Main title – The Effect of Ultraviolet B Radiation on Pain Modulation in Healthy Humans Student researchers: Matthew Carabetta, Alexander Sherlock, Madeleine O'keefe, Angeline Amrutha Rai

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## **Summary**

Past research has shown that chemical, thermal and electrical stimulation has generated ipsilateral forehead analgesia (on the same side as the condition) to the side of the painful stimulus in healthy participants. This is a sign of inhibitory pain controls descending from the central nervous system. In some of these studies, this was accompanied by enhanced excitability of the pain induced blink reflex. The inhibitory pain modulation is associated with areas of the brain projecting into the spinal cord to inhibit nociceptive, pain signals entering the spinal cord. In order to better understand pain processing in chronic pain sufferers we temporarily simulated limb pain which is inherent in many chronic pain syndromes. The aim of this experiment was to investigate the effect of ultraviolet B (UVB) exposure on pain processing in healthy volunteers. In this study we were interested in investigating the effects of pain mechanisms affecting areas of the body remote from the site of UVB exposure including the secondary site (2-3 cm from the site of primary UVB exposure), the contralateral arm and both sides of the forehead. Of particular interest was whether the same ipsilateral analgesia would present for UVB as it did during other superficial pain experiments.

Each of the 20 participants were tested during two sessions. The first session tested for baseline measures and was followed by UVB exposure. The two testing sessions were performed 24 hours apart to allow for the UVB to reach peak inflammation. At both sessions participants underwent psychophysical tests involving superficial mechanical, thermal and electrical stimulation (which was paired with startling acoustic stimuli). Participants were instructed in using a numerical pain scale to report pain and sharpness intensity. This pain scale was a rating of pain scored between 0 being 'no pain' and 10 being 'extreme pain'. Testing was done at designated test sites of the forearms and on each side of the forehead. The psychophysical tests quantified sensitivity to pain, sharpness and heat which was tested using a Von Frey monofilament (a semi-rigid nylon bristle) and a Neuropen pin. Pressure pain was measured using an electronic algometer with a broad tip, pushed, perpendicular to the testing sites until the participant reported pain. Heat pain was measured with a heat lamp held against the participants forearm for seven seconds at 44 degrees Celsius.

The nociceptive blink reflex to a supraorbital electrical stimuli was also employed and measured blink reflex integrated amplitude (physical extent) during both sessions to assess the effect of the UVB on this protective reflex. The electrical stimulus was delivered using concentric electrodes on each side of the forehead and the onset of the blink, from stimulus, was detected with surface electrodes attached under the lower eyelids, on the orbit. Electromyography signals were sampled and the blink reflex analysed for changes before and after UVB. In addition, earphones that delivered an acoustic startle stimulus simultaneously with the blinks was incorporated to strengthen the reflex. Participants also reported pain and sharpness ratings (described above) to the electrical stimulus.

Results of this study showed that two times the MED of UVB does not produce ipsilateral forehead analgesia in healthy participants after 24 hours; but rather hyperalgesia. Hyperalgesia in the affected forearm was also detected superficially following UVB and trends were observed that extended to the forehead. This implies activation of facilitatory controls that modulate pain. This finding was in parallel with an inhibitory effect on the R2 integrated amplitude of blink reflex ipsilateral to UVB conditioned forearm. These findings support previous findings of independent facilitatory and inhibitory controls. Additionally, this study supports the awareness that different experimental pain models are necessary in determining distinct pain modulation

processes. This distinction is particularly evident between the variety of results achieved from differing nociceptive stimuli and the strengths of their respective doses (UVB, HFS and Capsaicin). Which further supports research evidence highlighting differences in pain processing between deep tissue and cutaneous nociception. However, future research using a unilateral UVB is encouraged as a non-invasive, effective method for studying pain processing