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The associations between PMS, depressive symptoms, and skin barrier integrity

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Abstract

This master thesis investigates the associations between the premenstrual syndrome (PMS), depressive symptoms, and transepidermal water loss (TEWL) as an indicator of skin barrier integrity. Previous research indicates a possible connection between these variables, with PMS often associated with depression, suggesting shared etiological factors and depression acting as a transient symptom of PMS. Additionally, both PMS and depression have been associated with various skin disorders and inflammatory processes, potentially impacting skin barrier integrity. For this thesis, the data of 116 women who participated in a study within a larger experimental project focusing on stress, music listening, and wound healing, were analyzed. Depressive symptoms and PMS were assessed via online questionnaires, while skin barrier integrity was evaluated through transepidermal water loss measurements.
Contrary to initial hypotheses, the results could not show significant associations between depressive symptoms or PMS and TEWL. However, a significant moderate positive correlation was observed between PMS and depressive symptoms, consistent with existing literature. From these findings, it can be inferred that while there is a significant correlation between premenstrual syndrome and depressive symptoms, their relationship with skin barrier integrity, as measured by transepidermal water loss, remains unclear.

Keywords: *Premenstrual syndrome, depression, skin barrier integrity, transepidermal water loss, psychodermatology*

Contents

Introduction

The premenstrual syndrome

Introducing the premenstrual syndrome

Even long before the medical terminology of the premenstrual syndrome (PMS) emerged, the distressing symptoms experienced by women during their menstrual cycle were recognized. This dates back to the era of the ancient Egypt in 1900 BC, where depressive mood and hysterical disorders in women were suspected to be caused by spontaneous movements of the uterus within the female body. The term "hysterical" originates from the Greek word "hystera," meaning uterus, reflecting historical beliefs associating such disorders with the uterus (Ehrenreich & English, 2010). In the Greek mythology, Plato posited that the uterus becomes sorrowful when it fails to unite with the male and cannot facilitate the creation of new life. He therefore concludes that the absence of a conventual sex life leads to female madness, characterized by anxiety and melancholia (Tasca et al., 2012). The movement of the womb within the body was believed to potentially induce health problems by disrupting other organs (Dinsdale and Crespi, 2017). According to Hippocrates, the cure to the female hysteria lies in entering marriage and experiencing a fulfilling sexual life (Tasca et al., 2012). According to the myth of Euripides, a communal method of treating and preventing uterine melancholy is embodied in the Dionysian rituals, where consuming wine and even attending orgies were supposed to help healing the female madness.

In the Roman Empire, the physician Galen, one of the most famous figures in medical history, rejected the concept of a physically wandering womb. He clarified that although it might appear to move, the sensation was attributed to the tension of the membranes securing it in position, causing a slight elevation. Galen argued that the core issue lay in the "suffocation" of the womb due to the accumulation of menstrual blood or, more concerning, the female counterpart of "seed" merging with male sperm. The retention of this seed, he asserted, could lead to putrefaction and the release of vapors, which, in turn, could corrupt other organs (Simon, 2014). In Galen's work *Hippocratis libricum de humoribus*, he states: "I have examined many hysterical women, some stuporous, others with anxiety attacks [...]: the disease manifests itself with different symptoms, but always refers to the uterus." (Kofner, 2018).

Galen's therapeutic interventions for hysteria included purges, the administration of substances such as hellebore, mint, laudanum, belladonna extract, valerian, and various herbs,

but also engaging into marriage or avoiding stimuli that could bring excitement to a young woman (Tasca et al., 2012). During the Middle Ages, Hippocratic and Galenic teachings endured, while a notable advocate for women's health emerged: Trota de Ruggiero, one of the earliest gynecologists in the 11th century. In her two key works, she discussed the “female hysteria”, attributing it to the retention of blood or corrupt uterine humors (Chapa, 2016). Later, in the 16th century, Giovanni da Monte, a medical professor at University Ferrara, highlighted a potential link between menstruation and certain facets of depression (Bonuzzi, 1976), whilst in the 19th century, James Prichard, an English doctor, discussed "dysmenorrheal affections," noting that some females experience heightened nervous excitement during the menstrual period (Richardson, 1995). Prichard stated that at such times, individuals may exhibit morbid mental dispositions, including a capricious temper, heightened emotional excitability, bitterness, a tendency to quarrel with close relatives, and occasionally a melancholic state of mind approaching depression.

However, it was not until many centuries later that the first explicit discussions regarding discomforting symptoms occurring specifically in the days before menstruation arose. Karen Horney, a German psychoanalyst, stated that disruptions appear not only during menstruation but primarily in the premenstrual period. She describes those disruptions as: “Feelings of all degrees of unease, ranging from a general sense of heaviness and a feeling of overall displeasure or inhibition, a fluctuating self-esteem, melancholy, and a profound depressive mood. All this often goes along with irritability or anxiety.” (Horney, 1931). In the same year, Robert Frank, an American gynecologist, published a paper on the disturbances during the pre-menstrual phase that include heightened fatigue, diminished concentration, episodes of pain, severe headaches and noticeable swelling, particularly in the face, hands, and feet. Although all symptoms rapidly disappeared with the onset of menstrual flow, they recurred at different times as the end of the menstrual cycle approached (Frank, 1931). Dr. Frank is credited as the first to introduce the term for premenstrual symptoms, referring to them as “pre-menstrual tension”. Twenty years later, in 1953, Greene and Dalton describe the term “pre-menstrual tension” as inadequate and state it does not capture the condition’s complexity, as tension is just one of its many components (Greene and Dalton, 1953). The use of the term could lead to overlooking the disorder when tension is either not present or overshadowed by a more severe disturbance. Therefore, the authors introduced the term "premenstrual syndrome" (PMS), which remains the mainly used term to this day.

Defining and diagnosing PMS

In 1987, the American Psychiatric Association introduced a more severe form of PMS: The late luteal phase dysphoric disorder (LLPDD). This term was later renamed to the premenstrual dysphoric disorder (PMDD) and included as a psychiatric condition in the DSM5. However, the term premenstrual syndrome (PMS) is not formally acknowledged as an official medical diagnosis yet has become a part of everyday language (Schroll and Lauritzen, 2022). Since there are no agreed-upon criteria or a gold standard for validation, a consensus on the operationalization of clinically significant PMS is still lacking. Despite these challenges, it is generally accepted that PMS criteria should encompass minimum symptom severity and a progression of symptoms from the ovulatory to the luteal phase of the menstrual cycle (Dean et al., 2006). Both the American College of Obstetricians and Gynecologists Practice Guidelines and the International Classification of Diseases, 10th revision, outline criteria for PMS diagnosis, requiring a minimum of one physical or psychological premenstrual symptom that include: sudden appetite changes, weight gain, back pain, and skin problems to emotional upheavals such as headache, depression, anxiety, anger, fatigue, mood swings, and crying.

The PMDD diagnosis necessitates the presence of five out of eleven specified symptoms, with at least one being related to mood. These symptoms must be severe before menstruation and should alleviate after the onset of menses. Additionally, there must be significant functional impairment (Freeman et al., 2011).

There is ongoing discussion regarding the differentiation between premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD). While PMDD is recognized as a mental disorder in the DSM-5, PMS lacks this designation in diagnostic manuals. Consequently, the operationalization of PMS varies, with the diagnosis of PMDD requiring the maintenance of a symptom diary, a practice also suggested for PMS (Janda et al., 2016). To address this issue of diagnosing the severity of the symptoms, the International Society for Premenstrual Disorders suggests categorizing both premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD) as a continuum within the framework of premenstrual disorders (Yoshimi et al., 2022).

Primary care clinicians, who often handle patients with PMS, confront the challenge of evaluating a diverse range of nonspecific symptoms linked to PMS (Freeman et al., 2011),

since determining whether to address these cases from a gynecological or psychiatric perspective is often uncertain.

Schroll and Lauritsen (2022) mention the introduction of premenstrual dysphoric disorder (PMDD) into the DSM-5 in 2013 was a quite controversial step. While the inclusion was fervently supported by patients, psychiatrists, and the pharmaceutical industry, it faced criticism from psychologists and generalists who expressed concerns about potential overdiagnosis and pathologizing of normal hormonal changes.

Epidemiology

The prevalence estimates for the premenstrual syndrome vary widely due to factors like definition, study population, and research method. These differences affect the accuracy: a strict definition may yield high specificity but low sensitivity, underestimating prevalence, while a lenient one may show high sensitivity but low specificity, potentially overestimating prevalence (Dean et al., 2006).

However, an often-cited prevalence estimate states as follows: While affecting 85-90% of women of reproductive age, the severity of symptoms can range from mild to clinically significant, with 15-20% of women meeting the criteria for the premenstrual Syndrome. In severe cases, approximately 3-8% of women with heightened emotional symptoms may be diagnosed with premenstrual dysphoric disorder (PMDD), which is a psychiatric condition outlined in the DSM5 (Bertone-Johnson, 2016).

Etiology

The etiology of PMS remains a complex riddle, but past research suggests theories exploring hormonal fluctuations, particularly involving estrogen and progesterone, during the menstrual cycle. In the initial paper on premenstrual tension by Frank (1931), he hypothesized that the condition resulted from an excessive buildup of "the female sex hormone" (by which he meant estrogen) due to renal dysfunction. Frank proposed treatment options, suggesting the use of medication to enhance estrogen excretion or radiological procedures targeting the ovaries to reduce its production. In the 1950s, Dalton and Greene suggested the cause of PMS lies in a progesterone deficiency. The proposed to address the condition with injections or implantations of progesterone, or the oral ingestion of synthetic progestogens (Richardson, 1995). Thirty years later, in 1983, Dr. Guy Abraham, a research gynecologist and

endocrinologist, categorized the premenstrual syndrome (PMS) into four subgroups: PMS-A (anxiety), linked to high estrogen and low progesterone; PMS-C (carbohydrate craving), unclear etiology with increased appetite and sugar cravings; PMS-D (depression), associated with low estrogen and neurotransmitter breakdown and PMS-H (hyperhydration), involving water retention from elevated aldosterone (Abraham, 1983). Yet, Dr. Guy Abraham countered that Frank's explanation was insufficient. According to him, renal dysfunction would lead to an increased concentration of estrogen, which, in turn, would be biologically inactivated by the liver. (Chocano-Bedoya and Bertone-Johnson, 2013).

Nevertheless, none of these theories could be conclusively proven through empirical evidence. In recent years, various suggestions have emerged concerning nervous system sensitivity, genetic factors, and psychological elements such as stress (Goswami et al., 2023). PMDD is believed to be associated with hormonal changes, particularly those involving progesterone and its metabolite allopregnanolone. Genetic factors, including variations in the ESR1 gene, may play a role in contributing to PMDD. Studies using functional magnetic resonance imaging (fMRI) suggest the participation of neurotransmitters such as serotonin and GABA. The etiology of PMDD involves factors like stress exposure, genetic predisposition, and altered neuro steroid levels.

Yonkers, O'Brien & Eriksson (2008) propose that gonadal steroid hormones are implicated in PMS. They contend that while the drop in progesterone is often associated with symptoms, it fails to fully explain the variations observed in PMS, given the absence of symptoms during non-ovulatory cycles. Yonkers et al. suggest considering the preovulatory estradiol peak or postovulatory progesterone increase as potential triggers. There's a possibility that PMS patients exhibit increased sensitivity to normal hormonal fluctuations. Additionally, factors like body-mass index, stress, traumatic events, and thyroid indices may also contribute significantly.

Treatment

As scientific knowledge about menstrual symptoms has improved, so has the approach to treatment. While herbal remedies, nutrition, and relaxation continue to be viable treatment options today, there have been significant changes in the overall understanding and management of menstrual symptoms. The initial step in managing PMS still involves non-pharmacological approaches, including physical activity, cognitive behavioral therapy, herbal preparations, and social support. Additionally, treatment options include regular exercise,

healthy sleep patterns and stress management (Gudipally and Sharma, 2023). However, pharmacological therapies, particularly selective serotonin reuptake inhibitors (SSRIs), are often employed when non-pharmacological solutions prove ineffective.

Yonkers et al.'s research indicates that several clinical trials assessing selective serotonin inhibitors (SRIs) for managing PMS/PMDD have demonstrated significant benefits. The response rates typically range from 60–90% for active treatment compared to 30–40% for a placebo. Effective SRIs include the serotonergic tricyclic antidepressant clomipramine, selective SRIs like citalopram, escitalopram, fluoxetine, paroxetine, and sertraline, as well as the serotonin and noradrenaline reuptake inhibitor venlafaxine. SRIs not only reduce mood symptoms and somatic complaints but also enhance quality of life and social functioning. Many experts suggest considering SRIs as the first-line treatment for PMS patients with severe mood symptoms.

Furthermore, elevating the intake of complex carbohydrate supports the production of serotonin and the extract of Vitex agnus-castus is known to assist in alleviating mood swings. (Sureja et al., 2023). While there was a hypothesis that progesterone deficiency might contribute to PMS, attempts to supplement progesterone have not shown significant effects in past trials. Nevertheless, it is still utilized as a treatment in the UK (Yonkers, O'Brien & Eriksson, 2008).

Depression

Introducing Depression

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), a Major Depressive Episode diagnosis requires the presence of five or more symptoms over a consecutive 2-week period. Key symptoms include depressed mood or anhedonia (loss of interest or pleasure). Additional symptoms encompass changes in appetite or weight, disturbances in sleep patterns (insomnia or hypersomnia), psychomotor agitation or retardation, fatigue or loss of energy, diminished ability to think or concentrate, feelings of worthlessness or excessive guilt, and thoughts of suicidality (Tolentino and Schmidt, 2018). Regarding diagnoses, two general forms of depression are distinguished by the absence or presence of manic or hypomanic episodes: unipolar depression and bipolar disorder (Hammen & Watkins, 2018). Common forms of unipolar depression are the major depressive episode, persistent depressive disorder, premenstrual dysphoric disorder, and depression not otherwise specified (American Psychiatric Association, 2013). The criteria of the bipolar I disorder

involve at least one lifetime manic episode, while bipolar II disorder includes hypomania with depression (American Psychiatric Association, 2013). The form of depression in bipolar disorder shares similarities with unipolar depression but may have more frequent and abrupt episodes (Cuellar, Johnson, & Winters, 2005). However, diagnosing depression in youth requires careful consideration of family history and longitudinal assessment, as some initially diagnosed with unipolar depression may later develop bipolar disorder (Beesdo et al., 2009).

Epidemiology

According to the World Health Organization (WHO), depression affects approximately 3.8% of the global population, with varying prevalence across different demographic groups. Among adults, 5% experience depression, and there is a slightly higher incidence among women (6%) compared to men (4%). The prevalence increases to 5.7% among adults aged 60 and older, affecting around 280 million individuals worldwide. Notably, depression is about 50% more common in women than in men. Pregnant women and those in the postpartum period face a significant risk, with over 10% experiencing depression. Unfortunately, suicide claims the lives of over 700,000 people annually, ranking as the fourth leading cause of death among individuals aged 15 to 29 (World Health Organization, 2023). According to a survey on depression prevalence in the U.S., in 2020, 9.2% of Americans aged 12 years and older experienced a past-year major depressive episode. The highest prevalence was observed among young adults aged 18–25 years (17.2%), closely followed by adolescents aged 12–17 years (16.9%). Depression saw the most significant increase among adolescents and young adults, with an upward trend across various demographic groups, including sex, race/ethnicity, income, and education. Interestingly, depression prevalence remained stable among adults aged 35 years and older, and the rate of seeking help for depression consistently remained low throughout the study period (Goodwin at al., 2022).

Etiology

Depression is influenced by a combination of biological factors such as genetics and neurology, which interact with stress reactions and emotional processing. Environmental stressors, including acute events, chronic stress, and childhood adversity, contribute to the development of depression (England and Sim, 2009). Additionally, personal vulnerabilities,

encompassing cognitive and interpersonal factors, play a role in shaping an individual's susceptibility to depression.

Depression's familial link involves genetic and environmental factors, with about one-third of the risk attributed to genetic differences (Sullivan, Neale and Kendler 2000). The risk increases 2.5–3 times with a first-degree relative having depression and 5 to 16 times after highly threatening life events (Kendler et al., 1998). Research on genetics reveals a significant likelihood of major depressive disorder (MDD) in twins, especially in identical twins, indicating a strong genetic influence on MDD susceptibility (Sullivan, Neale and Kendler 2000). Notable genetic variations, especially in serotonin-related genes like 5-HTTLPR, are associated with an increased risk of depression in response to stress (Caspi et al., 2003). A prevalent neurobiological model of depression highlights dysregulation in the body's stress response, involving the HPA axis and related systems (England and Sim, 2009). Elevated cortisol levels, the primary stress hormone, are linked to depression, with sustained hypercortisolism impacting emotion regulation circuits. Genetic differences, particularly in the glucocorticoid receptor (GR), contribute to individual variations in the stress response. Furthermore, researchers investigated the connection between depression and the immune system, particularly proinflammatory cytokines. Their findings showed that chronic stress may trigger inflammation, leading to depressive symptoms (Miller and Blackwell, 2006). Cytokines, like interleukin-1β, interleukin-6 (IL-6), and tumor necrosis factor-α, play a role in this process, with downstream products like C-reactive protein (CRP) serving as an index of inflammation. Chronic stress is associated with elevated CRP and depression, suggesting a potential bidirectional relationship. Interestingly, a longitudinal study revealed that childhood maltreatment is linked to both depression and inflammation (Danese et al., 2008). Additionally, environmental factors play an important role in the etiology of depression. One crucial environmental factor is inconvenient life events. A study showed that recent stressors are more common in depressed individuals, with 80% of community depression cases preceded by major negative life events (Mazure, 1998). The impact of an acute event depends on its circumstances and subjective meaning to the individual. Losses, particularly those related to the sense of self, significantly contribute to depression (England and Sim, 2009). Immigrant and refugee populations, experiencing loss and isolation, often face high rates of depression (Essayagh, 2023). Another environmental factor is stress, sometimes even chronic stress. The relationship between stress and depression is bidirectional, as individuals with depression or a history of it experience more acute stressors, creating a cycle that can lead to recurring or chronic depression (England and Sim, 2002).

Treatment

Regarding treatment for depression, notable psychological interventions include cognitive-behavioral therapy, behavioral activation treatments, interpersonal psychotherapy, and problem-solving treatment (Cuijpers and Straten, 2009). In depression treatment, cognitive therapy targets changing how individuals perceive and interpret situations by reducing their significance, while behavioral therapy aims to identify and modify destructive behavioral patterns to foster improvement (Institute for Quality and Efficiency in Health Care, 2006). However, Cuijpers and Straten's findings (2009) suggest that integrating psychological interventions with medical approaches improves outcomes compared to utilizing psychological treatment alone. In terms of pharmacological solutions, approved medications for Major Depressive Disorder (MDD) include various classes, including SSRIs (selective serotonin reuptake inhibitor), SNRIs (Serotonin–norepinephrine reuptake inhibitor), serotonin modulators (e.g., trazodone), atypical antidepressants (e.g., bupropion, mirtazapine). SSRIs are often preferred as first-line treatments. Moreover, adjunctive medications like mood stabilizers and antipsychotics may also be used to enhance efficacy (Bains and Abdijadid, 2024).

Depression and its connection to PMS

Interestingly, depression shares a significant symptom with the premenstrual syndrome and the premenstrual dysphoric disorder: The experience of a depressed mood. However, a distinctive characteristic of PMS is that its symptoms usually fade away with the beginning of menstruation. Regarding potential long-term associations between depression and PMS, the primary focus lies in understanding whether individuals diagnosed with depression are at a heightened risk of experiencing more severe PMS symptoms, and vice versa. Past research on this matter has shown contrary results:

In Yonkers, Pearlstein and Rosenheck's study (2003), 22% of the women with premenstrual symptoms exhibited comorbid Major Depressive Disorder (MDD), and 5% had minor depressive disorder. Among those with potential premenstrual dysphoric disorder, 24% acknowledged experiencing suicidal thoughts at different levels (several days, more than half the days, or every day), with 20% endorsing these thoughts for several days.

Strine, Chapman and Ahluwalia's study (2005) used the data of women aged 18–55 years (n = 11,648) who participated in the 2002 National Health Interview Survey, a continuous,

computer-assisted personal interview of the noninstitutionalized U.S. population. They found that those with menstrual-related symptoms were significantly more prone to depression in the past 12 months compared to those without. Additionally, women with menstrual-related issues were notably more likely to report feelings of sadness, nervousness, restlessness, hopelessness, or worthlessness.

A study by Hurt et al. (1992) found that the risk of late luteal phase dysphoric disorder (LLPDD), was 14% higher in women with a history of psychological disorders. However, this presented a significant but relatively small risk for developing LLPDD. Interestingly, there was no increased risk for women who had previously experienced major depression. Additionally, there has been research, examining if the underlying causes and risk factors for PMS and depression are similar. Kendler et al.'s study (1998) on PMS and major depression found that these symptoms are moderately stable and largely heritable. Over six years, 1,312 female twins were assessed, revealing a 56% heritability rate for premenstrual symptoms. Family-environmental factors had no significant impact. The bivariate twin-measurement model found that genetic and environmental risk factors for lifetime major depression only had a modest impact on the development of premenstrual syndrome. Overall, it concluded that the genetic and environmental risk factors for premenstrual symptoms and major depression are not closely related. In 2011, Forrester-Knaus et al. conducted a study in 2011 exploring the relationship between premenstrual syndrome (PMS) and major depression in women of reproductive age. Analyzing data from the Swiss Health Survey 2007, they found that the prevalence of major depression was 11.3% in women with moderate PMS and 24.6% in those with severe PMS. Factors such as alcohol consumption, antidepressant use, oral contraceptive use, and work dissatisfaction were associated with varying risks for PMS and major depression. Furthermore, women experiencing both PMS and major depression were more significantly impaired, suggesting distinct yet co-occurring disorders. The study's findings show the importance of treating women with both major depression and PMS due to their heightened impairment. Forrester-Knaus et al. underline that detecting and treating comorbidity is crucial for understanding and addressing the co-occurrence of both conditions effectively.

Even though there are conflicting findings about the potential shared etiology of premenstrual symptoms and depression, it is obvious that the two disorders share similar symptoms. This can often lead to complications in differentiating and diagnosing the conditions. While some researchers emphasize similarities with anxiety disorders, particularly panic disorder, others argue the premenstrual dysphoric disorder should be seen as a variant

of depression. The preferred treatment, serotonin reuptake inhibitors (SRIs), is commonly used for depression and anxiety disorders. Landén & Eriksson's review (2003) concludes that PMDD is a distinct diagnostic entity, characterized by irritability and affect lability rather than depression or anxiety. The unique clinical profile of SRIs in PMDD, including a rapid onset of action, suggests a distinct serotonergic mechanism.

Skin disorders and their connection to depression

Adding another layer to the potential correlation between PMS and depression is the exploration of their connection to skin disorders. Past research has found positive associations between skin disorders and depression in children and adolescents (Teichgräber et al., 2021). In their study, Teichgräber et al. analyze 7,061 depression cases and matched controls. They found significant associations with atopic dermatitis/eczema, nail disorders, and hair loss. Another study explored the underdiagnosis of depression and anxiety among European patients with common skin diseases (Dalgard et al., 2015). In a cross-sectional multicenter study involving 3635 consultations, dermatologists' clinical assessments were compared to the Hospital Anxiety and Depression Scale (HADS). Results showed poor to fair agreement for all diagnosis categories, with dermatologists recognizing signs of depression or anxiety in 44.0% and 35.6% of cases.

Movrogiorgu et al. aimed to explore dermatological comorbidities in psychiatric patients outside dermatological treatment facilities. Analyzing data from over 17,000 patients with primary psychiatric disorders, they found that 1.24% (n=212) had additional dermatological diseases. Psoriasis (35.4%) and atopic dermatitis (22.6%) were the most prevalent among these patients, with infectious-parasitic skin diseases present in 13.2% of cases. Depressive illness was the most common mental disorder, affecting 42.5% of patients (Mavrogiorgou et al., 2020).

Basavaraj, Navya and Rashmi (2010) explain in their article that psychodermatologic disorders encompass the interplay between the mind and the skin, classified into psychosomatic, primary psychiatric, and secondary psychiatric disorders. Psychodermatology is further categorized into three types based on the relationship between skin diseases and mental disorders: 1) Psychophysiologic (psychosomatic) disorders, where skin diseases trigger various emotional states (such as stress) but are not directly associated with mental disorders (e.g., psoriasis, eczema); 2) primary psychiatric disorders leading to self-induced skin disorders (e.g., trichotillomania); and 3) secondary psychiatric disorders arising from

disfiguring skin conditions (e.g., ichthyosis, acne conglobata, vitiligo), potentially causing feelings of fear, depression, or suicidal thoughts.

Another study's findings confirm that individuals suffering from skin diseases have a higher risk of developing depressive symptoms alongside from social withdrawal, feelings of isolation, and an overall lower quality of life (Yew et al. 2020).

Depression and its connection to inflammation

Furthermore, investigations into the physiological underpinnings of depression have revealed the prominence of inflammatory processes, including elevated levels of innate immunity-associated inflammatory molecules.

According to Serafini et al. (2024), inflammation emerges as a significant feature not only in depression but also in suicidal behavior. Furthermore, Serafini et al. propose that including anti-inflammatory strategies for treating depression and preventing suicide could improve outcomes. Another study on suicidal behavior and inflammation (Brundin et al.,2019) highlights the increasing evidence linking immune system dysregulation to suicidality. Inflammatory conditions like traumatic brain injury, vitamin deficiency, autoimmune disorders, and infections are identified as potential triggers for suicidal behavior. These conditions elevate inflammatory mediators, disrupting the kynurenine pathway, HPA axis, and monoamine metabolism. These neurobiological effects may lead to profound alterations in emotion and behavior, contributing to suicide in vulnerable individuals. Dantzer et al. (2008) explain that during peripheral infection, pro-inflammatory cytokines induce sickness behavior in the brain. Therefore, prolonged immune activation in conditions like systemic infections can exacerbate sickness, leading to depressive symptoms in vulnerable individuals. This mechanism could contribute to the elevated prevalence of clinical depression in physically ill patients with no prior history of mental disorders.

Skin disorders and their connection to PMS

Past research has shown that also the premenstrual syndrome exhibits a noteworthy correlation with skin problems, for example specific conditions such as Cyclic Catamenial Dermatoses. This skin disorder manifests only during the premenstrual phase, characterized by unusual cyclic reactions influenced by hormones produced during the menstrual cycle. Kiriyama et al. (2003) conducted a study analyzing the data of 286 Japanese women with

atopic dermatitis were interviewed to assess the influence of the menstrual cycle on their skin lesions and to identify potential symptoms of premenstrual syndrome. The study's findings revealed that 47% of the patients experienced monthly skin deterioration, predominantly during the premenstrual week. The severity varied among participants, and all those with premenstrual skin aggravation also showed symptoms of premenstrual syndrome. In conclusion, premenstrual worsening of skin lesions is prevalent in about half of women with atopic dermatitis, closely associated with premenstrual syndrome.

Raghunath, Venables and Millington (2015) highlight in their review that pre-menstrual exacerbations of dermatoses are common, with evidence suggesting that estrogen is associated with improved skin thickness, water content, barrier function, and wound healing. Peak levels of progesterone correlate with dermatoses exacerbations like acne, psoriasis, eczema, and dermatitis. These reactions result from cyclical hormonal fluctuations, impacting immune and barrier functions.

However, like the depressive symptoms during the luteal phase, these perimenstrual skin reactions are also temporary. They typically disappear as the menstrual cycle progresses. This is why it is crucial to distinguish these temporary premenstrual skin disorders from the link between general sensitivity to PMS and immune parameters that persist throughout the menstrual cycle.

This distinction is supported by findings from Itsekson's studies (2004, 2019), which explore hypersensitivity reactions to sex hormones in patients with PMS and associated skin diseases. In 2004, Itsekson recruited ten patients with both PMS and skin diseases, ten with PMS only, and ten healthy women for his study. The goal was to investigate hypersensitivity reactions to sex hormones in these groups. Both immediate and delayed hypersensitivity reactions were observed in patients with PMS and PMS-related skin diseases. However, such reactions were not observed in healthy women. Furthermore, the study explored the effects of desensitization, a process to reduce hypersensitivity, on PMS symptoms and associated skin diseases. The results showed that desensitization led to a decrease in PMS symptoms and improvement in skin conditions related to PMS. This indicates a potential connection between hormonal fluctuations in PMS and skin hypersensitivity reactions. Itsekson conducted another study about female sex homones in 2019, to investigate hormonal skin testing as a diagnostic tool for sex hormone sensitivity in women with confirmed PMS. In the process, intradermal injections of Progesterone, Estradiol, Estrone, and Estriol were administered, and skin reactions correlated with PMS severity. Furthermore, desensitization via serial injections led to a significant improvement in PMS-related symptoms. This safe and sensitive diagnostic

approach holds therapeutic potential for resistant hormonal cycle-related syndromes. Both of Itsekson's studies reveal a potential connection between hormonal fluctuations in PMS and skin hypersensitivity reactions, suggesting long-term implications for skin health beyond transient perimenstrual reactions.

PMS and its connection to Inflammation

Additionally, investigations into inflammation in the context of PMS reveal potential connections: Several studies posit that inflammatory factors may exhibit elevated levels in women experiencing PMS. It is conceivable that alterations in immune function leading to chronic inflammation may directly play a role in expressing symptoms during the premenstrual period (Bertone-Johnson, 2016).

In Puder's et al. study (2006), the link between symptoms and inflammation across the menstrual cycle in normal weight and overweight women with and without premenstrual syndrome was examined. They found that regardless of weight, premenstrual symptoms correlated with inflammation markers like hs-CRP, independent of hormone levels, but not independent of the menstrual phase. Interestingly, inflammation was not strongest in the luteal phase but in the follicular phase. Their findings suggest that inflammation influences symptoms during the premenstrual phase.

Gold, Wells and Raiser (2016) investigated the relationship between inflammation and premenstrual symptoms in midlife women. Using data from the Study of Women's Health Across the Nation, they analyzed the high-sensitivity C-reactive protein (hs-CRP) as an inflammation biomarker. They found that higher hs-CRP levels were significantly associated with mood symptoms, abdominal cramps/back pain, appetite cravings/weight gain/bloating, and breast pain. However, no association was found with headaches or experiencing three or more premenstrual symptoms. These findings suggest potential clinical implications for using anti-inflammatory agents in treatment and prevention of premenstrual symptoms.

Skin barrier integrity and transepidermal water loss

Another marker of dermatological health is skin barrier integrity. The skin, a self-renewing organ, protects the body from external factors like mechanical, chemical, and microbial threats. It comprises two main layers: the outer epidermis and the deeper dermis. These layers collaborate to form the basement membrane, which separates and stabilizes

them. While both layers contribute to defense, the epidermis primarily maintains the body's internal environment by preventing water and nutrient loss and shielding against environmental hazards (Baroni et al., 2012).

The epidermis includes physical, chemical, biochemical, and immunological barriers. The physical barrier, primarily the stratum corneum (SC), is complemented by the nucleated epidermis, which provides additional components such as cell-cell junctions and cytoskeletal proteins. The chemical and biochemical barrier encompasses lipids, acids, enzymes, antimicrobial peptides, and macrophages. Lastly, the immunological barrier comprises humoral and cellular elements of the immune system (Proksch, Brandner and Jensen, 2008). The stratum corneum (SC) acts as the primary barrier, protecting against the penetration of chemicals and microbes, and resisting mechanical forces. It also regulates water release through transepidermal water loss (TEWL). Skin barrier function is typically assessed using TEWL, measured in grams per square meter per hour ($g/m^2/h$). TEWL is commonly evaluated in a controlled environment at room temperature (21°C) and 50% relative humidity. A standardized method involves acclimating the skin for 15 minutes before measurement using a thermohydrostatic chamber. However, TEWL measurements can vary due to factors like angle, temperature, and pressure. Additionally, controlling external factors like perspiration in the measurement environment can be challenging, leading to variability in results. As an alternative method, fluorescent video microscopy can visualize skin barrier function by observing how sodium fluorescein penetrates the stratum corneum (Uehara, Kusuhara & Nakamura, 2023). After applying sodium fluorescein to the skin, excess is removed, and penetration is observed with a fluorescent video microscope. This method correlates fluorescence intensity with TEWL, offering potential for evaluating skin barrier function. Another method involves using machine learning algorithms to estimate TEWL from skin images. While it seems promising, challenges include the need for standardized images and understanding the mechanism behind transepidermal water loss.

Skin barrier integrity and its connection to inflammation

Like PMS and depression, skin barrier integrity is also linked to inflammation. Strugar et al (2019) explains in their report that the compromission of the barrier can lead to a greater chance of harmful substances, irritants, and pathogens infiltrating the skin, triggering inflammation. This inflammation can cause various skin issues, including infections, allergic reactions, and long-term inflammatory conditions such as atopic dermatitis and contact

dermatitis. Additionally, a former study discovered that type 2 inflammation, which is associated with conditions like allergies and autoimmune diseases, might be connected to disruption of the skin barrier (Beck et al., 2022). The study's findings showed that skin barrier disruption induces type 2 inflammation, and conversely, type 2 inflammation exacerbates skin barrier disruption. Furthermore, Beck concludes that therapies targeting this inflammation may improve the compromised skin barrier in Atopic dermatitis. Another study aimed to find a new way to stop symptoms of atopic dermatitis by blocking a protein called PAR2 (Protease activated receptor 2). They discovered that a substance called NDGA (Nordihydroguaiaretic acid) can block PAR2 and reduce inflammation in skin cells. The findings propose that NDGA could be helpful in treating skin conditions like atopic dermatitis (Kim et al., 2012). Another study examined the relationship between skin inflammation, barrier function, and itching from dry skin using a mouse model (Zhu et al., 2023). Researchers tested a moisturizing spray with β-glucan and panthenol to alleviate itching. They induced chronic itching in mice and observed increased scratching and disrupted skin barrier function. The spray reduced scratching, improved the barrier function, and lowered inflammation, suggesting its potential for treating dry skin-induced itching. These results propose a connection between impaired skin barrier, dry skin, and inflammation.

Skin barrier integrity – a possible link to PMS and depression?

Skin barrier disruption may also cover associations with psychological disorders. Altemus et al.'s study (2001) examined the effects of psychological interview stress, sleep deprivation, and exercise on skin barrier function in women. The results showed that psychological stress and sleep deprivation delayed barrier function recovery and increased plasma levels of certain cytokines and natural killer cell activity. Exercise did not affect barrier function recovery but increased natural killer cell activity and circulating lymphocyte numbers. The results suggest that acute stress disrupts skin barrier function homeostasis, potentially through changes in cytokine secretion. Another study investigated the relationship between psychological stress, sleep quality, and skin health among medical students (Lyu et al., 2023). It found that as the semester progressed, sleep quality worsened and anxiety levels increased, correlating with impaired skin barrier function. Mental stress negatively impacted skin health through poor sleep and severe anxiety. The authors address the importance psychological well-being for maintaining healthy skin. A third study also explored how psychological stress affects skin barrier function. Stress increases glucocorticoids, which

harm the skin barrier. The study found that stress elevates an enzyme called 11ß-HSD1, leading to higher cortisol levels in the skin and worsened barrier function. Treating stress with SSRIs reduced 11ß-HSD1 levels and improved barrier function, suggesting a potential treatment for stress-related skin issues. Notably, SSRIs are also used as treatment for depression and PMS (Choe et al., 2018).

Research gap and Hypotheses

Given the established correlation between inflammation and the three variables under consideration - PMS, depression, and skin barrier integrity - it's plausible to suggest a potential interconnection among them. Previous research has identified several key relationships. For instance, several studies posit that inflammatory factors may exhibit elevated levels in women experiencing PMS, indicating a direct association to inflammation. Similarly, the physiological underpinnings of depression have revealed the prominence of inflammatory processes, suggesting that inflammation plays a significant role in the development and progression of depressive disorders. The connection between skin disorders and depression has also been documented, with evidence showing that these conditions can co-occur. Furthermore, interventions aimed at desensitization to reduce hypersensitivity to sex hormones in PMS patients have been observed to lead to a decrease in skin disorders, hinting at a direct link between PMS and skin health. Despite these findings, there remains a gap in the literature regarding the specific correlation between depression, PMS, and skin barrier integrity.

While existing studies have shed light on how mental stress can compromise the skin barrier, primarily through factors such as poor sleep and anxiety, the direct link to depression and PMS remains relatively underexplored. The intricate mechanisms by which these mental health conditions might impact the skin barrier, and how this could relate to symptoms of PMS and depression, represent areas ripe for investigation.

Additionally, the literature on PMS, skin health, and depression has predominantly concentrated on skin related and depressive symptoms occurring in the premenstrual phase. This is why it is important to explore the interactions between these factors throughout the entire menstrual cycle, in order to examine possible comorbidities of the conditions.

Moreover, it's essential to consider potential bidirectional relationships among these variables. For instance, while inflammation has been implicated in both PMS and depression,

it's also plausible that alterations in skin barrier integrity could influence the severity or frequency of depressive episodes and PMS symptoms. Understanding the complex interplay between these factors could offer valuable insights into new therapeutic interventions or preventive measures targeting multiple aspects of health simultaneously.

To fill this research gap, this thesis aims to explore the potential connections between the premenstrual syndrome, depressive symptoms, and skin barrier integrity. It is crucial to underscore that the study focuses on symptoms occurring throughout the entire menstrual cycle, rather than solely during the premenstrual phase. This distinction is important because the study evaluates participants during a menstrual cycle phase outside of the premenstrual window (i.e., during the follicular phase). Thus, the associations observed are relevant to the menstrual cycle as a whole, rather than being limited to the premenstrual phase.

Furthermore, due to constraints in participant selection, the study couldn't include individuals diagnosed with clinical depression. Consequently, the term "depressive symptoms" is utilized instead of "depression" throughout the entire thesis to accurately reflect the population under investigation.

This master thesis aimed to address the following research questions:

Research Question 1: Are depressive symptoms linked to impaired skin barrier integrity?

Hypothesis 1 (H1): Individuals reporting higher levels of depressive symptoms show higher transepidermal water loss, indicating decreased skin barrier integrity.

Research Question 2: Is PMS associated with impaired skin barrier integrity?

Hypothesis 2 (H2): Individuals reporting more severe PMS symptoms may show higher levels of transepidermal water loss, indicating potential skin barrier impairment.

Research Question 3: Is PMS associated with depressive symptoms?

Hypothesis 3 (H3): Individuals reporting more severe PMS symptoms may also report higher levels of depressive symptoms.

Given the uncertainty surrounding the direction of the effect, and the study's inability to discern a predetermined causal variable or dependent one, non-directional hypotheses were formulated to maintain impartiality and allow for open exploration of potential relationships between variables.

Methods

The data utilized for this master's thesis were gathered within an ongoing two-study project that is led by Dr. Majdandžić and Prof. Dr. Nater from the Music and Health Lab. The primary focus of the project revolves around whether individuals experience faster skin barrier recovery when exposed to music, particularly after it has been compromised through tape stripping. In addition to stress, heart rate, and salivary flow, the study also assessed depressive symptoms, the premenstrual syndrome, and tansepidermal water loss.
Notably, while both studies are identical, the second study (Study 2) includes the Trier Social Stress Test (TSST) to induce and measure acute stress, distinguishing it from the first study (Study 1).

Sample

Participants in both studies had to meet specific inclusion and exclusion criteria, ensuring a homogeneous sample for analysis. These criteria included being assigned female at birth, aged between 18 and 35 years, and maintaining a regular menstrual cycle. Additionally, participants were required to be free from chronic somatic illnesses, including cardiovascular diseases, respiratory diseases, liver diseases, hypertension, chronic pain, and metabolic diseases. Mental health history was also considered, with participants excluded if they had experienced depression, psychosis, anxiety disorders, or eating disorders within the last five years. Only individuals with no hearing impairment, chronic tinnitus, music-related professions, or absolute hearing were included to control for confounders related to music experience. Furthermore, participants had to adhere to specific criteria to enable tape-stripping, including the absence of chronic or acute inflammatory skin diseases, allergies to adhesive tape, or any skin abnormalities on the volar forearm. In Study 2, additional criteria were imposed, such as having no prior experience with the Trier Social Stress Test (TSST) and no personal relationship with the study team members. This was necessary to ensure consistency in the efficacy of the TSST across participants.

Recruitment efforts involved promoting the project via social media platforms, university mailing lists, and distributing flyers to attract potential participants. Information provided included basic criteria, contact details, and the financial incentive offered. Interested individuals underwent a telephone screening process to assess their compatibility with the study's criteria. Eligible participants received detailed information about the study procedures

and were asked to provide consent for their involvement. Additionally, participants were requested to provide details about their menstrual cycle to schedule testing during the follicular phase optimally. Before the testing date, participants were asked to complete a battery of questionnaires, accessible through a provided link. A reminder email was sent two days before the testing date to ensure participants' preparedness.

Assessing depressive symptoms

The Beck Depression Inventory (BDI) is a 21-item self-reporting questionnaire designed to assess the severity of depression in both normal and psychiatric populations. Developed by Beck and colleagues, the BDI underwent revisions in 1978, resulting in the BDI-IA, and in 1996, leading to the creation of the BDI-II (Beck et al., 1996). The BDI is grounded in the theory of negative cognitive distortions as central to depression. The BDI-II does not rely on any particular theory of depression and has been translated into several languages, with a shorter version, the BDI Fast Screen for Medical Patients (BDI-FS), available for primary care use. The BDI-II has been demonstrated to have very high measurement precision, with a reliability greater than .90 (Keller et al., 2022).
The questionnaire consists of 21 items, each scored on a 4-point scale ranging from 0 (absence of the symptom) to 3 (severe symptoms). Items cover affective, cognitive, somatic, and vegetative symptoms, aligning with the criteria for major depression outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994). The recall period for the BDI-II is the preceding two weeks (Beck et al., 1996).

Scores on the BDI-II range from 0 to 63, with higher scores indicating greater severity of depressive symptoms. In non-clinical populations, scores above 20 are indicative of depression. For individuals diagnosed with depression, scores fall into the following categories: 0–13 (minimal depression), 14–19 (mild depression), 20–28 (moderate depression), and 29–63 (severe depression).

The BDI-II demonstrates high construct validity and concurrent validity, with strong correlations observed between BDI scores and other measures of depression (Jackson-Koku, 2016). Criterion validity is supported by positive correlations with established depression assessment tools. The BDI-II also exhibits high test-retest reliability and internal consistency. The BDI-II is copyrighted, and the rights are held by Harcourt Assessment Incorporated (Pearson Education plc), under contract from the author. In occupational health, the BDI-II

can be used as a screening tool to detect depression in normal populations or as a tool to assess symptom severity in clinical populations.

Assessing PMS

Before participants arrived for testing, the premenstrual syndrome (PMS) was assessed using an online questionnaire, specifically, the PMS-1 questionnaire, developed by Ditzen et al. (2011), which has demonstrated high reliability with a Cronbach's alpha of .879.
The PMS questionnaire was designed based on the DSM-IV-TR criteria for the premenstrual dysphoric disorder. Each diagnostic criterion was subjectively formulated into one or more items (see DSM-IV-TR research criteria and corresponding formulations of the PMS questionnaire in Table 1). The DSM-IV-TR does not inquire about how many days before the onset of menstruation the symptoms occur and how long they last, as this is captured through prospective assessment. This fact is considered in the PMS questionnaire by using the phrase "(e.g., stronger) ... than usual" in the items. Equivalent to the calculation of symptoms in the DSM-IV-TR, each item could be rated as "applies" or "does not apply." The questionnaire comprised the "PMS in General" (PMS-iA) and "PMS at the Moment" (PMS-iM) scales. Scores on the PMS-iA scale served for the retrospective assessment of premenstrual symptoms during the last 12 months. PMS-iM referred to the current severity of symptoms. Scores between 0 and 30 could be obtained on both scales. It is important to note that for this study, the PMS-iA scale was used.

This questionnaire assesses premenstrual syndrome by asking women to self-report various psychological and physical symptoms experienced during different phases of their menstrual cycle. The questionnaire is divided into two parts: A symptom assessment and a functional impairment assessment.

The first part of the questionnaire asks respondents to indicate whether they are currently experiencing specific symptoms associated with PMS. These symptoms include mood changes (e.g., feeling sad, hopeless, irritable), anxiety, fatigue, changes in appetite and sleep patterns, physical symptoms (e.g., breast tenderness, headache, joint pain), and perceived changes in weight or body image. Respondents rate the intensity of each symptom on a scale ranging from "Does not apply" to "Applies strongly."

The second part of the questionnaire evaluates the extent to which these symptoms impair various aspects of the respondent's life. It assesses the impact of symptoms on work or

school performance, social activities, and interpersonal relationships. Respondents rate the extent of impairment on a scale ranging from "Not at all" to "Strongly."

Based on the PMS-I questionnaire provided, a participant is considered to have PMS if they report experiencing at least five symptoms as "trifft stark zu" (applies strongly) during the premenstrual phase. These symptoms should include a combination of both psychological and physical symptoms listed in the questionnaire. Therefore, PMS is identified when the participant experiences a significant impact from these symptoms during the premenstrual phase.

Table 1

DSM-IV-TR research criteria for PMDD, the corresponding formulations, and quality criteria of the original version of the PMS questionnaire, (Ditzen, 2011)

PMDD criteria	Items
clear depressive mood, feelings of hopelessness, self-deprecating thoughts	“My mood is significantly worse than usual.” “I feel more hopeless than usual.”
clear anxiety, tension, feelings of being tense and irritable	“I feel stronger anxiety than usual.” “I feel more irritable than usual.”
noticeable mood swings (sudden feelings of sadness or heightened sensitivity to rejection)	“I have to cry out of a sudden.” “I suddenly feel sad.” “I am struggling with rejection.”
persistent and pronounced anger, Increased interpersonal conflicts	“I get angry more often than usual.” “I get involved in more conflicts than usual.”
decreasing interest in usual activities (such as work, school, hobbies)	“I am less interested in things I usually do.”
subjective feeling of difficulty concentrating	"It seems like I'm having more difficulty concentrating than usual."
lethargy, easy fatigue, or significant loss of energy	“I get tired faster than usual.”

significant changes in appetite, eating when not hungry, craving for specific foods	"I eat more than usual." "I am craving specific foods."
excessive sleepiness and insomnia	"I sleep more than usual." "I sleep less than usual."
subjective feelings of being overwhelmed and losing control	"I feel like I have less control than usual."
other physical symptoms like breast tenderness or swelling, headaches, joint or muscle pain, feeling bloated, weight gain	"I feel tightness in my chest." "My chest feels swollen." "My head aches." "My joints ache." "I weigh more than usual."

Transepidermal water loss

Transepidermal water loss was evaluated using the TEWAMETER TM300 developed by Courage + Khazaka Electronic GmbH, to assess skin barrier integrity. The TEWAMETER TM300 utilizes electrical measurements to gauge the hydration level of the skin surface, specifically measuring the humidity lost through a designated area of the skin on the lower non-dominant inner arm, approximately two centimeters from the elbow crease.
The setup comprises a hollow cylinder housing two sensors capable of measuring the density gradient of water evaporation pressure at various locations on the skin surface, while accounting for temperature. The disparity between measurements from these sensors is computed using Fick's laws of diffusion, showing values in grams per hour per square meter (g/h/ m^2) (Ariffin & Hasham, 2020).

The measurements were conducted under controlled conditions, with room temperature maintained between 22.5 and 23.5 °C and a probe temperature set at 34°C. Participants and experimenters were instructed to minimize speaking or breathing near the probe and to remain as still as possible during measurements. To avoid interference, air conditioning was deactivated, and all doors were closed during the procedure. Each participant had a small area of their skin marked for measurement, divided into three distinct sections arranged vertically below each other. These sections were consistently measured during each session, with a fourth area serving as a control site. Measurements continued until 20 consecutive readings with a standard deviation of less than 0.5 were obtained by the

device. To assess the baseline TEWL-value, a mean value was calculated for the three designated areas, given the discrepancies between multiple measurements did not exceed 10 g/h/ m². This mean measurement served as the baseline TEWL-value, and therefore as indicator for the participants' dermatological health.

Procedure

The main project is divided into two studies, namely MuSkiBa I (short for Music - Skin Barrier) and MuSkiBa II. In the study MuSkiBa I associations between music and skin barrier recovery are being investigated, whereas in study MuSkiBa II mediating effects of acute psycho-social stress on the association between music and skin barrier recovery are examined. Since not the whole project is directly relevant to the research questions at hand only crucial parts of the studies will be discussed in detail, while a general overview will assist in placing given focus points in the timeline.

Participants completed various online questionnaires before testing, assessing depressive mood (BDI 2; Jackson-Koku, 2016) and PMS (PMS-I; Ditzen, 2011). Protective factors, coping strategies (Brief-COPE; Carver, 1997), emotion regulation (ERQ; Sala et al., 2012), and resilience (RS-11; Leppert et al., 2008) were also evaluated. Health and well-being were examined using the PHQ-D (Spitzer et al., 2000) for mental disorders, and somatic symptoms were assessed with the MFI-20 (Smets et al., 1995). Stress reactivity (TICS; Schulz & Schlotz, 1999), perceived stress (PSS; Cohen et al., 1983), childhood trauma (CTQ; Bernstein et al., 2003), and personality traits (NEO-FFI; Borkenau & Ostendorf, 1993) were also measured, along with self-esteem (RSES; Rosenberg et al., 1989).

In both studies MuSkiBa I and MuSkiBa II participants were randomly assigned to one of three groups: a music listening group, serving as the experimental condition, or to either an audiobook listening group or a silence group, both serving as control conditions. In each test session, a team of two individuals conducted the study: a study leader who provided instructions and guidance, and a female study assistant responsible for carrying out TEWL measurements. Specifically, since the procedure for measuring transepidermal water loss involves close physical contact and requires the assistant to touch the participant, it was considered that a male assistant could potentially induce stress or discomfort in the female participants.

MuskiBa I (Study 1)

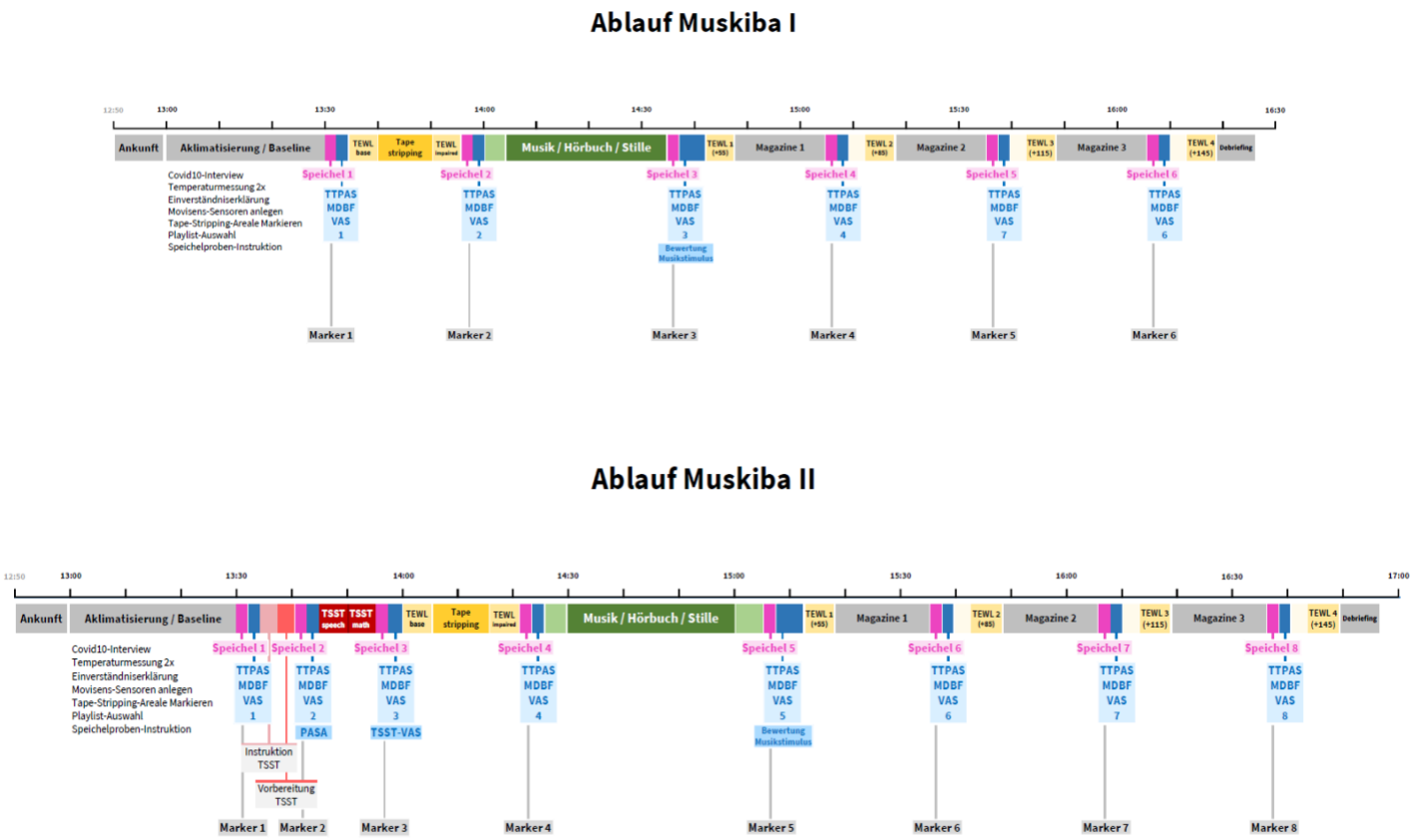
Every test session was scheduled to start at the exact same time of the day: 12:50pm. This specific timing was crucial to standardize cortisol level comparisons, as cortisol levels typically fluctuate throughout the day. Upon arrival, participants were greeted by the study leader and led to the designated testing rooms, where they underwent a thirty-minute acclimatization phase. During this time, participants were asked to sign the declaration of consent for the study. Following the acclimatization phase, participants were fitted with sensors for electrocardiogram data (placed on the solar plexus) and electrodermal activity (attached to the non-dominant hand). If participants were assigned to the music condition, they were asked to select a playlist from options such as classic music, lofi and other styles. Alternatively, if they were in the audiobook condition, they were prompted to choose a topic of interest from options like biology, physics, geography, or history. Subsequently, participants were instructed to collect saliva samples for two minutes without swallowing, followed by transferring the saliva into polypropylene tubes using a provided tube. A marker was set for EDA and ECG data, facilitating precise time marking for subsequent data analysis. TEWL measurements commenced, starting with a baseline measure, which held specific significance for the thesis. The tape stripping procedure followed, preceding another TEWL measure to evaluate skin barrier recovery. Afterwards, participants engaged in their designated activity (listening to music, an audiobook, or sitting in silence) for thirty minutes. Following this relaxation period, additional saliva samples and state questionnaires were administered, interspersed with thirty minutes of reading geo magazines to maintain relaxation. Finally, participants received a briefing about the study's objectives and remuneration, completing the study trial.

MuskiBa II (Study 2)

This study followed a similar procedure to Study 1, with the addition of the Trier Social Stress Test (TSST). After the acclimatization phase, participants were led into another test room at 1:20 PM. Here, two additional study assistants, one perceived as male (the active stressor) and the other perceived as female (the passive stressor), conducted the TSST. This test aimed to induce acute psychosocial stress in participants. Prior to the TSST, participants provided initial saliva samples and filled out state questionnaires. The study leader instructed participants to imagine they were applying for a job and had three minutes for preparation.

Following this preparation time, another saliva sample was taken, and additional state questionnaires were filled out. Participants then engaged in a simulated job interview, followed by a math exercise. Immediately after the TSST, participants provided another saliva sample and completed further questionnaires before returning to the testing room for TEWL measurements. The remainder of the study followed the same procedures as Study 1, allowing for a comprehensive investigation of the effects of acute psychosocial stress on skin barrier recovery, in addition to the influence of music exposure.

Figure 1



Note. The two timelines of Study 1 (Muskiba 1) and Study 2 (Muskiba 2)

Results

Statistical analysis

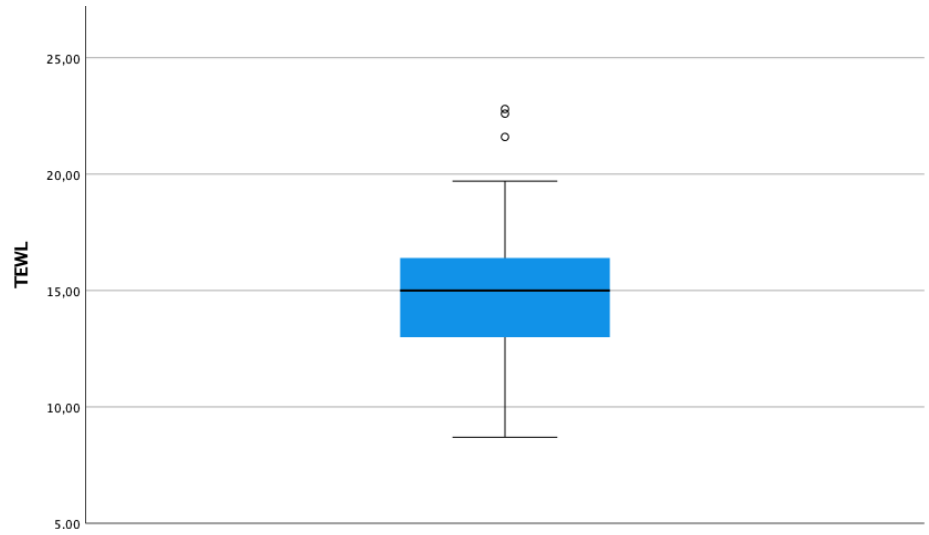
For statistical analysis the data from both studies, Muskiba I and Muskiba II was incorporated, using SPSS version 28 with a significance level set at $p < .01$ (two-tailed) for all tests. Firstly, a power analysis with G-Power (Faul et al., 2007) was conducted a priori. To be able to reliably find a correlation with a small effect size of 0.3, an $\alpha = 0.05$ and a power of 0.9 a sample size of at least 112 participants would be required. A two-sample t-test compared baseline transepidermal water loss values in Muskiba I and Muskiba II to ensure consistent TEWL values and unbiased data combination of both studies. Subsequently, outlier analyses were conducted to identify any data points that deviated significantly from the norm. Following the outlier exclusion, descriptive statistics were employed to evaluate the sociodemographic characteristics of the sample. Next, the distributions of each variable were analyzed, utilizing parameters such as the mean value (M), standard deviation (SD), and Cronbach's Alpha to evaluate construct reliability. The three hypotheses were assessed using Spearman's correlation analyses, while all parameters met the requirements of Spearman's correlation, being paired observations, and at least scaled ordinally.

Sample preparation

A two-sample t-test comparing baseline transepidermal water loss values in Muskiba I and Muskiba II groups showed no significant difference, indicating consistent TEWL values across studies. This finding is particularly important because the two studies differed in the events preceding the TEWL measurement. Muskiba II included a stress test, the Trier Social Stress Test (TSST), immediately before the TEWL measurement, which could potentially make participants nervous and lead to sweating or other physiological responses that might affect TEWL values. In contrast, Muskiba I did not include such a stress test before the measurement. Therefore, the lack of a significant difference in baseline TEWL values between the two groups indicates that the stress test did not have a measurable impact on the skin barrier function as assessed by TEWL. This allowed for unbiased data combination of the two studies. Outliers were identified through boxplot analyses, with three extreme outliers in TEWL (see Figure 2), likely due to measurement errors attributed to instrument sensitivity, were excluded as they far exceeded the typical range of 10-18 g/h/m². No significant outliers

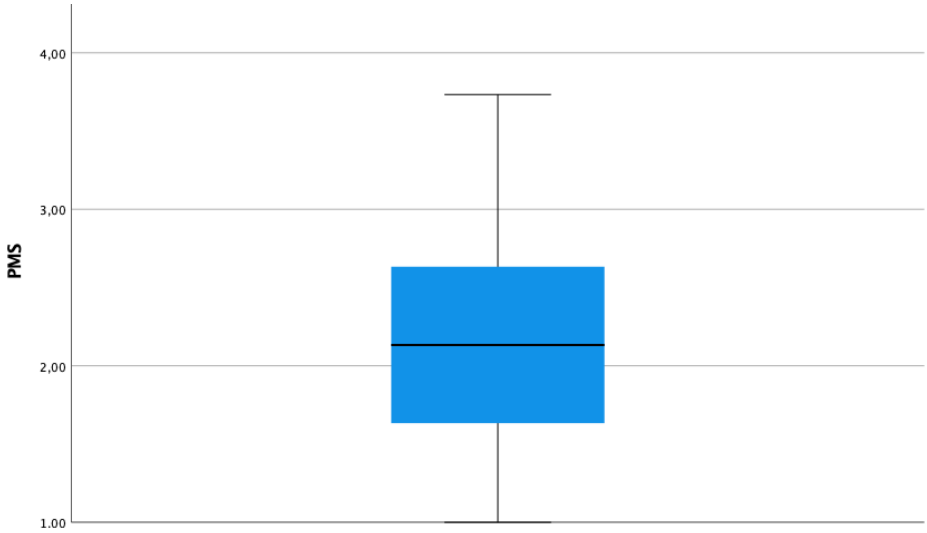
were found for PMS (Figure 3). For depressive symptoms, six outliers were found (see Figure 4). Those outliers were maintained in the statistical analyses for depressed mood, as participants with diagnosed depression had been previously excluded from the experiment. Excluding these outliers might potentially underestimate the observed effect size, given the already limited representation of individuals with clinically diagnosed depression

Figure 2



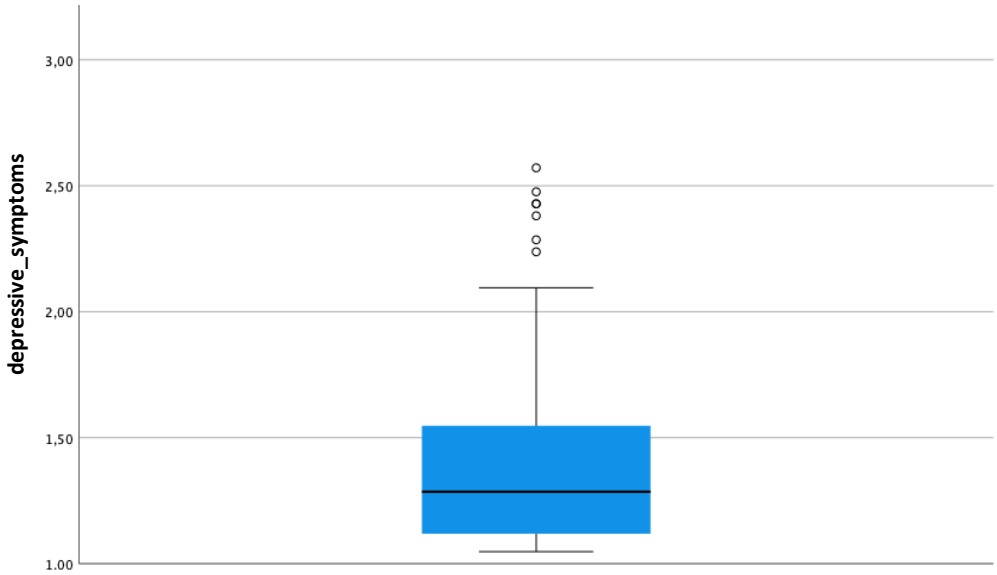
Note. Boxplot picturing the distribution of TEWL-scores, revealing three outliers

Figure 3



Note. Boxplot picturing the distribution of PMS-scores, revealing no outlie

Figure 4



Note. Boxplot picturing the distribution of "depressive symptoms"-scores, revealing six outliers

Sample description

Following the exclusion of outliers, data from a total of N=116 female participants were analyzed. These participants' age ranged from eighteen to thirty-five, with a mean age of 23.83 years (SD=3.75), while the BMI of the participants ranged from 17.58 to 25.39, with a mean BMI of 20.95 (SD=1.74). The majority of participants reported Austrian nationality (59%) or German nationality (21%), with the remaining participants representing various other nationalities.

In terms of education, 92% of the participants had completed at least Abitur/Matura, while the remainder reported different qualifications. Regarding occupation, the majority were either students (36%) or worked less than part-time (29%), with smaller percentages engaged in full-time work (6%), more than half-time work (12.5%), or being unemployed (8.3%). Most of the participants lived in a shared appartement (41%), with the remaining evenly split between living alone (19%), living with their partner (16%), or having a completely different housing arrangement (16%). Regarding their relationship status, 49% reported being in a relationship, 47% identified as single, 1% as married, 1% as separated/divorced, and 2% identified differently.

Table 2

Descriptive data of the sample

	Absolute values (n)	*Relative Values (%)*
Nationality		
Austria	68	59%
Germany	26	21%
Italy	1	1%
Ukraine	3	3%
Chile	1	1%
Croatia	3	3%
Hungary	4	3%
Luxembourg	1	1%
Columbia	1	1%
Albania	1	1%
Russia	1	1%
Serbia	2	2%
Swiss	1	1%
China	1	1%
France	1	1%
School education		
Secondary school	2	2%
Technical college entrance Qualification	4	3%
A-Levels	106	92%
Other	4	3%
Employment		
Fulltime	8	6%
At least part time	16	12%
Less than part time	37	29%
Student	46	36%
Unemployed	9	7%
Housing situation		

Alone	24	19%
Shared apartment	53	41%
With partner	18	14%
Other	21	16%
Relationship status		
In partnership	57	49%
Single	54	47%
Married	1	1%
Divorced	1	1%
Other	3	2%

Descriptive data of the parameters

The average score of PMS, measured by the "PMS", was 2.15 (SD= .674), ranging from 1 to 3,7 points, suggesting that on average, participants experienced PMS symptoms to a moderate degree. The mean score of "depressive symptoms" assessed by the "BDI" was 1,39 (ms; SD=.362), varying from 1,05 to 2,57 points, also indicating symptoms of a moderate degree. TEWL baseline scores showed a mean of 14.7 g/h/m2 (SD=2.47). Descriptive statistics of the parameters are shown in Table 3. Reliability analyses as Cronbach´s Alpha showed *r*.= .88 for PMS-1 and *r*. = .83 for BDI-I, indicating good (>.80) reliability for both scales.

Table 3

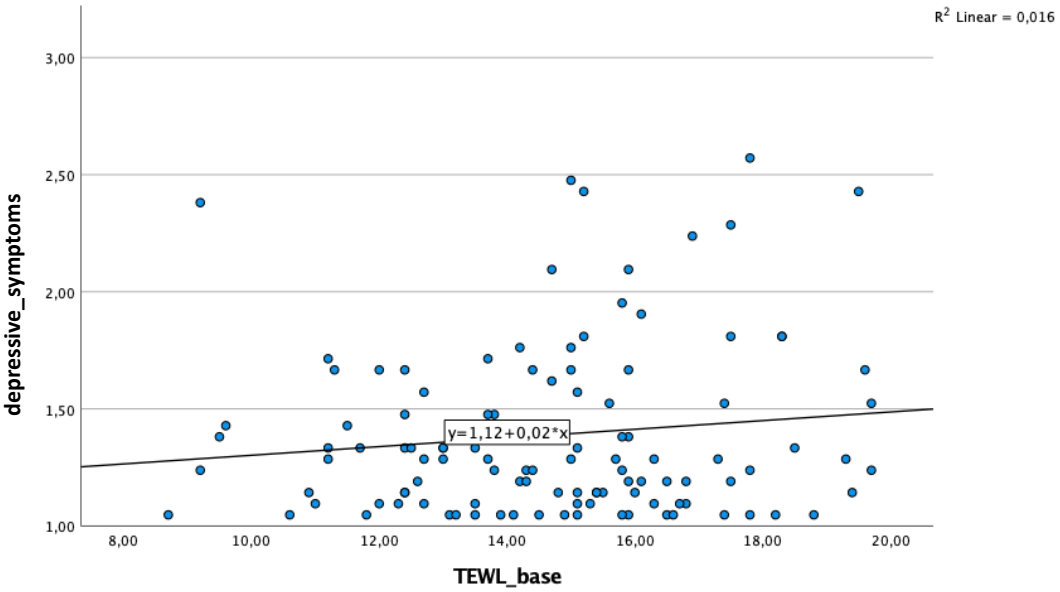
Descriptives of the scales

	PMS	*transepidermal water loss*	*depressed mood*
N	116	116	116
mean	2.15	14.70	1.39
STD	.67	2.47	.36
minimum	1.00	8.7	1.05
maximum	3.73	19.7	2.57

Depressive symptoms and transepidermal water oss

The first hypothesis explored the relationship between depressive symptoms and transepidermal water loss. Spearman analysis revealed no significant association (r = 0.055, p=0.558 [df = 114]) between the two variables, indicating that reported depressive symptoms and baseline TEWL did not predict each other. The scatterplot in Figure 8 confirms this, showing no discernible pattern among data points, with the regression line explaining only 1,6% of variance.

Figure 5



Note. Scatterplot of depressive symptoms and transepidermal water loss

Table 4

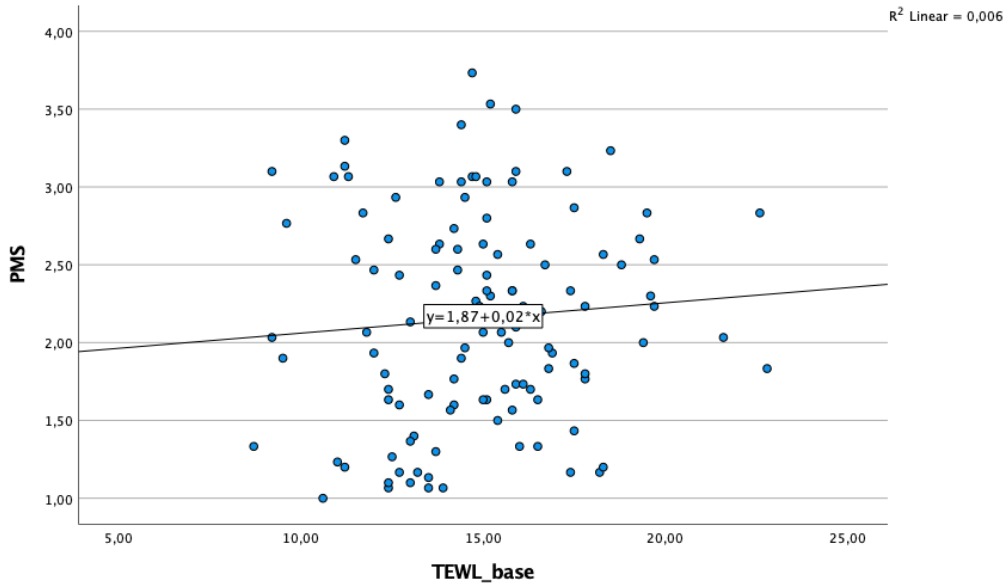
Spearman Correlation TEWL and depressive symptoms

	Correlation Coefficient	Sig. (2-tailed)	N
TEWL	1.000		116
depressive symptoms	0.055	.558.	116

PMS and transepidermal water loss

The second hypothesis investigated a possible link between the premenstrual syndrome and transepidermal water loss. However, Spearman correlation analysis found no significant correlation ($r = 0.085$, $p = 0.365$ [$df = 114$]), indicating no connection between reported PMS and baseline TEWL values. The scatterplot in Figure 9 also reflects this lack of correlation, with no discernible pattern among data points. The regression line explains only 0.6% of variance, likely coincidental.

Figure 6



Note. Scatterplot of PMS and transepidermal water loss

Table 5

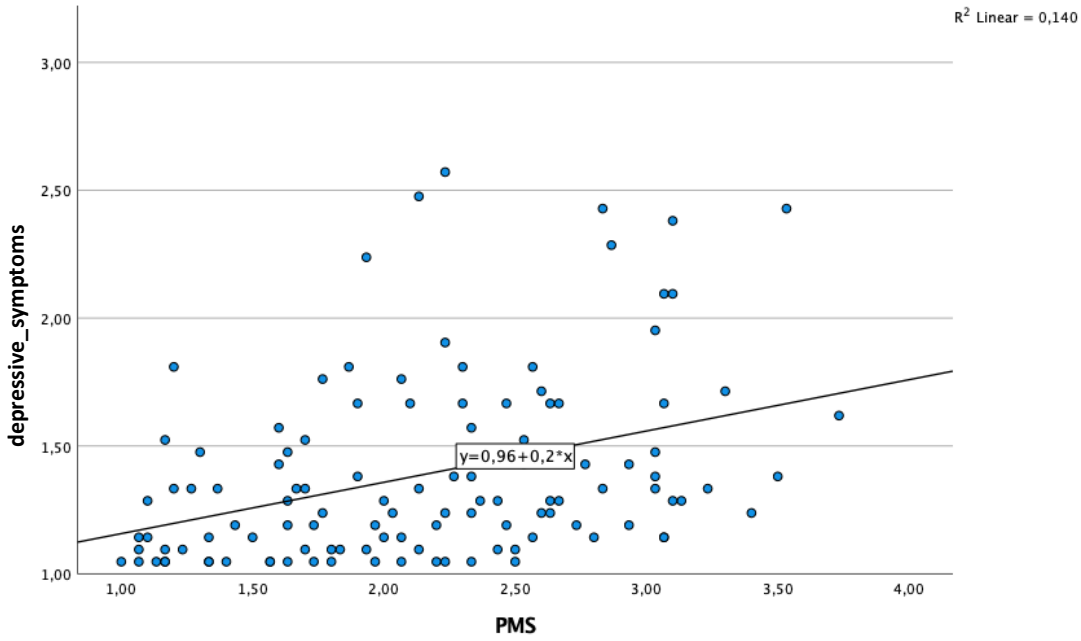
Spearman Correlation of TEWL and PMS

	Correlation Coefficient	Sig. (2-tailed)	N
TEWL	1.000		116
PMS	0.085	.365	116

PMS and depressive symptoms

The third hypothesis examined the relationship between PMS and depressive symptoms. The analysis revealed a significant and moderate correlation ($r = 0.415$, $p < 0.001$ [$df = 114$]), indicating that participants reporting higher PMS scores also reported higher levels of depressive symptoms, and vice versa. The regression line explains 14 % of variance in the data, suggesting a medium to moderate effect size for the observed correlation.

Figure 7



Note. Scatterplot of depressive symptoms and PMS

Table 6

Spearman Correlation of PMS and depressive symptoms

	Correlation Coefficient	Sig. (2-tailed)	N
depressive symptoms	1.000		116
PMS	0.415**	<.001	116

Discussion

This master's thesis was conducted to explore potential associations between the premenstrual syndrome, depressive symptoms, and skin barrier integrity. The overarching aim was to investigate whether these variables were correlated. The first and second hypotheses posited that persistent dermatological health issues throughout the menstrual cycle, characterized by elevated transepidermal water loss, could be linked to PMS and depressive symptoms, respectively. The third hypothesis suggested a correlation between depressive symptoms and PMS, implying a possible co-occurrence of these conditions. By examining these hypotheses, the study aimed to contribute to our understanding of the complex relationships between psychological well-being, menstrual cycle-related symptoms, and dermatological health. Insights gained from this research could potentially lead to improved interventions and therapeutic strategies addressing the moredimensional needs of individuals affected by PMS, depression, and skin issues.

Summary of the results

In summary, the reported levels of transepidermal water loss showed no significant correlation with either depressive symptoms or PMS, leading to the rejection of hypotheses 1 and 2. However, a significant association was observed between depressive symptoms and PMS, indicating a positive correlation. Therefore, hypothesis 3 could be supported and accepted.

Contextualizing the findings within the existing literature

Depressive symptoms and transepidermal water loss

The data from this thesis did not reveal any association between depressive symptoms and transepidermal water loss, suggesting a lack of direct connection between skin barrier integrity and depression. These findings contradict the assumption that a correlation could be plausible, given that earlier studies have identified effects with closely related constructs. While previous research has not specifically explored this correlation, both depression and skin health have been independently linked with inflammation, raising the possibility of a potential link between the two. Additionally, alternative links between psychological factors and skin barrier disruption have been documented. For instance, stress and sleep deprivation

have been associated with skin barrier dysfunction, suggesting that psychological factors may indeed influence skin integrity. Past studies have found that psychological stress, sleep deprivation and anxiety delay barrier function recovery and increase certain cytokines (Lyu et al., 2023; Altemus et al., 2001). Stress elevates an enzyme, 11ß-HSD1, worsening barrier function; treating stress with SSRIs improves it (Choe et al., 2018). Based on these findings, one might hypothesize that not only stress and sleep deprivation, as psychological factors, but also depressive symptoms could correlate with impaired skin barrier function.

Moreover, previous research has shown that skin disorders correlate with depression. Teichgräber et al. (2021) found associations between depression and atopic dermatitis, nail disorders, and hair loss, while Mavrogiorgou et al. (2020) reported common dermatological comorbidities in psychiatric patients. Lastly, depression is linked to inflammation, which affects skin barrier integrity. Inflammatory processes play a significant role in depression and suicidal behavior (Serafini et al., 2024; Brundin et al., 2019). Strugar et al. (2019) explain how inflammation compromises the skin barrier, leading to various skin issues. Moreover, Type 2 inflammation exacerbates skin barrier disruption, as shown in studies by Beck et al. (2022) and Zhu (2023). Given the established co-occurrence of depression and skin disorders, and the role of inflammation in the pathophysiology of depression, which also correlates with impaired skin barrier function, it is plausible to suggest that depressive symptoms and lower TEWL values could be correlated.

In conclusion, while this thesis did not establish a direct connection between depressive symptoms and skin barrier integrity, it is known that there are indeed connections between psychological factors, inflammation, and skin health. Further research is needed to fully understand these relationships.

PMS and transepidermal water loss

Once again, despite assumptions drawn from previous research findings, the current investigation did not identify a significant connection between transepidermal water loss and the premenstrual syndrome within the study sample. Previous research, while not directly investigating the connection between PMS and skin barrier integrity, has independently linked both PMS and skin health with inflammation. For example, studies by Bertone-Johnson (2016) and Puder et al. (2006) found correlations between PMS symptoms and inflammation markers like hs-CRP. Moreover, it has been found that inflammation can lead to an impaired skin barrier (Strugar et al., 2019). These findings collectively suggest a possible

correlation between PMS and skin disorders, with inflammation potentially serving as the 'bridge' between the two.

Itsekson's studies (2004, 2019) investigated hypersensitivity reactions to sex hormones in PMS patients with skin diseases. They found correlations between hormonal fluctuations, skin reactions, and PMS severity. Desensitization led to symptom improvement, indicating a potential connection between hormonal fluctuations, skin hypersensitivity, and PMS symptoms. The observation that desensitization to sex hormones, used to alleviate premenstrual symptoms, also leads to improvements in skin disorders, implies a potential association between skin disorders and premenstrual symptoms.
However, the thesis couldn't support this hypothesis 2, since no correlation between PMS and transepidermal water loss could be found. Again, further research is needed to explore the complex interactions between PMS, inflammation, skin health, and hormonal fluctuations.

Depressive symptoms and PMS

For hypothesis 3, a significant moderate association between premenstrual syndrome (PMS) and depressive symptoms was discovered. This suggests that individuals with higher levels of depressive symptoms also report higher levels of PMS symptoms, and conversely, individuals with higher levels of PMS symptoms also report higher levels of depressive mood. This finding substantiates the hypothesis proposing that depressive mood and PMS are correlated and tend to co-occur. With a sample size of 116 for both variables, the analysis is robust. Previous research supports the notion that depression and PMS can co-occur, suggesting a bidirectional relationship between the two. It's conceivable that women who experience PMS are at a higher risk of developing depression, or conversely, that depression predisposes individuals to experiencing PMS. This aligns with previous research indicating a bidirectional relationship between depression and PMS.

Several studies supported this notion. Yonkers, Pearlstein, and Rosenheck (2003) found comorbid Major Depressive Disorder (MDD) in women with premenstrual symptoms, with some reporting suicidal thoughts. Strine, Chapman, and Ahluwalia (2005) observed that women with menstrual-related symptoms were more prone to depression and reported feelings of sadness, nervousness, and hopelessness. Hurt et al. (1992) noted a higher risk of late luteal phase dysphoric disorder (LLPDD) in women with a history of psychological disorders, although the risk increase was relatively small. Furthermore, Forrester-Knaus et al.

(2011) found a higher prevalence of major depression in women with severe PMS, and those experiencing both conditions were significantly impaired.

Despite conflicting findings about shared etiology, it's evident that PMS and depression share similar symptoms, complicating diagnosis, and differentiation. Some argue that PMDD should be seen as a variant of depression, given the similarities in symptoms. The preferred treatment with serotonin reuptake inhibitors (SRIs) supports this notion.
In conclusion, hypothesis 3 is supported by the thesis findings, highlighting the connection between PMS and depressive symptoms. This understanding could lead to more integrated intervention approaches for individuals experiencing both conditions.

Limitations

All these interpretations should be considered with great caution because of some limitations that could potentially influence the results and distort the interpretation. Those limitations involve the study design and problems regarding generalizability and validity.

Study design

Firstly, the sample selection criteria and the study procedure were tailored to the specific focus of the study, which centered on investigating the recovery of the skin barrier over time in response to music and stress. Consequently, the study design did not prioritize the assessment of depression and the premenstrual syndrome as primary variables of interest, nor did it encompass a comprehensive evaluation of general skin health, such as baseline TEWL-values. The absence of participants diagnosed with depression or skin disorders, coupled with a retrospective assessment of PMS, could have impacted the study's ability to draw definitive conclusions regarding these variables. These limitations are discussed in greater detail in the following sections.

A critical limitation arises from the exclusion of participants with skin disorders, allergies, or irritations, resulting in a sample comprised solely of individuals with ostensibly "healthy skin." This restriction likely minimized variability in TEWL-values, thereby reducing the ability to detect significant effects related to skin barrier integrity and its associations with depression and PMS. Moreover, individuals with skin disorders or allergies may experience unique interactions between their skin health, mental health, and menstrual symptoms. Their exclusion from the study may have overlooked important insights into how

these factors intersect and influence one another. Another point for consideration is whether it would have been more advantageous to focus not only on the baseline values of transepidermal water loss, but also on the skin's recovery over time. A well-functioning skin recovery may serve as a more reliable indicator of dermatological health. However, it is important to acknowledge the many influences that can affect this recovery process. For instance, as part of the music and stress study, it is also investigated whether listening to music reduces stress and therefore has an impact on skin barrier recovery. Therefore, to minimize confounding variables, it would be advisable to only analyze the data of one single condition of the project (e.g., music, audiobook, or silence). In this context, it might be preferable not to choose the music condition, given the possibility that individuals who are musically inclined and have a deep appreciation for music may tend to experience faster skin healing compared to those who do not share such musical preferences. Nevertheless, both the audiobook and silent conditions also include external factors that could influence skin recovery. Conversely, the decision to focus on baseline values may be justified. Despite excluding participants with allergies, skin disorders, and irritations, heightened TEWL measurements could potentially signal underlying abnormalities, predisposing individuals to future skin issues rather than indicating present disorders.

Furthermore, the measurement of transepidermal water loss has inherent limitations. TEWL is a sensitive measure influenced by various factors, including environmental conditions such as humidity and airflow. Temperature may also affect the measurement by influencing sweating and room humidity, but it does not necessarily impact the validity of the measurement itself. Additionally, TEWL varies across different anatomical sites and is influenced by physiological factors such as sweat gland activity, skin temperature, and corneocyte properties (Peer, Burli and Maibach, 2022). Existing literature also highlights inconsistencies in TEWL measurements, particularly regarding its relationship with skin of color and aging (Kottner, Lichterfeld and. Blume-Peytavi, 2013). These methodological challenges underscore the need for cautious interpretation of TEWL data and consideration of potential confounding variables.

Moreover, the exclusion of participants with diagnosed mental disorders, while necessary for controlling confounding variables in the context of stress and music interventions, may have limited the generalizability of findings regarding depression. Including participants with depression could have provided valuable insights into the relationship between these variables, particularly considering the potential transient nature of depressive symptoms and its distinction from clinical depression. By using "depressive

symptoms" as a proxy for depression, the study may not fully capture the spectrum and severity of depressive symptoms experienced by individuals with clinical depression. This limitation could affect the accuracy of assessing the relationship between depressive symptoms and other variables, such as PMS and skin barrier integrity.

Additionally, the retrospective assessment of the premenstrual syndrome may introduce several biases, complicating the interpretation of reported symptoms. One significant concern is recall bias, wherein participants may inaccurately remember or exaggerate their symptoms when asked to recall them retrospectively. This bias can stem from the subjective nature of symptom perception and the passage of time between experiencing the symptoms and recalling them. Participants may unintentionally emphasize or downplay certain symptoms based on their current mood or perceptions of PMS symptoms.

Moreover, mood-congruent memory bias may influence recollection, wherein participants' current emotional state affects how they remember past experiences. For instance, individuals experiencing depressive symptoms at the time of recall may tend to recall PMS symptoms more negatively or vividly than they actually experienced them. This bias can skew the reported association between PMS symptoms and depressive mood, potentially leading to misleading conclusions about the relationship between the two variables.

Considering these biases, it would have been preferable to administer the PMS questionnaire during participants' premenstrual phase to obtain the most accurate results. However, it's worth noting that the correlation could potentially have been stronger if the PMS values had not been assessed retrospectively and if participants with diagnosed depression had not been excluded from the analysis.

Generalizability

The study's findings may lack generalizability to broader populations due to several factors inherent in the sample selection and study design.

Firstly, the exclusion of individuals with skin disorders, allergies, or irritations may have limited the representation of diverse skin health experiences within the sample. Individuals with skin conditions may experience unique interactions between their skin health, mental health, and menstrual symptoms, potentially impacting the observed associations between skin barrier integrity, depression, and PMS. By excluding this subgroup,

the study may not accurately reflect the diversity of experiences related to these variables in the general population.

Furthermore, the exclusion of participants with diagnosed depression may have restricted the applicability of the findings to individuals with clinical depression. Depression represents a significant portion of the population, and its exclusion from the study may result in findings that are not representative of the experiences of those with depression. This limitation reduces the study's relevance and applicability to clinical settings, where understanding the relationship between depression, PMS, and skin barrier integrity is crucial for informing interventions and treatment strategies. Moreover, the specific sample used for the study may limit its generalizability to the broader population. The inclusion criteria, such as having a regular menstrual cycle and being completely healthy, resulted in a homogenous sample that may not reflect the diversity of the population. Additionally, the predominance of participants with a certain level of education further limits the generalizability of the findings to populations with varying educational backgrounds.

Implications and suggestions for future research

The findings of this master's thesis have significant implications for both research and clinical practice in the fields of dermatology, psychology, and women's health. Despite the lack of direct associations between transepidermal water loss and the premenstrual syndrome, as well as TEWL and depressive symptoms, the observed correlation between depressive symptoms and PMS underscores the interconnectedness of psychological well-being and menstrual health. These implications pave the way for several key considerations:

Firstly, the absence of a significant relationship between TEWL and depressive symptoms suggests the need for a better approach to understanding the impact of mental health on skin barrier integrity. While previous literature has hinted at potential connections between inflammation, stress, and skin health, the current study's findings emphasize the complexity of these interactions. Future research should explore alternative markers of dermatological health beyond TEWL, such as skin hydration and elasticity, to improve the examination of skin barrier function in the context of mental health and well-being. Moreover, biomarkers like Beta-endorphin and IgE, measured via radioimmunoassay, have been identified as useful indicators for assessing itch intensity and disease severity in patients with atopic dermatitis (AD) (Lee, 2006). These biomarkers offer valuable insights into patient

conditions and treatment responses, providing a more comprehensive approach to studying skin health.

Additionally, Tang et al. (2023) revealed barriers to mental health care utilization among patients with skin diseases, highlighting factors such as lack of awareness about available services, concerns about treatment side effects, and confusion regarding mental health issues. This underscores the underutilization of mental health services in this population and underscores the importance of addressing knowledge gaps and concerns to improve access to mental health care for individuals with skin conditions. Further research in this area is warranted to develop interventions that effectively address the mental health needs of patients with skin diseases.

The lack of a significant relationship between TEWL and PMS highlights the necessity for further investigation into the effects of menstrual cycle-related symptoms on skin barrier integrity. Research should explore not only transient skin conditions like Atopic Dermatoses but also the potential for a heightened risk of overall skin health issues in individuals experiencing severe premenstrual symptoms. Enhancing integrated approaches to care for both skin disorder patients and those with PMS is imperative in addressing their unique needs and improving overall well-being.

Furthermore, the significant correlation between depressive symptoms and PMS underscores the importance of addressing the moredimensional needs of individuals experiencing menstrual cycle-related symptoms. By recognizing the co-occurrence of these conditions, healthcare providers can implement integrated interventions that target both psychological well-being and menstrual health. Exploring the neurobiological etiology of both disorders could reveal their potential similarities and the reasons for their co-occurrence. Additionally, investigating potential directional effects, such as whether PMS could be caused by depression or vice versa, could provide valuable insights. Conducting exploratory analyses in this regard could bring useful findings for understanding the complex interplay between depressive mood and PMS, ultimately informing more targeted interventions and treatment approaches.

Although our study did not detect any impact on transepidermal water loss, future research could investigate the treatment of skin conditions that have significant psychophysiological aspects. Techniques such as biofeedback, cognitive-behavioral methods, hypnosis, meditation, or progressive relaxation may complement traditional dermatological treatments by addressing underlying psychological factors contributing to skin issues (Shenefelt, 2010). Additionally, Jafferany (2007) proposes stress management, relaxation

techniques, benzodiazepines, and selective serotonin reuptake inhibitors (SSRIs) for skin conditions that occur with psychological disorders. Moreover, treating primary psychiatric disorders that negatively influence skin conditions can lead to improvements in dermatological health. Conversely, addressing secondary psychiatric disorders triggered by skin conditions could be important for moredimensional management.

Furthermore, the limitations of the study, including sample selection criteria and methodological considerations, could be improved in future research. Future studies could include more diverse samples that encompass a wider range of skin health experiences, menstrual symptoms, and psychological profiles.

Conclusion

In conclusion, this thesis investigated associations among premenstrual syndrome (PMS), depressive symptoms, and skin barrier integrity. While no significant correlations were detected between transepidermal water loss and either depressive symptoms or PMS, a notable positive correlation could be found between depressive symptoms and PMS. These findings underscore the interplay between psychological well-being and menstrual health, emphasizing the necessity of comprehensive approaches to address the multifaceted needs of individuals affected by these conditions. The absence of direct associations between TEWL and PMS or depressive symptoms highlights the complexity of understanding the impact of menstrual cycle-related symptoms on skin health. Future research should explore alternative markers of dermatological health beyond TEWL to gain a better understanding of skin barrier function in the context of menstrual health. Similarly, the lack of a direct correlation between TEWL and depressive symptoms underscores the need for further investigation into the mechanisms linking psychological distress and skin barrier integrity. Subsequent studies should investigate potential pathways through which psychological factors influence skin health, such as neuroendocrine regulation, immune function, and behavioral patterns. Overall, this thesis contributes to advancing our insights into the intricate relationships between psychological well-being, menstrual cycle-related symptoms, and dermatological health, suggesting the development of more effective interventions and treatment strategies.

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Zhu, Jia1; Wang, Yong-Fang1; Song, Sha-Sha1; Wu, Li-Li1; Chen, Yi1; Li, Xin-Yu1,*; Ju, Mei2,*. Alleviating Skin Barrier Disruption, Skin Inflammation, and Pruritus: A Moisturizing Spray Containing β-Glucan and Panthenol. *International Journal of Dermatology and Venereology*, *6*(1):p 1-8, March 2023. https://doi.org/10.1097/JD9.0000000000000248

Abstract German

Diese Masterarbeit untersucht die Zusammenhänge zwischen depressiven Symptomen, dem prämenstruellem Syndrom (PMS) und dem transepidermalen Wasserverlust (TEWL) als Indikator für die Stabilität der Hautbarriere. Vorangegangene Forschung deutet auf komplexe Korrelationen zwischen diesen Variablen hin, wobei PMS oft mit Depressionen in Verbindung gebracht wird, was auf gemeinsame ätiologische Faktoren hindeuten könnte. Außerdem wurden sowohl PMS als auch Depressionen mit verschiedenen Hauterkrankungen und entzündlichen Prozessen in Verbindung gebracht, die die Hautbarriere beeinträchtigen können. Für diese Masterarbeit wurden die Daten von 116 Frauen analysiert, die an einer Studie im Rahmen eines größeren experimentellen Projekts zu Stress, Musikhören und Wundheilung teilgenommen haben. Depressive Symptome und PMS wurden mithilfe von Online-Fragebögen erhoben, während die Stabilität der Hautbarriere durch den transepidermalen Wasserverlust gemessen wurde. Entgegen den ursprünglichen Hypothesen konnten die Ergebnisse keine signifikanten Zusammenhänge zwischen depressiver Stimmung oder PMS und TEWL zeigen. Dennoch wurde eine signifikante moderate positive Korrelation zwischen PMS und depressiven Symptomen beobachtet. Diese Ergebnisse deuten darauf hin, dass zwischen dem prämenstruellen Syndrom und depressiven Symptomen eine signifikante Korrelation besteht, während die Beziehung zu der Integrität der Hautbarriere, abgebildet durch den transepidermalen Wasserverlust, weiterhin unbewiesen bleibt.

Stichwörter: *Prämenstruelles Syndrom, Depression, Hautbarriere, transepidermaler Wasserverlust, ganzheitliche Behandlung, Psychodermatologie*

Figures

Tables

This master thesis was carried out within the overarching research project "Muskiba: Effects of music listening on stress and skin barrier recovery" led by Jasminka Majdandžić. Therefore, the contents of this thesis may incidentally show overlap (in terms of e.g. appendices) with theses by other students co-supervised by Jasminka Majdandžić.