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ABSTRACT SUBMISSION – DEADLINE 10 May 2013

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| Title (up to 30 words, Arial, 11 pt, single line spaced, in sentence case. Like this: | Inflammation and resolution in exercise-induced skeletal muscle injury: The effect of NSAID treatment on pro-inflammatory and anti-inflammatory/pro-resolving lipid mediators |
| Authors (Underline the <u>presenting author</u>) | <u>J Markworth</u> , L Vella, K Maddipati & D Cameron-Smith |
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| Select Your Abstract Session Theme Category | 7 Clinical prescriptions for exercise in athletes – what are the key messages for prevention of illness and adequate recovery for athletic populations? |
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ISEI Abstract –

“Clinical prescriptions for exercise in athletes- What are the key messages for prevention of illness and adequate recovery for athletic populations. Session theme number – 7”

Inflammation and resolution in exercise-induced skeletal muscle injury: The effect of NSAID treatment on pro-inflammatory and anti-inflammatory/pro-resolving lipid mediators.

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ABSTRACT

Current approaches in the treatment of exercise-induced muscle injury rely on the inhibition of pro-inflammatory pathways to alleviate cardinal signs of inflammation; redness, swelling, heat and pain. However, recent research suggests that the cellular events which occur early in acute inflammation engage an active and coordinated inflammatory resolution program characterised by a switch from pro-inflammatory mediators to production of active pro-resolution factors that govern the withdrawal of inflammation whilst facilitating tissue healing. This led to the identification of novel classes of anti-inflammatory/pro-resolving lipid mediators, including the lipoxins (LX), resolvins (Rv), and protectins (P), which may provide new targets in the treatment of inflammation. METHODS: Sixteen untrained male subjects (age 23 ± 0.7 yr, mass 88 ± 3.1 kg) were assigned to a placebo (PLA) (n=8) or ibuprofen (IBU) (n=8) group. Subjects completed a single bout of resistance exercise consisting of 3 sets of 8-12 repetitions of a squat, leg press and leg extensions at 80% 1RM. Intravenous blood samples were obtained at rest, at 30 min intervals between 0 and 3 h and again at 24 h post exercise. The IBU group orally consumed 400mg of the non-steroidal anti-inflammatory drug (NSAID) ibuprofen pre-exercise, and again at 5 and 10 h post exercise (1200 mg/day). Serum lipid mediator profiles were analysed via LC-MS targeted lipidomics. RESULTS: Acute exercise increased serum levels of pro-inflammatory eicosanoid species derived from both the COX-1 and 2 (prostaglandins: e.g. PGF_{2α}, PGE₂, PGD₂, TXB₂) and 5-LOX (leukotrienes: e.g. LTB₄, LTB₅) pathways. Additionally, heightened circulating levels of novel pro-resolving lipid mediators derived from arachidonic acid (LXA₄ and LXB₄), EPA (RvE₁) and DHA (RvD₁ and PD₁ isomer) were detected post-exercise. Both the pro-inflammatory COX-1 & 2/5-LOX responses, as well as pro-resolving lipid mediator biosynthesis were blunted by the administration of the COX-1 and 2 inhibiting NSAID ibuprofen. CONCLUSION: Pro-inflammatory eicosanoids as well as novel pro-resolving bioactive lipid mediators are acutely up regulated following unaccustomed resistance exercise; a response which is diminished by IBU treatment. We hypothesize that the active resolution of exercise-induced inflammation may be important in effective post-exercise recovery and that a shift from anti-inflammatory interventions towards those which promote active resolution may hasten natural withdrawal of inflammation whilst facilitating the successful repair and regeneration of damaged muscle tissue.