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# Review of Preclinical and Clinical Studies Supporting the Role of Polydeoxyribonucleotide in the Treatment of Tendon Disorders

Authors' Contribution:
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Tendon disorders are among the most common musculoskeletal conditions, accounting for 30% to 50% of all sports-related injuries. Injured tendons heal slowly and often fail to regain their original structural integrity and mechanical strength, creating significant challenges for physicians. Recently, investigations have reported that polydeoxyribonucleotide (PDRN) plays a key role in promoting tendon healing. For example, preclinical studies indicate that PDRN can enhance tendon repair by inhibiting inflammation and cell apoptosis while promoting collagen production. In clinical studies, the effectiveness and safety of PDRN were also confirmed for managing several conditions, including plantar fasciitis, epicondylitis, Achilles tendinopathy, pes anserine tendinopathy, and chronic rotator cuff disease. In light of these findings, this article aims to review the preclinical and clinical studies that support the role of PDRN in the treatment of tendon disorders. A search was conducted in Medline and PubMed from January 1994 to October 2024 to find relevant research. Ultimately, the review included 3 preclinical studies and 8 clinical studies, involving a total of 318 patients. In conclusion, PDRN is a promising therapeutic option for treating tendon disorders. However, further preclinical and clinical studies are needed to better understand its effects on tendon disorders and to support future clinical applications.

**Keywords:** Polydeoxyribonucleotides • Tendon Injuries • Tendinopathy • Tendons

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## Introduction

Tendon disorders are the most common musculoskeletal conditions that lead individuals to seek medical attention [1]. Tendon disorders vary from acute tendon injuries to chronic tendinopathies [2]. After acute tendon injuries, tendons undergo inadequate and slow healing, resulting in the formation of inferior scar tissue [3]. Moreover, the accumulation of microinjuries during loading, stemming from insufficient tendon healing after acute injuries, is a key factor that predisposes individuals to chronic degenerative tendinopathy [4]. This condition encompasses a wide range of chronic tendon disorders characterized by local pain, stiffness, and physical disability [5].

Current treatment options for tendon disorders can be broadly categorized into surgical and conservative approaches [6]. Conservative treatments are widely accepted as effective options for tendon disorders, as surgical management can lead to complications, such as infection, nerve damage, and scar formation [7]. First-line options for conservative treatment typically include physiotherapy techniques, such as eccentric exercises, extracorporeal shock wave therapy, and therapeutic ultrasound, as well as anti-inflammatory medications, including non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroid injections [8]. Despite advancements in these strategies, the outcomes of conservative treatments have often been unsatisfactory, leading to prolonged symptoms and a notable decrease in the personal productivity of patients [9].

To enhance the quality of tendon healing and functional outcomes, various novel approaches have been developed, among which polydeoxyribonucleotide (PDRN) has been reported to play a crucial role in stimulating the regenerative potential of injured tissue [10,11]. PDRN is a compound formed by deoxyribonucleotide polymers of different lengths ranging from 50 to 2000 base pairs and nucleosides [12]. They have a natural origin, from *Oncorhynchus keta* (chum salmon) and *Oncorhynchus mykiss* (salmon trout) sperm, by a standardized process to purify and sterilize the substance that allows PDRN to display a high proportion of deoxyribonucleic acid (DNA) without active proteins and peptides [13].

Recent studies have highlighted the positive impact of PDRN in treating tendon disorders [14]. For example, intra-articular injection of PDRN provided remarkable pain reduction and function recovery in patients with rotator cuff tendinopathy [15]. In vivo, PDRN inhibited inflammatory response and improved tendon regenerative healing in a rat model of Achilles tendon injury [16]. Furthermore, the systemic review by Bizzoca et al confirmed that PDRN is a promising therapeutic drug in the treatment of tendon disorders [17]. Therefore, in light of these previous findings, in this article, we aim to review the preclinical and clinical studies that support the role of PDRN in the treatment of tendon disorders.

## Material and Methods

### Search Strategy

The search strategy was as follows. (1) For the site search, articles were from PubMed, a database of papers on biomedical science. (2) Database was limited to MEDLINE. (3) Keywords were polydeoxyribonucleotides, tendon injuries, tendinopathy, conservative treatment, musculoskeletal pain. (4) Boolean algorithm was as follows: ("Polydeoxyribonucleotides") OR ("Tendon injuries" OR "Tendinopathy" OR "Conservative treatment" OR "Musculoskeletal pain"). (5) For the retrieval timeframe, we searched the selected in journals published from 1994 to 2024.

### Eligibility Criteria

Articles were included in this study based on the following criteria: (1) patients' pain was induced by tendon disorders; (2) the pain was persisted or increased for more than 1 month; (3) rotator cuff tendinopathy, lateral epicondylitis, and plantar fasciitis were included in this review.

Articles were excluded in this study based on patient age younger than 18 years and older than 65 years.

### Basic Information About Tendon Scar Healing and Tendinopathy

When a tendon is injured, the healing process typically involves the formation of scar tissue, which has characteristics that are inferior to those of intact tendons in terms of gross appearance, histology, and mechanical properties [18,19]. Tendon healing occurs in 3 distinct but overlapping phases: (1) a short inflammatory phase lasting a few days; (2) a proliferative phase lasting several weeks; and (3) a remodeling phase that can extend over several months or even years [20]. During the inflammatory phase, the injury site experiences increased vascular permeability, leading to an influx of erythrocytes and inflammatory cells, including macrophages, platelets, and neutrophils [21]. These cells release growth factors and chemo-attractants for endothelial cells, resulting in the formation of a hematoma [22,23]. After several days, the proliferative phase begins, characterized by the proliferation of tendon cells and the production of type III collagen at the injury site [24]. Finally, in the remodeling phase, the type III collagen synthesized during the proliferative phase is gradually replaced by mechanically stronger type I collagen, helping to restore the tendon's mechanical strength [25]. Despite these healing efforts, the process often fails to achieve complete regeneration of the injured tissue, leading to the formation of biomechanically inferior scar tissue [26]. Furthermore, excessive scar tissue proliferation between the tendon and adjacent tissues can result in adhesions, which hinder normal tendon





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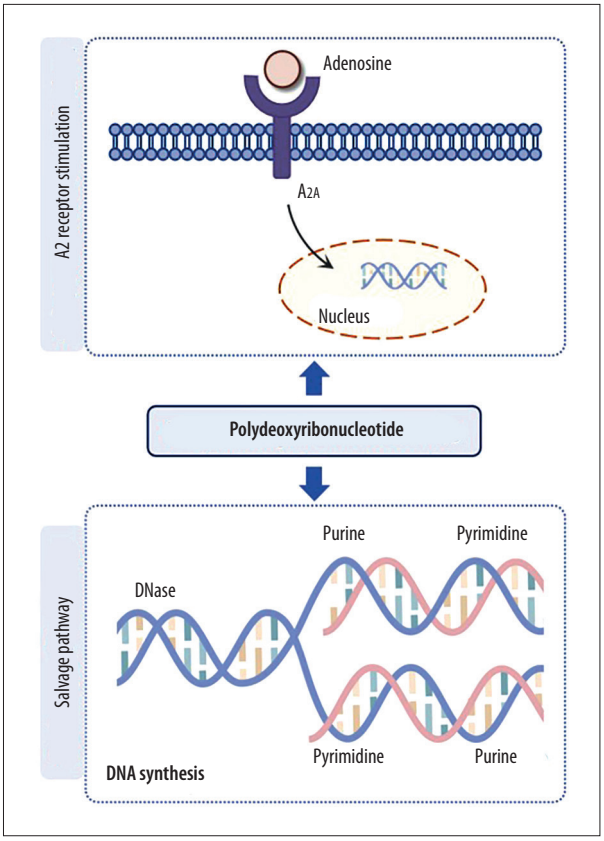


**Figure 1.** Schematic diagram summarizing the basis of polydeoxyribonucleotide (PDRN) efforts: the healing-promoting effect of PDRN appears to be mediated by the activation of adenosine A2A receptor or the "salvage pathway". Following tissue injuries, PDRN activates the adenosine A2A receptor, facilitating a more complete tissue regeneration process. On the other hand, it stimulates the salvage pathway to enhance DNA synthesis and repair, resulting in faster tissue regeneration. DNA – deoxyribonucleic acid; DNase – deoxyribonuclease. *Created with Adobe Photoshop software version 26.2.*

gliding and function [27]. If a tendon continues to experience microdamage after inadequate regenerative healing, local cells in the tendon tissue can undergo pathological changes, ultimately leading to the deterioration of tendon tissue and the progression to degenerative tendinopathy [28].

## The Basis of PDRN Efforts: The Activation of Adenosine A2A Receptor or the "Salvage Pathway"

The healing-promoting effect of PDRN is thought to be mediated by the activation of the adenosine A2A receptor and the "salvage pathway". Generally, following tissue injuries, PDRN activates the adenosine A2A receptor, facilitating a more complete tissue regeneration process. On the other hand, it stimulates the salvage pathway, leading to faster tissue regeneration (**Figure 1**).

Activating the adenosine A2A receptor is one of the primary pharmacological actions of PDRN. Adenosine activates 4 distinct receptors: A1, A2A, A2B, and A3 [29]. Among these, the activation of the adenosine A2A receptor plays a key role in regulating various physiological processes, including stimulating cell proliferation, promoting collagen production, and enhancing angiogenesis [30]. Research indicates that PDRN and adenosine can foster the growth of human skin fibroblasts in primary cultures [31]. However, the effect of PDRN is inhibited by 3,7-dimethyl-1-propargylxanthine, a known antagonist of the A2A receptor, whereas it is not affected by 8-cyclopentyl-1,3-dipropylxanthine, an antagonist of the A2B receptor [31]. This finding confirms that PDRN preferentially targets the adenosine A2A receptor [31]. This selectivity may stem from PDRN's resistance to 5'-exonuclease degradation, which results in small fragments that can bind to several types of purinergic receptors [32]. Under this ligand-receptor mechanism, the effects of PDRN can be more long-lasting than those of platelet-rich plasma, stem cells, or growth factors [33]. Additionally, PDRN can promote DNA synthesis and repair, rejuvenating cellular proliferation and growth in damaged or oxygen-deprived tissues through the "salvage pathway" [34]. In this process, PDRN is cleaved by active membrane enzymes, providing purine and pyrimidine rings for the salvage pathway [35]. As a result, this pathway recovers nucleosides and bases formed by damaged DNA and ribonucleic acid (RNA), which are then converted back into nucleosides and reincorporated into DNA [36]. Ultimately, PDRN reactivates normal cell growth and proliferation patterns by facilitating DNA formation [37]. Nevertheless, the detailed mechanisms still remain poorly understood.

## PDRN on Tendon Disorders Based on Preclinical Studies

This section reviews the literature on the biological effects and underlying mechanisms of PDRN on tendon disorders from a preclinical perspective. Mechanically, PDRN promotes tendon repair by inhibiting inflammatory responses, suppressing excessive apoptosis, and enhancing type I collagen production, which will be thoroughly described below (**Figure 2**).

## PDRN Inhibits Inflammatory Responses and Cell Apoptosis

Studies have recently demonstrated that inflammation plays a non-negligible role in the early initiation of tendinopathy [38]. After a tendon injury, the cytokine equilibrium in the tendon tissue is skewed toward the increased concentrations of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin 6 (IL-6) [22]. This imbalance leads to further destruction of the tendons. On the other hand, the physiological apoptotic process is also known as "programmed cell death" and plays a critical role in maintaining tendon homeostasis. However, excessive apoptosis has been involved in the deterioration of tendon tissue [39]. Caspases are critical





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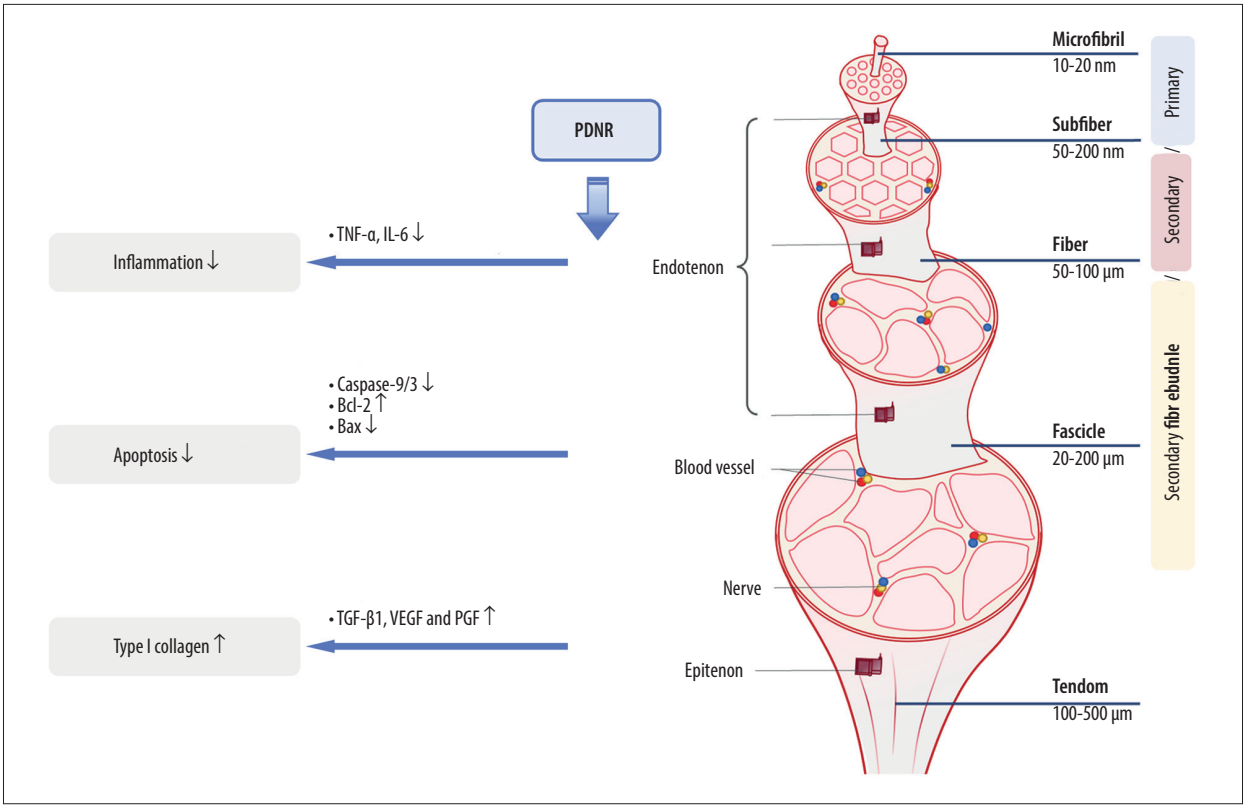


**Figure 2.** Schematic diagram summarizing the roles and mechanisms of polydeoxyribonucleotide (PDRN) in the treatment of tendon disorders based on preclinical investigations. PDRN inhibits inflammation by decreasing the expression of the pro-inflammatory cytokines, including TNF-α and IL-6, thereby enhancing tendon repair. PDRN suppresses cell apoptosis by reducing the expression of caspase-9/3 and Bax while increasing Bcl-2 expression, thereby promoting tendon healing. PDRN encourages type I collagen synthesis by enhancing the expression of TGF-β1, VEGF, and FGF, thereby improving tendon healing. PDRN – polydeoxyribonucleotide; ↑ – increase; ↓ – decrease; TNF-α – tumor necrosis factor alpha; IL-6 – interleukin 6; TGF-β1 – transforming growth factor-beta 1; VEGF – vascular endothelial growth factor; FGF – fibroblast growth factor. *Created with Adobe Photoshop software version 26.2.*

elements involved in cell apoptosis. Particularly, caspase 3 is thought to be an effector caspase as it is involved in breaking down proteins. In parallel, caspase 9 plays a supportive role in the execution phase of the process [40]. Additionally, B-cell lymphoma-2 (Bcl-2) and Bcl-2-associated X (Bax) are 2 proteins involved in the regulation of apoptosis. More specifically, Bcl-2 inhibits the apoptotic process, while Bax enhances apoptosis [41]. When pro-apoptotic proteins are expressed more than anti-apoptotic proteins, apoptosis occurs, eventually leading to deterioration of tendon tissue [42]. Therefore, the corresponding treatment for tendon disorders could inhibit excessive apoptosis and ameliorate inflammation. One study reported that every other day direct PDRN injections (100-μL volume) for 16 days (total of 6 times) favored the recovery of a rat Achilles tendon model. In this study, molecular analyses revealed that the rates of cleaved caspase 3-positive cells and caspase 9-positive cells, as well as the Bax vs Bcl-2 ratio, were increased after tendon injury of rats, which indicates that tendon injury exacerbates the apoptotic process. However, the PDRN-treatment group reduced the percentages of caspase 3- and caspase 9-positive cells and the Bax vs Bcl-2 ratio. Additionally, after the Achilles tendon injury, levels of pro-inflammatory cytokines, such as TNF-α and IL-6, were observed to rise. However, the treatment group receiving PDRN exhibited reduced levels of TNF-α and IL-6 [43]. Ultimately, the PDRN-treated group demonstrated enhanced tendon healing in a rat Achilles tendon model [43]. These findings suggest that PDRN promotes tendon repair by potentially inhibiting inflammation and suppressing cell apoptosis [43]. However, there is still limited research conducted for understanding of the complete mechanisms of action of PDRN as anti-inflammation and anti-apoptosis for tendon disorders. Nevertheless, the anti-inflammatory effects of PDRN are believed to be partly due to its ability to activate the adenosine A2A receptor, which then triggers the intracellular signaling pathway involving cyclic adenosine monophosphate (cAMP), protein kinase A (PKA), and cAMP response element binding (CREB) [44]. The process occurs as follows. First, the adenosine A2A receptor





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on immune cells forms complexes with the G protein's alpha (G-α) subunit (primarily the G protein alpha S [G-αs] subunit) and the G protein beta-gamma (G-β-G-γ) subunits [45]. When PDRN is introduced, it activates the adenosine A2A receptor, causing the G-αs subunit to dissociate from G-β-G-γ [46]. The freed G-αs then activates adenylate cyclase, specifically adenylate cyclase 1 and 9, which converts intracellular adenosine triphosphate into diphosphate and cAMP [47]. The generated cAMP functions as a second messenger, stimulating PKA activity [48]. PKA subsequently phosphorylates CREB, and the cAMP binds to proteins such as phosphorylated CREB and colostrum basic protein/p300 [49]. This series of events effectively inhibits the expression of pro-inflammatory factors like TNF-α and IL-6, while promoting the expression of anti-inflammatory factors such as IL-10 [50]. However, this mechanism of action of PDRN within tenocytes remains not fully understood.

### PDRN Promotes Type I Collagen Synthesis

Normal tendons are primarily made up of collagen type I, with a small amount of collagen type III present [51]. In tendinopathic tendons, there is an increased production of collagen type III, which is not adequately replaced by collagen type I over time [52]. This imbalance leads to reduced mechanical strength and can eventually result in tendon rupture, as collagen type III tends to form thin and randomly oriented fibers [53,54]. Thus, if the production of type I collagen is purposely and selectively increased, the mechanical strength of the damaged tendon tissue could be provoked [55]. The contribution of PDRN to tendon repair has also been reported to encourage the synthesis of type I collagen.

Kang et al examined the effect of PDRN in a rat model of Achilles tendon injury. This methodology included daily PDRN (8 mg/kg/day) injections instead of just 1 injection at the very beginning, as indicated previously. In the PDRN group, type I collagen was increased with a commensurate boost in the expression of transforming growth factor-beta 1 (TGF-β1), vascular endothelial growth factor (VEGF), and fibroblast growth factor (FGF) after 2 weeks, compared with the saline-treated group. Conversely, there was no significant difference in type III collagen production between the experimental and control groups. Also, in the PDRN group, the maximum load, tensile stress, and energy uptake improved remarkably [16]. Interestingly, an increased expression of TGF-β1, VEGF, and FGF is consistent with the previous studies showing that the A2A receptor promotes TGF-β1, VEGF, and FGF secretion and thereby encourages the production of type I collagen, suggesting that the favorable effect of PDRN on the production of type I collagen appears to be mediated by the activation of the adenosine A2A receptor [24,56,57]. Nevertheless, there is still limited research on the mechanisms by which PDRN promotes type I collagen production in the treatment of tendon disorders.

### PDRN on Tendon Disorders Based on Clinical Studies

The clinical manifestations of tendon disorders typically involve pain and dysfunction, which can significantly limit patients' abilities [58]. Tendinopathy is the most appropriate term to describe various chronic tendon pathologies encountered in clinical settings, including paratendinitis, tendinitis, and tendinosis, particularly when there is no biopsy-proven histopathological evidence [59]. In the upper extremities, the most common tendinopathies are rotator cuff (shoulder) tendinopathy and lateral elbow tendinopathy [60]. The most frequently affected tendons in the lower limbs are the plantar fascia, Achilles tendon, and patellar tendon [61]. Recently, PDRN has emerged as an effective treatment for tendinopathy in clinical trials [62]. However, PDRN has been examined to treat only rotator cuff tendinopathy, lateral epicondylitis of the humerus, plantar fasciitis, and pes anserine tendinopathy (**Table 1**). Therefore, clinical results are still lacking in terms of other tendinopathies.

### Rotator Cuff Tendinopathy

One clinical situation characterized by frequent occurrence in individuals over 60 years old is rotator cuff tendinopathy [63]. Three studies have investigated the therapeutic potential of PDRN injection as a therapeutic alternative against rotator cuff tendinopathy.

In a retrospective study by Ryu et al, the age of included patients was 53.4±10.0 years (n=131). Patients were selected who had rotator cuff lesions, including partial tear (involvement <50% of tendons) and tendinosis, based on magnetic resonance imaging and ultrasonography, along with symptoms that had persisted for at least 3 months and were refractory to other conservative treatments, including kinesitherapy and physiotherapy. Among them, 32 patients had received PDRN treatment weekly (3 mL PDRN mixed with 1 mL of 1% lidocaine). One week after treatment, patients demonstrated a significant reduction in pain, compared with before treatment, evaluated by the visual analog scale (VAS). Pain reduction remained stable at 1 month and 3 months, suggesting that the pain was significantly improved and relieved at 1 week and lasted for 3 months. The functional fraction of the shoulder assessed by single assessment numeric evaluation over the 1-week period after treatment improved the most at 1 month and 3 months after treatment. Shoulder disability measured by the Shoulder Pain and Disability Index (SPADI) after 1 month and 3 months decreased more than at 1 week after treatment. No adverse events, including post-treatment pain and infection, occurred during treatment. According to this report, PDRN contributed to safe improvements in function and pain of patients with refractory rotator cuff disorders for at least 3 months. However, one limitation of this study is the lack of a control group (conventional therapy) for comparison [15].





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**Table 1.** The effect of polydeoxyribonucleotide (PDRN) in tendon disorders based on clinical studies.

| Study | Disease type | Number of treatments | Dosage | Outcomes | Conclusions |
|---|---|---|---|---|---|
| Ryu et al | Rotator cuff tendinopathy | Once | 3 mL PDRN mixed with 1 mL of 1% lidocaine | Pain ↓<br>Shoulder disability ↓<br>Function ↑ | PDRN contributed to safe improvements in function and pain of patients with refractory rotator cuff disorders for at least 3 months |
| Yoon et al | Rotator cuff tendinopathy | Once | 5.625 mg/3 mL of PDRN mixed with 1% lidocaine 1 mL with a 23-gauge 6-cm needle | Pain ↓<br>Shoulder disability ↓<br>Function ↑ | Three months after treatment, the subjective pain, SPADI, and VAS demonstrated significantly improvements compared to the control group without adverse events |
| Do et al | Rotator cuff tendinopathy | Three times | 5.625 mg/3 mL of PDRN | Pain ↓<br>Shoulder disability ↓<br>Function ↑<br>Shoulder tear volume ↓<br>Range of motion ↑ | PDRN injection into torn area of supraspinatus tendon on US could be candidate for the safe and effective treatment on shoulder pain and limited range of motion in patients with rotator cuff tear |
| Lee and Park | Lateral epicondylitis | Once | 5.625mg/3 mL PDRN with a 27-gauge, 1.5-inch needle | Pain ↓<br>Symptom relief ↑ | After 2 weeks from PDRN injection, both patients reported significant pain relief. The 2 weeks after the PDRN injection showed that the hypervascularity of the common extensor tendon in both patients had been completely cured |
| Shim et al | Lateral epicondylitis | Once | 5.625 mg/3 mL | Pain ↓<br>Function ↓<br>Tendon tear volume↓ | PDRN injections combined with counterforce braces exhibited a greater improvement in pain and function within the 12-week follow-up |
| Lee et al | Plantar fasciitis | Once | 3 mL PDRN at 1-week intervals | Pain ↓<br>Function ↑<br>Symptom relief ↓ | PDRN injection could be an effective and safe option for plantar fasciitis and was comparable to corticosteroid injection after 6months follow up |
| Kim and Chung | Plantar fasciitis | Once | / | Pain ↓<br>Function ↑<br>Symptom relief ↑ | The PDRN group achieved a significant improvement in pain and function at 4 weeks after treatment, and this improvement continued until 12 weeks after treatment |
| Mun et al | Pes anserine tendinopathy | Once | 5.625 mg/3 mL | Pain ↓<br>Function ↑<br>Symptom relief ↑ | Follow-up for the patient was more than 8 months. She showed good improvement in pes anserine tendinopathy without any complications |

PDRN – polydeoxyribonucleotide; ↑ – increase; ↓ – decrease; SPADI – Shoulder Pain and Disability Index; VAS – visual analog scale; US – ultrasonography.





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By contrast, a retrospective and comparative study of conventional treatment and PDRN application was conducted by Yoon et al [64]. A total of 119 patients (aged from 40 to 65 years) were diagnosed with rotator cuff tendinopathy that had a duration of symptoms for at least 6 months and was refractory to the conventional conservative therapies for at least 1 month (including analgesics and resistance training). Among these, 62 patients chose PDRN treatment, and 57 continued opting for the above conservative treatments (control group). Ultrasound (10 to 13 MHz linear transducer)-guided PDRN injections of 5.625 mg/3 mL of PDRN mixed with 1% lidocaine 1 mL and administered with a 23-gauge 6-cm needle were used to treat those patients. Three injections were administrated into the supraspinatus tendons at weekly intervals. The main outcome measures included the subjective pain, SPADI, and VAS scores. Three months after treatment, the subjective pain, SPADI, and VAS scores demonstrated significant improvements, compared with the control group, and without adverse events. Therefore, based on the above 2 studies, PDRN injections demonstrate the greatest effect at 3 months after the treatment.

In a pilot study, Do et al reported the potential for the use of ultrasound-guided PDRN injections in patients with partial-thickness tear of supraspinatus tendons. Seventeen patients received ultrasound-guided PDRN injections (5.625 mg/3 mL of PDRN) at 0, 2, and 4 weeks, for a total of 3 injections. The VAS score, Disabilities of Arm, Hand, and Shoulder (DASH) score, range of motion in the shoulder, and ultrasonographic examinations were checked at 0, 6, and 12 weeks after the treatments. It was found that the mean VAS score, DASH score, and range of motion in the shoulders of those patients significantly improved after the treatments. Moreover, based on the ultrasound findings, shoulder tear volume decreased during the treatment period. No significant complications resulting from the PDRN injections, such as infection and skin injuries, were found during the treatment period [65].

Above all, these clinical investigations indicated that PDRN is highly associated with pain relief in patients with rotator cuff tendinopathy. However, some of these studies are retrospective; therefore, there could have been several limitations to collect records of outcome assessment comprehensively. Therefore, with the aim of displaying the effectiveness of PDRN, future investigations with randomized controlled trials are needed to overcome these limitations.

### Lateral Epicondylitis

Lateral epicondylitis, or tennis elbow, features pain in the lateral elbow, which is exacerbated by attempts to extend and supinate the wrist and hand against resistance. The injury involves the origin of the common extensor tendon on the lateral epicondyle of the humerus [66].

In the case study conducted by Lee et al, 2 patients with pain exacerbation experienced for 2 months of the right lateral elbow received ultrasound-guided PDRN injections (5.625mg/3 mL PDRN with a 27-gauge, 1.5-inch needle) into the joint extensor tendons. Two weeks after the injections, both patients demonstrated significant pain reduction, and at the 2-month follow-up, improved symptoms in the lateral epicondylitis were reported in both patients. It has been proposed that improved symptoms are attributed to the anti-inflammatory effect of PDRN, which is responsible for reducing pro-inflammatory cytokines such as TNF-α and IL-6 and increasing anti-inflammatory cytokines such as IL-10, as indicated in previous studies [67].

In a randomized controlled trial conducted by Shim et al, 69 patients with a clinical diagnosis of chronic lateral epicondylitis were allocated into 3 groups (21 patients remained in each group, and 6 patients were excluded). At the first visit, all 3 groups were taught to perform extensor muscle strengthening exercises with counterforce braces. Group 2 was injected with PDRN (5.625mg/3 mL), while group 3 was treated with extracorporeal shock-wave therapy (pressure 1.5 bar, frequency 4.0 Hz, number 500). Mayo elbow performance score (MEPS), VAS score, and ultrasound evaluations were assessed before and 6 and 12 weeks after the treatments. The ultrasound examinations include the common extensor tendon depth, common extensor tendon tear thickness, and color Doppler activity. The results showed that all 3 groups improved the mean VAS scores, MEPS, and ultrasound examinations (color Doppler activity, common extensor tendon depth, and common extensor tendon tear thickness). Among the 3 groups, the mean MEPS of group 2 remarkably improved more than that in groups 1 and 3 at 6 weeks, as well as more than group 2 at 12 weeks after the treatments. The mean common extensor tendon depth on ultrasound examinations of group 2 significantly improved more than group 1 and group 3 at 6 weeks, and that of groups 2 and 3 increased significantly more than group 1 at 12 weeks [68].

### Plantar Fasciitis

Lee et al compared the effectiveness of PDRN treatment and corticosteroid injection for plantar fasciitis in a prospective randomized clinical study, in which 44 patients with intractable plantar fasciitis were randomly allocated to PDRN (3 mL PDRN at 1-week intervals) or corticosteroid injection. The VAS pain score and the Manchester-Oxford foot questionnaire (MOXFQ) were conducted to evaluate pain at baseline, 1, 2, and 6 weeks, and 6 months. Ultrasonography was used to assess the echogenicity and thickness of plantar fascia. The results showed that in both groups, significant symptom relief was reported. However, there was no significant difference between the 2 groups using the VAS pain score at 6 months after treatment [69].





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Another prospective randomized study was conducted by Kim et al to evaluate the efficacy and safety of PDRN injection for the treatment of patients with plantar fasciitis. Forty patients diagnosed with plantar fasciitis were randomly assigned to a PRDN injection or saline group. The injections were performed at 1-week intervals for 3 weeks. Clinical evaluations using the VAS pain score and MOXFQ were performed at baseline and 4 and 12 weeks after treatment. Patients treated with PRDN injection exhibited significant improvements in VAS and MOXFQ scores at 4 weeks after treatment, with this effect persisting for 12 weeks after treatment, compared with the saline group. No injection-related complications were found after PRDN injection [70].

### Pes Anserine Tendinopathy

In a case report by Mun et al, a 50-year-old female patient was diagnosed with pes anserine tendinopathy, presenting with localized swelling on the medial side of her left knee. She experienced chronic, refractory pain in that area, particularly during daily activities, and rated her pain as 7 out of 10 on the numeric rating scale. At the time of her admission, the pain was so severe that it limited her abilities. Although she had been receiving physical therapy, including bandages and NSAIDs, she showed no signs of improvement. The patient declined a glucocorticoid bursa injection due to concerns about its effects and adverse effects. Consequently, the researchers performed an ultrasound-guided injection into the posterior bursa using a combination of 5.625 mg of PDRN with 3 mL of 1% lidocaine administered with a 26-G, 4-cm needle. At the 1-week follow-up after the injection, her numeric rating scale score decreased from 7 to 2. By the 2-week follow-up, she reported further significant pain reduction, with her numeric rating scale score dropping to 0. Follow-up assessments continued for over 8 months, during which the patient made a complete recovery. She experienced no pain and regained full range of motion in her left knee while walking. No adverse reactions or adverse effects were observed [71].

### Future Directions

Although the application of PDRN has demonstrated promising results in managing tendon disorders, much remains to be explored in this domain.

First, *Oncorhynchus keta* (chum salmon) and *Oncorhynchus mykiss* (salmon trout) sperm are very hard to obtain, since these organisms spawn only during the breeding season, which leads to PDRN being costly [72]. Recently, some studies investigated PDRN extracted from humans and plants [73]. However, the ethical problems of extracting PDRN from humans are great [73]. When PDRN is extracted from plants, including roses, broccoli, and aloe, additional processes are required to break the cell walls of plants [36,74]. Future studies are needed to solve this problem.

Second, all studies reviewed in this article used commercially manufactured PDRN rather than extracted PDRN [67]. Thus, the dosage application in those above experiments was fixed below 1.875 mg/mL, as PDRN is commercially provided at 5.625 mg/3 mL [67]. Therefore, more studies are needed to demonstrate the other dosages that may influence the effectiveness of PDRN.

Finally, in a study on a rabbit chronic full-thickness rotator cuff tendon tear model, Kwon et al investigated the effect of PDRN in combination with human umbilical cord blood-derived mesenchymal stem cells (UCB-MSCs). Specifically, 32 rabbits were randomly allocated into the following 4 groups: group 1 (0.2 mL saline); group 2 (0.2 mL PDRN); group 3 (0.2 mL UCB-MSCs); and group 4 (0.2 mL PDRN together with 0.2 mL UCB-MSCs). In group 4, the synthesis of type I collagen, cell proliferation, angiogenesis, walking distance, and walking speed were significantly improved at 6 weeks after treatment, compared with that in the other 3 groups, suggesting that the combination of UCB-MSCs and PDRN achieved more promising results in tendon disorders [75]. This suggests that the synergistic effect of PDRN and other therapies tends to produce a better result. Moreover, the same group of researchers investigated the synergic effects of PDRN in combination with microcurrent on the rotator cuff tear of a rabbit model. Rabbits (n=24) were randomly divided into 3 groups after full thickness: group 1 (0.2 mL saline); group 2 (0.2 mL PDRN); and group 3 (0.2 mL PDRN+microcurrent). The tendon type I collagen synthesis, angiogenesis, and walking parameters were significant in group 3, compared with the other 2 groups, indicating that the combination of PDRN with microcurrent is more potent [76].

In the future, we should concentrate on the effectiveness of combining PDRN with other therapies for treating tendinopathies, fully exploiting the synergistic effects of PDRN and these therapies so as to stimulate their therapeutic potential and thus better regulate tendon healing.

## Conclusions

Tendon disorders are recognized as a significant musculoskeletal issue, contributing to pain and disability in patients. Traditional treatments for these disorders include surgery, NSAIDs, eccentric exercise, extracorporeal shock wave therapy, and therapeutic ultrasound. Despite the advancements in these therapeutic options, the outcomes of these treatments have often been unsatisfactory. As noted, several preclinical and clinical investigations show that PDRN is an





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effective and promising drug for the treatment of tendon disorders. Pre-clinical evidence has revealed that the underlying mechanisms for the effects of PDRN on tendon disorders include inhibiting inflammatory responses, suppressing apoptosis, and enhancing collagen production. Moreover, clinical data has suggested that PRDN contributes to pain relief and function recovery in patients with rotator cuff tendinopathy, lateral epicondylitis, plantar fasciitis, and pes anserine tendinopathy. However, further research is needed to investigate optimal dosage, combination therapy of PDRN, and the ethical considerations surrounding the extraction of PDRN, to facilitate its future clinical use.

### Declaration of Figures' Authenticity

All figures submitted have been created by the authors, who confirm that the images are original with no duplication and have not been previously published in whole or in part

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