

Alleviating back pain in pregnancy: The role of collagen supplementation

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ABSTRACT

Background: Lower back pain (LBP) is a common and disabling complaint during pregnancy, particularly in the second and third trimesters. Changes in biomechanics, hormones, and neuromuscular function contribute to the development of LBP. Collagen, a major component of connective tissue, plays a crucial role in maintaining tissue integrity and may help alleviate musculoskeletal pain. **Material and Methods:** This review aims to evaluate the potential role of collagen supplementation in reducing lower back pain in pregnant women by summarizing current biochemical and clinical evidence. A comprehensive literature search was conducted to identify peer-reviewed articles published in English or Polish that investigated collagen supplementation or its physiological effects related to musculoskeletal health, especially in pregnancy. Non-human studies, case reports, and editorials were excluded. Relevant full texts were reviewed for data on collagen's mechanism of action, safety, and clinical outcomes in pregnant populations. **Results:** Hydrolyzed collagen peptides exhibit high bioavailability and promote fibroblast activity, extracellular matrix synthesis, and modulation of inflammatory pathways. These effects support tissue repair and joint stability, which are critical during pregnancy-related biomechanical stress. Although evidence from direct clinical trials on pregnant women is limited, studies in other populations suggest collagen supplementation may reduce musculoskeletal pain and improve function. No significant safety concerns have been reported. **Conclusion:** Collagen supplementation appears to be a promising adjunctive strategy for managing pregnancy-related lower back pain. However, well-designed clinical trials are needed to confirm its efficacy and safety in pregnant women.

Key words: Collagen, Lower back pain, Lumbopelvic pain, Pregnancy

INTRODUCTION

Spinal pain, particularly in the lumbar region, is a common complaint among pregnant women. The pathophysiology multifactorial etiology, including biomechanical and hormonal changes, as well as neuromuscular adaptations associated with pregnancy. Moreover, the posture, center of gravity and spinal curvatures change over the course of pregnancy. Lower back pain (LBP) is defined as pain and/or

discomfort located between the 12th rib and the gluteal fold [1]. According to a recent study up to 86% of women experience lumbopelvic pain during the third trimester of pregnancy [2,3]. The severity of pain usually reaches its peak between 24th and 36th weeks of gestation [1]. Hydrolyzed collagen supplements, which contain bioactive peptides with high absorption and bioavailability, have gained popularity in recent years as a nutritional strategy to support joint health and tissue repair [4]. Several studies in non-pregnant populations

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have demonstrated that collagen supplementation may improve skin elasticity, reduce joint pain, and promote cartilage regeneration [5,6]. In particular, collagen peptides have been shown to stimulate fibroblast activity, enhance extracellular matrix production, and exert anti-inflammatory effects—all of which are potentially beneficial in the context of pregnancy-induced tissue strain [7]. Despite its high prevalence and considerable impact on maternal quality of life, the physiological and biomechanical mechanisms contributing to pregnancy-related low back and pelvic pain are not yet fully understood [2]. This review aims to summarize current knowledge on the role of collagen in pregnancy, explore the potential mechanisms by which collagen supplementation may alleviate back pain, and highlight areas for future research.

MATERIAL AND METHODS

A comprehensive literature search was conducted to identify relevant studies on collagen supplementation and its effects on lower back pain (LBP), lumbopelvic pain, and connective tissue changes during pregnancy. Electronic databases including PubMed, Scopus, Web of Science, and Google Scholar were searched from inception through May 2025. Search terms included combinations of keywords such as collagen, hydrolyzed collagen, collagen peptides, pregnancy, lower back pain, lumbopelvic pain, and pregnancy-related pain. References of included articles and relevant reviews were hand-searched to identify additional pertinent studies. Articles were included if they were published in peer-reviewed journals and available in English or Polish. Following an initial screening of titles and abstracts for relevance, the full texts of selected articles were reviewed to identify those containing substantial data.

COLLAGEN: FROM MOLECULAR STRUCTURE TO TISSUE INTEGRITY

Collagen (COL) is one of the most widespread and abundant structural proteins [8]. Nearly 30 distinct types of collagen have been identified in nature (Table I). The collagen molecule exhibits a quaternary protein structure. It is predominantly composed of three amino acids: glycine, proline, and hydroxyproline. These amino acids polymerize into chains of approximately 1,000 residues, forming alpha chains. A single collagen molecule consists of three such alpha chains twisted

into a left-handed triple helix, with one complete turn occurring every three amino acids (Fig. 1). The structural stability of the collagen molecule is largely attributed to intramolecular hydrogen bonds formed between glycine residues in adjacent chains [9,10]. Thus, glycine is absolutely essential in every third position for the proper formation of the triple helix, due to its minimal side chain [11].

Collagen is synthesized by various types of connective tissue cells, including, among others, fibroblasts (predominant in soft tissues), osteoblasts (in bone), and chondrocytes (in cartilage) and is a key component of the extracellular matrix (ECM) and is present in connective tissue, skin, ligaments, bones, cartilage, and numerous other tissues. Additionally, it comprises about 65–80% of the dry weight of tendons. The formation of collagen cross-links is fundamental to the biomechanical properties of tendons, as it enhances their capacity to resist high tensile and shear forces [13-16].

FROM COLLAGEN TO PEPTIDES: CLARIFYING THE NOMENCLATURE

The terms hydrolyzed collagen (HC) and collagen peptides (CP) are frequently used interchangeably, though they refer to closely related, yet distinct, forms of processed collagen. Hydrolyzed collagen describes collagen that has been subjected to hydrolysis—a process that breaks down the native protein into smaller peptide chains. In turn, the end products of this process are known as collagen peptides, which are defined by their low molecular weight and enhanced bioavailability. This structural modification improves their digestibility and facilitates rapid absorption in the human gastrointestinal tract [9,21].

INFLAMMATORY MEDIATORS AND NOCICEPTOR SENSITIZATION IN JOINT PAIN

The body initiates an inflammatory reaction in response to tissue damage or infection, engaging a coordinated biological process involving the autonomic, somatosensory, vascular and immune systems. Key inflammatory agents - such as prostaglandins, pro-inflammatory cytokines, and chemokines - contribute to pain generation by directly stimulating nociceptors, which are primary sensory neurons specialized in

Table 1: Classification of Collagen Types and Their Distribution in Human Tissues, based on current structural and functional literature [17-20].

Class	Collagen Types	Primary Locations and Functions
Fibrillar collagens	I, II, III, V, XI	Type I: skin, tendons, bones, ligaments, cornea Type II: hyaline cartilage Type III: skin, blood vessels, internal organs Type V: skin, placenta, muscle fibers, bones Type XI: cartilage, vitreous body of the eye
Network-forming collagens	IV, VIII, X	Type IV: basement membranes (e.g., kidney, skin) Type VIII: corneal endothelium Type X: hypertrophic cartilage, endochondral ossification
FACIT (Fibril-Associated Collagens with Interrupted Triple Helices)	IX, XII, XIV, XIX, XXI	Type IX: cartilage, interacts with type II collagen Type XII: tendons, ligaments Type XIV: skin, tendons Type XIX: skeletal muscles Type XXI: tendons, ligaments
Multiplexing	XV, XVIII	Type XV: vascular basement membranes Type XVIII: basement membranes, precursor of endostatin
MACIT (Membrane-Associated Collagens with Transmembrane Domains)	XIII, XVII, XXIII, XXV	Type XIII: neuromuscular junctions Type XVII: hemidesmosomes in skin Type XXIII: various tissues Type XXV: brain
Beaded filament-forming collagens	VI	Type VI: skeletal muscle, skin, tendons
Anchoring fibrils	VII	Type VII: dermal-epidermal junctions
Other/less characterized types	XX, XXII, XXIV–XXVIII	Type XX: poorly defined function Type XXII: myotendinous junctions Type XXIV: bone Type XXV: brain Types XXVI–XXVIII: various tissues, functions under investigation

detecting harmful stimuli [22,23]. Cytokines represent a group of signaling proteins that play a pivotal role in the regulation and amplification of inflammatory processes, including the development of inflammatory pain. Under normal physiological conditions, cytokines are present at low levels throughout the body. However, during inflammatory responses, their concentrations can increase significantly, even by several orders of magnitude. These molecules are predominantly secreted by activated immune cells such as B lymphocytes, T lymphocytes, macrophages, and neutrophils. Nonetheless, non-immune cells, including endothelial cells and fibroblasts within the joint environment, may also contribute to cytokine production. Key proinflammatory cytokines, such as interleukin-6 (IL-6), interleukin-1 beta (IL-1 β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α) may increase the excitability of nociceptors located in the joint or promote the synthesis of prostaglandins, which enhance the transmission of pain signals [24].

THERAPEUTIC EFFECTS OF COLLAGEN SUPPLEMENTATION: A REVIEW OF CURRENT EVIDENCE

Maysa Alves Rodrigues Brandão-Rangel et al. conducted a study to evaluate the ability of collagen peptides to

stimulate the proliferation and activation of human fibroblasts and keratinocytes. The study also assessed their potential to suppress an inflammatory response induced by lipopolysaccharide (LPS), a known pro-inflammatory agent. Cells were treated with LPS (10 ng/mL) in combination with three increasing concentrations of collagen peptides. The parameters analyzed included cell proliferation, expression of pro-collagen-1 α by fibroblasts, and the secretion of key pro-inflammatory cytokines - interleukin-1 β , interleukin-6, interleukin-8 (IL-8), and tumor necrosis factor alpha (TNF- α) - by both cell types. The results demonstrated a significant increase in the proliferation of both fibroblasts and keratinocytes across all collagen peptide concentrations. However, only the highest dose (10 mg/mL) led to a notable increase in pro-collagen-1 α expression in fibroblasts, a precursor molecule in the process of collagen synthesis. In addition, collagen supplementation significantly reduced the secretion of IL-1 β , IL-6, IL-8, and TNF- α in fibroblasts. A similar anti-inflammatory effect was observed in keratinocytes, with marked reductions in LPS-induced IL-1 β , IL-6, IL-8, and TNF- α secretion. These findings confirm that collagen peptides not only exert anti-inflammatory effects but also promote the proliferation and activation of skin fibroblasts and keratinocytes, supporting their potential application in regenerative and dermatological therapies [7]. In order to verify the possible beneficial effects of undenatured type II

collagen in monosodium iodoacetate (MIA)-induced (osteoarthritis) OA, young and old rats were tested. In both age groups, all used doses of undenatured collagen reduced knee diameter. Moreover, the higher doses reduced the Mankin score, a histological scoring system designed to evaluate the severity of OA in articular cartilage. In addition, gait measurements were also improved compared to those of the MIA rats. The levels of most inflammatory and cartilage breakdown markers (i.e. IL-1 β , IL-6, TNF- α , etc.) both in serum as well as in knee were reduced, suggesting the efficacy of undenatured collagen in improving overall joint health in rats [25]. Anti-inflammatory action of collagen peptides has been studied in the Chinese giant salamander (CGS), one of the largest salamanders and amphibians in the world. In this study, skin collagens were extracted from the skin of CGS and tested using lipopolysaccharide (LPS)-induced RAW264.7 cells, a widely used mice macrophage cell line. As a result, the expression of inflammatory cytokines: IL-6, IL-1 β , TNF- α , chemokines were inhibited in these cells. It indicated that skin collagens may regulate macrophage cells [26]. There is a growing body of evidence supporting the use of hydrolyzed collagen supplementation for the relief of joint pain. P. Benito-Ruiz et al. conducted a randomized, double-blind, controlled trial involving 250 patients diagnosed with knee osteoarthritis. The participants supplemented with 10 grams of hydrolyzed collagen daily for a period of six months. Pain assessment using the Visual Analogue Scale (VAS) and the pain subscale of the WOMAC questionnaire demonstrated a significant reduction in osteoarthritic pain. Additionally, the most pronounced effects were observed in patients with the most advanced joint degeneration and the lowest habitual intake of meat protein [27]. Similar conclusions were reached by Chen CC et al. A group of 160 patients diagnosed with grade I–III knee osteoarthritis, based on the Kellgren–Lawrence criteria, and experiencing joint pain for at least three months, were randomly assigned with equal probability to one of four groups. Over a 24-week period, each group received one of the following interventions: hydrolyzed collagen type II (HC-II), essence of chicken combined with hydrolyzed collagen type II (EC-HC-II), glucosamine HCl, or placebo. The intervention was combined with resistance training. The present study discusses only the findings related to HC-II, which fall within the thematic scope of this paper. The results demonstrated that supplementation with HC-II significantly reduced pain intensity, as measured by the VAS, after just 14 days, to a greater

extent than placebo. In contrast, the EC-HC-II group showed potential benefits in improving fat-free mass and muscle strength, suggesting that EC-HC-II may represent a novel, comprehensive approach to supporting mobility by enhancing joint, muscle, and bone health in older adults [28]. In another randomized, double-blind, placebo-controlled clinical trial, the effects of collagen peptide supplementation on symptoms of knee osteoarthritis in elderly women were evaluated. Elderly women diagnosed with knee osteoarthritis were enrolled and randomly assigned to one of two groups. Participants in the intervention group were supplemented with bovine-derived collagen peptides (Peptan® B 2000, Rousselot), whereas those in the placebo group received maltodextrin. The observation period lasted six months. Outcomes were assessed at baseline, after three months, and after six months. The results demonstrated that collagen peptide supplementation led to a statistically significant reduction in perceived knee joint pain. To also evaluate the safety of collagen peptide use in patients, liver and kidney function parameters were assessed in blood and urine samples at baseline, three months, and six months. These parameters were within normal ranges in all participants prior to the intervention. After six months, the collagen peptide group showed a slight but significant improvement in liver parameters and blood urea nitrogen levels. A small increase in creatinine was observed in both the placebo and collagen groups, confirming the safety of administering 8 grams of collagen peptides daily over a six-month period [29].

PATHOPHYSIOLOGY OF LOWER BACK PAIN DURING PREGNANCY

Two large population studies conducted on pregnant women indicated that early menarche (≤ 13 years old), maternal age < 35 years old, high body mass index (≥ 25 kg/m²), the presence of LBP before the first pregnancy, emotional distress, physically demanding work and the use of oral contraceptives increased the risk of LBP during pregnancy [30,31]. Lack of exercise during pregnancy has also been suggested as a potential risk factor for LBP in late pregnancy [32]. It has been hypothesized that hormones such as estrogen, progesterone and relaxin are potential contributors to LBP development and intensity. Relaxin levels rise during the first trimester of pregnancy and play a crucial role in childbirth by promoting the relaxation of spinal and pelvic ligaments and joints to facilitate

delivery. It may predispose joints to non-traumatic injury by increasing peripheral ligament laxity and triggers spinal and pelvic instability, potentially causing pain [2,33,34]. However, a systematic review by Aldabe et al. reported inconsistent associations between relaxin levels and pregnancy-related LBP, highlighting the overall insufficient quality of the available evidence [35].

Studies on estrogen and progesterone effects on spinal pain are inconsistent. Despite estrogen's role in decreasing the fibroblasts proliferation and synthesis of procollagen, possibly increasing ligament laxity, a study conducted by Kristiansson et al. found no difference in estrogen levels between women reporting pelvic pain during pregnancy and those without such pain. The study also examined progesterone, a hormone involved in the relaxation of all smooth muscles and reported significantly higher progesterone levels among women experiencing spinal pain during pregnancy [36]. Based on current evidence, relaxin, estrogen, and progesterone may contribute to the development and severity of pregnancy-related low back and pelvic pain, however, further research is required to confirm this hypothesis. The most significant biomechanical changes occur in the lower trunk and pelvic regions in response to the increasing fetal load. Most pregnant women experience a small increase in lumbar lordosis and low back pain intensity, predominantly during the second and third trimesters, coinciding with significant fetal growth and hormonal changes [2]. Fetal growth induces significant alterations in body mass distribution and causes an anterior shift of the center of gravity. This shift leads to adaptive postural changes, which may result in increased loading of spinal structures (Fig. 2) [37,38]. However, according to a study conducted by Glinkowski WM et al., there is no significant relationship between posture and back pain during pregnancy, and the increased pain observed in women with greater lordosis and kyphosis angles is not statistically significant [3]. The question of how pregnancy causes back pain remains unresolved and requires further research.

MANAGEMENT AND TREATMENT OF LBP DURING PREGNANCY

Physical therapy focusing on stabilization exercises, particularly targeting the deep trunk muscles and pelvic floor, is strongly recommended. Manual therapy, including joint mobilization techniques, may be beneficial when performed by trained

professionals. The use of pelvic support belts can provide mechanical stability and symptom relief in selected cases. Pharmacological treatment is limited due to safety concerns during pregnancy. Therefore, non-pharmacological methods are prioritized. Patient education regarding posture, ergonomics, and activity modification is also crucial in reducing symptom burden and improving function. Importantly, bed rest and inactivity are discouraged, as they may worsen functional outcomes. A multidisciplinary approach, including physiotherapists, obstetricians, and pain specialists, is advised in more complex or persistent cases [40]. Similarly, Casagrande et al. highlight that a multidisciplinary, non-pharmacological approach is central to effective management. Exercise therapy, particularly targeting the core and pelvic stabilizers, has demonstrated positive outcomes, while pharmacologic interventions are typically limited to acetaminophen, given safety concerns related to non-steroidal anti-inflammatory drugs (NSAIDs) and opioids in pregnancy. Both sources underscore the necessity of early intervention, tailored treatment plans, and patient education to optimize maternal quality of life while minimizing functional disability [39]. Collectively, these guidelines and reviews support a holistic, individualized, and activity-focused treatment model for pregnancy-related LBP.

DISCUSSION

Collagen supplementation during pregnancy has garnered interest due to its potential benefits in supporting maternal tissue integrity and fetal development. Collagen, a primary structural protein, plays a crucial role in maintaining the strength and elasticity of skin, joints, and connective tissues. During pregnancy, the body's demand for collagen increases to accommodate the expanding uterus, growing fetus, and changes in maternal tissues. Emerging studies suggest that hydrolyzed collagen supplementation may enhance skin elasticity and hydration, potentially reducing the incidence of stretch marks and improving overall skin health in pregnant individuals [41]. Additionally, collagen may support joint health, which is particularly beneficial as pregnancy-related hormonal changes can lead to joint laxity and discomfort. However, it's important to note that while these findings are promising, comprehensive clinical trials focusing specifically on collagen supplementation during pregnancy are limited. A study by Tsutsui et al. investigated the expression patterns of fibrillar

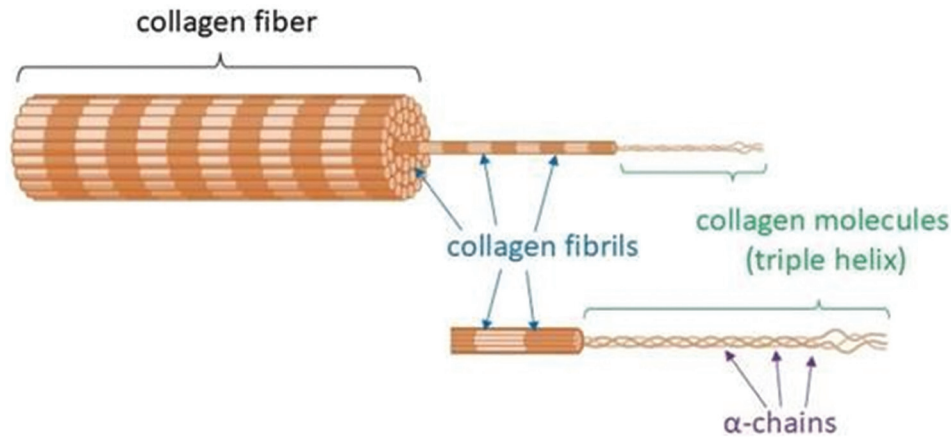


Figure 1: Schematic representation of collagen structure [12].

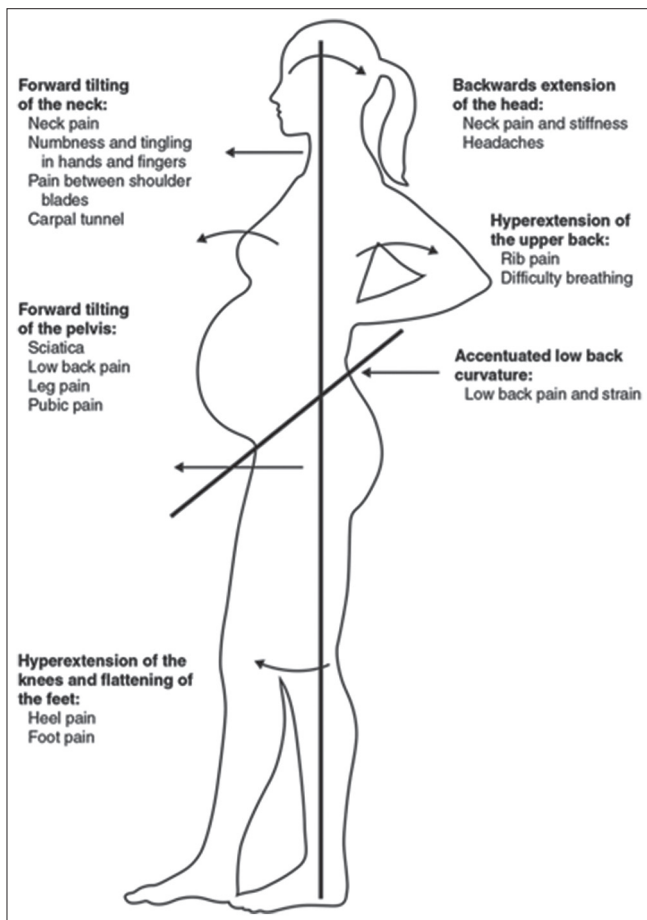


Figure 2: Musculoskeletal compensations during pregnancy [39].

collagen types I, III, and V in the mammary gland during pregnancy, lactation, and weaning in mice. It was observed that collagen type I was predominantly present during pregnancy, suggesting its role in providing structural support during mammary gland development. Collagen type III expression increased during lactation, which may be associated with the

gland's functional changes to facilitate milk production. Following weaning, collagen type V expression was notably upregulated, potentially contributing to tissue remodeling and involution processes. These findings highlight the dynamic regulation of specific collagen types in the mammary gland across different reproductive stages, underscoring their importance in tissue remodeling and function during pregnancy [42]. According to study by Jia-Wei Shi et al. collagen mat play a crucial role in trophoblast adhesion, proliferation, invasion and angiogenesis. Additionally, this study also underscores the critical role of collagen metabolism in cervical remodeling, a process essential for maintaining pregnancy and enabling successful delivery. The cervix must remain structurally intact throughout gestation to support the growing fetus yet undergo profound remodeling at term to permit passage through the birth canal. This paradox is regulated by dynamic changes in the ECM, particularly in collagen composition. Collagen types I and III serve as key structural components, providing tensile strength and elasticity to the cervical tissue. The progression of cervical remodeling is tightly linked to the reorganization of these ECM components. While human in vivo data are limited, findings from rodent models offer valuable insights. These studies demonstrate that collagen type I expression remains stable during early gestation, increases significantly toward term, and rapidly declines postpartum. Such temporal patterns suggest a tightly regulated mechanism of collagen metabolism that facilitates both the maintenance of cervical integrity during pregnancy and the structural transition required for labor. Further investigation in human tissues is warranted to validate these mechanisms and explore potential therapeutic targets for the prevention of preterm birth and cervical insufficiency [43-45].

While these findings collectively support the biological plausibility of collagen's beneficial role during pregnancy, particularly for connective tissue support and inflammation modulation, clinical data specifically targeting pregnant populations remain scarce. Most evidence is derived from studies on joint health in the elderly or osteoarthritis patients. Therefore, further research, especially randomized controlled trials in pregnant women, is essential to determine the efficacy, optimal dosing, and safety profile of collagen supplementation for reducing back pain and supporting connective tissue function during gestation.

CONCLUSIONS

Pregnancy-related lower back pain significantly impacts maternal quality of life and daily functioning. Collagen, as a fundamental component of connective tissues, has demonstrated potential benefits in supporting tissue repair, reducing inflammation, and enhancing musculoskeletal integrity. Current biochemical and clinical evidence suggests that collagen supplementation may help alleviate lower back pain associated with pregnancy by improving joint stability and soft tissue resilience. While direct clinical studies on pregnant women remain limited, available data from related populations indicate a favorable safety profile and potential therapeutic effects. Therefore, collagen supplementation represents a promising, low-risk adjunctive intervention for managing pregnancy-related back pain. Further rigorous, well-controlled clinical trials are necessary to establish definitive efficacy, optimal dosing, and safety parameters specific to pregnant populations.

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The figure 1 has been created in BioRender.com.

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