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Improving the Quality of Life among Postmenopausal Women in a Resource-limited Setting: A Review of Possible Role of Phytoestrogens

Emokpae M. A., Aikoriogie O., Babatunde E. M. and Emokpae L. A.

ABSTRACT

Background: Menopause is often accompanied by deleterious and degenerative health effects, such as arteriosclerosis, osteoporosis and atrophic changes the skin, suggesting that acceleration of aging is triggered by a lack of estrogen. This review seeks to discuss the possible use of phytoestrogens in combating the adverse health consequences associated with menopause.

Methods: Relevant literature from electronic search engines such as PubMed, Cochrane Library, Embase, Web of Science, CINAHL, and African Journals Online (AJOL) regarding the use of phytoestrogen to combat complications of menopause was obtained and reviewed. Keywords used in the search were combined which include Postmenopausal Women, Quality of Life, Resource-Limited Setting" OR "Low-Income Country and Health Outcomes. Studies were selected based on the pre-defined criteria, structured around the Population, concept and context.

Results: Studies have indicated a growing demand for alternative treatments for the symptoms and pathological processes associated with menopause. Research has supported the use of natural antioxidants such as soy isoflavones, extracts of Curcuma longa, β-carotene, and honey that are rich in phytoestrogens to ameliorate or prevent antioxidant deficiency associated with menopause. Natural phytoestrogens protect mitochondria against premature oxidative damage with loss of adenosine triphosphate synthesis and cellular functions. The consumed phytoestrogens act as sources of estrogen and antioxidants needed to prevent menopause-associated pathologies.

Conclusion: Natural phytoestrogens could be considered an alternative option for the management of menopause associated symptoms and pathologies in postmenopausal women in a resource-limited setting.

Key words: Female; Post-menopause; Antioxidants; Quality of Life

INTRODUCTION

Menopause is one of the most significant events in a woman's life, and it leads to several physiological changes that affect her life permanently. As a result of decrease in the estrogen concentration in the blood caused by menopause, reproductive capacity decreases, leading to complete cessation of ovarian function, decreased psychological function, poor memory or forgetfulness, vaginal dryness and urinary tract infections (1, 2). There are speculations about the symptoms that appear before, during, and after the onset of menopause. These symptoms constitute postmenopausal syndrome; they adversely impair the quality of life of postmenopausal women, and the management of these symptoms has become an important field of research (2).

Studies have reported that there are cultural and ethnic differences in the severity and frequency of menopausal symptoms). Factors responsible for the differences include genetics, dietary habits, level of activity, and daily exercise due to differences in the natural age of menopausal women (3,4). Abstinence from difficult tasks, cultural conflicts, belief and knowledge of the menopausal process, and the presence of stressors are among the factors influencing menopausal quality of life (4, 5). For example, the prevalence of hot flashes and night sweats was reported to be greater among African American and Caucasian women than among Asian women (6). It was recently observed that meal-based intervention may be an important strategy for overall health promotion in relatively healthy menopausal women (7). Furthermore, awareness of and access to menopause-related information and services are lacking in some African countriescountries, since menopause is often not discussed within families, communities, workplaces, or health-care settings (8). Also, chronic strain that exist due to a persistent inadequate allocation of health care resources, there is demand for care that surpasses standing supply in most health care facilities (8). Therefore, perimenopausal women in this setting need to know that the symptoms they experience are related to menopause and be aware of what lifestyle habits and diets that can improve their quality of life. This review seeks

*Correspondence: mathias.emokpae@uniben.edu
Department of Medical Laboratory Science, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria.
Full list of author information is available at the end of the article

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to discuss the possible use of phytoestrogens in combating the adverse health consequences associated with menopause.

MATERIALS AND METHODS

This article presents a systematic review (or scoping review) of the literature aimed at synthesizing current evidence regarding strategies to enhance the quality of life for postmenopausal women in resource-limited environments. The review adhered to a structured methodology to guarantee thorough and impartial data collection and synthesis.

A thorough systematic search was executed across several electronic databases: PubMed, Cochrane Library, Embase, Web of Science, CINAHL, and African Journals Online – AJOL The search strategy employed a combination of Medical Subject Headings (MeSH) terms along with free-text keywords pertinent to the population, intervention/concept, and setting. The search spanned from the year 2000 to October 2025, ensuring the inclusion of recent research.

Keywords were interconnected using Boolean operators (AND, OR) and included terms such as "Postmenopausal Women, Quality of Life, Resource-Limited Setting" OR "Low-Income Country and Health Outcomes." Studies were chosen based on pre-defined criteria, structured around the Population (studies involving postmenopausal women), Concept (research concentrating on interventions, strategies, programs, or factors directly related to enhancing or evaluating the women's Quality of Life (QOL), physical, or mental health outcomes), and Context (studies conducted in or specifically addressing resource-limited settings, such as countries classified as low or middle-income by the World Bank, or specific resource-poor geographical areas within any country) framework, suitable for a scoping/systematic review. Original research articles (e.g., randomized controlled trials, observational studies, qualitative studies) and existing reviews were included for a more extensive scoping.

Studies that focused solely on perimenopausal or premenopausal women, those conducted exclusively in high-income, resource-rich settings, editorials, commentaries, conference abstracts without full papers, or book chapters (unless they are highly relevant and the primary source is unavailable) were excluded.

Biochemistry of Menopause

Menopause is a natural step in the process of aging (9). Menopause is associated with a significant decrease in plasma concentrations of estrogen, increased levels of follicle-stimulated hormones and luteinizing hormones and alterations in other hormones, such as inhibins (10). These biochemical alterations coincide with the effects of aging, social and metabolic factors, daily activity and well-being (10). The adverse effects of menopause, which are attributed to a decrease in estrogen levels in the blood, can lead to alterations in the lipid profile, body mass index, and insulin levels and an increase in the risk of hypertension, cardiovascular disease, osteoporosis, diabetes mellitus, cancer, and other degenerative changes in postmenopausal women (11).

An increase in the production of free radicals after menopause, which is due to sudden alterations in hormonal status, has been reported (12, 13). There is enhanced oxidative stress and decreased antioxidant defense in postmenopausal women compared to premenopausal women, which can play an important role in the pathogenesis of the various diseases related to menopause. This may also explain why postmenopausal women are more susceptible to chronic diseases, such as atherosclerosis, Alzheimer's disease, and postmenopausal osteoporosis (14-16). Free radicals are potentially harmful to almost all biomolecules, including lipids, carbohydrates, and proteins (17, 18).

Oxidative stress can lead to damage to most major cellular components of the body, including proteins, DNA, and membrane lipids, possibly resulting in apoptosis and cell death (19). The lipids of cell membranes are the favorite targets of free radicals, which are oxidized, leading to lipid peroxidation. Lipid peroxidation is specifically dangerous for the cell because it propagates through a self-perpetuating chain reaction (19).

The human body requires both oxidant and antioxidant substances for normal metabolism, regulation of cellular functions, and signal transduction. Hence, each cell maintains a state of equilibrium between the oxidant and antioxidant species. The generation of oxidants more than the antioxidants present in the body leads to oxidative stress. Lipid peroxidation and other degradation products, such as malondialdehyde (MDA), are produced at increased levels in biological fluids. The enzyme superoxide dismutase (SOD) is the primary enzymatic antioxidant that catalyzes the degradation of superoxide anions to oxygen and hydrogen peroxide. Because superoxide is the principal free radical generated from several activities in the body, dismutation by SOD is vital for physiological functions. Other enzymatic antioxidant systems include glutathione peroxidase (GPx) and catalase (CAT), while nonenzymatic antioxidants include vitamin C and vitamin E (20).

The progressive loss of estrogen and its protective effects combined with deficient endogenous antioxidants exacerbates oxidative stress. To prevent oxidative damage, the human body possesses an antioxidant defense mechanism that can activate free radicals, metal chelation, and enzymatic activities to neutralize reactive species. Additionally, the consumption of dietary antioxidants can maintain an adequate concentration of antioxidants in the human body and hence mitigate oxidative stress (21).

Some metal elements, particularly calcium (Ca), magnesium (Mg), copper (Cu), manganese (Mn), and zinc (Zn), are essential for bone metabolism. Some trace minerals are co-

factors of many enzymes. Se is a cofactor of glutathione peroxidase, one of the most important enzymes among the defense enzymes against free radicals. Zn and Cu are integrated elements of superoxide dismutase (Cu/Zn SOD). Manganese superoxide dismutase (Mn-SOD) is a major enzyme responsible for the detoxification of ROS in mitochondria. MnSOD protects cells from harmful effects associated with the excess generation of nitric oxide (NO) and peroxynitrite in two ways: it reacts directly with reactive nitrogen species (RNS) and plays an active role in the detoxification of reactive oxygen species (ROS) by preventing the reactions of ROS with NO to form peroxynitrite (22). Reactive oxygen species (ROS) and lipid peroxides, which are produced by a free radical chain reaction, have been implicated in the pathogenesis of a variety of conditions, including menopause (23). Estrogen deficiency in postmenopausal women may be associated with postprandial hyperlipidemia and could limit peripheral glucose uptake. Recently, a study recommended monitoring risk factors for type 2 diabetes and CVD among postmenopausal women to reduce morbidity and mortality associated with estrogen decline (24).

Menopause is a unique event in women's lives that occurs approximately age 45-50 years, and women spend more than one-third of their lives in postmenopausal state (24). A higher incidence of metabolic syndrome, cardiovascular risk factors, and changes in lifestyle factors has been reported in menopausal women (25, 26). Changes in gut microbiome species were reported among postmenopausal women; four species were significantly abundant in postmenopausal women, while four other species were abundant among premenopausal women (24). The duration, intensity and impact of these symptoms differ from one person to another, and in different societies, some women might experience more severe symptoms that could deeply impact their personal and social performance as well as their quality of life, which might lead them to encounter multiple serious issues in life (27).

What is the quality of life?

According to the World Health Organization (WHO), quality of life (QoL) is the "individual's perception of their position in life in the context of culture and value systems in which they live about their goals, expectations, standards, and concerns" (28). Quality of life is a wide-ranging phenomenon that is affected in a complex way by an individual's physical health, psychological state, level of independence, social relationships, and personal beliefs, along with their relationship with the salient features of their environment (29, 30). Even though QoL is difficult to measure, several organizations have developed measurement scales that can be used; these include the "Healthy Days Measures" and "Patient-Reported Outcomes Measures (PROMS)". These are based on a patient's health condition as perceived by the individual. QoL is an important aspect of health that plays a significant role in the execution and evaluation of health interventions (31). Some have recently suggested the need for reproducible and accurate tools for its measurement (32).

Life expectancy has increased in several countries worldwide due to new developments in medical sciences and healthcare services (33). As a result, many women spend a third of their lives after menopause (34). Therefore, the QoL of postmenopausal women is of great public health interest (35). QOL after menopause can be influenced by a host of personal and social factors. Enabling factors such as access to information and participation in educational programs on the issues of menopause may improve the quality of life of postmenopausal women. In addition to increasing awareness, women exhibit improved attitudes, and they feel more confident, powerful, and valuable. These symptoms will decrease the severity of menopausal symptoms and thereby improve quality of life (36). Access to funds could also be a factor. Studies have shown that one of the most important factors in healthcare utilization is adequate income and access to financial resources (37, 38). In postmenopausal women, access to financial resources is also one of the factors affecting quality of life. Changes associated with menopause can affect a woman's quality of life. Approximately 50-80% of women complain of menopausal symptoms, which are the main determinants of a reduced health-related QoL (39). Postmenopausal women have a greater incidence of menopausal symptoms that significantly affect their QoL than premenopausal and perimenopausal women (40). In Nigeria, evidence suggests that approximately 50% of postmenopausal women have symptoms related to joint and muscular discomfort, and some researchers recommend paying close attention to comprehensive sexuality education for menopausal women to improve their QoL(41). Olowookere *et al*. (42) reported that 58.3% of their study participants had poor lifestyles, with 41.0% and 0.7% having moderate and good lifestyles, respectively. Overall, 66.4% of the women experienced moderate menopause-related symptoms, while 15.5% and 0.4% of the women experienced severe and very severe menopause-related symptoms, respectively. It was concluded that positive lifestyle modifications might ameliorate menopausal-related symptoms. Furthermore, others have recommended social support as the most important determinant of menopausal symptom severity. The enhancement of social support in Nigeria for women to cope better with menopausal symptoms and improve their overall quality of life was suggested (43).

Pathogenesis of Menopause

Although menopause is associated with changes in the hypothalamic and pituitary hormones that regulate the menstrual cycle, menopause is not a central event but rather a primary ovarian failure (42,43). At the level of the ovary, there is depletion of ovarian follicles. Therefore, the ovary is no longer able to respond to pituitary hormones, i.e., follicle stimulating hormone (FSH) and luteinizing hormone (LH), and

ovarian estrogen and progesterone production cease. Androgen production from the ovary continues beyond the menopausal transition because of the sparing of the stromal compartment (43,44). Menopausal women continue to have low levels of circulating estrogens, principally from peripheral aromatization of ovarian and adrenal androgens. Adipose tissue is a major site of aromatization, so obesity affects many of the sequelae of menopause. The ovarian-hypothalamic-pituitary axis remains intact during the menopausal transition (45). Thus, FSH levels increase in response to ovarian failure, and there is no negative feedback from the ovary. Atresia of the follicular apparatus, in particular granulosa cells, results in reduced production of estrogen and inhibin, leading to a reduction in inhibin levels and elevated FSH levels, which are cardinal signs of menopause (45,46).

The menopausal transition or perimenopause is a defined period beginning with the onset of irregular menstrual cycles until the last menstrual period and is marked by fluctuations in reproductive hormones (44). This period is characterized by menstrual irregularities, prolonged and heavy menstruation intermixed with episodes of amenorrhea, decreased fertility, vasomotor symptoms, and insomnia. Some of these symptoms may emerge four years before menses cease (42). During the menopausal transition, estrogen levels decline, and FSH and LH levels increase. The menopausal transition is characterized by variable cycle lengths and missed menses, whereas the postmenopausal period is characterized by amenorrhea. The menopausal transition begins with variability in menstrual cycle length accompanied by rising FSH levels and ends with the final menstrual period.

The ovary is the only source of oocytes, her primary source of estrogen and progesterone, and a major source of androgens. Ovarian cessation of progesterone production appears to have no clinical consequences except for the increased risk of endometrial proliferation, hyperplasia, and cancer associated with continued endogenous estrogen production or the administration of unopposed estrogen therapy in menopausal women (45).

Oxidative Damage as Pathogenesis of Menopause

Studies have confirmed the association between tmenopause and the development of chronic degenerative diseases, cardiovascular diseases (CVDs), Alzheimer's disease (AD) and osteoporosis among postmenopausal women (46, 47). The association between increased oxidative damage and menopause may be due to the loss of protective effects of estrogen in most organs or tissues where the estrogen receptors ERα and ERβ are expressed at different levels (48). These receptors are critical for the actions of both endogenous and exogenous estradiol administered in hormone replacement therapy (HRT) to ameliorate the effects of ovarian deficiency. To date, the benefits of HRT for women's health are still controversial, as available evidence suggests that its effectiveness in reducing the risk of CVD, incidence of osteoporosis and all-cause mortality is still questionable (47). At the metabolic level, estradiol has been demonstrated to lower the levels of atherogenic plasma lipids, improve endothelial function, lower inflammation, and increase neuronal survival, thus highlighting the complex modulatory actions of estradiol (45,46).

It is now well understood that estrogen exerts antioxidant effects by modulating the expression of antioxidant enzymes and nutritional antioxidants that apparently act via a similar mechanism (46,47). Based on this perspective, it is believed that the control of oxidant challenges and biological defenses in the body through adequate dietary supplementation could help alleviate the pathological consequences of menopause (47).

The combined effects of decreasing estrogen concentration and aging contribute to the increasing incidence of CVD in females (46). This was supported by evidence that premenopausal women have a lower incidence of CVD than age-matched men do, but this sex-linked advantage disappeared after menopause (46,47). However, some have suggested that the protective effects of estrogen against oxidative damage to cardiovascular, neuronal and bone tissue have not been scientifically validated in human disease (47). It is imperative to understand that increased oxidative stress is critical to the pathogenesis of vascular diseases and the harmful effects of free radicals on endothelial dysfunction, muscular proliferation and contraction, and the formation of atherosclerotic plaques. The loss of NO bioavailability that moderates endothelium-dependent relaxation is the main trigger of damage to vascular walls. Estrogen is able to guarantee adequate NO bioavailability by enhancing the production of NO via the induction of NO synthase expression or decreasing the generation of superoxide (47, 49).

Menopause is also associated with a high incidence of osteoporosis and risk of fracture. This is due to high bone turnover and loss of bone mass. During premenopausal life, estrogen protects bone against erosion by providing a critical balance between bone resorption and formation (10,25,47). The ability of estrogen to reduce oxidative stress was associated with the mechanisms underlying the beneficial effects of estrogen on bone cells. This difference may be attributed to estrogen receptor α-mediated activation of cytoplasmic kinases (10). Estrogen enhances glutathione reductase activity in the bone marrow, which is a rich source of mesenchymal stem cells in transgenic mice (47).

Similarly, approximately 40% of women undergoing menopause were reported to have high bone turnover with a rapid decrease in bone mass density. These individuals are commonly referred to as "fast-looser", and they are at high risk of developing overt osteoporosis (47).

Hormonal alterations in menopausal women trigger the production of proinflammatory mediators and induce

oxidative stress, leading to progressive neuronal damage (50). Evidence suggests that major depressive disorder (MDD) is 50% more common among women of reproductive age than among men, and approximately 20% of women may experience an episode of MDD at some point in their life (51). Furthermore, the risk of depression appears to double or even triple during the postmenopausal period compared to the perimenopausal period (50).

Menopause and Psychological Changes

As a result of differences in culture, dietary habits, and socioeconomic status, differences exist in the incidence of psychologic symptoms among postmenopausal women. Apart from the common symptoms of hot flashes, sweating, palpitations, dizziness, anxiety, irritability, and headache, the most common mental problems among Asian perimenopausal women were reported to be insomnia, irritability (51%) and stiff joints and pain (54). However, in Nigeria, the most common symptoms were pain in the joints (57%) among suburban women (53), joint discomfort (83.9%), physical exhaustion (67.4%), and depressive mood (64.0%) among urban women (54).

The marked decline in the levels of estrogen during menopause directly impact the brain and is responsible for the psychological and cognitive changes in women. It is important to recognize that these psychological symptoms are real and rooted in biological changes, and they can be amplified by other stressors (e.g., career demands, caregiving for aging parents, children leaving home). Effective remediations have been suggested using natural phytoestrogen (55-58).

Protective Effects of Natural Antioxidants against Menopausal Symptoms

Natural antioxidants, primarily sourced from diet and supplements, are essential in alleviating menopausal symptoms and reducing long-term health risks by directly counteracting oxidative stress (55). These natural antioxidants function through Free Radical Scavenging. They provide electrons to unstable reactive oxygen species (ROS), such as hydroxyl radicals ({OH}) and superoxide anions (O), effectively neutralizing them and preventing the chain reactions that can damage cellular components, including lipid peroxidation in cell membranes (56). Additionally, certain dietary antioxidants serve as cofactors for the body's own antioxidant enzymes, including Superoxide Dismutase (SOD), Catalase (CAT), and Glutathione Peroxidase (GPx), thereby enhancing the body's intrinsic defense mechanisms, which are often diminished post-menopause(57,58).

The protective benefits of antioxidants are evident in several critical areas, including cardiovascular health protection. The risk of cardiovascular disease (CVD) significantly escalates during menopause (26,56).

Table 1: Relevance of some Antioxidants to the amelioration of Menopausal symptoms

Antioxidant Compound	Source and Mechanism	Relevance to Menopause
Vitamin E (tocopherol)	Nuts, seeds, vegetable oils. Protects cell membranes from lipid peroxidation.	**Inhibits the oxidation of LDL cholesterol**, a key step in atherosclerosis (plaque formation), thereby reducing CVD risk [13,17].
Flavonoids/Polyphenols	Soy milk, Berries, green tea, dark chocolate, red wine. Potent direct free radical scavengers.	**Improves endothelial function** (blood vessel lining) by increasing the bioavailability of Nitric Oxide (NO), a vasodilator that is often reduced due to estrogen loss (55,56,59).
Omega-3 Fatty Acids	Oily fish (salmon, mackerel), flaxseed. Anti-inflammatory properties.	Reduces systemic inflammation and **improves arterial stiffness**, a marker of cardiovascular health deterioration in postmenopausal women (59,60).

Although not a direct hormonal therapy, antioxidant capacity is linked to the severity of symptoms like hot flashes and night sweats (10,48). Research has indicated a negative correlation between a diet rich in total antioxidant capacity and the severity of overall Menopause Rating Scale scores, which include hot flashes and night sweats, implying a systemic advantage from diminished oxidative stress [60]. Vitamin C, present in citrus fruits, peppers, and broccoli, has the ability to regenerate other antioxidants (such as Vitamin E) and aids adrenal function, which is vital for managing stress and mood fluctuations related to menopause (20,58).

Oxidative stress and persistent inflammation hasten bone deterioration. While vitamin D and calcium are fundamental for bone integrity, antioxidant vitamins and minerals (including selenium and zinc) which serve as crucial cofactors for the enzymes that regulate the oxidative environment within the bone matrix, thereby indirectly contributing to the mitigation of postmenopausal osteoporosis (21,23).

Integrating a diverse array of these natural compounds into the diet is the key approach for the prevention of menopausal symptoms:

A study conducted in Canada revealed that several postmenopausal women use complementary or alternative medicine to ameliorate menopausal syndrome, but they still have concerns regarding its cost and efficacy (60). The United States Food and Drug Administration (FDA) has approved estrogen-based therapy for several years to alleviate the most common postmenopausal symptoms (vasomotor) (61). Due to the deleterious effects of HRT, the FDA recommended the use of estrogen treatments for the amelioration of menopause-associated symptoms, and cautious use of HRT was advised for osteoporosis prevention (62). Nutraceuticals, including phytoestrogens and herbal derivatives, have recently attracted public interest because of their ability to mitigate menopausal symptoms (62,63). Some of the highlighted nutraceuticals include *Actaea racemosa* and *Valerian officinal* used to alleviate common menopausal symptoms and musculoskeletal pain, and *Panax ginseng* used to treat depression, insomnia and libido, while *Ginkgo biloba* has been used to improve attention deficit in postmenopausal women (63).

The relatively lower incidence (10–25%) of hot flush among postmenopausal women in Asian countries such as China, Japan, Korea and Taiwan compared to their counterparts in Western countries (60–90%) was attributed to the consumption of soybeans and other isoflavone derivatives (64). Isoflavones can exert estrogen-like effects and thus alleviate postmenopausal syndrome.

Isoflavones have been shown to possess antioxidative and anti-inflammatory properties and have been used in the management of diseases. Isoflavones have promising therapeutic efficacy in the management of several diseases (55,56). For example, human epithelial colorectal adenocarcinoma cells treated with genistein showed decreased interferon (IFN) -induced signal transducer and activator of transcription 1 (STAT1) phosphorylation (55), inhibited microglial activation by lipopolysaccharide (LPS) using genistein and daidzein (56), and reduced inflammatory markers albino rats after treatment with a soybean phytoestrogen-rich extract in 4-vinyl cyclohexane diepoxide-induced menopause (57). There was a reduction in inflammatory indicators (C-reactive protein, interleukin-6, and tumor necrosis factor-α) after treatment with different concentrations of soybean phytoestrogen-rich extract (57). The soybean phytoestrogen extract contains genistein, which is structurally similar to 17-hydroxylase estradiol (57,59). It can bind to estrogen receptors and carry out its functions within cells. Genistein has a very high affinity (87%) for estrogen receptor-beta (ERβ), which is approximately 20-30 times greater than the affinity of ER- for 17-estradiol (4%) (57). A molecular docking study revealed that the interaction energy between genistein and ERβ was lower than that between genistein and ERα (58). The administration of genistein can lead to enhanced genomic activities of the estrogen–eNOS receptor complex, which is associated with apoptosis, proliferation, and telomere activity (58). It was reported that ERβ can significantly induce the expression of Ki-67, as demonstrated by the interaction between ERβ/eNOS/17β-estradiol-genistein and the Ki-67 gene promoter. Additionally, genistein may be more effective at inducing the above interaction than 17β-estradiol is (58). This may be true judging from the lower energy required for interaction. Another proliferation marker reported to be significantly induced by genistein is the CyclinD1 gene. The authors observed that the ERβ/eNOS/17β-estradiol-genistein complex induced transcriptional activation of the CyclinD1 gene (58).

After evaluating the antioxidant properties of the soybean phytoestrogen-rich extract in 4-vinylcyclohexane diepoxide-induced menopausal rats, the authors reported that the antioxidant effects of the soybean phytoestrogen-rich extract increased in a dose-dependent manner in menopausal-induced Wistar rats. The administration of a high dose of soybean phytoestrogen-rich extract was more effective than estrogen therapy, and it was concluded that soybean may be a better alternative source of estrogen for mitigating oxidative stress in menopausal and postmenopausal women (59).

Curcuminoids (curcumins or turmeric) are a group of active compounds that have a wide range of pharmacological and biological activities, such as powerful free radical scavenging properties, increasing intracellular glutathione levels and reducing lipid peroxidation (65,66). Curcumin was reported to ameliorate testicular injury induced by cadmium toxicity and sperm indices in male albino rats (67). It was concluded that curcumin may improve spermatogenesis and sperm indices and reverse oxidative stress.

It was recently reported that ethanol root extract of turmeric was protective against impaired learning and memory loss induced by cadmium in adult Wistar rats (68). The authors used two standard memory tests (Y-maze and novel object recognition) to evaluate the protective effect of the ethanolic extract of turmeric root against cadmium-induced neurotoxicity in rats. The Y-maze test calculates the percentage of spontaneous alternation behavior, which is regarded as a measure of short-term spatial memory in rodents. This requires the animals to remember the arm most recently entered in a bid to alternate the choice of next arm entry (69). The authors observed that spontaneous alternation behavior among cadmium-treated rats was significantly lower than that among control rats. In addition, rats pretreated with ethanolic turmeric root extract exhibited significantly greater spontaneous alternation behavior than rats treated with cadmium alone, hence conferring protection against the cognitive deficit induced by cadmium in the Y-maze task (68). Similarly, the exploration of the new object was significantly lower in rats treated with cadmium alone than in non-

cadmium-treated rats. The degenerative changes in the cerebral and hippocampal structures were attributed to the susceptibility of the rats to cadmium neurotoxicity and the ability of cadmium to induce oxidative damage. However, supplementation with ethanolic turmeric root extract protected against histomorphological changes in the cerebrum and hippocampus of rats exposed to cadmium alone, and these sections showed that turmeric extract has the capacity to protect against neurodegeneration (68).

Conclusion: The reviewed scientific data indicate that postmenopausal women benefit from the consumption of natural antioxidants against common symptoms. Additionally, the consumption of more natural antioxidants is associated with a lower risk of developing cardiovascular diseases. Even though natural antioxidants may not be as effective as HRT in alleviating some menopausal-associated disorders, the satisfaction rate among postmenopausal women is very high. Due to the near absence of natural antioxidant toxicity coupled with its overall health benefits, it is considered an alternative option for the management of postmenopausal women.

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Author Details: Department of Medical Laboratory Science, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria.

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