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REVIEW

Neuromusculoskeletal disorders in pregnancy revisited: Insights and clinical implications

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ABSTRACT

Women face a higher risk of musculoskeletal disorders, particularly during and after pregnancy, due to complex hormonal and biomechanical changes. Pregnancy-associated hormonal fluctuations, primarily involving estrogen, progesterone, and relaxin, contribute to increased joint laxity, altered collagen dynamics, and ligamentous instability. Simultaneously, anatomical adaptations, including shifts in the center of gravity, increased lumbar lordosis, and altered gait mechanics, further strain the musculoskeletal system. These changes predispose pregnant women to conditions such as low back pain, pelvic girdle dysfunction, hip pathologies, and peripheral neuropathies, significantly impacting daily function and quality of life. While many of those disorders resolve postpartum, their long-term effects remain inadequately studied, raising concerns about potential chronic musculoskeletal complications, including osteoarthritis and persistent joint instability. Despite the prevalence of these conditions, there is a lack of standardized, evidence-based clinical pathways for assessment, early intervention, and postpartum rehabilitation. Current management strategies tend to focus on short-term symptomatic relief, such as analgesic use and activity modification, while often overlooking preventive strategies like routine musculoskeletal screening during prenatal visits, structured exercise programs tailored to pregnancy, and postpartum rehabilitation protocols. A well-tailored interdisciplinary collaboration is critical to address this gap. Obstetricians are well-positioned to identify early signs of musculoskeletal problems and initiate referrals. Physiatrists can develop and implement non-pharmacological treatment plans, including therapeutic exercise, physical therapy, and posture correction. They are also able to initiate targeted injections for musculoskeletal problems. Orthopedic surgeons provide expertise in diagnosing and managing structural abnormalities or persistent biomechanical dysfunction. Together, these disciplines can design comprehensive, longitudinal care models that prioritize both prevention and recovery. Research is still needed to delineate the long-term consequences of pregnancy on the musculoskeletal system and to establish preventive measures that enhance maternal health beyond the perinatal period. In this review, we address this gap by providing a comprehensive discussion regarding the effects of pregnancy and hormonal changes on the musculoskeletal system, as well as the commonplace conditions in daily clinical practice.

Keywords: Gestation, hormone, joint, ligament, tendon.

Women are at an elevated lifetime risk of developing musculoskeletal disorders, particularly affecting the spine, knee, shoulder, and foot.[1] Although the precise mechanisms underlying this sex-based disparity remain uncertain, potential contributing factors include variations in muscle strength, joint anatomy/alignment, ligamentous laxity, and hormonal effects.[2,3] These factors can be further exacerbated by pregnancy-related physiological changes.

During pregnancy, physiological changes in gait and posture, hormonal fluctuations, and increased joint laxity are observed.[4,5] Most peripheral and pelvic joints, including the wrist, finger, knee, ankle, pubic symphysis, and sacroiliac joints, are affected by ligamentous laxity due to hormonal

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and biomechanical changes during pregnancy. The center of gravity shifts anteriorly due to added trunk mass, resulting in compensatory adaptations in the body, such as an increase in lumbar lordosis, posterior sacral tilt, and cervical extension.[6] Concurrently elevated levels of relaxin, progesterone, and estrogen mediate structural changes in the pelvic ligaments to facilitate delivery. These hormonal effects extend to peripheral joints and connective tissues (tendons, cruciate ligaments, plantar fascia) and may result in ligamentous laxity, collagen remodeling and joint hypermobility.[7] These kinetic and dynamic changes collectively increase the risk of joint instability during pregnancy.

The increased risk is not limited to joint instability, but various other musculoskeletal problems also exhibit a higher prevalence. There is a notable lack of long-term prospective studies examining the persistence of pregnancy-related musculoskeletal changes in the postpartum period within the current literature. Moreover, the relationship between the increased risk of joint instability and female hormonal changes, as well as pregnancy, remains insufficiently investigated. In this review, we address this gap by providing a comprehensive discussion regarding the effects of pregnancy and hormonal changes on the musculoskeletal system, as well as the commonplace conditions in daily clinical practice.

HORMONAL EFFECTS ON THE MUSCULOSKELETAL SYSTEM

Estrogen, progesterone, relaxin, prolactin, and oxytocin are produced in high quantities during pregnancy. Therefore, understanding the effects of these hormones can provide a comprehensive approach to the musculoskeletal changes in the course of pregnancy. Estrogen, a sex hormone, affects multiple tissues, including those in the musculoskeletal system, such as muscles, bones, cartilage, joints, tendons, and ligaments.[8] Tendon and ligament injuries exhibit sex differences, a topic of growing interest among researchers.[8] Physiological and anatomical variations play a role in this disparity, with sex hormones being considered a significant contributing factor.

Estrogen and collagen dynamics in ligaments and tendons

Estrogen influences collagen content, structure, and metabolism in ligaments and tendons, thereby altering the mechanical properties of joints, particularly during pregnancy.[9] However, the effects of estrogen on collagen synthesis remain controversial. Studies have shown mixed results regarding estrogen's impact on collagen synthesis; i.e., while some indicate a negative impact,[10,11] others suggest either positive effects[12,13] or no significant effect.[14,15]

One study demonstrated that high estrogen levels increase collagen synthesis in three-dimensional (3D) ligaments engineered from human anterior cruciate ligament (ACL) cells.[13] However, the aforementioned study also reported that elevated estrogen inhibited the activity of lysyl oxidase, an enzyme responsible for collagen cross-linking, which reduced ligament stiffness.

On a related note, elevated estrogen levels have been linked to increased knee joint laxity. This phenomenon is observable even during the menstrual cycle, where fluctuations in estrogen correspond to changes in knee laxity.[16] Additionally, estrogen and progesterone together increase the expression of relaxin receptors in ligaments, further contributing to joint laxity. These factors may explain why ACL ruptures are significantly more common in female soccer players compared to their male counterparts,[17,18] as well as the inferior functional recovery after ACL reconstruction or meniscus surgery.[19,20] A further point of discussion is the lasting effects of these hormonal changes. Several reports have emphasized that passive stabilization of the knee may not recover fully at an evaluation made at four months postpartum, instead of a traditionally accepted six to 12 weeks.[21] Moreover, whether static sagittal and rotational stability ever return to baseline remains debatable.

In tendons, estrogen increases the fractional synthesis rate of collagen measured by proline isotope uptake in tendons, rather than increasing free collagen synthesis as indicated by N-terminal peptide of procollagen I in blood.[22] This suggests that estrogen primarily enhances collagen incorporation into tendons rather than promoting overall collagen production. However, animal studies have shown that lower estrogen levels result in reduced collagen synthesis in tendons.[23] Estrogen also increases insulin-like growth factor 1, which promotes collagen synthesis in tendons.[24,25] Additionally, estrogen reduces tendon stiffness by inhibiting lysyl oxidase activity, which may explain the lower incidence of tendon injuries among females.[13]

Hormonal changes and osteoarthritis (OA)

Women are at higher risk of developing OA, particularly after menopause, suggesting a potential relationship between hormonal changes and OA pathogenesis.[26,27] However, contemporary data remains divided as to whether estrogen has a protective or detrimental effect on cartilage health and OA. A cross-sectional study reported an association between higher estradiol levels and an increased prevalence of radiographic knee OA.[28] Conversely, two cohort studies identified lower estradiol levels as a risk factor for the development of radiographic knee OA[29] and for requiring knee arthroplasty.[30]

At the molecular level, estrogen and its receptors appear to play a critical role in OA pathogenesis. Estrogen-related receptor (ERR)-alpha has been shown to influence chondrocyte proliferation differently from ERR-gamma. Yet, both receptors contribute to cartilage degradation, osteophyte formation, and synovitis through inflammatory cytokines such as interleukin-1β, interleukin-6, and matrix metalloproteinase-13.[31] Despite these conflicting findings, it is undeniable that hormones significantly affect cartilage and may either protect against or contribute to the development of OA.

The effects of sex hormones on the musculoskeletal system remain a topic of debate for several reasons. First, each tendon, ligament, and joint have unique mechanical properties; therefore, it is difficult to generalize findings from regional studies to the entire body. Second, differences in sex hormones, their effects, and the menstrual cycle across species limit the applicability of animal study results to humans. Nevertheless, it is evident that sex hormones significantly impact the mechanical properties and composition of musculoskeletal tissues, particularly during pregnancy.

IMPACT OF PREGNANCY ON THE MUSCULOSKELETAL SYSTEM

Pregnancy is a temporary, but transformative condition that induces significant anatomical and physiological changes across various systems. Among these, the musculoskeletal system is one of the most affected, as it must adapt to support the additional weight of the fetus, enlarged breasts, and the growing gravid uterus, amounting to a total weight gain of 11.3 to 15.9 kg for women with a body mass index (BMI) of 18.5 to 25 kg/m^2.[32]

This increased abdominal weight causes anterior shift in the center of gravity, leading to a compensatory increase in lumbar lordosis. Lumbar lordosis increases significantly between the 17th-24th and 25th-32nd weeks of pregnancy, with a slight but non-significant increase persisting up to the fourth month postpartum. This indicates that spinal curvature has not yet returned to pre-pregnancy levels; however, longer-term prospective studies are needed to determine whether this alteration eventually normalizes after the postpartum period. A significant difference in lumbar lordosis was observed between nulliparous and multiparous women at 16 weeks of gestation, with greater curvature noted in multiparous women. Parity appeared to influence lordotic curvature during the final eight weeks of pregnancy: lumbar lordosis increased in multiparous women but decreased in nulliparous women. This finding is particularly noteworthy, as neither body weight nor ligamentous laxity, two factors typically associated with postural adaptations, differed significantly between women experiencing their first pregnancy and those with previous pregnancies.[33] Additionally, the increased breast mass contributes to greater thoracic kyphosis. To maintain pulmonary function, the rib cage expands laterally, increasing the circumference of the chest. These changes lead to the overstretching of the upper back muscles and shortening of the pectoral muscles, resulting in rounded shoulders during pregnancy.[6]

Additionally, the lower limbs are significantly impacted by pregnancy, as they exhibit kinetic chain characteristics where changes in one joint affect other joints. In the context of increased ligamentous laxity, the heights of the transverse and longitudinal arches of the foot decrease, leading to foot pronation and compensatory tibial internal rotation.[6,34] The pelvis tilts anteriorly to compensate for this tibial rotation and the increased flexion moment at the hip. To counterbalance the anteriorly added mass, the hip extensors and plantar flexors are overloaded to maintain postural stability.[6]

In terms of gait, pregnancy is associated with decreased stride length and increased step width. The latter increases the base of support, enhancing stability during gait. These alterations in bone alignment and muscle function are the primary contributors to the widespread neuromusculoskeletal pathologies observed in pregnant women.[6,35] While several studies have demonstrated these alterations during pregnancy, the more critical question remains as to whether they persist postpartum, potentially leaving women more susceptible to musculoskeletal disorders over their lifetime.

TABLE I
An overview of common musculoskeletal problems experienced during pregnancy

Disease/problem	Symptoms/significance	Diagnosis	Treatment
Low back pain	Pain affecting daily activities and sleep, experienced by 50-80% of women	Clinical evaluation, MRI if no pain relief	Rest, exercise, physical therapy, acetaminophen
Transient osteoporosis of pregnancy	Acute onset of hip pain during weight-bearing activities, typically in the third trimester	MRI to differentiate from avascular necrosis	Self-limiting condition, bed rest, acetaminophen
Diastasis of rectus abdominis	Increased abdominal gap >2.5 cm, overstretching of abdominal muscles	US to assess muscle gap and rule out hernias	Abdominal strengthening and pelvic exercises
Carpal tunnel syndrome	Pain, paresthesia, weakness, sensory loss	Clinical evaluation, US, electromyography	Wrist splints, exercises, physical therapy, injections, possible surgery in severe cases
De Quervain's tenosynovitis	Wrist/thumb pain, inflammation of the first dorsal compartment tendons	Clinical evaluation, US	Splinting, physical therapy, steroid injections for severe cases
Osteitis condensans ilii	Pelvic pain, sclerotic areas on iliac sides of sacroiliac joints	MRI to differentiate from inflammatory sacroiliitis	Acetaminophen and NSAIDs if appropriate postpartum, physical therapy
Joint laxity (hypermobility)	Increased joint motion, frequent ankle/knee sprains	Beighton score, extensometers, arthrometers, goniometers	Injury prevention, supportive care

MRI: Magnetic resonance imaging; US: Ultrasound; NSAID: Nonsteroidal anti-inflammatory drug.

COMMON MUSCULOSKELETAL PROBLEMS

Physicians encounter numerous neuromusculoskeletal problems due to the aforementioned hormonal and anatomical changes, as well as increased joint laxity during pregnancy. These issues encompass well-known pregnancy-related musculoskeletal disorders, such as low back, pelvic, and hip pain, diastasis of the rectus abdominis, peripheral neuropathies, tendon/ligament injuries, osteitis condensans ilii, and arthralgia/arthritis (Table I).

Diagnosis in musculoskeletal problems relies heavily on imaging, and pregnant patients pose particular difficulties in this aspect. Therefore, before defining these aforementioned conditions, key decision points in imaging should be made clear for diagnosis, particularly in the trauma setting. Although plain radiographs and computed tomography (CT) are mostly relied on in the emergency setting, imaging modalities free of ionizing radiation should be the first priority in pregnant patients. Such studies should be reserved for scenarios in which the result would alter immediate management; i.e., high-energy fractures, neurovascular compromise, unstable dislocations or a possible malignancy; after confirming that ultrasound or magnetic resonance imaging (MRI) cannot adequately answer the clinical question. Guidelines emphasize that single-region extremity radiographs or low-dose CT scans typically expose the fetus to <5 mGy, well below the cumulative 50 mGy teratogenic threshold with utilization of abdominal shielding and tight collimation.[36] It should still be kept in mind that no thresholds can be determined for stochastic effects such as childhood leukemia or solid tumors, and the fetus is the most radiosensitive during the first trimester. Therefore, “As low as reasonably achievable (ALARA)” principles should be kept paramount.[37]

Low back pain is experienced by 50 to 80% of women during pregnancy and can interfere with daily activities and disrupt sleep. It is primarily induced by altered mechanical effects, including increased lordosis and laxity of spinal ligaments. Notable risk factors include multiparity and younger maternal age. Conservative treatment remains the main approach, including rest, exercise, hot/cold therapy, and acetaminophen. Physical therapy including trunk-strengthening programs should be considered, if significant pain persists beyond six weeks.[38] If pain relief is not achieved, further imaging should be conducted.[39]

Hip pain, unless referred from the low back or pelvis, may result from transient osteoporosis of pregnancy (TOP) or avascular necrosis of the femoral head. Although hip is the most affected joint in TOP, involvement of the knee and talus has also been reported. Vascular, hormonal, and neural factors contribute to the etiology, with the primary complaint being hip pain during weight-bearing activities. The main treatment for this self-limiting condition is bed rest. Activity modulation in transient osteoporosis of the hip aims maintaining hip mobility and peri-articular muscle strength while the bone is protected. Although a non-weight-bearing period is critical, further consideration must be given to aquatic exercises early on, and a gradual strengthening program with protected ambulation using crutches once pain eases, to speed safe functional recovery.[40] Avascular necrosis of the femoral head is another potential cause of hip pain during weight-bearing. In differential diagnosis, MRI can be utilized, and anti-inflammatory medications, if appropriate, along with weight-bearing avoidance are considered for treatment.[39,41]

Diastasis of the rectus abdominis (DRA) refers to increased gap (>2.5 cm) between the two heads of the rectus abdominis muscle; i.e., gaps greater than those usually accepted as abnormal.[39] The pushing effect of the additional weight from the gravid uterus and the expanded rib cage causes overstretching of the abdominal muscles, potentially leading to DRA. Imaging modalities, particularly ultrasound, can be employed to rule out other pathologies, such as ventral hernias, and to measure the gap. Abdominal strengthening and pelvic exercises are the preferred treatment options except extreme cases.[39] Diastasis recti wider than 3 cm that fails to close after six to 12 months of targeted core rehabilitation can be evaluated for open or laparoscopic midline plication.[42]

Peripheral neuropathies, particularly carpal tunnel syndrome (CTS), affect approximately 31 to 62% of pregnant women, predominantly due to increased fluid retention during pregnancy.[43] Major complaints include pain, paresthesia, weakness, and sensory loss. Treatment may consist of wrist splints, exercises, physical therapy, and injections as necessary. Although majority of CTS in pregnancy is expected to resolve with standard conservative treatment strategies in the first month postpartum,[44] there are reports that it may persist for up to three years, underscoring the importance of appropriate treatment during the acute phase.[39,45] If conservative options fail and there is evidence of persistent, or recurrent nerve compression, surgical intervention should be considered, particularly when there is motor deficit.[46] However, local anesthetics with use of tourniquet should be favored and the necessary precautions related with pregnancy should be taken.[47,48]

De Quervain's tenosynovitis involves inflammation of the tendons in the first dorsal compartment (abductor pollicis longus and extensor pollicis brevis). Fluid retention and hormonal effects on collagen contribute to the pathophysiology.[49] Moreover, in the early postpartum period, the physical demands and repetitive motions while caring for the newborn can further exacerbate the risk.[50] For this self-limiting condition, splinting is often effective, while steroid injections may be reserved for more severe cases.[39,51] If splinting and activity modification fail, physical therapy including ergonomic coaching and targeted tendon-gliding therapy can also be considered, with surgery being reserved for refractory cases.[51]

Osteitis condensans ilii is a radiological term describing bilateral pelvic triangular sclerotic areas on the iliac sides of the sacroiliac joints. Contributing factors include additional weight in the abdomen and pelvis, as well as overloading during childbirth. It is crucial to differentiate this condition from inflammatory sacroiliitis. In osteitis condensans ilii, the sacroiliac joint space remains preserved, and no additional systemic complaints are typically observed, unlike in inflammatory sacroiliitis. The MRI can assist in confirming the diagnosis. As this condition predominantly occurs in the postpartum period, analgesics and physical therapy are often employed for treatment.[39]

Joint laxity or hypermobility refers to a condition in which a joint permits more motion than is considered normal. To measure joint laxity, the Beighton score system or tools such as extensometers, arthrometers, or goniometers can be utilized. In early childhood, joint laxity peaks and then declines rapidly into adulthood. However, in females, this decline may be interrupted or even reversed during pregnancy.[7] Pregnant women frequently seek outpatient care for ankle and knee sprains, as well as ligament injuries. Several studies have indicated that approximately 21% of injuries during pregnancy involve the ankle and 16% the knee.[52] Consequently, healthcare providers should be vigilant in assessing and managing these conditions to ensure maternal health and mobility.[53] Emergency ligament surgery should be limited to mechanical problems such as locked knee due to meniscus tears, multi-ligament injuries, or unstable ankle fracture-dislocations. Elite athletes or other critical workers whose instability remains uncontrolled by bracing may be considered for reconstruction surgery under regional anesthesia with continuous fetal monitoring.[54] In absence of data from large databases, such tiered algorithm can be considered reassuring where well-selected ligament reconstructions can be carried out safely in the peripartum period whenever maternal function or long-term joint preservation is genuinely at stake. Increased joint laxity during pregnancy is attributed to hormonal changes, particularly to relaxin. Relaxin is well-known for inducing ligamentous relaxation, thereby playing a crucial role in preparing the body for childbirth. However, studies investigating the potential relationship between hormonal changes and laxity have not revealed statistically significant correlations.[55,56]

Several studies have explored when the laxity begins to increase and peaks during pregnancy. According to previous studies, it remains stable in the first trimester, begins to increase in the second trimester, and peaks in the third trimester.[7,55,56] This pattern appears to correlate with serum hormone levels, suggesting that it is most likely associated with the cumulative effects of these hormones.[56] However, Dumas and Reid[57] demonstrated that knee laxity reached its maximum value during the first half of pregnancy and varied little throughout the rest of the pregnancy. Presenting a different outtake, Schauberger et al.[55] reported increased knee laxity during pregnancy, peaking at two weeks postpartum and decreasing slightly by six weeks postpartum. Similarly, Marnach et al.[56] and Lindgren and Kristiansson[58] corroborated these observations at six and thirteen weeks postpartum, respectively. Furthermore, Opala-Berdzik et al.[59] demonstrated that trunk flexibility increases during pregnancy, with a marked increase noted at six months postpartum. Although one study reported persistent laxity changes postpartum, the findings were conflicting, indicating that anterior knee laxity increased while posterior/coronal plane laxity decreased significantly.[21]

It is also essential to identify whether joint laxity is associated with complaints experienced by pregnant women. Two studies have yielded controversial results regarding the relationship between joint laxity and back pain. One study indicated that increased finger joint laxity and the number of previous pregnancies were associated with back pain during early pregnancy and persisting after childbirth.[58] Conversely, Ostgaard et al.[60] observed no significant correlation between joint laxity and back pain. Damen et al.[61] reported increased sacroiliac joint laxity during pregnancy as expected. More intriguingly, they found that pregnancy-related pelvic pain was not determined solely by the magnitude of sacroiliac joint laxity, but rather by the asymmetric laxity of the sacroiliac joints. Another study revealed significant decrease in arch height and arch rigidity index, accompanied by increase in foot length and arch drop, though the potential relationship between these findings and foot complaints was not investigated.[5]

It is also of utmost importance to consider whether the number of pregnancies influences laxity during pregnancy. Although the current literature offers limited data on this topic, the available studies present conflicting findings. Calguneri et al.[62] reported a significant difference in laxity at the end of pregnancy between women in their first and subsequent pregnancies, suggesting that parity may play a role in laxity progression. In contrast, Ostgaard et al.[60] observed a difference between nulliparous and multiparous women at 12 weeks of gestation, which later disappeared as pregnancy progressed, indicating a transient rather than sustained effect of parity. Furthermore, a study by Dumas et al.[33] found no significant difference in laxity between the two groups at any time point; however, their data collection began at 17 weeks of gestation, leaving the early pregnancy period unexamined. As such, their results do not necessarily contradict those of Ostgaard et al.[60] Of note, neither group showed a significant difference in the postpartum period, and both exhibited some recovery. These findings may suggest that ligamentous laxity increases more rapidly in subsequent pregnancies than in first pregnancies, rather than indicating a permanent increase in laxity following the first pregnancy.

Based on current literature, it is evident that most joints are affected by pregnancy in terms of laxity, leaving them more susceptible to injuries. Additionally, these joints may be sources of pain during pregnancy. Further studies are needed to identify which joints are affected during pregnancy, as specialized exercise programs or other preventive measures could be implemented before/during pregnancy for each joint. To illustrate, strengthening exercises for the plantar intrinsic muscles should be emphasized to mitigate decreases in arch height, and the use of arch-supporting orthoses should be considered. To prevent falls and other joint or ligament injuries caused by mechanical changes during pregnancy, balance, coordination, and proprioception exercises can be initiated.

FUTURE PERSPECTIVES

Given the complex pathophysiology of musculoskeletal disorders and the additional pregnancy-related challenges, it is imperative that gynecologists, physiatrists, and orthopedic surgeons promptly collaborate. Gynecologists are often the first point of contact for pregnant women and play a crucial role in the early recognition of musculoskeletal symptoms and risk factors. Their awareness of biomechanical and hormonal influences on the musculoskeletal system is key to timely referral. Physiatrists bring expertise in conservative management strategies such as posture correction, therapeutic exercise, activity modification, and pain management techniques including physical modalities and targeted injections. Their role is central in preventing chronicity and optimizing physical function throughout pregnancy and after delivery.

Orthopedic surgeons may become involved when structural abnormalities, such as spinal disc herniation, severe joint instability, or exacerbation of pre-existing musculoskeletal conditions, require specialized imaging, diagnosis, or surgical consultation. Although surgical interventions are rarely indicated during pregnancy, orthopedic input is valuable for postpartum follow-up and long-term structural assessment.

Effective communication and coordination between these specialties ensures that pregnant patients receive comprehensive, individualized care. This approach facilitates early intervention, reduces the risk of chronic pain syndromes, enhances postpartum recovery, and supports a faster return to normal daily activities. Furthermore, multidisciplinary collaboration is essential in developing and implementing clinical pathways, patient education programs, and long-term follow-up strategies which address both acute and persistent musculoskeletal concerns in the perinatal population.

It is evident that, due to the risks associated with radiation exposure from X-rays and CT, the extended examination times and the potential safety concerns of MRI during the first trimester,[63] ultrasound emerges as the preferred imaging modality for diagnosing musculoskeletal disorders in pregnancy.[64] That being said, physiatrists and orthopedic surgeons can utilize ultrasound as both a diagnostic and therapeutic tool for pregnant women, as localized treatment is inherently preferred over systemic treatment to minimize potential risks to the fetus.

Obviously, long-term effects of pregnancy on the female musculoskeletal system are insufficiently understood, with limited evidence available. To date, investigations have usually examined pregnant women only through the sixth month postpartum; however, longer-term prospective studies are needed to provide valuable insights into whether pregnancy-related musculoskeletal changes persist throughout life or eventually revert to baseline. Furthermore, it remains unclear whether multiple pregnancies render women more susceptible to tendon, ligament, or joint problems in the long term; therefore, long prospective studies are required to assess how the musculoskeletal system is affected after each/every pregnancy and whether the changes are temporary or permanent.

In conclusion, pregnancy imposes significant burden on the musculoskeletal system. Collaboration of relevant disciplines is noteworthy in clinical practice for prompt diagnosis and optimal management. Nevertheless, further research is indisputably needed for providing better insight into understanding these problems in the long term.

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