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An Epidemiological Survey of Post-Coital Psychological Symptoms in a UK Population Sample of Female Twins

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Postcoital psychological symptoms (PPS) is a virtually unexplored phenomenon in the female population even though women frequently complain about irritability and motiveless crying after intercourse and/or orgasm. The aim of this study was to explore the epidemiology and genetic influences of PPS in a UK population sample of women. 1,489 unselected female twins aged 18–85 completed questions on recent and persistent PPS and potential risk factors. Standard methods of quantitative genetic analysis were used to model latent genetic and environmental factors influencing variation in PPS. For identification of potential risk factors, regression analyses were conducted. Phenotypic variation in PPS was explored using a genetic variance component analysis (VCA) approach. We found 3.7% of women reported suffering from recent PPS and 7.7% from persistent PPS. Relationship satisfaction and experience of abuse were found to be independently associated with recent (OR 4.5, 95% CI 4.13–4.87and OR 1.3, 95% CI 1.02–1.34, respectively) and persistent PPS (OR 2.53, 95% CI 2.17–2.81and OR 1.16, 95% CI 1.09–1.26, respectively). VCA revealed that phenotypic variance was best explained by an additive genetic (AE) model, ascribing 28% (for recent PPS) and 26% (for persistent PPS) of phenotypic variance to additive genetic effects, with the rest being a result of individual experiences and random measurement error. To our knowledge, this is the first and largest study investigating the epidemiology of PPS. It seems that the most important targets for intervention and prevention occur outside of the family, such as relationship quality and satisfaction, and history of abuse.

■ **Keywords:** genetics, post-coital, twins, irritability, resentment

Postcoital psychological symptoms (PPS) is a virtually unexplored phenomenon in female populations. Despite a wealth of evidence from specific internet sites and forums (e.g., www.thenakedscientist.com) suggesting that irritability, crying and mood swings after sex seem to be common in females and males, to date no scientific study has tried to explore the nature of the phenomenon. In several case reports a similar condition — post-orgasmic illness syndrome (POIS) — has been described in men, consisting of symptoms such as anxiety, irritability, severe fatigue, headache (Ashby & Goldmeier, 2010; Waldinger & Schweitzer, 2002). However, male POIS includes mental and/or physical symptoms and seems to appear rapidly after ejaculation whereas reports from female sufferers describing their condition suggest that PPS can occur after sexual intercourse, both with and without orgasm. Because of the paucity of epidemiologic data, it is unclear whether PPS is caused by hormonal changes during sexual intercourse and/or after orgasm, which can lead to emotional highs and lows, similar to pre-menstrual syndrome, with no obvious emotional reason; or whether the symptoms occur because of psychological reasons such as resentment about the partner choice or feeling a low internal locus of control (e.g., feeling trapped in an unhappy relationship; Krueger et al., 2003; Rosenfeld et al., 2008).

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Several studies have shown hormonal changes to happen during sexual arousal and orgasm in women and men (Exton et al., 1999; Krueger et al., 2003). In an experimental study conducted on women by Exton et al. (1999), film-induced sexual arousal significantly increased plasma adrenaline and noradrenaline concentrations, whereas orgasm induced a large increase in plasma concentrations of prolactin, which remained elevated 60 minutes after completion of arousal. Additionally, small increases in LH and testosterone concentrations were observed. It is not unlikely that postcoital and/or postorgasmic endocrine alterations may cause symptoms of PPS in women, since a substantial amount of evidence exists proving effects of hormones on mood, depression, anxiety and so on (Dell & Stewart, 2000; Schmidt et al., 1998). Especially estrogen seems to affect mood, due to its ability to increase serotonin and beta-endorphins, and hence might also be involved in the pathogenesis of PPS. Apart from biological factors, research on female sexual dysfunction (FSD) has shown several psychological entities to correlate even more strongly with sexual functioning. The psychosocial risk factors affecting women's sexual functioning are broad and comprise interpersonal factors such as relationship imbalances or lack of trust, and emotional difficulties, such as stress or a history of sexual and physical abuse (Qureshi et al., 2007; Schnarch, 1997). It is therefore not unlikely that, similar to FSD, these psychological factors also contribute to the development of PPS.

Given the complete absence of epidemiologic knowledge about PPS, the aim of this study was to explore prevalence and etiology of PPS in a UK population sample of women. We further explored whether there is genetic variation (heritability) to the common causes of PPS suggesting an underlying biological basis to PPS.

Further research into the neurobiochemical sequelae of orgasm will be useful in understanding the pathological processes in these cases.

Material and Methods

Sample

Subjects were female monozygotic (MZ) and dizygotic (DZ) female twins drawn from the TwinsUK registry (Spector & Williams, 2006). The twins have undergone extensive clinical investigations and have been shown to be comparable with age-matched general population in terms of disease, prevalence of lifestyle and sexual behavioral characteristics (Andrew et al., 2001; Burri et al., 2009). The subjects were not selected on the basis of variables being studied. The project was approved by the St Thomas' Hospital research Ethics Committee and all twins provided informed consent. Zygosity was established by using standardized questions about physical similarity that have over 95% accuracy when judged against genotyping results (Peeters et al., 1998). In cases of uncertainty, zygosity was further confirmed by multiplex DNA genotyping (Von Wurmb-Schwark et al., 2004).

Measures

Data on recent and persistent PPS were collected in 2009 using a postal self-administered questionnaire. The questionnaire was made up of various validated questionnaires (including the Female Sexual Distress Scale – FSDS) and a question constructed specifically for this study to assess PPS, asking: 'Have you ever experienced postcoital resentment, i.e. symptoms of crying, mood swings, depressions etc. after having had sexual intercourse with your partner' (Derogatis et al., 2002). Response options were on a Likert-type scale, ranging from 'never' (1) to 'always' (5) with the supplementary option 'no sexual activity' (0). Because of extreme skewness and kurtosis, the variables were dichotomized. Based on the peak score of the item distribution a cut-off score of 4.1 was chosen for both, recent and persistent PPS.

Demographic, psychological, and interpersonal 'risk factor' measures. Data collection of the potential risk factors was carried out in waves between 2007 and 2009. Socio-demographic information on all twins including age and current marital status were obtained from the TwinsUK database. Mental and physical health were both classified as dichotomous traits on the basis of a subject's response to the question: 'Do you suffer from any conditions? (a) medical (b) psychological. If yes, please list'. Events of physical, emotional and sexual abuse were responded to on a *No* (0), *Yes* (1) dichotomous scale. Current and lifelong relationship satisfaction was assessed using a single item asking about overall relationship satisfaction in the last 4 weeks and on average over the whole lifespan, with response options ranging from *Very satisfied* (1) to *Not satisfied at all* (6). Data on anxiety was obtained from the 16-item self-report 'Anxiety Sensitivity Index' (Reiss et al., 1986). The Big Five personality dimensions and the related construct of emotional intelligence were assessed using the 'Ten-item Personality Index' and the 'Trait Emotional Intelligence Questionnaire' (Gosling et al., 2003; Petrides et al., 2004). All measures were selected for their documented association with sexual functioning and problems in women and their psychometric reliability, discriminative and construct validity (see cited references for further details).

Statistical Analysis

To determine the relationship between PPS and the different potential risk factors, univariate and stepwise backward multivariate logistic regression was used. All tests were 2-tailed. Unpaired 2-tailed Student's t test was used to test for differences between MZ and DZ twins for all continuous variables. Two-sample tests of proportions were performed to look for differences between DZ and MZ twin pair groups in dichotomous and ordinal vari-

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ables, including PPS. Non-independence of twin pairs was accounted for by using the cluster function for familial relatedness, which is a form of conditional regression.

Because of the commonly reported relationship between age and sexual problems, age was included as a confounder in the subsequent variance component analysis. For all analyses, a p value less than .05 (95% confidence interval not including '1') was considered statistically significant, unless stated otherwise. Data handling and preliminary heritability analyses were undertaken using STATA (Intercooled Stata for Windows 95, Version 5.0, 1997, StataCorp, College Station, TX) while all further genetic modeling was carried out with Mx software (Neale et al., 2006).

Twin Data and Genetic Modeling

Twin studies are the optimum epidemiological design to study and partition population variation of a trait into genetic and non-genetic — shared and unique environmental — components. The twin design assumes that MZ twins share 100% of their genes, whereas DZ twins share on average 50% of their genes. By contrast, environmental influences that contribute to familial resemblance (shared environment) are assumed to affect MZ and DZ twins equally, meaning that any greater similarity between MZ as compared to DZ twin pairs is attributed to genetic factors (Kyvik, 2000).

In this study, standard methods of quantitative genetic analysis were used to model latent genetic and environmental factors influencing sibling covariance for MZ and DZ twins (Posthuma et al., 2003). For a dichotomous trait, such as PPS, evidence for a genetic contribution (heritability) can be obtained by comparing the casewise concordance (CR) in MZ and DZ twins. Case-wise concordance describes the probability that a twin is affected, given that the co-twin is affected and is calculated from the number of concordant pairs (c) and discordant pairs (d) using the formula: $CR = 2c/(2c + d)$ (MacGregor, 2000). For dichotomous traits, the maximum likelihood modeling method is used, which assumes that variation in the underlying liability of the dichotomous trait is normally distributed in the population (Falconer & McKay, 1989). The correlation in liability among twins is estimated from the frequencies of concordant and discordant pairs using a multifactorial liability threshold model. The level of association within MZ and DZ twin pairs was further measured by employing tetrachoric correlation coefficients.

Quantitative genetic model fitting was used for the decomposition of the observed phenotypic variance into additive (A) and dominant (D) genetic components and shared (C) and unique (E) environmental components (Neale & Cardon, 1992). The latter also includes measurement error. Initial assessment of the components (A, D, C, and E), may suggest non-significant values in one or more component. In further analysis, the significance of each factor as components of the observed variance can be determined by removing each sequentially from the full model and testing the deterioration in fit of the various submodels using hierarchic chi-squared tests. In addition, the Akaike Information Criteria (AIC) is considered, with lower values indicating better fit. The most parsimonious model is then used to estimate the heritability, which is defined as the proportion of total phenotypic variation in a population that is attributable to genetic variation among individuals (Posthuma et al., 2003; Falconer & McKay, 1989). Where a sample consists of only MZ and DZ twin pairs reared together, D and C become confounded and so cannot be estimated together in one model.

Results

Sample Characteristics and Prevalence of PPS

The questionnaire, including the question on PPS, was sent to a total of 3,175 women (aged 18 to 85, mean age 56.2) and returned by 1,589 (50%). Of the 1,589 women who filled in the questionnaire, females who reported never having been sexually active were excluded from further analyses. Further, those reporting no partnered sexual activity during the past four weeks were excluded from analyses of recent PPS. After applying exclusion criteria, a total of 1,489 women were eligible for analyses of persistent PPS, comprising 244 MZ pairs, 189 DZ pairs and 623 women whose co-twins did not participate (41.8%). Data of 930 women, comprising 119 MZ pairs, 67 DZ pairs and 558 single twins were available for analyses of recent PPS.

Characteristics of the overall sample (n = 1489) and compared by zygosity are shown in Table 1. The mean age of participants in the study was 56.3 years (standard deviation [SD] 11.63; range 18–85 years). The MZ and DZ twin groups were well matched for most socio-demographic variables and potential risk factors for PPS, except for marital status where MZ twins were significantly more often married compared with DZ twins (54.9% vs. 46.5%; $p < .01$), who, on the other hand, reported more often being divorced (9.7% vs. 6.6%; $p < .05$). No significant differences in frequency of recent or persistent PPS were found between MZ and DZ twins.

Thirty-four women (3.7%) reported recent PPS, whereas persistent PPS was reported by 115 women (7.7%; Table 1). Of the women suffering from recent PPS, two in three reported being distressed about it (76.9%), whereas one in two women reporting persistent PPS also reported sexual distress in conjunction with PPS (55.5%). Most women (95.5%) suffering from recent PPS also reported persistent PPS; however, 40 (4.5%) women with current PPS didn't report persistent PPS.

Risk Factors

We assessed whether commonly reported risk factors for sexual problems were associated with PPS by performing

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TABLE 1

Lifestyle and Behavioral Risk Factors and Demographic Characteristics of Women Studied by Zygosity and as an Overall Sample

	Overall (n = 1489)			MZ (n = 757)			DZ (n = 732)			p value*
	Mean	*SD*	Range	Mean	*SD*	Range	Mean	*SD*	Range	
Age	56.30	11.63	18–85	55.92	12.26	20–82	56.62	10.94	18–85	0.24
Extraversion	3.50	1.58	1–7	3.52	1.58	1–7	3.48	1.58	1–7	0.19
Agreeableness	2.44	1.13	1–6	2.45	1.13	1–6	2.42	1.13	1–6	0.95
Emotional stability	3.31	1.40	1–7	3.20	1.38	1–7	3.41	1.41	1–7	0.07
Conscientiousness	1.96	0.97	1–6.5	1.84	0.85	1–6	2.08	1.07	1–6.5	0.53
Openness	3.11	1.25	1–7	3.13	1.25	1–7	3.08	1.25	1–7	0.44
Emotional intelligence	154.1	24.48	59–207	154.93	24.82	59–206	153.19	24.09	59–207	0.93
Relationship dissatisfaction	3.33	2.14	1–6	3.50	2.29	1–5	3.43	1.84	1–6	0.51
Anxiety sensitivity	14.02	9.88	0–60	13.30	9.09	0–53	14.77	10.59	0–60	0.06
	N	%		*N*	%		*N*	%		p value**
Marital status										
Single	87	5.85		40	5.28		47	6.42		0.32
Married	**756**	**57.11**		**416**	**54.95**		**340**	**46.45**		**0.01**
In relationship	468	31.42		221	29.19		247	33.74		0.09
Divorced	**121**	**8.20**		**50**	**6.60**		**71**	**9.69**		**0.03**
Widowed	57	3.86		30	3.96		27	3.68		0.76
Sexual abuse	157	10.54		74	9.77		83	11.34		0.41
Physical abuse	171	11.48		89	11.75		82	11.02		0.67
Emotional abuse	323	21.69		161	21.27		162	22.13		0.67
Medical conditions	236	15.85		110	14.53		126	17.21		0.15
Psychological conditions	152	10.21		72	9.51		84	11.47		0.16
Persistent PPS	115	7.69		61	8.06		54	7.38		0.62
	Overall (n = 930)			MZ (n = 492)			DZ (n = 438)			
Recent PPS	34	3.66		19	3.86		15	3.42		0.36

Note: DZ = dizygotic; MZ = monozygotic; *SD* = standard deviation. Significant differences between MZ and DZ twins are highlighted in bold type.
* Unpaired two-tailed t test and Mann-Whitney U-tests were used to test for mean differences.
** Two-sample test of proportions were used to explore differences in response frequencies.

regression analyses (Table 2). Age was found to be substantially correlated with recent but not with persistent PPS, with older women generally being more likely to report PPS compared to younger women (OR 1.06, 95% CI 1.04–1.07, OR 2.4, 95% CI 1.83–3.27, respectively). All variables significantly correlated with PPS in the univariate regression were entered in the multiple regression model (Table 2). A particularly strong association was found between relationship satisfaction and recent PPS (OR 4.49, 95% CI 4.13–4.87, $p < .001$). While experience of physical abuse was associated with recent PPS (OR 1.25, 95% CI 1.02–1.34, $p < .01$), experience of emotional abuse increased the odds of suffering from persistent PPS by 2.5-fold (OR 2.53, 95% CI 2.17–2.81, $p < .001$). Anxiety sensitive women were slightly more likely to report recent PPS (OR 1.05, 95% CI 1.02–1.08, $p < .05$). Contrary to the univariate analyses, no effects of personality emotional intelligence, or psychological conditions on recent or persistent PPS could be observed, (data not shown).

Genetic modeling

Overall, correlations for recent and persistent PPS were significantly higher in MZ compared with DZ twins for recent (70% vs. 40%; $p < .05$) and persistent PPS (78% vs. 70%; $p < .05$), thus a moderate heritability could be expected for these phenotypes (Table 3). Tetrachoric correlations further supported these findings. MZ correlations (r) were consistently higher for both phenotypes, indicating an influence of A on both variables. Based on the CR's, ACE and ADE models were fitted to both phenotypes.

Variance component analysis found that phenotypic variance in recent and persistent PPS was best explained by an AE model, ascribing 28% (for recent PPS) and 26% (for persistent PPS) of phenotypic variance to additive genetic effects (Table 4). The source of familial resemblance according to the χ^2 test could not be distinguished between A and C for both phenotypes but when comparing the AIC fit indices, an AE model was suggested to be the most parsimonious model. Most of the phenotypic variance, however, was explained by unique environmental factors and random error. The confidence intervals for estimation of heritability and especially environmental influence tended to be relatively narrow across both phenotypes, implying adequate precision of our point estimates (Table 4).

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TABLE 2

Univariate and Multivariate Logistic Regression Analysis of Potential Risk Factors for Recent and Persistent PPS

	Univariate analysis			Multivariate analysis		
	OR	*p* value	R^2	OR	*p* value	R^2
Recent PPS (*n* = 930)						0.70
Age	1.06 (1.04–1.07)	**	0.06	1.01 (0.87–1.18)	0.69	
Education	0.93 (0.88–0.98)	*	0.00	0.95 (0.89–0.99)	0.36	
Menopause	2.44 (1.83–3.27)	**	0.02	1.09 (0.75–1.32)	0.88	
Physical abuse	1.61 (1.27–1.98)	*	0.01	**1.25 (1.02–1.34)**	*	
Psychological condition	1.53 (1.01–2.33)	*	0.00	1.85 (1.39–2.14)	0.32	
Extraversion	1.16 (1.07–1.26)	**	0.01	1.03 (0.85–1.21)	0.74	
EI	0.98 (0.97–0.99)	**	0.02	1.00 (0.99–1.01)	0.57	
Relationship dissatisfaction	3.93 (3.40–4.55)	**	0.65	**4.49 (4.13–4.87)**	**	
AS	1.01 (1.00–1.02)	*	0.00	**1.05 (1.02–1.08)**	**0.05**	
Persistent PPS (*n* = 1489)						0.09
Sexual abuse	2.07 (1.37–3.12)	**	0.01	1.37 (0.69–1.86)	0.43	
Emotional abuse	2.99 (2.21–4.05)	**	0.05	**2.53 (2.17–2.81)**	**	
Physical abuse	2.10 (0.14–3.11)	**	0.01	1.55 (0.78–2.41)	0.36	
Psychological conditions	2.46 (1.62–3.71)	**	0.01	1.12 (0.68–1.76)	0.08	
Emotional stability	1.23 (1.12–1.33)	**	0.02	—	—	
EI	0.98 (0.97–0.99)	**	0.02	0.98 (0.97–0.99)	0.06	
Relationship dissatisfaction	1.10 (1.04–1.17)	*	0.01	**1.16 (1.09–1.26)**	**	
AS	1.03 (1.02–1.05)	**	0.02	1.01 (0.99–1.04)	0.44	
OCB	1.02 (1.09–1.04)	*	0.01	1.00 (0.94–1.08)	0.78	

Note: Only significant variables in the univariate model are displayed and were entered into the multivariate model. Significant results of the multivariate regression are shown in bold type. Familial relatedness was accounted for by using conditional regression. Abbreviations: OR = Odds ratio; AS = Anxiety sensitivity; EI = Emotional intelligence; OCB= Obsessive compulsive behavior.
*p value < .05; **p value < .001

Discussion

To our knowledge, this is the first population study to date investigating prevalence of recent and persistent PPS, as well as socio-demographic, health behavioral and psychological risk factors. The present study is further the first to quantify and compare genetic and environmental contribution to the variance in PPS.

Only 3.7% of women reported recent PPS, whereas persistent PPS was reported by around twice as many women (7.7%). Although low in comparison with the prevalence figures often reported for other sexual problems (ranging up to 60%), PPS is clearly not a rare occurrence, despite the lack of research interest (Rosen et al., 1993; Hayes et al., 2006). It is unknown whether PPS is caused by biological factors, such as hormonal changes during and after sexual intercourse and/or orgasm, or whether psychological and emotional factors contribute to the development of PPS. The results of our regression analyses indicated that mainly psychological and interpersonal determinants (i.e., anxiety, experience of abuse and relationship satisfaction) were associated with PPS; whereas, for example, socio-demographic characteristics or personality factors showed no significant effect on PPS.

Similarly, the results of the genetic variance component analysis pointed towards a strong effect of unique environment (and potentially classification error) on the development of PPS. Modest heritability estimates of 28% for recent PPS and 26% for persistent PPS were found, reflecting the overall tendency of low or moderate heritability estimates found for behavioral traits, compared to anatomo-physiologic traits that usually display significantly higher heritabilities (Burri et al., 2009). The results are further consistent with previous twin studies showing a moderate heritability to sexual problems (Dawood et al., 2005; Dunn et al., 2005; Witting et al., 2009). Shared environmental influences were not significant, suggesting that the shared family environment (e.g., conveyed attitudes towards sexuality) during upbringing is negligible for adult women's sexual function or experience of PPS. Overall it seems that bio-physiological or genetic influences explain only a small fraction of the phenotypic variance, implying an important role of contextual or interpersonal factors in the emergence of PPS. Indeed, relationship dissatisfaction turned out to be the main predictor for recent and persistent PPS, increasing the odds for reporting for example of recent PPS by 4.5-fold. These findings are in line with recent research, emphasizing the impact of interpersonal factors in both the development and the maintenance of sexual problems (Nicolosi et al., 2003; Qureshi et al., 2007; Schnarch, 1997). In addition, a history of abuse turned out to be an important contributor to recent and persistent PPS. There are several pathways on how a history of abuse and PPS or negative post-coital affect may go together. Victimized or abused women may develop emotional or psychological problems, such as a heightened anxiety about sexual contacts,

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TABLE 3

Case-Wise Concordance and Tetrachoric Correlations for Recent and Persistent PPS

Phenotype	Zyg	Total*	CR (95% CI)	*p* value	Tetrachoric correlation
Recent PPS	MZ	119	0.70 (0.64–0.76)		0.34
	DZ	67	0.40 (0.40–0.58)	0.00	0.01
Persistent PPS	MZ	244	0.78 (0.74–0.84)		0.41
	DZ	189	0.70 (0.64–0.77)	0.03	0.09

Note: * Total pairs; Abbreviations: Zyg = Zygosity, CR = Case-wise concordance

which may have long-term effect on women's sexual behavior and function. Stressful life events such as abusive experiences might further contribute to disrupted interpersonal and intimate bonding behavior; hence, indirectly lead to the feeling of resentment described here after sexual intercourse. It is further plausible that women with abusive history may be more prone to enter relationships in which they do not always feel in control of their experience, or assertive about their wants/needs, and perhaps may be prone to resentment.

We further found an association between self-reported anxiety and recent, but not persistent, PPS. These findings parallel previous studies that reported association between high anxiety and depression scores and general sexual problems in epidemiological and community samples of women (Bradford & Meston, 2006; Dunn et al., 1999; Frohlich & Meston, 2002). These studies suggest that generalized anxiety or affective problems increase maladaptive attention towards quality of sexual performance and body image that may be imperfectly related to actual sexual activity. We suggest that PPS may be a consequence of a woman's overall high anxiety level. Women may experience anxiety about sex (which again can be rooted in traumas or past experiences or in relationship problems) that makes her uncomfortable during sexual activities, so that afterwards a flood of emotional response is triggered. Our observation is in accordance with the high prevalences of FSD observed in women with anxiety disorders. For example, in a large community epidemiological survey, Dunn and colleagues (1999) found women with moderate-to-high scores of self-reported anxiety to be at a significantly higher risk for a variety of sexual problems.

Although similar risk factors were identified for recent and persistent PPS, the amount of phenotypic variance explained by these factors differed significantly for recent and persistent PPS. While accounting for 70% of the variance in recent PPS, relationship satisfaction and previous events of physical abuse only explained 10% of the variability in persistent PPS, suggesting that there are other unknown factors that contribute to the phenotypic expression of persistent PPS.

Although age did not emerge as a significant independent risk factor neither for recent nor persistent PPS, a possible effect of menopause on PPS cannot be ruled out. Using a commonly applied cut-off of 50 years to classify women into pre- and postmenopausal suggested that 70% of women in this study were post-menopausal (Te Velde et al., 1998). It is therefore not unlikely that endocrine alterations occurring during menopause could have a potential effect on PPS — which would also explain the considerable prevalence rates we found. This assumption is somehow supported by the fact that 4.5% ($n = 40$) of women without persistent PPS reported current PPS, with 38 of these women being postmenopausal. Menopausal transition and especially surgically induced menopause (e.g., due to hysterectomy or ovariectomy) has long been known to impair women's sexual functioning, mostly due to the related decrease in estrogen levels (Dennerstein et al., 2002; Nappi, 2007). Many women may develop symp-

TABLE 4

Genetic Model Fitting Results for Univariate Analysis of Recent and Persistent PPS

Model	A (95% CI)	C/D (95% CI)	E (95% CI)	χ^2 (df)	*p* value	AIC
Recent PPS (n = 866)						
ACE	0.28 (0.00–0.46)	0.00 (0.00–0.00)	0.71 (0.53–0.90)	(861)	—	-524.85
ADE	0.00 (0.00–0.00)	0.33 (0.00–0.51)	0.66 (0.48–0.87)	(861)	—	-526.31
AE	**0.28 (0.09–0.46)**	**—**	**0.71 (0.53–0.90)**	**1.46 (862)**	**.23**	**-526.85**
E	—	—	—	23.65 (863)	.00	-524.43
Persistent PPS (n = 372)						
ACE	0.26 (0.08–0.37)	0.00 (0.00–0.14)	0.73 (0.62–0.83)	(1339)	—	3698.66
ADE	0.00 (0.00–0.33)	0.29 (0.00–0.39)	0.70 (0.60–0.81)	(1339)	—	3696.32
AE	**0.26 (0.16–0.37)**	**—**	**0.73 (0.62–0.83)**	**2.34 (1340)**	**.12**	**3696.66**
E	—	—	—	24.18 (1341)	.00	3699.55

Note: Best-fit models are set in bold.
A = Additive genetic effects; D = Dominant genetic effects; C = Common environmental effects; E = Unique environmental effects and random error; The variance component E is conflated with the random error in the ordinary least squares regression and therefore the confidence intervals for E cannot be explicitly estimated using DF methods. However, the point estimate for E can be estimated by subtracting the other estimated variance components from 1. AIC = Akaike Information Criterion. AIC describes the model with best goodness-of-fit combined with Parsimony. The submodel with the lowest AIC is the best fitting; df = change in degrees of freedom between submodel and full model; χ2 = chi-square goodness-of-fit statistic; P = probability that Dχ2 is zero; CI = confidence interval.

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toms of depression, anxiety, and mood swings as estrogen levels begin to fluctuate during perimenopausal years and further decline at menopause, offering a potential cause-effect mechanism for PPS.

The present findings should be considered in light of several methodological limitations, particularly our assessment method. We would ideally have used a more detailed questionnaire in order to improve accuracy in ascertaining our 'PPS' phenotype. Especially the time window between cessation of coitus and occurrence of the symptoms needs to be properly defined, as well as the duration of the symptoms and the role of orgasm in the development of PPS. It is important to note that our study assessed symptoms occurring post-coitally (with or without experienced orgasm) and not specifically after orgasm, hence do not cover a post-orgasmic condition. Given the hormonal changes that happen at the time of orgasm, which tend to be different from sexual excitement, this could be important (Exton et al., 1999; Krueger et al., 2003). To address this issue we conducted post-hoc analysis using a questionnaire sent to the twins in 2008 and asking about frequency of orgasm during intercourse. This revealed that 21 (18% — in accordance with estimate figures found in other studies such as Dunn et al., 2005) of the 115 women with PPS reported never having experienced orgasm during vaginal intercourse, indicating that symptoms of PPS can occur independently from orgasm. The term 'resentment' could further have been misinterpreted by some participants, but the fact that we clearly specified the symptoms (irritability, depression, crying, mood swings) makes a misinterpretation of the question and the terminology unlikely. Furthermore, several other multidimensional constructs — for example, extroversion or anxiety — have been successfully ascertained with simple questions in the past, similar to this study (Hampson, 2005).

Another general study limitation is the fact that we were not able to directly determine the direction of causality between our 'risk measures' and PPS. Further prospective research is needed to further investigate the link between PPS and, for example, relationship satisfaction. We were further unable to assess some potential confounders such as, for example, duration of relationship, due to unavailability of the data. Duration of relationship might substantially impact relationship dissatisfaction and thus confound the association between PPS and relationship dissatisfaction. Our response rate was relatively low (50%) compared with other medical surveys, but respectable compared with other sex surveys (Baileys et al., 2000; Dunne et al., 2000; Witting et al., 2008). Underreporting bias is commonly known to affect the results of questionnaire surveys on sexual function and behavior and arise from personal concerns about social destigmatisation and from social desirability (Bancroft & Coles, 1976; Goldstein et al., 2005). If underreporting was existent in our study, then the resulting prevalence estimates of PPS would tend to be slightly underestimated. However, this would be true for every study in this field.

Conclusion

In spite of potential methodological limitations, the prevalence of PPS found in our study and the fact that a considerable proportion (up to half) of these women felt distressed about the condition, clearly indicates the need for more research on the nature of this phenomenon. Our results show that PPS is influenced primarily by interpersonal and contextual variables. It seems that the most important targets for intervention and prevention are factors such as relationship quality and satisfaction and history of emotional or physical abuse. Despite identification of both genetic and environmental contributions, many of the precise factors involved remain to be uncovered.

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