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Diminished anticipatory pleasure predicts anhedonia in women with endometriosis: a case-control study

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Abstract

Background Anhedonia, a diminished response to pleasurable stimuli extensively studied in mental disorders and chronic pain, has been reported as prevalent among patients with deep endometriosis. Anhedonia is considered a multifaceted transdiagnostic construct involving cognitive, affective, and behavioral processes related to the anticipation and experience of pleasure. In this study, we aim to expand on previous findings by exploring the presence of anhedonia in patients with ultrasound diagnosed (UD) endometriosis compared to a control group, and by examining how its anticipatory and consummatory components contribute to the loss of pleasure in this condition.

Methods A total of 216 patients with UD endometriosis and 229 controls were recruited from a hospital gynecological service. Sociodemographic and clinical information was collected, and participants completed an online survey assessing anhedonia using a gold-standard measure, the Snaith-Hamilton Pleasure Scale (SHAPS). To assess anticipatory and consummatory components, the Temporal Experience of Pleasure Scale (TEPS) was administered, along with additional psychological measures of mood and anxiety. A moderated multiple regression model was performed to predict anhedonia scores based on TEPS subscales and diagnosis status. An additional model including anxiety, depression and dysmenorrhea was also conducted. Analyses were conducted using R version 4.4.3 and RStudio, with p-values set at < 0.05 .

Results Clinically pathological anhedonia was more prevalent in patients with UD endometriosis than in controls (31% vs. 15%, $p < .001$). UD endometriosis participants also reported greater menstrual pain, more chronic pain comorbidities, higher anxiety, and increased use of mental health services and hormonal treatments (all $p < .05$). In moderated regression analyses, UD endometriosis diagnosis, lower anticipatory pleasure, and lower consummatory

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pleasure significantly predicted anhedonia ($p < .05$), while depression was not predictive. A significant diagnosis $\times$ anticipatory pleasure interaction ($p < .001$) indicated stronger impairment among patients with UD endometriosis. This effect remained after adjusting for relevant covariates (anxiety, depression and dysmenorrhea). Depression was not predictive of anhedonia levels.

Conclusions Our findings confirm that patients with UD endometriosis consistently show deficits in deriving pleasure from everyday life experiences independently from depressive symptoms and directly influenced by anxiety and marginally dysmenorrhea. Notably, a specific impairment in anticipatory pleasure suggests a disruption in cognitive-affective processing associated with this chronic inflammatory condition. These results support the relevance for multidisciplinary care in UD endometriosis and highlight the transdisciplinary value of assessing anhedonia. Interventions targeting cognitive and affective processes may be essential for restoring health and improving quality of life in this patient population.

Keywords Anhedonia, Endometriosis, Anticipatory reward, Consummatory reward, Dysmenorrhea, SHAPS, TEPS, Pleasure, Hedonic capacity.

Introduction

Avoiding pain is a fundamental driver of human behavior. From an early age, mechanisms aimed at minimizing discomfort and reinforcing the associated learning are established and become hardwired into the brain. In most individuals, pain avoidance is intrinsically associated with survival mechanisms and the maintenance of health and well-being [1, 2]. However, when pain becomes chronic or cyclically inescapable, despite ongoing efforts to eliminate it, previously adaptive behaviors and cognitions may deteriorate leading to increased negative affect and long-term maladaptive changes [3]. In an effort to manage pain, some individuals withdraw from various aspects of daily life, avoiding social interactions, physical activity, certain foods, and even previous enjoyable hobbies. The impact of chronic pain on daily functioning is profound, often triggering a downward spiral in which pleasure and reward processes can be disrupted [4, 5]. This psychological phenomenon—the deterioration of the capacity to feel pleasure—is known as anhedonia.

In recent years, several studies have demonstrated a higher prevalence of anhedonia among individuals with chronic pain compared to healthy controls [4–6]. Notably, a recent study found that anhedonia is abnormally prevalent in women with deep endometriosis (DE) [7]. DE is the most severe phenotype of endometriosis, a chronic systemic inflammatory condition characterized by the presence of endometrial-like tissue outside the uterine cavity, associated with cyclic and chronic pelvic pain, infertility, and a significant impact on mental health [8–15]. According to the study, approximately 30% of patients with deep endometriosis exhibit clinically significant levels of anhedonia, comparable to those observed in other chronic pain populations. This research also revealed a strong association between anhedonia and all pain-related symptoms of deep endometriosis, with pain emerging as a significant predictor of anhedonia in this population. Furthermore, the findings highlighted the

detrimental impact of severe chronic pain, as patients in this subgroup not only presented with the highest levels of anhedonia but also exhibited increased rates of depression and, most notably, anxiety. These results underscore the value of multidisciplinary care in deep endometriosis, integrating psychological and pain management strategies to address the complex interaction of symptoms.

Although traditionally regarded as a symptom of depression and residual schizophrenia, anhedonia has been demonstrated to be transdiagnostic and associated with symptom severity across mental disorders, with potential to explain prevalent psychiatric comorbidities, such as anxiety and depressive disorders, among others [16–21]. Converging evidence also points to a common neural substrate across psychiatric conditions [22]. These findings support examining anhedonia as a cross-cutting dimension across diagnostic categories.

Anhedonia is not a unitary construct, rather it reflects a broader impairment that affects several complex psychological processes, ranging from reward anticipation to experiential engagement, and also implicating learning and memory. In line with this, anhedonia can be parsed into two distinct components: anticipatory and consummatory. The former refers to the motivational or “wanting” state that drives behavior toward environmental or social rewarding stimuli, while the latter corresponds to the actual pleasure experienced during satiation—the “liking” state [23, 24]. Both components of reward processing have been shown to rely on distinct neural circuits [25, 26]. Functional magnetic resonance imaging (fMRI) studies have revealed that reward anticipation is associated with activation in regions such as the anterior cingulate cortex and the ventral striatum, whereas the experience of reward (consummation) primarily engages the orbitofrontal and medial prefrontal cortices, as well as the anterior and posterior cingulate cortices, as demonstrated by a recent systematic review and meta-analysis [27]. Importantly, pleasure circuits overlap with those

involved in the affective and motivational dimensions of pain and its relief, providing neural evidence of pain–reward interactions [1, 28].

This temporal distinction between anticipatory and consummatory components of anhedonia has been well established in both healthy individuals and populations with mental disorders, highlighting the particularly critical role of impaired anticipation in these samples as a key aspect in understanding anhedonia. In individuals with depression, deficits in reward anticipation have been consistently observed and are often linked to reduced motivation, whereas in some cases, no significant differences with controls have been found regarding consummatory pleasure [29, 30]. Interestingly, a recent study showed that anticipatory anhedonia predicted depressive symptoms one week later, and that greater anticipatory deficits strengthened the association between anxiety and depression [17], two well-documented comorbid clinical entities in chronic pain as well as in other populations [31–34]. In addition, the importance of anticipation has also been reported in dysphoric individuals [35] as well as in patients with eating disorders, where again reduced pleasure appears to result primarily from impairments in anticipatory rather than consummatory hedonic capacity [36, 37]. Particularly relevant to the present research, maladaptive reward processes have been proposed as a shared diathesis for both depression and chronic pain, acting as mutually reinforcing mechanisms and potentially explaining their frequent comorbidity [38].

In summary, although there is psychometric evidence of impaired general hedonic functioning in DE, no study to date has investigated how anhedonia presents in UD endometriosis patients compared to a control group without endometriosis. Therefore, the primary objective of this study is to compare levels of anhedonia between the two groups and to assess whether observed differences are consistent with previous research. Additionally, we will explore, for the first time, how the anticipatory and consummatory components of pleasure manifest in each group, and how these components contribute to anhedonia. In this sense, we hypothesize that patients will show impairments in both components of reward processing, with a distinctive impairment pattern relative to controls. Finally, we will explore how anxiety, depression and dysmenorrhea, along with anticipatory and consummatory pleasure, are associated with anhedonia. These findings may serve as a first step toward better understanding the psychological comorbidities of endometriosis, potentially guiding future treatment strategies.

Materials and methods

Participants

Participants with UD endometriosis and controls, all of legal age, were recruited at the gynecological department

of a tertiary university hospital. Prior to agreeing to participate, they were informed that the objective of the study was the psychological characterization of endometriosis. Participation was open to individuals with a UD endometriosis and those without the condition. Patient diagnosis was confirmed via high-resolution transvaginal ultrasound. Controls comprised individuals consulting for contraception, and those diagnosed with benign gynecological conditions, excluding UD endometriosis. All participants in the control group underwent high-resolution transvaginal ultrasound, which confirmed the absence of UD endometriosis lesions. Informed consent was obtained from all participants, who then completed an online survey assessing self-reported sociodemographic, health-related, and psychological variables relevant to the study objectives. The study received ethical approval from our institution prior to participant recruitment (HCB/2021/0674). Recruitment was carried out consecutively over a three-month period, from April to June 2024. All procedures involving human participants were conducted in accordance with the ethical standards of the Ethics Committee for Drug Research of Hospital Clínic and with the 1964 Helsinki Declaration and its later amendments.

Participants were excluded if they had been diagnosed with past or present malignancy, severe psychiatric conditions (including psychosis, bipolar disorder, and substance use disorder), neurodegenerative diseases, cardiovascular or systemic illnesses and menopausal status. Neurodegenerative and psychiatric disorders were excluded due to their possible influence on behavioral assessments.

Measures

Anhedonia was assessed using the Snaith-Hamilton Pleasure Scale (SHAPS), which is considered a gold standard measure of anhedonia. The SHAPS consists of 14 items covering four domains of hedonic experience: interests, social interactions, food/drink, and sensory experiences. Example items include “*I would enjoy my favorite meal*” or “*I would enjoy being with family and friends*”. Each item is self-rated on a four-point Likert scale indicating the level of agreement with each statement (“*definitely agree*,” “*agree*,” “*disagree*,” and “*strongly disagree*”). Total scores range from 14 to 56, with higher scores reflecting greater levels of anhedonia. The internal reliability of the SHAPS has previously been demonstrated in chronic pain samples, with alpha coefficients ranging from 0.78 to 0.92. An alternative scoring method for the SHAPS collapses “*agree*” responses into a score of 1 and “*disagree*” responses into a score of 0, yielding a total score from 0 to 14. In this binary scoring format, higher scores reflect greater levels of anhedonia. A score of 3 or above has been shown to differentiate normal hedonic tone from

clinically significant anhedonia, supporting a more comprehensive assessment and its feasibility in pathological samples [4, 5, 39]. Hereafter, we refer to the long scoring format as SHAPS (1–4) and the short format as SHAPS (0–1).

Anticipatory and consummatory components of hedonic capacity were measured using the Temporal Experience of Pleasure Scale (TEPS) [40]. The TEPS was developed as a trait-level measure designed to capture individual differences in both anticipatory and consummatory experiences of pleasure. It consists of two subscales reflecting these two components. The Anticipatory subscale comprises 8 items, while the Consummatory includes 10 items. Each item presents a statement about likeable experiences, and participants are asked to rate how true each statement is for them on a 6-point scale ranging from “very false for me” to “very true for me”. Higher scores on each subscale indicate greater hedonic capacity. Examples of items from the anticipatory and consummatory subscales, respectively, include: “When I think about eating my favorite food, I can almost taste how good it is” and “I appreciate the beauty of a fresh snowfall.”

Depression and anxiety were assessed using the Hospital Anxiety and Depression Scale (HADS). The HADS is a 14-item self-report questionnaire comprising two subscales of 7 items each measuring depression and anxiety. Respondents rate each item based on how frequently they had experienced each statement during the past week. The HADS has been previously validated in samples with endometriosis [41]. Each subscale yields a score ranging from 0 to 21, with higher scores indicating greater severity of depressive or anxiety symptoms. Because the depression subscale largely reflects anhedonia [21, 42–44], and to avoid overlap with our primary outcome, we computed a modified non-anhedonic depression score, following previous studies [21, 42] by excluding the anhedonia-related items (“I still enjoy the things I used to enjoy,” “I can laugh and see the funny side of things,” “I feel cheerful,” “I look forward with enjoyment to things,” and “I can enjoy a good book, radio or TV programme”). The final non-anhedonic depression score was based on the remaining items (“I feel as if I am slowed down” and “I have lost interest in my appearance”).

A single item assessing the intensity of dysmenorrhea was used as a proxy for endometriosis-associated pain. Respondents rated their pain on an 11-point Numeric Rating Scale (NRS), ranging from 0 to 10, where 0 represents the complete absence of pain and 10 indicates the worst pain imaginable. The 11-point NRS is a widely used and validated tool for assessing both pain and chronic pain severity [45, 46] and has also been previously used in endometriosis samples to assess perceived symptom severity [7].

Data analysis

Descriptive and questionnaire's internal consistency analysis

Descriptive analyses were conducted to summarize both quantitative and qualitative variables. For quantitative variables and outcomes, we calculated means and standard deviations. For qualitative variables, we presented absolute frequencies and percentages. Group differences in quantitative variables were assessed using t-tests, and comparisons of qualitative variables were conducted using χ^2 tests. Statistical significance was set at $p < .05$.

Cronbach's alpha coefficients were calculated to assess the internal consistency of the main psychometric scales (SHAPS and TEPS).

Samples comparison analysis and effect sizes calculation

To compare the main quantitative outcome variables between cases and control groups, we conducted independent-samples t-tests. Effect sizes are reported using Cohen's δ to provide a standardized measure of group differences.

Exploring the relationship between anticipatory and consummatory components of reward and further psychological variables: using moderated multiple linear regression to predict anhedonia

We conducted an a priori multiple linear regression analysis to examine whether the anticipatory and consummatory components of pleasure predicted levels of anhedonia, as measured by SHAPS scores in its continuous short-form version (SHAPS 0–1). The initial model included TEPS anticipatory, TEPS consummatory, GROUP, and the interaction terms (TEPS anticipatory $\times$ GROUP and TEPS consummatory $\times$ GROUP). To improve interpretability and model parsimony, we applied a backward elimination procedure, sequentially removing non-significant predictors.

Prior to analysis, the dataset was screened for missing values. Model assumptions (linearity, homoscedasticity, and multicollinearity) were evaluated using residual diagnostics and variance inflation factors (VIF). Model fit was assessed using R^2 and Adjusted R^2 , and the overall model significance was tested using an F-test. All analyses were performed in R (version 4.4.3) and RStudio.

To further explore the impact of anxiety, depression, and dysmenorrhea on anhedonia scores, and building on the results of the a priori model, we conducted an additional linear regression including the HADS anxiety subscale, HADS non-anhedonic depression subscale, and the NRS measure of dysmenorrhea as predictors. All new variables were entered as continuous predictors.

Table 1 Demographic and clinical variables among women with UD endometriosis and controls

	UD Endometriosis (N = 216)	Non-endometriosis controls (N = 229)	Statistic	p-value
DEMOGRAPHIC VARIABLES				
Age	36.88 (6.48)	32.54 (8.93)	$t(416.21) = 5.89$	< 0.001*
Years of education	21.23 (2.80)	21.36 (2.68)	$t(437.09) = -0.52$	0.606
Civil Status (<i>in a relationship</i>)	170 (78.70%)	159 (69.43%)	$\chi^2(1) = 4.49$	0.026*
Children	63 (29.17%)	66 (28.82%)	$\chi^2(1) < 0.01$	> 0.999
CLINICAL VARIABLES				
Previous Gynecological surgery	122 (56.48%)	41 (17.90%)	$\chi^2(2) = 75.94$	< 0.001*
Hormonal treatment	137 (63.43%)	47 (20.52%)	$\chi^2(1) = 82.60$	< 0.001*
Mental health treatment	125 (58.14%)	88 (38.43%)	$\chi^2(1) = 16.48$	< 0.001*
Other diagnoses with chronic pain	64 (29.63%)	45 (19.65%)	$\chi^2(1) = 5.98$	0.014*

Absolute frequencies and percentages are presented for categorical variables and means and standard deviations (SD) for quantitative variables. Differences between groups were assessed using the Chi-square test for categorical variables and *t* tests for quantitative variables. $p < .05^*$

Table 2 Self-reported endometriosis-related and psychological variables among women with UD endometriosis and controls

	UD Endometriosis (N = 216)	Non-endometriosis controls (N = 229)	Statistic	p-value
Dysmenorrhea, NRS score	7.90 (2.31)	5.05 (2.86)	$t(432.34) = 11.57$	< 0.001*
Dysmenorrhea (NRS ≥ 7)	187 (86.57%)	88 (38.43%)	$\chi^2(1) = 11.23$	< 0.001*
Anhedonia - SHAPS (1–4)	25.56 (7.83)	22.05 (6.59)	$t(420.88) = 5.10$	< 0.001*
Anhedonia - SHAPS (0–1)	2.28 (3.20)	1.18 (2.14)	$t(372.44) = 4.21$	< 0.001*
Clinical Anhedonia, SHAPS (0–1) ≥ 3	67 (31.02%)	39 (17.03%)	$\chi^2(1) = 11.98$	< 0.001*
TEPS anticipatory	3.80 (0.99)	4.14 (0.86)	$t(424.17) = -3.84$	< 0.001*
TEPS consummatory	4.50 (0.96)	4.71 (0.95)	$t(438.5) = -2.29$	0.023*
Anxiety (HADS)	10.71 (4.18)	8.59 (4.24)	$t(442.13) = 5.30$	< 0.001*
Anhedonic Depression (HADS)	7.46 (4.23)	4.52 (3.52)	$t(418.75) = 7.93$	< 0.001*
Non-anhedonic Depression (HADS)	2.7 (1.5)	1.7 (1.3)	$t(415.01) = 7.02$	< 0.001*

Absolute frequencies and percentages are presented for categorical variables, and means and standard deviations (SD) for quantitative variables. Differences between groups were assessed using the Chi-square test for categorical variables and the *t* test for continuous variables. $p < .05^*$

Results

Sample description

A total of 216 women with UD endometriosis and 229 control participants were included in the study (see Table 1). In the control group, most participants were seeking contraception (64.2%; $n = 147$), while 35.8% ($n = 82$) consulted for common benign gynecological conditions that were asymptomatic, such as unilateral simple ovarian cysts (7%), dermoid ovarian cysts (3.5%), uterine fibroids (19.2%), or polycystic ovarian syndrome (6.1%). In all cases, adenomyosis and endometriosis lesions were excluded through transvaginal examination and transvaginal ultrasound.

The mean age in the UD endometriosis group was 37 years, compared to 32.5 years in the control group. The UD endometriosis group was significantly older than the control group ($t(416.21) = 5.98$, $p < .001$). The two groups did not differ significantly in terms of educational attainment. The majority of participants reported being in a relationship, with a higher percentage of participants partnered in the UD endometriosis group ($\chi^2(1, N = 445) = 4.49$, $p < .05$). Overall, 71% of participants were childless. A history of gynecological surgery was reported

by more than half of the women with UD endometriosis, compared to only 19% of controls ($\chi^2(1, N = 445) = 71.26$, $p < .001$). Hormonal treatment was used by 63% of women with UD endometriosis and 20% of controls, also showing a statistically significant difference ($\chi^2(1, N = 445) = 82.60$, $p < .001$). The prevalence of other diagnoses associated with chronic pain was higher in the UD endometriosis group ($\chi^2(1, N = 445) = 5.98$, $p = .014$). At the time of assessment, 58.1% of women with UD endometriosis were receiving some form of mental health treatment, compared to a lower percentage (38.4%) in the control group ($\chi^2(1, N = 445) = 16.48$, $p < .001$).

Regarding the endometriosis-related clinical and psychological profile of the participants (see Table 2), patients with UD endometriosis reported significantly higher levels of menstrual pain, with a mean score of 8 ($SD = 2.30$), and nearly 87% exceeded the clinical severity threshold [47]. In contrast, the control group reported a mean pain level of 5 ($SD = 2.80$), with pain scores being significantly higher in the UD endometriosis group ($t(432.34) = 11.57$, $p < .001$). In terms of psychological distress, the UD endometriosis group had a mean depression score of 7.46 ($SD = 4.23$) and a mean anxiety score

of 10.71 ($SD = 4.18$), compared to 4.52 ($SD = 3.52$) and 8.60 ($SD = 4.24$), respectively, in the control group. Both depression and anxiety scores were significantly higher in the UD endometriosis group ($t(418.75) = 7.93$, $p < .001$ for depression; $t(442.13) = 5.30$, $p < .001$ for anxiety). Neither group reached the average clinical cut-off score for depressive disorder [48].

Presence of anhedonia

Mean anhedonia scores in the UD endometriosis group were 25.56 on the SHAPS (1–4) scale and 2.30 on the alternative 0–1 scoring system, with 31% of patients reaching clinical levels of anhedonia. In contrast, the control group showed lower scores: 22.00 ($SD = 6.60$) on the 1–4 scale and 1.20 ($SD = 2.10$) on the 0–1 scale. These differences were statistically significant for both scoring methods ($t(420.88) = 5.10$, $p < .001$ for the 1–4 scale; $t(372.44) = 4.21$, $p < .001$ for the 0–1 scale; see Fig. 1). The percentage of individuals reaching clinical levels of anhedonia was also higher in the UD endometriosis group ($\chi^2(1, N = 445) = 11.88$, $p < .001$). Effect sizes, as measured by Cohen's δ , were 0.47 and 0.40, indicating medium effects for both scoring formats. The SHAPS (1–4) version demonstrated excellent internal consistency, with a Cronbach's alpha of 0.94.

UD endometriosis participants also reported significantly lower scores on both the anticipatory and consummatory components of the TEPS ($t(424.17) = -3.84$, $p < .001$, and $t(438.5) = -2.29$, $p < .001$, respectively; see Table 2). TEPS scores were averaged to facilitate comparison, as the two subscales consist of different numbers of items (see Materials and Methods). Internal consistency was acceptable, with a Cronbach's alpha of 0.75 for

the anticipatory subscale and 0.80 for the consummatory subscale.

Moderated multiple regression model of anhedonia scores

We first conducted a linear regression analysis to assess the influence of potential sociodemographic and clinical covariates on the outcome variable (SHAPS). The demographic and somatic clinical variables that were statistically different between groups (age, presence of other chronic pain diagnoses, and hormonal treatment) were included as covariates, along with the primary predictors of interest: GROUP (0 = control, 1 = patient), TEPS anticipatory, TEPS consummatory, and their interaction terms, as specified in the a priori model. None of the covariates were statistically significant predictors of SHAPS scores and were therefore excluded from subsequent analyses.

Afterwards we tested our a priori regression model with the SHAPS (0–1) as the continuous dependent variable. Our predictors were GROUP (UD endometriosis – controls), both anticipatory and consummatory TEPS scores, as well as their interactions with GROUP. The regression equation was defined as: $SHAPS = GROUP + TEPS \text{ anticipatory} + TEPS \text{ consummatory} + (GROUP \times TEPS \text{ anticipatory}) + (GROUP \times TEPS \text{ consummatory})$. This model significantly predicted anhedonia scores, $F(5, 438) = 67.63$, $p < .001$, explaining approximately 43.6% of the variance (Adjusted $R^2 = 0.429$). Two cases were excluded from the analysis due to missing data. Residual diagnostics confirmed that model assumptions were adequately met. Variance inflation factors (VIFs) for the continuous predictors indicated no concerns regarding multicollinearity (TEPS anticipatory = 3.06; TEPS consummatory = 2.61).

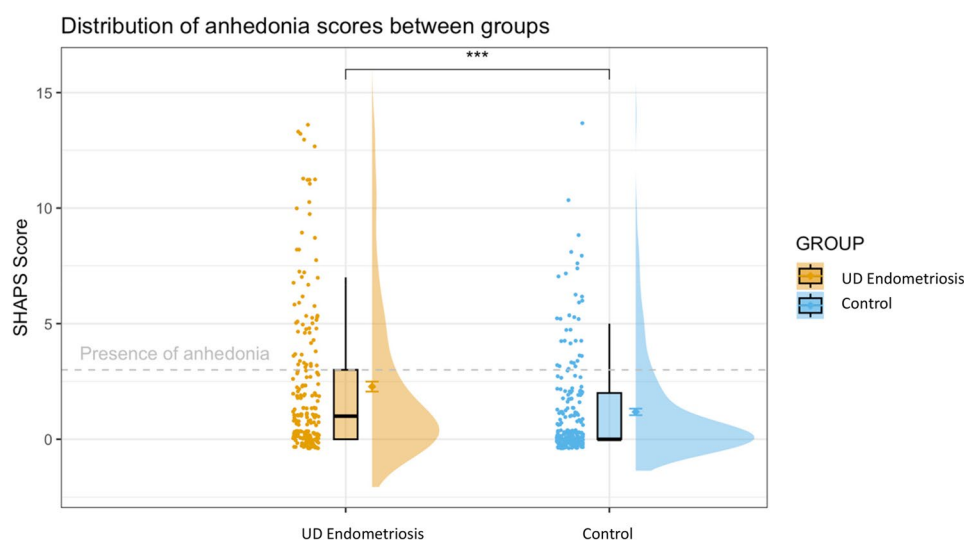


Fig. 1 Raincloud plot of anhedonia scores in women with UD endometriosis and control participants. The UD endometriosis group is shown in yellow and the control group in blue. The plot displays raw data points, boxplots, and density plots for the SHAPS anhedonia scores (short scoring format). Mean and standard deviation for each group are also depicted on the density plots. Differences between groups are statistically significant at $p < .001$

Table 3 Results of two linear regression models with SHAPS 0–1 (short scoring format) as the dependent variable

	Estimate (β)	SE	95% CI	p
<i>Predictors 'a priori' model</i>				
Intercept	7.72	0.78	[6.19–9.25]	< 0.001*
Group	4.71	1.04	[2.67–6.76]	< 0.01*
TEPS anticipatory	−0.52	0.18	[−0.87 – −0.16]	< 0.001*
TEPS consummatory	−0.93	0.16	[−1.25 – −0.61]	< 0.001*
T. Anticipatory x Group	−0.67	0.25	[−1.17 – −0.18]	< 0.001*
Consummatory p. x Group	−0.33	0.24	[−0.81 – −0.15]	0.18
<i>Predictors final 'a priori' model</i>				
Intercept	8.08	0.73	[6.65–9.52]	< 0.001*
Group	3.93	0.86	[2.23–5.63]	< 0.001*
TEPS anticipatory	−0.44	0.17	[−0.77 – −0.10]	< 0.01*
TEPS consummatory	−1.08	0.12	[−1.32 – −0.84]	< 0.001*
T. Anticipatory x Group	−0.86	0.21	[−1.27 – −0.44]	< 0.001*
<i>Predictors Extended model</i>				
Intercept	4.01	0.96	[2.08–5.94]	< 0.001*
Group	4.82	1.38	[2.16–7.47]	< 0.01*
TEPS anticipatory	−0.30	0.17	[−0.64–0.05]	0.09
TEPS consummatory	−0.73	0.16	[−1.05 – −0.42]	< 0.001*
Anxiety	0.17	0.03	[0.09–0.24]	< 0.001*
Non-anhedonic depression	0.18	0.12	[−0.07–0.43]	0.15
Dysmenorrhea	−0.08	0.05	[−0.18–0.01]	0.09
T. anticipatory x Group	−0.63	0.025	[−1.11 – −0.15]	< 0.01*
T. consummatory x Group	−0.40	0.023	[−0.86–0.07]	0.09
Anxiety x Group	−0.08	0.05	[−0.19–0.02]	0.12
Non-anhedonic Depression x G.	0.20	0.16	[−0.11–0.52]	0.20
Dysmenorrhea x Group	0.13	0.08	[−0.02–0.28]	0.08

The *a priori* model, the final *a priori* model obtained through backward elimination, and the extended model are presented. The table reports unstandardized regression coefficients (β), standard errors (SE), 95% confidence intervals (CI), and p-values for each regressor and the intercept. Statistically significant results are indicated by $p < 0.05^*$

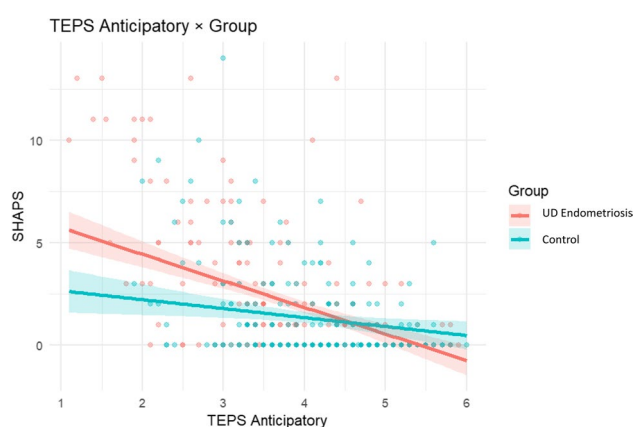


Fig. 2 Interaction effect between anticipatory pleasure and group (UD endometriosis vs. control) on predicted SHAPS scores. The control group is shown in turquoise, and the UD endometriosis group in red. Shaded areas represent the 95% confidence intervals (CI) of the predicted anhedonia scores as a function of TEPS anticipatory measures.

All variables in the model were statistically significant predictors of Anhedonia scores as well as the interaction between TEPS anticipatory and GROUP (see Table 3a priori model). The interaction between TEPS consummatory and GROUP was non-significant and was therefore

excluded from the follow-up model in order to enhance interpretability. The final *a priori* model significantly predicted anhedonia scores, $F(4, 439) = 83.93$, $p < .001$, explaining approximately 43.3% of the variance (Adjusted $R^2 = 0.428$) (see Table 3final 'a priori' model). Residual diagnostics confirmed that model assumptions were adequately met. Variance inflation factors (VIFs) for the continuous predictors indicated no concerns regarding multicollinearity (TEPS anticipatory = 2.73; TEPS consummatory = 1.44). The final model showed that, holding anticipatory and consummatory pleasure constant, having a UD endometriosis diagnosis significantly predicted SHAPS scores, with an increase of 3.93 points ($\beta = 3.93$, $p < .001$). A significant interaction between TEPS anticipatory and GROUP was found ($\beta = -0.86$, $p < .001$), indicating that the relationship between anticipatory pleasure and anhedonia differed by diagnostic group (see Fig. 2). In the control group, both pleasure dimensions were significant predictors of anhedonia: lower TEPS consummatory scores ($\beta = -1.08$, $p < .001$) and lower TEPS anticipatory scores ($\beta = -0.44$, $p = .01$) were associated with higher SHAPS scores. The UD endometriosis group shared the same slope for TEPS consummatory, but

TEPS anticipatory had a much greater impact on SHAPS scores, with a derived slope of $\beta = -1.30$ (i.e., $\beta = -0.44 + -0.86$) (see Fig. 2).

Finally, we conducted an extended regression model including anxiety, depression, and dysmenorrhea to examine their contribution to anhedonia scores, together with anticipatory and consummatory pleasure. Given the significant interactions identified in previous models and our objective of understanding the moderating role of UD endometriosis diagnosis, we also tested the interaction effects of these new predictors with GROUP. Anxiety was measured by the HADS anxiety subscale, depression by the non-anhedonic component of the HADS, and dysmenorrhea by the NRS scale.

Prior to estimating the model, we examined multicollinearity. VIFs for a model including only main effects revealed strong collinearity between GROUP and dysmenorrhea (GROUP VIF=8.7; Dysmenorrhea VIF=11.5). After centering the dysmenorrhea variable, all VIFs fell below 1.7, indicating no problematic collinearity.

The extended model significantly predicted anhedonia scores, $F(11, 427)=40.25$, $p < .0001$, explaining approximately 50% of the variance (Adjusted $R^2 = 0.49$) (see Table 3, *extended model*). Significant main effects were observed for UD endometriosis diagnosis ($\beta=4.82$, $p < .001$), lower consummatory pleasure ($\beta = -0.73$, $p < .001$), and higher anxiety ($\beta=0.17$, $p < .001$), all predicting increased anhedonia scores at statistically significant levels. Although anticipatory pleasure lost significance as a main effect compared to the previous model, its interaction with UD endometriosis diagnosis remained significant, suggesting a distinctive anticipatory cognitive pattern associated with diagnostic status. Non-anhedonic depression was not predictive of anhedonia once anxiety and dysmenorrhea were included. Finally, a marginally significant positive interaction between dysmenorrhea and UD endometriosis diagnosis was identified ($\beta=0.13$, $p=.08$), indicating that greater menstrual pain in women with UD endometriosis was associated with higher anhedonia scores.

Discussion

Anhedonia, a behavioral trait characterized by a diminished response to reward, has previously been reported as prevalent among patients with Deep Endometriosis [7]. Building on these earlier findings, the present study demonstrates that anhedonia levels are significantly higher also in women with UD endometriosis compared to controls without the condition (see Fig. 1), with 31% of patients exhibiting clinically relevant hedonic deficits. This significant difference was further supported by our results, which identified the presence of UD endometriosis as the strongest and most significant predictor

of elevated anhedonia scores across all regression models (see Table 3). Interestingly, the present study identified, for the first time to our knowledge, that in the UD endometriosis population studied in our sample, anhedonia is predicted by a distinctive and specific pattern of reward-related deficiencies, characterized by greater difficulties in anticipating pleasurable life experiences. Regarding further distress measures, anxiety emerged as a significant main predictor, whereas depression showed no impact on anhedonia scores. Finally, a marginal interaction effect involving dysmenorrhea was found, suggesting that in UD endometriosis, increased menstrual pain may contribute to higher levels of anhedonia.

The present findings are consistent with previous research on the prevalence of anhedonia in DE. In the current study, patients with UD endometriosis exhibited a mean anhedonia score of 25.5 and 2.3 on the long and short formats of the SHAPS respectively, which are comparable to the means previously reported in DE by Mallorquí and colleagues (24.2 and 2, respectively) [7]. It is important to note that the present sample likely provides a more naturalistic observation of anhedonia in UD endometriosis, compared to the earlier study based on a sample of individuals with Deep Endometriosis who were undergoing multimodal treatment. As such, the current findings may offer greater external validity and a more representative picture of anhedonia in the broader UD endometriosis population.

In recent years, studies on endometriosis have consistently highlighted the impact of the disease on mental health and quality of life. The present study corroborates these findings, as not only anhedonia but also depression and anxiety show consistently higher levels among patients with UD endometriosis. Furthermore, the fact that almost 60% of patients are engaged in some form of mental health treatment reflects the value of addressing UD endometriosis through a multidisciplinary approach. Interestingly, a recent systematic review on the use of multidisciplinary team care for endometriosis indicated that psychological approaches were the most frequently suggested additions to improve multidisciplinary care outcomes [49]. Multidisciplinary interventions have demonstrated improvements in aspects such as quality of life, chronic pelvic pain, and psychological comorbidities, with patients also reporting higher levels of satisfaction with their treatment. Moreover, growing evidence is progressively clarifying the psychological profile of endometriosis, identifying key therapeutic targets such as catastrophizing, rumination, low-control illness perceptions, anhedonia, and mood and anxiety alterations [7, 11, 50, 51].

The results of this study advance our understanding of anhedonia as a transdiagnostic behavioral correlate of mental health and somatic systemic diseases such as UD

endometriosis. Our results show that anhedonia is specifically increased by anxiety, whereas depression shows no impact on hedonic capacity, highlighting the distinctiveness of these constructs. This reveals a more nuanced profile of psychological distress in UD endometriosis, suggesting that anxiety, depression, and anhedonia may be interrelated but follow distinct pathways that require further investigation to elucidate comorbidities. In addition, we also found a marginal effect of menstrual pain on anhedonia, indicating that, along with anxiety and other pleasure-related impairments, it may contribute separately to the psychological burden of UD endometriosis through mechanisms not fully explained by depression.

Interestingly, our results demonstrated, for the first time to our knowledge, that patients with UD endometriosis differ from controls without UD endometriosis in the specific aspects of reward processing that are impaired. In particular, while both consummatory and anticipatory components contribute significantly to anhedonia in UD endometriosis, a reduced capacity for anticipation may highlight how cognitive processes deteriorate in the context of chronic inflammation and pain. Alternatively, as suggested by Rizvi et al. (2021), anticipatory reward deficits could reflect a pre-existing cognitive vulnerability that precedes the onset of the disease and contributes to the risk of developing chronic pain and depression. However, given the cross-sectional nature of the present paper, this possibility could not be explored and remains a subject for future research.

In this sense, diminished anticipatory pleasure in UD endometriosis could also be explained as a narrowing of the range of experiences anticipated and perceived as pleasurable. Interestingly, this effect has been described in both chronic pain and affective disorders, where individuals show reduced sensitivity to natural rewards and an attentional focus on pain relief or threat avoidance [52–54]. This perspective could help in understanding the behavior of UD endometriosis patients with elevated levels of psychological distress and anhedonia. In these cases, motivation may become focused almost exclusively on finding a way to eradicate suffering and mostly pain. Clinicians working with UD endometriosis and chronic pelvic pain patients may recognize this pattern, where individuals invest considerable time and effort into seeking pain relief, which appears to become the dominant valued reward. Such an extreme narrowing of the reward experience could lead to a loss of motivation for stimuli that were previously perceived as rewarding representing a plausible hypothesis that requires further investigation.

The findings presented highlight the importance of addressing reward-related deficits, particularly, anticipatory pleasure, as part of a therapeutic strategy. In recent years, specific psychological therapies aimed at increasing positive affect and enhancing reward responsiveness

have been developed, showing promising results for psychological outcomes relevant to endometriosis [55–57]. These approaches may hold potential for individuals with endometriosis, and future research could valuably explore their feasibility and effectiveness in this population.

From a methodological perspective, one strength of our study lies in the recruitment of a large number of participants with and without UD endometriosis, all of whom underwent clinical evaluation and specialized sonography to confirm or rule out the diagnosis.

An important conceptual gap concerns sexual pleasure, which remains strikingly understudied in endometriosis despite its clinical relevance. Mallorquí et al. (2025) [7] showed that its loss is detrimental for DE patients, assessing it with a BDI-II item since the SHAPS does not capture this dimension. Sexual pleasure is also overlooked in standard instruments such as the Female Sexual Function Index (FSFI) [58], and recent literature has emphasized the need to address this relevant gap [59]. Our findings on anhedonia further support expanding research on sexual pleasure in endometriosis.

Several methodological limitations should also be acknowledged in the present study. First, chronic pelvic pain and other relevant endometriosis symptoms, known to influence anhedonia levels, were not assessed. Only menstrual pain intensity was recorded, and its clinical significance as a proxy for overall endometriosis-related pain is limited. Future studies should incorporate more detailed clinical variables to allow for more precise and nuanced conclusions. Additionally, recruiting both groups from a gynecological service among individuals seeking medical attention, and framing the study around endometriosis, may have introduced a self-selection bias, potentially leading to an overestimation of distress levels in both groups. Furthermore, the fact that the non-endometriosis control group comprised participants with common gynecological conditions could have influenced our results by potentially increasing distress levels in the controls. Future studies including participants without gynecological conditions may reveal larger between-group differences and improve the generalizability of the findings.

Finally, it is important to mention that the HADS is largely composed of anhedonia-related items while covering fewer cognitive and behavioral aspects of depression such as suicidal thoughts, sadness, guilt, or helplessness. Although it allows for the assessment of depressive symptoms independently from somatic complaints such as fatigue, sleep disturbances, poor appetite, or anergia, other depression scales should be considered in future studies addressing anhedonia and depression in UD endometriosis. For instance, the Beck Depression Inventory - II (BDI-II) [60], widely used in

endometriosis research, provides a more comprehensive evaluation including psychological, affective, and cognitive symptoms, while the Center for Epidemiologic Studies Depression Scale (CES-D) [61] also captures the social dimension of depression, which may be particularly relevant for characterizing depression. Both would allow researchers to isolate different components of depression and move beyond the limits of our cross-sectional findings by clarifying the relative independence of anhedonia from depression in UD endometriosis.

In summary, our findings confirm that patients with UD endometriosis exhibit consistently higher levels of anhedonia compared to controls regardless of depression, corroborating recent findings in deep endometriosis populations. Notably, patients in the present study display a specific deficit in anticipatory pleasure, which underscores the significance of cognitive processes that may be compromised in UD endometriosis. Anticipatory cognitive processes are crucial for engaging in health-promoting and goal-directed behaviors, that are central to affective regulation. Targeting cognitive and affective processes is essential for restoring the health and quality of life of endometriosis patients and is particularly important for preventing the reinforcement of maladaptive psychological patterns that contribute to chronic distress and may hinder patients' ability to experience pain relief through functional reward-related mechanisms.

Abbreviations

UD Endometriosis	Ultrasound Diagnosed Endometriosis
SHAPS	Snaith Hamilton Pleasure Scale
TEPS	Temporal Experience of Pleasure Scale
DE	Deep Endometriosis
fMRI	Functional Magnetic Resonance Imaging
HADS	Hospital Anxiety and Depression Scales
NRS	Numeric rating scale
SD	Standard deviation
VIF	Variance Inflation Factor
SE	Standard error
CI	Confidence Interval

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Authors' contributions

MAMZ, FC, and AM designed the study protocol. MAMZ and FC supervised the study. CM and MAMZ collected the data. ES and AM conducted all statistical analyses and prepared the figures and tables. AM drafted the manuscript. LQM and MG reviewed the first draft and contributed to refining the gynecological scientific terminology and perspectives. BC, MAMZ, and FC supervised the final versions of the manuscript and provided expert advice on the content.

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Data availability

The data supporting this study are not publicly available to preserve individuals' privacy under the European General Data Protection Regulation (GDPR), but may be obtained from the corresponding author upon reasonable request and appropriate permissions.

Declarations

Ethics approval and consent to participate

The study received ethical approval from the Research Ethics Committee on Medicinal Products of the Hospital Clínic of Barcelona prior to participant recruitment (HCB/2021/0674). All procedures involving human participants were conducted in accordance with the ethical standards of the Ethics Committee for Drug Research of Hospital Clínic and with the 1964 Helsinki Declaration and its later amendments. All participants provided informed consent before participating in the study.

All authors consented to participate in this study and agreed to the preparation and submission of this manuscript.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

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