Premenstrual Dysphoric Disorder – Review of Actual Findings about Mental Disorders Related to Menstrual Cycle and Possibilities of their Therapy

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Abstract: It is known that mood disorders in women explicitly relates to estrogen production. Except for these findings phenomenon as Premenstrual Syndrome and Premenstrual Dysphoric Disorder, directly connected to menstrual cycle in women, is widely discussed. Premenstrual dysphoric disorder (PMDD) is a set of subjectively unpleasant mental and somatic symptoms. It appears in luteal phase of ovarian cycle. During menstruation it remits and disappears up to one week from its termination. DSM IV classified PMDD into the category of "Other specific depressive disorders" and further revision DSM IV-TR classifies PMDD as a separate strictly defined psychiatric diagnosis. The International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) does not include any specific category as PMDD or similar. The closest category F38.8 does not represent the core of the phenomenon because it relates only to general depressive symptomatology and does not give specific diagnostic criteria to menstrual cycle related mood disorders (Grady-Weliky, 2003). In the presented article, possible effectivity of PMDD treatment with the focus to antidepressants of SSRI type (Serotonin selective reuptake inhibitors) is discussed. In spite of interesting and significant findings, the treatment of PMDD and accordingly PMS is above all multidisciplinary question and it must be treated like that.

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Introduction

Reproductive hormones show relation to the changes of moods, anxiety, intrapsychic tension and emotional lability. The changes of ovarian hormones may influence accumulation of serotonin in the brain and its receptor availability and further they are connected to metabolism of other neurotransmitters. Another findings show that gonadal steroids significantly influence central nervous system. Menstrual cycle is accompanied with substantial changes of hormonal levels (Campagne and Campagne, 2007; Cunningham et al., 2009). From the beginning of the cycle to the ovulation, the levels of estrogens increase up to ten times. Conversely in the second half of the cycle the levels of progesterone raise up to thirty times above the level at the beginning of the cycle (Ford et al., 2009). Before the menstruation itself the levels of both steroids are decreasing. The steroids are bound to hypophyseal gonadotropins, hypothalamic gonadoliberin, which are accompanied by the changes of dopamine, endorphin and serotonin system. During the menstrual cycle emotions may change (in the second half, depressive moods and irritability is culminating), sexual behaviour, cognitive functions and sensory perception as well (Johnson, 1987; Rubinow et al., 1988; Reame et al., 1992; Pearlstein, 1995; Ford et al., 2009). One of the most well-studied mood disorders, with respect to the influence of ovarian steroids on mood, is PMDD.

The term of "premenstrual syndrome" (PMS) is recommend for use in similar difficulties in women who does not meet all PMDD criteria (Steiner and Wilkins, 1996; Steiner et al., 1999; Steiner and Pearlstein, 2000) and it is more like the subject of gynaecology while the diagnosis of PMDD should be treated by psychiatrists (Raboch et al., 2001).

Clinical picture of Premenstrual Syndrome (PMS)

PMS appears in the period of several days up to one week before menstruation. Among symptoms related to mood changes the most frequently observed is its decline, depression, dysphoria, anxiety, affective lability, persistent irritability, paranoid perception, hyperprosexia (Campagne and Campagne, 2007; Stahl, 2008; Weisz and Knaapen, 2009).

Regarding behavioural changes during PMS, among the most frequent symptoms belong increased appetite, sleep disorders (insomnia and hypersomnia), fatigue, feeling of lack of energy, decrease of interest for every day activity a relations, vacillation (Upshur, 1986; Grady-Weliky, 2003; Stahl, 2008).

Among somatic symptoms of PMS tension in breast, cephalgia, artrodynia and myalgia, paresthesia, dizziness, engagement of liquids in body with oncoides, dermatic manifestations like acne, and changes of hair quality (Grady-Weliky, 2003; Rapkin, 2008) are usually classified.

Described symptoms of PMS appear during luteal phase of the cycle and usually disappear during the first days of menstruation. The diagnostics depends on presence of specific symptoms, always in the week before menstruation with the

strict absence of the symptom in the second week of the menstruation cycle. This is necessary to be proved by prospect observation in the period of at least three month. In general population, clinically significant PMS is rare and its aetiology is not yet clear. Comparing women suffering PMS and women without these symptoms several significant differences have been found: abnormal regulation of serotonin in thrombocytes (Steiner and Pearlstein, 2000), changes in levels of melatonin and beta-endorphins, changes in cortisol response, disorders of thyreoidea function, changes of Mg concentration in erythrocytes, disorders in growth hormones release, sleep disorders (Rapkin, 2003).

During premenstrual period onset of depressive episode may be observed. Approximately 65% of women with unipolar depression experienced PMS. Women with PMS also more frequently suffer from postpartum depressions (Grady-Weliky, 2003). Women suffering from migranoid headache also frequently observe cephalgia during PMS (Hutchinson and Silberstein, 2008) and increased risk of asthmatic seizure in the course of PMS was proved in asthmatic women. Fluctuation of estrogens and progesterone levels in the period of PMS may influence insulin concentration in diabetic's women (Pearlstein, 1995; Halbreich et al., 2003).

PMS may be caused or worsened by stress, selected genetic factors, age (PMS is more often in women older than 30 years of age), number of children, alcohol abuse, excessive use of sugar and caffeine, occurrence of hypothyroidism (Grady-Weliky, 2003; Halbreich and Endicott, 2003; Stahl, 2008).

For DSM IV classification of "other specific depressive disorders", at least 5 symptoms and at least 1 symptom of mood disorders should be proved prospectively at least in the period of three month. Regarding differential diagnostics, following disorders must be excluded: disorder of thyreoidea, migraine, chronic fatigue syndrome, syndrome of irritable colon, seizure disorders, anaemia, endometriosis, psychoactive substance abuse and other mental disorders. For screening diagnostics, several rating scales can be used: Daily Rating Form (DRF), Moos Menstrual Distress Questionnaire (MDQ), Daily Rating Scale (DRS) or Calendar of Premenstrual Experiences (COPE) (Halbreich and Endicott, 2003; Cunningham et al., 2009).

PMS in contrast to PMDD is not exactly defined as a disorder. The term of "premenstrual syndrome" (PMS) is recommend for use in similar difficulties in women who do not meet all PMDD criteria (Steiner and Wilkins, 1996; Steiner et al., 1999; Steiner and Pearlstein, 2000) and it is more like the subject of gynaecology while the diagnosis of PMDD should be treated by psychiatrists (Raboch et al., 2001).

Pathophysiology of Premenstrual Dysphoric Disorder (PMDD)
Trimonoaminergic modulators (triple monoamine modulators or TMMs)
An increasing number of agents now appear to modulate the Trimonoaminergic neurotransmitter system of 5HT, NE, and DA by mechanism other than inhibition

of monoamine transporters and in a manner that may be more effective when given with a monoamine transport inhibitor rather than as a monotherapy. These therapeutic interventions range from hormones to vitamins, medical foods, ions, electrical and magnetic brain stimulation, and even psychotherapy (Stahl, 2008).

The hormone estrogen has a profound impact on mood and on the Trimonoaminergic neurotransmitter system (Table 1) and thus it can be considered as a trimonoaminergic modulator (TMM). Estrogen also modulates the activity of other neurotransmitters, including GABA and glutamate, as it will be discussed below. Many of estrogen's effects upon various neurotransmitter systems appear to be the result of estrogen binding to nuclear hormone receptors, known as estrogen receptors. Receptors for estrogen may also exit in neuronal cell membranes, but

Table 1 – DSM-IV-TR research criteria for premenstrual dysphoric disorder (Schmidt and Rubinow, 2006)

DSM-IV-TR research criteria for premenstrual dysphoric disorder

A. In most menstrual cycles during the past year, five (or more) of the following symptoms were present for most of the time during the last week of the luteal phase, began to remit within a few days after the onset of the follicular phase, and were absent in the week postmenses, with at least one of the symptoms being either (1), (2), (3), or (4):
  1. markedly depressed mood, feelings of hopelessness, or self-deprecating thoughts
  2. marked anxiety, tension, feelings of being "keyed up," or "on edge"
  3. marked affective lability (e.g. feeling suddenly sad or tearful or increased sensitivity to rejection)
  4. persistent and marked anger or irritability or increased interpersonal conflicts
  5. decreased interest in usual activities (e.g. work, school, friends, hobbies)
  6. subjective sense of difficulty in concentrating
  7. lethargy, easy fatigability, or marked lack of energy
  8. marked change in appetite, overeating, or specific food cravings
  9. hypersomnia or insomnia
  10. a subjective sense of being overwhelmed or out of control
  11. other physical symptoms, such as breast tenderness or swelling, headaches, joint or muscle pain, a sensation of "bloating", weight gain

Note: In menstruating females, the luteal phase corresponds to the period between ovulation and the onset of menses, and the follicular phase begins with menses. In nonmenstruating females (e.g. those who underwent hysterectomy), the timing of luteal and follicular phases may require measurement of circulating reproductive hormones.
B. The disturbance markedly interferes with work or school or with usual social activities and relationships with others.
C. Avoidance of social activities, decreased productivity and efficiency at work or school.
D. The disturbance is not merely an exacerbation of the symptoms of another disorder, such as Major Depressive Disorder, Panic Disorder, Dysthymic Disorder, or a Personality Disorder (although it may be superimposed on any of these disorders).
E. Criteria A, B, and C must be confirmed by prospective daily ratings during at least two consecutive symptomatic cycles. (The diagnosis may be made provisionally prior to this confirmation.)

these have not been yet well characterized. However, it is well established that nuclear ligand receptors specific for estrogen are transformed into nuclear ligand-activated transcription factors when estrogen binds to them (Stahl, 2008).

Estrogen modulates gene expression by binding to nuclear hormone receptors for estrogen i.e. "estrogen receptors". Receptors for estrogen differ from tissue to tissue and they may differ between the brain regions. In addition to various subtypes of estrogen receptors, there are also nuclear hormone receptors for progesterone and androgens as well as for other steroids such as glucocorticoids and mineralocorticoids (Stahl, 2008). Unlike neurotransmitter receptors located on neuronal membranes, nuclear ligand-activated receptors for estrogen are located in the neuronal cell nucleus, so estrogen must penetrate the neuronal membrane to find its receptors, which are located near the genes that estrogen influences. These genes are called estrogen response elements. Activation of estrogen response elements by estrogen requires receptor "dimerization", when estrogen binds to them; this form of an active transcription factor capable of turning on estrogen response elements. Once estrogen receptors are activated as transcription factors, they activate gene expression in the neuron by binding to estrogen response elements in the neuron's DNA (Stahl, 2008).

Gene products that are regulated by estrogen include trophic factors such as brain-derived neurotrophic factor (BDNF) as well as neurotransmitter synthesizing and metabolizing enzymes and various neurotransmitter receptors. Dramatic evidence of estrogen's trophic properties can be observed in hypothalamic and hippocampal neurons in adult female experimental animals within days and across a single ovarian cycle (Klingler, 2008; Pearlstein et al., 2009). During the early phase of the cycle, estradiol levels rise, cause formation of dendritic spines specifically on pyramidal neurons in the hippocampus and on neurons in the ventromedial hypothalamus of female rats. Progesterone administration potentiates dendritic spine formation, so the spine density is the greatest when both estrogen and progesterone peak just after the first half of the cycle. However, once estrogen levels fall significantly and progesterone continues to raise, the presence of progesterone without estrogen triggers down regulation of these spines by the end of the menstrual cycle (Riecher-Rössler, 2009).

One hypothesis explaining mechanism of this cyclical formation and loss of dendritic spines relays on estrogen that regulates spine formation occurring when neurons are active and that reverses the process when neurons are inactive, known as "activity-dependent" dendritic spine formation. As estrogen levels rise and fall due to the ovarian cycle, estrogen can cause a corresponding cyclical rather than continuous activation of neurons in certain brain areas. The cyclical activation of these neurons is explained by the fact that estrogen exerts a cyclical inhibitory influence on GABA interneurons (Stahl, 2008; Ford et al., 2009).

When estrogen activates pyramidal neurons, these neurons release glutamate. As estrogen levels rise during the first half of the menstrual cycle, so does pyramidal

neuron activation by glutamate from other pyramidal neurons as estrogen levels fall due to the last half of the ovarian cycle, pyramidal cells lose their activation (Jones and López, 2006).

There is a cyclical formation of dendritic spines that is the consequence of these cyclical changes in estrogen levels for a single ovarian cycle. Thus, at the beginning of the cycle, estrogen levels are low, so GABA interneurons are active. When GABA interneurons are active, they inhibit pyramidal neurons. However, as estrogen levels rise due to the first half of the cycle, GABA interneurons are progressively inhibited, causing progressive disinhibition of pyramidal neurons (Jones and López, 2006; Stahl, 2008).

Disinhibited pyramidal neurons release glutamate. Glutamate then interacts at a number of glutamate receptors, including postsynaptic NMDA receptors on other pyramidal neurons. Sustained activation of NMDA receptors can trigger long-term potentiation and trophic changes in postsynaptic neurons, including the formation of dendritic spines by the end of the cycle. Once estrogen levels fall by the end of the cycle, glutamate neurons again become inactive, and activity-dependent dendritic spine formation is not maintained (Klingler, 2008).

Estrogen levels shift rather dramatically across the female life cycle in relation to various types of reproductive events (Figure 1). Such shifts are also linked to the onset or recurrence of major depressive episodes. In men, the incidence of depression rises in puberty and then is essentially constant throughout life, in spite of a slowly declining testosterone level from age twenty-five onward (Pearlstein and Steiner, 2008; Cunningham et al., 2009).

By contrast, in women, the incidence of depression in many of them mirrors their changes in estrogen across the life cycle. That is, as estrogen levels rise due to puberty, the incidence of depression skyrockets in women; then, after menopause, it falls again. Thus, women have the same frequency of depression as man before puberty and after menopause. However, due to their childbearing years, when estrogen is high and cycling, the incidence of depression in women is two to three times higher than it is in men (Stahl, 2008; Halbreich, 2009).
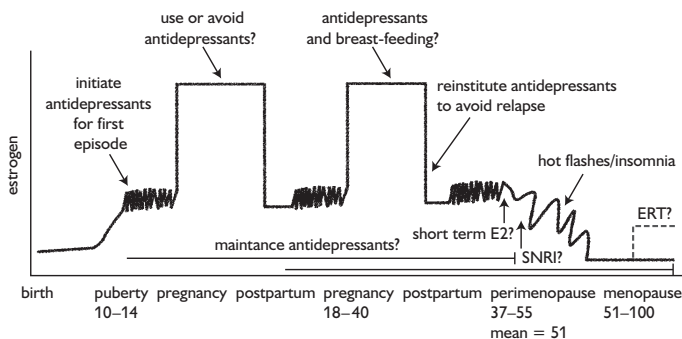

Figure 1 – Estrogen levels fluctuations during the female life (Stahl, 2008).

Premenstrual Dysphoric Disorder

As estrogen levels first begin to rise and then cycle due to puberty, first episodes of depression often begin. Unfortunately these episodes are frequently unrecognized and untreated (Grady-Weliky, 2003; Rapkin and Winer, 2009).

Throughout the childbearing years, most women experience some irritability, during the late luteal phase just prior to menstrual flow of menstrual cycles; however, if this is actually incapacitating, it may form mood disorder known as premenstrual dysphoric disorder (PMDD) or as a premenstrual syndrome (PMS) (Klingler, 2008).

Clinical picture of Premenstrual Dysphoric Disorder (PMDD)

The disorder was originally defined in the appendix of DSM IV among suggestions of next categories. In the revision of DSM in 1987 it was stated as "dysphoric disorder of late luteal phase" (Steiner and Wilkins, 1996; Schatzberg and Nemeroff, 2004). As the first, historically related term, "premenstrual tension", used by American physician R. T. Frank in 1931 (Miller et al., 1999) can be considered. Later on the term "premenstrual syndrome" can be found (Johnson, 1987; Endicott et al., 1999). Nowadays both terms can be found in medical literature. Most of the mentioned and related terms are usually used without clear conventions or rules. Despite the fact that premenstrual dysphoric disorder (PMDD) is according to the criteria of DSM-IV-TR defined quite exactly (Steiner, 2000; Chromý, 2001; Pidrman and Látalová, 2001).

PMDD is characterized by a constellation of affective and somatic symptoms that are manifested during the late luteal phase of the menstrual cycle and resolve shortly after the onset of menses (Steiner, 2000). Unlike other mood disorders, the mood disturbances associated with PMDD are cyclical and tightly linked to the menstrual cycle; hence the occurrence of symptoms ceases during pregnancy and after menopause (Stein et al., 2005). Premenstrual syndrome (PMS) is a common condition among menstruating women, with prevalence estimates ranging from 30% to more than 60% (Stein et al., 2005). PMDD is a severe form or a subgroup of PMS that affects only about 3–9% of women (Halbreich, 2009). Although symptoms may appear anytime after menarche, the average age of onset for PMDD

Table 2 – Trimonoamine modulators (TMMs) (Stahl, 2008)

Trimonoamine modulators (TMMs)
folate
L-MTHF (L-methyl-tetrahydrofolate)
estrogen
estrogen replacement therapy
thyroid hormones (T3/T4)
lithium
brain stimulation
psychotherapy

is the mid 20s, and women generally do not seek treatment until their 30s. Women commonly report that their symptoms gradually worsen with age, until the onset of the menopause (Stein et al., 2005). Although opinions about incidence are not comprehensive (Čepický, 2001, 2005), PMDD is more strictly defined (Table 2) than PMS, with the emphasis on mood and behaviour symptoms and functional impairment (Pearlstein et al., 2009). According to DSM-IV-R, at least 5 out of possible 11 symptoms are required, and at least one of them must be a core mood symptom – irritability/anger, depressed mood, anxiety, or mood swings. The symptoms must be severe enough to cause impairment in social or occupational functioning. The presence and severity of symptoms and their cyclical relation to the luteal phase must be confirmed by prospective daily ratings for at least two consecutive menstrual cycles. Prospective documentation of symptoms is a critical step in diagnosis, given that a significant number of women who seek treatment for premenstrual emotional symptoms actually have premenstrual exacerbation of another underlying psychiatric condition – usually, a depressive or an anxiety disorder (Stein et al., 2005). The luteal phase of menstrual cycle is a period of increased vulnerability in some women for the onset of a new depressive episode or for exacerbation of symptoms of an ongoing episode (Stahl, 2008). Premenstrual exacerbation of symptoms also may be seen with a variety of other psychiatric disorders and general medical conditions (e.g. anxiety disorders, eating disorders, substance abuse, migraines, asthma, seizures). The high prevalence of depressive and anxiety disorders found in women thought to have PMS or PMDD highlights the need for clinicians to be aware of this overlap in presenting complaints and hence to be better able to assess patients, clarify the diagnosis, and initiate appropriate treatment (Stein et al., 2005).

The term of "premenstrual syndrome" is recommend for using in similar difficulties in women who does not meet all PMDD criteria (Steiner and Wilkins, 1996; Steiner et al., 1999; Steiner and Pearlstein, 2000) and it is more likely the subject of gynaecology while the diagnosis of PMDD should be treated by psychiatrists (Raboch et al., 2001).

Therapy of Premenstrual Dysphoric Disorder (PMDD)
Current literature recommends (Čepický et al., 2005, 2006) several steps for effective and interdisciplinary therapy of PMDD. Among other the mostly recommended are:

1. Change of life style is usually recommendation of the first choice (Grady-Weliky, 2003; Rapkin, 2003; Ballagh and Heyl, 2008; Weisz and Knaapen, 2009). The use of non-pharmacological procedures as dietary arrangements (reduction of caffeine, salt, alcohol, smoking), regular physical activity, relaxation, hypnosis and psychotherapy are recommended (Rapkin, 2003; Stahl, 2008; Daley, 2009; Lustyk et al., 2009).

2. Pharmacotherapy – hormonal anticonceptive – above all substances containing drospirenon (progestin with anti-mineralocorticoid effect), in the case of non usage or failure of this approach, symptomatic treatment should be taken – above all inhibitors of prostaglandins in pain symptoms, bromocriptin or vitamin E in mastopathy, anti oedematous agents as spironolacton in tumescence (Cunningham et al., 2009; Georgantopoulou and Field, 2009).

3. Alternative medication and supplemental treatment with diuretics, inhibitors of prolactin, beta-blockers and vitamin B6 in dosage of 100 mg daily, calcium, effective may be also the cod-liver oil (Freeman et al., 1996; Ghanbari et al., 2009).

4. Hormonal treatment is considered as well. Already mentioned use of anticonceptive drugs for ovulation inhibition can be connected to impairment of symptoms in some women. Use of androgenous drugs can cause higher incidence of depressive syndrome. Use of estradiol implants and agonists of gonadotropins releasing hormones is despite possible improvement in some women connected to the risk of masculinisation and weight increase. An extreme form of PMS and PMDD treatment is the blockade of menstrual cycle with super agonists of gonadoliberin (Stahl, 2008; Weisz and Knaapen, 2009).

5. Use of antidepressants has been confirmed as the treatment of the first choice, especially SSRI and SNRI antidepressants, namely sertralin, fluoxetin, paroxetin (Pearlstein et al., 2009). It was found that these antidepressants are effective in short term use – 3–7 days in premenstrual period. Continual use may have higher effectivity (Pearlstein and Steiner, 2008; Pearlstein et al., 2009; Schneider and Popik, 2009). SSRI antidepressants are recommended for their fast effect in the treatment so far psychotherapy may have long term effect (Rapkin, 2003).

6. The role of GABA in PMS and PMDD has not been defined clearly, although some women refer improvement of selected symptoms, as a reduction of tension, anxiety, irritability and hostility after the use of GABA agonist alprazolam, which is considered to be the only adjacent treatment (Stahl, 2008).

7. For positive influence of PMS and PMDD symptomatic can be used buspiron – partial agonist of 5-HT1 (Stahl, 2008).

PMDD can be treated cyclically with oral contraceptive hormones or alternatively with antidepressants, sometimes just during the luteal phase. In some patients, this end-of-cycle worsening is in reality the unmasking of a mood disorder that is actually present during the whole cycle but significantly worsens at the end of the cycle that it becomes obvious as a phenomenon called "menstrual magnification" (Figure 1). This may be a harbinger of further worsening or may also represent state of incomplete recovery from the previous episode of depression (Upshur, 1986; Rapkin, 2003). One of the best-documented effective treatments is the elimination of menstrual cycling with leuprolide, a GnRH agonist that improves mood (Schatzberg and Nemeroff, 2004). A number of studies found that leuprolide

was highly effective in reducing symptom severity and cyclicity in PMDD patients (Schatzberg and Nemeroff, 2004), although an increased rate of depressive-like symptoms has also been reported during leuprolide treatment (Schatzberg and Nemeroff, 2004). Leuprolide also leads to hypoestrogenism, which affects both bone density and cardiovascular disease; thus, it is necessary to add both steroid hormones. In the study by Mortola et al. (1991), just the addition of a placebo, with the suggestion that it might make mood symptoms worse, caused a significant worsening in mood symptoms. However, addition of conjugated equine estrogen with or without medroxyprogesterone acetate (MPA) while patients were still on leuprolide did not lead to a relapse of depressive symptoms. Not all studies have agreed that progesterone can be added back without significant worsening of symptoms. Schmidt and colleagues studied a group of women with PMDD whose symptoms were significantly improved by leuprolide, as well as a control group with no previous mood symptoms who were also taking leuprolide (Schatzberg and Nemeroff, 2004). They demonstrated a return of symptoms following administration of estradiol or progesterone but not with placebo. In the control women, none of the hormone replacements altered mood (Schmidt and Rubinow, 2006). Finally, progesterone itself has been used for the treatment of PMDD despite its documented lack of effectiveness (Schatzberg and Nemeroff, 2004).

Discussion and Conclusion
In the previous work (Žukov et al., 2009) the authors studied incidence of PMDD in the group of female patients (n=43) treated for bipolar affective disorder for six month with SSRI antidepressants. Incidence of PMDD was in the observed group substantially smaller than in the non clinical population untreated with SSRI antidepressants. This finding confirms the hypothesis of serotonin dysregulation in PMDD. Occurrence of PMDD in the observed group treated for bipolar affective disorder was not different from the control group. All women in the observed group have been medicated with at least maintenance dosage of SSRI antidepressants and there were 5 more serious relapses of the basic psychiatric disorder (bipolar affective disorder) connected with hospitalization. During the treatment with SSRI antidepressants there was significant decrease of symptoms typical for PMDD. Despite these findings, the treatment of PMDD with SSRI antidepressants is an efficacious psychiatric medical solution that must always be accompanied by life style change and necessary dietary interventions (Grady-Weliky, 2003; Rapkin, 2003; Ballagh and Heyl, 2008; Stahl, 2008; Daley, 2009; Weisz and Knaapen, 2009).

In the treatment of PMDD it is necessary to stress the need for complex history information, multidisciplinary approach (gynaecology, endocrinology, psychiatry) and namely changes of the life style (i.e. dietary changes) (Ford et al., 2009; Weisz and Knaapen, 2009; Žukov et al., 2009). Further research interest should concentrate at the understanding of physiological relations and consequences of PMDD as well

as at the practical recommendations for clinical practice (Steiner and Wilkins, 1996; Čepický et al., 2005; Stein et al., 2005).

Inclusion of PMDD into DSM-IV-R has started discussion within medical society, but it also helped to start intensive research in this area (Čepický, 2001; Raboch et al., 2001).

Necessary condition for diagnosing PMS is the prospective observation of occurrence and intensity of symptoms at least in two or three menstrual cycles. The symptoms must appear in luteal phase of each cycle and it must not appear in the second week of the menstrual cycle. The diagnosis of PMDD is strictly assessed according to DSM-IV-R, while PMS is a subject of gynaecologists (Raboch and Zvolský, 2001; Čepický, 2005; Čepický et al., 2006).

In conclusion, it is necessary to emphasize, that the menstruation cycle is a physiological process, hence if the intensity of psychic symptoms does not exceed the border diagnostically designated for PMS or especially for PMDD, it is not necessary to consider any medical intervention.

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