

# Ibuprofen intake increases exercise time to exhaustion: A possible role for preventing exercise-induced fatigue

Lima F.D.

Stamm D.N.

Della Pace I.D.

Ribeiro L.R.

Rambo L.M.

Bresciani G.

Ferreira J.

Rossato M.F.

Silva M.A.

Pereira M.E.

Ineu R.P.

Santos A.R.

Bobinski F.

Figuera M.R.

Royes L.F.F.

Although the intake of nonsteroidal anti-inflammatory drugs (NSAIDs) intake by athletes prevents soreness, little is known concerning their role in exercise performance. This study assessed the effects of ibuprofen intake on an exhaustive protocol test after 6 weeks of swimming training in rats. Animals were divided into sedentary and training groups. After training, animals were subdivided into two subsets: saline or ibuprofen. Afterwards, three repeated swimming bouts were performed by the groups. Ibuprofen (15 mg/kg) was administered once a day. Pain measurements were performed and inflammatory and oxidative stress parameters were assayed in cerebral cortex and gastrocnemius muscle. Training, ibuprofen administration, or both combined ( $P < 0.05$ ;  $211 \pm 18s$ ,  $200 \pm 31s$ , and  $279 \pm 23s$ ) increased exercise time to exhaustion. Training decreased the

acetylcholinesterase (AChE) activity ( $P < 0.05$ ;  $149 \pm 11$ ) in cerebral cortex. Ibuprofen intake decreased the AChE activity after exhaustive protocol test in trained and sedentary rats ( $P < 0.05$ ;  $270 \pm 60$ ;  $171 \pm 38$ ; and  $273 \pm 29$ ). It also prevented neuronal tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) and interleukin (IL 1 $\beta$ ) increase. Fatigue elicited by this exhaustive protocol may involve disturbances of the central nervous system. Additive anti-inflammatory effects of exercise and ibuprofen intake support the hypothesis that this combination may constitute a more effective approach. In addition, ergogenic aids may be a useful means to prevent exercise-induced fatigue. © 2015 John Wiley & Sons A/S. Published by John Wiley & Sons Ltd

AChE activity

Exercise training

exhaustive exercise

inflammation

NSAIDs intake

acetylcholinesterase

ibuprofen

interleukin 1beta

nonsteroid antiinflammatory agent

reactive oxygen metabolite

tumor necrosis factor

animal

animal experiment

brain cortex

drug effects

endurance

fatigue

male

metabolism

nerve cell

oxidative stress

pain

pain measurement

physiology

protein carbonylation

randomization

rat

skeletal muscle

swimming

Wistar rat

Acetylcholinesterase

Animals

Anti-Inflammatory Agents, Non-Steroidal

Cerebral Cortex

Fatigue

Ibuprofen

Interleukin-1beta

Male

Muscle, Skeletal

Neurons

Oxidative Stress

Pain

Pain Measurement

Physical Conditioning, Animal

Physical Endurance

Protein Carbonylation

Random Allocation

Rats

Rats, Wistar

Reactive Oxygen Species

Swimming

Tumor Necrosis Factor-alpha