PT&H

Arthrose

Physiotherapie Tschopp & Hilfiker

Marielle Tschopp & Roger Hilfiker

Wer sind wir:

Marielle Tschopp, aufgewachsen in Leukerbad, Physioschule Leukerbad 1992 bis 1996

→Spital Visp
→ Rehazentrum Leukerbad
→ Leitung Spital Zofingen
→ Eigene Praxis in Leukerbad
→ Leitung Spitalzentrum Oberwallis
→ Rückenzentrum in Thun
→ Eigene Praxis seit März 2023
→ Forschungserfahrung HES-SO

Roger Hilfiker, aufgwachsen im Berner Oberland, Physioschule in Leukerbad (1990 – 1994)

→ Spital Brig
→ Privatpraxis Mathieu & Mottier Sion
→ Praxis Zanella Bellwald Hilfiker, Brig
→ Rehazentrum Leukerbad
→ HES-SO
→ Paraplegikerforschung Nottwil
→ BFH
→ HES-SO
→ Eigene Praxis seit März 2023

Was macht Arthrose?

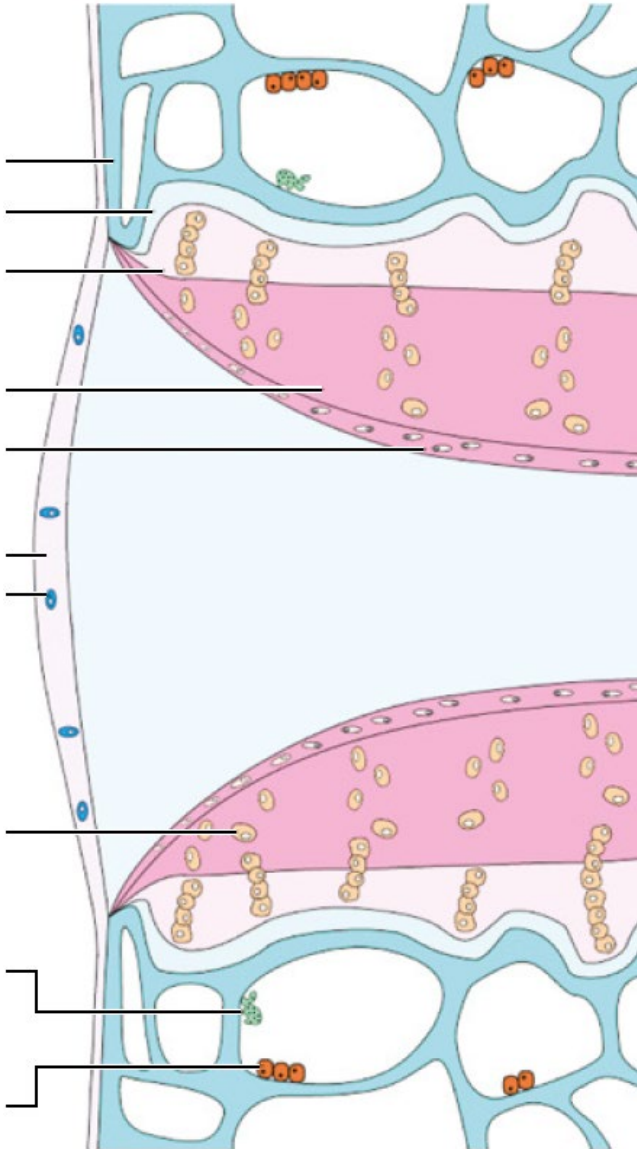
- Typische Symptome: Anlaufschmerzen, z.B. am Morgen oder nach längerem Sitzen.
- Steifigkeit in den Gelenken, Beweglichkeitsverlust.
- In aktivierten Phasen (Entzündungsphase), kann es auch zu Ruhe- oder Nachtschmerz kommen. Es kann auch zu einer Schwellung im Gelenk kommen.

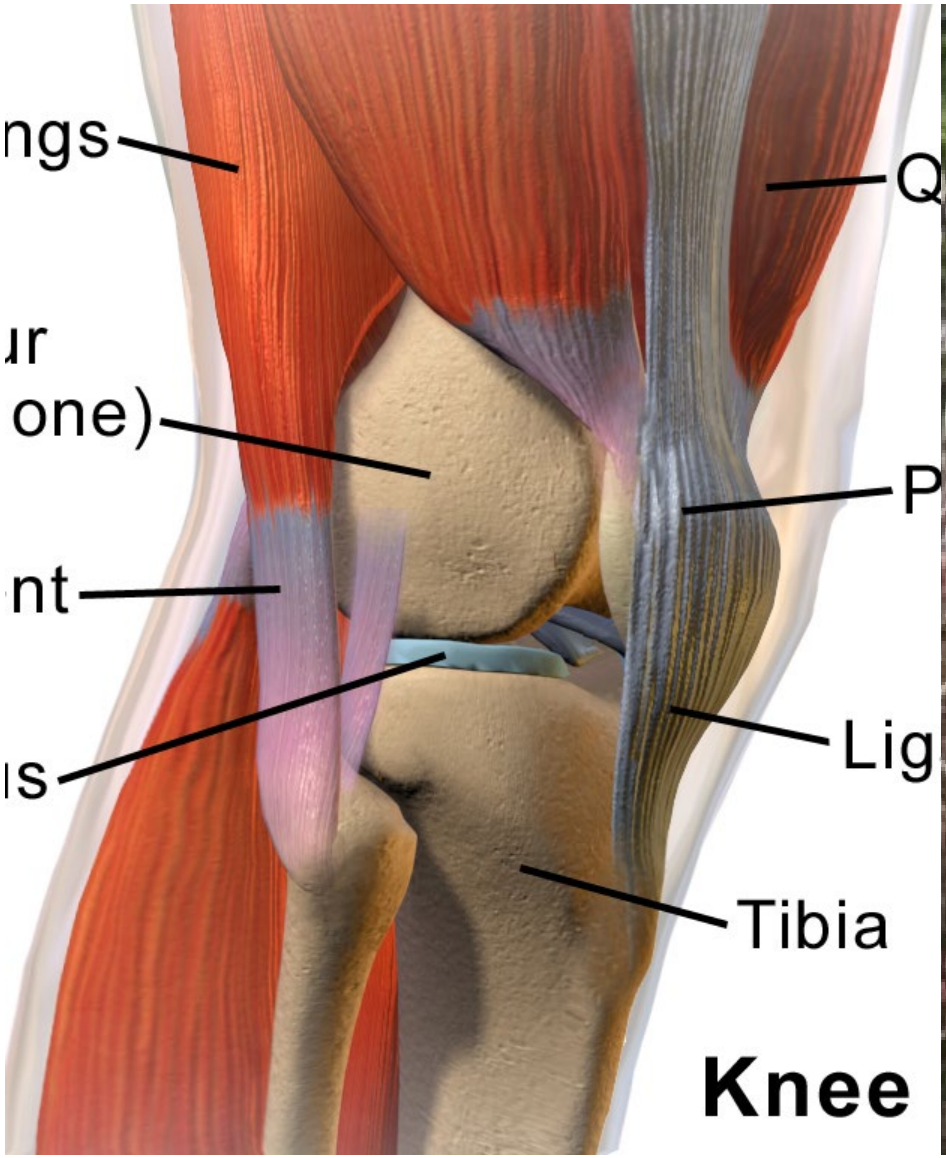
Was ist Arthrose?

- Arthrose ist ein Ungleichgewicht zwischen Auf- und Abbau aller Gelenkstrukturen.

- Verschiedene Stressfaktoren wie mechanische Überbelastung oder unterschwellige Entzündungen führen zu einem gesteigerten Abbau des Knorpels und zu Veränderungen im Knochen, der Gelenkskapsel und anderen Strukturen.


Arthrose ist nicht Abrieb wie bei einem Reifen

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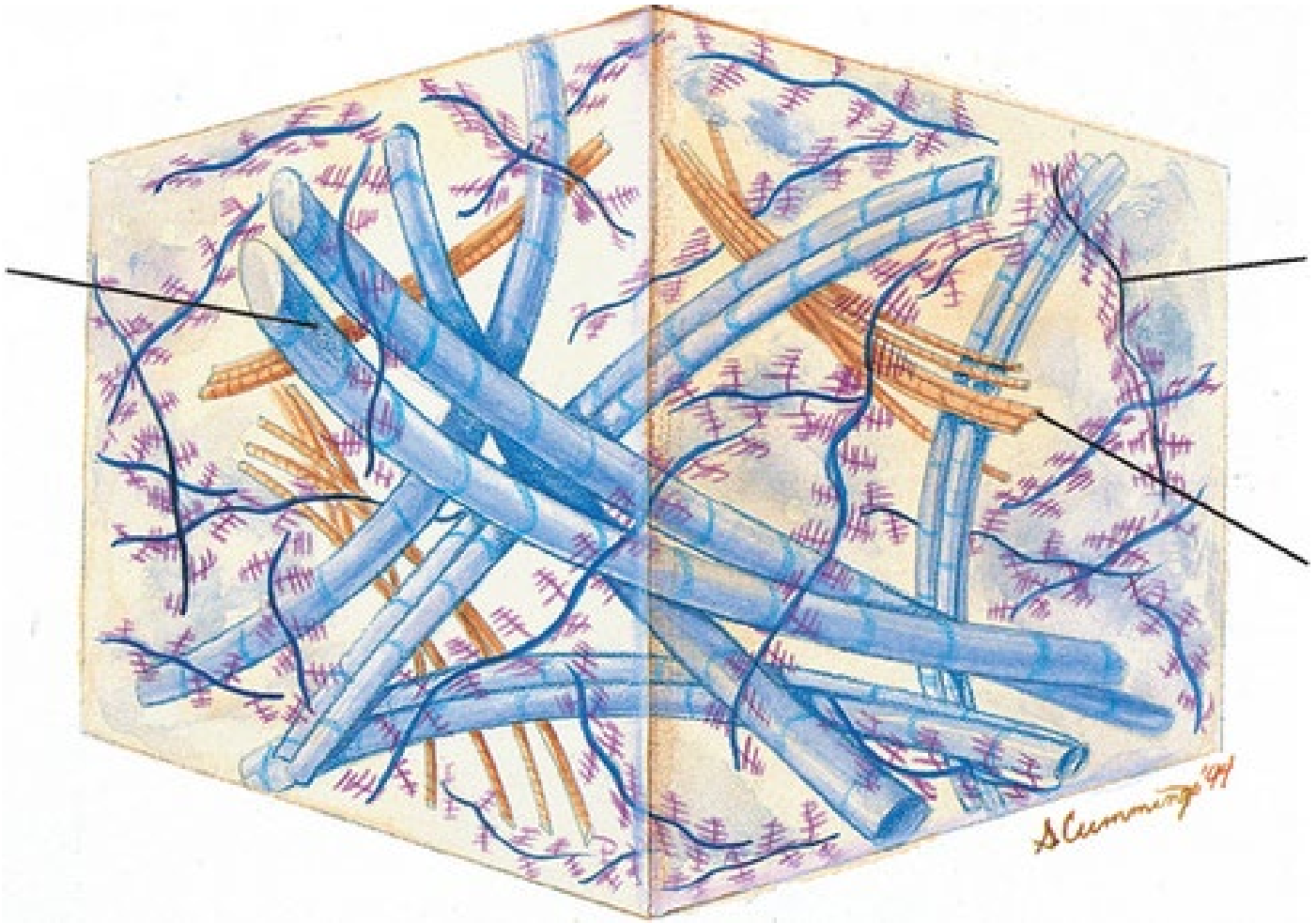
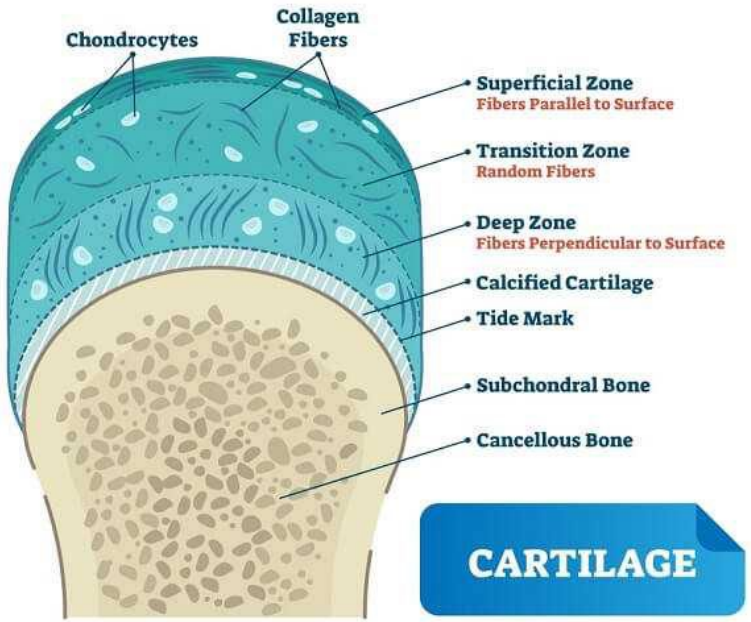
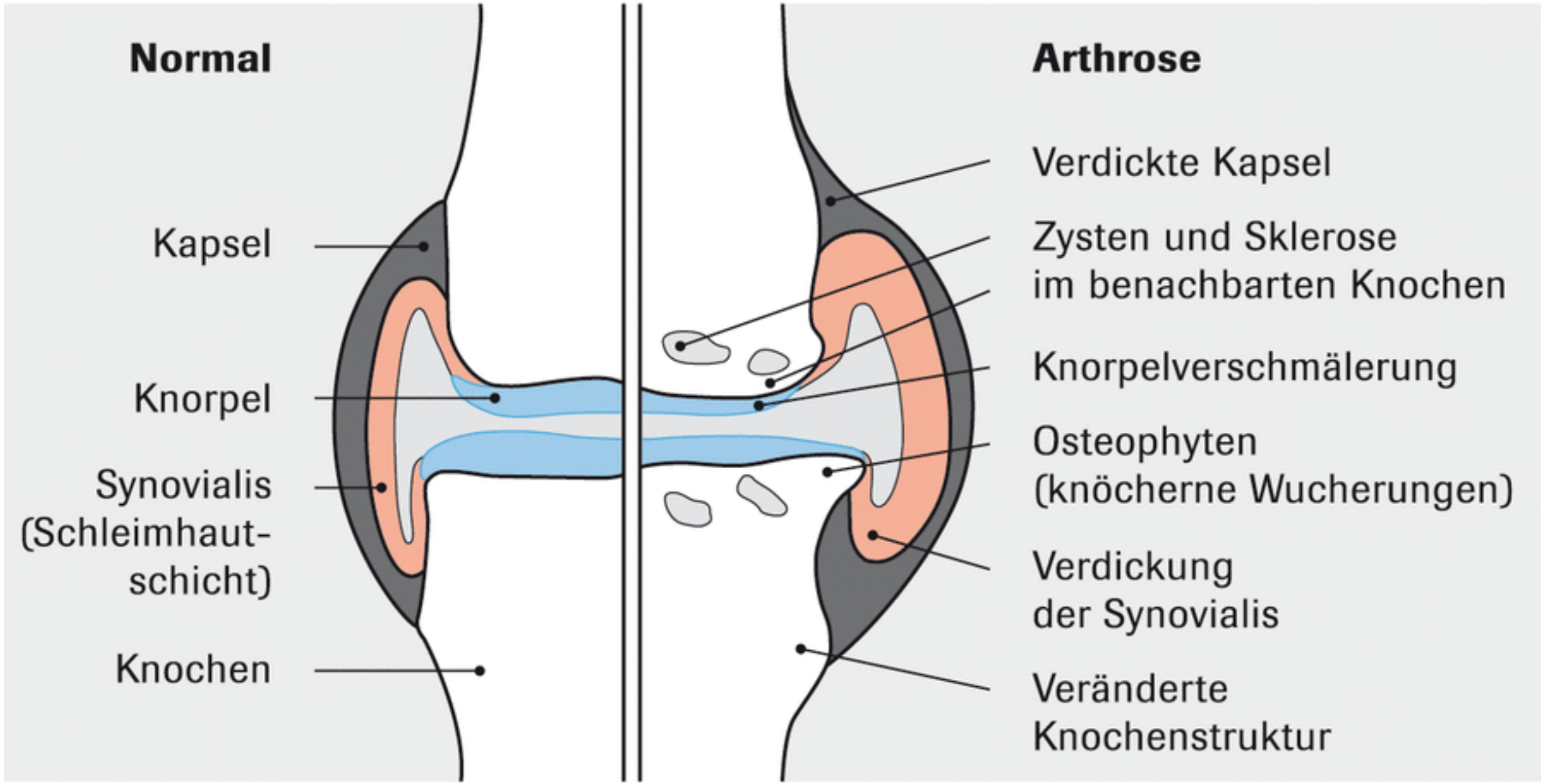
Knee

Der Knorpel braucht Bewegung, Belastung und Entlastung, um optimal ernährt zu werden.

→ Er ist nicht durchblutet und hat keine Nerven.
→ Ernährung über Gelenksflüssigkeit
→ Druck und Entlastung stimuliert Knorpelzellen, fördert die Diffusion der Gelenksflüssigkeit in den Knorpel und bindet Wasser im Knorpel.





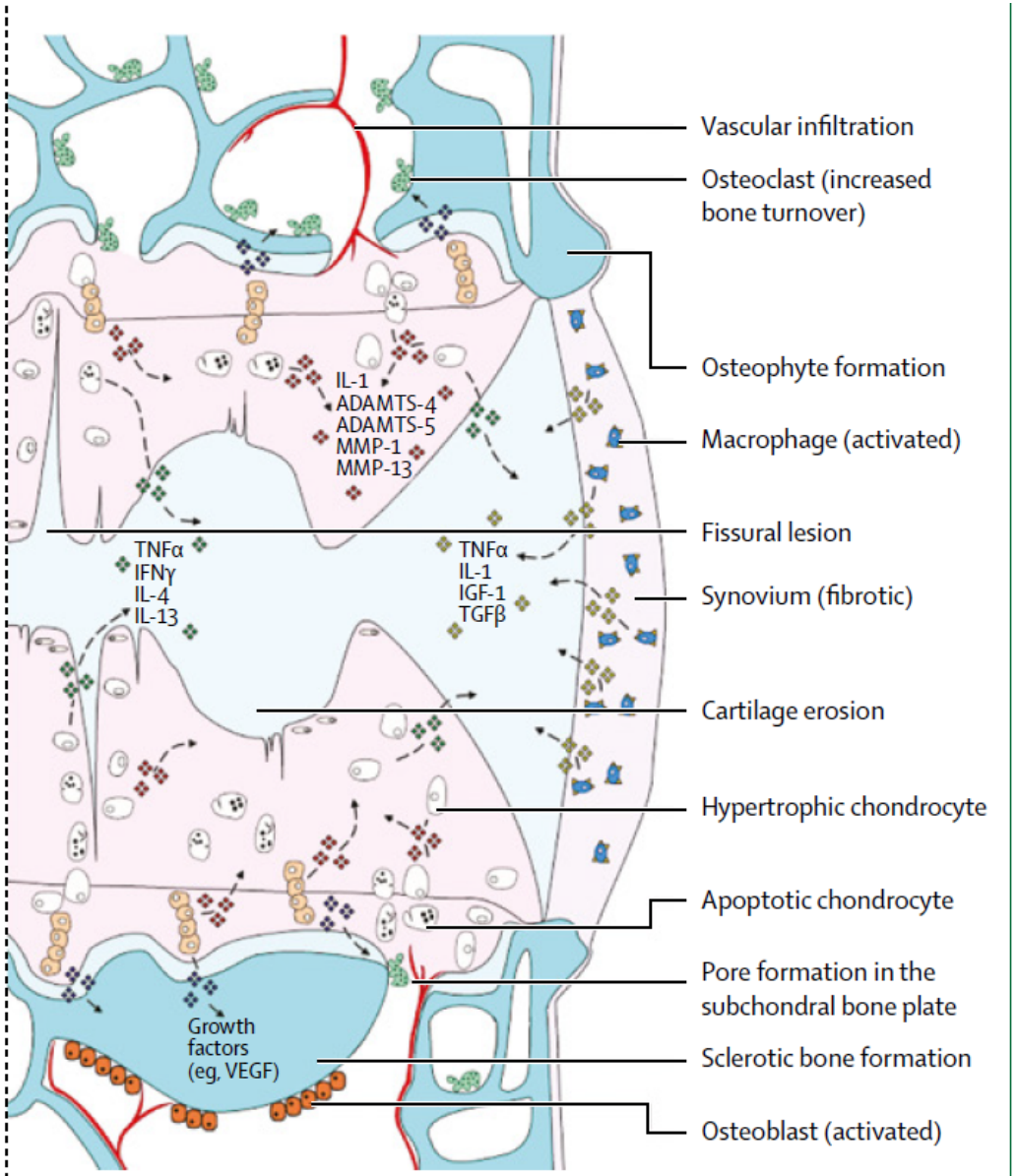

Normal
Kapsel
Knorpel
Synovialis
(Schleimhaut-
schicht)
Knochen
Arthrose
Verdickte Kapsel
Zysten und Sklerose
im benachbarten Knochen
Knorpelverschmälerung
Osteophyten
(knöcherne Wucherungen)
Verdickung
der Synovialis
Veränderte
Knochenstruktur

Im Gegensatz zu den Reifen ist der Knorpel eine lebendige Struktur

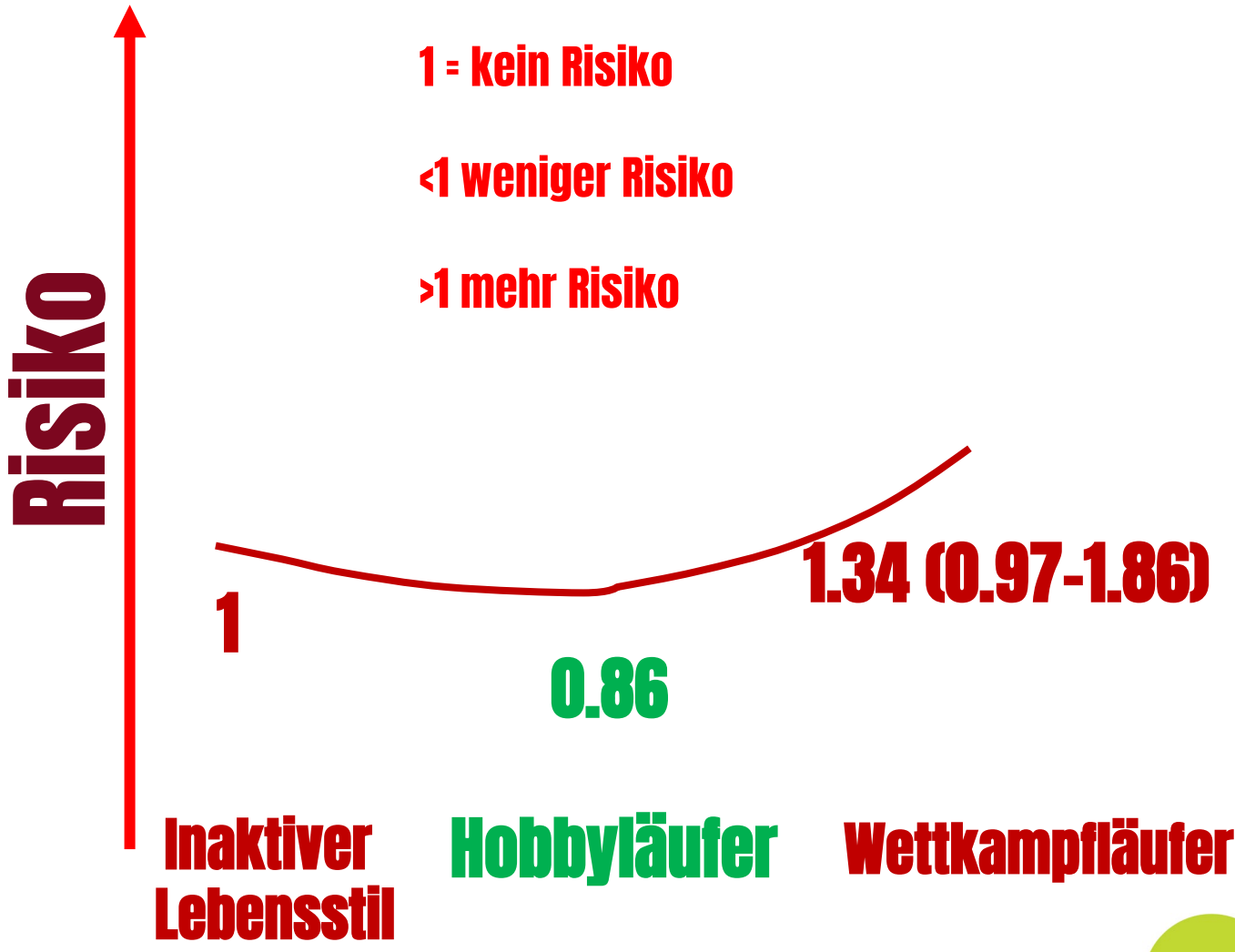
Die Knorpelzellen brauchen Reize - Druck und Entlastung → Bewegung

Das Gelenk - nicht nur der Knorpel - braucht Bewegung
Zu wenig schadet - zu viel schadet

Nicht Abrieb sondern komplexes Zusammenspiel verschiedener Botenstoffe und Enzyme (“Katalysatoren”)

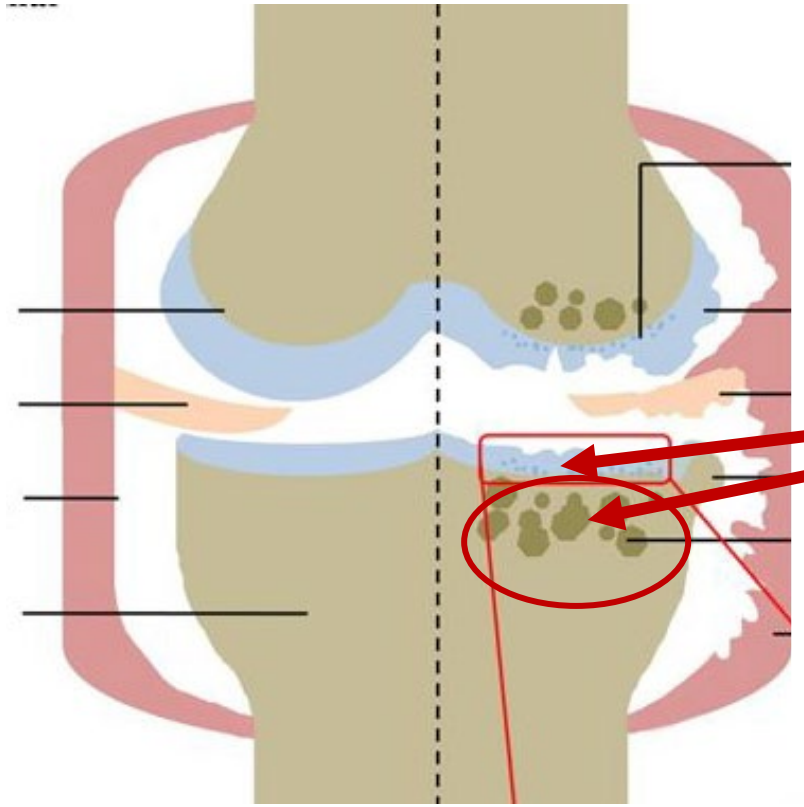


Sport und Arthrose: Ist Sport ein Risikofaktor?



Alentorn-Geli, E., Samuelsson, K., Musahl, V., Green, C. L., Bhandari, M., & Karlsson, J. (2017). The association of recreational and competitive running with hip and knee osteoarthritis: a systematic review and meta-analysis. *journal of orthopaedic & sports physical therapy*, *47*(6), 373-390. 10.2519/jospt.2017.7137

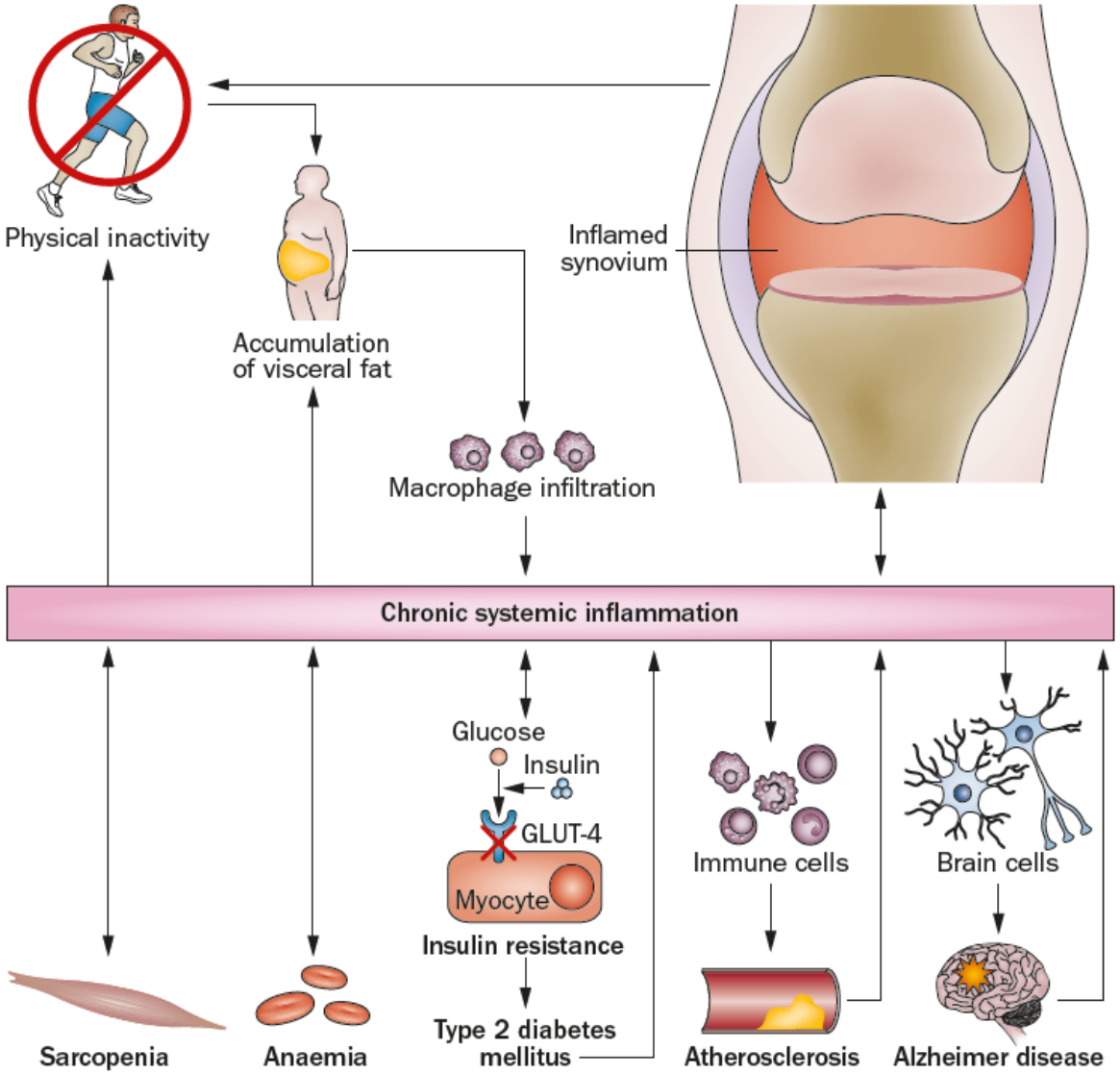
Verletzungen sind ein Risiko



Verletzungen am Knorpel oder an den Bändern oder Menisken führen zu Problemen wie Instabilität, Muskelschwäche, chronischer niedriggradiger Entzündung.

Bei Mikroverletzungen kommt es zu einem gestörten Gleichgewicht von abbauenden und aufbauenden Prozessen.

Aber die Vorteile von körperlicher Aktivität und Sport überwiegen



Sport reguliert **entzündungsfördernde** und **entzündungshemmende** Botenstoffe

Sport hat **positive Effekte** auf Risiken für **kardiovaskuläre Probleme**

Sport **reduziert viszerales Fett**

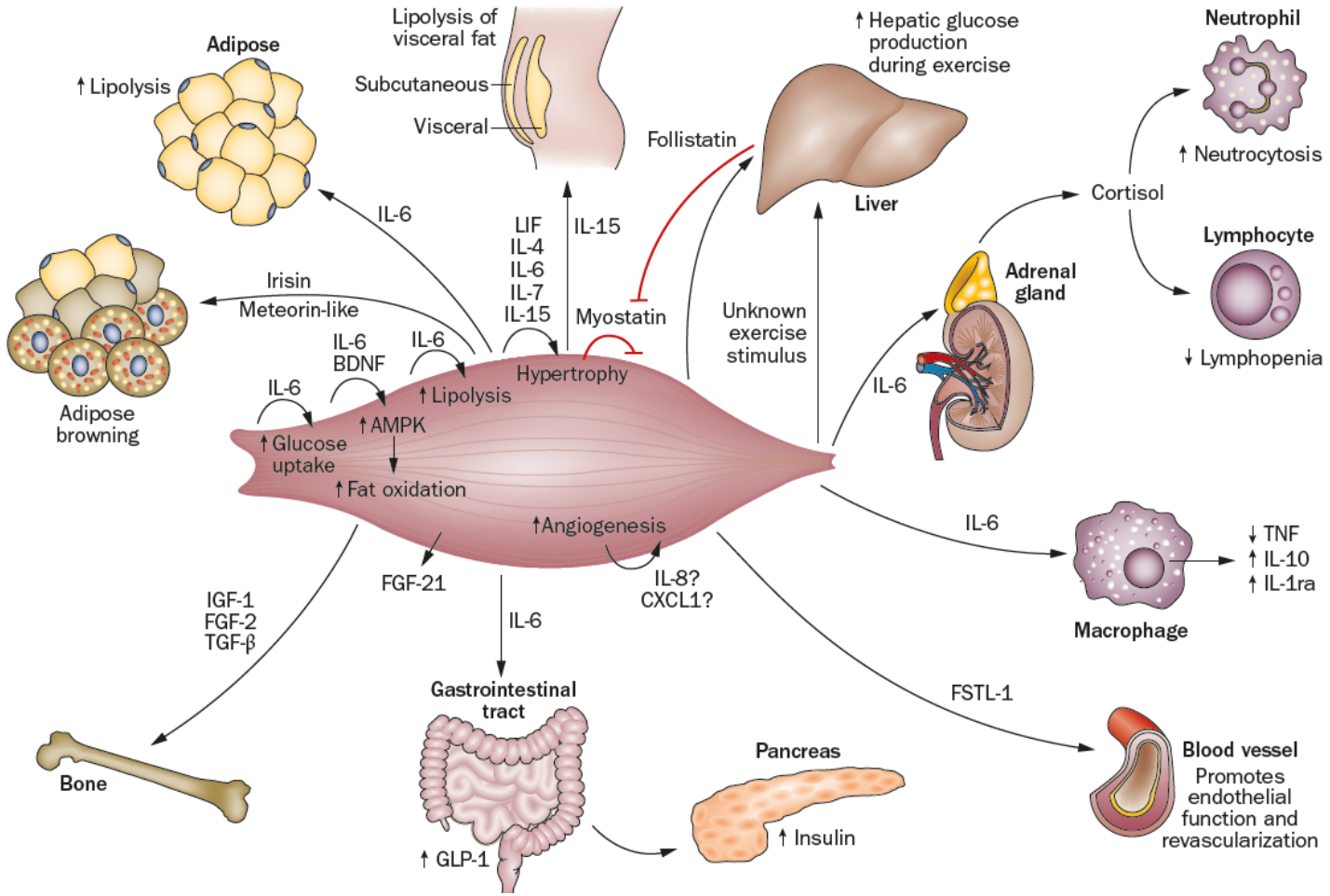
Sport hat **positive** Effekte auf **Blutzucker**

Sport hilft, die Gelenke zu **stabilisieren** → besserer **Schutz** durch bessere **Muskulatur** und bessere **Kontrolle**

Sport hat **positive** Wirkung auf **Gehirn**

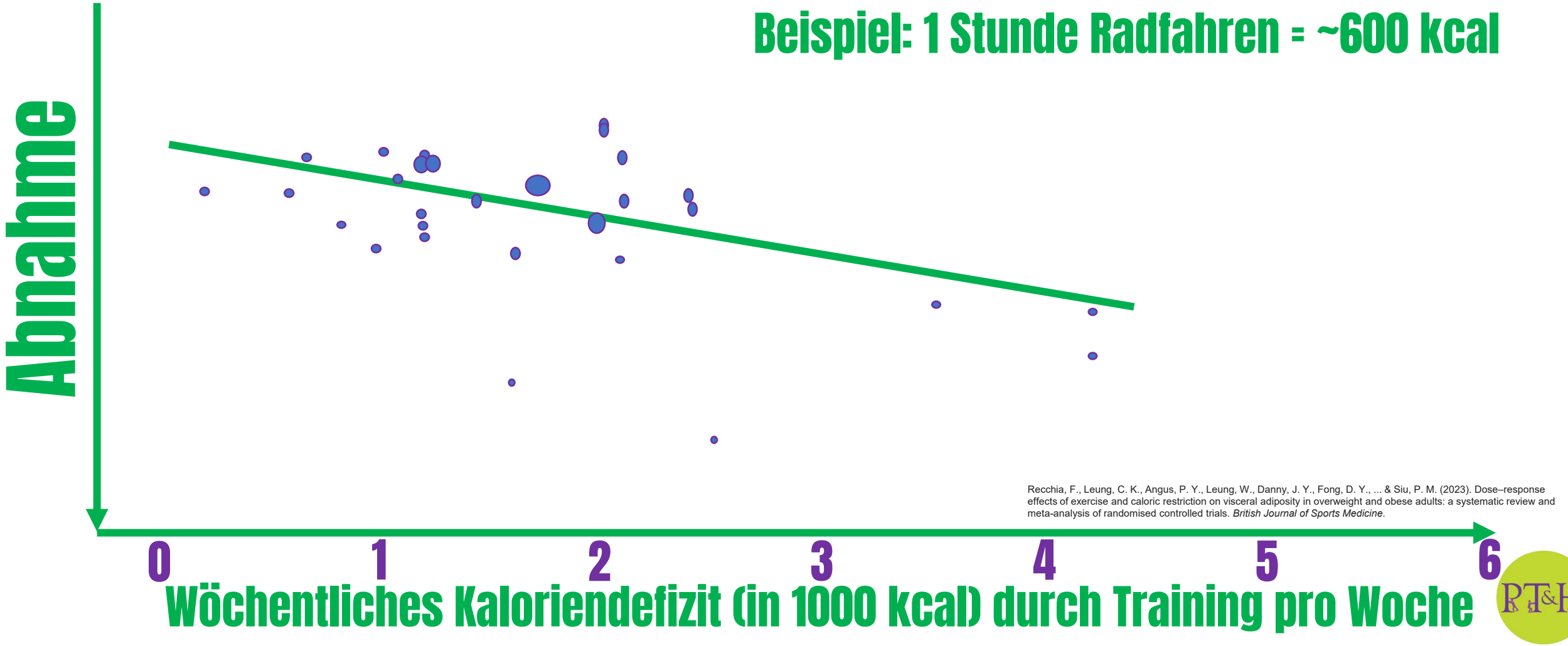
Benatti, F. B., & Pedersen, B. K. (2015). Exercise as an anti-inflammatory therapy for rheumatic diseases—myokine regulation. *Nature reviews rheumatology*, *11*(2), 86.

Der Muskel produziert gute Botenstoffe



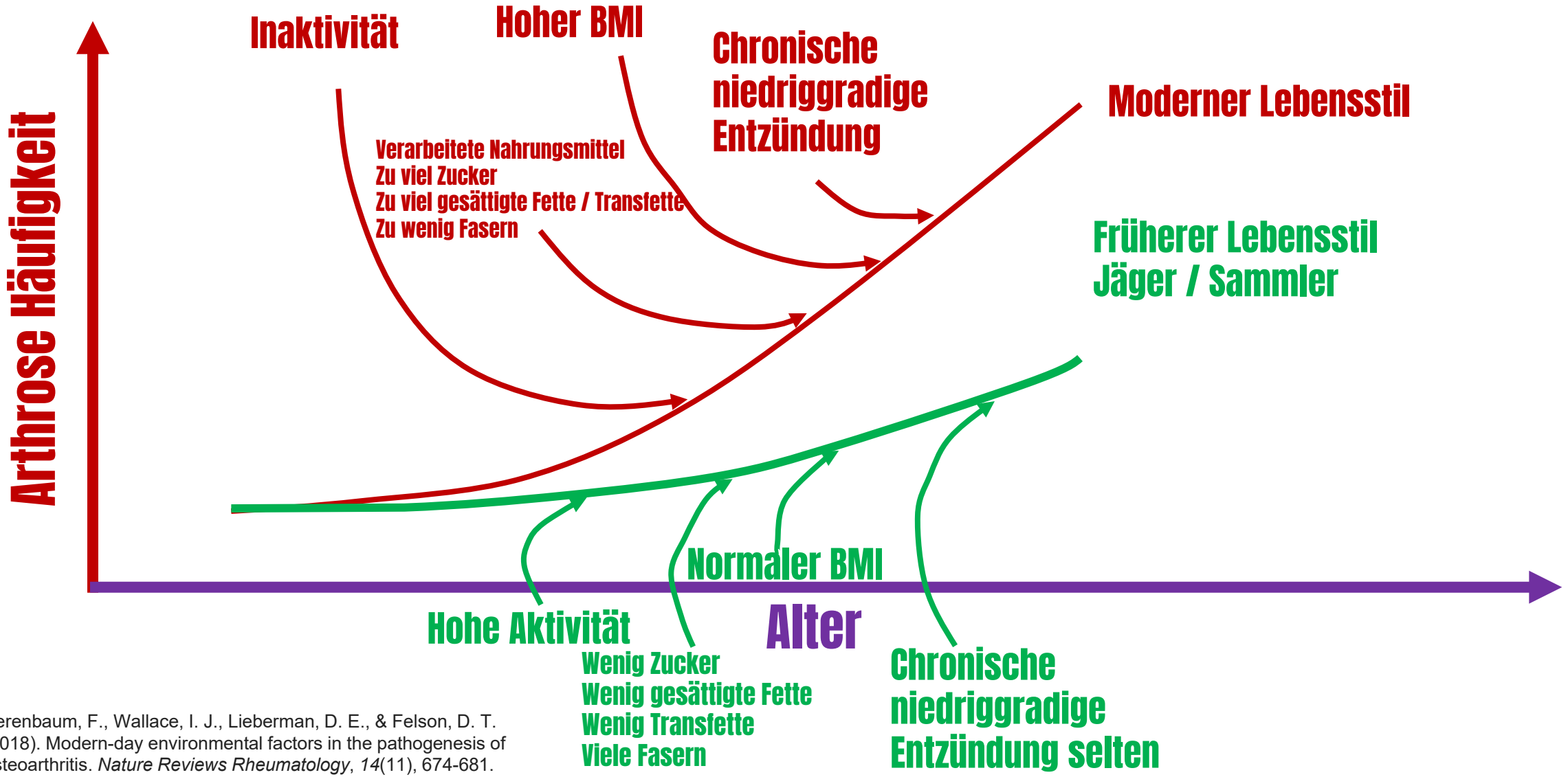
Benatti, F. B., & Pedersen, B. K. (2015). Exercise as an anti-inflammatory therapy for rheumatic diseases—myokine regulation. *Nature reviews rheumatology*, *11*(2), 86.

Effekt von körperlicher Belastung / Training auf das viszerale Fett



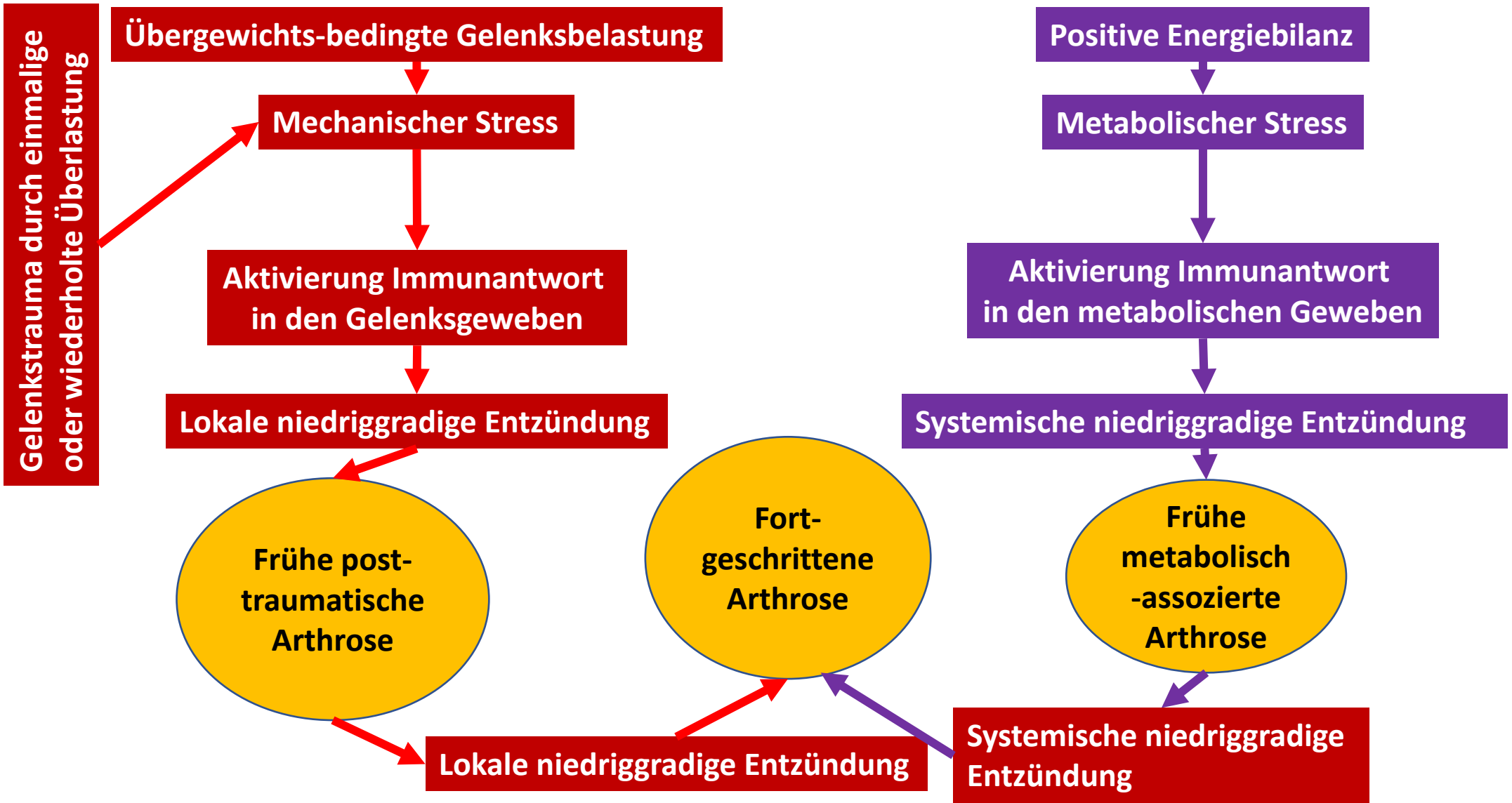
Recchia, F., Leung, C. K., Angus, P. Y., Leung, W., Danny, J. Y., Fong, D. Y., ... & Siu, P. M. (2023). Dose–response effects of exercise and caloric restriction on visceral adiposity in overweight and obese adults: a systematic review and meta-analysis of randomised controlled trials. *British Journal of Sports Medicine*.

Risikofaktoren für Arthrose

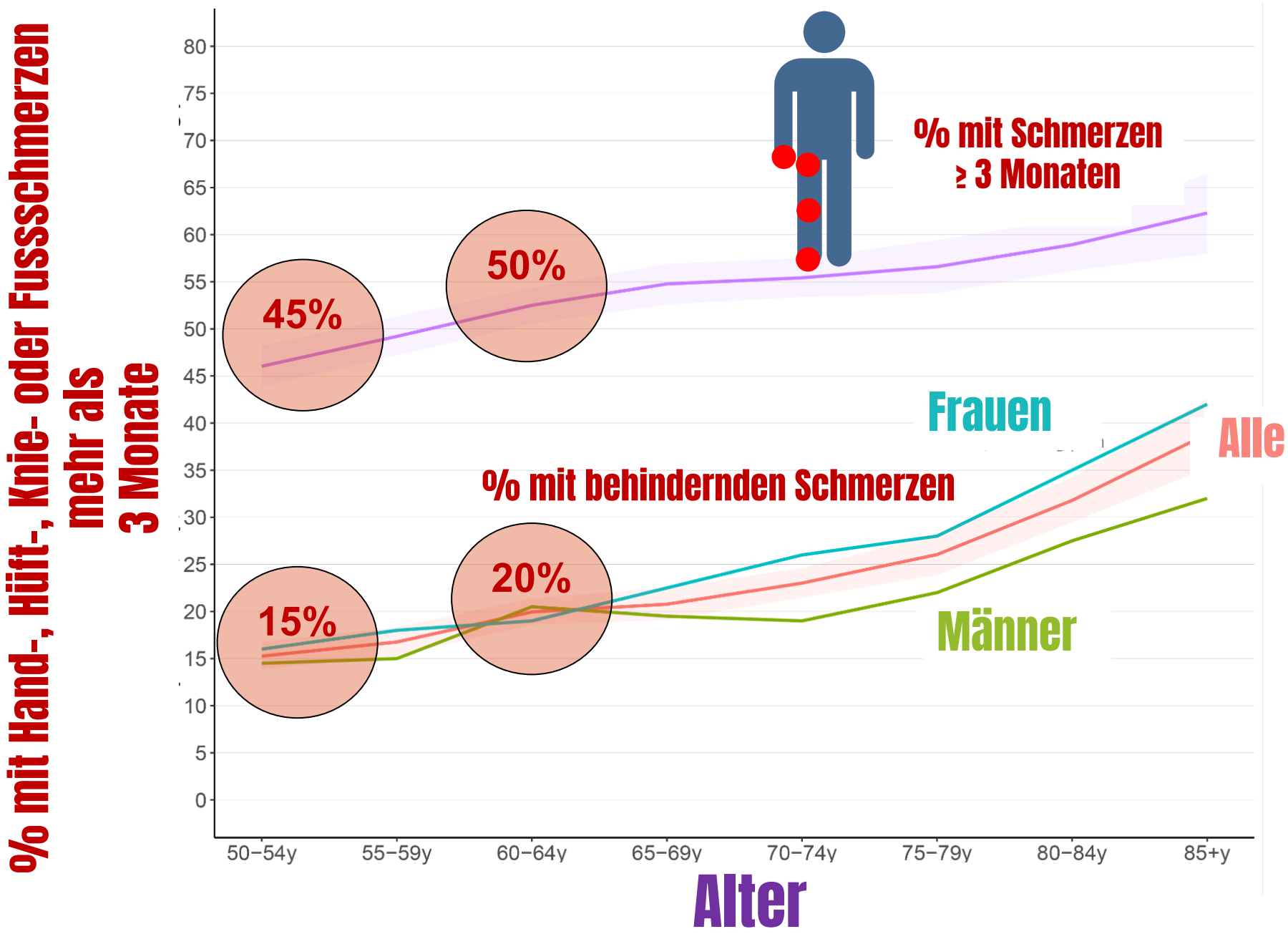


Berenbaum, F., Wallace, I. J., Lieberman, D. E., & Felson, D. T. (2018). Modern-day environmental factors in the pathogenesis of osteoarthritis. *Nature Reviews Rheumatology*, *14*(11), 674-681.

Mechanische und metabolische Belastung

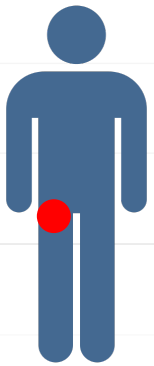


Wie häufig ist Arthrose?



Roger-Hilfiker-Diagramm mit Daten von Thomas et al., 2014

Radiologische Arthrose häufiger als symptomatische



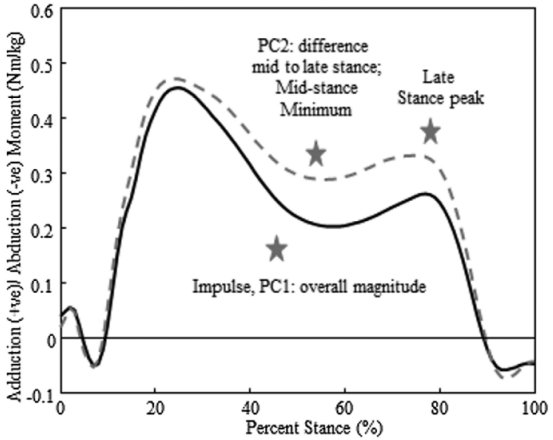
Daten aus der Framingham-Studie

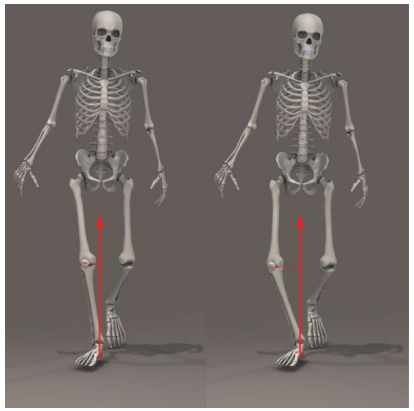
→ In anderen Studien ist die Prävalenz bei Frauen höher.

Roger-Hilfiker-Diagramm mit Daten von Kim et al., 2014

Zusammenhang Röntgenbild und Symptome

- Es gibt einen Zusammenhang
- Doch es gibt viele Menschen mit strukturellen Veränderungen (Röntgenbild) ohne Beschwerden
- In der Physiotherapie behandeln wir nicht das Röntgenbild
- Bei gleichem Röntgenbild kann es Unterschiede geben, die wir behandeln.



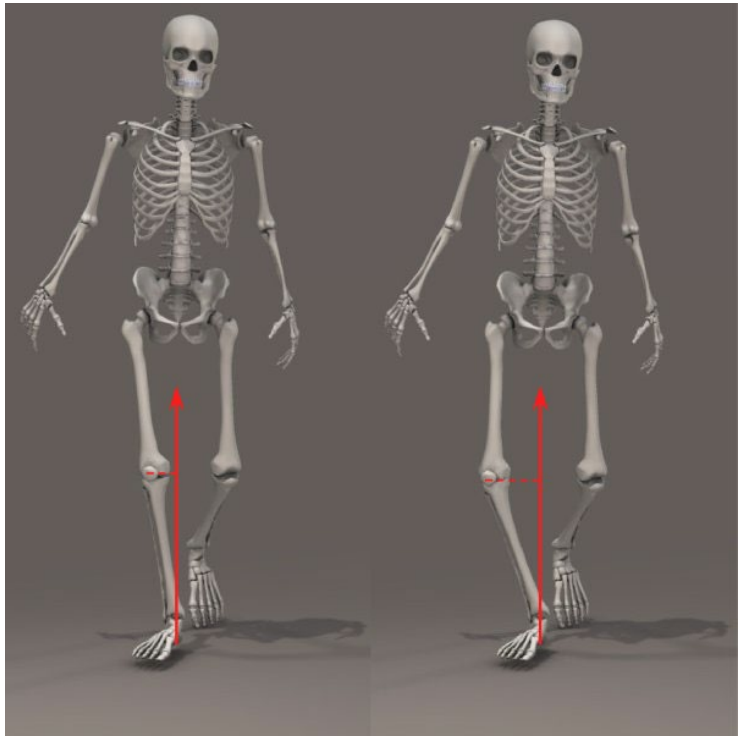


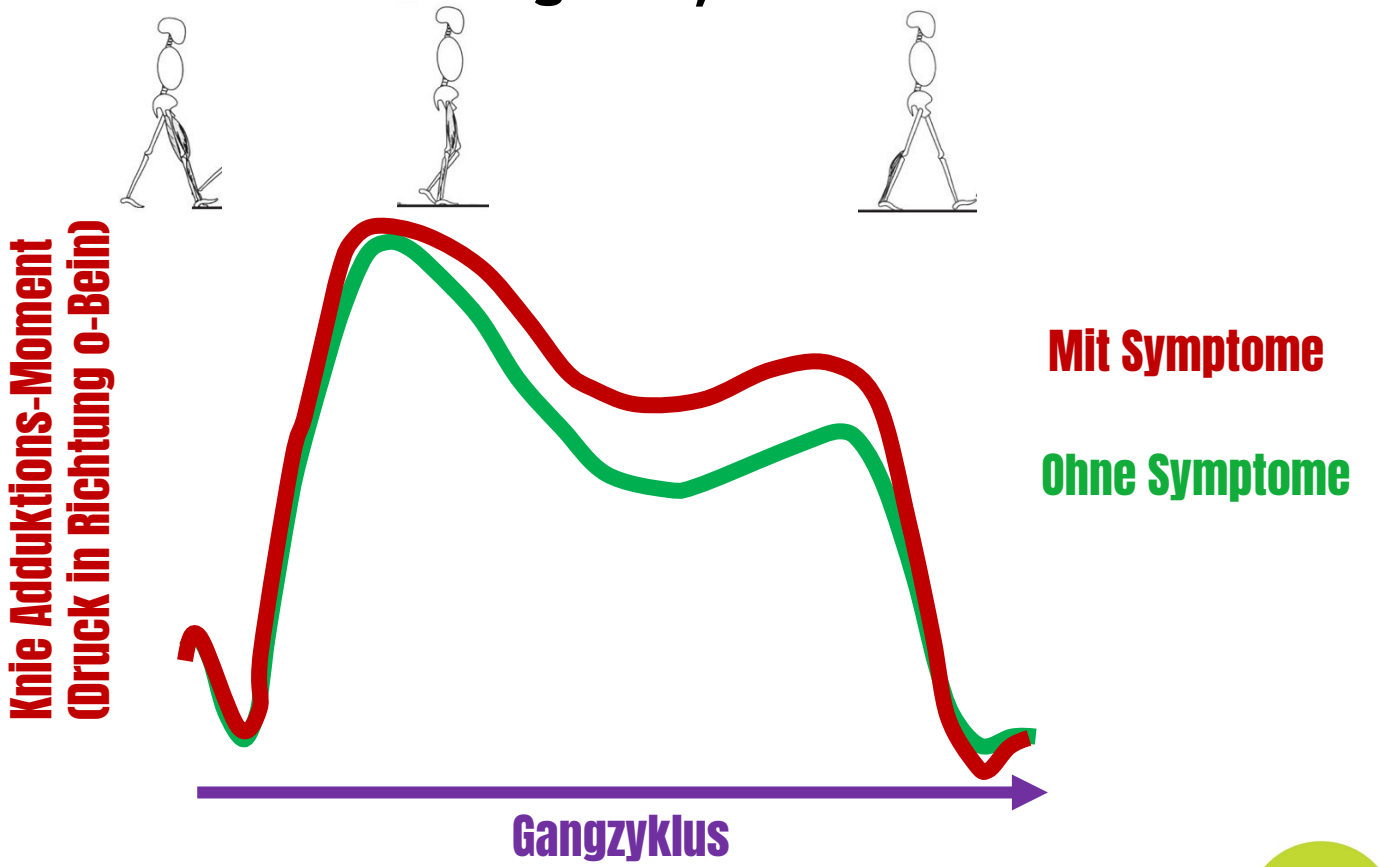
Neogi, T., Felson, D., Niu, J., Nevitt, M., Lewis, C. E., Aliabadi, P., ... & Zhang, Y. (2009). Association between radiographic features of knee osteoarthritis and pain: results from two cohort studies. *Bmj*, *339*.

Astephen Wilson, J. L., Stanish, W. D., & Hubley-Kozey, C. L. (2017). Asymptomatic and symptomatic individuals with the same radiographic evidence of knee osteoarthritis walk with different knee moments and muscle activity. *Journal of Orthopaedic Research*, *35*(8), 1661-1670.

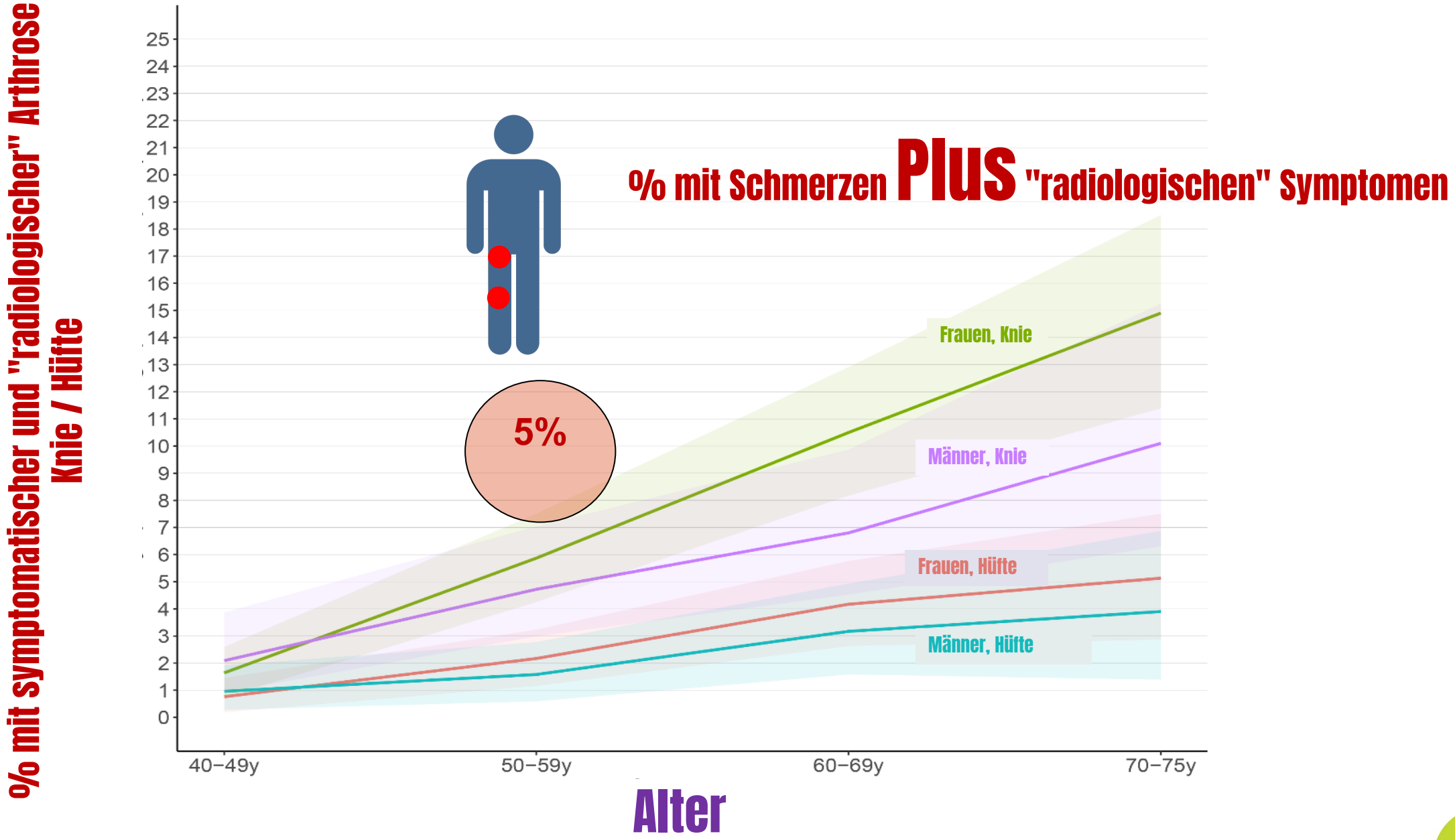
Zusammenhang Röntgenbild und Symptome

- Bei gleichem Röntgenbild kann es andere Unterschiede geben, die wir behandeln.





Astephen Wilson, J. L., Stanish, W. D., & Hubley-Kozey, C. L. (2017). Asymptomatic and symptomatic individuals with the same radiographic evidence of knee osteoarthritis walk with different knee moments and muscle activity. *Journal of Orthopaedic Research*, *35*(8), 1661-1670.



Roger-Hilfiker-Diagramm mit Daten von Guillemin et al., 2011

Warum haben Frauen häufiger Arthrose?

- Nicht alle Gründe bekannt
- Hormonelle Gründe, vor allem nach Menopause (Östrogen ⬇)
- BMI → Frauen haben ein anderes Verhältnis von Fett / Muskulatur
- Anatomie → Frauen haben breitere Hüften → Ausrichtung der Beine → mehr Belastung auf Knie
- Verletzungsrisiko → Hormonschwankungen, lockere Bandstrukturen, weniger Muskulatur
- Genetik → gewisse Gene haben mehr Auswirkungen bei Frauen

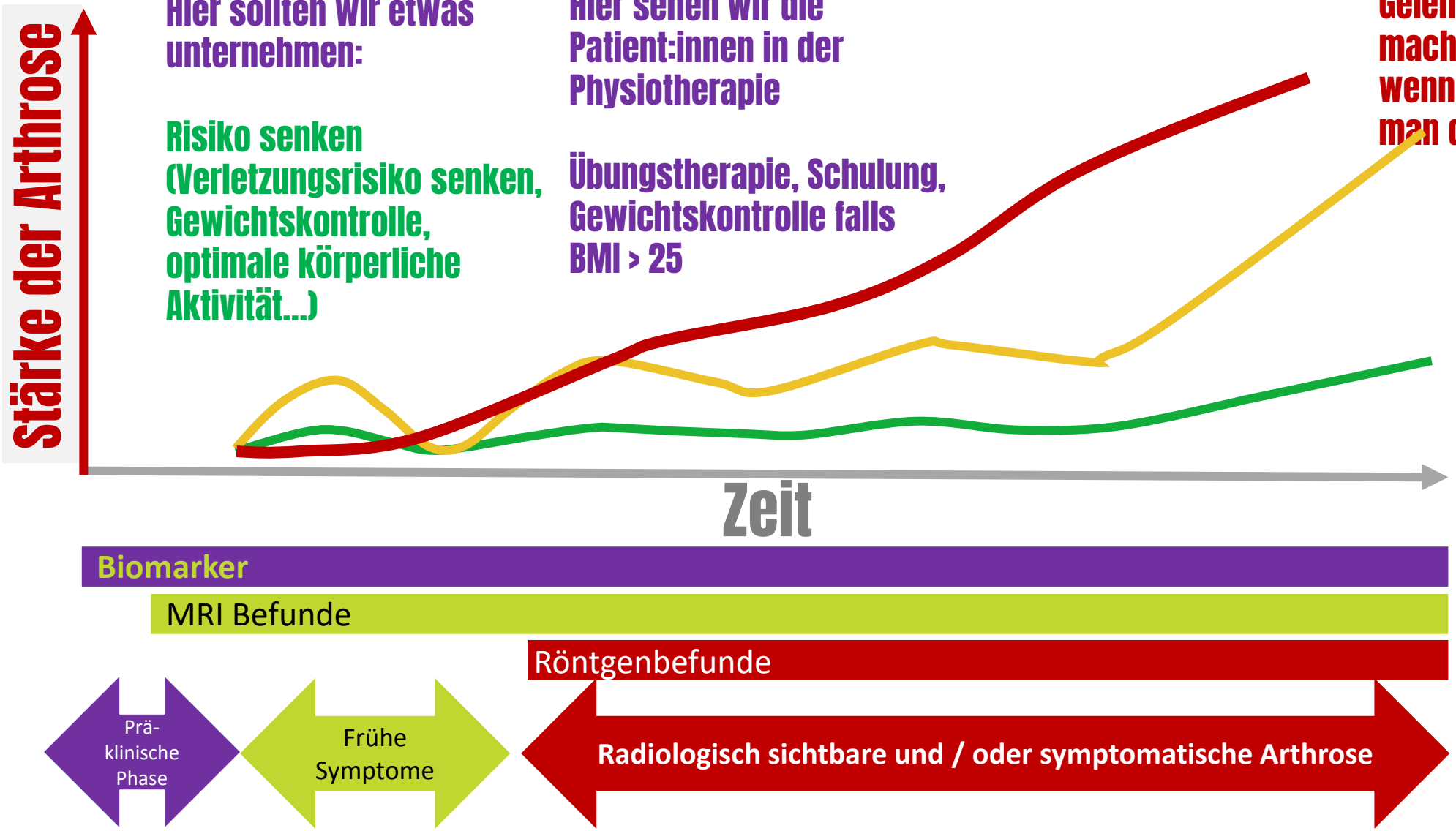
Hier sollten wir etwas unternehmen:

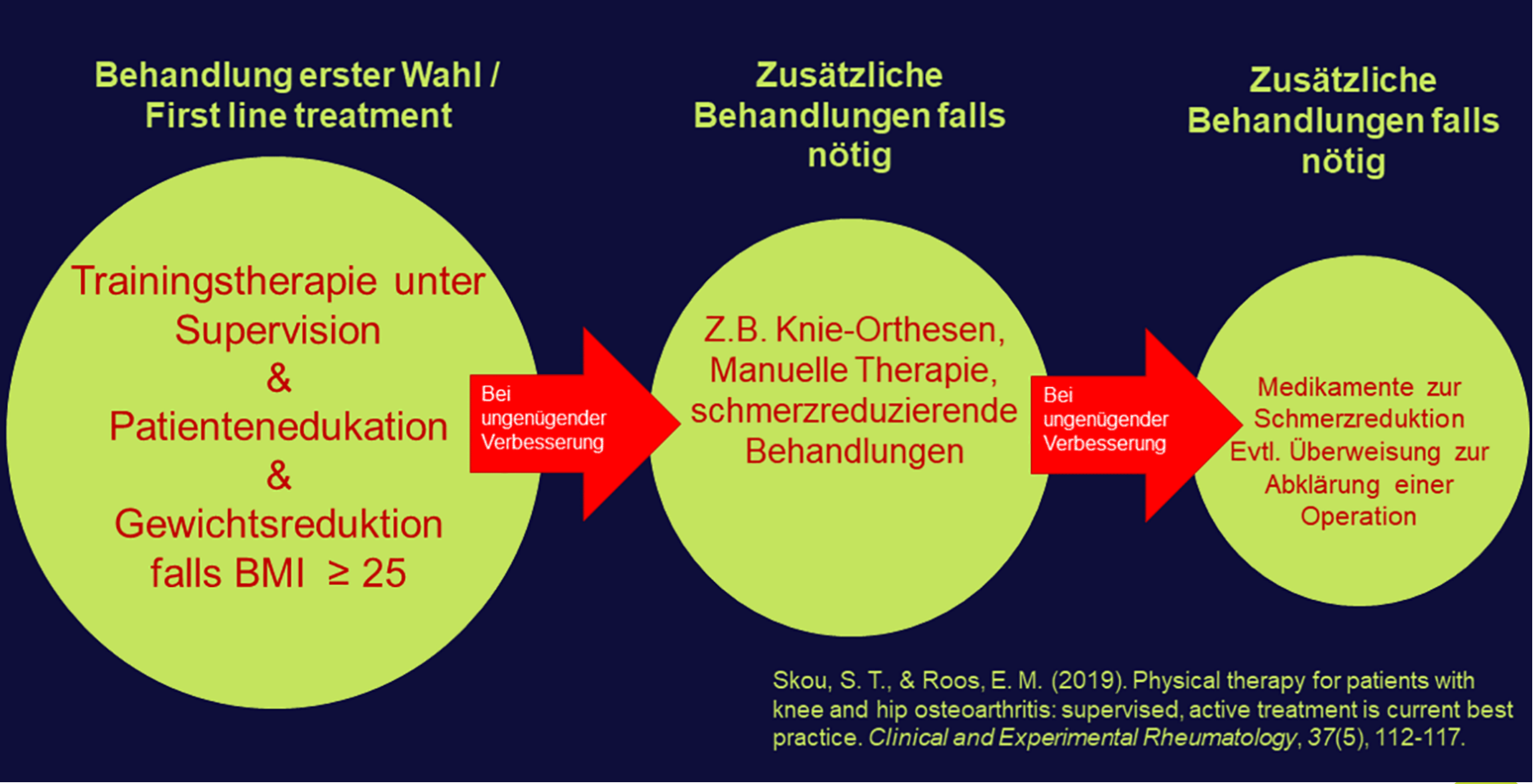
Risiko senken (Verletzungsrisiko senken, Gewichtskontrolle, optimale körperliche Aktivität...)

Hier sehen wir die Patient:innen in der Physiotherapie

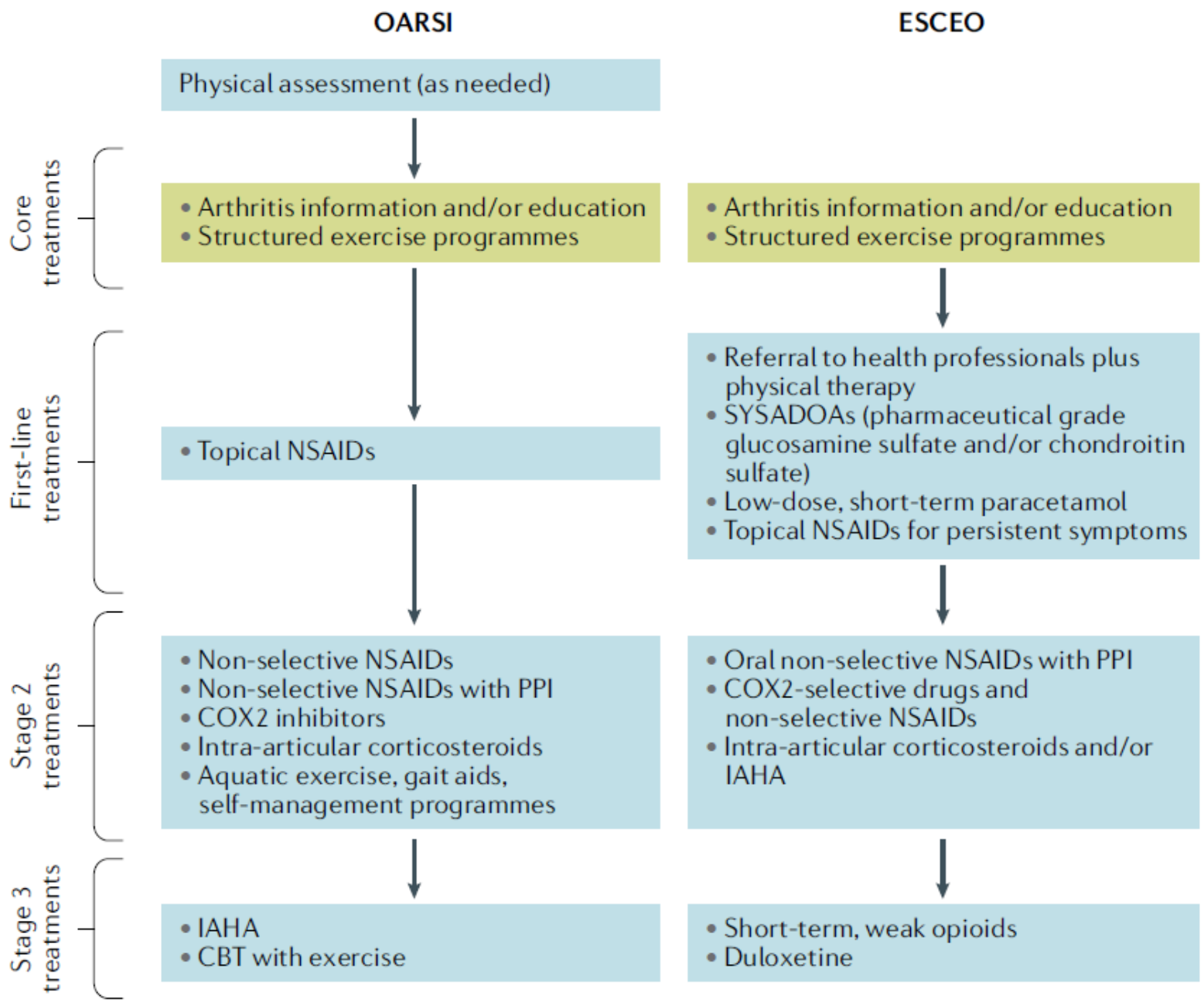
Übungstherapie, Schulung, Gewichtskontrolle falls BMI > 25

Nicht alle benötigen einen Gelenksersatz! Aber es macht ja auch Hoffnung, wenn man weiss, dass man diese Option hat





Skou, S. T., & Roos, E. M. (2019). Physical therapy for patients with knee and hip osteoarthritis: supervised, active treatment is current best practice. *Clinical and Experimental Rheumatology*, *37*(5), 112-117.



Nur als «Beweis», dass wir uns das nicht aus den Fingern gesogen haben.

Internationale Experte (nicht Physiotherapeuten, sondern Ärtze) geben diese Empfehlungen ab.

Arden, N. K., Perry, T. A., Bannuru, R. R., Bruyère, O., Cooper, C., Haugen, I. K., ... & Reginster, J. Y. (2021). Non-surgical management of knee osteoarthritis: comparison of ESCEO and OARSI 2019 guidelines. *Nature Reviews Rheumatology*, *17*(1), 59-66.
https://doi.org/10.1038/s41584-020-00523-9

Was machen wir mit Arthrose?

- Übungstherapie → Beweglichkeit, Gleichgewicht, Bewegungskontrolle, Krafttraining.
- Falls notwendig: Manuelle Therapie (zur Verbesserung der Beweglichkeit)
- Patientenschulung (Information)
- Individuelle Physiotherapie oder GLA:D Programm (je nach Wunsch der Patient:innen oder des Arztes).

1. Einzelsitzung
Untersuchung
Fragebogen ausfüllen

2. Einzelsitzung
Tests
Ziele setzen
Einführung Übungsprogramm

3. Einzelsitzung
Einführung Übungsprogramm
Unterlagen abgeben

2 Gruppensitzungen
Patientenschulung

Arthrose:
12 Gruppensitzungen
Übungsprogramm
2 x pro Woche für 6 Wochen

Rücken:
12 Gruppensitzungen
Übungsprogramm
2 x pro Woche für 4 Wochen
1 x pro Woche + 1 x Heimprogramm für 4 Wochen

4. Einzelsitzung
Abschlussgespräch
Tests
Evaluation
Abschlussbericht

Ablauf GLA:D Arthrose und GLA:D Rücken

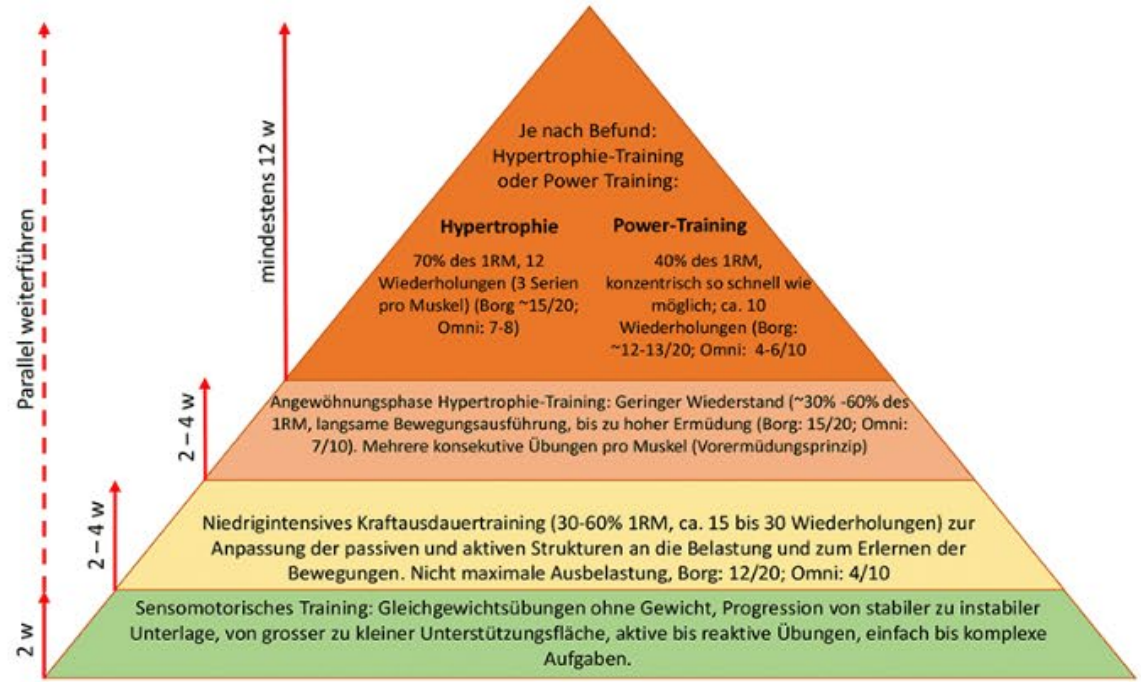
PT&H

Physiotherapie Tschopp & Hilfiker
Englisch-Gruss-Strasse 32
3902 Glis

Was kommt nach GLA:D?

Sie lernen im GLA:D Programm, selbständig zu trainieren.

Sie können jedoch auch bei uns ein progressives Krafttraining durchführen, oder in einem Fitnesszenter trainieren.



Beispiel individueller Therapie

- Wenn wir beobachten, dass jemand die Beinlängsachse nicht optimal stabilisieren kann → eher neuromuskuläres Training

- Wenn dies nicht der Fall ist → Aufbau progressives Krafttraining

- → Achtung: Krafttraining benötigt einen gut geplanten Aufbau, damit es nicht zu Überlastungen kommt.


Danke für Ihr Interesse!
cargo

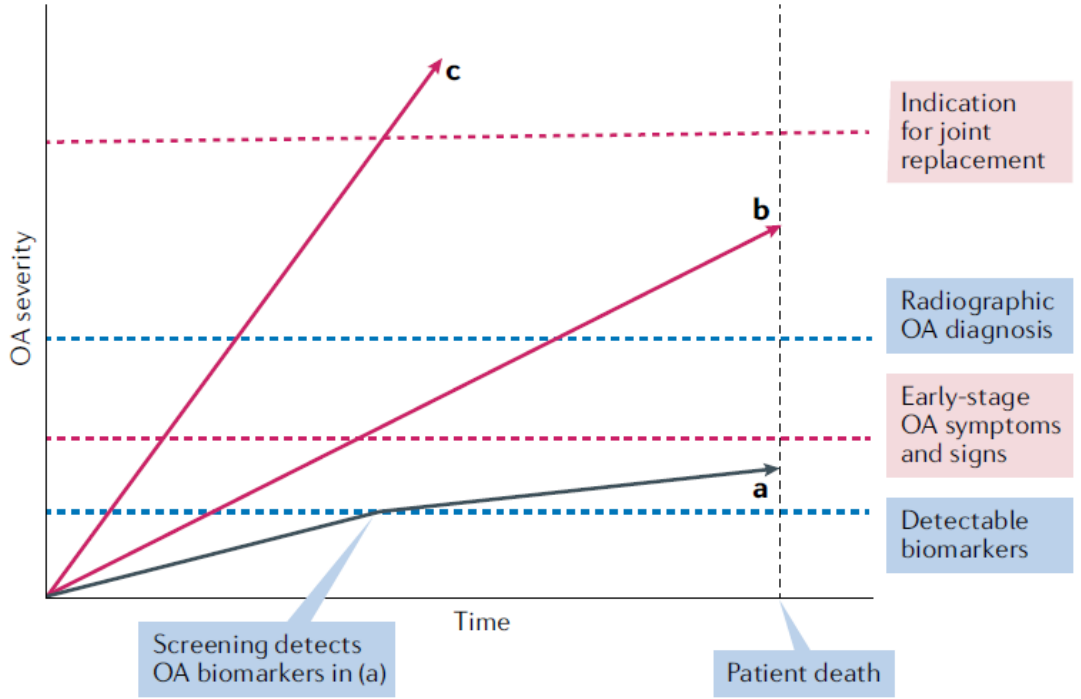


Fig. 5 | **The potential for overdiagnosis and overtreatment of OA.** In this schematic, the vertical axis represents 'global' osteoarthritis (OA) disease severity and the horizontal axis represents time (and patient age). The two horizontal dashed red lines represent the degree of severity (disease stage) at which symptoms begin, or become so severe that joint replacement surgery may be indicated. The lower horizontal dashed blue line represents the disease stage at which biomarkers (MRI or molecular biomarkers) might first detect an increased risk for disease, and the upper horizontal dashed blue line when a diagnosis of radiographic OA can be made using plain radiography. The vertical dashed line represents the time at which death could take place. The arrows labelled a, b and c represent OA disease trajectories with different rates of disease development. In this schematic, individuals along trajectory a, whose disease progresses slowly or not at all, would be over-diagnosed by biomarkers; they would never experience symptoms and any treatment would be overtreatment. Individuals along trajectories b and c could benefit from both having their symptoms recognized as early-stage OA and receiving beneficial symptomatic treatment before the point at which a radiographic diagnosis would be made. Future research could show whether OA treatment at this early stage can modify the development of further symptoms and structural joint changes for individuals along trajectories b or c, thus providing OA disease modification.

https://doi.org/10.1038/s41584-021-00673-4

ACR classification criteria for knee OA

Clinical and laboratory criteria	Clinical and radiographic criteria	Clinical criteria
• Knee pain At least five of the following: • Age >50 years • Stiffness <30 min • Crepitus • Bony tenderness • Bony enlargement • No palpable warmth • ESR <40 mm/h • RF <1:40 • Synovial fluid analysis indicative of OA	• Knee pain • Osteophytes At least one of the following: • Age >50 years • Stiffness <30 min • Crepitus	• Knee pain At least three of the following: • Age >50 years • Stiffness <30 min • Crepitus • Bony tenderness • Bony enlargement • No palpable warmth

Proposed classification criteria for early-stage knee OA

Using MRI or arthroscopic findings	Without MRI data

• Knee pain: at least two episodes of pain for >10 days in the past year
• Standard radiography: KL grade 0 or 1 or 2 (osteophytes only)
At least one of the following:
• Arthroscopy: ICRS grade I–IV in at least two compartments or grade II–IV in one compartment with surrounding softening and swelling
• MRI: at least two of the following:
  • At least grade 2 BLOKS for size of cartilage loss
  • At least grade 2 BLOKS for percentage full-thickness cartilage loss
  • Signs of meniscal degeneration
  • At least grade 2 BLOKS for size of bone marrow lesions
	• Patient-based questionnaires (KOOS): 2 out of the 4 KOOS subscales need to score 'positive' (≤85%)
	• Clinical examination: at least one of the following needs to be present:
	  • Joint line tenderness
	  • Crepitus
	• Radiography: KL grade 0–1 standing, weight bearing (at least two projections: posteroanterior fixed-flexion and skyline for patellofemoral OA)

Fig. 2 | **Comparison of newly proposed classification criteria for early-stage knee OA and the ACR classification criteria for knee OA.** Criteria for diagnosis and classification are related and could overlap in the features included, but diagnostic criteria focus on case finding and management of the individual patient in clinical practice whereas classification criteria aim to define disease with the goal of specifying homogeneous patient groups for studies of epidemiology, natural history and disease mechanisms and, importantly, for interventional studies. In contrast to diagnostic criteria, classification criteria typically aim to achieve high specificity and allow for lower sensitivity, and are thus less inclusive but more sharply defined. BLOKS, Boston Leeds Osteoarthritis Knee Score; ESR, erythrocyte sedimentation rate; ICRS, International Cartilage Repair Society; KL, Kellgren & Lawrence; KOOS, Knee Injury and Osteoarthritis Outcome Score; OA, osteoarthritis; RF, rheumatoid factor.

https://doi.org/10.1038/s41584-021-00673-4

Table 1 | Cytokines involved in the pathophysiology of OA

Cytokine	Activities in OA
TNF	Elevated in OA synovial fluid, synovial membrane, subchondral bone and cartilage Suppresses the synthesis of proteoglycan, link protein and type II collagen in chondrocytes[18] Stimulates the release of MMP-1, MMP-3 and MMP-13[27,28] Induces the production of IL-6[33] and chemokines such as IL-8,[34] MCP-1[35] and CCL5 (also known as RANTES)[36]
IL-1β	Elevated in OA synovial fluid, synovial membrane, subchondral bone and cartilage Suppresses type II collagen[20,21] and aggrecan expression[22] Stimulates the release of MMP-1, MMP-3 and MMP-13[27,28] Induces the production of IL-6[33] and chemokines such as IL-8,[34] MCP-1[35] and CCL5[36]
IL-6	Elevated in synovial fluid and sera from patients with OA[81] Upregulates MMP-1 and MMP-13 expression in combination with IL-1β and oncostatin[82,83] Reduces expression of type II collagen[85]
IL-15	Elevated in synovial fluid in early OA Suggested association with MMP-1 and MMP-3[104]
IL-17	Induces IL-1β, TNF and IL-6 production Upregulates NO and MMPs[105] Downregulates proteoglycan levels[106]
IL-18	Elevated in human OA chondrocytes Structurally similar to the IL-1 protein family Acts through both IL-1-dependent and IL-1-independent pathways in OA[107]
IL-21	Elevated in synovial fluid in early OA
LIF	Elevated in OA synovial membrane and fluid Produced in response to IL-1β and TNF Stimulates cartilage proteoglycan degradation, MMP synthesis and NO production[108]
Chemokines (CCL5, IL-8, GROα and MCP-1)	Elevated in OA tissues Induce iNOS, MMP-1 and IL-6 Stimulate proteoglycan depletion[36,108]
Anti-inflammatory cytokines (IL-4, IL-10 and IL-13)	Elevated in OA tissues Inhibit IL-1β, TNF and proteases Upregulate IL-1Ra and TIMP production[39]

Abbreviations: CCL, CC-chemokine ligand; GRO, growth-related oncogene; IL, interleukin; IL-1Ra, IL-1 receptor antagonist; iNOS, inducible nitric oxide synthase; LIF, leukemia inhibitory factor; MCP, monocyte chemotactic protein; MMP, matrix metalloproteinase; NO, nitric oxide; OA, osteoarthritis; RANTES, regulated upon activation, normal T cell expressed and secreted; TIMP, tissue inhibitor of metalloproteinases; TNF, tumor necrosis factor.

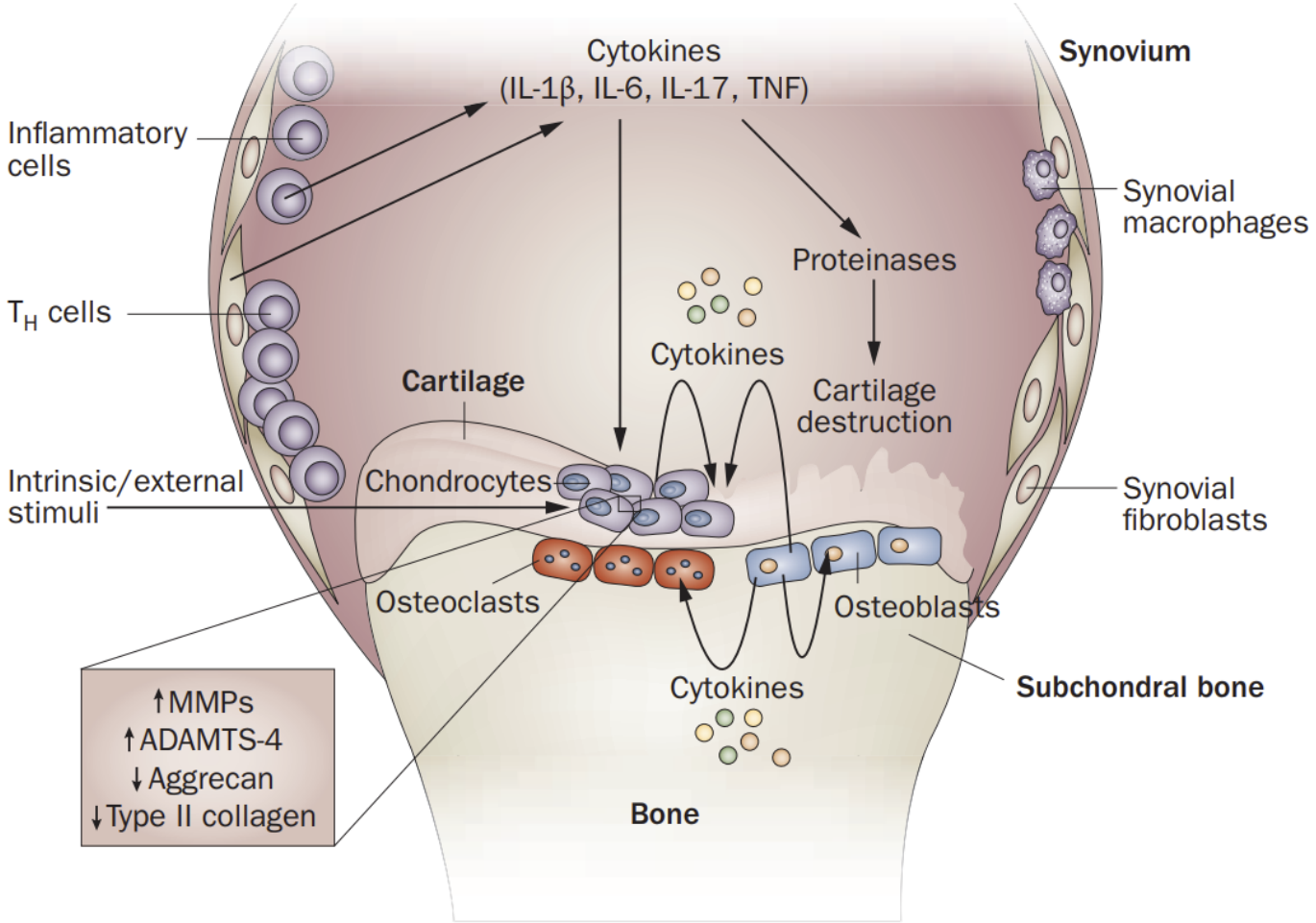


Figure 1 | The role of proinflammatory cytokines in the pathophysiology of OA. The levels of proinflammatory cytokines, including IL-1β, TNF and IL-6, are elevated in OA. These cytokines contribute to the pathogenesis of OA through several mechanisms including downregulation of anabolic events and upregulation of catabolic and inflammatory responses, effects that result in structural damage to the OA joint. Abbreviations: ADAMTS, a disintegrin-like and metalloproteinase with thrombospondin type 1 motifs; IL, interleukin; MMP, matrix metalloproteinase; OA, osteoarthritis; TNF, tumor necrosis factor.

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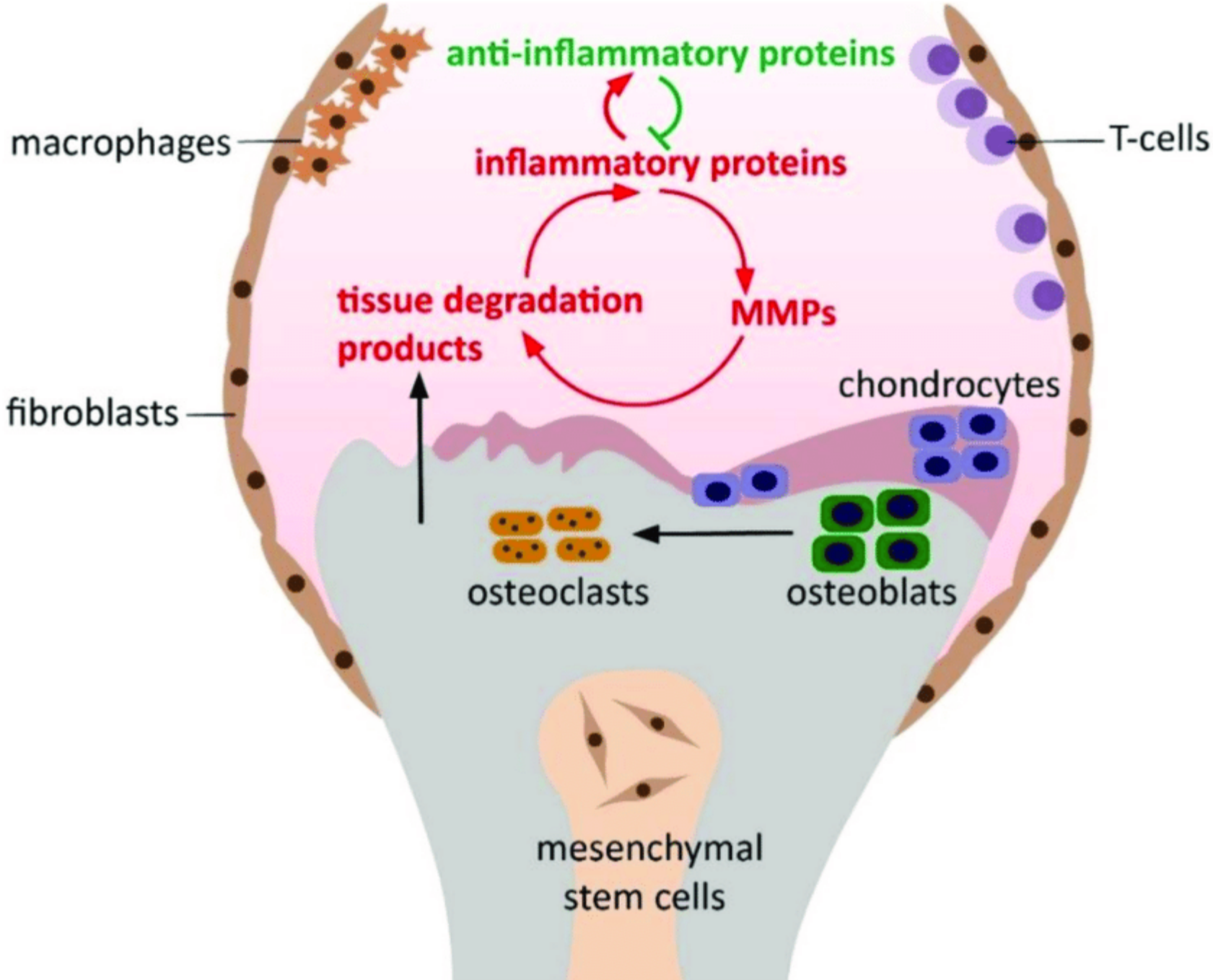

anti-inflammatory proteins
macrophages
T-cells
inflammatory proteins
tissue degradation products
MMPs
fibroblasts
chondrocytes
osteoclasts
osteoblats
mesenchymal stem cells

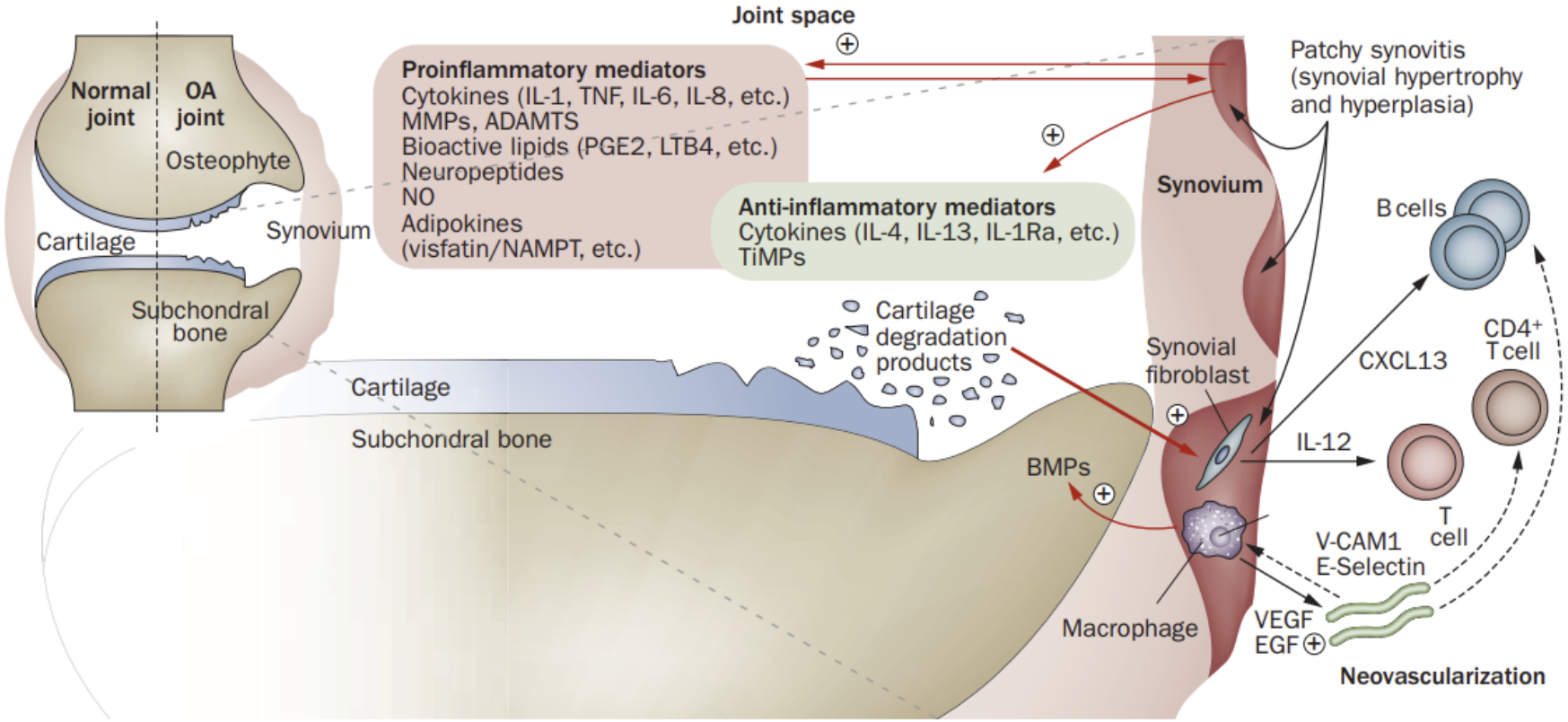


Figure 1 | Involvement of the synovium in OA pathophysiology. Products of cartilage breakdown that are released into the synovial fluid are phagocytosed by synovial cells, amplifying synovial inflammation. In turn, activated synovial cells in the inflamed synovium produce catabolic and proinflammatory mediators that lead to excess production of the proteolytic enzymes responsible for cartilage breakdown, creating a positive feedback loop. The inflammatory response is amplified by activated synovial T cells, B cells and infiltrating macrophages. To counteract this inflammatory response, the synovium and cartilage may produce anti-inflammatory cytokines. In addition to these effects on cartilage inflammation and breakdown, the inflamed synovium contributes to the formation of osteophytes via BMPs. Abbreviations: ADAMTS, a disintegrin and metalloproteinase with thrombospondin motifs; BMP, bone morphogenetic protein; CCL2, CC-chemokine ligand 2; CXCL13, CXC-chemokine ligand 13; EGF, endothelial growth factor; GM-CSF, granulocyte-macrophage colony-stimulating factor; IL, interleukin; IL-1Ra, IL-1 receptor antagonist; LIF, leukemia inhibitory factor; LTB4, leukotriene B4; MMP, matrix metalloproteinase; NAMPT, nicotinamide phosphoribosyl transferase (also called visfatin); NO, nitric oxide; NGF, nerve growth factor; OA, osteoarthritis; PGE2, prostaglandin E2; TIMP, tissue inhibitor of metalloproteinase; TNF, tumor necrosis factor; VCAM-1, vascular cell adhesion molecule 1; VEGF, vascular endothelial growth factor.

doi:10.1038/nrrheum.2010.159

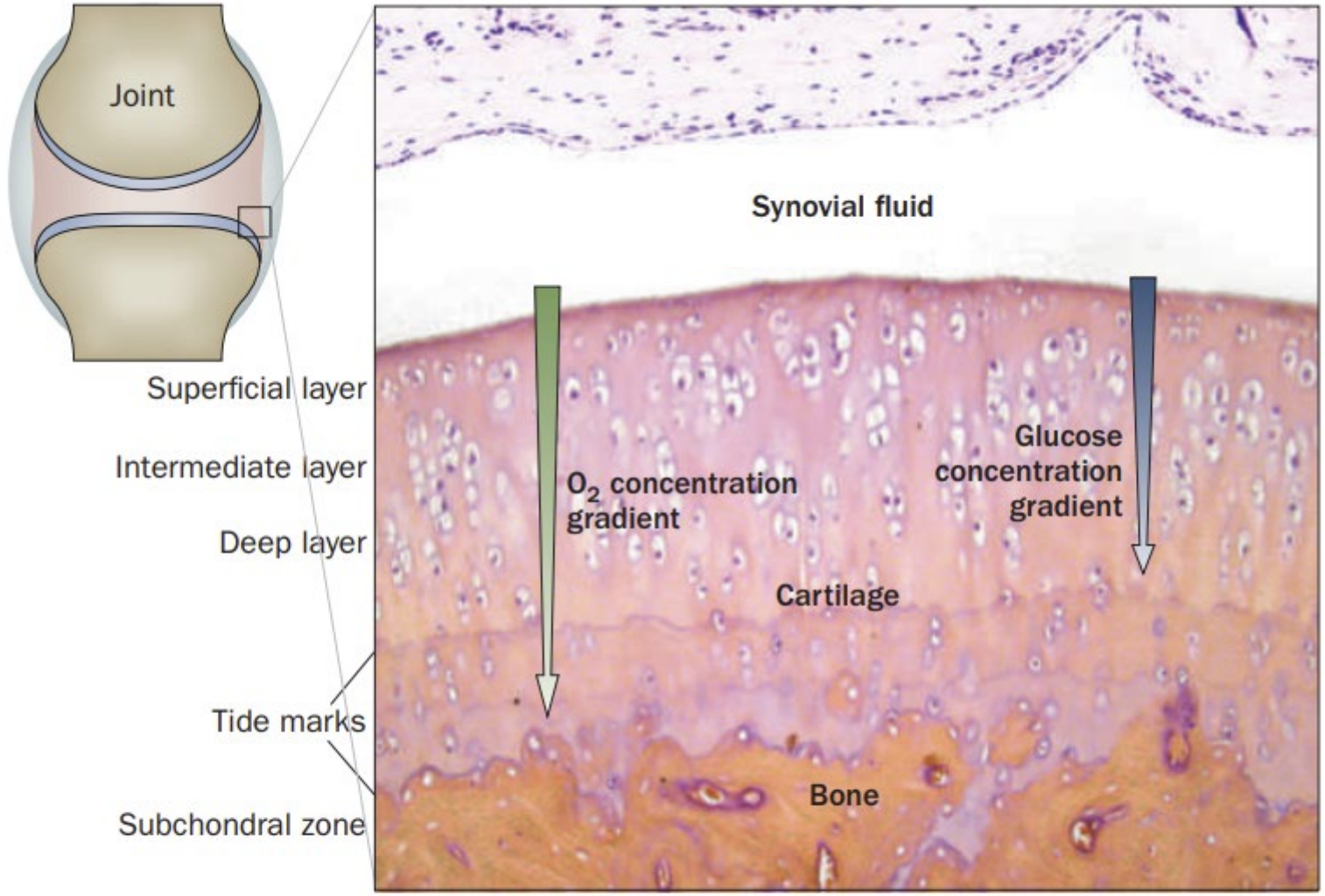


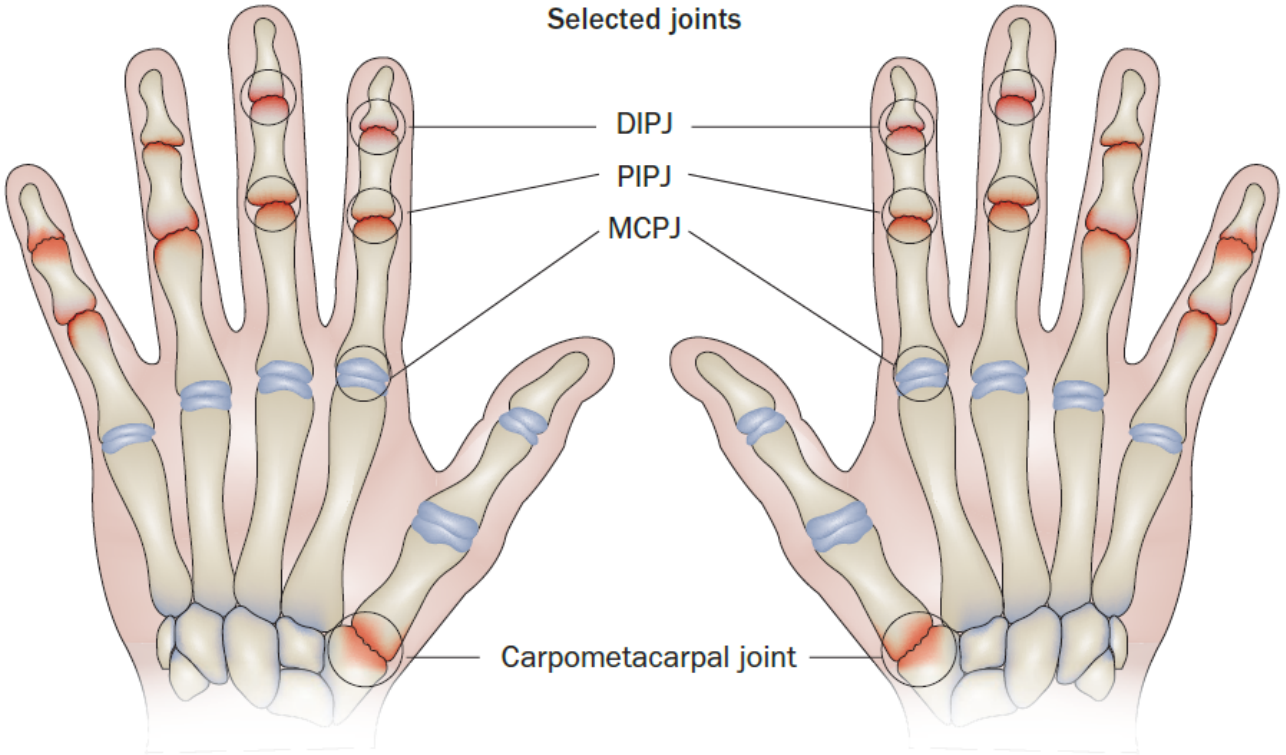
Figure 2 | Diffusion of glucose and oxygen into articular cartilage. Articular cartilage is avascular, which means that chondrocytes must obtain nutrients and oxygen via diffusion from the synovial fluid. Thus, these two compounds show a gradient of concentrations in the cartilage, being lower in the deeper layers than at the surface.

Chondrozyten

- Die Zellen des Gelenkknorpels können Knorpelabbauende Stoffe (matrix metalloproteinases) und entzündungsfördernde Stoffe(hautpsächlich Interleukin 1 und TNF) produzieren.
- Sie produzieren auch andere Stoffe, wie Prostaglandine, NO, und Reaktive Sauerstoffspezies, die auch in Verbindung mit Arthrose stehen.

doi:10.1038/nrrheum.2012.135

Handarthrose



ACR criteria

Hand pain, aching or stiffness
+
Three of the following four criteria

- Hard tissue enlargement of at least two of 10 selected joints
- Hard tissue enlargement of at least two DIPJs
- Swelling of fewer than three MCPJs
- Deformity of at least one of 10 selected joints

Figure 1 | Classification criteria of the ACR for hand OA. Abbreviations: ACR, American College of Rheumatology; DIPJ, distal interphalangeal joint; MCPJ, metacarpophalangeal joint; PIPJ, proximal interphalangeal joint; OA, osteoarthritis. Permission obtained to adapt ACR criteria from John Wiley and Sons © Altman, R. *et al.* The American College of Rheumatology criteria for the classification of osteoarthritis of the hand. *Arthritis Rheum.* **33**, 1601–1610 (1990).

Table 1 | Prevalence, risk factors, disease course and clinical burden in hand OA subsets

Hand OA subset	Prevalence	Risk factors	Disease course	Clinical burden
Nodal hand OA	Radiographic OA: up to 81% of elderly population[8,15] Symptomatic OA: varies from 2.0–20.4%[15–20]	Age[15,17,18,21,23] Female sex[24] High bone mineral density[26–28] Obesity[29] Mechanical or occupational factors and sports[32–36] Familial factors[37–42] Genetic factors: *RBFOX1* (also known as *A2BP1*) variant (rs716508);[43] mutation in *MATN3*;[44] aggrecan VNTR polymorphism;[45] IL-1 region (*IL1B* and possibly *IL1RN*)[46]	Heterogeneous in general Considered as a slow process[59] After 10 years: 90% and 74% progression of osteophytes and JSN, respectively[61]	Variable: mild to high Potential influence on pain, joint mobility, grip strength, functional ability and aesthetics[48,49,97]
Thumb base OA	Radiographic age-adjusted 1st CMCJ OA: 7% in men, 15% in women[21] Above 55 years of age: 35.8%[8] Symptomatic 1st CMCJ OA in elderly: 1.9–4.1%[16,73]	Hypermobility[74–76] Obesity[21,72,77] Mechanical factors[35,78–81] Familial factors[82,83] Genetic factors: mutations in *MATN3*[44,84,85]	Rarely studied After 10 years: radiographic progression in 1st CMCJ in 38% for osteophytes and in 48% for JSN[61]	Relative contribution of thumb base OA in hand OA is controversial Affects pain, grip strength, disability[8,68,72,77,87–89]
Erosive OA	In elderly population: 2.8%[93] Symptomatic: 6.9–10.2%[93,95]	Familial factors[92,97] Genetic factors: *IL1B* 5810;[101] MS α1-antitrypsin;[102] *HLA-DRB1*07* allele[103] Obesity[93]	No information in literature	Higher clinical burden (more pain, disability, stiffness) and worse outcome than nonerosive hand OA, eventually leading to instability and ankylosis[50,95,96,104]

Abbreviations: CMCJ, carpometacarpal joint; JSN, joint space narrowing; MATN3, matrillin-3; OA, osteoarthritis; RBFOX1, RNA binding protein fox-1 homolog 1; VNTR, variable number of tandem repeats.

https://doi.org/10.1038/s41584-018-0073-x

Lebensstil und Arthrose

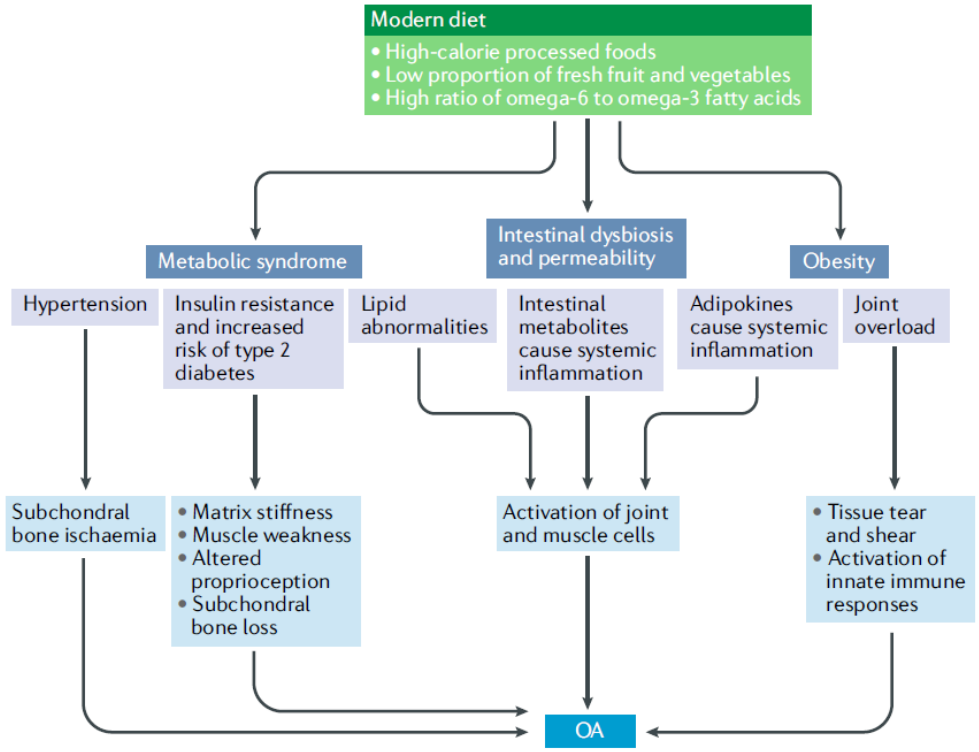


Fig. 3 | **Diet as a mismatch factor.** The deleterious effects of modern diets on osteoarthritis (OA) arise from increased adiposity and body mass, which leads to joint overload, a well-established risk factor for OA. Moreover, high caloric intake, a low proportion of fresh fruit and vegetables and a high ratio of omega-6 to omega-3 fatty acids, which are all hallmarks of a modern diet, participate in an increase in intestinal dysbiosis and permeability. These intestinal alterations increase systemic low-grade inflammation, a biological response suggested to trigger joint cell activation in OA. Modern diets are also considered the main cause of metabolic syndrome, which includes hypertension, insulin resistance and lipid abnormalities. Each of these pathologies could indirectly have an important function in OA pathogenesis through deleterious effects on joint tissues.

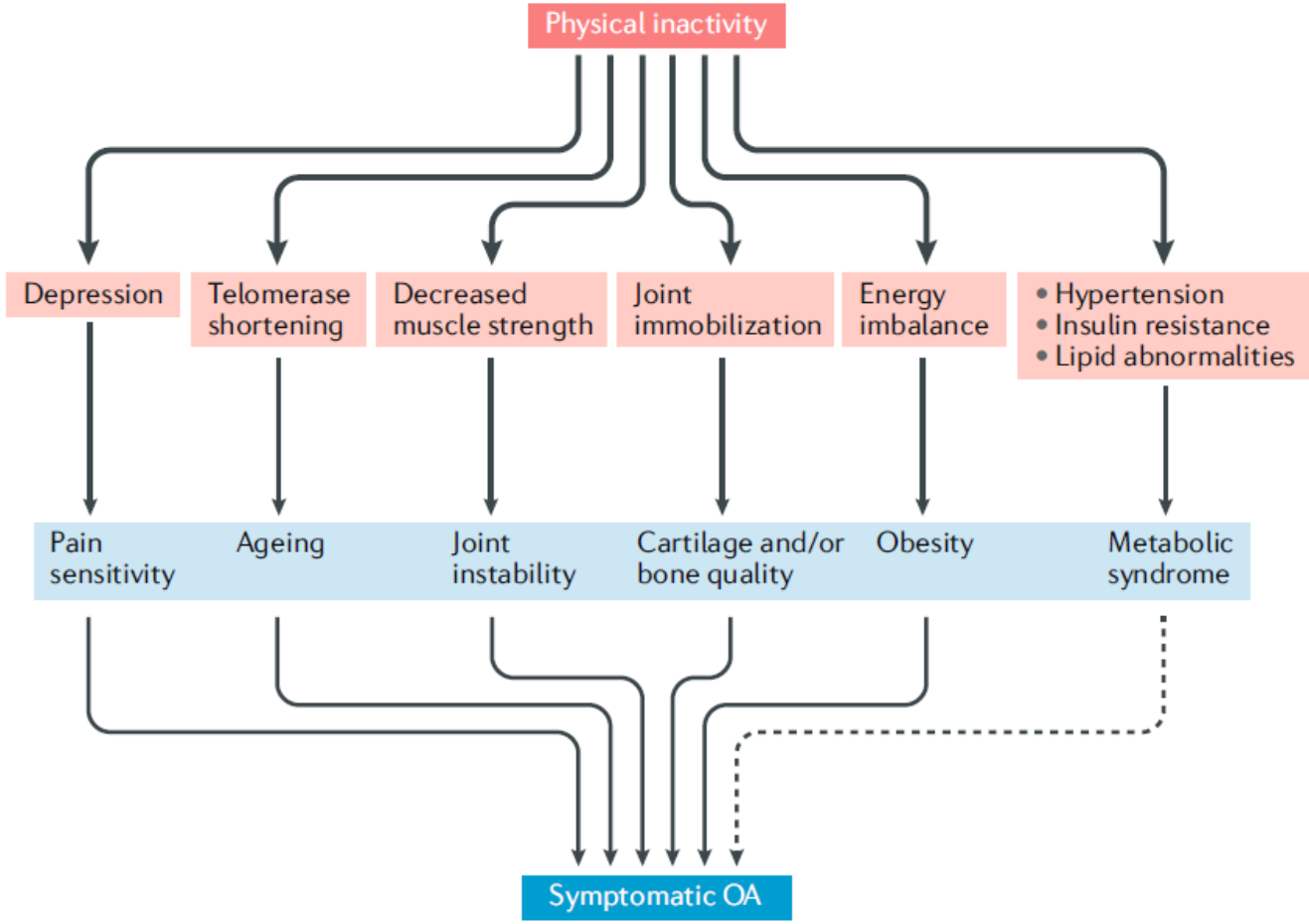


Fig. 4 | **Physical inactivity as a mismatch factor.** A sedentary lifestyle and physical inactivity might initiate and aggravate osteoarthritis (OA) and its symptoms via a variety of pathways.

Krafttraining oder Neuromuskuläres Training

- Falls beim Stehen oder Gehen Tendenz zu X- oder O-Beinen → Neuromuskuläres Training
- Falls beim Stehen oder Gehen keine Tendenz zu X- oder O-Beinen → Starten mit sensomotorischem Training, kurze Phase neuromuskuläres Training, danach Krafttraining.

Sensomotorisches Training: Fokus auf Gleichgewicht und Bewegungsqualität. Gleichgewichtsübungen.
Neuromuskuläres Training: Fokus auf Bewegungsqualität. ~15 Wiederholungen
Krafttraining: Gewicht so, dass nach 10 Wiederholungen starke Müdigkeit in der Muskulatur vorhanden ist

→ Achtung: Eher

Bennell, K. L., Dobson, F., Roos, E. M., Skou, S. T., Hodges, P., Wrigley, T. V., ... & Hinman, R. S. (2015). Influence of biomechanical characteristics on pain and function outcomes from exercise in medial knee osteoarthritis and varus malalignment: exploratory analyses from a randomized controlled trial. *Arthritis care & research*, *67*(9), 1281-1288. https://doi.org/10.1002/acr.22558

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