Premature and Delayed Ejaculation: Genetic and Environmental Effects in a Population-Based Sample of Finnish Twins

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ABSTRACT

Introduction. A number of different theoretical approaches to understanding the etiology of ejaculatory dysfunction have been proposed, but no behavior genetic study has yet, to our knowledge, been conducted to explore the genetic and environmental influences on ejaculatory dysfunction.

Aim. The aim of the present study was to explore the genetic and environmental effects on premature (PE) and delayed (DE) ejaculation in a population-based sample.

Methods. The genetic and environmental influences on PE and DE were investigated in a population-based sample of 1,196 Finnish male twins, age 33–43 years, with 91 identical and 110 complete twin pairs. Several different aspects of ejaculatory function were measured by a self-report questionnaire (e.g., latency time, subjective experience of ejaculatory control). Factor analyses distinguished two subcomponents of ejaculatory function, and subsequently, composite variables measuring PE and DE were created. Structural equation modeling was performed on the composite variables.

Main Outcome Measures. Measurement of genetic and environmental effects on PE and DE.

Results. The results suggested moderate genetic influence (28%) on PE, but not on DE (0%). There was a moderate familial effect on DE with shared environmental effects accounting for 24% of the variance. However, omission of the shared environmental component did not directly result in a significantly decreased model fit for DE, and omission of the additive genetic component did not directly result in a significantly decreased fit for the PE model.

Conclusions. The findings from the present study provide useful information regarding the etiology and understanding of ejaculatory dysfunction. **Jern P, Santtila P, Witting K, Alanko K, Harlaar N, Johansson A, von der Pahlen B, Varjonen M, Vikström N, Ålgars M, and Sandnabba K. Premature and delayed ejaculation: Genetic and environmental effects in a population-based sample of Finnish twins. J Sex Med 2007;4:1739–1749.**

Key Words. Premature Ejaculation; Delayed Ejaculation; Genetic Effects; Twin Study

Introduction

Premature ejaculation (PE), also referred to as early or rapid ejaculation, is, together with erectile dysfunction, widely regarded as the most common sexual dysfunction in males [1]. Problems with ejaculatory function can be viewed as a continuum varying as a function of latency, where lifelong PE is present on the one extreme, and lifelong delayed ejaculation (DE) or inability to achieve ejaculation occurs on the other [2].

The prevalence estimates of PE vary widely. Montorsi [3], in a review of recent studies, suggested a global prevalence of PE of approximately 30% across age groups as well as different cultures.

Waldinger and his associates propose that PE should be defined according to the intravaginal ejaculation latency time [4,5]. All men with a latency of less than 1 minute would have definite PE, and men with a latency varying between 1 and 1.5 minutes would have probable PE. Using these definitions, 0.5% of males would have definite PE and 2.5% probable PE. These definitional suggestions are based on stopwatch-assessed intravaginal ejaculation latency times, that is, latency from vaginal penetration to ejaculation. In a multinational

population survey [4], 500 couples were recruited from five different countries. The enrolled men were all older than 18 years of age and in stable heterosexual relationships. The median latency was 5.4 minutes and decreased significantly with age. Some other studies using self-report methodologies have found longer mean latencies: 13.6 minutes [6] and 7.9 minutes [7]. However, contrary to median values, means would be easily affected by unusually long latencies, which makes a direct comparison impossible. The mean percentage of ejaculations that took place prior to penetration varied from 5.6% [7] to 9.9% [6]. These two indicators of PE, latency, and ejaculation prior to penetration, were moderately negatively associated.

DE is a less common sexual dysfunction in males, and few studies have looked into its etiology [8]. Rosen suggested that the disorder may be associated with a variety of surgical or medical conditions and the use of anti-adrenergic or neuroleptic drugs. In recent studies, approximate prevalence estimates of 1–4% in sexually active men have been reported [9,10], while Nathan found the prevalence of DE in the general male population to be around 0.15% [11]. Waldinger and Schweitzer point out that DE "always appears the least expressed sexual complaint" [12] (pp. 77–78). In the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), DE is termed male orgasmic disorder and defined as "a persistent delay in, or absence of, orgasm in a male following a normal sexual excitement phase during sexual activity that the clinician, taking into account the person's age, judges to be adequate in focus, intensity, and duration." This definition has been criticized by Waldinger and Schweitzer for its failure to separate orgasm from ejaculation.

Waldinger has stressed the importance of neurobiological factors in the etiology of PE and DE [2,12,13]. In his ejaculation distribution theory, he has emphasized the clinical relevance of ejaculation latency. According to the theory, there is normal biological variation in ejaculation latency in men that, in part, reflects the influence of genetic factors. A small subgroup of any random sample of men is expected to have PE, whereas another subgroup is expected to have DE with the majority falling in between these two extremes. Biological factors are assumed to be more influential at the extremes, whereas the role of psychosocial factors in affecting ejaculation latency is assumed to be more important in the middle of the distribution [2].

According to the ejaculation distribution theory, genetic factors play an important role in determining ejaculation latency. Waldinger suggested that lifelong PE, in particular, is a result of a genetically determined endophenotype, that is, a hereditary characteristic that is normally associated with—but is not a direct symptom of—the condition, as it precedes it in the causal chain [2]. By his definition, PE and DE have a stable pattern during life, are present for the total duration of an individual's life, and occur in both heterosexual and homosexual men. The direct evidence for the involvement of genetic factors in determining ejaculation latency or other indicators of PE or DE is, however, scant. Animal studies have revealed that mice lacking the gene for endothelial nitric oxide synthase develop symptoms similar to PE, while mice lacking the gene for heme oxygenase-2 develop DE or anejaculation [14]. In an early review of 1130 cases of PE in humans, Schapiro noted, in addition to other observations, that PE tends to be familial [15]. This conclusion was supported in a more recent study by Waldinger et al., which reported on 14 men who asked their male relatives about ejaculation latency [16]. Eleven of the men had a male relative with relevant information available. Ten of these had an ejaculation latency of less than 1 minute. The odds of the problem co-occurring between family members were much higher than those expected solely on the basis of population prevalence rates. The authors concluded that this observation supports the role of genetics in affecting PE. While plausible, familial resemblance in itself cannot prove genetic influences as individuals who are genetically related also often share their environment to some extent. Therefore, shared environmental factors could also explain familial occurrence of PE.

In the present study, the aim was to investigate the possibility of genetic influences on a number of commonly used indicators of PE and DE. In addition to investigating whether genetic factors are involved, the role of shared environmental factors on the indicators was explored. Basic behavior genetic design, the twin design, was applied to answer the research questions. This design is, besides adoption studies, the major method used to disentangle latent genetic from environmental sources of resemblance. Identical (MZ) twins are genetically identical. If genetic factors are important for a trait, MZ twins must be more similar than fraternal (DZ) twins who are, on average, 0.50 similar genetically. Making the so-called equal environments assumption, that is, that both