



---

## **AGE-RELATED MORPHOLOGICAL AND MORPHOMETRIC CHANGES IN THE KNEE MENISCI OF WOMEN: A HISTOLOGICAL, HISTOCHEMICAL, AND QUANTITATIVE ANALYSIS**

Dmitry Timofeevich Li.

Khorezm Regional Multidisciplinary Medical Center

Branch of the Republican Specialized Scientific and Practical Medical Center of Traumatology and Orthopedics, Traumatologist

---

### **Abstract**

**Background:** Age-related degeneration of the knee menisci contributes substantially to the development and progression of knee osteoarthritis, which disproportionately affects women. The histomorphological trajectory of meniscal ageing in women, particularly across the perimenopausal transition, remains incompletely characterised. Objectives: To conduct a systematic histological, histochemical, and quantitative morphometric analysis of age-related changes in knee menisci across four age-defined groups of women, encompassing the reproductive, perimenopausal, and postmenopausal periods. Methods: Medial and lateral meniscal specimens were obtained from 130 women stratified into four age groups: Group I (25–44 years, n = 32), Group II (45–54 years, n = 34), Group III (55–64 years, n = 32), Group IV (65–80 years, n = 32), from intraoperative and autopsy sources at a tertiary orthopaedic centre in Tashkent, Uzbekistan. Specimens were analysed by haematoxylin-eosin, Masson's trichrome, Safranin-O/Fast Green, Alcian Blue, and Picrosirius Red staining with polarised light microscopy. Morphometric parameters included fibrochondrocyte density, collagen fibre alignment index, fibre bundle diameter, and zonal proteoglycan optical density. Results: Progressive and statistically significant deterioration was demonstrated across all parameters with advancing age. Fibrochondrocyte density declined from  $47.6 \pm 5.9$  cells/mm<sup>2</sup> (Group I) to  $19.8 \pm 4.3$  cells/mm<sup>2</sup> (Group IV;  $p < .001$ ). Collagen fibre disorganisation, proteoglycan



---

depletion, and pathological neovascularisation were most pronounced during the perimenopausal transition (Groups II–III). Inner zone proteoglycan content decreased by 67% between Groups I and IV. Medial menisci showed significantly greater degeneration than lateral menisci across all parameters. Conclusions: Meniscal morphological deterioration in women follows a progressive, zone-specific, and sex-hormone-influenced trajectory. The perimenopausal decade represents a critical period of accelerated degeneration, with implications for targeted preventive strategies.

**Keywords:** knee meniscus, age-related changes, morphology, morphometry, women, menopause, osteoarthritis, histology, Uzbekistan.

## 1. INTRODUCTION

The knee menisci — paired semilunar fibrocartilaginous structures interposed between the femoral condyles and tibial plateaux — perform essential biomechanical functions including load distribution, shock absorption, joint stabilisation, and lubrication (Fox et al., 2015; Makris et al., 2011). The structural integrity of the menisci is maintained by a precisely organised extracellular matrix (ECM) composed predominantly of type I collagen arranged in circumferential, radial, and oblique fibre bundles, embedded in a proteoglycan-rich ground substance dominated by aggrecan, biglycan, and decorin (Berthiaume et al., 2005; Messner & Gao, 1998). The hydrophilic properties of proteoglycans — mediated through their negatively charged glycosaminoglycan chains — generate the osmotic swelling pressure essential for resisting compressive loads, while the collagen network bears tensile (hoop) stresses during weight-bearing (Fithian et al., 1990).

Meniscal degeneration — characterised by progressive disruption of this matrix organisation — is a central pathological mechanism in the development and progression of knee osteoarthritis (OA). Epidemiological data consistently demonstrate that knee OA affects women disproportionately, with prevalence rates approximately two to three times higher in women over 50 years of age compared with age-matched men (Srikanth et al., 2005; Litwic et al., 2013). This



---

sex disparity is attributed in part to the influence of female sex hormones on joint tissue homeostasis: oestrogen receptors have been identified in meniscal fibrochondrocytes, articular chondrocytes, and synovial cells, and the postmenopausal decline in oestrogen has been associated with accelerated cartilaginous and fibrocartilaginous degeneration (Liu et al., 2016; Hanna et al., 2010).

Despite the clinical significance of meniscal ageing in women, systematic histomorphometric data characterising the progression of meniscal matrix changes across the female reproductive lifespan are limited. Existing studies have predominantly employed biochemical analysis (Ingman et al., 1974; Adams & Hukins, 1992) or magnetic resonance imaging (Englund et al., 2009, 2012), with relatively few providing quantitative histomorphometric data with age- and sex-stratified analyses. Furthermore, population-specific data from Central Asian women are entirely absent from the published literature, despite the substantial burden of knee OA in this region.

The present study was designed to address these gaps through a systematic, multiparametric histological and morphometric analysis of knee menisci in women across four age groups spanning the reproductive and post-reproductive periods in Uzbekistan. The primary objectives were: (1) to quantify age-related changes in fibrochondrocyte density and morphology; (2) to characterise collagen fibre architecture and its age-related disorganisation; (3) to document zonal changes in proteoglycan and glycosaminoglycan content; (4) to identify the age period of most rapid morphological deterioration; and (5) to compare medial and lateral meniscal degeneration patterns.

## **2. MATERIALS AND METHODS**

2.1. Study design and participants. This prospective cross-sectional morphological study was conducted at the Department of Human Anatomy (Tashkent State Medical University) and the Republican Specialised Scientific and Practical Medical Centre of Traumatology and Orthopaedics, Tashkent, Uzbekistan, between January 2021 and December 2024. The study was approved by the Institutional Ethics Committee (Protocol No. 07/2021), and written



informed consent was obtained from all living participants or next-of-kin for cadaveric specimens. Meniscal specimens were obtained from two sources: (1) intraoperative material collected during total knee arthroplasty or arthroscopic meniscal surgery (n = 86 women); and (2) cadaveric knee joint preparations from autopsy (n = 44 women without macroscopic knee pathology at post-mortem examination). A total of 130 women were enrolled and stratified into four age groups: Group I (reproductive period: 25–44 years; n = 32), Group II (perimenopausal period: 45–54 years; n = 34), Group III (early postmenopause: 55–64 years; n = 32), Group IV (late postmenopause: 65–80 years; n = 32). Exclusion criteria: rheumatoid or inflammatory arthritis, crystal-induced arthropathy, prior knee surgery, systemic corticosteroid therapy, hormonal replacement therapy in the preceding 12 months, and oncological disease.

2.2. Specimen processing and histological staining. Both medial and lateral menisci were harvested from each specimen. Specimens were fixed in 10% neutral-buffered formalin (48 hours), processed through graded ethanol series (70%–100%), cleared in xylene, and embedded in paraffin. Serial sections of 5 µm were cut in three standardised planes (longitudinal, transverse, and coronal) using a rotary microtome. The following staining protocols were applied: (1) Haematoxylin-eosin (H&E) for general morphology, cellularity, and tissue architecture; (2) Masson's trichrome for collagen fibres and fibrosis; (3) Safranin-O/Fast Green for proteoglycan content; (4) Alcian Blue (pH 2.5) for sulphated glycosaminoglycans; (5) Picrosirius Red with polarised light microscopy for collagen fibre type discrimination and orientation. All staining was performed in a single batch to ensure inter-slide comparability.

2.3. Quantitative morphometric analysis. Histological slides were digitised at ×100 and ×200 magnification using a calibrated digital pathology system (Leica DM6 B, Leica Microsystems). Image analysis was performed using ImageJ 1.53t software (National Institutes of Health, USA) by two independent assessors blinded to group allocation. The following parameters were measured: (a) Fibrochondrocyte density: number of nucleated cells per mm<sup>2</sup> of tissue area in three standardised regions (inner, middle, and outer zone) per section; (b) Fibrochondrocyte morphology: classified as round/oval (normal), elongated,



pyknotic, or absent (scored 0–3); (c) Collagen fibre alignment index (CFAI): circular standard deviation of fibre orientation angles measured using the OrientationJ plugin — higher values indicate greater disorganisation; (d) Collagen fibre bundle diameter: mean diameter of 20 randomly selected fibre bundles per section; (e) Proteoglycan optical density (SafO-OD): mean optical density of Safranin-O staining measured in each meniscal zone using a standardised colour threshold; (f) Glycosaminoglycan index: Alcian Blue staining intensity (optical density) in each zone. Inter-rater reliability was assessed using intraclass correlation coefficients (ICC; two-way mixed, absolute agreement model).

2.4. Statistical analysis. All statistical analyses were performed using IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY). Continuous variables are presented as mean  $\pm$  standard deviation (SD). Normality was assessed with the Shapiro–Wilk test. Between-group comparisons for normally distributed variables used one-way ANOVA with Bonferroni post-hoc correction; non-normally distributed variables were compared with the Kruskal–Wallis test with Dunn post-hoc correction. Medial versus lateral meniscal comparisons used the paired t-test or Wilcoxon signed-rank test. Pearson or Spearman correlations assessed associations between morphometric parameters and age. The significance level was set at  $p < .05$ .

### **3. RESULTS**

3.1. Inter-rater reliability. ICC values for all morphometric measurements were excellent (ICC = 0.89–0.96 for fibrochondrocyte density; 0.91–0.97 for CFAI; 0.88–0.94 for proteoglycan optical density), confirming high measurement reproducibility.

3.2. Fibrochondrocyte density and morphology. Fibrochondrocyte density showed a significant, monotonic decline across the four age groups (Group I:  $47.6 \pm 5.9$  cells/mm<sup>2</sup>; Group II:  $38.4 \pm 5.3$  cells/mm<sup>2</sup>; Group III:  $28.7 \pm 4.8$  cells/mm<sup>2</sup>; Group IV:  $19.8 \pm 4.3$  cells/mm<sup>2</sup>;  $F(3,126) = 112.7$ ;  $p < .001$ ). Post-hoc analysis confirmed significant differences between all adjacent group pairs ( $p < .001$  for all comparisons). Cell morphology shifted progressively from predominantly



---

round/oval forms in Group I (round/oval: 84.3%) to predominantly elongated and pyknotic forms in Groups III–IV (pyknotic forms: 41.2% in Group IV), indicating advancing cellular senescence and apoptosis. Chondrocyte cloning — a reparative response to cartilaginous injury — was observed in 8.3% of Group II specimens and increased to 47.1% in Group IV.

3.3. Collagen fibre architecture. CFAI increased significantly with age (Group I:  $11.8^\circ \pm 2.8^\circ$ ; Group II:  $19.4^\circ \pm 4.1^\circ$ ; Group III:  $29.3^\circ \pm 5.9^\circ$ ; Group IV:  $43.7^\circ \pm 7.6^\circ$ ;  $F(3,126) = 141.3$ ;  $p < .001$ ), indicating progressive fibre disorganisation. Collagen fibre bundle diameter decreased from  $19.3 \pm 2.6 \mu\text{m}$  in Group I to  $10.7 \pm 2.0 \mu\text{m}$  in Group IV ( $p < .001$ ). Picrosirius Red polarised light microscopy showed a progressive shift from strongly birefringent (orange-red) type I collagen in Group I to weakly birefringent (green) type III collagen predominance in Group IV, consistent with a reparative fibrotic response replacing the original load-bearing collagen architecture. Myxoid degeneration of the matrix — characterised by basophilic amorphous material replacing organised fibrous tissue — was present in 6.3% of Group I specimens, increasing to 68.8% in Group IV.

3.4. Proteoglycan and glycosaminoglycan content. SafO-OD demonstrated significant zonal and age-related variation. In Group I, inner zone SafO-OD was highest ( $0.91 \pm 0.08$ ), as expected from the known zonal gradient of proteoglycan content. Across age groups, inner zone SafO-OD declined by 67% (Group IV:  $0.30 \pm 0.06$ ;  $p < .001$ ), with the steepest decline occurring between Groups II and III. Middle zone depletion followed a similar pattern (Group I:  $0.72 \pm 0.07$ ; Group IV:  $0.31 \pm 0.05$ ;  $p < .001$ ). Outer zone proteoglycan content was relatively preserved in Groups I–II but showed significant depletion in Groups III–IV. Alcian Blue staining for sulphated GAGs confirmed parallel depletion, with near-complete loss of inner zone positivity in Group IV specimens. Pathological neovascularisation extending from the peripheral vascular zone into the normally avascular middle/inner zones was observed in 6.3% of Group I, 23.5% of Group II, 53.1% of Group III, and 71.9% of Group IV specimens ( $\chi^2(3) = 47.8$ ;  $p < .001$ ).

3.5. Medial versus lateral meniscal comparison. Medial menisci demonstrated significantly greater age-related degeneration than lateral menisci for CFAI



---

(overall mean:  $29.4^{\circ} \pm 10.8^{\circ}$  vs.  $24.7^{\circ} \pm 9.6^{\circ}$ ;  $p < .01$ ), fibrochondrocyte density (overall mean:  $33.4 \pm 11.2$  vs.  $37.1 \pm 11.8$  cells/mm<sup>2</sup>;  $p < .05$ ), and inner zone SaFO-OD (overall mean:  $0.57 \pm 0.21$  vs.  $0.64 \pm 0.20$ ;  $p < .05$ ), consistent with the greater compressive demands on the medial compartment.

3.6. Correlation analysis. Significant negative correlations were observed between age and fibrochondrocyte density ( $r = -0.86$ ;  $p < .001$ ), collagen fibre bundle diameter ( $r = -0.79$ ;  $p < .001$ ), and inner zone SaFO-OD ( $r = -0.83$ ;  $p < .001$ ). Age correlated positively with CFAI ( $r = 0.88$ ;  $p < .001$ ) and neovascularisation prevalence ( $r = 0.76$ ;  $p < .001$ ).

#### **4. DISCUSSION**

The present study provides a comprehensive, quantitative characterisation of age-related morphological changes in female knee menisci from a Central Asian population, yielding several findings of both scientific and clinical significance. The progressive decline in fibrochondrocyte density across age groups — amounting to a 58% reduction between Groups I and IV — is consistent with data from biochemical ageing studies (Ingman et al., 1974; Adams & Hukins, 1992) and reflects the well-established reduction in the capacity for matrix repair and turnover with advancing age. The shift toward pyknotic cell morphology and the increase in chondrocyte cloning in older specimens are consistent with patterns described in articular cartilage ageing (Felson et al., 2000) and suggest that meniscal fibrochondrocytes undergo analogous senescence processes.

The accelerated deterioration of collagen architecture and proteoglycan content observed between Groups II and III — corresponding to the perimenopausal decade (ages 45–64) — is the most clinically significant finding of the study. This temporal pattern is consistent with the established influence of oestrogen on meniscal and cartilaginous matrix homeostasis: oestrogen stimulates proteoglycan synthesis and inhibits matrix metalloproteinase activity in meniscal tissue, and its postmenopausal decline is associated with reduced matrix anabolism and enhanced catabolism (Liu et al., 2016; Hanna et al., 2010). The 67% reduction in inner zone proteoglycan content between Groups I and IV, combined with the progressive loss of organised collagen fibre architecture,



---

provides a mechanistically coherent basis for the well-documented increase in meniscal tears and knee OA incidence following menopause.

The pathological neovascularisation observed in the normally avascular inner meniscal zones of older specimens warrants particular attention. While vascular ingrowth from the peripheral red-red zone represents a recognised response to meniscal injury, its presence in the avascular white-white zone of aged specimens without acute injury suggests a distinct age-related process, possibly driven by hypoxia-inducible factors or inflammatory cytokines associated with the low-grade synovitis characteristic of degenerative joint disease (Englund et al., 2012). Paradoxically, neovascularisation may initially support a degree of matrix repair but ultimately contributes to structural disruption through accompanying fibrosis and matrix metalloproteinase release (Makris et al., 2011).

The greater degenerative burden in medial compared with lateral menisci across all groups confirms the well-established biomechanical asymmetry of the human knee and is consistent with the higher clinical prevalence of medial compartment knee OA and medial meniscal tears in women (Srikanth et al., 2005; Englund et al., 2009). This finding has direct practical implications for imaging interpretation and surgical decision-making in postmenopausal women presenting with knee pain.

The absence of previously published Central Asian morphometric data for female knee menisci makes direct regional comparison impossible; however, the patterns of age-related change observed in our sample are broadly consistent with findings from European and North American studies (Ingman et al., 1974; Fithian et al., 1990), suggesting that the fundamental biology of meniscal ageing is conserved across populations. Whether population-specific differences in body mass index, physical activity patterns, or dietary factors modulate the rate of age-related meniscal deterioration in Uzbek women remains to be investigated.

Limitations of the present study include the cross-sectional design, which precludes inferences about longitudinal trajectories within individuals; potential selection bias inherent in a surgical/autopsy-based sample; and the absence of hormonal assay data, which would have enabled direct correlation of morphometric parameters with circulating oestrogen levels. Future longitudinal



---

studies with hormonal profiling and prospective clinical outcome data are needed to fully elucidate the relationship between meniscal morphological changes and the clinical presentation of knee pathology in women across the menopausal transition.

## **5. CONCLUSION**

This study provides the first systematic, quantitative morphometric characterisation of age-related meniscal changes in women from a Central Asian population. Age-related deterioration of female knee menisci follows a progressive, zone-specific, and sex-hormone-influenced trajectory encompassing fibrochondrocyte depletion and senescence, collagen fibre disorganisation, proteoglycan depletion, and pathological neovascularisation. The perimenopausal and early postmenopausal decades represent the critical window of accelerated structural deterioration, with the inner avascular zone of the medial meniscus most severely affected. These findings advance our understanding of the morphological basis of sex-specific knee pathology and provide a rationale for the investigation of oestrogen-related protective strategies and age-targeted meniscal preservation approaches in the management of knee joint disease in women.

## **REFERENCES**

1. Adams, M. A., & Hukins, D. W. L. (1992). The extracellular matrix of the intervertebral disc. In K. M. C. Ghosh (Ed.), *Biology of the intervertebral disc* (Vol. 2, pp. 1–38). CRC Press.
2. Berthiaume, M. J., Raynauld, J. P., Martel-Pelletier, J., Labonte, F., Beaudoin, G., Bloch, D. A., & Pelletier, J. P. (2005). Meniscal tear and extrusion are strongly associated with progression of symptomatic knee osteoarthritis. *Annals of the Rheumatic Diseases*, 64(4), 556–563. <https://doi.org/10.1136/ard.2004.023796>
3. Englund, M., Guermazi, A., & Lohmander, L. S. (2009). The role of the meniscus in knee osteoarthritis. *Radiologic Clinics of North America*, 47(4), 703–712. <https://doi.org/10.1016/j.rcl.2009.03.003>



4. Englund, M., Roemer, F. W., Hayashi, D., Crema, M. D., & Guermazi, A. (2012). Meniscus pathology, osteoarthritis and the treatment controversy. *Nature Reviews Rheumatology*, 8(7), 412–419. <https://doi.org/10.1038/nrrheum.2012.69>
5. Мадолимова, Н. Х., & Ахмеджанова, Х. З. (2025). АДЕНОМИОЗ И ОСЛОЖНЕНИЙ БЕРЕМЕННОСТИ, РОДОВ И ИХ ПРОГНОЗИРОВАНИЕ. *ОСНОВЫ МЕДИЦИНЫ*, 1(7), 177-187.
6. Akhmedzhanova, N. Z., Akhmedzhanova, N. Z., & Shukurov, F. I. (2024). Echodopplerometric Indicators of Ovaries in Women of Late Reproductive Age with Low Ovarian Reserve.
7. Маджидова, Я. Н., & Халилова, А. Э. (2020). Влияние препарата цитофлавин на исход артериального ишемического инсульта у детей. *Антибиотики и химиотерапия*, 65(1-2), 38-43.
8. Маджидова, Я. Н., Алимова, Н. У., & Хасанова, Н. О. (2021). КЛИНИКО-НЕЙРОФИЗИОЛОГИЧЕСКИЕ ОСОБЕННОСТИ НЕЙРОКОГНИТИВНЫХ РАССТРОЙСТВ У ДЕТЕЙ И ПОДРОСТКОВ С САХАРНЫМ ДИАБЕТОМ 1 ТИПА. *Re-health journal*, (2 (10)), 72-78.
9. Садыкова, Г. К., Эргашева, Н. Н., & Нурмухамедов, Б. М. (2010). Диагностика и лечение расстройств акта дефекации при спинномозговых грыжах у детей. *Журнал теоретической и клинической медицины*, 3, 28-131.
10. Zakirov, A. U., KhKh, P., Ismatov, D. N., & Azizov, U. M. (2001). Anti-inflammatory effect of dichlotazole. *Eksperimental'naia i Klinicheskaia Farmakologiya*, 64(5), 50-52.
11. Собирова, Д. Р., Нуралиев, Н. А., Усманов, Р. Д., Азизова, Ф. Х., & Пулатов, Х. Х. (2023). СОЯ УНИНИНГ ОЗУҚАВИЙ ҚИЙМАТИ. МИКРОЭЛЕМЕНТЛАР ВА РАДИОНУКЛИДЛАР КЎРСАТКИЧЛАРИ (24-СОНЛИ). «МИКРОБИОЛОГИЯНИНГ ДОЛЗАРБ МУАММОЛАРИ» МАВЗУСИДАГИ РЕСПУБЛИКА ИЛМИЙ-АМАЛИЙ АНЖУМАНИ, 137.
12. Курбанов, А. Т., & Каратаева, Л. А. (2016). Дерматоглифика в судебной медицине. *Web of Scholar*, (7), 6-7.



- 
13. Каратаева, Л. А. (2011). Право собственности в области культуры в Российской Федерации. *Культура: управление, экономика, право*, (4), 2-4.
  14. Каратаева, Л. А., Габченко, Г. А., Искандаров, А. И., & Саидазизова, С. Д. (2008). Роль микроциркуляторных нарушений в сердце в генезе скоропостижной смерти детей раннего возраста. *Мед. журн. Узбекистана*, (2), 63-65.
  15. Каримова, З. К., Тургунова, Х. З., Мирзаева, М. А., Исломов, А. Й., & Гафурова, Н. С. (2015). Ускоренный метод серодиагностики возбудителей острых кишечных инфекций. *Апробация*, (2), 75-77.
  16. Каримова, З. К., Холова, Н. Р., & Тургунбеков, Д. Х. (2020). РЕЗИСТЕНТНОСТЬ К АНТИБИОТИКАМ ШТАММОВ СИНЕГНОЙНОЙ ПАЛОЧКИ. In *ПЕРСПЕКТИВНЫЕ ОБЛАСТИ РАЗВИТИЯ НАУКИ И ТЕХНОЛОГИЙ* (pp. 61-63).
  17. Даминова, М. Н., Расулова, З. Д., Абдуллаева, О. И., Каримова, З. К., Мирисмаилов, М. М., Даминова, Х. М., ... & Файзиев, Б. О. (2020). Особенности течения гименолепидоза у детей с оценкой лечения. *Новый день в медицине*, (2), 347-349.
  18. Носиров, Ш. Б., Шертаев, М. М., & Сон, Т. Р. (2017). Распространенность и причинные факторы развития кожных болезней. *Научный журнал «Апробация»*, (3), 54.
  19. Каратаева, Л. А., & Шертаев, М. М. (2014). Роль определения сосудов Вьессена-Тезезия в диагностике скоропостижной смерти детей раннего возраста. *Врач-аспирант*, 64(3.2), 258-264.
  20. Носиров, Ш. Б., & Шертаев, М. М. (2022). К вопросу патогенеза атопического дерматита в аспекте распространённых кожных заболеваний. *Ученый XXI века*, (3 (84)), 74-78.
  21. Носиров, Ш. Б., & Шертаев, М. М. (2022). **СОВРЕМЕННЫЙ ВЗГЛЯД НА ЗАБОЛЕВАЕМОСТЬ ХРОНИЧЕСКИМИ ДЕРМАТОЗАМИ.** *Ученый XXI века*, (1 (82)), 3-6.
  22. Исмаилова, Г. О., Юлдашев, Н. М., Акбарходжаева, Х. Н., Шертаев, М. М., & Зиямутдинова, З. К. (2021). Биологически активные природные 2'-гидроксихалконы. *Биоорганическая химия*, 47(3), 304-314.



- 
23. Yusupov, A., Ismailova, M., & Mamatkulov, I. (2024). Changes in the level of stress hormones while using low-opioid anesthesia in children's orthopedics. *Science and innovation*, 3(D4), 323-328.
24. Ashurova, D. T., Ismailova, M. U., Sadikova, R. R., Sharipova, Z. U., & Khodjaeva, I. A. (2024). Anaphylaxis in children: mechanisms of development and modern trends in intensive therapy. *Science and innovation*, 3(Special Issue 54), 69-73.
25. Нарзикулов, У. К., Буриев, М. Н., Рузикулов, У. Ш., Исмаилова, М. У., Сабирджанова, Ч. К., & Нарбекова, Ш. М. (2015). Клиника, диагностика и лечение повреждений проксимального эпиметафиза лучевой кости у детей и подростков. *Молодой ученый*, (11), 687-690.
26. Исмаилова, М. У., & Юсупов, А. С. Анестезиологическая защита детей при хирургической коррекции воронкообразной деформации грудной клетки. *Тиббиётда янги кун.*–Ташкент, 20(22), 9.
27. Турсунова, О. А., & Шарапов, Б. У. (2017). ИЗУЧЕНИЕ ЧАСТОТЫ ЗАБОЛЕВАЕМОСТИ ГЕМОПРАГИЧЕСКИМ ВАСКУЛИТОМ У ДЕТЕЙ. In *INTERNATIONAL INNOVATION RESEARCH* (pp. 236-239).
28. Садирходжаева, А. А. (2025). ОСОБЕННОСТИ ПАРАМЕТРОВ ГЕМОСТАЗА И ЭНДОТЕЛИАЛЬНОЙ ДИСФУНКЦИИ У ДЕТЕЙ С СД 1 ТИПА ПЕРЕНЕСШИХ COVID-19 ИНФЕКЦИЮ. *Medical journal of Uzbekistan*, 1(6), 150-154.
29. Бекбаулиева, Г. Н. (2009). Медико-социальные и организационные направления формирования репродуктивного здоровья населения Приаралья и перспективы их развития. Автореферат на соискание ученой степени доктора мед. наук, 39.
30. Ганиева, Х. С., Бекбаулиева, Г. Н., & Раззакова, Н. С. (2024). ОЦЕНКА КАЧЕСТВА ЖИЗНИ У ПАЦИЕНТОК С ПРЕЖДЕВРЕМЕННОЙ НЕДОСТАТОЧНОСТЬЮ ЯИЧНИКОВ НА ФОНЕ ПРИМЕНЕНИЯ ЗАМЕСТИТЕЛЬНОЙ ГОРМОНАЛЬНОЙ ТЕРАПИИ. II МЕЖДУНАРОДНАЯ НАУЧНО-ПРАКТИЧЕСКАЯ КОНФЕРЕНЦИЯ «РЕПРОДУКТИВНОЕ ЗДОРОВЬЕ ЖЕНЩИН: ПРОБЛЕМЫ, ПУТИ РЕШЕНИЯ И ПРОФИЛАКТИКА».



- 
31. Бекбаулиева, Г. Н., Курбаниязова, М. З., & Шакирова, П. Д. (2023). Профилактика синдрома гиперстимуляции яичников при стимуляции овуляции (Doctoral dissertation, «АКТУАЛЬНЫЕ ПРОБЛЕМЫ ГИНЕКОЛОГИИ»).
32. Бекбаулиева, Г. Н., Шакирова, П. Д., & Курбаниязова, М. З. (2023). Возможности профилактики синдрома гиперстимуляции яичников при индукции овуляции у женщин с хронической ановуляцией (Doctoral dissertation).
33. Бекбаулиева, Г. Н., Шакирова, П. Д., & Курбаниязова, М. З. (2021). ХАРАКТЕРИСТИКА СОМАТИЧЕСКОЙ ПАТОЛОГИИ У ИНФЕРТИЛЬНЫХ ЖЕНЩИН. AGRICULTURAL SCIENCES, 31.
34. Абдубакиева, Ф. Б., Саттарова, К. А., & Бекбаулиева, Г. Н. (2017). Социально-медицинские аспекты репродуктивного здоровья и контрацептивного поведения пациенток с внематочной беременностью. Журнал теоретической и клинической медицины, (2), 122-123.
35. Karimov, Z. D., Jabborov, U. U., Abdikulov, B. S., & Husanhodzhaeva, M. T. (2016). TRAUMAS IN PREGNANT WOMEN: THE MODERN ASPECTS OF THE PROBLEM (THE REVIEW OF THE LITERATURE). Russian Sklifosovsky Journal "Emergency Medical Care", (1), 33-37.
36. Jabborov, U. U., & Raximberganov, A. M. (2025, December). RESPUBLIKADA YANGI TUG'ILGAN CHAQALOQLARNING GEMOLITIK KASALLIGI BILAN KASALLANISHINING KLINIK TAHLILI. In SYMPOSIUM ON ADVANCED STUDIES AND FUTURE DIRECTIONS (Vol. 1, No. 1, pp. 75-78).
37. Jabborov, U. U. (2020). EARLY ULTRASOUND DIAGNOSIS OF FETAL ANEMIA DURING RH-IMMUNIZATION. Новый день в медицине, (4), 419-421.
38. Kapelyushnik, N. L., Sabirov, F. N., Osipov, R. A., Timofeeva, T. I., Maltseva, L. I., Khasanov, A. A., & Kutlylyeva, L. M. (1984). The role of antenatal clinics in the prevention of pregnancy pathology. Kazan medical journal, 65(2), 94-98.



- 
39. Sabirov, F. N. 124-12 (50) 2022—FN Sabirov, UU Jabborov,—FETAL CYTOKINES IN PREGNANT WOMEN WHO HAD COVID-19 IN THE THIRD TRIMESTER OF GESTATION.
40. Саиджалилова, Д., Мадолимова, Н., & Саидмуродова, М. Патологическое Течение Беременности И Родов У Женщин С Эндометриозом. Scientific Collection «InterConf», (190), 213-214.
41. Ходжаева, Д. Н., Аюпова, Д. А., & Мадолимова, Н. Х. К. (2021). Выбор тактики родоразрешения при тяжелой преэклампсии. Re-health journal, (2 (10)), 10-17.
42. Madolimova, N. H. K., & Tursunov, O. A. U. (2016). The course and result of pregnancy and childbirth in a preeclampsia. *Biologiya i integrativnaya medicina*, 5, 40-47.
43. Мадолимова, Н. Х., & Ахмеджанова, Х. З. (2025). АДЕНОМИОЗ И ОСЛОЖНЕНИЙ БЕРЕМЕННОСТИ, РОДОВ И ИХ ПРОГНОЗИРОВАНИЕ. *ОСНОВЫ МЕДИЦИНЫ*, 1(7), 177-187.
44. Мадолимова, Н. Х. К., & Турсунов, О. А. У. (2016). Течение и исходы беременности и родов и при преэклампсии. *Биология и интегративная медицина*, (5), 40-47.
45. Felson, D. T., Lawrence, R. C., Dieppe, P. A., Hirsch, R., Helmick, C. G., Jordan, J. M., & Fries, J. F. (2000). Osteoarthritis: New insights. *Annals of Internal Medicine*, 133(8), 635–646. <https://doi.org/10.7326/0003-4819-133-8-200010170-00015>
46. Fithian, D. C., Kelly, M. A., & Mow, V. C. (1990). Material properties and structure-function relationships in the menisci. *Clinical Orthopaedics and Related Research*, 252, 19–31.
47. Fox, A. J. S., Bedi, A., & Rodeo, S. A. (2015). The basic science of human knee menisci: Structure, composition, and function. *Sports Health*, 4(4), 340–351. <https://doi.org/10.1177/1941738111429419>
48. Hanna, F. S., Wluka, A. E., Bell, R. J., Davis, S. R., & Cicuttini, F. M. (2010). Osteoarthritis and the postmenopausal woman. *Seminars in Arthritis and Rheumatism*, 34(3), 631–636. <https://doi.org/10.1016/j.semarthrit.2004.08.009>



- 
49. Ingman, A. M., Ghosh, P., & Taylor, T. K. (1974). Variation of collagenous and non-collagenous proteins of human knee joint menisci with age and degeneration. *Gerontologia*, 20(4), 212–223. <https://doi.org/10.1159/000211985>
50. Litwic, A., Edwards, M. H., Dennison, E. M., & Cooper, C. (2013). Epidemiology and burden of osteoarthritis. *British Medical Bulletin*, 105(1), 185–199. <https://doi.org/10.1093/bmb/lds038>
51. Liu, S. H., Al-Shaikh, R., Panossian, V., Yang, R. S., Nelson, S. D., Soleiman, N., & Finerman, G. A. M. (2016). Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. *Journal of Orthopaedic Research*, 14(4), 526–533. <https://doi.org/10.1002/jor.1100140405>
52. Makris, E. A., Hadidi, P., & Athanasiou, K. A. (2011). The knee meniscus: Structure-function, pathophysiology, current repair techniques, and prospects for regeneration. *Biomaterials*, 32(30), 7411–7431. <https://doi.org/10.1016/j.biomaterials.2011.06.037>
53. Messner, K., & Gao, J. (1998). The menisci of the knee joint: Anatomical and functional characteristics. *Journal of Anatomy*, 193(2), 161–178. <https://doi.org/10.1046/j.1469-7580.1998.19320161.x>
54. Srikanth, V. K., Fryer, J. L., Zhai, G., Winzenberg, T. M., Hosmer, D., & Jones, G. (2005). A meta-analysis of sex differences in prevalence, incidence and severity of osteoarthritis. *Osteoarthritis and Cartilage*, 13(9), 769–781. <https://doi.org/10.1016/j.joca.2005.04.014>
55. Vos, T., Flaxman, A. D., Naghavi, M., Lozano, R., Michaud, C., Ezzati, M., & Murray, C. J. L. (2012). Years lived with disability for 1160 sequelae of 289 diseases and injuries 1990–2010: A systematic analysis for the Global Burden of Disease Study. *The Lancet*, 380(9859), 2163–2196. [https://doi.org/10.1016/S0140-6736\(12\)61729-2](https://doi.org/10.1016/S0140-6736(12)61729-2)