Sideridis Aristotelis , MD

Orthopaedic Surgeon

ΚΛΙΝΙΚΗ
ΑΓΙΟΣ
ΛΟΥΚΑΣ

1926
ΠΑΟΚ

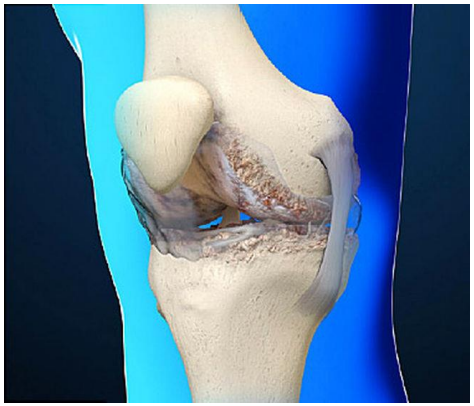
Medical stuff

The MIS
THESSALONIKI MINIMALLY INVASIVE SURGERY
ΟΡΘΟΠΑΙΔΙΚΟ ΚΕΝΤΡΟ

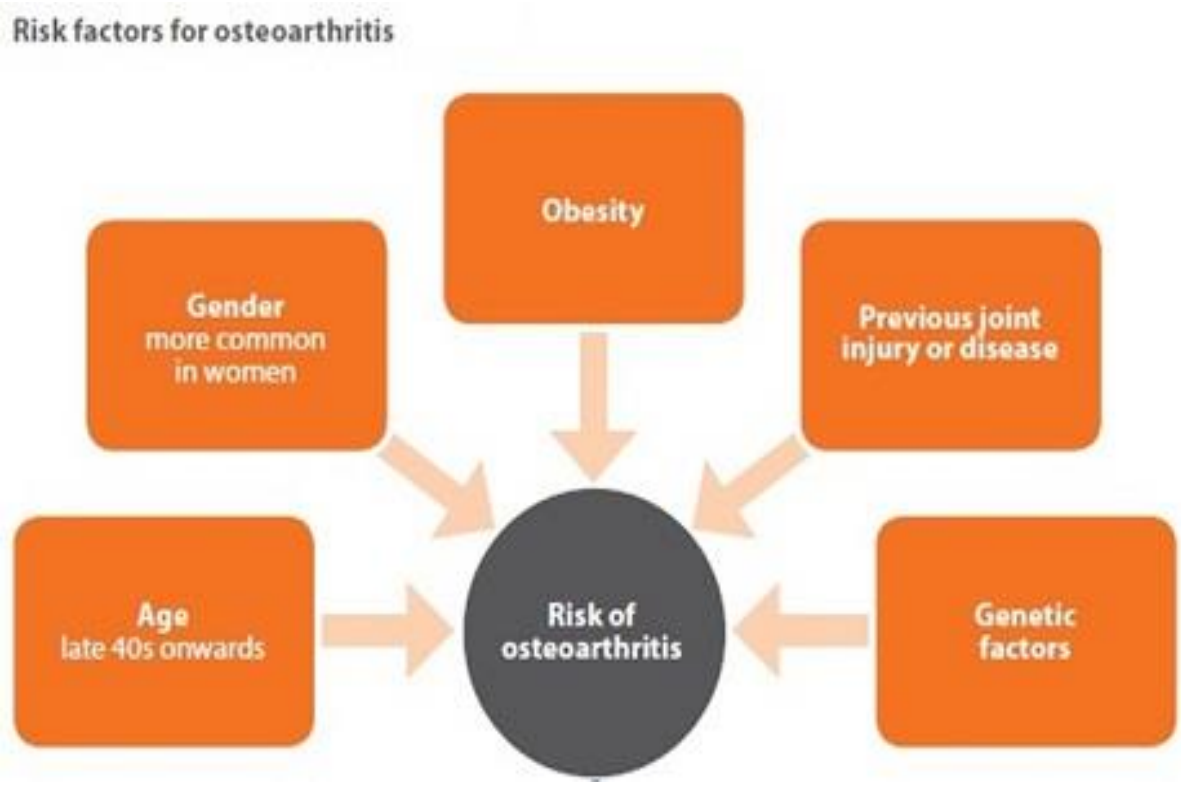
Disclosures

- Lecture fees: Arthrex
- Research grant: Amgen, European Commission, Gallenica S.A.
- Consultancy Agreement: -

Είναι η οστεοαρθρίτιδα των ηλικιωμένων το άθροισμα των κακώσεων του μυοσκελετικού συστήματος της νεαρής ηλικίας;



Osteoarthritis (OA), is a progressive disorder of the joints caused by gradual loss of cartilage and resulting in the development of bony spurs and cysts at the margins of the joints.



Joint Injury, Repair, and Remodeling Roles in Post-Traumatic Osteoarthritis
Joseph A. Buckwalter MD; and Thomas D. Brown, PhD

Osteoarthritis causes

- **Age –** Muscles weakening and body being less able to heal itself.
- **Gender –** For most joints, especially the hips and hands, osteoarthritis is more common and more severe in women.
- **Obesity –** Being overweight is an important factor in causing osteoarthritis, especially in knees.
- **Joint injury –** A major injury on a joint may lead to osteoarthritis.
- **Joint abnormalities –** Born with abnormalities or developed them in childhood, it can lead to earlier and severe osteoarthritis. (Perthes' disease)
- **Genetic factors –** Nodal osteoarthritis, particularly affects the hands of middle-aged women, although it's not yet clear which genes are involved. Genetic factors play a smaller, but still important, part in osteoarthritis of the hip and knee.
- **Other types of joint disease –** Sometimes osteoarthritis is a result of damage from a different kind of joint disease, such as rheumatoid arthritis or gout.

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J Orthop Res. 2011 Jun; 29(6): 802–809.
Published online 2011 Feb 11.

POST-TRAUMATIC OSTEOARTHRITIS: IMPROVED UNDERSTANDING AND OPPORTUNITIES FOR EARLY INTERVENTION

Donald D. Anderson, PhD, Susan Chubinskaya, PhD, Farshid Guilak, PhD, James A. Martin, PhD, Theodore R. Oegema, PhD, Steven A. Olson, MD, and Joseph A. Buckwalter, MS, MD

Acute-impact joint injuries initiate a sequence of biologic events that cause the progressive joint degeneration that leads to PTOA.

Gelber AC, Hochberg MC, Mead LA, et al. Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. Ann Intern Med. 2000;133:321–328.

- Joint surface **incongruity and instability** that follow some joint injuries play a critical role in **PTOA**, a problem that also needs attention.
- The time course over which clinically measurable OA develops is highly variable, ranging from **two to five years** in the case of certain articular fractures, **to decades** for less severe joint injuries, and that is another problem that requires attention.

Roos H, Lauren M, Adalberth T, et al. Knee osteoarthritis after meniscectomy: prevalence of radiographic changes after twenty-one years, compared with matched controls. Arthritis Rheum. 2008;41:687–693.

- 24% of those who had a knee injury during adolescence and young adulthood developed **knee OA**, compared with 6% of those who did not have a knee injury.
- People who suffered knee ligamentous and meniscal injuries had a **ten-fold increased risk of OA** as compared with those who did not have a joint injury.

Weigel DP, Marsh JL. High-energy fractures of the tibial plateau. Knee function after longer follow-up. J Bone Joint Surg Am. 2012;84:1541–1551

- Between 23% and 44% of patients develop knee OA after articular fractures of the knee and
- >50% of patients with fractures of the distal tibial articular surface develop ankle OA

Brown TD, Johnston JC, Saltzman CL, et al. Posttraumatic osteoarthritis: a first estimate of incidence, prevalence, and burden of disease. J Orthop Trauma. 2016;20:739–744.

- A study of patients presenting with disabling hip, knee, and ankle OA showed that 1.6% of patients with **hip** OA, 39.8% of patients with **knee** OA, and 79.5% of patients with **ankl**e OA had a verified history of one or more joint injuries

Joint Injury in Young Adults and Risk for Subsequent Knee and Hip Osteoarthritis

Allan C. Gelber, MD, MPH, PhD; Marc C. Hochberg, MD, MPH; Lucy A. Mead, ScM; Nae-Yuh Wang, MS, PhD; Fredrick M. Wigley, MD; Michael J. Klag, MD, MPH

Results:

Over a median follow-up of 36 years, 141 participants reported joint injuries (knee alone [*n* = 111], hip alone [*n* = 16], or knee and hip [*n* = 14]) and 96 developed osteoarthritis (knee alone [*n* = 64], hip alone [*n* = 27], or knee and hip [*n*= 5]). The cumulative incidence of knee osteoarthritis by 65 years of age was 13.9% in participants who had a knee injury during adolescence and young adulthood and 6.0% in those who did not (*P* = 0.0045) (relative risk, 2.95 [95% CI, 1.35 to 6.45]). Joint injury at cohort entry or during follow-up substantially increased the risk for subsequent osteoarthritis at that site (relative risk, 5.17 [CI, 3.07 to 8.71] and 3.50 [CI, 0.84 to 14.69] for knee and hip, respectively). Results were similar for persons with osteoarthritis confirmed by radiographs and symptoms.

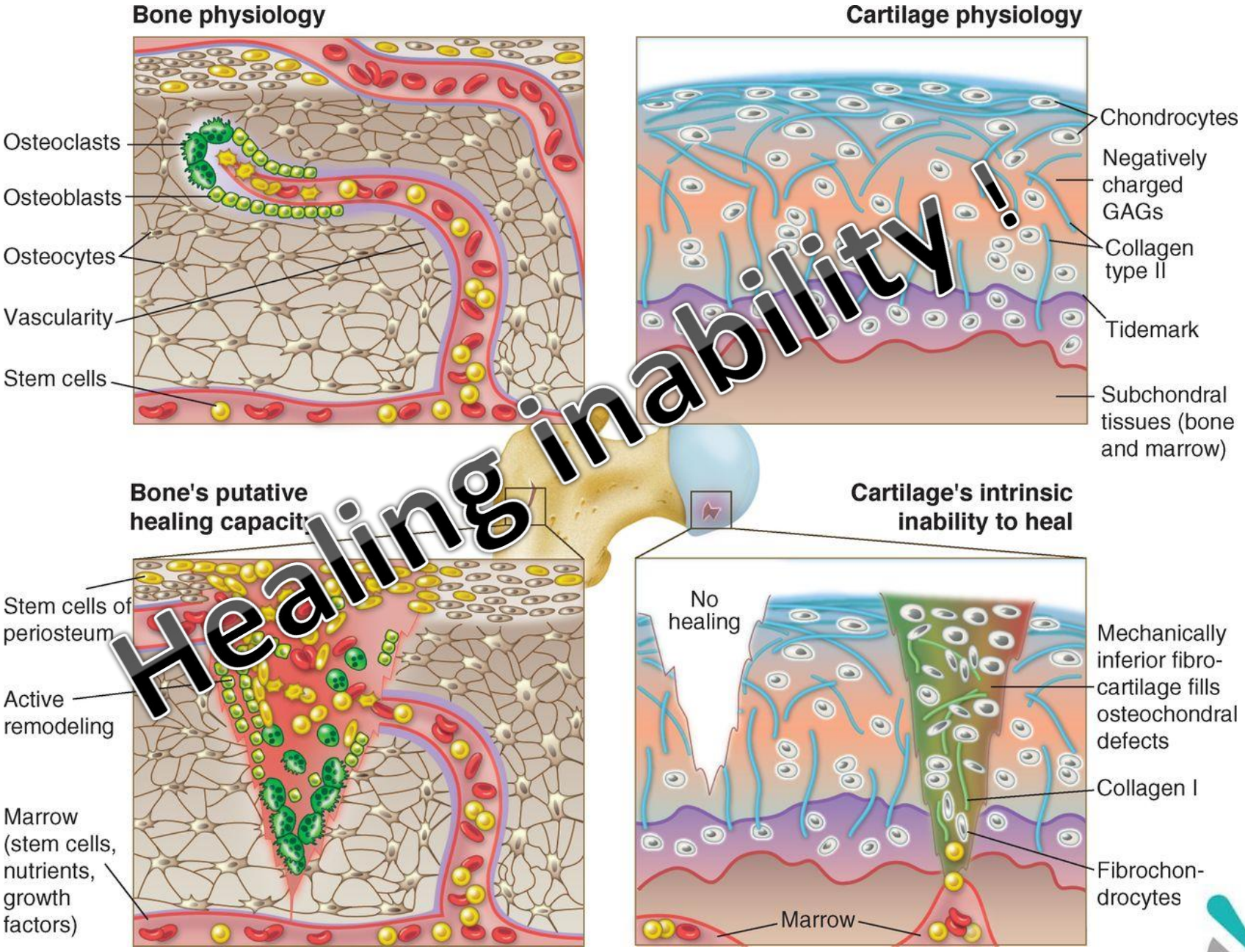
Conclusions:

Young adults with hip & knee injuries are at considerably increased risk for osteoarthritis later in life and should be targeted in the primary prevention of osteoarthritis.

Post-traumatic arthritis: overview on pathogenic mechanisms and role of inflammation

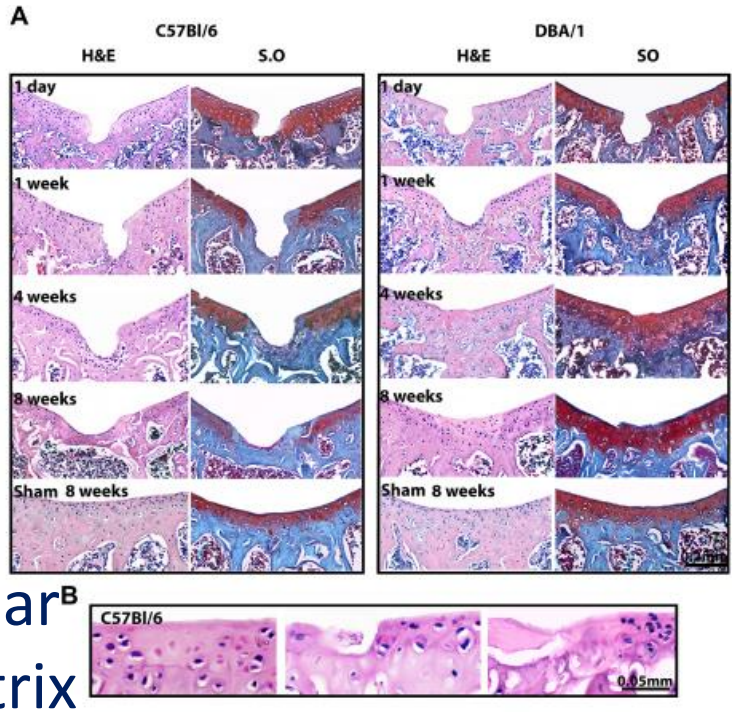
Leonardo Punzi, Paola Galozzi, Roberto Luisetto, Marta Favero, Roberta Ramonda, Francesca Oliviero and Anna Scanu 2017 Reum. I

- A history of physical trauma may also be found in patients with **chronic inflammatory arthritis** with the percentage varying from 2% to 25%.
- Trauma is one of the most frequent causes of recurrence of acute attack in **gout and pseudogout** as a consequence of crystal shedding.
- Furthermore, it has been reported that patients who experienced significant joint injuries have an increased risk of developing **calcium pyrophosphate** and basic calcium phosphate crystal deposits.
- Different studies support the idea that a previous physical trauma may also be considered pathogenically relevant in other categories of inflammatory chronic arthritis, including **rheumatoid arthritis (RA), juvenile chronic arthritis**, juvenile or adult onset Still's disease and **spondiloarthritis,** especially PsA.


Bone physiology
Osteoclasts
Osteoblasts
Osteocytes
Vascularity
Stem cells
Cartilage physiology
Chondrocytes
Negatively charged GAGs
Collagen type II
Tidemark
Subchondral tissues (bone and marrow)
Healing inability !
Bone's putative healing capacity
Stem cells of periosteum
Active remodeling
Marrow (stem cells, nutrients, growth factors)
Cartilage's intrinsic inability to heal
No healing
Mechanically inferior fibro-cartilage fills osteochondral defects
Collagen I
Fibrochon-drocytes
Marrow

Superficial Injuries of Cartilage

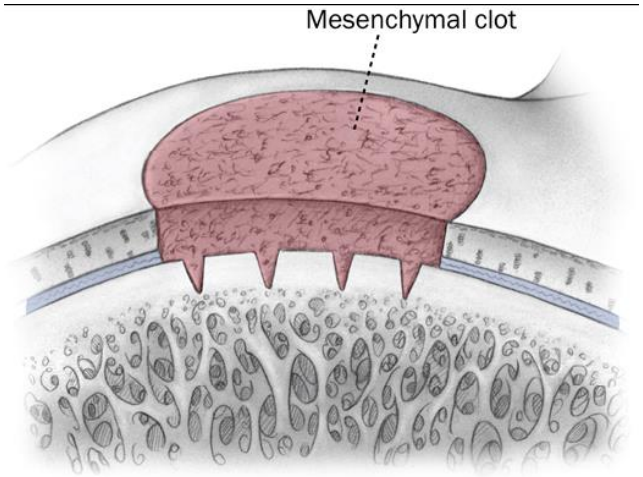
- Defects remain unchanged – no blood clot formation, limited cellular response
- Fibrine clot formation
- Fibroblasts with collagen fibers replace clot
- 1 month : Some chondrocytes appear
- 2 months : Lesion is covered by matrix (collagen 1)
- 6 months : No more healing process fibrocartilage thin and soft



A novel in vivo murine model of cartilage regeneration. Age and strain-dependent outcome after joint surface injury Osteoarthritis and Cartilage 17(6):695-704 · December 2008

Subchondral bone injury

- Blood clot - granulation tissue
- Stem cells fron subchondral bone migrate
- Collagen type 1 & 2
- Hyaline like cartilage (depends on size)
- Thinner than normal
- Osteophytes
- MF

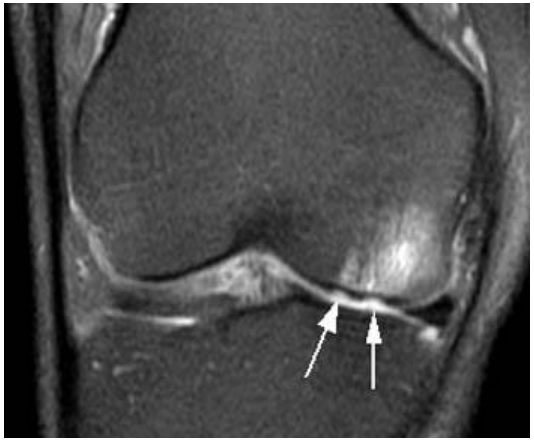


Orthop Clin North Am. 2005 Oct;28(5):847-64.
Cartilage wound healing. An overview

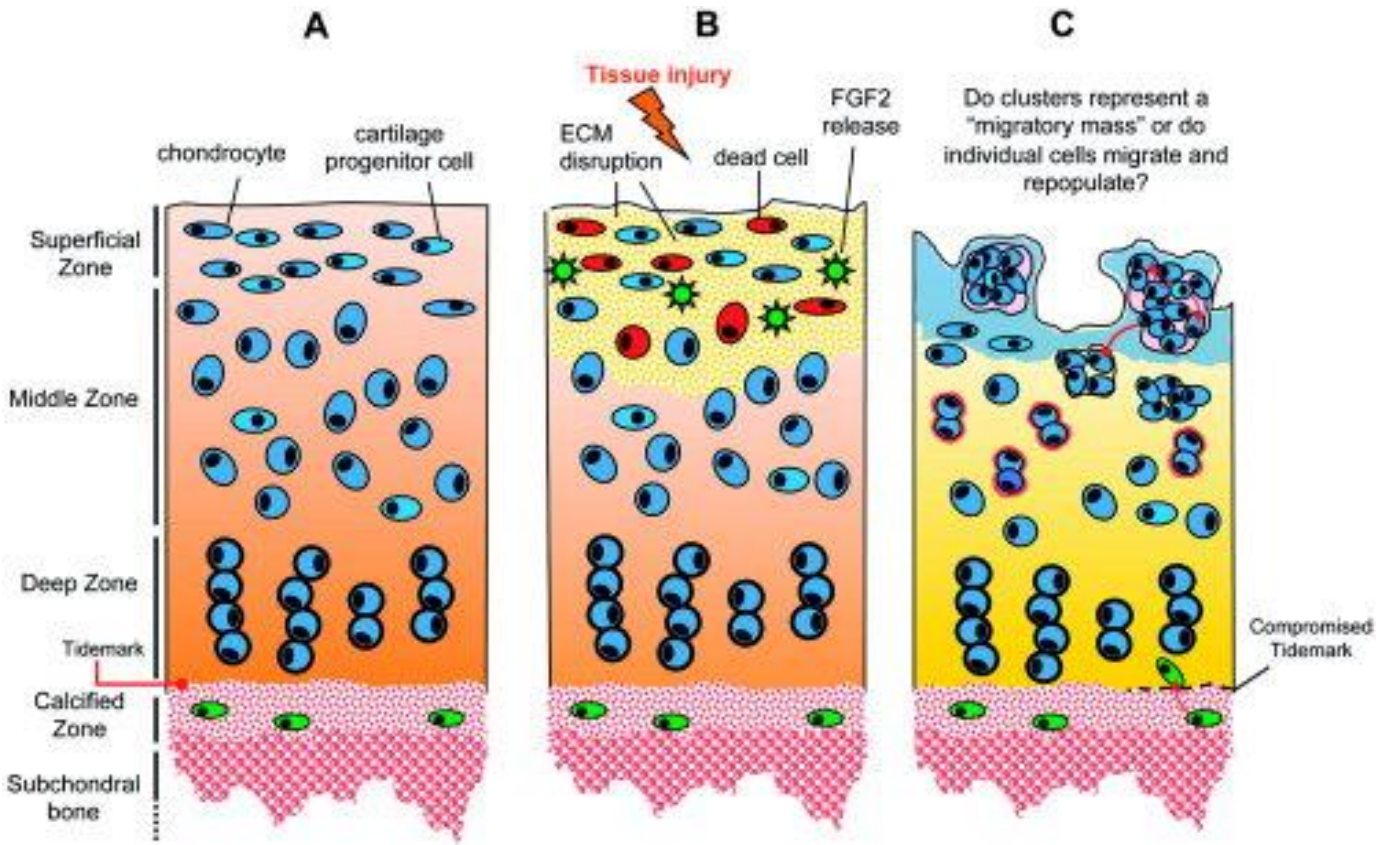
- Cartilage wound healing is a tentative balance between deposition of **type I collagen in the form of scar tissue** and **repair by expression of type II collagen and proteoglycans**.
- Small full-thickness cartilage defects are replaced by **fibrocartilage,** whereas partial-thickness defects are either normally repaired by deposition of **fibrous scar tissue or not healed at all**.

Types of joint injury

1) damage to cells and/or matrices that **does not include macroscopic structural disruption** of cartilage or bone,
2) damage to cells and/or matrices along **with macroscopic structural disruption** of articular cartilage without displaced bone fracture (these injuries may be associated with microfractures of the calcified cartilage and in some instances of the subchondral or trabecular bone), and



3) **displaced fractures** of the articular surface extending through cartilage and bone.


A
B
C
chondrocyte
cartilage progenitor cell
Tissue Injury
ECM disruption
dead cell
FGF2 release
Do clusters represent a "migratory mass" or do individual cells migrate and repopulate?
Superficial Zone
Middle Zone
Deep Zone
Tidemark
Calcified Zone
Subchondral bone
Compromised Tidemark

Furman BD, Strand J, Hembree WC, et al. Joint degeneration following closed intraarticular fracture in the mouse knee: a model of posttraumatic arthritis. J Orthop Res. 2007;25:578–592

- articular fracture causes significant **chondrocyte death** at the fracture.
- such an injury leads to the development of osteoarthritis-like changes in the **cartilage, bone, synovium, and other joint tissues**
- **rapid changes in serum and synovial fluid** concentrations of cartilage biomarkers and pro-inflammatory cytokines

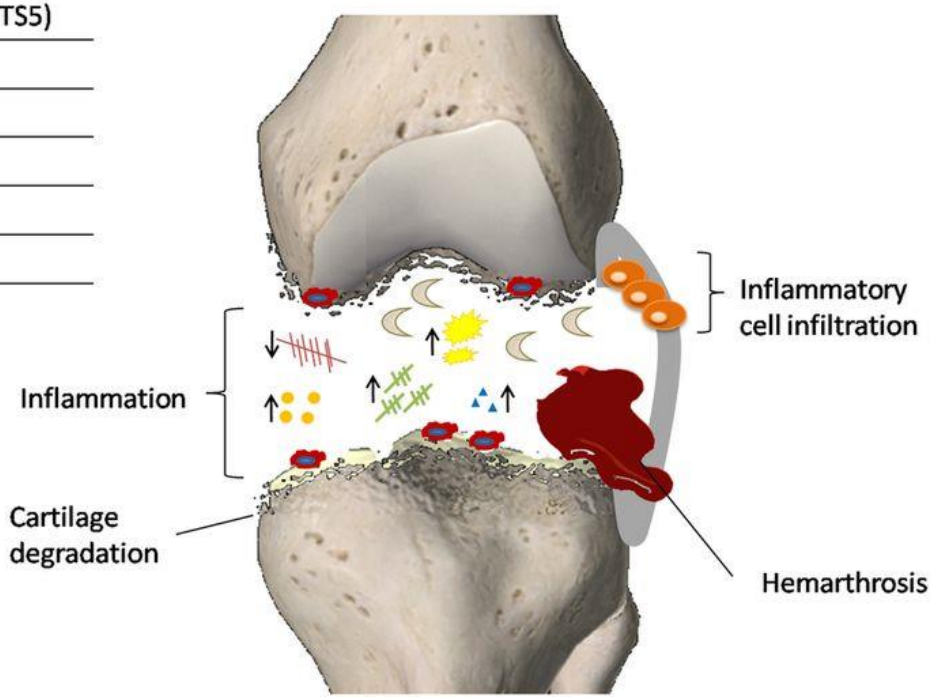
Martin JA, McCabe D, Walter M, et al. N-acetylcysteine inhibits post-impact chondrocyte death in osteochondral explants. J Bone Joint Surg Am. 2009;91:1890–1897.

- that impact injury stimulates **release of oxygen free radicals from chondrocytes**, which leads to progressive chondrocyte damage and matrix degradation
- superoxide released by damaged components of the mitochondrial electron transport chain that **mitochondrial dysfunction could be responsible for chondrocyte mortality in joint injuries** that involve blunt trauma to articular surfaces

Post-traumatic arthritis: overview on pathogenic mechanisms and role of inflammation

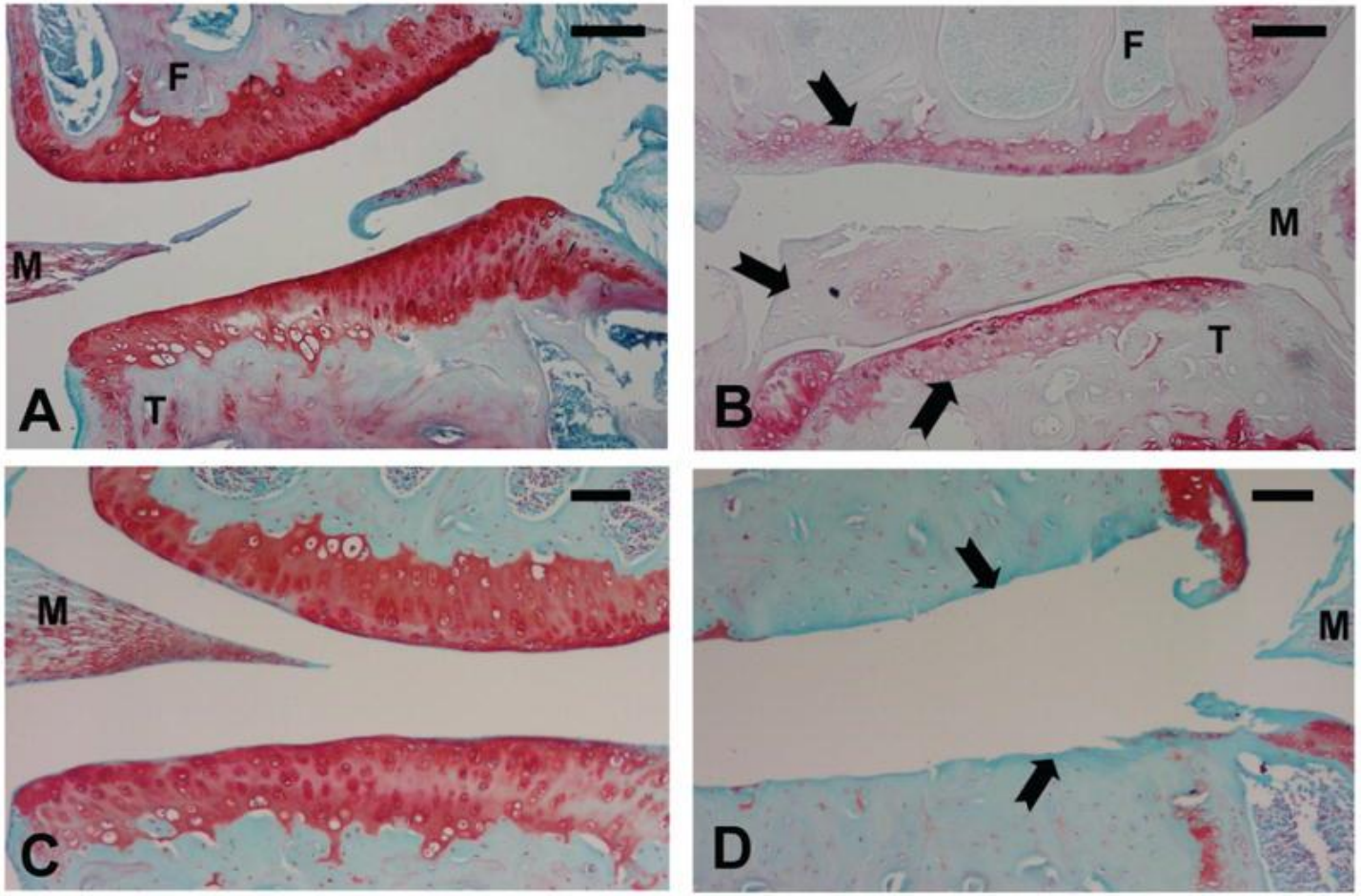
Leonardo Punzi, Paola Galozzi, Roberto Luisetto, Marta Favero, Roberta Ramonda, Francesca Oliviero and Anna Scanu 2017

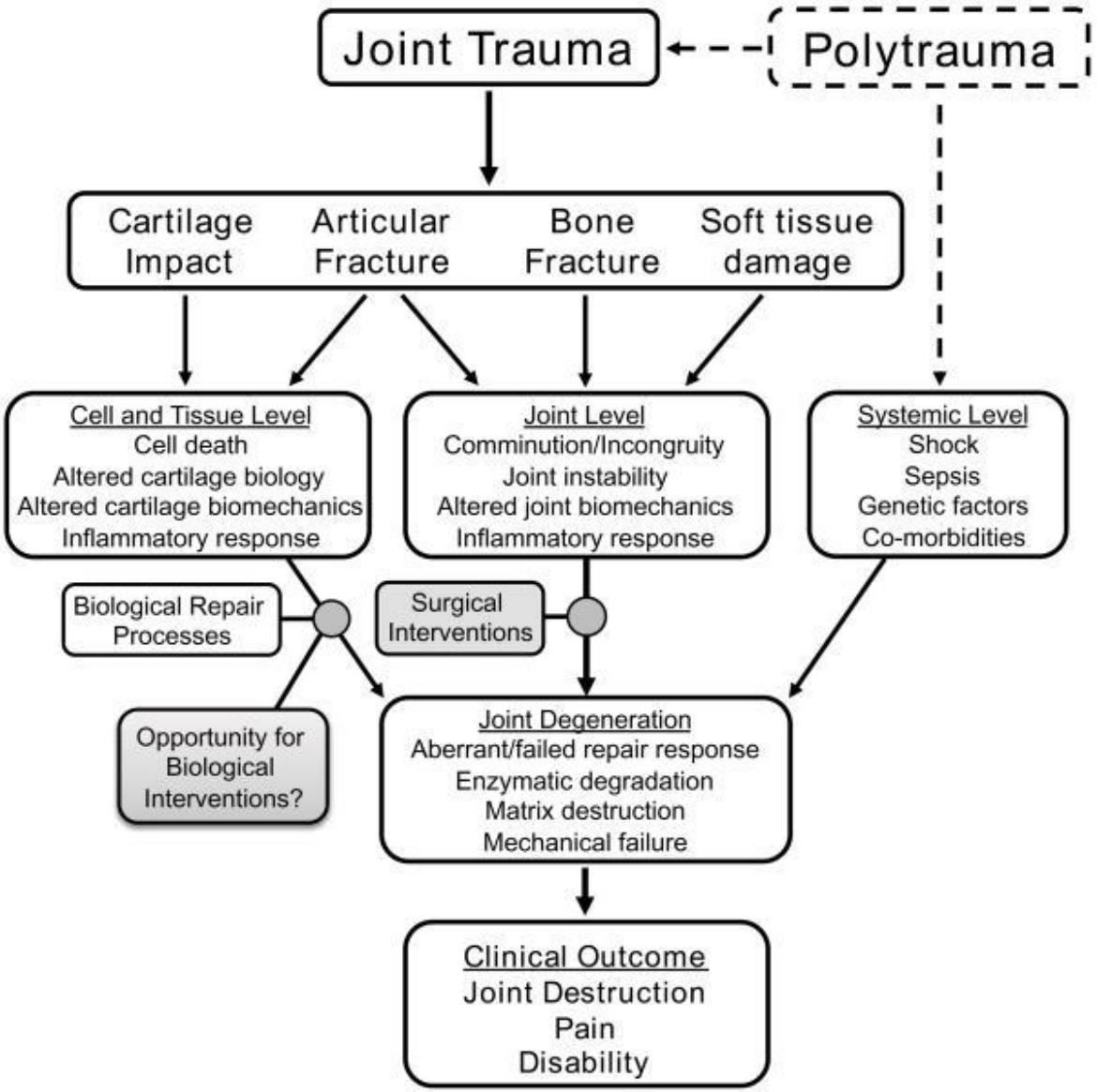
	Cytokines (IL-1, IL-6, IL-8, TNF)
	Metalloproteinase (MMP-1, -3, -8, -9, -13; ADAM-TS5)
	Proteoglycan
	Chondrocyte death
ROS	Reactive oxidant species ($O_2^{\bullet-}$)
	Lubricin
	Cartilage fragments
	Inflammatory cells (macrophages, lymphocytes)



J Orthop Res. 2011 Jun; 29(6): 802–809

Histologic sections of control (left) and experimentally fractured (right) mouse knee joints at 8 (A,B) and 50 (C,D) wks following joint injury





J Orthop Res. 2011 Jun; 29(6): 802–809.
Published online 2011 Feb 11

Prevention ?

Biochemical Prevention?

- Anti-inflammatory interventions may represent the best available opportunity to intervene early in the acute post-traumatic period. **Anticytokine therapy** has demonstrated a marked efficacy as preventative agents of the long-term onset of chronic PTA. IL-1 inhibition, through knockout of IL-1β**, intra-articular injection or adenoviral transfer** of IL-1Ra and retroviral transduction to overexpress IL-1Ra, is resulted therapeutically effective in animal models of surgically induced PTA.
- **Blocking of TNF** increased the production of lubricin and decreased the release of GAG, resulting in a chondroprotective effect in a rat model . However, although cytokines play a role in the post-traumatic acute phase, different studies performed in mouse models assert that intra-articular inhibition of IL-1, rather than TNF, **may reduce the development of chronic PTA**.
- Despite the use of all these agents has proven effective in reducing the progression of chronic PTA in animal models, only one **small randomised pilot clinical trial** has been conducted. Currently, IL-1Ra is the only agent that has been used as an anticytokine approach in patients with acute PTA. In this study, it has been observed **that IL-1Ra injected intra-articularly within 30 days of ACL injury** (n=6) reduced pain and improved function at 2 weeks compared to placebo (n=5).Although this strategy has proved to be efficacious in the early postinjury phase, the results obtained have not been confirmed in larger studies***. Moreover, the ability of IL-1 inhibition to prevent the long-term onset of chronic PTA has not been demonstrated***

- ***ICRS MACAO 2018***.

What's Evidence based?

- ACL reconstruction
- Meniscus Repair
- Cartilage Repair

ACL Injury

- In a population of patients with ACL tear, high levels of **IL-1ß, IL-6 and IL-8** have been found in the SF collected within 48 hours after injury . Interestingly, they observed persistent lower levels of the **IL-1 receptor antagonist (IL-1Ra)** hypothesising an imbalance of this protecting factor induced by trauma at least at disease onset.

- Raised SF concentrations of IL-6 after joint injury along with those of **monocyte chemoattractant protein (MCP)-1**, **macrophage inflammatory protein-1ß and interferon (IFN)-γ** indicate a postinjury activation of inflammatory pathways in cells of the synovium and cartilage.

- Early signs of articular cartilage degradation, such as elevated **MMPs** production**, collagen-type II peptide release**, **proteoglycan degradation** and bone marker release, appear in the SF from patien following knee injury.

ACL rupture complications leading to OA

- Functional problems in the ACL instability, in activities requiring pivoting can result in intraarticular damage, in particular **meniscal tears** and **cartilage lesions**.
- The majority of patients with ACL deficient knees are able to walk normally however, indicated that up to **44% of patients will develop significant functional disability** affecting their ability to perform activities of daily living.

Open Orthop J. 2012; 6: 295–300. Published online 2012 Jul 27.

Anterior Cruciate Ligament Rupture and Osteoarthritis Progression

James Min-Leong Wong, Tanvir Khan, Chethan S Jayadev, Wasim Khan and David Johnstone

- The relationship between **osteoarthritis and ACL rupture** is one of the main reasons cited for advocating ACL reconstruction in the young, active patient but is difficult to define. There is a great variability in the reported rate of osteoarthritis after ACL rupture in the published literature, ranging between **34% to 86%** .

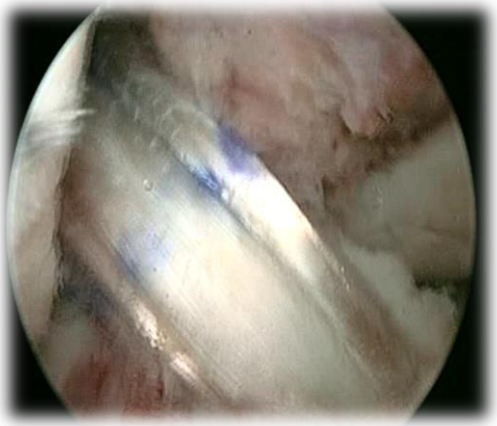
10TH Biennial
ISAKOS CONGRESS 2015
JUNE 7–11, 2015 LYON, FRANCE

Return to soccer following ACL reconstruction

TheMIS Orthopaedic Center E. Papacostas, E. Papasoulis, I. Terzidis, A. Sideridis
St. Luke' s Hospital, Thessaloniki HELLAS

January 2004 – September 2017
Single Surgical technique
Same post-op rehabilitation protocol

- 116 patients
- 119 knees
- 116 included
- Age: 22.7 (14 – 38)
- Tegner score 7: 56 9: 60

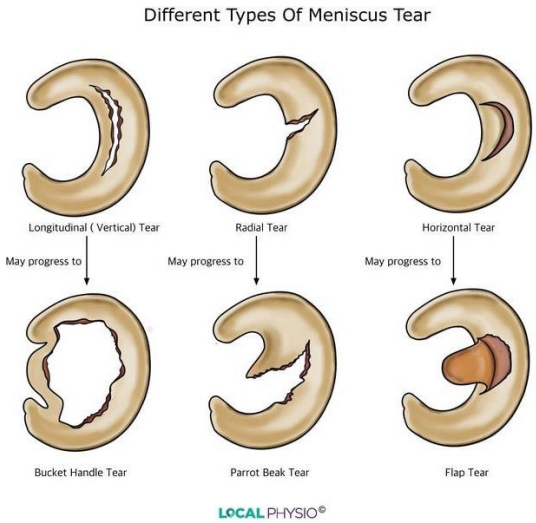


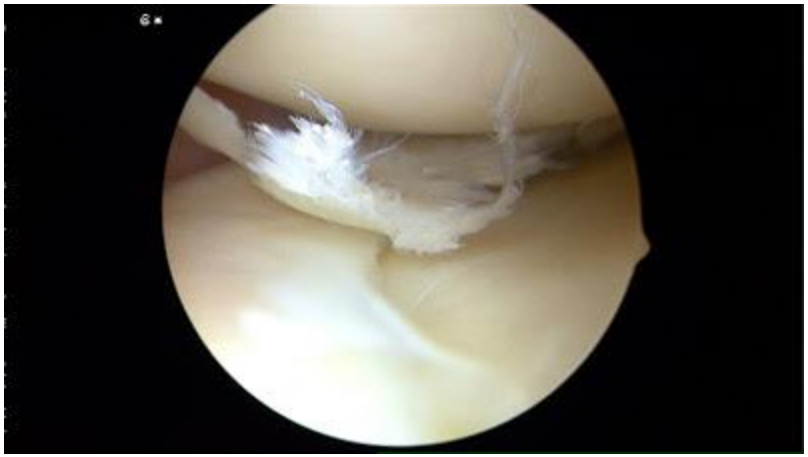
1 FAILED ATTEMPT TO RETURN TO SPORTS at 7 months post-op
115 RETURNED TO SPORTS

111 SAME LEVEL

4 DETERIORATED FROM TEGNER 9 TO TEGNER 7

Meniscus injury





- Knee osteoarthritis in young adults is **common after meniscus injury**. In women who sustained a meniscus injury in soccer, 51% (mean age 31) had radiographic changes after 12 years. Men, 41% (mean age 36) had mild osteoarthritis after 14 years.
- An injury to the menisci during middle age resulting in a horizontal tear is more probably the **first signal of an already ongoing osteoarthritis process of the knee**.

Curr Opin Rheumatol. 2005 Mar;17(2):195-200.
Joint injury causes knee osteoarthritis in young adults.
Roos EM.

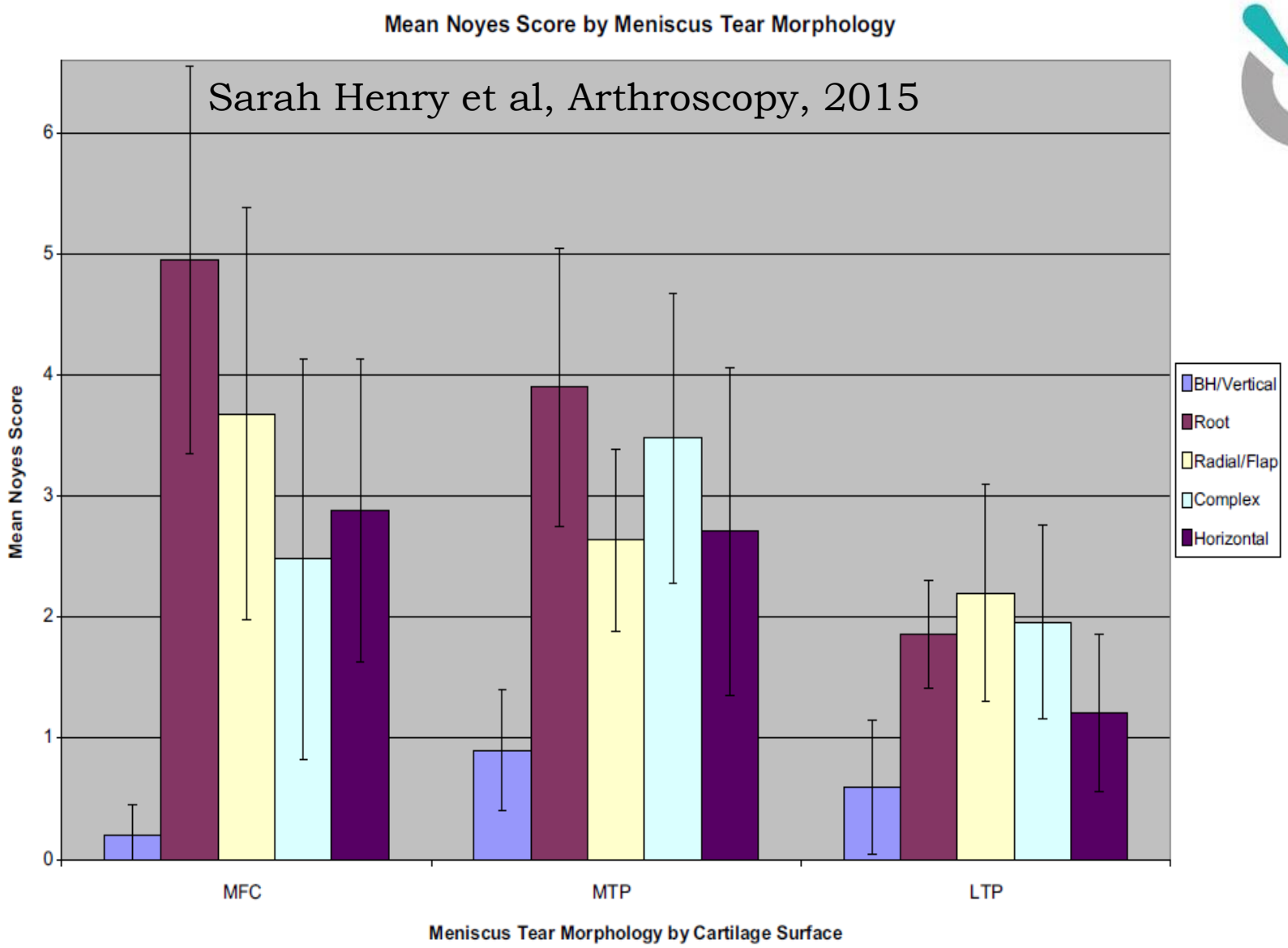


FIGURE 2. Mean Noyes scores for each meniscus tear morphology on MFC, MTP, and LTP surfaces. The mean Noyes score (y-axis) represents severity of cartilage degeneration, with a higher score representing more severe cartilage wear. The Noyes score is displayed for each type of meniscus tear (BH/vertical, root, radial/flap, complex, and horizontal) for each surface of the knee (MFC, MTP, and LTP).

Meniscectomy

- Removal of part or all of the load-bearing meniscus is also associated with **knee OA**.
- In a review of 41 studies of surgically treated isolated meniscus tears (mean age 30 years, with adolescents included in some studies), radiographic knee **OA was present in approximately 50% of those who had undergone meniscectomy 10 to 20 years earlier**.
- In long-term follow up of **young athletes undergoing meniscus surgery, more than 50% developed knee OA** with accompanying pain and physical decline.

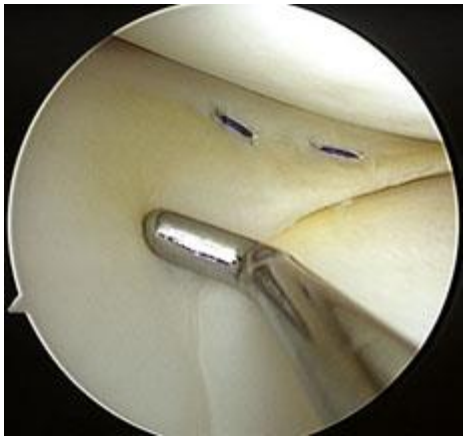
Sarah Henry et al, Arthroscopy, article in press 2015

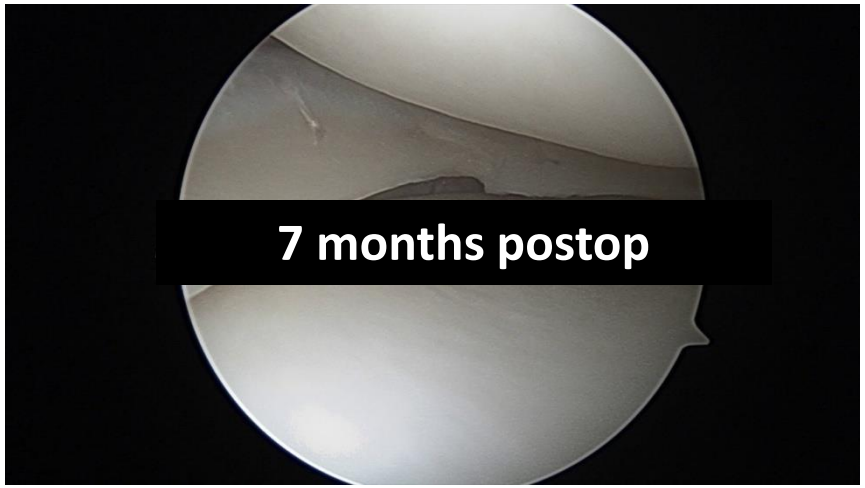
Shave the meniscus

- TheMIS Orthopaedic Center
- 2003-2015
- 1 κέντρο – 3 χειρουργοί
- 185 ασθενείς
- 191 γόνατα
- 192 συρραφέντες ρήξεις μηνίσκων
- Έσω μηνίσκος: 88 (62%)
- Έξω μηνίσκος: 54 (38%)
- Red – red zone: 89 (63 %)
- Red – white zone: 52 (37%)
- 22 αποτυχίες συρραφής
  - 11,4% Μηνισκεκτομή

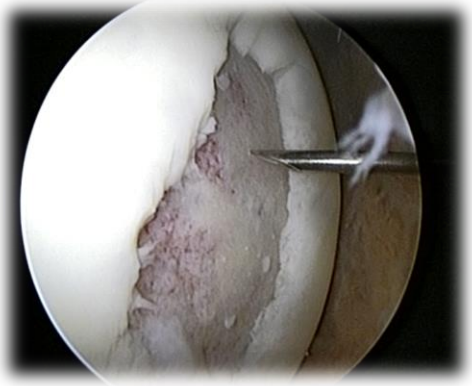
Shave the meniscus

- Επιστροφή σε αθλητική δραστηριότητα: 7,4 μήνες (4-14)
- Η επιτυχία της all-inside τεχνικής συρραφής του μηνίσκου σε αθλητές στη σειρά μας ήταν 88,6% και δεν σχετιζόταν με το μέγεθος της βλάβης ή την ταυτόχρονη αποκατάσταση του ΠΧΣ
- Καλή ακτινολογική εικόνα στο μακρό follow up (5,5 έτη)





Cartilage Lesions
is it really a problem??



So silent

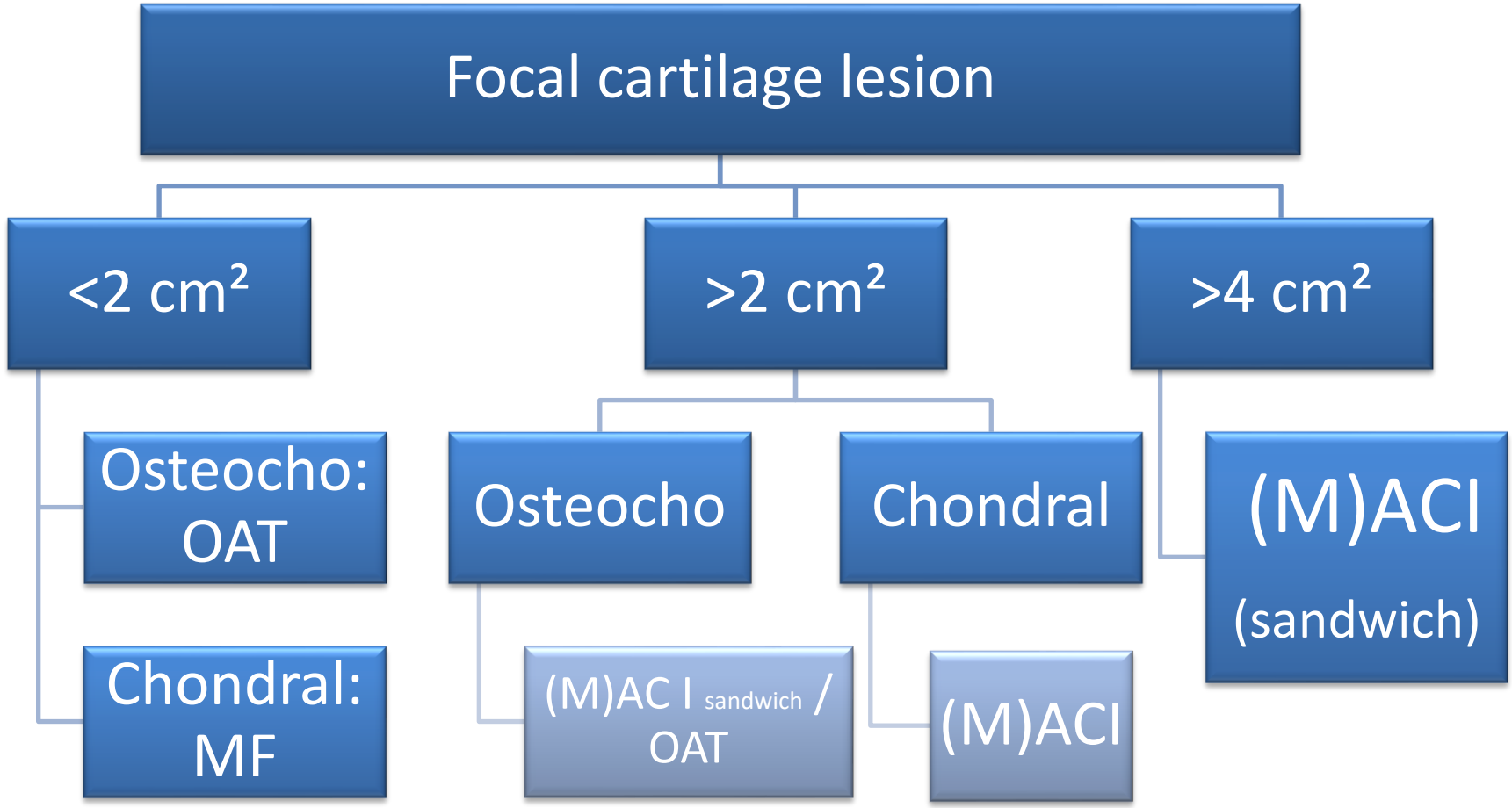
So common

67% of arthroscopies

> 65% lead to PTOA

Australian burden 2017 – WHO ref. p2014

Bekkers Cartilage Sup 2012



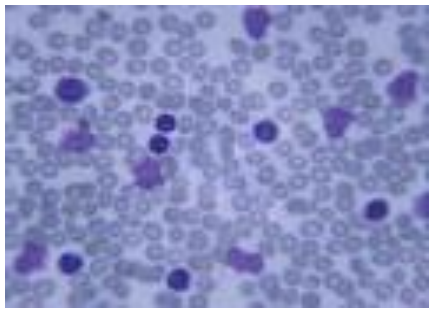
Bekkers AJSM 2009
Gudas Arthroscopy 2005
Bentley JBJS Br 2003

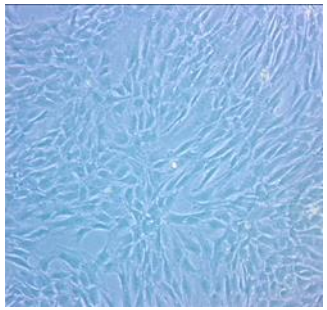
Knutsen JBJS Am 2007
Kon AJSM 2009
Saris AJSM Sup 2009

3rd generation ACI

HIV, HBSag, HCV, Shyphilis

Culture (1st phase)

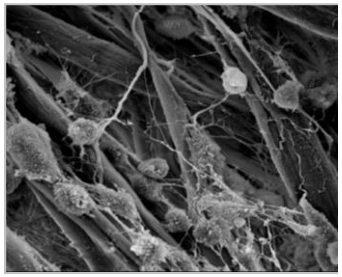




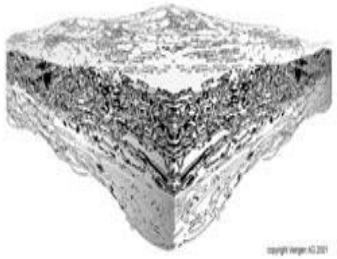
- Chondrocyte release by enzymatic digestion
- Expansion in culture from 100.000 to 20 million cells
- Cryopreservation

3rd generation ACI

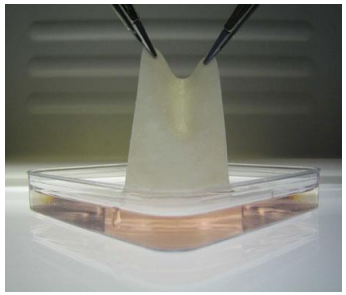
2nd phase – determination of implantation date



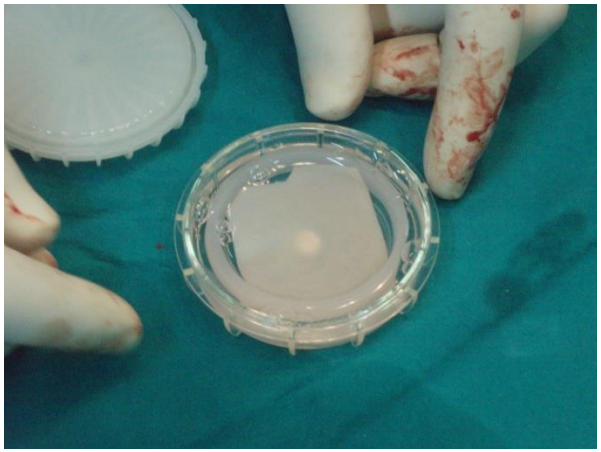
•Cells seeded in biomembrane (collagen I-III, scaffold)

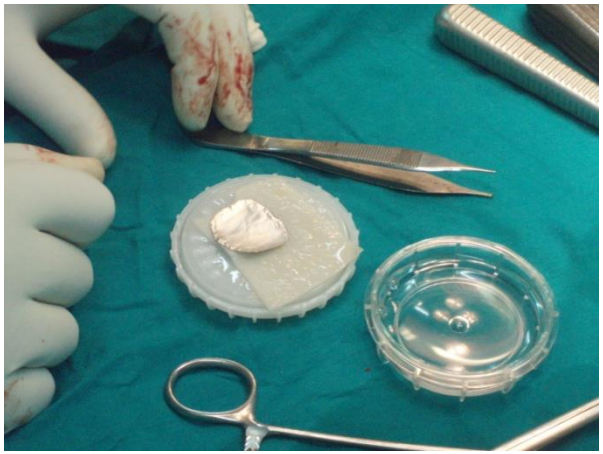


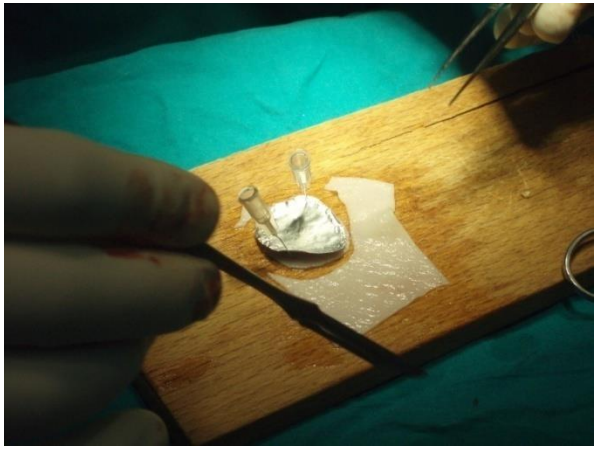
•Further cells expansion

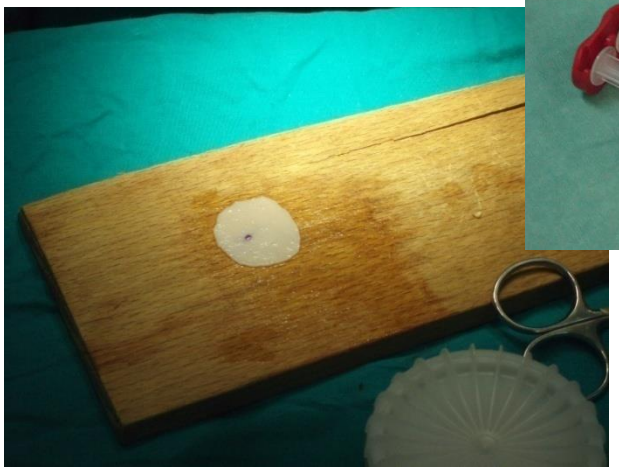


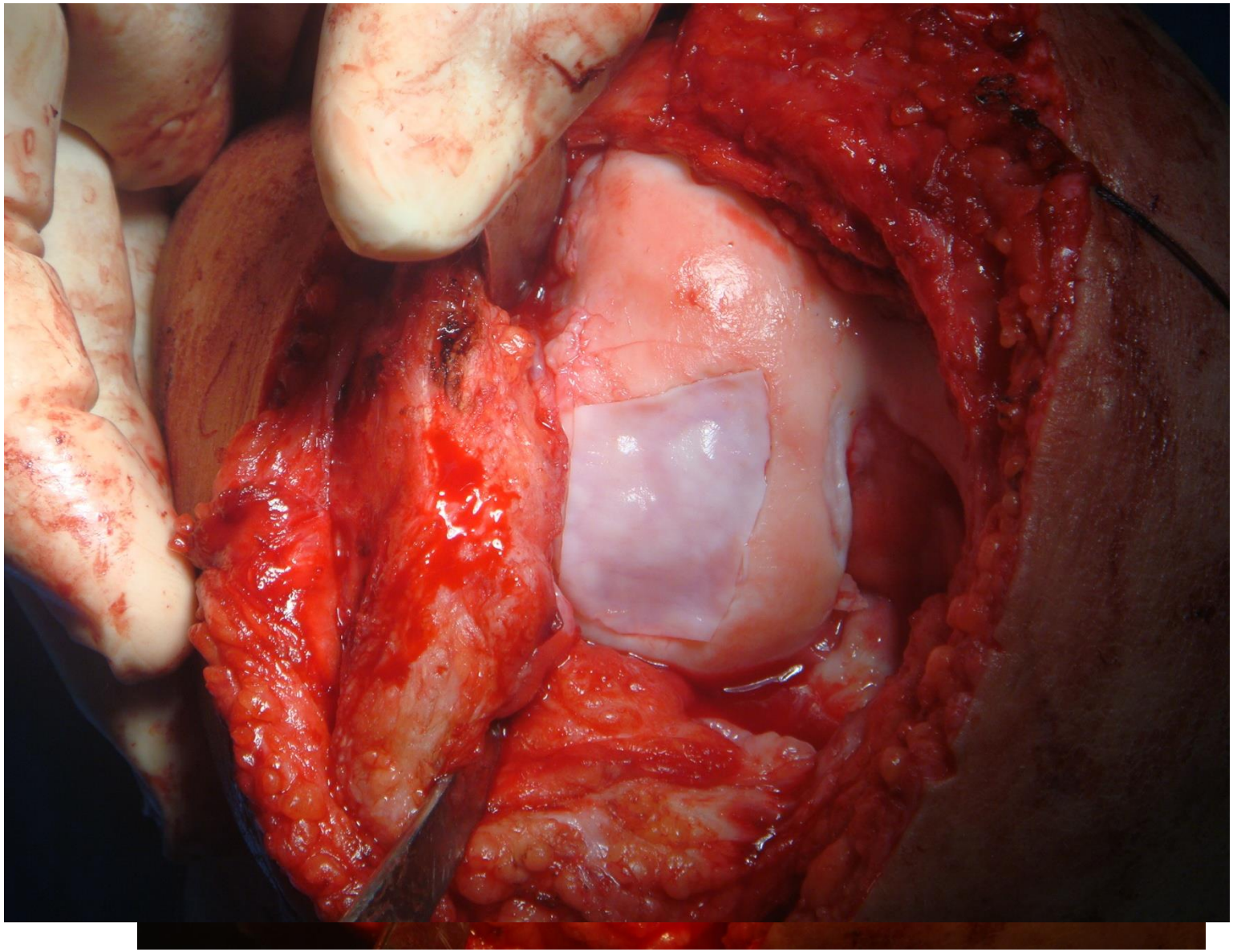
• Membrane final form



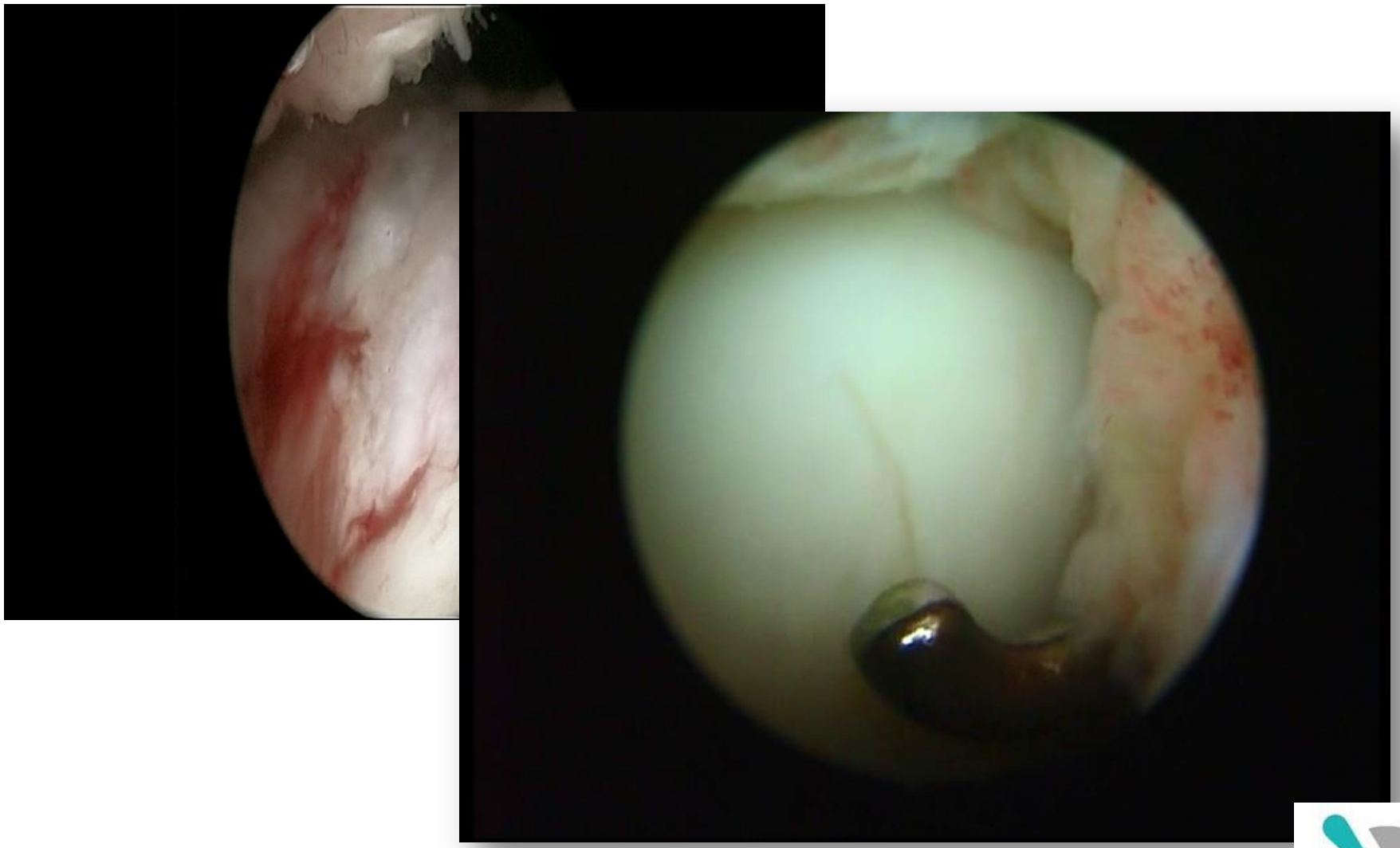






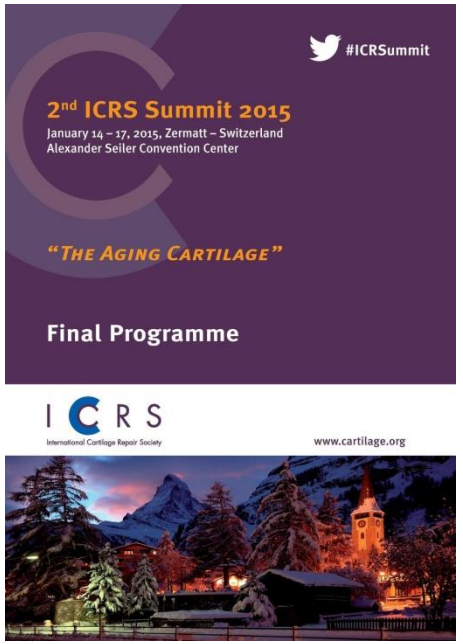


2nd look

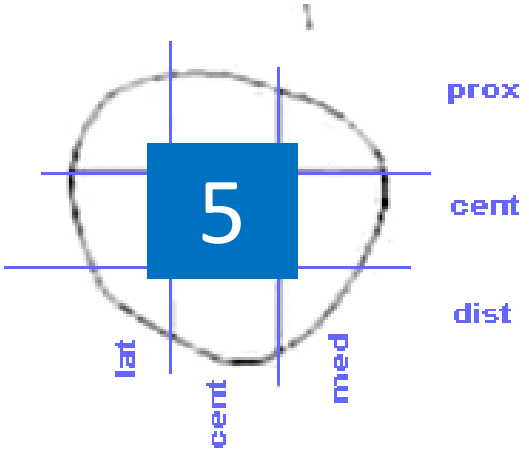


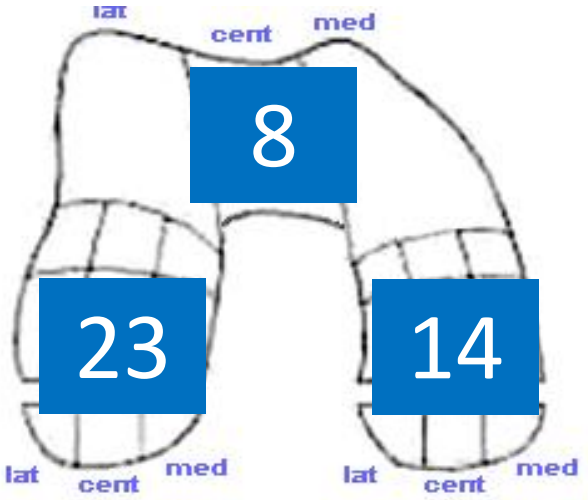
Our Experience
Results 5 years

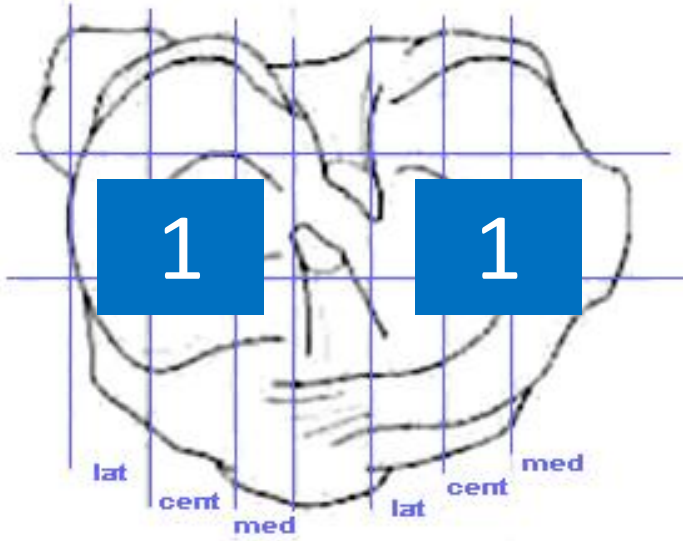
- 39 pts
- 24 male, 15 female
- Age: 28,63 (±10,12) yo
- 27 athletes



Location





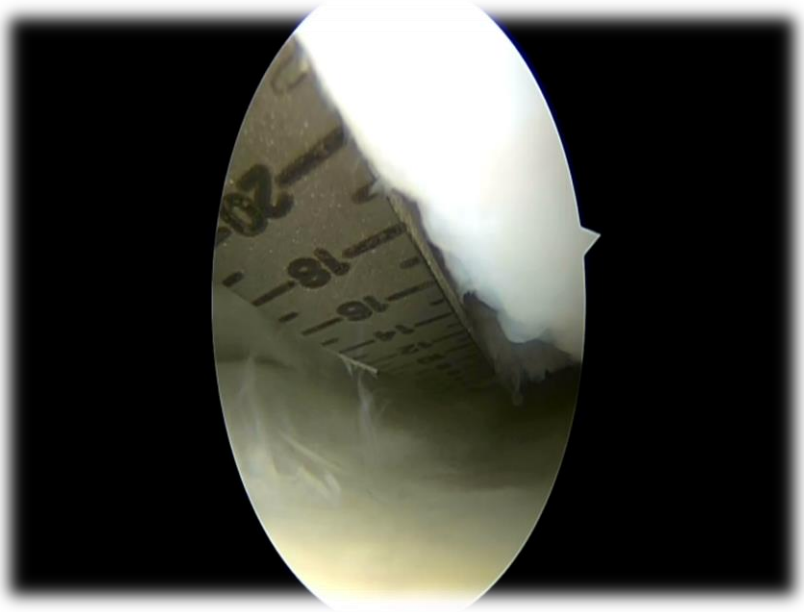


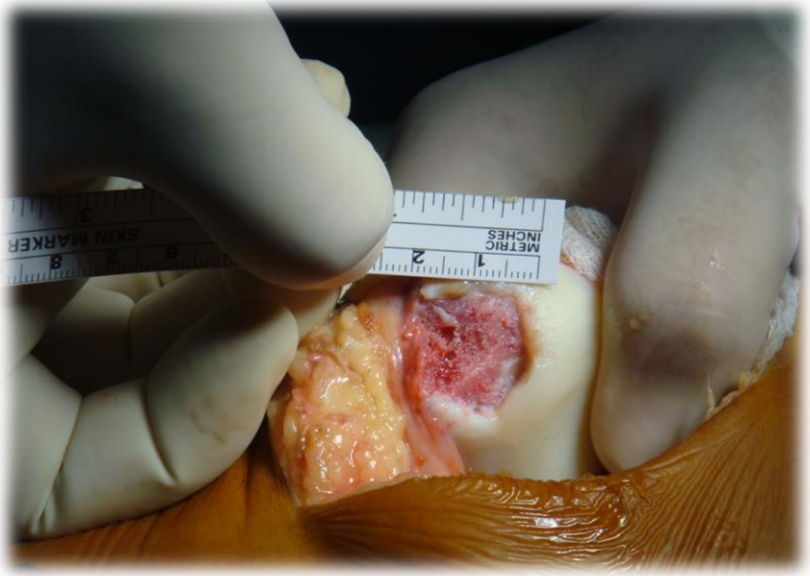
52 lesions

Defect size 5Y

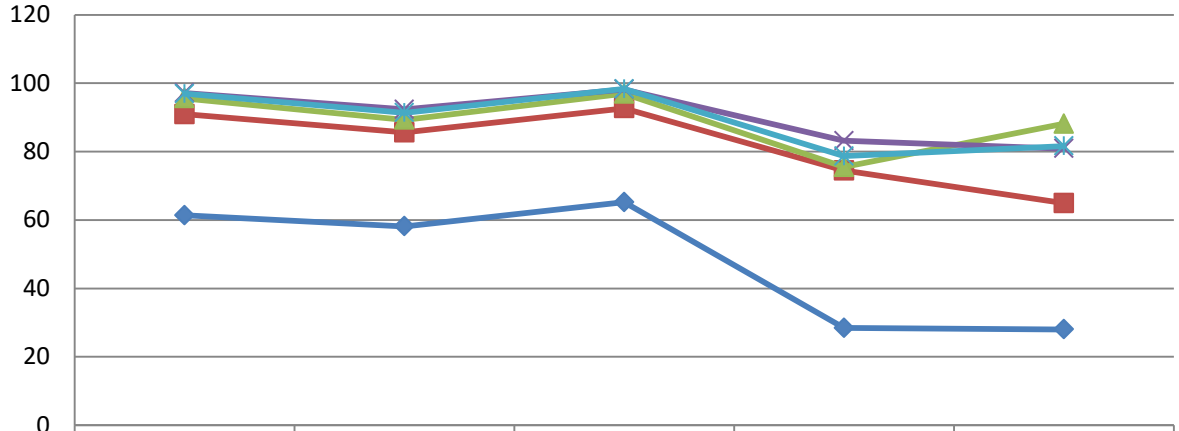
6,63 cm^2

± **2,18** cm^2



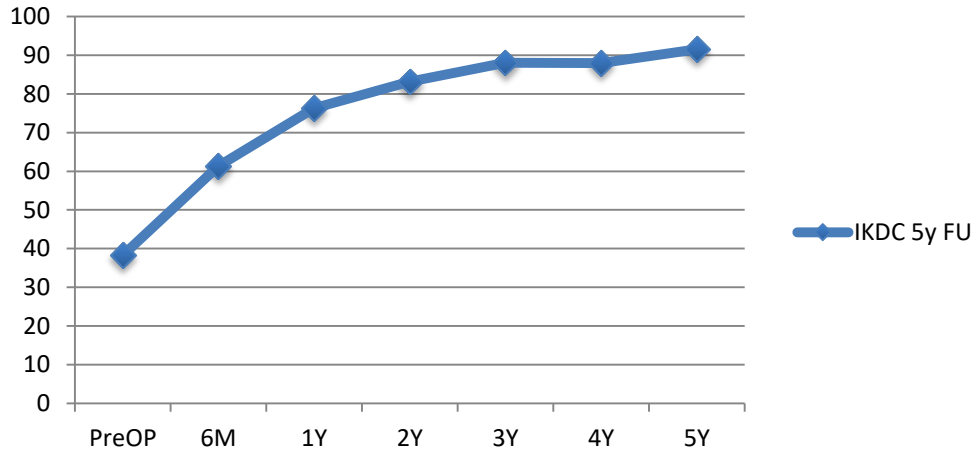


5 YEARS FU

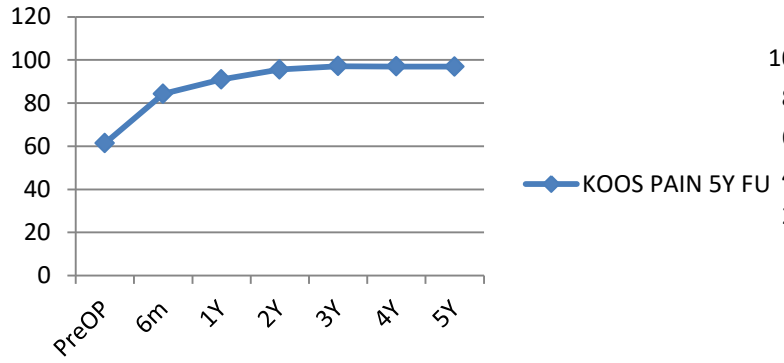


	PAIN	SYMPTOMS	ADL	SPORTS	QOL
PreOP	61,36	58,15	65,21	28,42	28
1Y	90,94	85,58	92,68	74,47	64,94
2Y	95,57	89,31	96,94	75,52	88,13
3Y	97,16	92,36	98,21	83,15	80,87
4Y	96,94	91,36	98,31	78,68	81,61

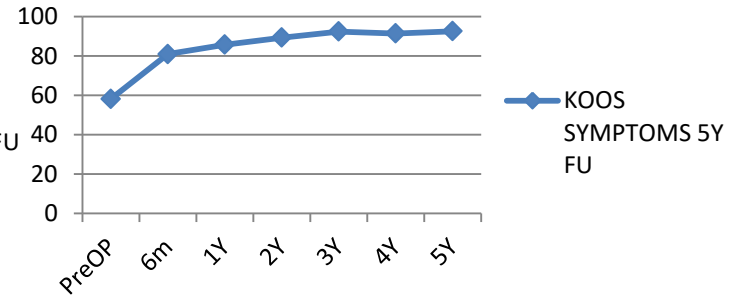
IKDC 5y FU



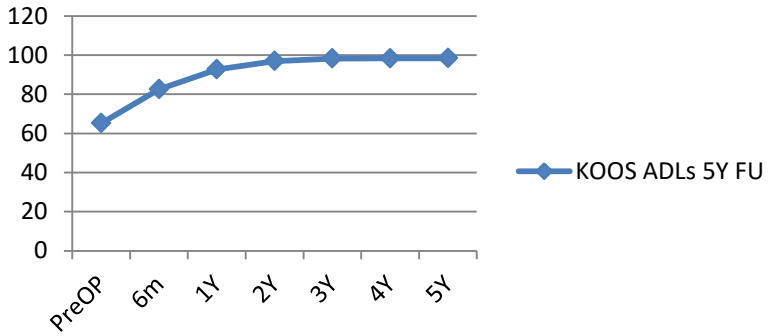
KOOS PAIN 5Y FU



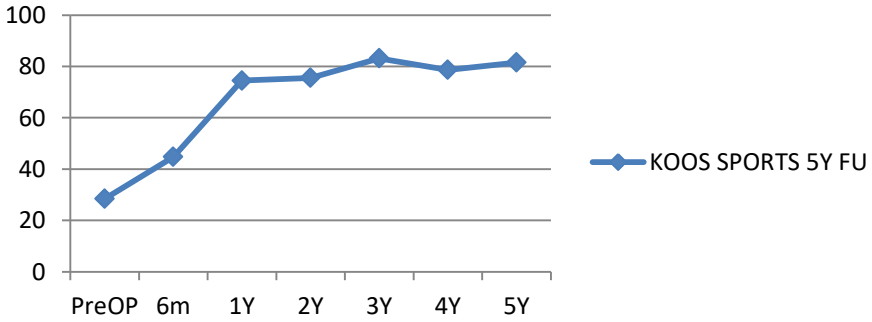
KOOS SYMPTOMS 5Y FU



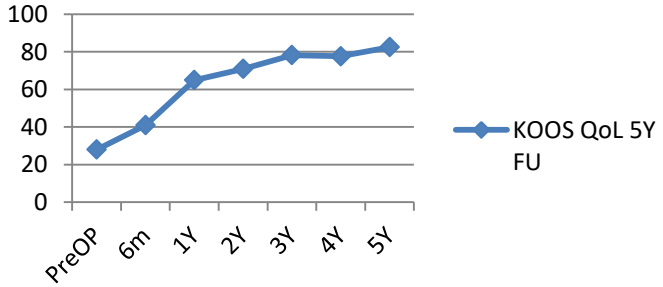
KOOS ADLs 5Y FU



KOOS SPORTS 5Y FU



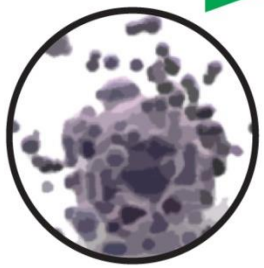
KOOS QoL 5Y FU



What is a Stem Cell?

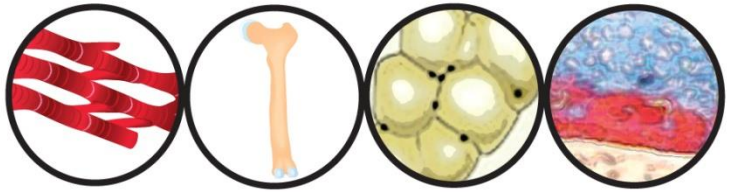
A mesenchymal stem cell is a primitive cell with the ability to:

Reduce Inflammation

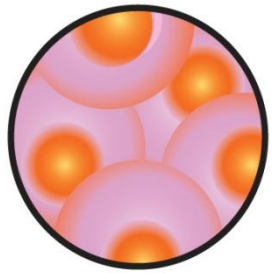


Fight Apoptosis (Cell Death)

Differentiate into Multiple Tissues



Muscle Bone Fat Cartilage



Self Replicate

World Journal of Stem Cells

Submit a Manuscript: http://www.wjgnet.com/esps/
Help Desk: http://www.wjgnet.com/esps/helpdesk.aspx
DOI: 10.4252/wjsc.v6.i3.344

World J Stem Cells 2014 July 26; 6(3): 344-354
ISSN 1948-0210 (online)

REVIEW

Mesenchymal stem cells as a potent cell source for articular cartilage regeneration

1. Expression of cartilage hypertrophy markers

   (Sekiya 2002, Mwale 2006, Pelltari 2006)

   – Hypertrophy ➔ Ossification

2. Thinner regenerated cartilage (Koga 2009)

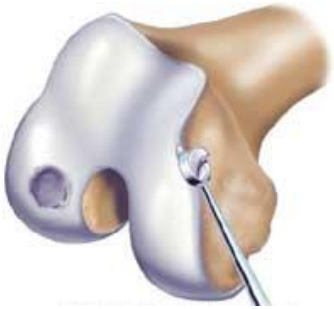
Overcome 1&2:

Co-Culture MSCs with mature chondrocytes

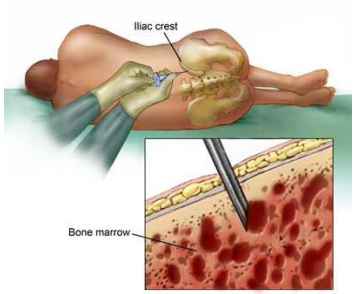
(Bial 2011)

R&D Concept

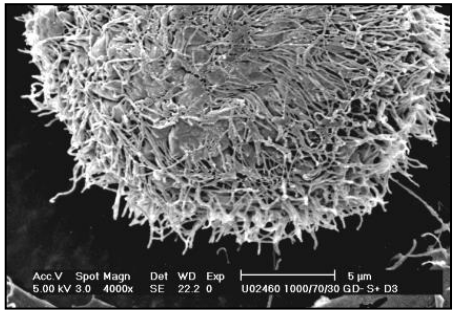
The beginning of INSTRUCT



Cartilage biopsy

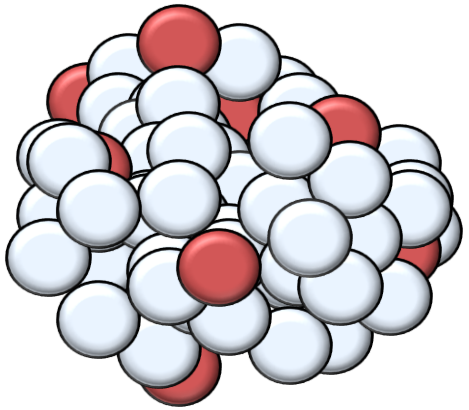


Bone marrow biopsy



Freshly isolated
Primary Chondrocytes (PCs)

MonoNucleated Cells (MNCs)



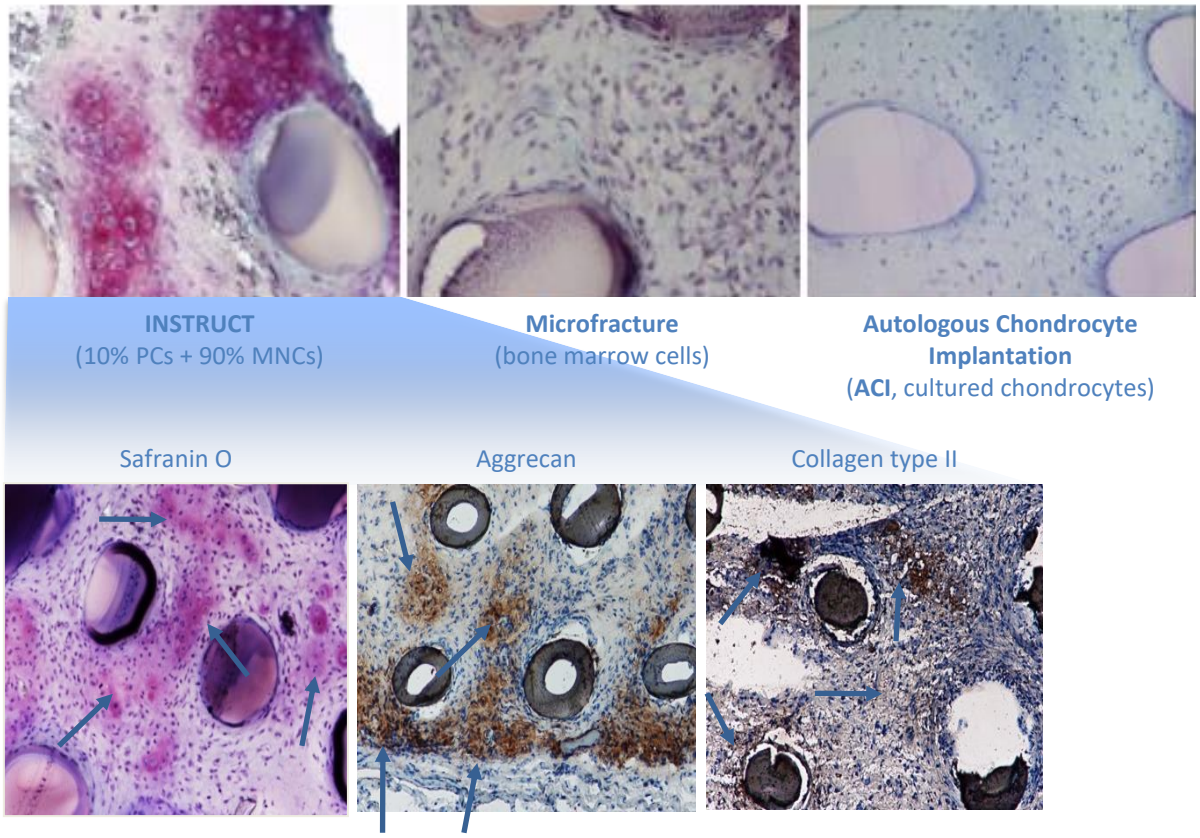
1 – 10% PCs

99 – 90% MNCs

Courtesy of CellCoTec

R&D Concept

PCs combined with MNCs enhance cartilage tissue formation



Courtesy of CellCoTec

Can we create a better standard of care?

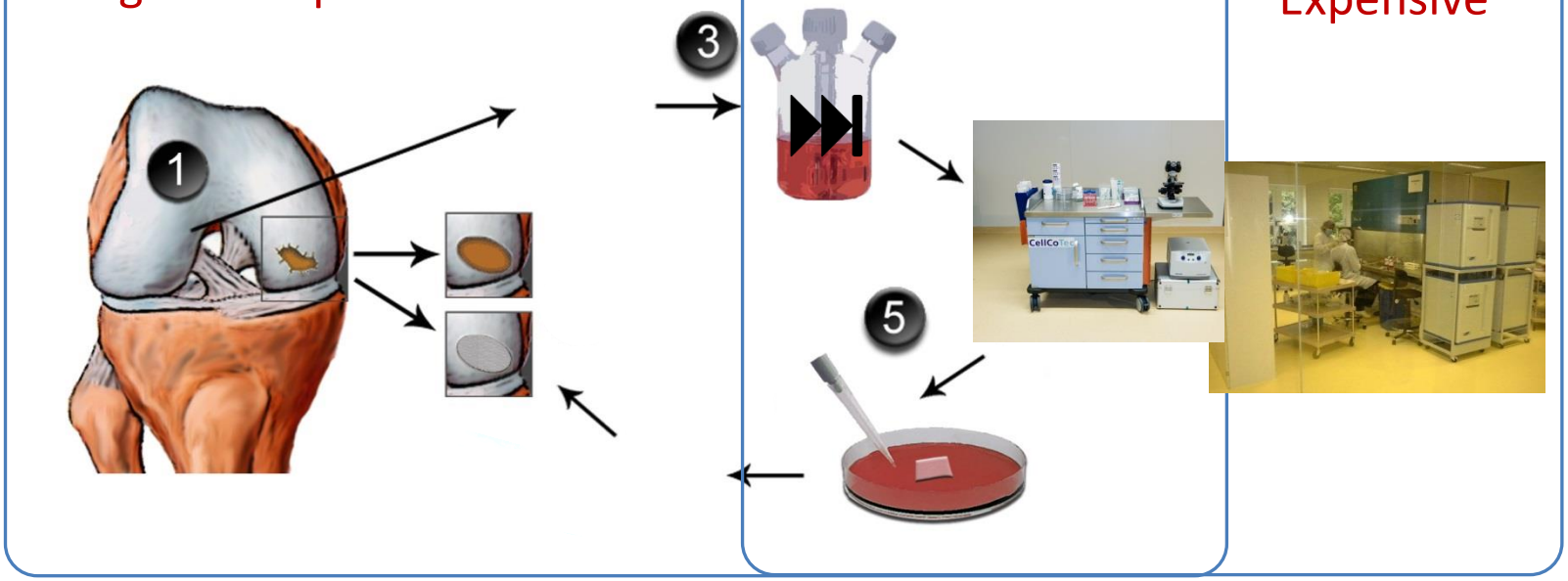
Toward a Single Stage Concept

Two Stage Concept

Operating Theater

GMP Facility

Expensive

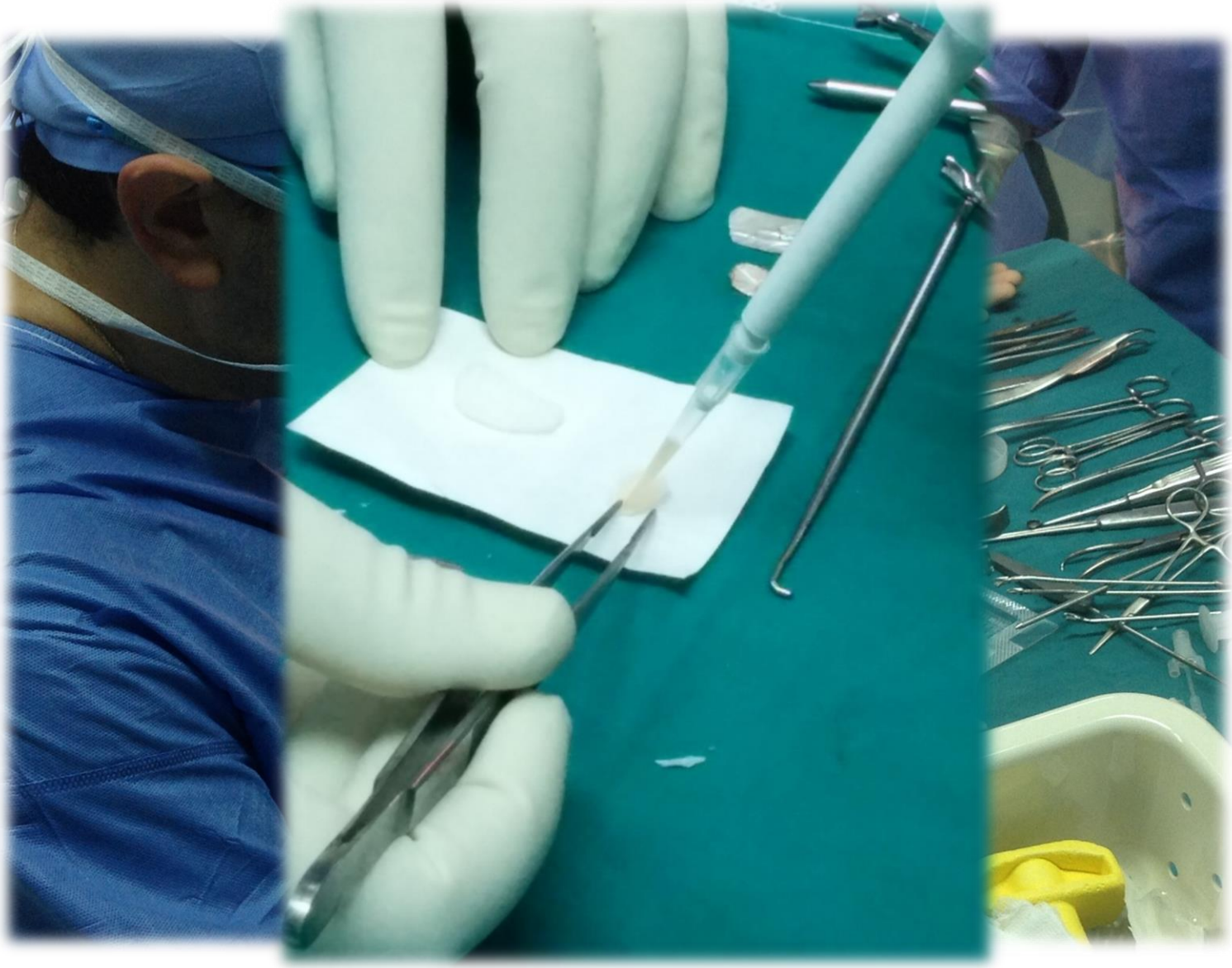


Logistically Complicated

Courtesy of CellCoTec

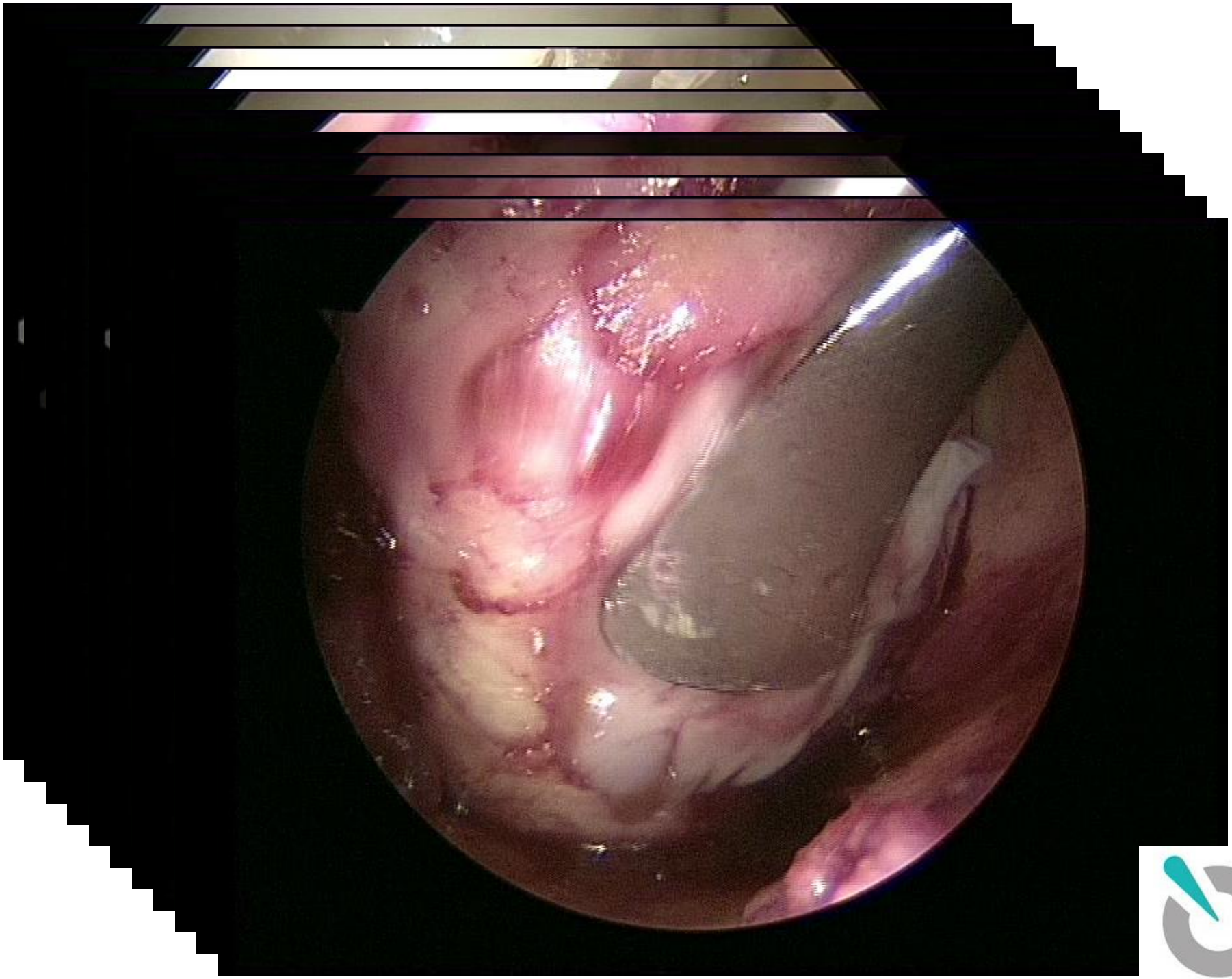
Portable Cell Therapy Lab

Courtesy of CellCoTec



CartiONE™

ONE-STEP, CELL-BASED CARTILAGE REPAIR



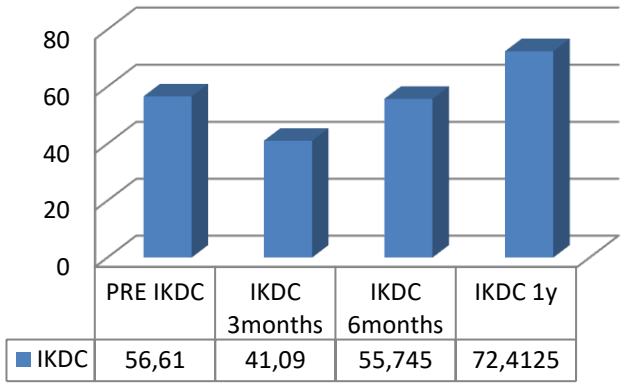
Early experience

One year clinical results of 18 consecutive cases

TheMIS Orthopaedic Center

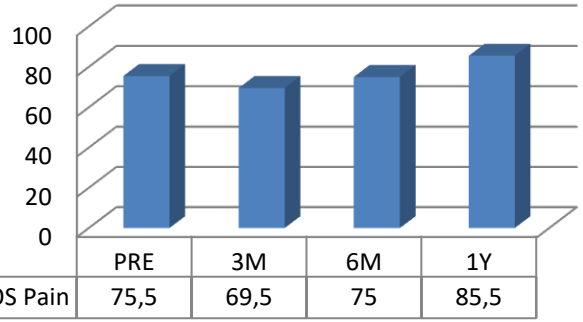
Thessaloniki, HELLAS

IKDC



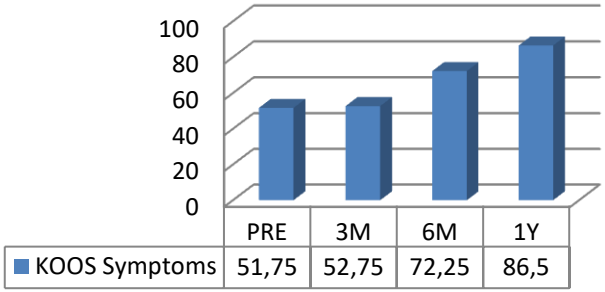
	PRE IKDC	IKDC 3months	IKDC 6months	IKDC 1y
IKDC	56,61	41,09	55,745	72,4125

KOOS Pain



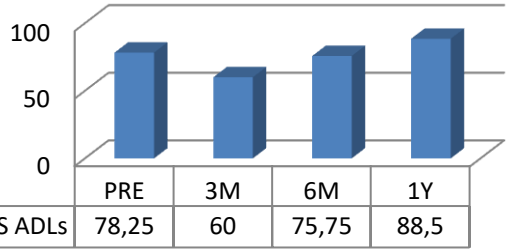
	PRE	3M	6M	1Y
KOOS Pain	75,5	69,5	75	85,5

KOOS Symptoms

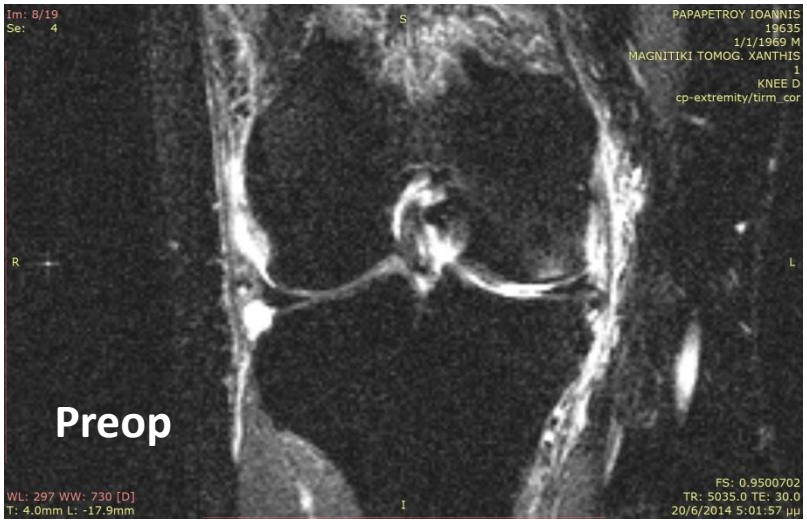


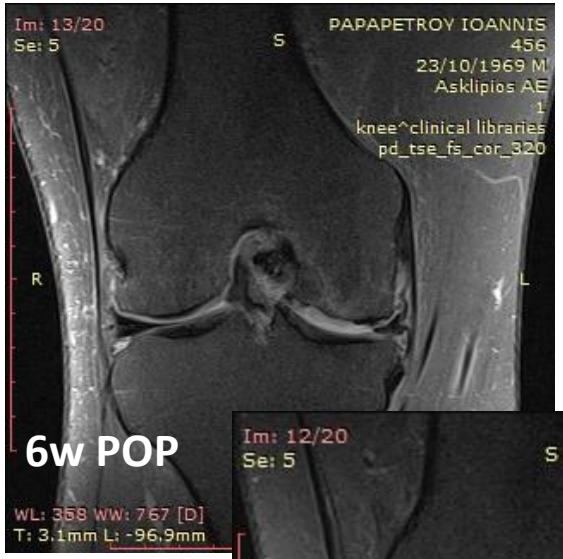
	PRE	3M	6M	1Y
KOOS Symptoms	51,75	52,75	72,25	86,5

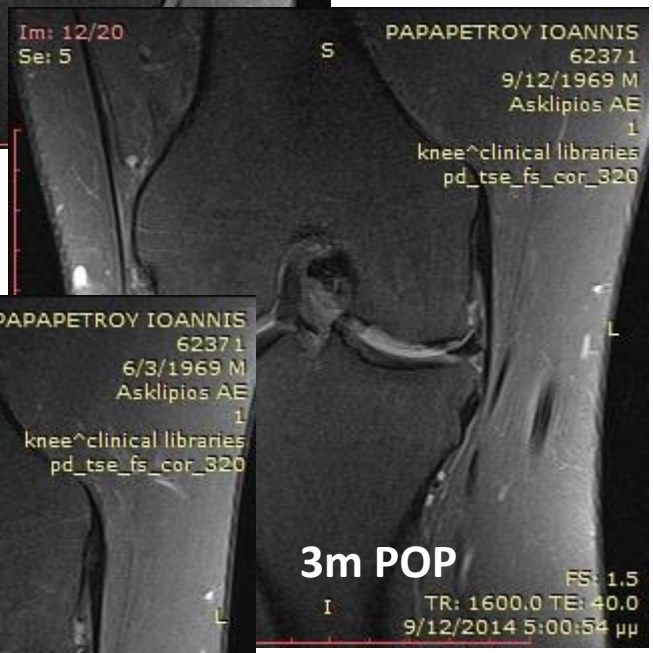
KOOS ADLs

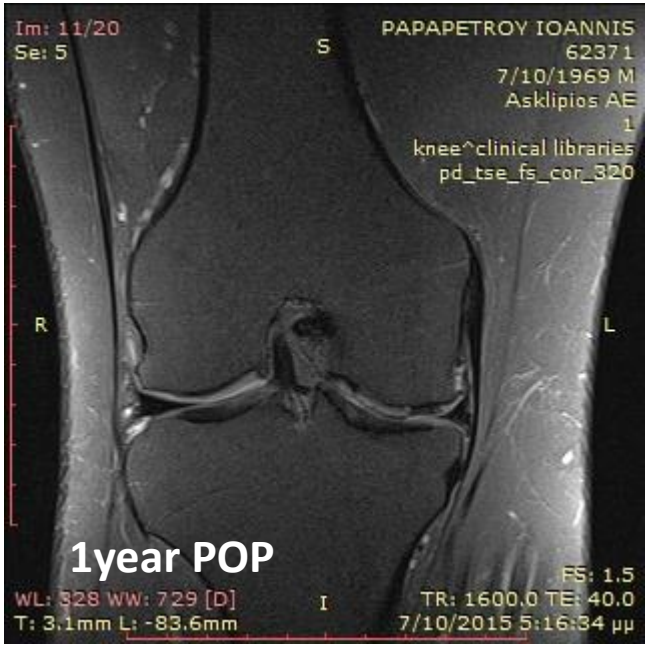


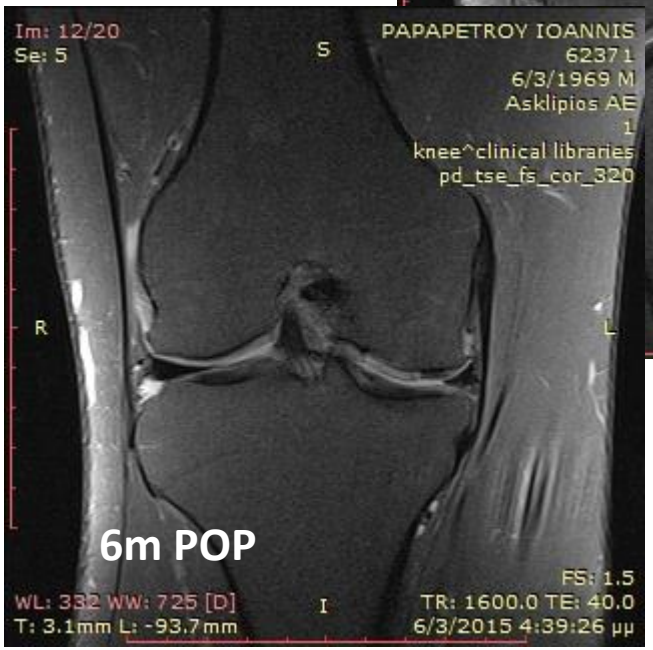
	PRE	3M	6M	1Y
KOOS ADLs	78,25	60	75,75	88,5


Im: 8/19
Se: 4
S
PAPAPETROY IOANNIS
19635
1/1/1969 M
MAGNITIKI TOMOG. XANTHIS
1
KNEE D
cp-extremity/tirm_cor
R
L
Preop
WL: 297 WW: 730 [D]
T: 4.0mm L: -17.9mm
I
FS: 0.9500702
TR: 5035.0 TE: 30.0
20/6/2014 5:01:57 μμ


Im: 13/20
Se: 5
S
PAPAPETROY IOANNIS
456
23/10/1969 M
Asklipios AE
1
knee^clinical libraries
pd_tse_fs_cor_320
R
L
6w POP
WL: 358 WW: 767 [D]
T: 3.1mm L: -96.9mm


Im: 12/20
Se: 5
S
PAPAPETROY IOANNIS
62371
9/12/1969 M
Asklipios AE
1
knee^clinical libraries
pd_tse_fs_cor_320
L
3m POP
I
FS: 1.5
TR: 1600.0 TE: 40.0
9/12/2014 5:00:54 μμ


Im: 11/20
Se: 5
S
PAPAPETROY IOANNIS
62371
7/10/1969 M
Asklipios AE
1
knee^clinical libraries
pd_tse_fs_cor_320
R
L
1year POP
WL: 328 WW: 729 [D]
T: 3.1mm L: -83.6mm
I
FS: 1.5
TR: 1600.0 TE: 40.0
7/10/2015 5:16:34 μμ


Im: 12/20
Se: 5
S
PAPAPETROY IOANNIS
62371
6/3/1969 M
Asklipios AE
1
knee^clinical libraries
pd_tse_fs_cor_320
R
L
6m POP
WL: 332 WW: 725 [D]
T: 3.1mm L: -93.7mm
I
FS: 1.5
TR: 1600.0 TE: 40.0
6/3/2015 4:39:26 μμ

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