USE OF NON-PROHIBITED MEDICATION IN SPORTS

Dr. med. Maximilian Schindler & Dr. med. Philippe Tscholl



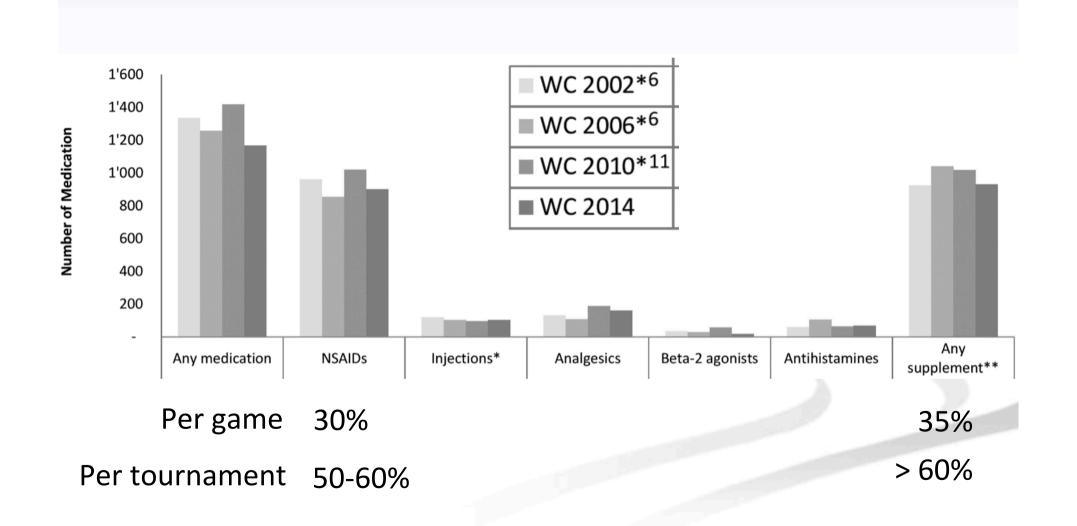






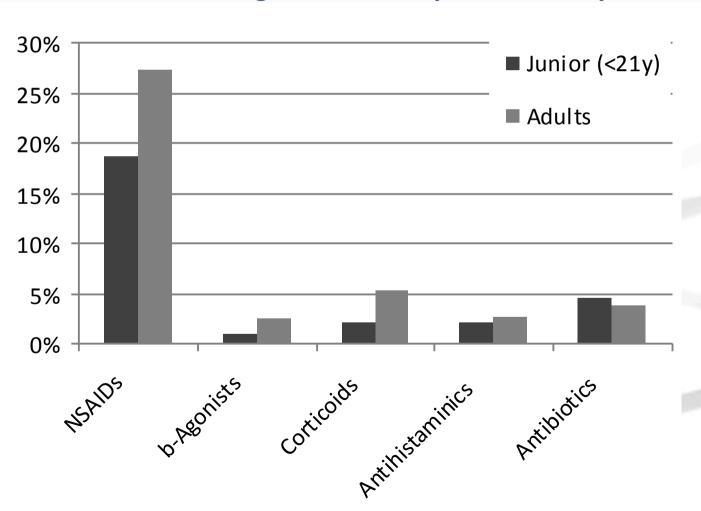


Use of medication in FIFA WC



The use of medication in elite athletes

% of athletes using medication prior to competition



Marathon – Bonn / El Andalus

47-61 % were using pain-medication (60% NSAIDs) 11% started with pain

> 30% were not aware of potential side-effects

Source

OTC 22%, friends 56%, physician 6%

Citations of professional footballers

"Prior to every match one, prior to competition two – sometimes more."

"I used to use pain medication like sweets."

Why are painkillers so frequently used?

Therapeutic use

Recreational use

Performance enhancement

Associated factors

Pubmed-based review

"The use of medication in professional athletes 72 h prior to competition".

- → 8 publications
 - 2003 2010, published in AJSM, BJSM, IJSM, CJSM
- \rightarrow 33'233 athletes

track & field, football, winter and summer olympics

Associated factors

Team physician

Origin

Age

Type of sport

Gender (f > m)

Associated factors

Team physician Type of sport

Origin Gender (f > m)

Age

2002 FIFA World Cup™

22 out of 23 were using pain medication throughout the championship

Allopurinol in 20% of the players of one team

Non-associated factors

Reported injuries

Team success

Starting formation, bench-players

In-vs. out- of competition

Why are painkillers so frequently used?

Therapeutic use

Recreational use

Performance enhancement

Medical indication

Making sports participation possible

Improving sports performance

Doping

NSAIDs – the largest group

Indication

analgesic, anti-inflammatory, antipyretic

Mode of action

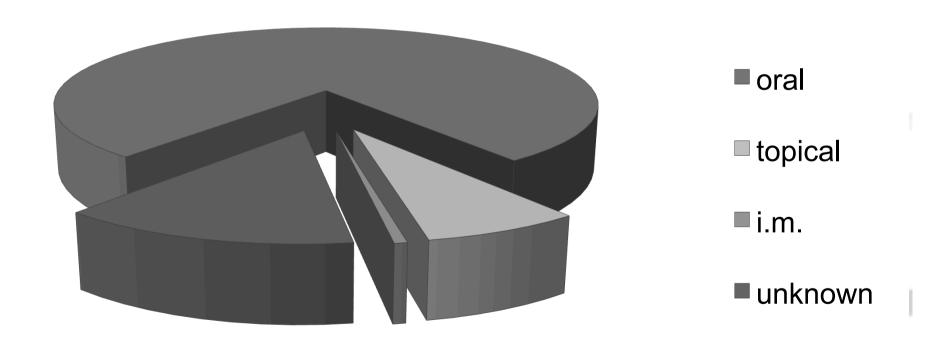
Inhibiting prostaglandin synthesis

Application

per oral, transdermal, intramuscular

Application

During FIFA World Championships



Local application

Diclofenac-Gel is effective in acute soft-tissue injuries.

10-20 fold concentration in soft-tissues than in synovial fluid and serum

Higher concentration in soft tissues (tendon, muscle periost) via patch than per oral application (less in bone and plasma).

NSAIDs – side effects

Dyspepsia in 20 %

Decreased renal blood flow

Hyponatremia in long extensive sports activities?

Potentially decreased respiratory function
In asthmatics

Non-steroidal anti-inflammatory drugs

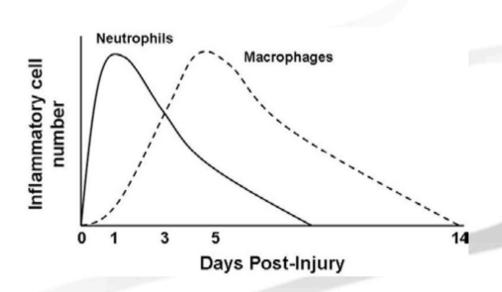
MUSCULAR TISSUE

Inflammatory process

Neutrophile Granulocytes

Additional tissue damage due to early inflammatory phase

Knock-out mice have no decreased tissue healing

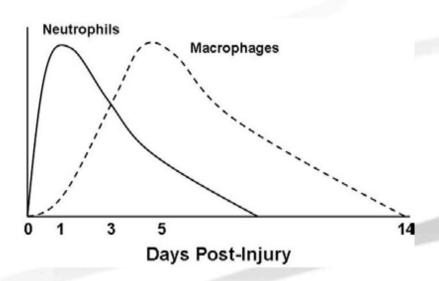


Inflammatory process

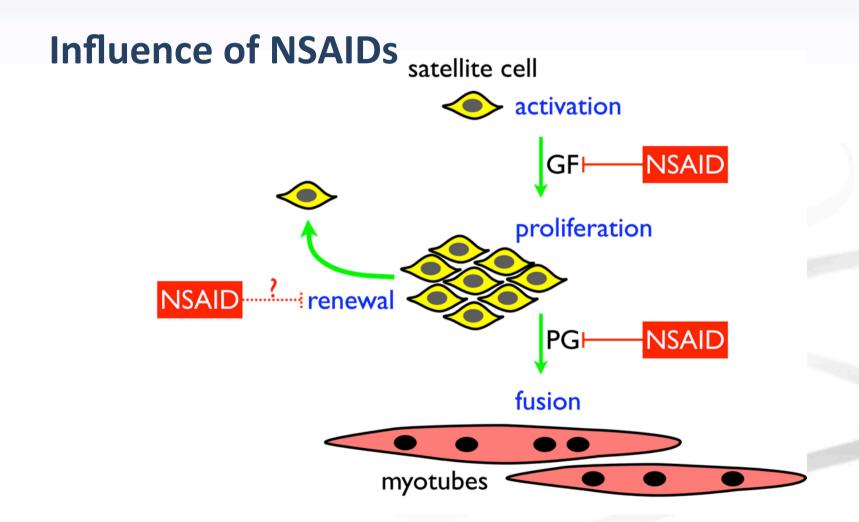
Macrophage cells

Phagocytosis of the cell damage

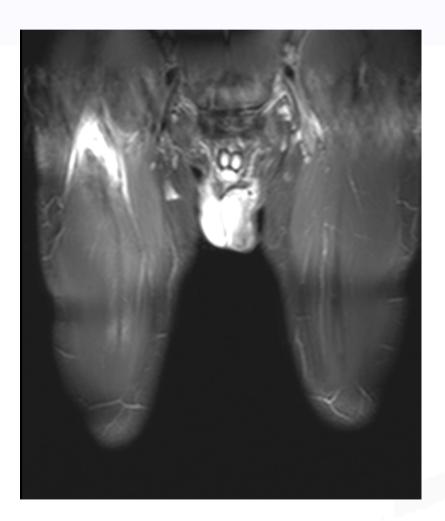
Macrophage cells stimulate IL-6, IGF-1 (which are both important for muscular adaptation to exercise) by producing PGE2 and radicals



Proliferation process



Clinical relevance



DOMS

→ earlier RTS with NSAIDs

Contusion

Intramuscular lesions

Myotendinous injury

Clinical relevance

Potential early benefit in structural muscle damage (eccentric work-out?)

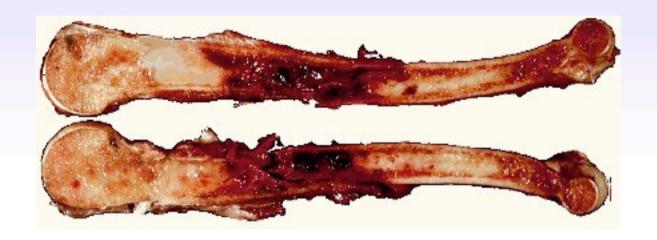
Decreased hypertrophic adaptation process 50-70%

1200mg Ibuprofen per day (no influence measured at 400mg daily)

Lower activity of satellite cells

Long-term intake with negative influence on muscle adaptation process (beneficial in older patients?)

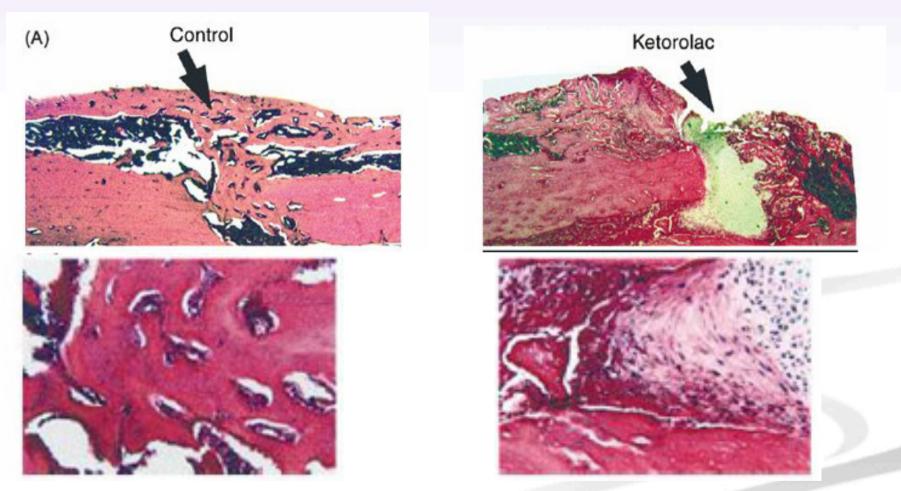
Ideal intake during 2 (-3) days



Non-steroidal anti-inflammatory drugs

Bone

Fracture healing



Also by inhibiting PGE pathway, which activates osteoblasts and osteoclasts

Fracture healing

Analgesic effect or side-effect?

Acetaminophen however has no influence on bone quality in fracture healing

Celecoxib has a dose-dependant interaction with decreased bone microstructure

Clinical relevance

Prophylactic use of Indomethacin (heterotopic ossification) lead to significantly higher non-union rates (26% vs 7%).

Same tendency in *retrospective* analysis of humeral fractures and spinal fusion.

Non-steroidal anti-inflammatory drugs

Tendon

Adaptation to exercise

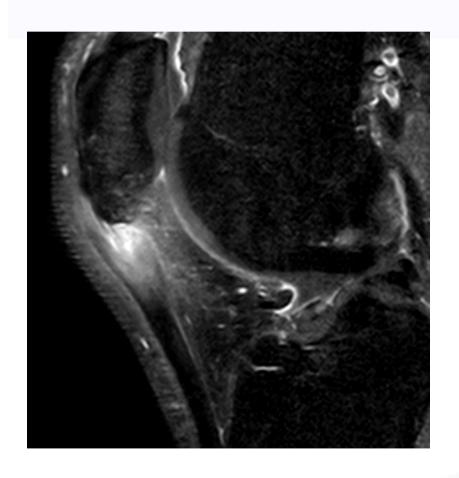
Inflammatory process is driven by PGE2, IGF, IL-6 but also PDGF

Influence of NSAIDs

Decreased blood flow of up to 30% (PGE2)

Decreased peritendinous collagen formation (after 3 days of NSAIDs)

Tendinopathy



(almost) no inflammatory cells

Rather a degenerative process

"poor inflammatory process may be the cause"

Clinical relevance

Adaptation to exercise

Inflammatory reaction after exercise is inhibited

Decreased growth (thickness)

Mechanical properties are identical

Any benefit?

Not in chronic tendinopathy

For acute pain and peritendinous fibrin production

Hand surgery

Non-steroidal anti-inflammatory drugs

Ligaments

Ligamentous injury

NSAIDs and COX-2

Less pain, earlier return, higher loads possible, BUT...

Decreased ROM, more swelling, more instability after 14 days.

Decreased mechanical properties (laxity); 32% less strength on MCL



The Dilemma

"There is inflammation without healing, but there is no healing without inflammation"

Non-steroidal anti-inflammatory drugs

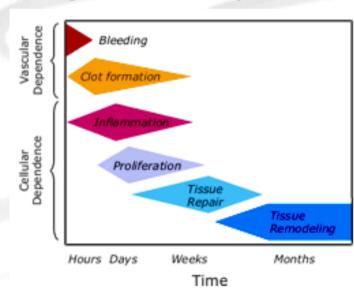
CONCLUSIONS

- Excessive use of medication, especially of NSAIDs
- → Athletes (and physicians) are not quite informed?
- NSAIDs are no purely pain-killing agents

Healing and adaptation to exercise might be compromised

No longer than 2-3 days

Should be avoided in early fracture healing





Dr. med. Philippe Tscholl & Maximilian Schindler









