we do not accept proposals that remain exclusively heterosexual, and as such, any development of these criteria must emphasize penetrative activity (vaginal, anal, or oral) and should not cite intravaginal ejaculation as the only parameter but rather vaginal, anal, oral, and/or masturbatory ejaculation time. Lifelong vs. acquired and global vs. situational can be retained. The concept of associated factors replacing etiological subtypes warrants further attention and consensus. Moving toward sexological diagnoses rather than psychiatric or “organic” will provide a further challenge for those attempting to define the group of conditions that affect sexual response and performance.

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The basis of characterizing male orgasmic disorder in the various DSMs has had the problem of confusing the mechanism of ejaculation with emission of semen with the mechanism of orgasm. It is received wisdom that these are separate neurobiological mechanisms, yet the literature to support this is difficult to find and rarely quoted despite the demands of evidence-based medicine. Kinsey et al. [1] highlighted their separation in the 1948 male survey. They stated “orgasm may occur without the emission of semen” (p. 158), exemplars being preadolescent males and in a few adults (11 in 4,102 histories, an incidence of 0.3%). However, they also added that “while the climax is thus clearly possible without ejaculation it is doubtful if ejaculation can ordinarily occur without a preceding climax” and that “it is difficult to understand what mechanism could produce ejaculation without a precedent orgasm” (p. 159). We would regard these statements today as authority based and not evidence based. Later, in their second study [2] on the female, they listed the evidence (pp. 635–6) for concluding that they were separate mechanisms, namely

1. orgasms occur without ejaculation in preadolescent males;
2. in some adult males, orgasm does not occur until a few seconds after ejaculation;
3. a few adult males who are anatomically incapable of ejaculation have orgasms;
4. males who have been prostatectomized experience orgasm; and
5. men who are deficient in gonadal hormones cannot ejaculate, but some have orgasms.

Later additions [3] include

1. paraplegics who can have so-called “phantom orgasms” without ejaculation;
2. men who claim to have multiple orgasms before finally ejaculating (see Levin [4] for references); and
3. men with the condition “anesthetic ejaculation” can ejaculate but do not have an orgasmic sensation [5].
Inspecting this list shows that much of the evidence for identifying the different mechanisms for ejaculation and orgasm comes either from pathophysiological conditions or immature males rather than from strong empirical studies. It is of course difficult and possibly unethical in normal males to try and separate experimentally the two mechanisms. Comparing the brain scans of males who ejaculate but who do not have orgasms (anhedonics) and those who can have orgasms but do not ejaculate until the final one may be a possibility. If they are really different, it would add considerable weight to the separate mechanism concept.

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In andrology practice, concern about ejaculation—and in particular about premature ejaculation (PE)—is quite common. In August 2007, a panel of experts of the International Society for Sexual Medicine reviewed the criteria for the definition of PE [6].

Retarded, inhibited, or delayed ejaculation (DE) is another ejaculatory dysfunction, although less frequently referred than PE. Unfortunately, the definition of DE is still not completely clear. An evidence-based widespread definition of a medical condition is the cornerstone to correctly identifying and treating subjects affected. In addition, it would help clinicians to correctly stratify the problem and to compare data from different studies. A classic example is what happened with the erectile dysfunction (ED) definition. Different methods used for the definition of ED have been considered as possible confounding factors for study comparisons. In 1993, the National Institute of Health Consensus Conference defined ED as the persistent inability to achieve and/or maintain a penile erection adequate for satisfactory sexual activity [7]. This definition is still the reference point in the field.

In this issue of The Journal of Sexual Medicine, Segraves reviewed the current literature emphasizing the problems included in the current version of DE definition of the DSM IV TR and recommending what should be corrected during the development of DSM V consensus statement.

First of all, they suggested using the term “delayed ejaculation” instead of “male orgasmic disorder” because it better reflects the pathogenesis of the disorder. In fact, although orgasm and ejaculation usually occur simultaneously in men, they are two separate phenomena. In line with this point, it is also important to emphasize that DE should not be confused with the more common condition known as retrograde (or dry) ejaculation. In the latter case, ejaculatory bolus goes back into the bladder, instead of out the urethra because of incompetence of the bladder neck. Remarkably, in retrograde ejaculation, although there is no appearance of an anterograde ejaculate, orgasm is still present.

In addition, a better clarification of the DE-associated psychological factors has also been suggested and that the possible presence of an organic etiology may totally explain the ejaculatory problem. In particular, associated medical features include serotonergic and antidopaminergic pharmacological agents as well as surgical procedures and peripheral neuropathies impairing the ejaculatory reflex neurological pathway. Interestingly, it has also been reported that even the hormonal system may influence ejaculatory timing. In particular, we demonstrated for the first time an association between hypogonadism and DE [8], while Jannini et al. [9] reported a higher prevalence of DE in subjects with hypothyroidism.

Finally, the lack of sufficient information to designate an absolute time delay for the diagnosis of DE and the necessity of treatment progress monitoring have also been emphasized.

In conclusion, a more accurate, evidence-based definition of DE is required. Specific expert recommendations should be taken into account during the development of the DSM V consensus statement.

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It is an atypical experience to find oneself in complete agreement with every recommendation offered in an article by another sexual medicine expert. However, I fully support the recommendations outlined in Robert T. Segraves’ draft of the DSM V revisions for the male orgasmic disorder (MOD) section, including the “delayed ejaculation” renaming, the criteria for diagnosis, and especially the new use of associated considerations to replace subtyping by etiology. Nonetheless, below are two additional points of commentary.
**Nomenclature.** MOD is better used to describe numerous conditions including, but not limited to, premature ejaculation, diminished ejaculatory amount, force, intensity, sensation, as well as delayed ejaculation (DE) [10]. DE is a better name for the previously labeled “MOD,” allowing for official diagnostic nomenclature to be congruent with current usage. However, eventually, the U.S. regulatory authority (FDA) will approve a drug for DE. FDA approval will trigger direct-to-the-consumer advertising, resulting in “DE” being the name of choice as erectile dysfunction was successfully relabeled “ED.” No doubt such a process will be repeated, making any further discussion moot.

**Etiology.** It is agreed that the current DSM IV subtypes (e.g., due to psychological, combined, etc.) should be discontinued [11]. However, my rationale for such discontinuation is different from that offered in this DSM V draft document. Etiology is always “mental and physical.” What varies is the extent to which each of the individual “mental and physical” factors contributes to the end-point sexual response. In our eagerness to classify, we tend toward a reductionism, which impacts the accuracy of our data interpretations and distorts our subsequent theorizing and conclusions. Genetically predetermined ejaculatory thresholds have a profound impact on ejaculatory ease and latency time, and do distribute in a manner similar to a number of human characteristics. However, human experience is better explained when one postulates that such thresholds predetermine a range of response rather than an exact point; a “scatter cloud” rather than a “trigger point” would be a better metaphor. The ease and timing of a particular ejaculation and orgasm would be a function of a variety of psychosocial–behavioral and cultural factors impacting that biologically predetermined range [12]. This integrated biopsychosocial–cultural model would account not merely for the difference between each man’s ease, quality, and latency of ejaculation/orgasm but also for the well-documented intraindividual range on those same metrics. There is considerable variation in any given man’s range of response, depending on type of stimulation, partner variables, age, context and developmental history, and so on. The neurobiological threshold model [13] does not fully account for such variation, but a biopsychosocial–cultural model (which incorporates the concept neurobiological threshold) does explain the full range of such variation both between and within given individuals, and thus provides a better theoretical basis for future research [12].

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