



Arthralgias, bodily aches and pains and somatic complaints in midlife women: etiology, pathophysiology and differential diagnosis

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Somatic symptoms characterized by arthralgias, bodily aches and pains, musculoskeletal pain and joint pain have been investigated in a number of menopause and depression studies. Although depression is one of the most common causes of bodily aches and pains, and arthralgias, these same symptoms are also commonly associated with a natural menopause, surgical menopause and menopause induced by chemotherapy in breast cancer treatment. Somatic symptoms in the absence of definitive medical diagnoses result in these patients receiving various diagnoses and labels – ‘medically unexplained symptoms’, ‘worried well’, as well as various Diagnostic and Statistical Manual of Mental Disorders (4th edition) somatoform diagnoses. Osteoarthritis and joint pain increase in prevalence from premenopausal- to menopausal-aged women with hormonal change implicated in their etiology. The current research on the relationships among menopause, depression, nociceptive mechanisms, perception and pain in the distressed midlife patient is discussed. The amelioration and management of pain symptoms in the menopausal and postmenopausal woman, with or without comorbid depression, have been elusive and difficult problems for clinicians. Familiarity with the differential diagnosis, pathophysiology and evidence-based treatment for such patients is crucial to their proper care.

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Materials & methods

Studies were retrieved using both Cochrane and PubMed searches. A literature search review was performed to identify randomized controlled trials, some of which were also double-blind, evidence-based review articles and meta-analyses, as well as evidence-based guidelines relevant to the topics and symptoms reviewed. Reference lists from these articles were also examined. As is standard, studies that used methodology consistent with evidence-based medicine were used. Given the current limits of some areas of research, we also included less reliable evidence when higher levels were not available. The limitations of these studies and their application to clinical treatment are indicated.

The most common somatic symptoms attributed to menopause include thermoregulatory problems, such as hot flashes and night sweats, sleep complaints, vaginal dryness, urinary complaints, sexual dysfunction and

uterine bleeding [1]. Some somatic symptoms attributed traditionally to menopause, such as back pain, tiredness, and stiff and painful joints, have not been shown to be specifically associated with menopause [1], and Nelson

et al. (2005), in their detailed evidence-based analysis, concluded that these somatic symptoms, as well as bodily pain and poorer general health, were greater in menopausal women in some studies but not in others [1–3]. On the other hand, back pain, tiredness and arthralgias are often the predominant presenting symptoms in depression [2,4–13], especially in women [14]. The most and least common causes for bodily aches and pains in the midlife patient are presented in BOX 1, and the most and least common causes for back pain and stiff, painful joints in BOX 2. Depression and menopause are two of the more concurrent conditions associated with these complaints and the focus of this article, although other factors remain part of the differential diagnosis for these distressed midlife patients.

Somatic symptoms & menopausal status

Arthralgias, musculoskeletal pain, bodily aches & pains

Arthralgias, bodily aches and pains, back pain and diffuse musculoskeletal pain, along with other somatic symptoms of menopause, have been investigated in a number of cross-sectional and longitudinal studies. In the Australian Longitudinal Study of Women's Health (ALSWH) the bodily-pain category of the short form (SF)-36 health survey significantly increased during the perimenopausal transition when compared with premenopause [18,19]. In this study, the perimenopausal group was found to have the lowest physical functioning and physical role limitation. Ho *et al.* (2003) conducted a random telephone survey of 2145 pre-, peri- and post-menopausal women aged 44–55 years living in Hong Kong in 1996 using a 22-item somatic-complaint checklist adapted from Avis *et al.* (1993) [20,21] and modified to require only a binary yes/no response (based on studies of questionnaires in Asian cultures). With a response rate of 40.4%, the perimenopausal group reported the most complaints of backaches, headaches, joint aches and stiffness, with

joint aches and stiffness having the largest difference between the pre- and post-menopausal groups [21]. Dugan *et al.* (2006) studied the relationship between menopausal status and musculoskeletal pain in 2218 women at their third annual follow-up examination in the Study of Women's Health Across the Nation (SWAN). Using the Aches and Pains scale, the menopausal women reported increased pain symptoms compared with the premenopausal women [22], with results evaluated controlling for the effects of depression, body mass index (BMI) and smoking. The association between aches and pains and late perimenopause were diminished when depression and smoking were added to the statistical model. Owing to the cross-sectional analyses, investigators could not distinguish whether the aches and pains were a function of depression or of menopausal status.

Quality of life

Mishra *et al.* (2006) in a prospective longitudinal population-based cohort of 1525 British women followed 19 times from birth to 43 years of age and then annually from ages 47 to 54 years ('the 1946 British Birth Cohort', also known as the Medical Research Council National Survey of Health and Development), found that stress and lack of physical activity were both significantly associated with weight gain during perimenopause [23]. Decline in physical health, energy level, body weight, and work and family stress were the most significant factors resulting in decline in quality of life. Perimenopausal women were also more likely to have complaints concerning physical health than premenopausal women.

Kumari *et al.* (2005), in a prospective study of 2489 women followed through phases 3, 4, 5 and 6 of the Whitehall II study [24], used the SF-36 health survey to evaluate perceived health status and quality of life. The SF-36 is a widely used generic measure of self-reported health status, which may be administered face-to-face or by telephone, takes 10–12 min to administer and uses a score range of 0–100, a higher score implying better health. The 36 items are divided into eight scales that measure limitations in physical, social and role activities due to poor health or bodily pain, as well as frequency and intensity of feeling states, thereby surveying a range of self-perceptions regarding mental health, bodily pain, fatigue and energy level. For the perimenopausal women, greater severity of symptoms was found to be associated with a greater reported decline in quality of life [24]. A statistical association between more severe reported symptoms and a greater reported decline in physical functioning and bodily pain was also found for these perimenopausal women. Those perimenopausal women who reported vasomotor symptoms had a mean decline of 3.3 SF-36 points related to physical functioning. A statistical association between vasomotor symptoms and functional decline for all eight scales of the SF-36 – general health perceptions, physical functioning, physical role limitation, general mental health, emotional role limitation, vitality and social functioning – was found for both the peri- and post-menopausal women. Hormonal therapy (HT) did not change these reported associations between menopausal depression and decline in SF-36, nor did excluding women who had undergone hysterectomy.

Box 1. Differential diagnosis of bodily aches and pains in the middle-aged woman.

More common causes

- Depression
- Menopausal transition (varying evidence)
- High body mass index
- Fibromyalgia [15]

Less common causes

- Chronic infections (e.g., brucellosis and hepatitis C)
- Endocrine disorders (e.g., hypothyroidism and hyperparathyroidism)
- Neoplastic disease (e.g., myeloma, metastatic breast cancer and lung disease [16])

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Aromatase-inhibitor treatment in women with breast cancer

There are reports of increased complaints of arthritis, arthralgia and muscle pain in breast cancer patients whose estrogen has been greatly reduced by aromatase inhibitors [25–27]. Aromatase inhibitors markedly suppress plasma estrogen levels by blocking conversion of androgens to estrogen by means of the enzyme aromatase [25–27]. The current use of aromatase inhibitors does not include premenopausal women but are used after menopause or after a hypothalamic amenorrhea induced through gonadotropic-releasing hormone (GnRH) analogues, synthetic peptide drugs developed to mimic hypothalamic GnRH. Women treated with aromatase inhibitors have reported more arthralgias than women on placebo or tamoxifen, a selective estrogen receptor modulator (SERM) that acts as an estrogen antagonist in women of reproductive age and causes a menopause-like status [25]. Both tamoxifen and anastrozole, an aromatase inhibitor, have been observed to cause musculoskeletal disorders [27]. In one report, anastrozole induced arthralgias at a rate of 27.8% and tamoxifen at a rate of 21.3% [26]. In the National Cancer Institute of Canada clinical trials, letrozole, another aromatase inhibitor, was compared with placebo in a study performed after 5 years of treatment with tamoxifen [28]. The placebo arm of this study thus represented an iatrogenically induced menopause-like state [28]. Joint pain was 21.3% versus 16.6%, and muscle ache 15% versus 12%. Bodily pain and vitality were greater in the aromatase-inhibitor group (letrozole), as well as in the placebo group. Patients treated with leuprolide, a GnRH analogue that suppresses ovarian function and is used to treat leiomyomas and other conditions, developed arthralgias and myalgias within 3–7 weeks of treatment and vaginal dryness within 2 weeks [29]. It is unclear whether or not the myalgias, arthralgias and pain reported in natural menopause, menopause due to an aromatase inhibitor and menopause due to agents, such as tamoxifen and leuprolide, are all produced by similar mechanisms [25]. Felson and Cummings (2005) have proposed two hormonal mechanisms, diffuse pain due to estrogen depletion is resolved by estrogen replacement or that nociceptive sensitivity is due to rapid loss of estrogen [25].

Nociceptive & pain modulatory mechanisms: evidence for the role of estrogen

There is considerable evidence from animal studies suggesting roles for estradiol, progesterone and luteinizing hormone in nociceptive and pain-modulating mechanisms [30–36]. However, in humans, studies of changes in a woman’s tolerance of and sensitivity to painful stimuli due to hormonal changes have mixed results. An early review concluded that experimental pain intensity was highest during times of increasing estradiol and progesterone [37]. Another review concluded that women tolerated more pain in response to a variety of experimental pain conditions (e.g., pressure, temperature and ischemic stimuli) during times of lowest estradiol and progesterone levels [38]. De Leeuw and colleagues (2006) studied nine healthy, pain-free women (mean age: 26 years) using whole-brain functional

Box 2. Differential diagnosis of back pain, stiff and painful joints in the middle-aged woman.

Common causes

- Depression
- Menopause (variable evidence – see text)
- Chemical menopause secondary to tamoxifen or aromatase inhibitors
- Back sprain/strain or disc problem, sciatica

Less common causes

- Osteoarthritis
- Spinal stenosis
- Rheumatoid arthritis
- Aortic aneurysm
- Duodenal ulcer
- Kidney stones or pyelonephritis
- Pancreatitis
- Hip osteoarthritis [17]

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(f)MRIs during periods with high and low estrogen levels, finding no significant difference in pain thresholds [39]. While the magnitude of activation of the anterior part of the anterior cingulate, cerebellum and precuneus showed significant differences, these differences may have failed to reach statistical significance because of the small sample size.

Both Fillingim *et al.* (2000) and Sherman *et al.* (2006), in their evidence-based analyses, felt that methodological differences in the human experimental pain literature have prevented scientific replication of earlier findings and thus any definitive understanding of the effects of female reproductive hormones on experimental pain [40,41]. Sherman and Leresche (2006) suggest that future research studies adopt a standardized approach to defining and naming critical times in the menstrual cycle, direct measurement of hormones daily during the study period, excluding women using exogenous hormones and standardization of the pain stimulus [41].

Association of osteoarthritis onset & estrogen decline

There are reports linking the loss of estrogen at the peak of menopause with the development of osteoarthritis (OA), an illness with a higher prevalence in women that usually develops from the age of 40 to 50 years [42–44]. Nadkar *et al.* (1999) reported on case series of 100 consecutive patients, 50 female and 50 male, for OA and the relationship of this to menopause in women. The onset of OA occurred before the age of 50 years in 58% of the women but in only 20% of the men [45]. Sowers *et al.* (2000) reported the prevalence of knee OA was 15% in

women aged 29–52 years. Sowers *et al.* (2006) also found that both lower endogenous estradiol levels and lower levels of one of its metabolites, 2-hydroxyestrone (OHE), were associated with incident OA of the knee, even after adjusting for age, injury and BMI. Several clinical studies suggest a protective effect of hormone therapy in the presentation of OA [46,47].

Mechanisms for arthralgias with estrogen decline

Several mechanisms have been proposed to link estrogen and menopause status to pain and arthralgias. These include estrogen impact on arachidonic metabolism, the impact through altered receptor binding and the impact on structural proteins, such as collagens [48]. Sowers (2006), Weidler (2004) and Castagnetta (2003) have suggested that alterations in estrogen metabolism may play a role in pain and inflammation rather than in the onset and progression of OA, systemic lupus erythematosus and rheumatoid arthritis specifically [48–50]; 2-hydroxyestrone (2-OHE) is thought to modulate prostaglandins [51]. Weidler *et al.* (2004) have reported lower 2-OHE concentrations in patients with systemic lupus erythematosus and rheumatoid arthritis [49]. The study of estrone metabolites, particularly the ratio of 2-OHE to 16 α -hydroxyestrone (16-OHE) is an important area of research with possible implications for breast cancer risk [50,52] and chronic inflammatory diseases [49]. The impact of diet [49,50] and lifestyle [53] on these estrone metabolites continues to be investigated with much interest.

Dietrich and colleagues (2006) found that estrogen receptor (ER)- β , as opposed to ER α , is expressed in normal human synovia and may therefore play a role in synovial membrane function [54]. The implications for this research on arthralgias, as well as the effect of different estrogen receptor polymorphisms, has yet to be explored fully.

Type II collagen is a primary structural protein of articular cartilage matrixes. Oestergaard *et al.* (2006), in their rat study of the effects of estrogen therapy on type II collagen (CII) turnover, suggested that estrogen counters the acceleration of CII degradation, as well as related structural changes [55]. Whether or not estrogen protects a woman's joints from CII degradation and whether such benefits are affected by the timing of estrogen administration relative to menopause onset, are being actively investigated.

Depressive illness: evidence for the development of bodily aches/pains & somatic symptoms in the midlife woman

When depression is undiagnosed or inadequately managed, it often presents with somatic and neurobehavioral complaints similar to those seen in menopause [25]. Independent of stage of life, including menopause, affective disorders may be characterized by physical as well as psychological symptoms [37,56]. Furthermore, generalized anxiety disorder also often presents with somatic symptoms troubling to the patient.

The prevalence of somatic complaints as the presenting symptoms of depression and anxiety in women varies from culture to culture and has been found to be higher in some

minorities and in new immigrants [57]. In some cultures, the Western concept of depression may in fact not exist and if a woman from such a culture is asked if she is depressed, she may deny it while at the same time reporting multiple somatic complaints [14].

Corruble *et al.* (2004) found that up to 76% of depressed patients complained of backache and chest pain [4], and distressing physical complaints have been found to be four-times more common in depressed than nondepressed patients [5]. Studies in the primary-care setting suggest that a similar presentation rate might be found in gynecological or other nonpsychiatric outpatient settings [6,7]. A correlation between number of physical symptoms and a depressive disorder was also found by Kroenke *et al.* (1994) in their study of 1000 adult primary-care patients in four primary-care settings, 631 selected randomly and 369 selected by convenience sampling [6]. Tylee *et al.* (2005), in their evidence-based review of the role of somatic symptoms in depression in primary care, concluded that most depressed primary-care patients present with somatic symptoms [10] and found similar results in their analysis of Phase II of the Depression Research in European Society study (DEPRESS II) [11]. Somatic complaints represented two out of three of the most commonly reported symptoms of depression: tired/no energy/listless and broken sleep/decreased sleep [11]. Simon *et al.* (1999), using data from the WHO study of psychological problems in general healthcare, found that 69% of participants who met criteria for depression approached their primary-care clinician complaining of somatic symptoms alone [7]. In a US study, Bair *et al.* (2004) found that 69% of depressed patients reported general aches and pain [12]. Demyttenaere *et al.* (2006) evaluated reports from respondents to the WHO cross-sectional, population-based study of 21,425 representative, randomized, noninstitutionalized adults in Belgium, France, Germany, Italy The Netherlands and Spain [13]. The authors found that painful physical symptoms were reported by 50% of respondents with major depressive disorder (MDD) but only by 29% of respondents without MDD.

Physical symptoms in the absence of an identifiable medical disorder may be a somatic presentation of a primary depressive or anxiety disorder [6]. Chronic pain has been found to be a risk factor for depression and, conversely, depression for chronic pain [5,58]. Somatic complaints have also been found to predict future episodes of depression in women [59]. When somatic symptoms are the primary presentation, neither clinician nor patient may realize that depression is the primary disorder [8,9]. Chronic pain has also been found to result in a higher prevalence of anxiety and depression diagnoses [58]. Katon and colleagues (2001) reported that patients with multisomatoform pains have three-times the lifetime prevalence of depression and anxiety compared with the general population [60]. Some studies of patients with medically unexplained symptoms have found somatic symptoms in the absence of concurrent depression and/or anxiety, and therefore, the assumption that patients with medically unexplained symptoms are simply depressed and/or anxious with predominantly somatic symptoms is not always warranted [61,62].

Nociceptive & pain-modulatory mechanisms***Chronic pain triggering depression & anxiety***

Ongoing research is exploring the mechanisms by which chronic pain results in anxiety. Max *et al.* (2006) hypothesize that depression and anxiety are triggered or worsened by pain and are mediated by anatomical and neurochemical links that differ in part from those mediating depression and anxiety disorders that occur independent of pain [63]. They further propose that in some individuals, pain–mood genes alter the effects of pain upon mood, thus affecting the degree of depression 1 year after the onset of pain. This hypothesis involves a gene–environment interaction model in which particular genes, in the presence of specified stressors, will affect the development of depression. To test the hypothesis for pain, they examined a prospective cohort study, the Maine Lumbar Spine Study [64], for which 162 of 277 surgically treated patients had contributed DNA. No polymorphism affecting the development of pain and depression was identified in this study.

Nackley and colleagues (2005) found that individuals with lower levels of catechol-*O*-methyltransferase (COMT), an enzyme that metabolizes catecholamines and estrogens, displayed heightened mechanical and thermal pain sensitivity, as well as increased risk of development of temporomandibular joint disorder [65], effects associated with *COMT* gene variants that result in higher catecholamine levels as a function of reduced COMT enzyme activity. Their study consisted of administering a COMT inhibitor to rats and measuring response to mechanical and thermal pain [55], with findings that confirmed the relationship of low COMT activity, higher catecholamines and heightened pain sensitivity, phenomena that could be blocked by the nonselective β -adrenergic antagonist, propranolol [55]. This type of research may help to elucidate individual variabilities in pain responsiveness, novel approaches to pain reduction and management, and the pathophysiology of the development of depression and anxiety with chronic pain. The reader is referred to a review by Edwards (2006) on some of these current research findings [66].

Antidepressant treatment & the role of mood disorders

It has been proposed that 5-hydroxytryptamine (5-HT; serotonin) and norepinephrine (NE) modulate brainstem–dorsal horn nociceptive transmission through descending inhibitory pain pathways [67,68]. Tricyclic antidepressants have historically been prescribed for the treatment of pain, and have dual activity, even though they are not as robust as the newer antidepressants in terms of 5-HT agonism. They also produce many untoward interactions leading to unwanted side effects [69–72]. Serotonergic antidepressants have not generally been found effective for the treatment of pain [71–74] and although selective serotonin- and NE-reuptake (dual-transmitter) antidepressants (serotonergic noradrenergic reuptake inhibitors [SNRIs]) have been found efficacious in the treatment of pain [75], their mechanisms are not fully understood.

Animal studies of persistent pain have shown that the combination of 5-HT and NE antidepressants is as efficacious for persistent pain as tricyclic antidepressants [76]. Venlafaxine, a

SNRI, has been shown to be efficacious in the treatment of neuropathic pain [77]. A second SNRI, duloxetine, has been found efficacious in treating painful somatic symptoms of depression [78–80]; one double-blind, placebo-controlled study also found that it reduced diabetic peripheral neuropathy pain in the absence of depression [81] and two double-blind trials found it useful in the treatment of fibromyalgia with or without depression [82,83].

Ongoing studies are currently examining the efficacy of both newer selective SNRIs and selective serotonergic reuptake inhibitors (SSRIs) in resolving the somatic complaints of depression, as well as in returning patients to their usual quality of life and work efficacy; a complete review of these studies is beyond the scope of this article. Two studies suggest the superiority of SNRIs in treating the depressed patient with a predominance of painful somatic symptoms [78,84], but more studies are needed to discern differences in treatment efficacy and remission in outpatient depressed populations with and without a preponderance of painful somatic symptoms. Denninger *et al.* (2006) examined the resolution of somatic symptoms of depression using fluoxetine, a SSRI, in an 8-week, open-label trial of 170 MDD outpatients with an average age of 40 years. Notably, there were no differences in baseline somatic scores with regard to gender. They found that good resolution of somatic symptoms correlated with an improvement in depression [85]. However, this study was not placebo controlled and as such this important research question would be well served by additional studies with placebo controls.

Dynamic interactions: depression & menopausal symptoms (somatic & psychological)

Both somatic and psychological symptoms of depression and anxiety can be similar to those attributed, correctly or incorrectly, to the menopausal transition. Distressing somatic complaints, such as general aches and pains [12,13], back/chest pain and arthralgias [2,4–10,12,13], tiredness/lack of energy/listlessness [11], sexual dysfunction [86–94] and sleep disturbance [95–97], are associated with depression. Menopause, natural or induced, has been associated with arthralgias, bodily aches and pains, back pain, diffuse musculoskeletal pain [1–3,18,19,22,24–27], poor sleep [1,98–101] and sexual dysfunction [102–111]. Depression and/or anxiety are among the most common causes of disturbed sleep [95], with 50–90% of those diagnosed with depression complaining of sleep problems [96,97]. Mood, stress, fatigue and/or anxiety problems may lead to sexual dysfunction during the menopausal transition and beyond, with both contemporaneous factors and menopausal status also playing roles [102,103,112].

For patients with comorbidities, there may be a dynamic interaction between different somatic symptoms associated with both depression and menopause. Vasomotor symptoms have been found to be associated with the menopausal transition to varying degrees, from none to severe, and lasting for a short period of time to many years [113–115]. Women with depression have a higher prevalence of vasomotor symptoms [116] regardless of prior history of depression [117]. Anxiety has

also been reported as a risk factor for hot flashes [118]. Sleep problems have been reported both as secondary to hot flashes [1,98–101] and as secondary to menopause, independent of hot flashes [1]. There remains some controversy in the field with regard to whether hot flashes disturb sleep [119] and to whether the menopausal transition directly affects sleep quality in some women [120]. TABLE 1 illustrates the somatic neurobehavioral symptoms that are similar for depression as well as for symptomatic menopause.

The range of mood problems, from minor depression to MDD, as well as the physical and psychological symptoms a woman may complain of, can be quite difficult to distinguish, particularly in the presence of disturbing menopausal symptoms, such as night sweats and/or sleep disturbance. For some women, particularly those with a history of mood disorders, menopause may exacerbate symptoms of depression and depression may exacerbate symptoms of menopause (e.g., hot flashes). Depression itself, whether minor or major, can be *de novo* due to menopause, independent of it or due to a dynamic interaction of risk factors for depression and menopause.

Expert commentary

Somatic symptoms, such as back pain, diffuse arthralgias, and aches and pains, can occur in both depression and menopause. The confluence of somatic and psychological symptoms for both a symptomatic menopause and a depressive illness may confound both the patient and clinician. The pathophysiology for pain mechanisms, the role of estrogen

and the co-occurrence of depression can contribute to the occurrence and perception of pain and arthralgia are all areas requiring further research.

Care for the symptomatic and distressed midlife woman with or without psychiatric comorbidity requires the careful development of evidence-based differential diagnoses, as well as a multifaceted treatment plan that includes interventions with the greatest likelihood of ameliorating the distressing symptoms that interfere with these women’s lives as individuals, the wellbeing of their families and their ability to function in the workplace.

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Table 1. Comparison of somatic and neurobehavioral symptoms seen in both menopause and depression.

<i>Perimenopause and early postmenopausal</i>	<i>Depressive disorder</i>
Hot flashes	Hot flashes are not usual but appear to be worsened by depression in some women
Sleep disturbance	Sleep disturbance
Severe tiredness	Fatigue/loss of energy
Back pain	Back pain
Stiff and painful joints	Stiff and painful joints
Bodily pain	Bodily pain
Worse general health	Worse general health
Reduced sexual responsiveness, libido or frequency of sexual activity (variable from woman to woman across transition) [37,121]; sexual disinterest or dysfunction, vaginal dryness in 1/3 of patients	Reduced sexual responsiveness, libido or frequency of sexual activity
<i>Neurobehavioral symptoms</i>	
Change in cognitive function related to ↓sleep or ↓mood or ↑ anxiety, or exacerbation of an underlying cognitive problem	↓Attention, ↑ distractibility, both perceived as loss of cognitive function
↓Mood	↓Mood
↓Quality of life if symptomatic	↓Quality of life

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Content & views

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Appendix

- **CME post test** and method of participation.

CME post-test and method of participation

Release date:	1st November 2007
Expiration date:	1st November 2008
CME units:	1 unit ACCME, and AAFP CMA PRA category 1 credits™ or CEU
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Processing fee:	US\$5/ACCME unit
Target audience, curricular goals, educational objectives and accreditation/credit designation:	go to www.afwh.org for a complete description of credit available or to the CME section of this supplement

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CME post-test and method of participation

CME/CEU online post-test (www.afwh.org)

Select an answer for each question. You must achieve a test score of 70% or greater to earn credit

1. *Which of the following are the most common types of somatic symptoms typically attributed to the menopause by patients? (a, b, c, all)*
 - a. Hot flashes and night sweats
 - b. Sleep complaints, tiredness
 - c. Back pain, stiff and painful joints

 2. *Which are the most common conditions found to cause back pain, tiredness, arthralgias and bodily aches and pains? (a, a & b, all)*
 - a. Depression
 - b. High BMI
 - c. Fibromyalgia

 3. *A decline in which of these factors was found to be associated with a decline in quality of life for women in the menopausal transition as concluded by the prospective longitudinal population-based cohort of 1525 British women followed 19-times from birth to 43 years of age and then annually from ages 47 to 54 years ('the 1946 British Birth Cohort', also known as the Medical Research Council National Survey of Health and Development)? (a & c, abc, all)*
 - a. Decline in physical health
 - b. Decline in energy level
 - c. Decline work and family stress
 - d. Increase in body weight

 4. *Breast cancer patients treated with aromatase inhibitors have reported more of which of the following symptoms? (a or b, abc, all)*
 - a. Arthralgias
 - b. Myalgias
 - c. Vaginal dryness
 - d. Dry mouth
-