Predictors of Sexual Desire Disorders in Women

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ABSTRACT

Introduction. A historic belief was that testosterone was the "hormone of desire." However, recent data, which show either minimal or no significant correlation between testosterone levels and women's sexual desire, suggest that nonhormonal variables may play a key role.

Aim. To compare women with hypoactive sexual desire disorder (HSDD) and those with the recently proposed more symptomatic desire disorder, Sexual Desire/Interest Disorder (SDID), on the relative contribution of hormonal vs. nonhormonal variables.

Methods. Women with HSDD (N = 58, mean age 52.5) or SDID (N = 52, mean age 50.9) participated in a biopsychosocial assessment in which six nonhormonal domains were evaluated for the degree of involvement in the current low desire complaints. Participants provided a serum sample of hormones analyzed by gas chromatography-mass spectrometry or liquid chromatography/mass spectrometry/mass spectrometry.

Main Outcome Measures. Logistic regression was used to assess the ability of variables (nonhormonal: history of sexual abuse, developmental history, psychosexual history, psychiatric status, medical history, and sexual/relationship-related factors; hormonal: dehydroepiandrosterone [DHEA], 5-diol, 4-dione, testosterone, 5-α-dihydrotestosterone, androsterone glucuronide, 3α-diol-3G, 3α-diol-17G, and DHEA-S; and demographic: age, relationship length) to predict group membership.

Results. Women with SDID had significantly lower sexual desire and arousal scores, but the groups did not differ on relationship satisfaction or mood. Addition of the hormonal variables to the two demographic variables (age, relationship length) did not significantly increase predictive capability. However, the addition of the six nonhormonal variables to these two sets of predictors significantly increased ability to predict group status. Developmental history, psychiatric history, and psychosexual history added significantly to the predictive capability provided by the basic model when examined individually.

Conclusions. Nonhormonal variables added significant predictive capability to the basic model, highlighting the importance of their assessment clinically where women commonly have SDID in addition to HSDD, and emphasizing the importance of addressing psychological factors in treatment. **Brotto LA, Petkau AJ, Labrie F, and Basson R. Predictors of sexual desire disorders in women. J Sex Med 2011;8:742–753.**

Key Words. Sexual Desire; Hypoactive Sexual Desire Disorder; Sexual Dysfunction; Hormones; Psychological Factors

Introduction

In light of increasing data on the variable nature of women's sexual response [1–3], there is ongoing debate regarding optimal definitions of sexual dysfunction—especially of sexual desire disorder [4,5]. Hypoactive sexual desire disorder (HSDD) is currently defined by the American Psychiatric Association's Diagnostic and Statistical Manual IV-TR (DSM-IV-TR) as "persistently or recurrently deficient (or absent) sexual fantasies and desire for sexual activity" which causes "marked distress or interpersonal difficulty" [6]. This definition of HSDD has been criticized for being too narrowly focused on sexual behavior [1,2,5,7] and sexual fantasies, as the latter are often

deliberately evoked as a means of boosting sexual arousal [8,9]. In response to these criticisms, an international multidisciplinary group of sexuality researchers and clinicians proposed that Sexual Desire/Interest Disorder (SDID), defined by low sexual desire, absent sexual fantasies, *and a* lack of "responsive desire," may more accurately capture sexual desire concerns in women [10]. The proposed inclusion of lack of responsive desire in the definition is based on numerous findings testing the Incentive Motivation Model [11] which posits that all sexual desires are responsive and there is no such thing as "spontaneous desire"; even seemingly spontaneous desire occurs in response to sexual stimuli.

Because this definition of SDID captures the women who neither experience any sexual desire prior to sexual activity nor any desire/excitement during a sexual encounter [12], women with SDID may experience a "more severe" form of desire dysfunction than women with DSM-IV-TR-defined HSDD. Empirical validation of SDID does not yet exist, although a recent review of the evidence of components of women's satisfying sexual experiences concluded that low/absent desire prior to engaging in sex plus the absence of sexual fantasies (i.e., criteria listed in the DSM-IV-TR definition of HSDD) did not preclude satisfaction nor merit the label of "disorder" [5]. Not unexpectedly, women who identified themselves with a model of sexual response which emphasizes responsive sexual desire were more likely to score lower on the desire subscale of the Female Sexual Function Index which is based on DSM-IV-TR criteria [13].

Desire decreases with age [14,15]. Women may nevertheless have satisfactory sexual experiences if they are motivated to start without initial desire but do become sufficiently aroused. Motivation is complex, varied, and dependant on many factors [16–18]. Therefore, how willingly a given woman without anticipatory desire embarks on sexual activity will be determined by these factors. Once sexually engaged, again, multiple factors modulate the brain's processing of the sexual stimuli [5,12,17,18] such that arousal and desire may or may not be experienced. Thus, we hypothesized that SDID would have a different etiology than HSDD, and the goal of the present study was to begin to examine whether SDID and HSDD are distinct clinical syndromes by testing whether they have differing (potentially etiological) correlates.

A variety of studies have explored hormonal and nonhormonal (e.g., sociocultural or psychological) predictors of low sexual desire in women. However, recent understanding of women's varied experience of "desire" and common conflation of desire and arousal [9,19] calls into question what actually has been examined. Small studies have reported that an ovulatory increase in sexual motivation coincides with the brief elevation in serum testosterone, thus reinforcing a long believed role for testosterone in promoting women's desire [20–23]. In contrast, larger studies with a broad cross-section of ages found no significant correlation between testosterone and measures of sexual functioning [24–26]. Until recently, no studies have used mass spectrometry methods, which afford the most accuracy [27]. Thus, there has remained uncertainty over the role of hormone deficit, and in particular androgens, in women's complaints of low desire. In the only large study that assessed HSDD among participants, there was no significant difference in mass spectrometry-measured serum testosterone levels between the clinical group (N = 121) and a sexually healthy control group (N = 124) [28]. Moreover, androgen metabolites—the major one being androsterone glucuronide (ADT-G), were similar in women with HSDD and controls. Androgen metabolites reflect the total androgen pool [29], including testosterone of ovarian origin plus testosterone made intracellularly in peripheral tissues from dehydroepiandrosterone (DHEA) and other precursor hormones of mainly adrenal origin [30].

In addition to the influence of testosterone, other precursors of androgens, such as DHEA, DHEA sulfate (DHEA-S), androstenedione (A4), and the most potent natural androgen 5-α-dihydrotestosterone (DHT), have been studied for their potential role in women's sexual desire. In a large cross-sectional study, low sexual desire was significantly associated with having a DHEA-S level below the 10th percentile [24], even though the majority of women with low DHEA-S did not report low desire. A more recent cross-sectional study of 245 women found significantly lower DHEA-S and near significantly lower DHEA in women with HSDD compared to controls [28]. Because neither mass spectrometry serum testosterone nor its metabolites differed significantly between the groups, the authors concluded that alternative explanations for the lower DHEA were needed, and that it may reflect dysregulation in the hypothalamic-pituitary-adrenocortical axis related to past stress.

Research examining the psychosocial correlates of low desire has identified attitudes, negative cog-

nitions, mood, well-being, self-image, feelings for partner, partner sexual functioning, and distress about the relationship as well as about one's own sexuality [31–34], to name a few factors, as being significantly associated with sexual desire in women. In a recent large study of Brazilian women, cardiovascular disease, breast cancer, posttraumatic stress disorder, less education, being older, being married, and inadequate sexual information during childhood were associated with increased risk of having HSDD [35]. Although this body of research has been useful for informing clinical practice and generating research directions, in general, such an approach of examining correlates individually provides an incomplete picture of the pathophysiology of low sexual desire.

There has been little research to date examining the relative effects of hormonal vs. nonhormonal predictors of women's sexual functioning, and no studies have examined these relative effects in predicting to HSDD vs. SDID groups. One exception to the first item is the prospective and longitudinal Melbourne Women's Midlife Health Project, which assessed a variety of hormonal, individual, and relational variables separately, and relative to one another, on women's sexual functioning. Women's positive feelings for a partner had significant effects on sexual desire, and served to buffer against the deleterious effects of menopausal symptoms [33]. Whereas vaginal dryness and dyspareunia had significant negative effects on sexual desire, there were no effects of any hormonal variable. Because the effects of menopausal symptoms on desire were only indirect (via well-being and sexual responsivity), the authors argued that the analysis of predictors of women's sexual desire must incorporate analytic models that can accommodate many variables (rather than a few). A later study found the single most predictive variable in current sexual response (a composite score of sexual desire, arousal, enjoyment, and orgasm) [36], and current sexual desire [37] was the woman's prior level of sexual response.

In a study of 740 American women aged 45–94, sexual desire was measured by two questions: "How frequently do you feel sexual desire?" and "How frequently do you have sexual thoughts, fantasies, or erotic dreams?" [38]. Using a two-stage regression with biomedical and relational predictors entered in stage 1 and the psychological/attitudinal predictors additionally entered in stage 2, the most significant predictors were attitudes toward sex, age, and having a sexual partner. The effect of age on desire was not accounted for by illness or medication use.

Overall, the literature confirms the importance of psychological and relational predictors of desire. Given SDID is defined as both a lack of desire according to the DSM-IV-TR (as lack of fantasies and desire for sex) as well as an inability to experience sexual desire in response to sexual activity and/or exposure to sexually competent stimuli; we predicted that women with SDID may show more involvement of nonhormonal variables compared to women with HSDD.

In light of the literature showing a lack of correlation between testosterone and women's sexual desire, we hypothesized a lack of correlation between the hormonal and nonhormonal variables, and significant positive correlations among the nonhormonal variables. The nonhormonal variables studied were: developmental history (significant life experiences as a child and adolescent), psychosexual history, psychiatric history and current status, history of sexual abuse, current relationship/contextual factors, and medical contributors. Participants in this study were those who participated in an earlier study comparing women with and without HSDD on androgen levels [28]; thus, the present study represents secondary/exploratory analyses. An exploratory analysis was done, without any a priori predictions, about which of the nonhormonal variables were predictive of group membership.

Methods

Participants

From January 2005 to March 2009, women being assessed for low sexual desire and/or low sexual arousal at a sexual medicine treatment center in a large Canadian metropolitan city were eligible to participate. The sexual dysfunction must have been present for at least 6 months and acquired after age 35. Because participants in this study were the subject of a primary paper comparing women with and without sexual desire disorder on androgen levels [28], factors known to result in significantly altered androgen activity were used as exclusion criteria. These included: current Major Depressive Episode as assessed by interview and completion of the Beck Depression Inventory (BDI-II) [39] using a cutoff score of 19 (where scores greater than 19 denote moderate to severe depression [40]), smoking, body mass index <18.5 or >29.9, current use of any hormone (e.g., oral contraceptives, hormonal replacement therapy) or

medications known to affect sexual function including antidepressants, presence of medical conditions known to interfere with sexual function, situational sexual dysfunction (i.e., the dysfunction is partner- or context-specific), chronic dyspareunia, substance abuse, cigarette smoking, severe relationship discord, or lack of English fluency. Women with and without partners were eligible.

Measures

Self-Report Measures of Sexual Response, Mood, and Relationship Satisfaction

Sexual desire was measured with the *Sexual Interest and Desire Inventory* (SIDI) [41], a 14-item questionnaire that assesses sexual desire in women including one nonscored item assessing intercourse frequency. Possible total scores range from 0–51, with higher scores indicating higher levels of sexual interest. The SIDI has excellent internal consistency (Cronbach's $\alpha = 0.90$). Item–total correlations are high for "Receptivity," "Initiation," "Desire–frequency," "Desire–satisfaction," "Desire–distress," and "Thoughts–positive" ($r \geq 0.7$); good for "Relationship–sexual," "Affection," "Arousal–ease," and "Arousal–continuation" ($r > 0.5$); and poor for the orgasm item ($r = 0.1$) [41].

Sexual arousal was measured with the *Detailed Assessment of Sexual Arousal* (DASA), an unpublished questionnaire that has been found to significantly differentiate aspects of sexual arousal in women [42]. Subscales include "Mental excitement," "Genital tingling/throbbing," "Genital wetness," and "Pleasant genital sensations." Because the DASA is an unvalidated instrument, data on its psychometric properties are not available.

The *Dyadic Adjustment Scale* (DAS) [43] is considered the gold standard in measuring relationship adjustment. It consists of 32 items measuring four domains, "Dyadic Consensus," "Dyadic Satisfaction," "Dyadic Cohesion," and "Affectional Expression." Total score range is from 0–151, with higher scores indicating higher levels of dyadic adjustment. Total score reliability (Cronbach's alpha) is 0.96, with subscales ranging from 0.94 to 0.73 [43].

The BDI-II [39] is a 21-item inventory that measures the degree of depressive symptoms with items specifically designed to be consistent with criteria for major depressive disorder, as defined by the DSM-IV-TR [6]. Items are rated on a 4-point Likert scale (0–3) with higher scores indicating a greater severity of depressive symptoms, and a score range of 0–63. The BDI-II has good internal consistency (Cronbach's $\alpha = 0.81$) and concurrent validity ($r = 0.74$).

The *Positive and Negative Affect Scale* (PANAS) [44] is a 20-item measure of positive and negative mood. It has high internal consistency (Cronbach's alpha), with a range of 0.86 to 0.90 on the Positive Affect subscale, and 0.84 to 0.87 for the Negative Affect subscale. Test–retest reliability when administered over an 8-week retest interval was good at 0.68 for positive affect and 0.71 for negative affect [44].

Clinical Interview

Participants took part in a 2-hour "semi"-structured interview with their partner (or alone, if there was no ongoing relationship), each being seen separately as well as together. In addition to the assessment of sexual desire, motivation, arousal/excitement, genital arousal/wetness, genital sexual sensitivity, orgasm, pleasure, and genital pain, the clinician assessed a variety of contextual factors that were potentially associated with the sexual complaints. These six domains included: (i) current sexual and relationship context (relationship discord, communication, intimacy, partner-related sexual difficulties, sexual skills, sexual environment); (ii) psychosexual history (i.e., sexual debut, past sexual experiences); (iii) developmental history (i.e., assessment of early attachment relationships, significant events as a child); (iv) history of sexual abuse; (v) current and past psychiatric status (i.e., diagnoses of Axis I and II disorders, use of psychotropic medications, life stressors); and (vi) current and past medical status (including medications, major medical illnesses, surgeries). The clinician-determined influence of each of these six domains on the current sexual complaints was rated on a 1 (if the domain was nonexistent or if it was deemed unlikely to contribute to the current sexual concerns) to 7 (if the domain was deemed to potentially fully account for the current sexual concerns) Likert scale. Of note, item (vi) was usually rated as a 1 given the exclusion criterion of significant medical comorbidity, but occasionally, past medical factors had unexpectedly contributed. Clinicians involved in recruitment (five in total) attended a 2-hour training workshop at the start of the study to ensure consistency in rating.

Hormonal Domains

We measured serum steroid levels of DHEA, 5-diol, 4-dione, T, and DHT, which were analyzed

by gas chromatography-mass spectrometry. ADT-G, 3α-diol-3G, 3α-diol-17G, and DHEA-S were analyzed by liquid chromatography/mass spectrometry/mass spectrometry using TurboIonSpray as per methods described previously [29,45–47].

Procedure

As previously described [28], participants in this study were either seeking treatment at a large Canadian tertiary care treatment center for sexual dysfunction or they responded to online and in-print advertisements in the community and via a hospital list-serve (87.3% of participants were recruited from the treatment center). For women who were seeking treatment, the study was described to them at the end of their clinical interview. Women were diagnosed with HSDD alone (D1), HSDD plus SDID (D2), or genital sexual arousal disorder based on the clinical interview. Only women with HSDD and HSDD plus SDID were analyzed in the current paper due to a small sample size for women with exclusively genital arousal complaints. Eligible women went on to receive any of a variety of forms of treatment for the presenting complaints following completion of the assessment (e.g., couple sex therapy; group therapy comprising four sessions of cognitive behavior therapy, mindfulness, and education; individual treatment; one-session education; or bibliotherapy). We have previously published data on the groups' androgen and metabolite levels in comparison to a recruited sexually healthy control group [28]. Here, we focus on the nonhormonal variables not previously reported.

Women who responded to advertisements were first screened via telephone by a masters level study coordinator who explained the procedures and determined eligibility criteria. If prospective participants passed the telephone screen, they were then scheduled for the same 2-hour clinical assessment as the women recruited from our treatment center; however, the former were not requesting, nor were they offered, treatment.

Information on the venepuncture procedure was provided following the clinical interview for all participants. Written instructions were included in the questionnaire package given to each participant, and women were asked to attend a local laboratory on any weekday morning between 8 AM and 10 AM (on days 8–10 of the menstrual cycle when relevant), where 5 mL of serum would be withdrawn. Technicians at the laboratory were given instructions to package the withdrawn serum on dry ice, and the samples were sent to the laboratory of the third author for analysis. Following the clinical interview, all women were given the questionnaire battery to take home and complete and mail back to the study coordinator in a stamped self-addressed envelope that was provided.

Steroid measurements were performed at the Laboratory of Molecular Endocrinology and Oncology, Laval University Hospital Research Centre, Quebec City, Canada, under Good Laboratory Practice-validated methodology. Extraction and analysis of conjugated and nonconjugated steroids were performed as previously described [28].

No remuneration was given; women were provided with written results and explanation of their hormone analyses and a copy was sent to the family physician if requested. Approval for the study was obtained from the University of British Columbia Clinical Research Ethics Board.

Statistical Methods

Comparisons of groups (HSDD alone—D1 vs. HSDD plus SDID—D2) were based on Student's *t*-test and analysis of covariance (for continuous variables) and Fisher's exact test (for categorical variables). Associations between continuous variables were assessed using Pearson correlation coefficients. Logistic regressions were used to assess the ability of variables to predict membership in D2 vs. D1; that is, the logistic regression model fits were for the log odds of being in group D2 vs. being in group D1. Interest focused on three sets of variables: Set 1 (demographics)—age and relationship length; Set 2 (hormonal)—nine hormonal variables of main interest; Set 3 (nonhormonal)—six nonhormonal variables of interest (i.e., the six domains assessed during the clinical interview). Comparisons of nested logistic regression fits were based on the likelihood ratio (LR) chi-square test and the significance of individual regression coefficients were assessed using Wald tests.

Results

Demographic Characteristics

A total of 110 participants were included with a mean age of 51.7 years. Of 108 women who provided information on relationship status, 97.2% were currently in a heterosexual relationship with a mean duration of 21.5 years. Ethnic composition was 95.1% Euro-Canadian. Further demographic

Table 1 Demographic characteristics of participants

Variable	HSDD only (D1)	HSDD plus SDID (D2)
Age (years): n, mean (SD)	58 52.5 (7.1)	52 50.9 (7.8)
Relationship length (years): n, mean (SD)*	54 21.3 (10.9)	51 21.6 (10.7)
Highest level of education: n (%)		
Less than high school	0 (0%)	2 (4%)
High school	7 (14%)	7 (14%)
Some college/university	29 (58%)	32 (64%)
Postgraduate level	14 (28%)	9 (18%)
Relationship status: n (%)		
Single/separated/divorced	3 (5.7%)	3 (6.0%)
Married or common-law	50 (94.3%)	47 (94.0%)
Ethnicity: n (%)		
Euro-Canadian	50 (96.2%)	47 (94.0%)
Asian	2 (3.8%)	2 (4.0%)
Biracial	0 (0.0%)	1 (2.0%)

*For women who were in a relationship at the time of the study.

information, by group, is provided in Table 1. The two groups did not significantly differ for any of these demographic variables; notably, there was no difference in age between the two groups ($P = 0.24$).

Self-Report Measures of Mood, Sexual, and Relationship Function

As shown in Table 2, women with SDID had significantly lower scores on the SIDI compared to women with HSDD, as predicted. SIDI scores in the SDID group were lower, on average, for all of the DASA subscales and significantly so on the first two (mental excitement and genital tingling) ($P = 0.017$ and 0.029, respectively), while the other two were nearly statistically significant. There were, *however*, no significant group differences on any of the DAS subscales (although, on each subscale, the women with SDID had lower scores, on average), PANAS, or on the BDI-II.

Nonhormonal Data

As shown in Table 3, women with SDID had significantly higher Likert ratings on developmental history ($P = 0.015$), psychiatric history ($P = 0.001$), and psychosexual history domains ($P = 0.007$), indicating greater hypothesized involvement of these factors in the current sexual complaints compared to the women with HSDD alone. There were no significant group differences in history of sexual abuse, medical history, or contextual factors.

Of the 110 participants, 105 (56 in the D1 group and 49 in the D2 group) provided complete data on all the variables to be used in the logistic regression analyses. All results that follow are based on these N = 105 "complete cases".

Correlations Between Hormonal and Nonhormonal Variables

In the full group of participants, all of the hormonal variables were significantly and positively correlated with one another (data not presented). For the nonhormonal variables, abuse was significantly positively correlated with developmental history; developmental history was also significantly positively correlated with psychiatric history, context, and psychosexual history; psychiatric history was also significantly positively corre-

Table 2 Group differences on scores from the Sexual Interest and Desire Inventory (SIDI), Detailed Assessment of Sexual Arousal (DASA), Dyadic Adjustment Scale (DAS), Beck Depression Inventory (BDI-II), and Positive and Negative Affect Scale (PANAS)

Variable	HSDD only (D1) n mean (SD)	HSDD plus SDID (D2) n mean (SD)	*t*	*P*
SIDI*	57 19.1 (8.8)	50 13.1 (7.3)	–3.84	0.0002
DASA-mental excitement†	54 3.8 (1.3)	48 3.1 (1.5)	–2.44	0.017
DASA-genital tingling†	54 3.4 (1.3)	48 2.8 (1.6)	–2.21	0.029
DASA-genital wetness†	53 2.9 (1.5)	48 2.4 (1.4)	–1.75	0.083
DASA-genital pleasure†	53 3.7 (1.6)	48 3.2 (1.7)	–1.72	0.089
DAS-consensus‡	53 50.8 (6.3)	48 48.3 (7.2)	–1.87	0.064
DAS-satisfaction‡	53 38.4 (5.8)	48 37.7 (6.2)	–0.65	0.52
DAS-cohesion‡	53 15.5 (3.4)	48 14.2 (4.8)	–1.54	0.13
DAS-affectional expression‡	53 7.4 (2.8)	48 6.7 (2.9)	–1.29	0.20
BDI-II§	53 8.5 (5.4)	50 8.0 (6.0)	–0.50	0.62
PANAS-positive¶	54 31.7 (9.1)	49 30.4 (8.0)	–0.72	0.47
PANAS-negative¶	54 14.4 (6.5)	48 15.8 (7.3)	0.98	0.33

*Higher scores denote more sexual desire on the SIDI. Scale range: 0–51.
†Higher scores denote more sexual arousal on the DASA. Scale range: 1–7.
‡Higher scores denote better relationship adjustment on the DAS. Scale range: consensus 0–65, satisfaction 0–50, cohesion 0–24, affectional expression 0–12.
§Higher scores denote more depressive symptoms on the BDI-II. Scale range: 0–63.
¶Higher scores denote more emotional affect on the PANAS. Scale range: 10–50.

Table 3 Group differences on scores from the six nonhormonal variables: Abuse, Developmental history (Dev-hist), Medical history (Medical-hist), Psychiatric history (Psych-hist), Context, and Psychosexual history (Psych-sex)

Variable	HSDD only (D1) n mean (SD)	HSDD plus SDID (D2) n mean (SD)	*t*	*p*
Abuse	58 1.8 (1.6)	52 2.0 (1.8)	0.43	0.67
Dev-hist	58 3.6 (2.0)	52 4.5 (1.8)	2.47	0.015
Med-hist	58 3.2 (2.1)	51 3.5 (2.1)	0.73	0.46
Psych-hist	58 3.6 (1.7)	51 4.7 (1.8)	3.33	0.001
Context	58 4.7 (2.3)	52 5.3 (1.9)	1.31	0.19
Psych-sex	58 2.7 (1.9)	49 3.7 (1.8)	2.75	0.007

Ratings were assigned during a comprehensive biopsychosocial interview. Higher ratings reflect more clinician-estimated involvement of that domain in the presenting sexual desire complaints. Scale range: 0–7.

lated with psychosexual history; and context was also significantly positively correlated with psychosexual history (Table 4). In contrast, the only significant cross-correlation between hormonal and nonhormonal variables was between 4-dione and medical history ($r = -0.21$, $P = 0.031$; data not shown).

Prediction of SDID vs. HSDD Group Status Based on Demographic, Hormonal, and Nonhormonal Variables Separately

A logistic regression based on only age and relationship length provided no predictive capability (LR test, d.f. = 2, $P = 0.43$). This was also the case when using only the nine hormonal predictors (LR test, d.f. = 9, $P = 0.62$). In contrast, using only the six nonhormonal variables yielded significant predictive capability (LR test, d.f. = 6, $P = 0.021$). In this six-variable fit, only psychiatric history was significant (Wald test, $P = 0.034$), indicating that women with a higher psychiatric history domain score were more likely to have been diagnosed with SDID compared to HSDD. In particular, for any fixed set of values of the other five nonhormonal predictors, the estimated odds ratio for a one-unit increase in the psychiatric history domain score was 1.32 (95% confidence interval [CI]: 1.02 to 1.70).

Prediction of SDID vs. HSDD Group Status Based on Demographic, Hormonal, and Nonhormonal Variables Collectively

To assess the additional predictive capability of the nonhormonal variables, we then fit two further logistic regressions in which the nine hormonal variables were first added to the demographic variables (age and relationship duration), followed by the further addition of the six nonhormonal variables. Adding all nine hormonal variables to the demographic variables did not significantly increase ability to predict group status (LR test, d.f. = 9, $P = 0.54$). However, when the six nonhormonal variables were added to the two demographic and nine hormonal variables, we found a significant increase in the ability to predict to group status (LR test, d.f. = 6, $P = 0.037$). However, none of the six nonhormonal variables were individually significant in this 17-variable fit (only psychiatric history approached significance: Wald test, $P = 0.066$), likely owing to the simultaneous fitting of many intercorrelated predictors.

Two approaches were then carried out to identify the source of the additional predictive capability provided by the set of six nonhormonal variables. In the first approach, each of the nonhormonal variables was separately added to the basic model that included the two demographic and nine hormonal variables. In these separate analyses, we found that each of developmental history ($P = 0.017$), psychiatric history ($P = 0.004$), and psychosexual history ($P = 0.012$) added significantly to the predictive capability provided by the basic model; women with higher scores on each of these three nonhormonal variables were more likely to be diagnosed with SDID compared to HSDD.

In the second approach, a stepwise forward model selection procedure based on Akaike's Information Criterion considered all members of the set of six nonhormonal variables for addition to the basic model that included the two demographic and nine hormonal variables. In this analysis, both psychiatric history (β estimate = 0.30,

Table 4 Correlation matrix for nonhormonal variables for the full sample of women (N = 105)

	Abuse	Developmental history	Medical history	Psychiatric history	Context
Developmental history	**0.25**	—	—	—	—
Medical history	0.02	–0.08	—	—	—
Psychiatric history	0.01	**0.33**	0.04	—	—
Context	–0.05	**0.22**	0.03	0.18	—
Psychosexual history	0.12	**0.43**	0.12	**0.35**	**0.25**

Correlations in bold are significant at $P < 0.05$.

standard error [SE] = 0.14, $P = 0.032$) and psychosexual history (β estimate = 0.21, SE = 0.13, $P = 0.11$) were included in the final fitted model, with both regression coefficients indicating that women with higher scores on these nonhormonal variables were more likely to be diagnosed with SDID compared to HSDD. In particular, for any fixed set of values of the other predictors, the estimated odds ratio for a one-unit increase in the psychiatric history domain score was 1.35 (95% CI: 1.03 to 1.78) and for a one-unit increase in the psychosexual history domain score was 1.23 (95% CI: 0.95 to 1.59).

Discussion

This study represents, to our knowledge, the first empirical report on the characteristics of otherwise physically healthy women with a diagnosis of HSDD (as per the DSM-IV-TR; [6]) vs. women meeting criteria for SDID (as per the International Consultation 2003 [10]). Although SDID is not a currently accepted diagnosis as per the American Psychiatric Association, the criteria for SDID parallel those proposed for Sexual Interest/Arousal Disorder for DSM-5 which focus on the "responsive" nature of sexual desire [5]. Here, we have described the relative contributions of hormonal and nonhormonal variables in predicting group membership. Participants with HSDD alone were comparable to the participants with SDID on demographic variables in terms of age, relationship duration, level of education, and ethnicity. There were also no significant group differences on relationship satisfaction or mood. The full sample can be described as a predominately middle-aged, Euro-Canadian, nondepressed, educated group of women in long-term relationships and without medical conditions likely to impair sexual function; thus, conclusions about the larger population of (treatment-seeking) women must be made tentatively.

As predicted, there was evidence of more significant symptoms of low desire and less mental sexual excitement among women with SDID, when measured with self-report questionnaires that asked women to reflect on the previous weeks. Given that SDID is defined as having symptoms of HSDD (i.e., reduced or absent sexual fantasies and desire for sexual activity) plus the inability to become sexually excited in response to sexual stimuli (i.e., to not experience "responsive" desire), lower scores on these two domains of sexual functioning are not surprising.

In addition, women with SDID had significantly lower scores on the genital tingling subscale of the DASA. Current DSM-IV-TR criteria for HSDD and female sexual arousal disorder do not assess genital tingling, and available epidemiological studies on sexual problem frequency have not yet assessed this symptom; thus, the extent to which genital tingling is impaired among women with sexual dysfunction is unknown. It is possible that the impairment in mental sexual excitement seen in women with SDID leads to an inability to detect genital tingling—however, genital congestion underlying the latter may not, in fact, be impaired. Data suggesting the lack of concordance between genital and subjective sexual excitement in women [48] support this speculation.

In the basic model, age, and relationship length did not significantly predict to HSDD vs. SDID. Thus, although relationship duration and age are significantly negatively associated with low desire [49], our findings suggest that these variables may not be helpful for predicting whether women also lack responsive desire and have a more "severe" form of desire dysfunction.

The present data confirmed our hypothesis of a greater contribution of nonhormonal variables in predicting group membership to SDID compared to HSDD; the nine hormonal variables did not significantly add to the prediction of group after age and relationship duration were included in our logistic regression analyses. However, compared to women with HSDD, women with SDID had greater clinician-rated involvement of developmental history, psychiatric history, and psychosexual history in accounting for their current symptoms of sexual dysfunction (Table 3) and these differences were reflected in significant findings for those variables when individually added to the base model. This suggests that these three variables individually may be important for determining the degree and breadth of desire dysfunction such that more negative early childhood factors, more past and/or present psychiatric symptoms, and a more negative sexual history are each associated with less ability to trigger sexual desire. To explore this further, we conducted an exploratory analysis to assess which of the nonhormonal variables were most predictive of group membership. Stepwise model selection indicated that psychiatric history was the primary contributor to this enhanced predictive capability with psychosexual history also included in the selected model but not contributing significantly.

Unlike prior studies that have explored the relative effects of hormonal and nonhormonal variables in predicting sexual responsivity [36], our study used a clinician-determined rating of nonhormonal involvement across six domains and we also assessed a past history of these factors in addition to their current involvement. This represents a strength of the current study given that each participant underwent a detailed in-person biopsychosocial interview alone and together with her partner. Prior research, which has relied on brief self-report questionnaires to assess these often complex constructs, may have inadequately assessed some of these domains and therefore understated, or even missed, their involvement.

Psychiatric history was the sole nonhormonal variable that significantly predicted group membership when all demographic, hormonal, and nonhormonal variables were considered collectively. Psychiatric factors have long been implicated in women's low sexual desire, and a recent review supported the role of depressive, anxious, psychotic, bipolar, and personality disorder symptoms in women's sexual dysfunction [50]. In the Global Study of Sexual Attitudes and Behaviors, depression was the single strongest predictor of every sexual dysfunction in both men and women [32]; the odds ratio of depression in predicting low sexual desire in women ranged from 1.3 to 2.2. In a recent large cross-sectional representative study of American women, the prevalence of all sexual dysfunctions, and in particular desire disorder, was higher among those women with clinical depression [51].

In Bancroft et al.'s large epidemiological study of distress about sex, emotional well-being (which included psychiatric factors) was one of the strongest predictors of sexual distress [31]. Anxiety, another facet of psychiatric history assessed in our sample, is also highly comorbid with sexual dysfunction in women [52,53]. The impact of psychiatric status is bidirectional such that increasing sexual symptoms may further lower mood, increase anxiety, and other psychiatric symptoms. In this current study, we found that for every one unit increase in the woman's psychiatric history score (during her interview), the odds of her being in the SDID group increased by 35% (95% CI: 3% to 78%).

Although they did not add significant predictive ability when considered with all other variables, developmental history and psychosexual history significantly predicted group membership when considered individually. What are the means by which these two variables might be predictive? Bowlby's Attachment Theory [54] indicates that there is a universal human tendency to seek closeness to another person and to feel secure when that person is present. Problems in early parent–child attachment may therefore lead to problems in how the child later develops intimate relationships as an adult. Dysfunctional very early relationships with caregivers can have a large variety of psychological consequences among which later sexual dysfunction maybe one aspect. Indeed, there is evidence that attachment-related concerns about acceptance and closeness have a strong influence on sexual experiences [55], sexual and relationship satisfaction [56], and sexual communication [57] within a romantic relationship. Because early attachment was a major aspect of the developmental history assessed in the current study, one might conclude that for women with more negative early attachment relationships, they may be especially vulnerable to developing a more severe form of sexual desire disorder as an adult.

The finding that psychosexual history was a significant predictor to the SDID group when considered individually is consistent with recent research which has found that psychosexual history was a significant predictor of having HSDD vs. no HSDD [35] in that those women who received adequate sex education as a child were less likely to have HSDD. In our study, this domain included the woman's perception and experience of first sexual experiences and all those leading to the current (sexual) relationship. Some studies have shown a negative impact of early sexual debut on adult sexual functioning, particularly for women [58,59], although having a large number of past sexual partners is not necessarily associated with greater risk of sexual dysfunction [60]. Because emotions experienced during early sexual encounters may be particularly influential [61,62], our findings suggest that, perhaps, early sexual experiences in which the woman had especially strong negative affect (e.g., a partner may have had poor sexual skills or ridiculed her sexual technique or inability to respond adequately) may lead to a more severe form of sexual desire disorder. Because "psychosexual history" included a number of different aspects of the woman's past sexual history (but did not include history of sexual abuse), the precise mechanisms by which it is associated with SDID are unknown.

There are limitations in this study that must be considered. First, our biopsychosocial interview, although a standard component of assessment for

all patients presenting to our treatment center, has not been empirically tested for its validity or reliability. Thus, there may be slight differences among the assessors in how ratings were determined. Because all assessors underwent a 2-hour training workshop at the start of data collection and have worked extensively together as cotherapists, the impact of interrater variance was likely minimal. One must also recognize the highly selective sample of women seeking treatment in a tertiary care center given that many factors which are commonly associated with sexual dysfunction (e.g., antidepressant use, medication use, etc.) were exclusion criteria (note that some but not all women had previously sought treatment from a primary care or other specialty provider). Additionally, approximately 95% of the participants were from Euro-Canadian ethnic backgrounds, and there are ethnic differences in the prevalence and correlates of sexual desire [32,63]. Thus, caution should be exercised in generalizing the findings to the larger population of women seeking treatment for sexual dysfunction. However, we wished to examine factors influencing sexual disorders rather than sexual symptoms from relationship discord, mood disorder, medications, and debility from medical illness. When the latter are present, they are the focus of therapy and any diagnosis of innate sexual disorder is deferred. Post hoc power analyses indicated that this study may not have been adequately powered to detect effects of the nonhormonal predictors of the magnitudes that appear to be present in this population[1], as the size of this study was determined by our original study that was adequately powered for its primary analyses of interest (i.e., comparing women with and without sexual desire disorder on androgens and their metabolites) [28]. Thus, future studies based on larger samples of women are needed in order to draw firm conclusions about predictors of HSDD vs. SDID. Finally, the studied sexual category, SDID, is not a formally recognized diagnostic category and it has not undergone the appropriate field trials that would be necessary to validate it. In this study, we conceptualized SDID as a "more severe" form of HSDD given that it includes the lack of responsive sexual desire in addition to reduced/absent interest in sex and sexual fantasies.

[1]For the largest model fit, for example, where all the demographic, hormonal and non-hormonal predictors were included, the odds ratios for the individual predictors would have had to be at least 1.4 to 1.5 to be detectable with 80% power.

Conclusion

Our findings contribute important information to the literature on sexual desire disorder in women. They suggest that psychosexual but not hormonal variables are more predictive of severity of desire dysfunction, that hormonal variables alone do not predict group status, and that among the nonhormonal factors, psychiatric status is the single strongest predictor of having SDID. Moreover, psychosexual history and developmental history are also predictors of SDID when considered individually. The clinical implications of the findings are clear: these nonhormonal factors must be assessed among women presenting with concerns about their sexual desire as they may play an important etiological role in more severe disorders of sexual desire. Although it remains to be studied, the findings also point to the importance of addressing these aspects of the woman's biopsychosocial context during treatment.

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