HOW heatwave IMPROVES MUSCLE RECOVERY – THE SCIENCE

Transient Receptor Ion Potential channels (TRP or pronounced TRiP) are expressed in almost every cell type in both excitable and non-excitable tissues. TRP channels are present in all cellular membranes, with the exception of the nuclear envelope and mitochondria.

Most TRP channels are localized in the plasma membrane, where they have an essential role in the influx and/or transcellular machinery that transports Ca^{2+}, Mg^{2+} and trace metal ions, and they modulate the driving force for ion entry.

These contributions are essential for several physiological processes, ranging from pure sensory functions including nociception, homeostatic functions to many other motile functions, such as muscle contraction and vasomotor control.

TRP ion channels convert energy into action potentials in somatosensory nociceptors. There are 28 different TRP channels and each plays a different role. For instance, TRPM8 relates to mechanisms of sensing cold, TRPV1 contributes to heat and inflammation sensations, and TRPA1 facilitates many signaling pathways like sensory transduction, nociception and inflammation.

Recent studies indicate that TRPV1 & TRPA1 is activated by a number of reactive and non-reactive compounds and considered as a 'chemo-sensor' in the body. TRPA1 antagonists are effective in blocking pain behaviours induced by inflammation.

The perception of pain throughout the body arises when neural signals originating from the terminals of nociceptors are propagated to second-order neurons in the spinal cord or brainstem, whereupon they are transmitted to specific higher order brain areas. Recent studies have begun to elucidate some of the molecular mechanisms underlying the transduction of noxious stimuli. Many stimuli have been found to activate ion channels present on nociceptor terminals that act as molecular transducers to depolarize these neurons, thereby setting off nociceptive impulses along the pain pathways. In particular, inhibiting TRPV1 has been shown to have this therapeutic value.

TRPV1 receptors are found mainly in the nociceptive neurons of the peripheral nervous system, but they have also been described in many other tissues, including the central nervous system. TRPV1 is involved in the transmission and modulation of pain, as well as the integration of diverse painful stimuli.

Upon tissue damage and the consequent inflammation, a number of inflammatory mediators, such as various prostaglandins and bradykinin, are released. These agents increase the sensitivity of nociceptors to noxious stimuli. This manifests as an increased sensitivity to hyperalgesia in response to allodynia. Most sensitizing pro-inflammatory agents activate the phospholipase C pathway. Phosphorylation of TRPV1 by protein kinase C have been shown to play a role in sensitization of TRPV1.

Upon prolonged exposure to **heatwave**, TRPV1 activity decreases, a phenomenon called *desensitization*. Extracellular calcium ions are required for this phenomenon; thus, influx of calcium and the consequential increase of intracellular calcium mediate this effect, leading to alleviation of pain via the subsequent decrease in the TRPV1 mediated release of inflammatory molecules following exposures to the stimuli ingredients in **heatwave**.

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