Ageing in modern society has created an inverse population pyramid in most Western countries. As a consequence, degenerative diseases have become a huge part of the pathologies seen in general practice. One of the main pathologies seen in ageing population is arthrosis, a degenerative pathology of the cartilage and in later stages also involving the bone, causing various degrees of dysfunctions of the affected joints.
Rheumatic diseases: classification

- Inflammatory rheumatism: arthritis
- Degenerative rheumatism: arthrosis
- Rheumatism of the soft tissue: e.g. tendonitis
- Rheumatism of the bone: osteoporosis
- Para-rheumatic diseases: gout

Rheumatism is a general term that refers to any painful disorder of joints, muscles or connective tissues. To rheumatological disorders belong also any condition that afflicts the connective tissues of various organs including the heart, lungs, bones, kidneys and skin.

A rheumatic disorder is marked by:

- Inflammation and/or
- Degeneration
- Metabolic derangement

of the connective tissue structures of the body, especially the joints and related structures, including muscles, bursae, tendons, and fibrous tissue.

Therefore rheumatism is a “family name” for a large number of diseases that afflict especially the locomotor system. Five classes are generally distinguished:

Inflammatory rheumatism: rheumatic diseases where inflammation prevails such as rheumatoid arthritis (an auto-immune disease).

Degenerative rheumatism: degenerative disorders of the locomotor system such as arthrosis (subject of this lecture) Cell death and loss of structure and tissue is at the level of the joints, more precisely the cartilage initially, later may extend to the bone structures.

Rheumatism of the soft tissues: pathologies like carpal tunnel syndrome, periarthritis of the shoulder, polymyalgia rheumatica and fibromyalgia are examples of soft tissue rheumatism.

Rheumatism of the bone: degenerative pathologies of the bone itself. Main example here is osteoporosis.

Para-rheumatic diseases is a group of locomotor pathologies that cannot be classified under the 4 former classes (e.g. gout).
What is arthrosis?

Arthrosis is a premature and excessive wearing away of the layers of cartilage on the bone, or even complete loss of the cartilage. In simple cases this loss of cartilage can be detected by x-rays, which reveal the degeneration of the joint capsule. Magnetic resonance imaging (MRI) is even better and more accurate. The final stages of an arthrosis disorder are accompanied by a visible deformation of the joint, which is why some forms are referred to as arthrosis deformans. Also, because of narrowing of the joint cavity and bony build-up on the edge, mobility is increasingly restricted, which can even cause an almost complete immobilization of the joint.

Arthrosis progresses in exacerbations and remissions. During the inactive phase, the joint is not swollen or particularly painful, although it is considerably less resilient. Additional injuries or overtaxing can then quickly trigger an active or inflamed condition, whereby swelling and pain are prevalent – sometimes even when at rest.
Within the group of the rheumatic diseases, the prevalence of arthrosis in Western countries is about 70%. Beside a high prevalence, it is also creating a heavy burden on health budgets for the society. Conventional treatments for arthrosis is primarily symptomatologic and focuses on pain and inflammation treatment. Beside a few chondro-protectives (e.g. hyaluronic acid injections) and surgical interventions (e.g. prothesis), common treatment of arthrosis is not effective in the long term.
Characteristics & symptoms

- Stiffness
- Start pain
- Pain in movement, especially when loaded
- Inflammation with periods of latency
- Cartilage degeneration
- Bone deformation at the level of the joint

The symptoms of arthrosis include a feeling of stiffness in the joint, especially in the beginning of a movement (e.g. walking after laying down). Often occurring is also the so called “start-pain” in the beginning of a movement. This pain decreases after a while as the movement itself seems to “lubricate” the joint.

Movement with weight bearing is more painful than a normal movement (e.g. carrying a heavy bag or going upstairs). In reactivated arthrosis inflammatory processes are obvious. Beside the degenerative aspects of the arthrosis, an acute arthritic type of reaction is often simultaneously present.
Arthrosis can be seen as a status of imbalance between the amount of new cartilage generated by the cooperation of synovial cells (hyaluronic acid) and chondrocytes (collagen and proteoglycans/glycosaminoglycans) and the degeneration of the cartilage by wear and tear, and various forms of enzymatic processes.

When a person is young, this balance is in favor of the generation of cartilage, the reason why we do not usually see arthrosis in young people. In adults the balance, little by little, shifts in favor of the degeneration of the cartilage, developing the condition of arthrosis.

In the final stages, when the cartilage is completely destroyed and sub-chondral bone touches sub-chondral bone, deformations of the bone-ends will become evident.
4 clinical-radiological stages of arthrosis are distinguished (1):

• Stage I: Mild clinical symptoms appear. Discrete sclerosis can be seen on the X-ray picture of the affected joint, the articular space and the condyle remain intact.

• Stage II: The mobility of the joint decreases because of the pain at the start of a movement and the rigidity of the joint. On the X-ray film small cysts and osteophytes may appear.

Radiologically, arthrosis may be classified into 4 successive stages.

In stage 1, arthrosis starts. Beside a discrete sclerosis there is not much to see on RX. Clinical symptoms are mild, appear and disappear in function of more or less loaded movement and activities. Important is that the clinical symptoms are varying very much in function of the use of the joint and in contrary to what would be commonly thought, unloaded movement (walking, swimming,….) is curative and ameliorating the condition of the joint.

Stage 2 Initial signs indicative of some impairment of mobility. The stiffness after rest, at the onset of a movement, gets worse. There is also movement start-pain and loss of strength (stairs, getting up from a kneeling position, etc…). On X-Rays the first fine osteophytes appear and even small subchondral cysts might be present.
4 clinical-radiological stages of arthrosis are distinguished (2):

- **Stage III:** The mobility of the joint becomes significantly limited. Secondary acute inflammation may occur. Subchondral cysts can be observed on the X-ray picture. The articular space becomes irregularly narrowed.

- **Stage IV:** Severe deformation of the joint: cystic degeneration, osteophytes, extremely narrow articular space. Bone on bone.

In **stage 3** Signs of strong mobility inhibition, partly by stiffness and start-pain, partly by changed physical aspects like narrowed synovial space and beginning of joint deformation. Strength is limited and inflammation may occur and make the picture even more complex. On X-Rays osteophytes and cysts are normally present.

**Stage 4** The final stage in which flexibility of the joint is gone. The patient strongly compensates for the immobile joint. There is recurrent inflammation and muscle decompensation. On X-Rays are visible signs of osteophytes, cysts, and the synovial space is extremely narrowed or even completely obliterated (subchondral bone contact).
As arthrosis is a degenerative pathology, mainly characterized by loss of tissue and cell death (chondrocytes), it will be located at the right of the Regulation/Compensation Division, in the 5th phase of the Disease Evolution Table, the Degeneration phase.

Since bones and joints differentiate from the mesodermal embryonic layer, arthrosis, therefore can be found at the mesodermal cavodermal level on the table.

Thus, from a Homotoxicological point of view, arthrosis is a cavodermal degenerative pathology.

Typically of conditions in the inflammatory phase, this is characterized by inflammation and an overactive repair process (osteophytes)
Consequences

- Without therapy, the condition tends to deteriorate.
- Inflammatory pattern should be seen as an attempt for regressive vicariation. The organism tries to eliminate endogenous homotoxins over an inflammation (arthritis on arthrosis)
- Suppression therapy will promote the chronic state.

The fact that arthrosis is located at the 5th phase on the Disease Evolution Table (DET) has some particular clinical and therapeutic implications.

Time is against arthrosis, because with time the status of the patient gets worse. There is no spontaneous regeneration of the joint and the progression of the degeneration can accelerate due to weight bearing load and general stressing of the joint. There may be temporary and limited spontaneous remissions, but the evolution is progressive.

In reactivated arthrosis we may see inflammatory processes that, in contrary to conventional medical approach, should be seen as attempts of the organism to eliminate some form of irritating factor or toxin (homotoxin) out of the synovial space through the process of inflammation. Although the inflammation will further damage the cartilage due to enzymatic processes, on short term it eliminates disturbing factors like fragmented parts of cartilage and other “homotoxins”. Suppression of these inflammatory processes, instead of inflammation regulation (immunomodulation), is therefore in the long term not the best strategy. Suppression therapy will eventually lead the patient into a rebound and accelerated chronic degenerative process.
What is the physiopathological process behind arthrosis? We will see that the interpretation of this process is different in conventional and anti-homotoxic medicine.
• Cartilage is dense matrix
  • no blood vessels
  • no nerve ends
  • no lymphatic vessels

Cartilage is very similar to a “compressed” matrix. The main components of the matrix are collagen, glycosaminoglycans and hyaluronic acid. The chondrocytes generate the matrix structure, using hyaluronic acid molecules that are provided by the synovial cells. By pressure and release, due to pressure and movement of the joint, the so called “sponge effect”, allow the nutrients and building blocks to be absorbed by the cartilage structure and during the release (pressure) phase byproducts of metabolism and homotoxins are expelled. This is why movement and exercise is so important in preventing and/or reducing the onset of degenerative processes, especially for individuals that may be predisposed to the condition and should be aided by early detoxification programs.

Cartilage contains no blood vessels nor lymphatic vessels. There are no nerve endings within the cartilage structure. All nutrients, defense cells and any other substance needed are provided by the synovial membrane through the synovial liquid. Toxic substances that may reach or leave the matrix also have to follow the same routes, as do medications.
Compared to the goldfish in an aquarium the quality of nutrition of the cartilage depends directly on the quality of the synovial liquid it is surrounded with.

The cartilage can be compared to the goldfish in the aquarium. Its life quality fully depends on the purity of the water in which they are swimming. The cartilage fully depends on the components available in the synovial liquid it is bathed.
• Cartilage is supplied with nutrients by the components in the synovial fluid that by the compression and decompression with joint movement will move fluids in and out of the structure.

Unloaded movement is healthy for the arthrosic joint as pressure and release mechanisms activate the "sponge effect", maintaining the local homeostasis.
Collagen structure in the cartilage

- **The basal zone**: a sclerosed zone which connects to the subchondral bone
- **The vertical zone**: the collagen fibres are directed (vertically) away from the subchondral bone
- **The flexible zone**: the vertical zone ends in horizontal direction and transfers into
- **The horizontal zone**: runs parallel to the surface

Collagen fibers within the cartilage are not distributed in a chaotic pattern, but are very well structured to create a basic layer in which the finer structures of proteoglycans are built in. To form a smooth surface of cartilage on the one hand, an elastic and buffering character of mid-structure on the other, and finally a well embedded connection into the subchondral bone, the collagen is structured in 4 phases.
On the left figure we see the 4 zones of collagen structure. Absorption of shocks and pressure is possible for vertical as well as for horizontal impact. In between the collagen fibers a fine web of proteoglycans guarantees a hydrophilic structure, needed to maintain the cartilage’s humidity and flexibility. Desiccated cartilage breaks under the smallest pressure. Well lubricated cartilage is extremely flexible.
At a certain age a stressing of the joint might lead to abrasions of cartilage with a wearing off of fragments from the cartilage surface, and ending up “floating” around in the synovial liquid. Movement will push the fragmented parts to the outside of the joint, irritating there the synovial membrane. Often this irritation triggers an inflammatory process, accompanied by the release of characteristic enzymes, meant to dissolve the fragment. As the cartilage surface is made up of the same components as the the fragment of cartilage, the enzymes that should act on the fragment will also damage the cartilage surface.

If not enough recovery time is taken to regenerate the damaged cartilage, recurrent stressing will end up in a vicious circle, degenerating the cartilage structure more and more. Strain, inflammation, and recurrence are the 3 main factors that will tend to perpetuate the progression of arthrosis.
Loose Parts of Cartilage in the Joint are endogenous Homotoxins

Physiological processes to eliminate the abraded parts should be seen as purposeful defense.

From the point of view of homotoxicology the fragmented parts of cartilage in the synovial space are considered as endogenous homotoxins. Although “self-made“ material they disturb the normal functioning of the joint. The elimination of the fragments by enzymatic processes joining the inflammation is a purposeful biological defense mechanism and should be respected, even supported and/or controlled, by all means.

The strategy should therefore be an immunomodulation and organ support ameliorating the life quality of the patient without blocking the elimination process, and support the regeneration mechanisms available as much as possible (amelioration of the function of the synovial cells and chondrocytes).
The four main components of cartilage (building blocks) can trigger the release of four corresponding enzymes. These enzymes are the so-called matrix metalloproteinases. One of the triggers for the MMP’s are pro-inflammatory cytokines.

**Mucopolysaccharidase** - will catalyze the hydrolysis of the glycosaminoglycans (also called mucopolysaccharides)

**Protease** - will catalyze the splitting of protein structures into smaller peptides and amino acids

**Collagenase** - will hydrolyze the collagen fibers.

**Hyaluronidase** will degrade hyaluronic acid (a jellylike aminoglycan).

Hyaline cartilage in the joints contains mainly collagen whereas elastic cartilage is found in the ear and glottis.
There are some major differences between the composition of old cartilage (aged individuals) and arthrotic cartilage. The main differences can be found in the protein and chondroitin sulfate content.

**Components:**

<table>
<thead>
<tr>
<th></th>
<th>Old Cartilage:</th>
<th>Arthrotic Cartilage:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chondroitin sulphate</td>
<td>Low content</td>
<td>Normal content</td>
</tr>
<tr>
<td>Proteoglycans (size)</td>
<td>Smaller</td>
<td>Normal size</td>
</tr>
<tr>
<td>Protein content</td>
<td>High</td>
<td>Low</td>
</tr>
</tbody>
</table>

Chondroitin sulphate (glycosaminoglycan or GAG) is low in old cartilage but normal in arthrotic cartilage.

The size of the proteoglycans is rather small in old cartilage but remains normal in arthrotic cartilage.

Protein content in old cartilage is high in old cartilage but low in arthrotic one. All this compared to normal cartilage.

Although there are some characteristic changes in arthrotic cartilage, the damage to the cartilage is mainly caused by enzymes accompanying inflammatory processes in recurrent strain and stressing of the joint.
Before describing the antihomotoxic therapeutic approach, we will enlighten some conventional ways of thinking and its therapeutic approach when it comes to the treatment of arthrosis.
Conventional approach

- **NSAIDs** were developed to cure **acute inflammation**. The objective of their application was short-term based. The extent to which the side effects appear is in function and proportion of the duration of application.

The main medication used in active arthrosis are the non-stereoidal anti-inflammatory drugs or NSAIDs. It is a class of diverse molecules (salicylates, ibuprofen, paracetamol, oxycans, COX-2-inhibitors, etc), mostly acting as non-selective inhibitors at the level of the enzyme cyclo-oxygenase, inhibiting the production of pro-inflammatory prostaglandines.

NSAIDs were developed to treat acute inflammatory patterns and certainly not chronic degenerative pathologies, even if they are accompanied by inflammation. The variety of side effects often seen with prolonged use of NSAIDs is generally accepted as normal. As with most conventional drugs, the prevalence of side effects is directly related to applied dose and duration of administration. Arthrosis in a CHRONIC degenerative pathology and the use of NSAIDs is often necessarily a long term application, causing long term suppression of regulation mechanisms and an increased risk of serious side effects.

This criticism is valuable as well for the first generation of NSAIDs (prostaglandin synthesis inhibitors), as for the second generation (COX-II inhibitors).
Conventional approach

- Observation confirms mistake:
  - Arthritis medication is used for arthrosis

NSAIDs were developed for acute inflammation treatments, and thus applied also in the treatment of acute arthritis (which is an acute inflammatory reaction), but we can easily confirm the mistake that arthritic medication is also used for the treatment of arthrosis (which is not an acute inflammatory process, but a chronic degenerative one, even if sometimes some signs of acute inflammation may be present) From the point of view of homotoxicology this is difficult to accept as arthritis is in a cavodermal inflammation phase (second phase on the DET) and arthrosis is a degeneration phase (fifth phase on the DET). This discrepancy in itself demonstrates the usefulness and the importance of the DET.
Side effects are not minor, as generally thought, with the chronic use of NSAIDs. In ageing individuals especially, increased risks for complications by those side-effects are real, as often there is already an underlying heart, liver or kidney problem. The blood platelet aggregation disturbance (easier and longer bleeding), water retention (heart failure), gastro-intestinal problems (burning gastritis, ulcus formation) and inhibited liver and/or kidney disturbances places these elderly patients at a very high risk.
Repeatedly, the Food and Drug Administration (FDA - USA), known as one of the most severe and critical observers of the pharmaceutical market when it comes to patient-protection, has warned about the (mis-) use of NSAIDs. They estimated that 20% of all suspected adverse drug reactions had to do with NSAIDs. 400,000 severe adverse events were reported in 1994, and it is estimated that in the USA alone about 20,000 people died in 1994 due to the use of NSAIDs. Within this group gastro-intestinal bleeding was the major cause for the number one death following NSAIDs-use/abuse.
When it comes to homotoxicology, the arthrosis treatment approach is very different from the conventional one. As it is a chronic degenerative pathology all strategies of the 3 pillars of antihomotoxic therapy are to be employed.
Antihomotoxic treatment

• To clean the terrain of the patient
• To regulate the inflammation process at the beginning of the inflammatory chain (no suppression)
• To sustain the affected tissue
• To regenerate where possible
• To improve the life quality of the patient

The combination approach with the 3 pillar strategy of antihomotoxic treatment of arthrosis will create:

• A “cleansing of the terrain”. The extra-cellular environment will be cleared to ameliorate transport of nutrients, mediators, etc. but also to facilitate the elimination of metabolic byproducts and other possible toxins.
• The inflammation process is regulated in such a way that the physiological characteristics remain, but the destructive and negative aspects are reduced to a minimum. Through such regulation we eliminate the causal homotoxins but inhibit the symptomatology.
• The tissue that is undergoing damage is supported. Conditions are created in which better functioning synovial cells and chondrocytes are supported in their physiological functions. A better regenerative function of the cartilage will tend to inhibit the degenerative effects of the process of arthrosis and attempts to slow down or reduce the damaging process.
Although a direct correlation between the coordinates on the DET and a medication are in principle not possible, it has been shown with Zeel. Degenerative pathologies of the joint only appear on the table at the cavodermal degeneration phase, and Zeel is specifically used for these indications.

But Zeel alone will not be enough to solve the arthrotic condition.
The 3 Pillars of antihomotoxic Treatment of Arthrosis

1. Drainage and Detoxification: Detox-Kit
2. Immunomodulation: Traumeel
3. Specific cell and organ support: Zeel

We should start with a general cleanse of the ECM, not only at the level of the affected joint. For this, the Detox-Kit is the most appropriate therapy (see IAH AC Drainage and Detoxification).

In activated arthrosis, immunomodulation is accomplished with Traumeel. Many corrective effects of Traumeel are known, such as: Inhibition of the pro-inflammatory mediators IL-1, TNF-alpha and IL-8 just to mention a few. Traumeel will also increase the release of TGF-beta by Treg cells (TH-3 cells), inhibiting by this mediator the pro-inflammatory TH-1 and TH-2 cells. Zeel T contains also a number of inflammation regulating plant extracts, as well as suis organ components that are related to the joint. In addition, essential catalysts present in the remedy will enhance the cellular oxygenation of synovial cells and chondrocytes. In those countries were Zeel T is not available, Zeel comp. can be complemented for cell and organ support by Coenzyme compositum, containing the main catalysts of the Krebs-cycle for enhancing the synovial cells and chondrocytes metabolic functions.
Abstract

- First 2 weeks: Traumeel + Zeel T injection (*) + oral use of Traumeel tablets. Inflammation prevails.
- Start Drainage and Detoxification from first day of therapy on (Detox-Kit)
- From the 3rd week on: Zeel T injection + oral Zeel T tablets.
- Local application of Traumeel ointment in the first 2 weeks, then ointment of Zeel.

(*) If Zeel T is not available in your market use Zeel comp. and add Coenzyme compositum

The summary of a therapy outline for the treatment of arthrosis should look like the one on the slide above. (Zeel T is not available in all countries. Furthermore, we should stipulate that this should be injected as a peri-articular injection or i.m or s.c. as ia application has been taken off the market for both Zeel T and Zeel comp N.)
Of course, in function of the accompanying pathology and symptoms presented by the patient, other preparations can be added on an individual basis.

Coenzyme compositum and Ubichinon compositum will facilitate the cellular energy. The medication contain respectively the catalysts and quinones needed for this purpose.

Dulcamara Homaccord is administered when aggravation of the rheumatic condition is weather related (worse in cold and humid weather, as is often seen in arthrosic patients).

The following homaccords have specific tropisms or topographic characteristics: Colocynthis-Homaccord in lumbar region, low back pain or lumbar spondylosis. Ferrum-Homaccord in periartthritis of the shoulder and shoulder-arm syndrome. Gelsemium-Homaccord in neck pain and occipital headache (whiplash)

Other medications can be added in function of the individual presenting conditions of each patient and are documented in specific antihomotoxic lectures.
• As a biological being man deserves a biological approach

Biologically therapy can offer a comprehensive treatment for osteoarthritis, including immune regulation and organ strengthening.

Through the addition of catalysts, which increase the energy formation in the cell, further support is awarded to the cartilage.

In the light of numerous adverse events reported with the use of conventional NSAID’s antihomotoxic treatment offers a viable alternative.